



Whole Genome Sequencing Test Report

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Letter to Customers

Dear Clients,

Hello!

First of all, we would like to thank you from the bottom of our hearts for your trust in us. It is our pleasure to inform you that your customized personal genomic analysis has been successfully completed. This is not only an in-depth decoding of your life information, but also a new milestone in your personal identity. On this basis, we further analyze your personal genomic information, complete the detection and evaluation report of hereditary diseases, and form relevant health management guidance recommendations. We look forward to helping you understand the meaning of life and health at a higher level and improve your quality of life.

In order to ensure that you can fully understand and be satisfied with our services, we hereby remind you to read the following important information carefully to ensure an accurate understanding of our services:

Our genetic testing services rely on the current world's leading high-throughput sequencing technology, as well as the most advanced bioinformatics analysis technology system we have mastered.

We provide personal genomic outcome test reports designed to:

Assist in analysing whether an individual is at risk for hereditary diseases.

Provide a reference for assessing whether an individual is in a disease carrier state.

Assists in predicting whether a family member or family member is at risk of a certain genetic disease and whether they are carriers of the same type of disease.

Please note that personal genomic information is highly sensitive personal privacy. We promise that we will keep this information safe and promise that we will not use it for any other purpose without your authorization. If the information is leaked due to personal reasons and all the consequences that may arise (including but not limited to possible social discrimination), you will be responsible for it. The original text of the genetic test report is in English. In order to facilitate reading and understanding, we provide VIP customers with a Chinese translation version. Please note that the translated version is for comprehension purposes only and all matters not covered shall be based on the original English text.

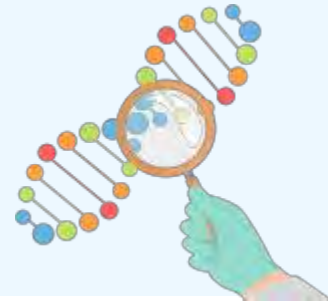
In addition, genetic disease assessment reports based on the analysis of personal genomic information may cause psychological stress or other mental burdens on you. Please understand that all such potential impacts will be borne by you and that our company is only obliged to inform.

It is important to emphasize that this report should not be used by employers or insurance companies to force you to use it as a reference for career choices and insurance. Under Canada's Genetic Nondiscrimination Act (GNA), the use of an individual's genetic test results as a basis for decision-making in areas such as educational preference selection, employment, insurance, etc. is prohibited.

Finally, we promise that the personal genomic testing services we provide will always be at the forefront of scientific and technological development, whether it is in the technological advancement of sequencing platforms or the application and transformation of the world's biological genomics scientific research results, we will remain at the forefront. We will do our best to help you analyze, evaluate and solve various process problems that may be encountered due to the development of biogenetic science research and sequencing technology.

Thank you again for your trust and support. Wishing you and your family good health!

Sincerely,
Ebovir Team



Please log in to the ebogenes.com in time and make an appointment on the official website for our follow-up online or offline professional report interpretation.



EBOGENES

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Ancestry Analysis

4 Reports

My Ancestry

Ancestry Composition	63.58% Northern Han Chinese
Paternal Haplogroup	Unknown
Maternal Haplogroup	Z3
Neanderthal Ancestry	3.313 %

Ancestry Analysis

Everybody's own unique ancestral history is encrypted in their DNA. The following WE report helps to decode your story by analyzing hundreds of thousands of your autosomal polymorphisms.

My Ancestry Composition

Chinese	94.08%
● Northern Han Chinese	63.58%
● Mongolian	30.20%
● She	0.28%
● Others	0.02%
Northeast Asian	2.91%
● Korean	2.73%
● Yakut	0.17%
● Others	0.01%
Central Asian	1.57%
● Kyrgyz	1.46%
● Others	0.11%
European	0.83%
● English	0.83%
American	0.57%
● Inuit	0.56%
● Others	0.01%
Others	0.04%

My Ancestry Timeline

Genomic information allows us to know our ethnic makeup and to better understand ourselves from the ancestral level. However, apart from the specific ethnic components in our DNA, we may also want to know when these genes were passed on to us. WE estimates the approximate time each ancestral component was mixed into your genome to help interpret how the ancestral footprints have shaped you.

In general, homologous recombination during human reproduction makes the above calculations very difficult. Thus, we introduce two presuppositions:

1. Each ancestral component is derived from a single ancestor with pure ancestral makeup.
2. On average, each generation passes ~50% of their genetic materials to the next generation.

1 - 2 Generation	1970 — 1945	Northern Han Chinese
2 - 3 Generation	1945 — 1920	Mongolian
6 - 7 Generation	1845 — 1820	Korean
7 - 8 Generation	1820 — 1795	English
7 - 8 Generation	1820 — 1795	Kyrgyz
8 - 9 Generation	1795 — 1770	Inuit

The Basics of Ancestry Analysis

Genetic characteristics are passed on to successive generations via DNA replication. Human gametes are produced by a process called meiosis, during which homologous recombination occurs. This leads to the random exchange of genetic materials between two strands of DNA inherited from each parent. This complex process not only enables parental DNA to be passed on to offspring, but also generates mutations

to ensure genetic diversity in a population. Single nucleotide polymorphisms (SNPs) are the most common among the mutations. These SNPs have a strong ethnic specificity and can be used to reflect the genetic characteristics of a population.

Method of Ancestry Analysis

WE's supervised machine learning algorithm is based on the Admixture ancestor analysis tool developed by the University of California, Los Angeles (UCLA). The algorithm compares your autosomal DNA information with 42 reference populations in the WE database, and quantifies the similarities between your genome and the 42 reference DNA.

Paternal Y-DNA haplogroup

Molecular anthropologists have established a haplogroup tree of human populations based on particular variants of the Y chromosome, which is passed on from fathers to son. WE helps to trace your paternal origin, evolution and migration history according to your Y-DNA haplogroup inference.

My Paternal Haplogroup

Unknown

Why Is It Unknown?

The paternal lineage is based on analysis of the Y chromosome, which only exists in males and is strictly passed from father to son. As females do not have the Y chromosome, female users would not have a paternal haplogroup result.

You can link the paternal haplogroup results from your father, brother or other direct male relatives in your family if they have their DNA tested.

Note for female users: The Y chromosome of your son is inherited from his own father, not your father.

The Basics of Paternal Lineage Analysis

The Y Chromosome (Chr) is the sex-determining chromosome in humans - only males have the Y Chr and it's passed from father to son. The Y Chr generally does not undergo homologous recombination and is inherited almost identically between father and son. But due to copying errors during DNA replication, roughly 2 mutations in the Y Chr is produced per generation. Such Y-chromosomal mutations accumulate in the evolutionary history of human populations for tens of thousands of years, and are used by molecular anthropologists to infer paternal lineages.

Studies have shown that modern humans originate from Africa. According to recent estimates, the common ancestor of all males - the Y-chromosomal Adam - appeared in Africa about 236,000 years ago (or 275,000 years), while the most recent common ancestry of Eurasia males has been traced back to about 69,000 years ago.

Based on the Y Chr data of different populations, molecular anthropologists have established a tree-like Y-DNA haplogroup map. Each branch of the tree corresponds to a haplogroup where Y chromosomes contain

similar mutations. WE can accurately predict your Y-DNA haplogroup through the comparison between your DNA test results and the reference Y-DNA haplogroup tree. This allows us to trace the origin, evolution and migration history of your paternal lineage.

Method of Paternal Lineage Analysis

Referring to the Y-DNA haplogroup tree published by ISOGG, WE developed a method which can predict Y-DNA haplogroup, quickly and accurately. Specifically, by comparing the Y Chromosome test result to each haplogroup on the tree, probability scores are evaluated, and the highest scored haplogroup will be assigned as the best matching Y-DNA haplogroup for the user. After continuous optimization of the scoring system, WE now offers highly accurate Y-DNA haplogroup results and details of all detected variants on the ISOGG reference tree, which can be accessed conveniently.

Maternal MT-DNA haplogroup

Molecular anthropologists have established a human mitochondrial DNA (MT-DNA) haplogroup tree based on particular variants of the MT-DNA, which is generally only passed from mother to offspring. WE helps to trace your maternal origin, evolution and migration history according to your MT-DNA haplogroup inference.

My Maternal Haplogroup

Z3

Brief Introduction to My Haplogroup



About My Haplogroup

Haplogroup Z, sister group of Haplogroup C, D, G, subclade of Haplogroup M



Time of Origin

45,000 Years Ago



Mainly Distributed Areas

North Europe, Russia and East Asia



Representative Populations

Chinese, Korean and Saami

Migratory Track



Principles of Maternal Lineage Analysis

MT-DNA is a kind of unique circular DNA outside the nucleus. Unlike the inheritance of the Y Chr of paternal lineage, the MT-DNA is generally only from the egg provided by the mother. The MT-DNA does not undergo homologous recombination and is inherited almost identically between the offspring and his/her mother. The MT-DNA is a circular DNA resembling prokaryotic genes that mutates more frequently than the Y Chr during replication. Such mitochondrial mutations accumulate in the evolutionary history of the human populations for tens of thousands of years, and are used by molecular anthropologists to infer maternal lineages.

Studies have shown that modern humans originate from Africa. According to the maternal lineage estimations, the most recent common ancestor - the mitochondrial Eve - appeared in Africa about 200,000 years ago.

Based on MT data of different populations, molecular anthropologists have established a tree-like MT-DNA haplogroup map. Each branch of the tree represents a MT haplogroup with similar mutations. WE can accurately predict your MT-DNA haplogroup through comparison between your DNA test results and the reference MT-DNA haplogroup tree, so as to trace the origin, evolution and migration history of your maternal lineage.

Method of Maternal Lineage Analysis

WE refers to the MT haplotype typing method published by the Chinese Academy of Sciences to predict users' MT-DNA haplogroup. Specifically, by comparing the user's MT-DNA result to each haplogroup on the MT-DNA haplogroup tree published by phylotree, probability scores are evaluated, and the highest scored haplogroup will be assigned as the best matching MT-DNA haplogroup for the user. WE now offers highly accurate MT-DNA haplogroup results and details of all detected variants on the phylotree reference tree, which can be accessed conveniently.

Neanderthal Variants Percentage

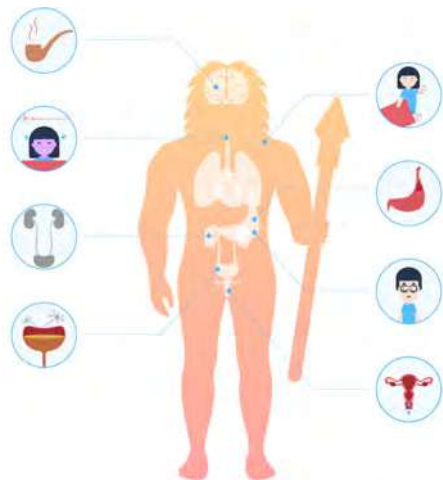
The Neanderthals are a group of extinct ancient humans, but they have passed on genetic information through gene flow with ancestors of modern humans. WE attempts to explore the secrets of human origin by analyzing the percentage of Neanderthal variants in your genome.

My percentage of Neanderthal variants is

3.313 %

100% of WE users are similar to me

Health risks possibly related to Neanderthal variants



Tobacco Use Disorder

Negative

It is a neuropsychiatric disease, which is also known as tobacco dependence. When smokers become addicted to nicotine, their physical manifestations are increased tolerance and withdrawal symptoms, and their behavior is manifested as loss of control.

Protein-Calorie Malnutrition

Negative

It is a nutritional deficiency disease caused by insufficient food supply or diseases. Wasting and kwashiorkor syndrome are the main clinical manifestations. Weight loss is due to the long-term lack of calories, protein and other nutrients in the diet, or there is malfunction in patient's digestion, absorption and utilization of food.

Functional Disorders of Bladder

Not Detected

It is urinary dysfunctions caused by damage to the central or peripheral nerves that control urination. It is a relatively common disease and is sometimes misdiagnosed as urinary tract infection in clinical practice.

Symptoms Involving Urinary System

Negative

Diseases can occur in various organs of the urinary system (kidneys, ureters, bladder, urethra) and affect the entire system. It can be caused by pathological changes in other systems of the body, and can also affect other systems or even the whole body.

Stress Urinary Incontinence in Female

Negative

It refers to the involuntary leakage of urine from the external urethra when the abdominal pressure increases due to sneezing or coughing in women. The major symptom is involuntary urination when abdominal pressure increases, such as during coughing, sneezing, and laughing. Body signs include increased abdominal pressure, observable involuntary outflow of urine from the urethra.

Obstructive Sleep Apnea

Negative

It is characterized by recurrent episodes of complete or partial obstruction of the upper airway leading to reduced or absent breathing during sleep. The airflow through the mouth and nose may stop for 10 seconds or more, and is accompanied by decreased blood oxygen saturation during each episode. There can be 30 or more episodes during a 7-hour sleep in an adult at night.

Sleep Related Movement Disorders

Negative

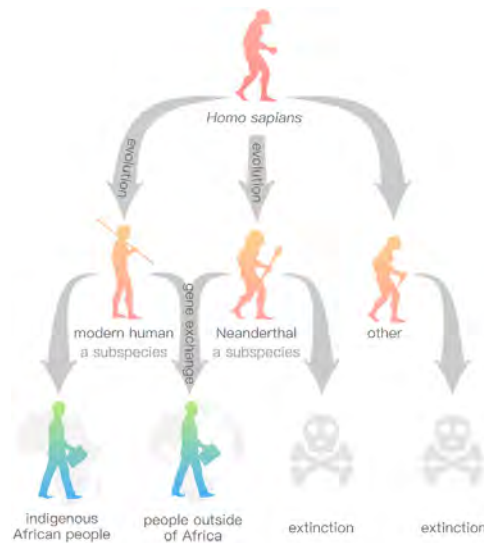
It is characterized by excessive limb movement when a person is about to fall asleep. Physical activity of the limbs usually will not affect sleep, but if this occurs too frequently, it may lead to periodic limb twitch disorder, which in turn leads to insomnia.

Gastroparesis

Negative

It is also known as paralysis or weakness of the stomach, which is a group of clinical symptoms characterized by delayed gastric emptying. Main symptoms include early satiety, epigastric postprandial fullness, nausea, episodic retching, vomiting, weight loss, etc.

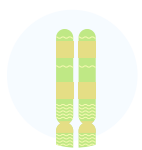
Who Were the Neanderthals?



Based on current anthropological research, both modern humans and the Neanderthals are subspecies of *Homo sapiens*.

Studies show that the Neanderthals and modern humans outside of Africa share 1%~4% of their total genomes. Scientists believe that about 100,000 to 50,000 years ago, one branch of the modern humans came out of Africa and mixed with the Neanderthals in the Middle East. The mixed descendants are scattered throughout Europe, Asia, the Americas, and Oceania. But the modern Africans don't have any Neanderthal genes, because their ancestors stayed in Africa.

Genes Tested



Nicotine Dependence: SLC6A11

rs901033, where the non-neanderthal allele is C and the Neanderthal allele is T. Studies found that individuals with the T allele had an increased risk of nicotine dependence.



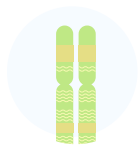
Protein-Caloric Malnutrition: SLC35F3

rs12049593, where the non-neanderthal allele is C, and the Neanderthal allele is T. Studies have found that individuals with the T allele have an increased risk of protein-caloric malnutrition.



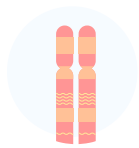
Bladder Dysfunction: DAB1

rs17115796, where the non-neanderthal allele is T, and the Neanderthal allele is C. Studies have found that individuals with the C allele have an increased risk of bladder dysfunction.



Urinary Symptoms: STIM1

rs11030043, where the non-neanderthal allele is A, and the Neanderthal allele is G. Studies have found that individuals with the G allele have an increased risk of urinary symptoms.



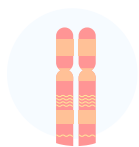
Obstructive Sleep Apnea: PIK3C2G

rs7133666, where the non-neanderthal allele is C, and the Neanderthal allele is A. Studies found that individuals with the A allele have an increased risk of obstructive sleep apnea.



Sleep Limb Hyperactivity Disorder: PKP4

rs3771635, where the non-neanderthal allele is T and the Neanderthal allele is C. Studies found that individuals with the C allele have a reduced risk of developing sleep hyperactivity disorder.



Gastroparesis: SOX5

rs4963700, where the non-neanderthal allele is T and the Neanderthal allele is C. Studies found that individuals with the C allele have an increased risk of gastroparesis.

Details of Variants

Report	Gene loci	My Gene	Description
Tobacco Use Disorder	RS901033	CC	no rise in the risk of nicotine dependence
Protein-Calorie Malnutrition	RS12049593	CC	no rise in the risk of protein-calorie deficiency malnutrition
Functional Disorders of Bladder	RS17115796	CT	higher risk of bladder dysfunction
Symptoms Involving Urinary System	RS11030043	AA	no rising in the risk of urinary symptoms
Stress Urinary Incontinence in Female	RS17766531	TT	no rising in the risk of stress urinary incontinence in female

Method of Neanderthal Variants Analysis

WE uses AdmixTools, the ancestry analysis toolkit developed by Harvard University, to calculate your Neanderthal genetic composition by comparing your genome sequences with the Neanderthal sequences.



Sport Genes

17 Reports



Basal Metabolism

Total Calorie Intake Tendency	Slightly Low
Total Carbohydrate Intake	Moderate
Saturated Fat Sensitivity	Slightly Low
Carbohydrate Sensitivity	Moderate
Caffeine Sensitivity	Low



Physical Characteristics

Explosive Power	Moderate
Endurance	Moderate



Weight Management

Influences of Exercise on Weight	Moderate
Influences of Diet on Weight	Slightly Small



Exercise Reaction

Heart Rate Improvement	Slightly Large
Maximal Oxygen Uptake (VO2max) Improvement	Moderate
Motivation to Exercise	Slightly Low
Muscle Endurance Improvement from Exercise	Large
Temperature Increasing Rate During Exercise	Slow

 Sport Protection

Anterior Cruciate Ligament (ACL) Injury Risk	Slightly Low
Achilles Tendon Injury Risk	Slightly Low
Lumbar Disc Injury Risk	Moderate

Caffeine Sensitivity

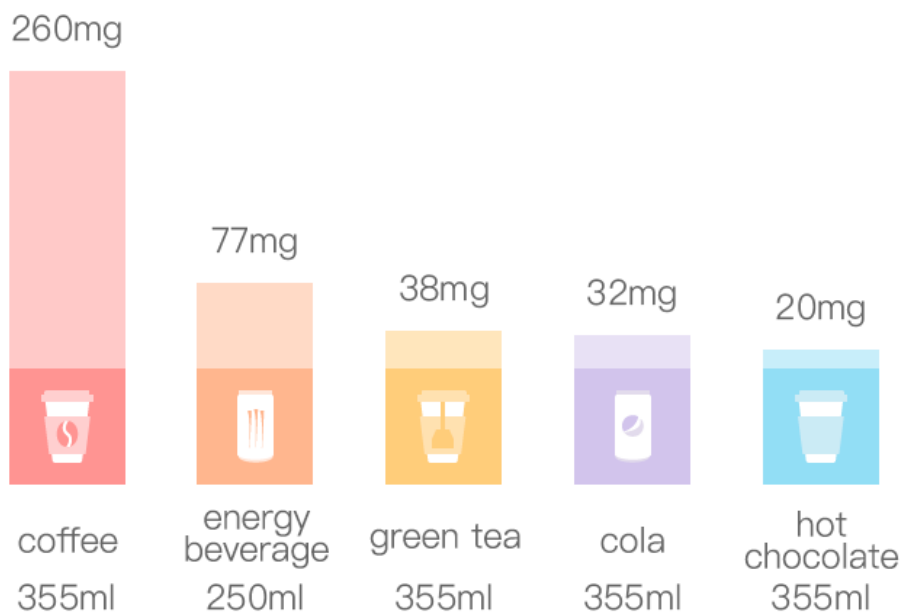
Caffeine sensitivity is related to the speed of caffeine metabolism. People with higher sensitivity should be more careful to prevent excessive caffeine consumption.

My Caffeine Sensitivity

Low

38.9% of We users are similar to me

Graph of caffeine content in common beverages



Knowledge

Metabolic profile

Caffeine is a xanthine alkaloid compound that acts as a central nervous stimulant. It can increase the activity of neurons in multiple brain regions by promoting the release of norepinephrine.

Caffeine sensitivity is related to the rate of caffeine metabolism. Caffeine is mainly oxidized in the liver by the cytochrome oxidase system. The *CYP1A2* gene is responsible for removing caffeine from the body. The high sensitivity of caffeine means that caffeine has a slower metabolic rate in the body of such people, and it stays in the body for a longer time.

Control caffeine intake

Most energy drinks and sports supplements contain caffeine, because caffeine can increase nerve excitability and bring better exercise performance. However, it should be noted that some studies have shown that because the sudden increase of adrenaline secretion will accelerate the heart rate and increase blood pressure, people with higher caffeine sensitivity will also have a greater risk of myocardial infarction after intaking excessive caffeine intake.

Therefore, the higher the sensitivity, the more important it is to control the intake of energy drinks to prevent the discomfort caused by large amounts of caffeine in the body for a long time.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS762551	CYP1A2	AA	not very sensitive to caffeine

Test details

Applicable situation

The caffeine sensitivity item uses a polymorphic locus on the *CYP1A2* gene: rs762551.

Notification

1. The test is mainly based on the research of Caucasian, and other populations are only for reference.
2. The test may not cover all genes or loci that related to the caffeine sensitivity.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all

users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

The test only evaluates your caffeine sensitivity from the genetic level, and does not include other factors such as age, liver function, dietary habits and so on. Therefore, the test results may not be equal to your actual caffeine sensitivity.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci related to caffeine sensitivity.

Carbohydrate Sensitivity

Carbohydrate sensitivity can be simply understood as the amount of calories the body gets from carbohydrates.

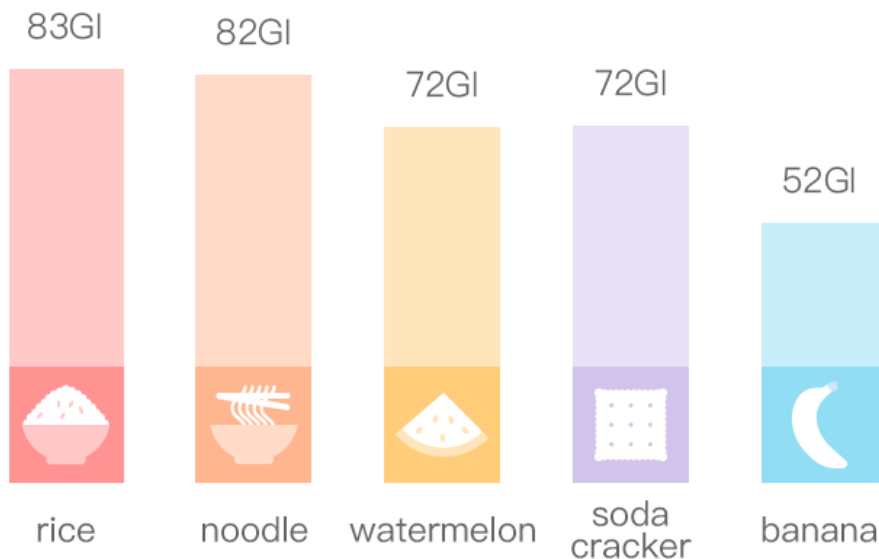
My Carbohydrate Sensitivity

Moderate

28.8% of We users are similar to me

Common foods with a high glycemic index

glycemic index: a 0–100 value used to measure how much one specific food increases blood glucose level. It represents the relative speed and capacity of rising blood glucose level, compared to glucose (a score of 100), after consuming that food.



Knowledge



Carbohydrates and blood sugar regulation

After entering the human body, carbohydrates from food are digested and decomposed into Monosaccharides to enter the blood circulation, which will raise blood sugar levels. At this time, the body regulates the secretion of insulin to lower the blood sugar levels in the body.

What is Glycemic Index (GI)

After different foods enter the gastrointestinal tract, the amount and speed of the final products such as glucose entering the blood are also different due to the difference digestion speed and absorption degree, so they have different effects on blood glucose level. Glycemic index (GI) is used to measure the impact of food containing the same amount of carbohydrates on blood glucose concentration. Generally, we call food with glycemic index greater than 70 as high GI food and food with glycemic index less than 55 as low GI food.

GI and diseases

If you eat a lot of high GI food for a long time, you will need to secrete a large amount of insulin regularly. After a certain period of time accumulation, human cells may become resistant to insulin. The consequence is that they can no longer effectively reduce blood sugar levels, leading to a series of problems such as obesity, diabetes, fatty liver and so on.

Therefore, people with high carbohydrate sensitivity should not only control their carbohydrate intake, but also choose foods with low glycemic index as much as possible.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS12255372	TCF7L2	GG	not very sensitive to carbohydrates
RS1801282	PPARG	CC	high carbohydrate sensitivity
RS4402960	IGF2BP2	GT	relatively sensitive to carbohydrates
RS5219	KCNJ11	CT	relatively sensitive to carbohydrates
RS7903146	TCF7L2	CC	not very sensitive to carbohydrates

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The carbohydrate sensitivity item uses six polymorphic loci on *TCF7L2* and *IGF2BP2* genes: rs5219, rs9939609, rs1801282, rs12255372, and rs4402960.

 Notification

1. The test is mainly based on the research of Caucasian and Asian, and other populations are only for reference.
2. The test does not cover all genes or loci that related to the carbohydrate sensitivity.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

 Notification

The test only evaluates your carbohydrate sensitivity from the genetic level, and does not include other factors such as family history of diabetes, regular exercise and so on. Therefore, the test results may not be equal to your actual carbohydrate sensitivity.

 Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

 Limit of detection

The test may not include all loci related to carbohydrate sensitivity.

Saturated Fat Sensitivity

Higher saturated fat sensitivity means that consumption of the same amount of saturated fat results in higher energy, so attention must be paid to the proportion of saturated fat in the daily diet.

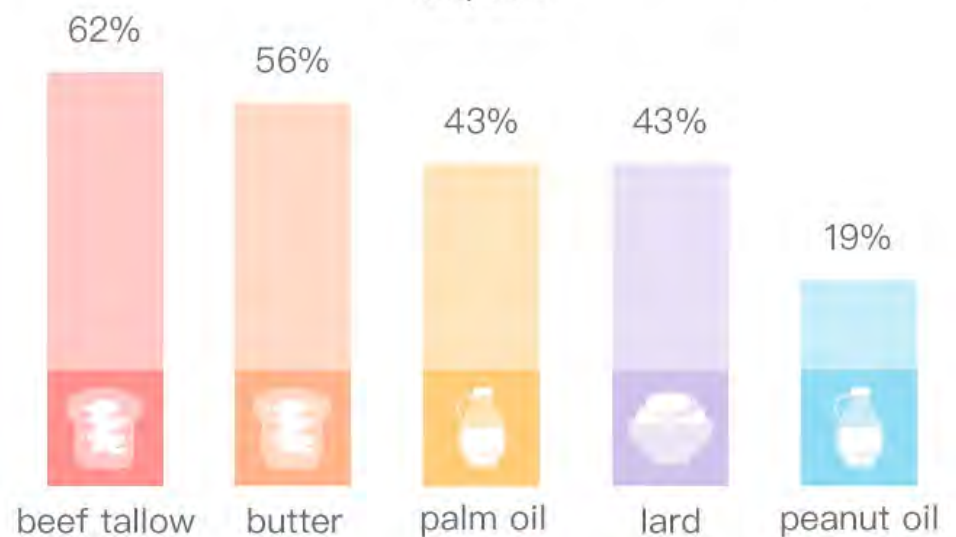
My Saturated Fat Sensitivity

Slightly Low

10.6% of We users are similar to me

Common edible oils rich in saturated fatty acids

saturated fat is made of saturated fatty acids and glycerol
the mass of saturated fat increases with the mass of saturated fatty acid



Knowledge

What is saturated fat

Fat is one of the important sources of human energy, It can provide essential fatty acids (fatty acids that can only be ingested through food but cannot be synthesized by itself, such as linoleic acid),

which is conducive to the digestion and absorption of fat-soluble vitamins. Fat is composed of glycerin and fatty acids. According to the chemical structure, fatty acids without unsaturated bonds are called saturated fatty acids, and the corresponding fat is saturated fat.

Problems caused by excessive saturated fat

In common foods, butter, cheese and animal oil contain more saturated fat. The human need to intake a certain amount of saturated fat to maintain health. However, the intake of saturated fat will lead to the increase of low-density lipoprotein (easier to stick to the inner wall of blood vessels and increase the risk of vascular obstruction). Therefore, excessive intake of saturated fat may lead to obesity, hypertension, atherosclerosis, etc.

Recommended intake

The World Health Organization recommends that the intake of fat should account for less than 30% of the total energy, and the intake of fat should be changed from saturated fat to unsaturated fat as much as possible. People with high sensitivity to saturated fats should strictly control the content of saturated fatty acids in their food if they need to reduce fat.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1799986	LRP1	CC	not very sensitive to saturated fats
RS1800629	TNF, LTA	GG	not very sensitive to saturated fats
RS328	LPL	CG	relatively sensitive to saturated fats
RS5082	APOA2	AA	not very sensitive to saturated fats

Test details

Applicable situation

The saturated fat sensitivity item uses four polymorphic loci on genes such as *TNF*, *LRP1* and *APOA2*: rs1799986, rs1800629, rs328, and rs5082.

Notification

1. The test is mainly based on the research of Caucasian and Asian, and other populations are only for reference.

2. The test may not cover all genes or loci that related to the saturated fat sensitivity.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

The test only evaluates your sensitivity to saturated fat from the genetic level, and does not include other factors. Therefore, the test results may not be equal to your actual sensitivity to saturated fat.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

The test may not include all loci related to saturated fat sensitivity.

Total Carbohydrate Intake

Those who have a greater tendency to consume carbohydrates need to pay more attention to controlling their daily intake of carbohydrates.

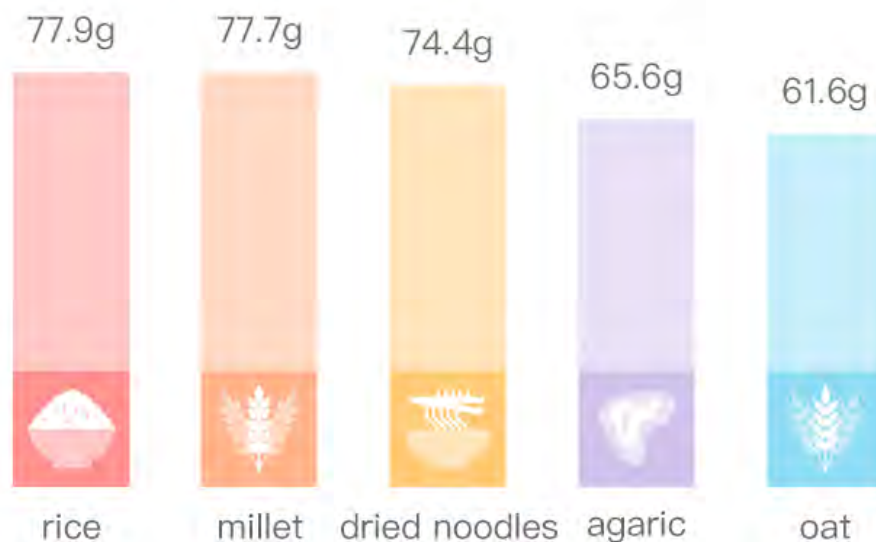
My Total Carbohydrate Intake

Moderate

67.1% of We users are similar to me

Common carbohydrate rich foods

carbohydrate content per 100g edible part



Knowledge

What are carbohydrates

In food science, carbohydrates refer to starch-rich foods (such as bread and cereals) or simple sugar foods (edible sugar). After entering the human body, carbohydrates can be stored in the form of glycogen, or they can be metabolized under aerobic or anaerobic conditions to produce energy, and they can also be converted into fat for storage in the body. They are the main energy source for

human daily life activities. The World Health Organization recommends that in a reasonable diet, the energy from carbohydrates should account for 55% to 65% of the total energy intake.

Recommended intake

The naturally occurring sugars in intact fruits and vegetables belong to endogenous sugars, while sucrose (white sugar, brown sugar), sweet additives (such as starch hydrolyzed products such as glucose syrup, maltose syrup, corn syrup, etc.), honey and concentrated fruit juices used in the food industry all belong to free sugars. Due to the great impact of free sugar on dental caries and cardiovascular diseases, the World Health Organization (WHO) recommends that the intake of free sugar should be limited to less than 10% of dietary energy intake.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS5082	APOA2	AA	inclined to consume more carbohydrates
RS35874116	TAS1R2	TT	no tendency to consume an increased amount of carbohydrates

Test details

Applicable situation

The carbohydrate intake tendency item uses two polymorphic loci on *APOA2* and *TAS1R2* genes: rs5082 and rs35874116.

Notification

1. The test is mainly based on the research of Caucasian, and other populations are only for reference.
2. The test may not cover all genes or loci that related to the tendency of carbohydrate intake.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all

users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

The test only evaluates your carbohydrate intake tendency from the genetic level, and does not include the influence of environment and other factors. Therefore, the test results may not be equal to your actual carbohydrate intake.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci related to carbohydrate intake tendency.

Total Calorie Intake Tendency

Human body consumes energy which can be measured by Calorie. The higher the total Calorie intake tendency, the greater the likelihood a person is to consume more food. If weight loss is required, this population must pay special attention to how much they eat and exercise can help with avoiding Calorie intake.

My Total Calorie Intake Tendency

Slightly Low

63% of We users are similar to me

Knowledge



Energy source

The carbohydrates, fats and proteins in food will be decomposed and absorbed in the human body to provide energy for normal activities.



BMI and obesity

If the energy intake from food is more than the energy consumed by daily life and exercise, the weight will increase over time. When the energy intake from food is less than the energy consumed by daily life and exercise, the weight will be reduced. The degree of body obesity can be roughly estimated by the body mass index (BMI for short, which is defined as the weight calculated in kilograms divided by the square of the height calculated in meters). The definition of overweight and obesity made by the World Health Organization for adults is: BMI ≥ 25 as overweight, and BMI ≥ 30 as obesity.



Recommended calorie intake

The dietary guidelines for Chinese residents recommend that adult women should intake 1800-2200 kcal per day, and adult men should intake 2200-2600 kcal per day. Studies have found that individuals with certain genotypes tend to intake higher energy in their daily diet, and their BMI index is often higher. Therefore, people with a higher tendency of total calorie intake should control their appetite more strictly if they want to reduce fat.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS5082	APOA2	AA	no tendency to consume an increased amount of calorie
RS9939609	FTO	TT	no tendency to consume an increased amount of calorie

Test details

Applicable situation

The total calorie intake tendency item uses two polymorphic loci on *FTO* and *APOA2* genes: rs9939609 and rs5082.

Notification

1. The test is mainly based on the research of Caucasian, and other populations are only for reference.
2. The test may not cover all genes or loci that related to the tendency of total calorie intake.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

The test only evaluates your total calorie intake tendency from the genetic level, and does not include the influence of environment and other factors. Therefore, the test results may not be equal to your actual calorie intake.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

The test may not include all loci related to total calorie intake tendency.

Endurance

Endurance refers to one's ability to conduct heavy physical activities in a relatively long period of time. Endurance is critical for sports like marathon or skiing.

My Endurance

Moderate

35.4% of We users are similar to me

Suggestions

- Strength of endurance is related to a variety of Genetic variation, your endurance is **Moderate**
- Your response to higher intensity Aerobic exercise training is ordinary and it is not recommended to focus on this type of training.
- In addition to targeted training, the following methods may help improve endurance:
 - Studies have shown that ingestion of certain **Caffeine** can improve performance in sports such as swimming.
 - Food rich in **Nitrate**, such as beetroot juice, can reduce the exercise oxygen consumption of 5.4%, extending running time about 15%.
 - Participating in meditation or yoga can reduce the level of pressure and improve endurance performance slightly.

*If you need additional dietary supplements, it is recommended that you seek help to authoritative health experts or clinicians.

Knowledge



What is endurance

Endurance refers to the endurance ability of the body to carry out intense physical activities for a long time. It is an important athletic ability. Endurance is especially important in marathon, skiing and other long-time and moderate-intensity endurance events. In addition to training and nutrition, genetics also play an important role in endurance performance.



Genes and endurance

Studies have found that gene polymorphism has a great influence on tolerance. For example, *ADRB2* encodes the adrenergic β_2 receptor, which is involved in the negative regulation of smooth muscle cell contraction, the positive regulation of skeletal muscle, and the positive regulation of muscle contraction and tube relaxation. Research shows that *ADRB2* may affect tolerance performance by mobilizing fat in fat cells and participating in energy metabolism.

Advice on exercise

Strong endurance is more likely to perform well in long-time and moderate-intensity endurance sports. When you exercise, you will benefit more from moderate-intensity aerobic exercise (such as aerobic monotherapy, etc.).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1042713	ADRB2	GG	not significantly helpful for endurance performance
RS17602729	AMPD1	GG	significantly helpful for endurance performance
RS1799945	HFE	CG	slightly helpful for endurance performance
RS1800849	UCP3	AG	helpful for endurance performance
RS1815739	ACTN3	CC	not significantly helpful for endurance performance

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The muscle endurance improvement item uses the 9 polymorphic loci on *ADRB2*, *PPARA* gene: rs1799945, rs1800849, rs1042713, rs17602729, and rs2016520.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that related to endurance.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

The test only evaluates your endurance at the genetic level, and does not involve consideration of age, gender, BMI and other factors. Therefore, the test result may not be equivalent to your actual endurance.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci related to endurance.

Explosive Power

Skeletal muscle can generate massive power and strength in a short period of time. In sports like sprint, one's ability to generate such power is extremely important.

My Explosive Power

Moderate

49.9% of We users are similar to me

Suggestions

- The strength of explosive force is related to a variety of Gene variation, your comprehensive explosive power is **Moderate**
- You can have a general training response to high intensity resistance training. It is not recommended that you emphasis on this type of training
- Studies have shown that testosterone levels in men are associated with explosive force, and Increasing testosterone levels may increase explosive force to some extent

* If you need to use additional dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Knowledge

What is explosive power

Explosive power is the ability of skeletal muscle to burst out huge energy in a short time, which is an important athletic ability. In short time and high intensity speed and power events such as sprint, short distance swimming and so on, explosive power is the key. In addition to training and nutrition, genetics also play an important role in explosive exercise performance.

Genes and explosive power

It is found that gene polymorphism has a great influence on explosive power. For example, *ACTN3* directs the synthesis of α -actinin-3. This gene is located on human chromosome 11. Under normal circumstances, the 577th nucleotide of exon 16 is C, which directs the encoding of *ACTN3* protein. When C base mutates to T base and the arginine chain codon at position 577 changes to stop codon, the loss of α -actinin-3 will affect muscle stretching speed and reduce explosive power.

Advice on exercise

With strong explosive power, it is easier to perform well in speed and power sports. When it comes to exercise, the benefits are more pronounced after more intense resistance exercises (such as weight lifting).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS11549465	HIF1A	CC	not significantly helpful for explosive power performance
RS17602729	AMPD1	GG	significantly helpful for explosive power performance
RS1800795	intergenic	GG	significantly helpful for explosive power performance
RS1801131	MTHFR	GT	helpful for explosive power performance
RS1801282	PPARG	CC	not significantly helpful for explosive power performance

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The explosive force item uses 10 polymorphic loci on *ACTN3*, *AGT*, *NOS3* gene: rs1815739, rs2070744, rs1801282, rs11549465, and rs17602729.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all the genes or loci that related to the explosive force.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

The test only evaluates your explosive force at the genetic level, not including age, gender, BMI and other factors. Therefore, the test results may not be the same as your actual display of explosive force.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci related to explosive force.

Influences of Diet on Weight

Eating habit is key to controlling weight. Long-term intake of foods that are high in fats and calories can easily lead to obesity and also cause certain nutritional deficiencies. Poor eating habits such as regular overeating, nighttime snacking and having too many snacks are also important causes of obesity.

My Influences of Diet on Weight

Slightly Small

39.1% of We users are similar to me

Suggestions

- The effect of diet on weight is related to a variety of Gene mutations. Diet has **Less** effect on your weight.
- Diet alone has less effect on weight control. It is suggested to combine with exercise
- Dietary adjustment and Dietary restriction contribute to improve the effect of weight control
- Avoid Abnormal eating behaviors such as overeating and nighttime eating

* If you need dietary supplement, it is recommended that you consult an authoritative health professional or clinician for help.

Knowledge

Benefits of diet

Unhealthy eating habits can contribute to obesity. Long-term ingestion of high-calorie and high-fat foods can easily lead to obesity and deficiency of certain nutrients. Besides, bad eating habits, such as frequent overeating, night meal and snacking habits are also important causes of obesity.

Genes and the effect of diet on weight

In fact, after the same dietary restriction, the effect of weight control is different. Some people's weight will change significantly, while others may not that is related to the genetic factors according to the research. Genome-wide association analysis (GWAS) showed that that *APOA5*, *PPARG* and *PPARG* gene were significantly correlated with the weight change after dietary restriction. Further research shown that *PPARG* gene is a transcription factor to participate in the regulation of lipid

differentiation, utilization, metabolism and obesity, *APOA5* gene regulates the activity of lipoprotein lipase and is intimately connected to the blood lipid metabolism and *APOA2* gene is associated with visceral fat accumulation and triglyceride-rich lipoprotein metabolism.

What can I do

If diet has a greater effect on weight, it is better to control your weight through diet. If you need to lose weight, it is suggested to select appropriate food matching based on the other test results (such as carbohydrate intolerance and saturated fatty acid intolerance).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1801282	PPARG	CC	dietary restriction may have a slightly poor impact on weight loss
RS5082	APOA2	AA	dietary restriction may have a slightly poor impact on weight loss
RS662799	APOA5	AA	dietary restriction may have a slightly poor impact on weight loss

Test details

Applicable situation

The effect of exercise on weight item uses three polymorphic loci on the *APOA5*, *PPARG* and *APOA2*: rs662799, rs1801282 and rs5082.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that related to the effect of diet on weight.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all

users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

The test evaluates the effect of diet on weight at gene levels and does not include the other factors such as age, sex, exercise habits, etc. Therefore, the test result may not be equivalent to the actual impact of diet on weight.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci that related to the effect of diet on weight.

Influences of Exercise on Weight

Exercise is a scientific and healthy way to controlling weight. Usually the greater the amount and longer the duration of exercise, the more sugar and fat consumed.

My Influences of Exercise on Weight

Moderate

36.8% of We users are similar to me

Suggestions

- The effect of exercise on weight is related to a variety of Gene mutations. Exercise has **Medium** effect on your weight.
- Exercise alone has medium effect on weight control. It is suggested to combine with other methods:
 - Appropriately increase the exercise time and frequency
 - Select the appropriate Sports tonics to improve the exercise effect
 - Dietary adjustment, Caloric restriction
- Please try to choose different sports forms for enjoying sports and developing exercise habit.

* If you need dietary supplement, it is recommended that you consult an authoritative health professional or clinician for help.

Knowledge

Benefits of exercise

Regular exercise has significant health benefits, such as reducing the risk of cardiovascular disease, type 2 diabetes, even some cancers, and the most benefit is weight control.

Genes and the effect of exercise on weight

In fact, after the same time and exercise intensity, the effect of weight control is different. Some people's weight will change significantly, while others may not that is related to the genetic factors according to the research. Genome-wide association analysis (GWAS) showed that that *FTO* and *PPARG* gene were significantly correlated with the weight change after exercise.

What can I do

If exercise has a greater effect on weight, it is better to control your weight through exercise. If you need to lose weight, it is suggested to select appropriate sports and actively participate in exercise based on the test results of other sports (such as explosive power and endurance).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1800588	LIPC	CC	a consistent amount of exercise has a relatively good impact on weight loss
RS1801282	PPARG	CC	a consistent amount of exercise has a relatively good impact on weight loss
RS9939609	FTO	TT	a consistent amount of exercise has a poor impact on weight loss

Test details

Applicable situation

The effect of exercise on weight item uses three polymorphic loci on the *FTO* and *PPARG* gene: rs9939609, rs1801282 and rs1800588.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that related to the effect of exercise on weight.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

The test evaluates the effect of exercise on weight at gene levels and does not include the other factors such as age, sex, eating habits, etc. Therefore, the test result may not be equivalent to the actual impact of exercise on weight.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci that related to the effect of exercise on weight.

Temperature Increasing Rate During Exercise

The speed at which body temperature increases during exercise reflects how efficiently body temperature increases during exercise of the same intensity. In general, the higher the indicator, the faster the optimal exercise temperature is reached, that is, a shorter warm-up time is required.

My Temperature Increasing Rate During Exercise

Slow

38.3% of We users are similar to me

Knowledge



Brief introduction

During aerobic exercise, the body will produce a series of physiological changes, such as increased heart rate, blood pressure, temperature. One 2012 study has revealed that increased temperature is related to heredity during exercise.



Research findings

200 participants completed a 30-minute submaximal (exercise intensity is the same as 65% maximal oxygen capacity) aerobic exercise on the treadmill, then researchers measured their ear temperature before exercise, 10 minutes, 20 minutes and 30 minutes after exercise. The result shown that people with rs2253206 in the *CREB1* gene had minimum temperature change after exercise, while people with AA and AG genotype had larger temperature change.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2253206	intergenic	GG	insignificant increase in body temperature during exercise, requires a slightly long warm-up

Test details

Applicable situation

The temperature increasing rate during exercise item uses one polymorphic locus on the *CREB1* gene: rs2253206.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that related to the temperature increasing rate during exercise.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

The test evaluates the temperature increasing rate during exercise at gene levels and does not include the influence of environment and other factors. Therefore, the test result may not be equivalent to the temperature increasing after actual exercises.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci that related to the temperature increasing rate during exercise.

Muscle Endurance Improvement from Exercise

This value is used to measure the significance of a person's increase in muscle endurance from exercise. The smaller the increase in muscle endurance, the more care should be taken not to blindly increase the intensity of exercise.

My Muscle Endurance Improvement from Exercise

Large

19% of We users are similar to me

Suggestions

- The strength of muscle endurance increasing with a variety of Gene Mutations, and your overall muscle endurance is **Great**
- Post-exercise muscle soreness can significantly improve after a sustained period of exercise
- The following methods may help with soreness in addition to regular exercise:
 - Healthcare should be carried out after exercise, such as proper stretching massage, timely hydration and protein intake, and repeated ice bath
 - Eat more Protective Foods such as watermelon, cherries, sports supplements containing glutamine, etc
 - Wearing high-performance clothing to help stabilize muscles, prevent minor injuries, improve circulation and speed up the recovery
 - Get more sleep because the body will produce growth hormone to help tissues grow and repair during sleep

* If you need to use additional dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Knowledge



What is muscle endurance

Muscle soreness and fatigue will follow after a long time of exercise (such as an 800-meter race) due to the shift of supply energy to the anaerobic system (phosphagen and glycolytic for energy supply) and the production of lactic acid with the increase of exercise intensity. Critical value at which lactic acid begins to accumulate is lactate threshold, also named anaerobic threshold. The higher of lactate threshold, the later muscle soreness occurs after exercise and the muscle endurance is higher.

Gene and muscle endurance improvement

Exercise can improve muscle endurance. However, research shows that different people have different effects on improving muscle endurance after exercise, which may be related to *PPARD* and *PPARGC1A* gene. Mitochondrial function and the types of muscle fibers in different populations lead to the differences in muscle endurance increasing (i.e. lactate threshold increased).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2267668	PPARD	AA	significantly increased muscular endurance after exercise
RS8192678	PPARGC1A	CC	significantly increased muscular endurance after exercise

Test details

Applicable situation

The improvement of muscle endurance item uses two polymorphic loci on the *PPARD* and *PPARGC1A* gene: rs2267668 and rs8192678.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that related to the improvement of muscle endurance.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

The test evaluates your muscle endurance improvement at gene levels and does not include the influence of training and other factors. Therefore, the test result may not be equivalent to the increase in muscle endurance after actual exercises.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci that related to the improvement of muscle endurance.

Motivation to Exercise

Motivation to exercise refers to a person's subjective initiative to exercise.

My Motivation to Exercise

Slightly Low

34.9% of We users are similar to me

Suggestions

1. Find an exercise partner to supervise each other, which will help keep exercising.
2. Choose diversified training items during exercise, reduce the boring feeling of training, and help to keep exercising.

Knowledge

The benefits of exercise

Prolonged sitting for a long time can lead to a series of health problems such as obesity, type 2 diabetes, and cardiovascular diseases. Exercise can bring benefits such as lowering blood pressure, reducing the level of low-density lipoprotein cholesterol levels in plasma, increasing the level of high-density lipoprotein cholesterol in plasma, and improving heart function, etc. It is considered to be a kind of help in the way of cardiovascular health.

Genes and motivation to exercise

In the past, people believed that participation in exercise was a trait influenced by environmental factors. Now, twin studies have shown that genes also play a very important role in the matter of adult participation in exercise. In a recent study, researchers conducted a genome-wide association study (GWAS) on a large sample of nearly 3,000 people. By analyzing the results of genetic testing of people who exercise frequently in their leisure time and people who lack exercise, they found genetic loci that were significantly associated with leisure-time exercise behavior.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS10887741	PAPSS2	CT	slightly high motivation to exercise, tends to participate in exercise in spare time
RS12612420	intergenic	GG	poor motivation to exercise, not inclined to exercise in spare time
RS8097348	intergenic	AA	poor motivation to exercise, not inclined to exercise in spare time

Test details

Applicable situation

The exercise motivation item uses the *PAPSS2* gene and 3 polymorphic loci of gene: rs10887741, rs12612420, and rs8097348.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that related to exercise motivation.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

The test only evaluates your exercise motivation at the genetic level, and does not include the influence of other factors such as the environment and whether you are injured. Therefore, the test result may not be equivalent to your actual participation in exercises.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

The test may not include all loci related to exercise motivation.

Maximal Oxygen Uptake (VO2max) Improvement

Maximal Oxygen Uptake (VO2max) refers to the maximum volume of oxygen one's body can deliver to skeletal muscle in a given period of time.

My Maximal Oxygen Uptake (VO2max) Improvement

Moderate

9.3% of We users are similar to me

Knowledge



What can the VO2max reflect

The VO2max can vary greatly between individuals. The VO2max can reflect the aerobic exercise capacity, and a high level of the VO2max often means a high level of aerobic exercise performance. For example, the VO2max of endurance Olympic athletes can reach 75ml/kg/min, while the average person without training can only reach less than 30ml/kg/min.



Genes and VO2max improvement

The VO2max is also used to measure the adaptability of cardio-pulmonary function. Studies have shown that regular training is the most effective way to improve cardio-pulmonary function adaptability. However, for different people, the increase in the VO2max brought about by regular training is also very different. In one study, the researchers performed aerobics sport training on 742 volunteers for 20 weeks. After the training, the average increase of volunteers' VO2max reached 400ml/min. However, there are huge individual differences. Volunteers with low level of increase can only increase less than 100ml/min, while those with high level of increase reach 700ml/min. This is mainly caused by genetic factors.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS6552828	ACSL1	GG	no significant increase in maximum oxygen uptake after exercise
RS1535628	intergenic	AA	significantly increased maximum oxygen uptake after exercise
RS4973706	intergenic	TT	no significant increase in maximum oxygen uptake after exercise
RS884736	CAMTA1	CC	no significant increase in maximum oxygen uptake after exercise
RS6090314	intergenic	GG	no significant increase in maximum oxygen uptake after exercise

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The VO2max improvement item uses genes such as *ACSL1*, *CD44*, *CAMTA1* and 12 polymorphic loci on these genes: rs6552828, rs1535628, rs884736, rs6090314, and rs10500872.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that related to VO2max improvement.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

-
1. The test only evaluates your VO2max improvement at the genetic level, and does not involve consideration of age, gender, and other factors. Therefore, the test result may not be equivalent to your actual VO2max improvement.
 2. In addition, due to the difference of the study population, the VO2max improvement value range in the literature is for reference only.
 3. If necessary, please consult an authoritative sports coach for professional training guidance with the goal of improving VO2max.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci related to VO2max improvement.

Heart Rate Improvement

Heart rate is a commonly used measurement for one's cardiorespiratory capacity.

More and more evidence proves that regular exercises could improve cardiorespiratory capacity and the speed of heart rate increment during exercises.

My Heart Rate Improvement

Slightly Large

38.5% of We users are similar to me

Knowledge

Sports and heart rate

During exercise, the heart supplies oxygen to the body by pumping out blood. The higher the exercise intensity, the more oxygen the body needs, and the heart needs to pump blood at a faster speed, thus leading to the pressure of the heart. The specific characterization is that the blood pressure is increased, and the frequency of heartbeat is accelerated (that is, the heart rate is increased).

What is heart rate recovery

Studies have shown that regular exercises can improve heart function. That is to say, when you perform exercises of the same intensity, the increase of the heart rate will be lower than the increase in the untrained stage, and the pressure on the heart will be smaller. This is heart recovery rate.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2253206	intergenic	GG	significantly helpful for improving heart rate after exercise
RS13387495	ERBB4	TT	not significantly helpful for improving heart rate after exercise

Test details

Applicable situation

The heart recovery rate item uses 2 polymorphic loci on the *ERBB4* gene: rs2253206 and rs13387495.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that related to heart recovery rate.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

1. The test only evaluates your heart recovery rate at the genetic level, and does not involve consideration of age, gender, and other factors. Therefore, the test result may not be equivalent to your actual heart recovery rate. In addition, the research results based on the detection are only applicable to heart rate improvement under regular submaximal exercises, and not applicable to heart rate changes caused by strenuous exercise. Therefore, the test result may not be equivalent to your actual heart recovery rate.
2. If necessary, please consult an authoritative sports coach for professional training guidance with the goal of improving heart rate.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci related to heart recovery rate.

Lumbar Disc Injury Risk

If lumbar disc is likely to develop injury, it is especially important to pay attention to controlling intensity during weightlifting and exercises that put a lot of stress on the waist.

My Lumbar Disc Injury Risk

Moderate

30.2% of We users are similar to me

Postures that can reduce pressure on the lumbar spine



maintain a neutral and straight spine position



making chair close to desk helps maintain good posture



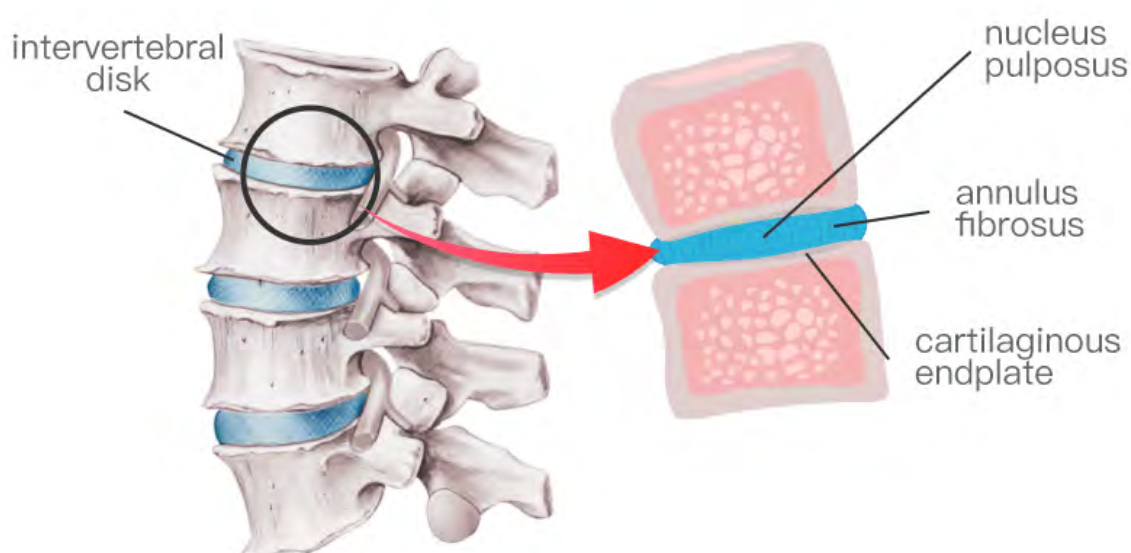
reduce lumbar disc pressure by bending knee and hip joints



Knowledge

Physiological structure

Located between two vertebral bodies, the lumbar intervertebral disc is a structure with hydrodynamic characteristics. It is composed of cartilage endplate, annulus fibrosus and nucleus pulposus. Nucleus pulposus is an elastic colloidal material, which can alleviate the impact. The annulus fibrosus is composed of multi-layer fibrous cartilage rings arranged in a ring, which surrounds the nucleus pulposus to prevent the nucleus pulposus from protruding outward. The annulus fibrosus connects the adjacent vertebral body so that it can move slightly. However, when the movement reaches a certain limit, the annulus fibrosus becomes tense, which plays the role of controlling the ligament and limiting the rotational movement; The cartilage end plate is located on the upper and lower sides of the intervertebral disc. It is composed of fibrocartilage, one on the upper and one on the lower side of the vertebral body. The cartilage endplate protects the vertebral bone, buffers the pressure, connects the vertebral body and the intervertebral disc, and plays the role of nutrient exchange for the intervertebral disc.



Causes of injury

Excessive lumbar load or lumbar movement can easily lead to the injury of lumbar intervertebral disc. When the lumbar intervertebral disc is seriously injured, the nerves can be compressed and lead to limb numbness. Therefore, whether standing or sitting, you should keep the back with the tail vertebrae in a straight line as far as possible to avoid lumbar injury.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS1052576	CASP9	CC	significantly increases lumbar disc protection
RS11549467	HIF1A	GG	not helpful for lumbar disc protection
RS1800795	intergenic	GG	not significantly helpful for lumbar disc protection
RS1800896	intergenic	TT	significantly helpful for lumbar disc protection
RS2073711	CILP	AG	significantly helpful for lumbar disc protection

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The lumbar intervertebral disc injury risk item uses twelve polymorphic loci on genes such as *COL9A3*, *VDR* and *MMP2*: rs731236, rs763110, rs917997, rs151058, and rs2287037.

Notification

1. The test is mainly based on the research of Caucasian and Asian, and other populations are only for reference.
2. The test does not cover all genes or loci that related to the risk of lumbar intervertebral disc injury.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

-
1. The test only evaluates the possibility of your lumbar intervertebral disc injury from the genetic level, and does not include other factors such as age, environment, external force impact and so on.
 2. If you think you may have lumbar spine problems, please consult an authoritative health expert or clinician in time.

Study population

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

The test may not include all loci related to the risk of lumbar intervertebral disc injury.

Achilles Tendon Injury Risk

If people have risk of Achilles tendon injury, they should warm up and pay attention to protecting the ankles when running or performing movements that require a bigger load on the Achilles tendon.

My Achilles Tendon Injury Risk

Slightly Low

13.7% of We users are similar to me

Knowledge



Physiological structure

Tendons are the connective tissue that connects muscles to bones. The main component of tendons is collagen.

The Achilles tendon is a tendon located on the back of the ankle bone, connecting the heel with the muscles on the back of the calf. The main function of the Achilles tendon is calf flexion and plantar flexion of the foot. The length of the Achilles tendon varies greatly from person to person. The mechanical properties of tendons depend on the diameter and direction of collagen fibers. Collagen fibers are parallel to each other and closely aligned with each other. The collagen fibers in the tendon have a certain degree of flexibility. In addition, since the tendon is a multi-linked structure composed of many independent fibers and bundles, rather than a rod-shaped structure, this property also contributes to its flexibility. When the Achilles tendon is pulled beyond the threshold, injury may occur. The injury of the Achilles tendon can lead to inflammation, and severely rupture, which seriously affects athletic ability. The risk of Achilles tendon injury is significantly associated with genes encoding metalloproteinase 3 and collagen.



Causes of injury

In addition to genetic factors, sports such as sprinting can also easily lead to d Achilles tendon injury. The maximum load on the Achilles tendon is about 3.9 times of the body weight when walking and 7.7 times of the body weight when running. Therefore, Achilles tendon injury is more common in sprinters and hurdlers.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1045485	CASP8	CG	helpful for Achilles tendon protection
RS1143627	IL1B	AG	significantly helpful for Achilles tendon protection
RS16944	IL1B	AG	significantly helpful for Achilles tendon protection
RS1800795	intergenic	GG	not significantly helpful for Achilles tendon protection
RS2104772	TNC	AA	not significantly helpful for Achilles tendon protection

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The Achilles tendon injury risk item uses eight polymorphic loci from genes like *COL5A1*, *TNC* and *MMP3*: rs16944, rs1045485, rs2104772, rs591058, and rs3834129.

Notification

1. The test is mainly based on the research of Caucasian, and other populations are only for reference.
2. The test may not cover all genes or loci that related to the risk of Achilles tendon injury.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

1. The test only evaluates the possibility of your Achilles tendon injury at the genetic level, and does not include the influence of other factors such as age, environment, external force impact, etc.
2. If you think you may have Achilles tendon problems, please consult an authoritative health expert or clinician in time.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci related to the Achilles tendon injury risk.

Anterior Cruciate Ligament (ACL) Injury Risk

Knee anterior cruciate ligament (ACL) prevents the tibia from moving forward to the femur, so it often breaks during intense torsional movement and ACL injury is very common among athletes. People with risk of ACL injury should avoid sudden change of direction in movement during exercises.

My Anterior Cruciate Ligament (ACL) Injury Risk

Slightly Low

16.8% of We users are similar to me

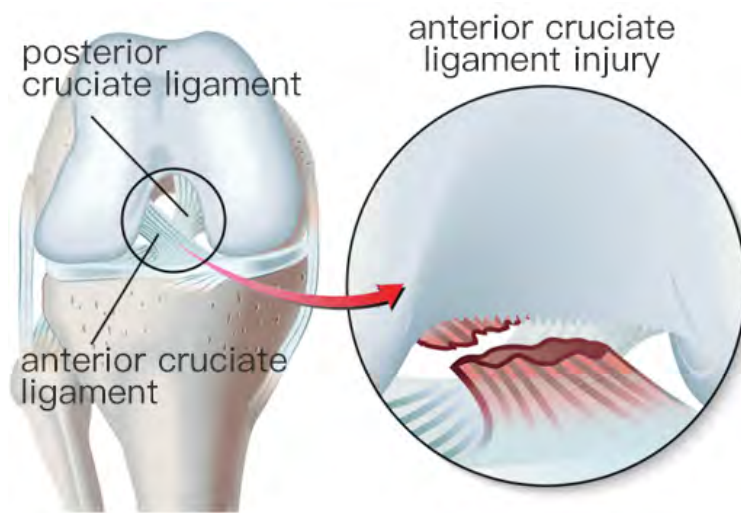
Knowledge



Physiological structure

Ligaments are flexible elastic connective tissue that attaches to the movable part of the bone, connecting the bone and limiting its range of activity to avoid injury.

The anterior cruciate ligament, also known as the anterior cruciate ligament, is located at the knee joint. It starts from the front of the tibial intercondylar bulge, and attaches to the medial surface of the lateral femoral condyle obliquely backward and upward, connecting the femur and tibia. The anterior cruciate ligament prevents the tibia from moving forward to the front of the femur. When the knee joint is fully bent, the anterior cruciate ligament stretches most tightly. When the ligament is stretched beyond the threshold, injury may occur. If the anterior cruciate ligament of the knee joint is injured, it will greatly affect the stability of the knee joint and may lead to the injury of meniscus and other structures.



Causes of injury

The risk of knee anterior cruciate ligament injury is significantly related to genes encoding disaccharide chain protein and collagen. In addition to genetic factors, the rapid change of direction or excessive torsion of tibia during exercise can easily lead to the injury of anterior cruciate ligament. Anterior cruciate ligament injury of the knee is more common among skiers, footballers, and basketball players.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1107946	intergenic	CC	significantly helpful for acl protection
RS1126499	BGN	CT	helpful for acl protection
RS1570360	VEGFA	GG	helpful for acl protection
RS1800012	COL1A1	CC	not significantly helpful for acl protection
RS1800255	COL3A1	GG	significantly helpful for acl protection

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The knee anterior cruciate ligament injury risk item uses nine polymorphic loci on genes such as *COL3A1*, *COL12A1* and *BGN*: rs1800255, rs1042103, rs970547, rs679620, and rs1570360.

Notification

1. The test is mainly based on the research of Caucasian, and other populations are only for reference.
2. The test may not cover all genes or loci that related to the risk of knee anterior cruciate ligament injury.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

1. The test only evaluates the possibility of your knee anterior cruciate ligament injury from the genetic level, and does not include other factors such as age, environment, external force impact and so on.
2. If you think you may have knee problems, please consult an authoritative health expert or clinician in time.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

The test may not include all loci related to the risk of knee anterior cruciate ligament injury.



Nutrient Metabolism

16 Reports

Metabolic Capacity

Lactose Metabolism

Weak



Caffeine Metabolism

Strong



Alcohol Metabolism

Moderate, hard to get dizzy and alcohol flush



Mineral Requirements

Calcium Nutritional Requirement

Normal, normal Calcium intake



Iron Nutritional Requirement

Slightly High



Zinc Nutritional Requirement

High



Magnesium Nutritional Requirement

Slightly High



Vitamin Requirements

Folate Nutritional Requirement

Slightly High, recommend supplementary folate intake



Vitamin E Nutritional Requirement

Slightly High



Vitamin B12 Nutritional Requirement

Slightly High



Vitamin A Nutritional Requirement

Slightly High, consider supplementary Vitamin A intake



Vitamin C Nutritional Requirement

Slightly High



Vitamin K Nutritional Requirement

High, consider supplementary Vitamin K intake



Vitamin B6 Nutritional Requirement

Slightly High



Vitamin D Nutritional Requirement

Slightly High



Vitamin B2 Nutritional Requirement

Normal



Lactose Metabolism

Lactose only exists in mammalian milk in nature, which is very important for children's intellectual development. Lactose metabolism is closely related to lactose intolerance, a condition in which the body does not produce lactase that breaks down lactose.

My Lactose Metabolism

Weak

Intake recommendations

- Your Lactose metabolism is **weak**. After drinking a lot of milk on an empty stomach, Adverse reactions such as gastrointestinal discomfort may occur
- There is a mutation in your *MCM6* gene, which causes the body's lactase activity to be insufficient to break down the lactose in milk
- Choose to drink Yogurt and other Lactose intolerant substitutes
- You can drink milk with cereals or Lactase
- Choose low-lactose milk (such as Shuhua milk) or lactose-free milk powder
- Not suitable Drink milk on an empty stomach
- Some people with lactose intolerance drink 200 ml of milk a day without any discomfort, and this ability can also be trained

List of suitable substitutes for lactose intolerant



lactose-free milk



yogurt



cheese



soy milk

Knowledge



Risks of consuming too much lactose in lactose-intolerant people

The symptoms of gastrointestinal discomfort caused by drinking milk and eating dairy products are mainly due to the lack of lactase in the intestine that can decompose lactose. The undecomposed lactose is continuously fermented by bacteria in the intestine, causing bloating, diarrhea and abdominal pain, severe cases may vomit and dehydration.



Lactose metabolism

Since lactose is ingested from the mouth, it does not undergo any chemical changes through the role of digestive glands such as the pharynx, stomach, gallbladder, and pancreas. Until the intestinal lumen side of the brush border of the small intestine mucosa, lactose is hydrolyzed into two monosaccharides, galactose and glucose, under the catalysis of lactase, and absorbed by the small intestine into the normal blood circulation. Then, under the regulation of uridine diphosphate galactose (UDP-Gal) 4-epimerase, most of the galactose enters the liver and turns into glucose, and a small amount is metabolized by red blood cells or excreted in the urine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS182549	MCM6	CC	weak lactose metabolism
RS4988235	MCM6	GG	weak lactose metabolism

Test details

Applicable situation

The lactose metabolism test item uses 2 polymorphic loci on the *MCM6* gene. The test result shows that the user's genotype combination at the two polymorphic loci detected and the lactose metabolism situation represented by this test cannot represent the user's actual lactose metabolism situation.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technology and the level of scientific cognition, and the test may not cover all genes or loci that related to the lactose metabolism.

How to use the test results

Notification

1. The test results are not applicable to clinical diagnosis.
2. An individual's nutritional metabolism is the result of the interaction of genes, environment, and living habits.
3. The test result only predicts your lactose metabolism from the genetic level, because it does not involve consideration of other factors, it does not represent your true situation. If you have nutritional supplement-related needs, you can consult authoritative nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutritional advice.

Test content

The test includes rs4988235 and rs182549, these 2 polymorphic loci related to lactose metabolism.



Study population

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the lactose metabolism.

Caffeine Metabolism

Caffeine is a central nervous stimulant, which can temporarily drive away drowsiness and restore energy. It is clinically used to treat neurasthenia and coma resuscitation.

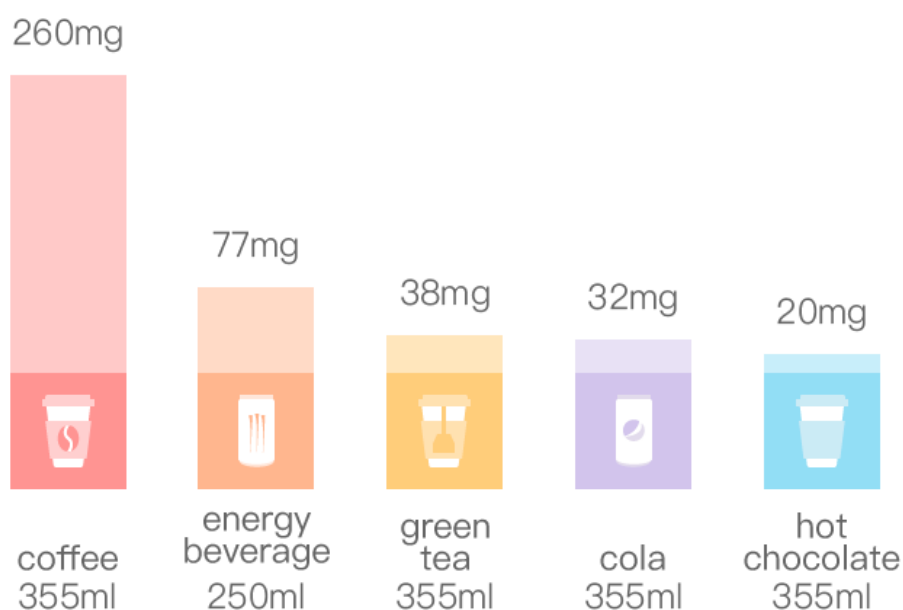
My Caffeine Metabolism

Strong

Intake recommendations

- Your Caffeine metabolism is high, it is appropriate to drink Caffeinated drinks no more than Normal intake (e.g., no more than two large cups of American coffee).
- The best time to drink coffee is: 9:30-11:30 AM in the morning or before 4:00 PM in the afternoon.
- Excessive drinking may also cause **insomnia**, **palpitation** and other neuroallergic reactions.
- Very hot coffee is not suitable for drinking; Hot drinks ($>65^{\circ}\text{C}$) are carcinogenic.
- Drink coffee with Milk to reduce the discoloration of teeth.
- Special population (such as pregnant women) need to pay attention to reduce the intake.

Graph of caffeine content in common beverages



Knowledge

Risks of caffeine consumption

1. Intensify hypertension.
2. Induced osteoporosis.
3. May cause abortion in the parturient.
4. For people who are prone to anxiety, it is easy to cause nervous reactions such as anxiety, palpitations, and sweating of palms.
5. For some people, it is easy to cause insomnia or poor sleep..

Benefits of caffeine consumption

1. It can boost the spirit, improve thinking and memory, and restore muscle fatigue.
2. Acting on the cardiovascular system can improve heart function, dilate blood vessels and promote blood circulation.
3. For the gastrointestinal system, it can help digestion and fat decomposition.

Caffeine metabolism

Caffeine is decomposed in the liver to produce three primary metabolites xanthine (84%), theobromine (12%) and theophylline (4%). Caffeine is completely absorbed by the stomach and small intestine within 45 minutes after ingestion. After absorption, it will be distributed in all organs of the body. The transformation process conforms to the first-order reaction of chemical kinetics. These compounds are further metabolized and finally excreted through urine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS762551	CYP1A2	AA	not very sensitive to caffeine

Test details

Applicable situation

The caffeine metabolism item provided uses a polymorphic locus of the *CYP1A2* gene. The test results indicate that the genotype results of the detected 1 polymorphic locus and the caffeine metabolism represented by this test cannot represent the true caffeine metabolism of the test.

 Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technology and level of scientific cognition, the test may not cover all genes or sites related to caffeine metabolism.

How to use the test results

 Notification

1. The test results are not applicable to clinical diagnosis.
2. An individual's nutritional metabolism is the result of the interaction of genes, environment, and living habits.
3. The test result only predicts your caffeine metabolism from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation. If you have nutritional supplement-related needs, you can consult an authoritative nutritionist, clinical doctor or professional to obtain professional nutritional advice based on the results of this genetic test.

 Test content

The test includes a polymorphic locus, rs762551, associated with caffeine metabolism.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci related to caffeine metabolism.

Folate Nutritional Requirement

Folate is an essential nutrient for the production of normal red blood cells and the synthesis of DNA. It is particularly important for pregnant women and can prevent fetal neural tube malformations.

My Folate Nutritional Requirement

Slightly High, recommend supplementary folate intake

Intake recommendations

- Your folate nutritional requirement is **Slightly High**, it is recommended to take in Folate-rich foods such as green leafy vegetables, or Extra supplements, or **L-Methylfolate**
- There is a mutation in your *MTHFR* gene, excessive supplementation may cause Adverse reactions, you should avoid excessive intake of folate, or use **L-Methylfolate** instead
- There is a mutation in your *MTHFR* gene. When you choose to supplement with folate, you should pay special attention to [^Folate overdose symptoms] such as insomnia. If it occurs, you should reduce the dose
- If you experience Folate deficiency symptoms such as fatigue, oral ulceration, etc., you should pay attention to folic acid or L-Methylfolate supplementation
- Sufficient intake of **Vitamin B12/B2/B6** helps Folate metabolism; if you choose to supplement with folate or L-Methylfolate, you should also pay attention to the supplement of **vitamin B12**
- Pregnant women's needs for folate is high, so you should pay attention to folic acid or L-Methylfolate supplementation

* If you have additional folate supplement needs, please consult your doctor or professional nutritionists for the specific dosage

Folate rich foods

nutrient content per 100g edible part



Knowledge

Problems caused by deficiency

Folate can help cells in the body produce and maintain DNA. It can also help produce blood cells with vitamins B6, B12 and C. Lack of any of the above vitamins can cause anemia. Severe folate deficiency causes megaloblastic anemia. Early symptoms of folate deficiency may include:

1. Fatigue
2. Gray hair
3. Aphthous ulcers
4. Failure to thrive
5. Swollen tongue

Problems caused by excessive intake

Since the human body does not store folate, folate is generally safe for the human body. However, excessive intake of supplements may cause insomnia.

Metabolism

Under the effect of methylenetetrahydrofolate reductase (MTHFR) and methionine synthase reductase (MTRR), folate participates in two important aspects of metabolism in the human body. One is the synthesis of DNA, and folate is a key cofactor involved in the synthesis of nucleobases in the human body. The other is involved in the conversion of some amino acids, folate can add a 'one-carbon unit' chemical group to the harmful homocysteine to eliminate harm.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1801131	MTHFR	GT	reduced folic acid metabolic enzyme activity
RS1801133	MTHFR	GG	normal folic acid metabolic enzyme activity
RS1801394	MTRR	AG	reduced folic acid metabolic enzyme activity

Test details

Applicable situation

The folate nutritional requirement item uses 3 polymorphic loci on the *MTHFR* and the *MTRR* gene. The test results only indicate the folate nutritional requirement result from the genotype combinations of the user at the 3 polymorphic loci detected. The test cannot represent the user's actual folate nutritional requirement.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technology and the level of scientific cognition, the test does not cover all genes or loci associated with folate nutritional requirement.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. An individual's nutritional requirement is the result of the interaction of genes, environment, and living habits.
3. The test results only predict your folate nutritional requirement from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status. If you have nutritional supplement-related needs, you can consult authoritative nutritionists,

clinicians or professionals based on the results of this genetic test to obtain professional nutritional supplementary advice, or follow the doctor's advice for clinically qualified inspection and test.

Test content

The test includes 3 polymorphic loci associated with folate nutritional requirement, such as rs1801131, rs1801133 and rs1801394.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci associated with folate nutritional requirement.

Calcium Nutritional Requirement

Calcium is the most abundant mineral in the human body, which is mainly used to support the structure and hardness of bones and teeth.

My Calcium Nutritional Requirement

Normal, normal Calcium intake

Intake recommendations

- Your calcium nutritional requirement is **normal**. It is recommended to consume Calcium-rich foods such as dairy products and seafood Normally.
- If there is joint pain, cramps, osteoporosis and other Calcium deficiency symptoms, attention should be paid to supplement calcium and **vitamin D**.
- **Vitamin D** helps the small intestine absorb calcium, and exposure to sunlight helps **vitamin D** synthesis.
- Children and adolescents or middle-aged and elderly people have higher calcium requirements and should pay attention to calcium intake.
- The elderly and those with dyspepsia can easily cause flatulence when using calcium carbonate. Organic calcium supplements should be preferred.

*For additional calcium and vitamin D supplements, consult your doctor or professional nutritionist the specific dosage.

Calcium rich foods

nutrient content per 100g edible part



Knowledge

Problems caused by deficiency

Most of the calcium in the body is in the form of calcium phosphate in bone. Those who are chronically deficient in calcium may develop osteoporosis.

Early symptoms of osteoporosis include:

1. Severe back pain
2. Shrinking height
3. Myasthenia

Other symptoms of calcium deficiency include depression, high blood pressure, restlessness, tingling and burning sensation in the hands, feet and toes, etc.

Problems caused by excessive intake

Increasing calcium intake for a short period of time usually does not cause problems. However, long-term higher levels of calcium intake can cause constipation and increase the risk of kidney stones in some people.

Metabolism

Generally, 20%-30% of the calcium ingested through food is absorbed into the stomach and intestines, and the unabsorbed calcium is excreted in the feces. Calcium metabolism is mainly regulated by vitamin D, which enhances calcium reabsorption in the small intestine and is closely involved in plasma calcium homeostasis. Calcium in the body exists in two forms: More than 99% of

calcium is combined with phosphorus to form bone salts in the form of hydroxyapatite, which is present in bones and teeth, while about 1% of calcium is present as ions in soft tissues, extracellular and blood, collectively known as miscible calcium pools. The above two kinds of calcium maintain a dynamic equilibrium state. Calcium in the bone is continuously released from osteoclasts into the calcium pool, and calcium ions in the calcium pool are deposited in osteoblasts to form new bone.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1042636	CASR	AA	normal serum calcium concentration
RS1544410	VDR	CC	normal absorption of calcium

Test details

Applicable situation

The calcium nutritional requirement test item uses 2 polymorphic loci in the *VDR* and *CASR* genes. This test result indicates the calcium nutritional requirement represented by the genotype combination of the 2 polymorphic loci detected by the user, but the test could not represent the actual calcium nutritional requirement of the user.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to calcium nutritional requirement.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. An individual's nutritional requirement is the result of the interaction of genes, environment, and living habits.

3. The test result only predicts your calcium nutritional requirement at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true status. If you have nutritional supplement-related needs, you can consult authoritative nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutrition advice.

 Test content

The test includes 2 polymorphic loci associated with the calcium nutritional requirement, such as rs1544410 and rs1042636.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to calcium nutritional requirement.

Alcohol Metabolism

The human body mainly relies on alcohol dehydrogenase and aldehyde dehydrogenase to decompose alcohol into carbon dioxide and water step by step. Therefore, these two key enzymes determine the metabolic rate of alcohol.

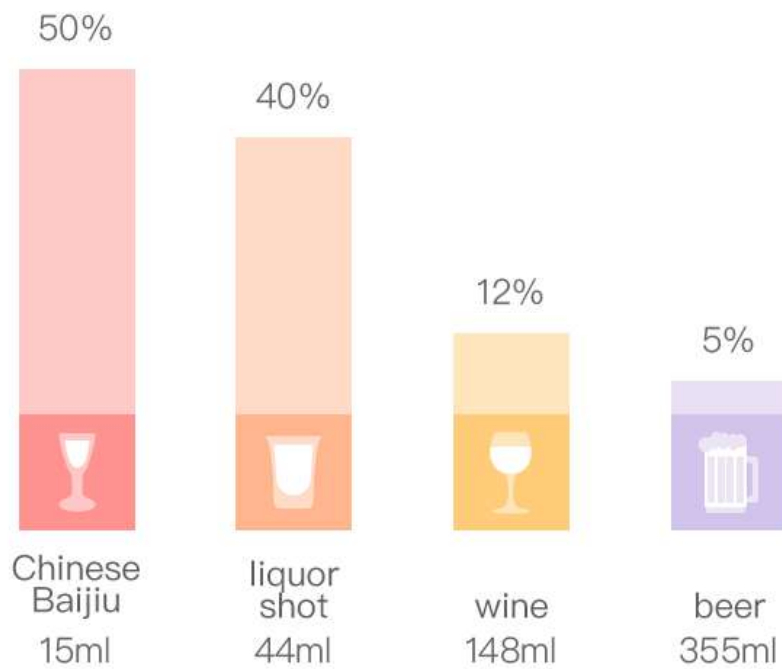
My Alcohol Metabolism

Moderate, hard to get dizzy and alcohol flush

Intake recommendations

- Your level of alcohol metabolism is **moderate**. It is recommended not to Drink too much.
- Your Alcohol dehydrogenase (ADH1B) gene has a small amount of mutations. The conversion of alcohol to acetaldehyde is slower and you are slightly drunk, but you generally do not flush, and the risk of acetaldehyde poisoning is low.
- In case of dizziness, palpitation, vomiting, tachycardia and other Adverse reactions, please stop drinking alcohol in time.
- **Cephalosporins, aspirin, warfarin** and other Drugs may react with alcohol, so be careful to drink alcohol during medication.
- Pregnant women, minors, patients with liver disease, gout patients should avoid drinking alcohol, which may cause Serious problems.
- Alcohol can affect the absorption of nutrients such as **vitamin C, vitamin B12** and **folic acid**. Take appropriate amounts after drinking.

Comparison of alcohol percentage in drinks



Knowledge

Risks of alcohol consumption

1. Alcohol interferes with communication between the brain and other organs, which can affect sleep quality, mood and coordination ability.
2. Alcohol can damage heart health and increase the risk of cancer.
3. Drinking alcohol can also cause adverse effects on the liver, pancreas and immune system.
4. Alcohol affects the absorption of essential nutrients such as vitamin C, vitamin B12 and folic acid.

Benefits of moderate alcohol consumption

According to a large population cohort study, there is no minimum dose of alcohol harmful to the human body. Any level of alcohol intake may cause health hazards. Please stay away from alcoholic beverages. Especially do not drink and drive.

Alcohol metabolism

More than 95% of alcohol (ethanol) consumed is catabolized in the body except for a very small amount excreted by respiration and urine, and the liver is an important organ for ethanol metabolism. The catabolism of alcohol in the human body depends mainly on two enzymes: one is ethanol dehydrogenase, the other is acetaldehyde dehydrogenase. Alcohol dehydrogenase can oxidize ethanol to acetaldehyde. And acetaldehyde dehydrogenase can oxidize acetaldehyde to acetic acid.

Alcohol metabolism and alcohol allergy are different

Allergic reaction is a pathological reaction caused by the body's immune system producing a specific immune response. It is the result of the interaction between external factors (allergens) and self factors (allergic constitution). Alcohols, esters, aldehydes and other substances in alcoholic beverages can be allergens, which in some people can 'trigger' an immune response that can lead to rashes and itchy skin. At this time, the patient should stop drinking immediately, and see a doctor or take medication in time depending on the condition.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1229984	ADH1B	CT	reduced ethanol metabolism
RS2066702	ADH1B	GG	normal ethanol metabolism
RS671	ALDH2	GG	normal ethanol metabolism

Test details

Applicable situation

The alcohol metabolism test item uses three polymorphic loci on the *ALDH2* and *ADH1B* genes. This test result indicates the alcohol metabolism represented by the genotype combination of the three polymorphic loci detected by the user. But the test could not represent the user's real alcohol metabolism.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to alcohol metabolism.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.

-
2. An individual's nutritional metabolism is the result of the interaction of genes, environment, and living habits.
 3. The test result only predicts your alcohol metabolism at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true status. If you have nutritional supplement-related needs, you can consult authoritative nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutrition advice.

Test content

The test includes 3 polymorphic loci associated with the alcohol metabolism, such as rs1229984, rs671 and rs2066702.

Study population

The basis of the test is mainly from the research of Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to alcohol metabolism.

Vitamin E Nutritional Requirement

Vitamin E is a fat-soluble vitamin. Its hydrolysate is one of the most important antioxidants, which can delay aging, reduce the generation of wrinkles, reduce cell oxygen consumption etc.

My Vitamin E Nutritional Requirement

Slightly High

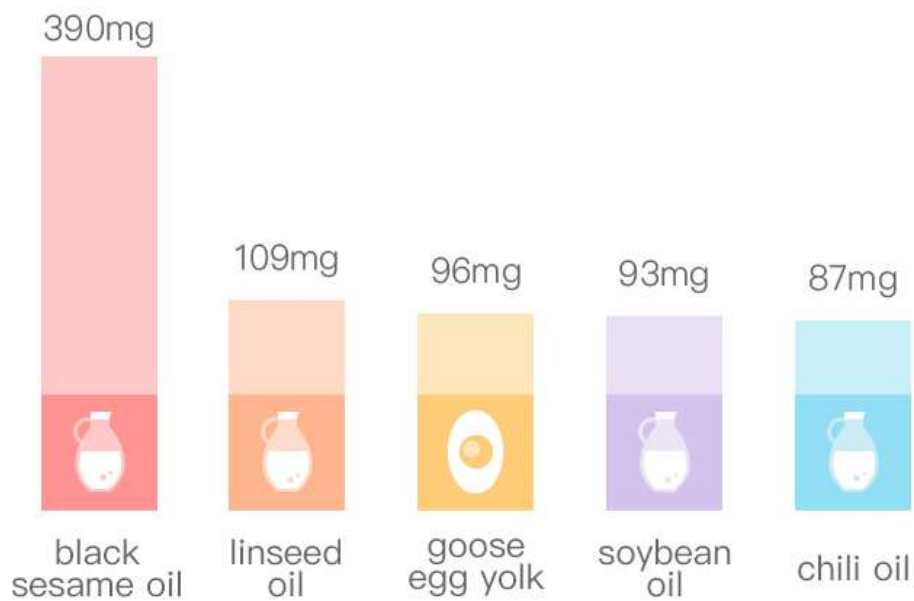
Intake recommendations

- Your vitamin E requirement is **slightly higher**. It is recommended to have Foods rich in vitamin E such as vegetable oils, nuts and leafy vegetables, or Additional supplement of vitamin E
- Your plasma vitamin E concentration is associated with gene variation, you can take vitamin E supplement appropriately
- You should pay attention to the levels of **Serum alpha-tocopherol** or **Effective serum vitamin E** available on your physical examination to guide vitamin E intake
- Vitamin E supplements should be taken if muscle pain or weakness, limb discoordination, etc. Symptoms of vitamin E deficiency occur
- People with long-term hunger, vegetarian diet and patients with hepatobiliary, pancreatic and intestinal diseases have higher requirements, and should pay attention to vitamin E intake
- If symptoms of vitamin E Poisoning such as headache, fatigue, diarrhea occur, reducing intake should be taken into consideration
- People taking anticoagulant drugs should take vitamin E with caution, may increase Risk of bleeding

* If you need additional vitamin E, please consult your doctor or professional nutrition consultant for specific dosage

Vitamin E rich foods

nutrient content per 100g edible part



Knowledge

Problems caused by deficiency

1. Muscle pain or weakness
2. Poor balance
3. Visual impairment
4. Loss of tendon reflex
5. Proprioception loss (sensation of movement in muscles, tendons, joints, etc.)
6. Early symptoms of anemia include fatigue, shortness of breath, bad temper, dizziness, headache, and difficulty concentrating
7. Retinopathy causes blurred vision and can lead to blindness in severe cases

Problems caused by excessive intake

Taking too much vitamin E can increase the risk of bleeding. In the long run, this can increase the risk of brain bleeding and stroke. Vitamin E supplements may have side effects in patients who are taking blood thinners and other medications, so consult your doctor before taking supplements.

Metabolism

Normally, vitamin E is absorbed through the gut as chylomicron into the circulatory system and transported to specific tissues and organs by specific proteins produced by the liver. If you take too

much vitamin E, since there is limited capacity for certain proteins, too much of it will be excreted in the stool and urine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2108622	CYP4F2	CC	low absorption of vitamin E
RS964184	ZPR1	CC	low absorption of vitamin E
RS6994076	intergenic	TT	normal vitamin E nutritional requirement

Test details

Applicable situation

The vitamin E nutritional requirement item uses 3 polymorphic loci on genes such as *ZPR1*. The test results indicate the vitamin E nutritional requirement represented by the genotype combination of the 3 polymorphic loci tested by the user, but the test does not mean the user's actual vitamin E nutritional requirement.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to vitamin E nutritional requirement.

How to use the test results

Notification

1. The test results are not applicable to clinical diagnosis.
2. An individual's nutritional requirement is the result of the interaction of genes, environment, and living habits.
3. The test results only predict your vitamin E nutritional requirement from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status. If

you have nutritional supplement-related needs, you can consult an authoritative nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutritional advice.

Test content

The test uses 3 polymorphic loci associated with vitamin E nutritional requirement including rs964184.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to vitamin E nutritional requirement.

Iron Nutritional Requirement

Iron is an essential component of human cells. It has hematopoietic function, participates in the production of hemoglobin and the synthesis of cytochrome and various enzymes, and promotes growth. Iron also transports and carries nutrients in the blood.

My Iron Nutritional Requirement

Slightly High

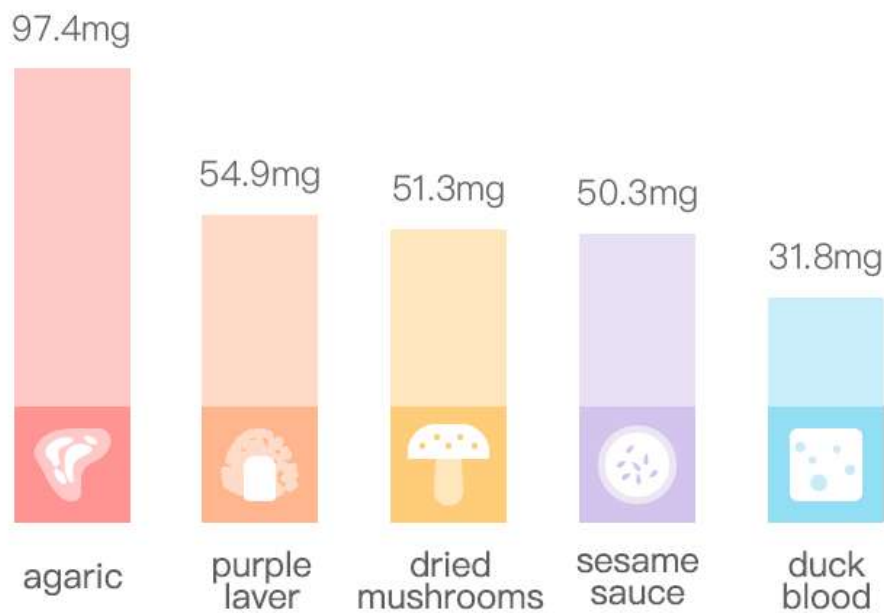
Intake recommendations

- Your iron requirement is **slightly higher**. It is recommended to take Iron-rich food, such as agaric fungus and duck blood, or Additional supplement of iron
- You have a mutation in the *TMPRSS6* gene and may have a low serum iron concentration
- You should pay attention to **Serum iron** and other Biochemical indicators in the physical examination to prevent the occurrence of iron deficiency anemia
- If anemia, pallor, etc. Iron deficiency symptoms occur, attention should be paid to iron supplement
- Iron needs of children, pregnant women are high and iron intake should be noted
- Have more fresh vegetables and fruits rich in **Vitamin C**, which can increase iron absorption
- If joint pain, liver disease, etc. Symptoms of excessive iron intake, reducing iron intake should be considered

* If you need additional iron, please consult your doctor or professional nutrition consultant for specific dosage

Iron rich foods

nutrient content per 100g edible part



Knowledge

Problems caused by deficiency

Iron deficiency can lead to anemia. Most of the time, symptoms start mild and develop slowly. Symptoms may include:

1. Feeling grumpy
2. Feeling tired more than usual
3. Headache
4. Difficulty in concentrating on matters

As anemia gets worse, symptoms may include: 1. Brittle nail disease 2. Dizziness when standing up 3. Pale skin 4. Shortness of breath 5. The tongue hurts

Problems caused by excessive intake

Consuming too much iron can cause iron overload, and early symptoms may include fatigue, joint pain, abdominal pain, and reduced libido. Later symptoms may include arthritis, liver disease, diabetes and abnormal skin tone.

Metabolism

Iron metabolism is the process in which iron is absorbed by organisms, transported, distributed, stored, utilized, transformed and excreted in organisms. The trivalent iron in food is converted to ferrous iron by gastric acid, which is absorbed into the duodenum and upper jejunum and then

transported into cells. Iron is eliminated daily mainly by gastrointestinal epithelial cells, bile, etc. This process is well controlled in advanced animals, including humans, to maintain a relative balance between iron absorption and excretion.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS12718598	IKZF1	CT	slightly high iron requirement
RS1800961	HNF4A	CC	high iron requirement
RS4820268	TMPRSS6	AG	slightly high iron requirement
RS855791	TMPRSS6	AG	slightly high iron requirement
RS57659670	DUOX2	TT	normal iron requirement

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The iron nutritional requirement item uses 6 polymorphic loci on the *TMPRSS6*, *IKZF1*, *SLCO1B1* and *HNF4A* genes. The test results indicate the iron nutritional requirement represented by the genotype combination of the 6 polymorphic loci tested by the user, but the test does not reflect the user's actual iron nutritional requirement.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to iron nutritional requirement.

How to use the test results

Notification

-
1. The test results are not applicable to clinical diagnosis.
 2. An individual's nutritional requirement is the result of the interaction of genes, environment, and living habits.
 3. The test results only predict your iron nutritional requirement from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status. If you have nutritional supplement-related needs, you can consult an authoritative nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutritional advice.

Test content

The test includes 6 polymorphic loci, such as rs4820268 and rs855791, associated with iron nutritional requirement.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to iron nutritional requirement.

Vitamin B12 Nutritional Requirement

Vitamin B12 is the only vitamin that contains metal. It is involved in the manufacture of bone marrow erythrocytes and preventing pernicious anemia.

My Vitamin B12 Nutritional Requirement

Slightly High

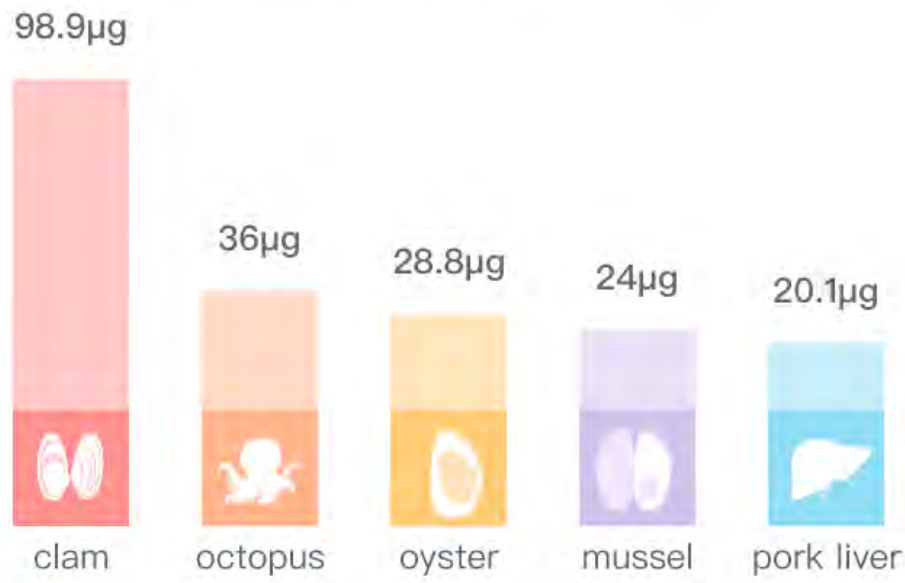
Intake recommendations

- Your vitamin B12 requirement is **slightly higher**. It is recommended to have eggs, meat, etc. Foods rich in vitamin B12 or Additional supplement for vitamin B12
- Vitamin B12 supplements should be taken if pallor, mouth ulcers etc. Symptoms of vitamin B12 deficiency occur
- Vegetarians are very low in vitamin B12, **Vegetarians** should take vitamin B12 supplements
- Vitamin B12 and **Vitamins B2, B6** are involved with Folic acid metabolism
- Pregnant and lactating women who have a high need for folic acid should also Appropriately increase their intake of vitamin B12 if folic acid supplements are taken
- If allergy, folic acid deficiency, etc. Symptoms of vitamin B12 overdose occur, intake should be reduced

* For additional vitamin B12 supplements, please consult your doctor or professional nutrition consultant for specific dosage

Vitamin B12 rich foods

nutrient content per 100g edible part



Knowledge

Problems caused by deficiency

1. Fatigue, dizziness when standing up or exerting force
2. Shortness of breath during exercise
3. Swelling, redness of tongue or bleeding gums
4. Difficulty concentrating
5. Paled face
6. Diarrhea or constipation
7. Dementia (in severe cases)
8. Depression
9. Ataxia
10. Numbness in hands and feet

Problems caused by excessive intake

Vitamin B12 is one of the least needed daily in the human body, and excess vitamin B12 can have toxic side effects. Allergic reactions such as asthma, urticaria, eczema, facial swelling, and chills have been reported with excessive vitamin B12 injection, as well as nerve irritation, precardiac pain, and palpitations. Too much vitamin B12 can also lead to a deficiency in folic acid.

Metabolism

Vitamin B12 in food combines with protein and enters the human digestive tract, where it is released under the action of gastric acid, pepsin and trypsin and binds to a glycoprotein endogenous factor (IF) secreted by gastric mucosal cells. Vitamin B12-IF complex is absorbed in the ileum. Vitamin B12 is stored in small amounts, about 2~3mg in the liver. It is discharged mainly from urine and partly from bile.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1047781	FUT2	AT	slightly high vitamin B12 requirement
RS10515552	intergenic	TT	high vitamin B12 requirement
RS12377462	intergenic	CT	slightly high vitamin B12 requirement
RS2298585	MS4A3	CC	high vitamin B12 requirement
RS3760775	intergenic	TT	normal vitamin B12 requirement

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The vitamin B12 nutritional requirement item uses 12 polymorphic loci on genes such as *MS4A3*, *CLYBL*, and *FUT2*. The test results indicate the vitamin B12 nutritional requirement represented by the genotype combination of the 12 polymorphic loci tested by the user, but the test does not mean the user's actual vitamin B12 nutritional requirement.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to vitamin B12 nutritional requirement.

How to use the test results

Notification

1. The test results are not applicable to clinical diagnosis.
2. An individual's nutritional requirement is the result of the interaction of genes, environment, and living habits.
3. The test results only predict your vitamin B12 nutritional requirement from the genetic level.
Since the test does not involve consideration of other factors, it does not mean your true health status. If you have nutritional supplement-related needs, you can consult an authoritative nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutritional advice.

Test content

The test includes 12 polymorphic loci associated with vitamin B12 nutritional requirement, including rs41281112, rs2298585 and rs1047781.

Population samples

The basis of the test is mainly from the research of Chinese, Indian and European.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to vitamin B12 nutritional requirement.

Vitamin A Nutritional Requirement

Also known as retinol, Vitamin A is closely associated with low light vision of the eyes. It can also maintain normal epithelial tissue differentiation and inhibit skin keratosis.

My Vitamin A Nutritional Requirement

**Slightly High, consider
supplementary Vitamin A intake**

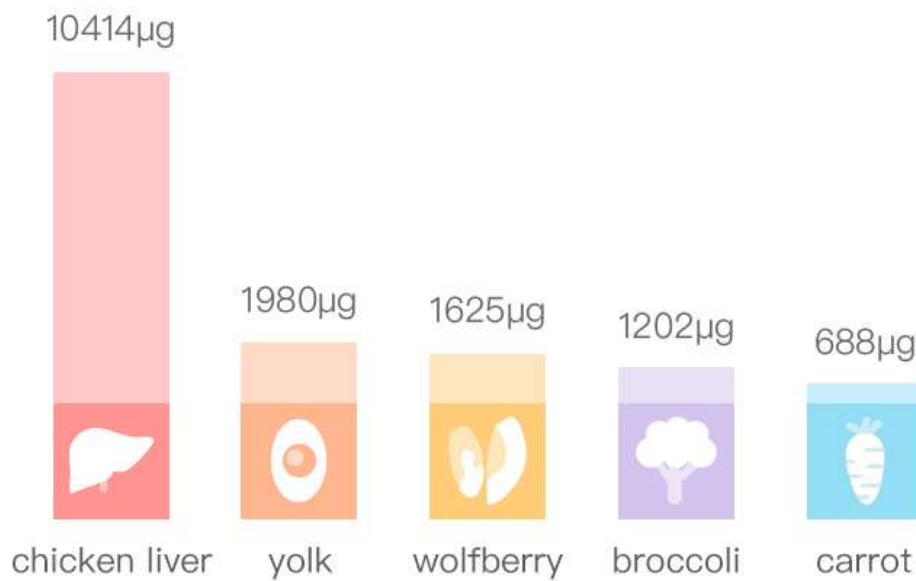
Intake recommendations

- Your vitamin A requirement is **slightly higher**. It is recommended to take animal liver, orange vegetables, etc. Food rich in vitamin A or β -carotene or Additional supplement Vitamin A and β -carotene.
- You have a mutation in the upstream loci of the *BCMO1* gene. Excessive intake of β -carotene should be avoided as excessive supplementation may cause Adverse reactions
- You have a mutation in the upstream site of the *BCMO1* gene. When you choose to take extra vitamin A, you should pay special attention to dry skin, peeling, muscle pain and other Vitamin A overdose symptoms, and reduce the use of vitamin A if it occurs
- If you have dry eyes, night blindness, etc. Vitamin A deficiency symptoms, attention should be paid to supplement vitamin A
- Moderate intake of **β -carotene** helps Vitamin A production
- Pregnant women should strictly control the intake of vitamin A, excess may lead to fetal malformation, but supplementation of **β -carotene** is safer

* For additional β -carotene or Vitamin A supplements, consult your doctor or professional nutrition consultant for specific dosage

Vitamin A rich foods

nutrient content per 100g edible part



Knowledge

Problems caused by deficiency

One of the basic functions of vitamin A is to help the formation of pigment in the retina. Typical symptoms of vitamin A deficiency are dry eyes and night blindness. Vitamin A deficiency can make symptoms of low iron levels worse and lead to anemia. In addition to these symptoms, vitamin A deficiency can lead to dry skin, hair loss, and an increased risk of diarrhea and measles and death.

Problems caused by excessive intake

Because vitamin A is fat-soluble, it will accumulate in the liver, resulting in hypervitaminosis, which includes the following symptoms:

1. Diplopia
2. Dry skin and lips or cracked lips
3. The hair loss
4. Headache, dizziness, nausea
5. Muscle pain, swelling and pain at long bone joints
6. Ataxia: Acute overdose of large amounts of vitamin A can lead to coma or death

Metabolism

In the body, vitamin A comes in three main forms: retinol, retinaldehyde and retinoic acid. When vitamin A accumulates in the liver or travels in the blood, it appears mainly as retinol; When it helps us see in the eye, it's retinaldehyde; As they regulate gene expression and control the cell's life cycle,

they often turn into retinoic acid on the spot. Vitamin A in the animal products we eat is often retinol (fatty acids combined with retinol, such as retinol palmitate and retinol acetate), which is reconverted to retinol in the body.

Plant-based foods contain no vitamin A at all, but many of them contain another compound: carotenoids. This is a large family of compounds with more than 600 members, such as α -carotene, β -carotene, β -cryptoflavin, lycopene, keratin, lutein and zeaxanthin. The first three are the main ones we can use to efficiently synthesize vitamin A (retinaldehyde) in our bodies, especially β -carotene.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS11645428	BCMO1	GG	low vitamin A conversion efficiency
RS6420424	PKD1L2	AA	low vitamin A conversion efficiency
RS6564851	BCMO1	TT	normal vitamin A conversion efficiency

Test details

Applicable situation

The vitamin A nutritional requirement item uses 3 polymorphic loci on *BCMO1* gene. The test results indicate vitamin A nutritional requirement represented by the genotype combination of the 3 polymorphic loci of the user, but the test does not reflect the user's actual vitamin A nutritional requirement.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to vitamin A nutritional requirement.

How to use the test results

Notification

-
1. The test results are not applicable to clinical diagnosis.
 2. An individual's nutritional requirement is the result of the interaction of genes, environment, and living habits.
 3. The test results only predict your vitamin A nutritional requirement from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status. If you have nutritional supplement-related needs, you can consult an authoritative nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutritional advice.

Test content

The test includes 3 polymorphic loci associated with vitamin A nutritional requirement: rs6420424, rs6564851, and rs11645428.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to vitamin A nutritional requirement.

Vitamin C Nutritional Requirement

Vitamin C is a water-soluble vitamin that participates in the metabolism of substances in the body and has antioxidant capacity.

My Vitamin C Nutritional Requirement

Slightly High

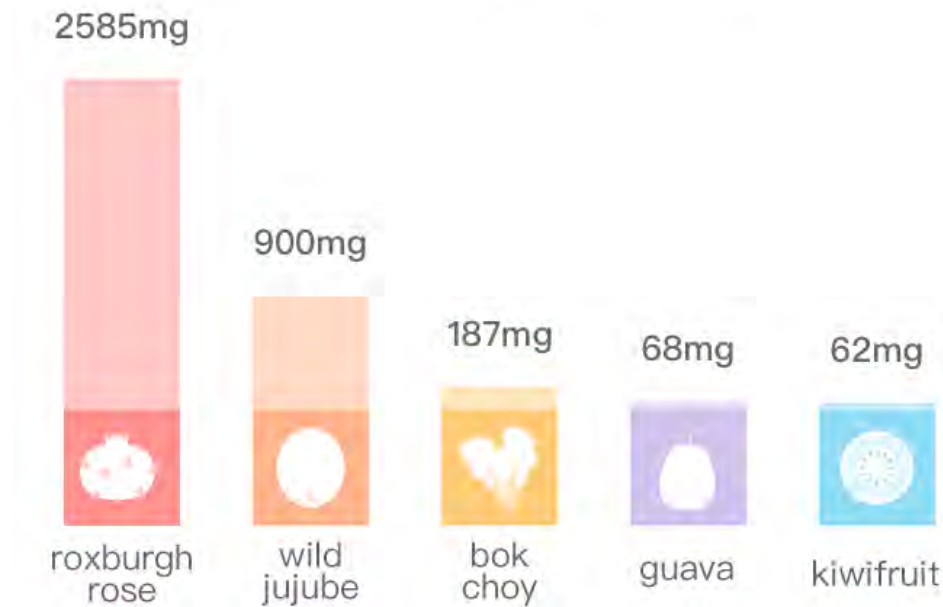
Intake recommendations

- Your vitamin C nutritional requirement is **slightly higher**, it is recommended to eat fresh vegetables, fruits and other Foods rich in vitamin C, or take in Additional supplement of vitamin C.
- There are multiple mutations in your vitamin C transporter gene, and your plasma vitamin C concentration may be low.
- Pay attention to improving Cooking methods to reduce the loss of vitamin C.
- Vitamin C supplements should be taken if Symptoms of vitamin C deficiency such as fatigue, irritability, weight loss, muscle or joint pain occur.
- Vitamin C intake is with higher requirement with respect to Pregnancy, breastfeeding, inflammation, surgery, smoking and other conditions.
- If Symptoms of vitamin C toxicity such as abdominal distention and diarrhea occur, consider reducing vitamin C intake.
- Vitamin C helps different types of white blood cells to play an immune role, also known as "immune Vitamins".

* If you need additional vitamin C supplement, please consult your doctor or professional nutrition consultant for specific dosage

Vitamin C rich foods

nutrient content per 100g edible part



Knowledge

Problems caused by deficiency

The human body cannot actively synthesize vitamin C, but it has a certain storage capacity. Therefore, short-term vitamin C deficiency will not immediately appear corresponding symptoms. When the diet lacks fresh vegetables and fruits, or absorption disorders, long-term use of specific drugs and alcoholism, it may lead to vitamin C deficiency. The following symptoms are common:

1. Weakness, depression and pale complexion; Children may not gain weight and be irritable.
2. Iron absorption disorder, anemia, etc.
3. Bleeding, manifested as skin ecchymosis, purpura, loose teeth, epistaxis, or even intracranial hemorrhage, convulsion, shock and death.

Problems caused by excessive intake

After excessive vitamin C intake, it will be quickly excreted from the urine, so there is only very low acute toxicity. Ingestion of more than 2-3 grams may cause indigestion. Other symptoms of overdose may include nausea, abdominal pain and diarrhea.

Metabolism

Vitamin C is also called ascorbic acid. The human body cannot synthesize vitamin C. It usually needs to be ingested from food, generally absorbed above the small intestine and finally excreted from urine. vitamin C can participate in the metabolism of many substances in the body and plays an important role in maintaining immune function and vascular integrity. At the same time, as an

antioxidant, it can inhibit the formation of tyrosinase and has the effect of whitening and lightening spots.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS11950646	SLC23A1	GG	high vitamin C nutritional requirement
RS33972313	SLC23A1	CC	normal vitamin C nutritional requirement
RS6053005	SLC23A2	TT	normal vitamin C nutritional requirement
RS6133175	SLC23A2	AG	high vitamin C nutritional requirement

Test details

Applicable situation

The vitamin C nutritional requirement item uses 4 polymorphic loci on and between genes such as *SLC23A1* and *SLC23A2*, etc. The test result shows the vitamin C nutritional requirement represented by the genotype combination at the four polymorphic loci tested by the user, and the test cannot represent the user's real vitamin C nutritional requirement.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technology and the level of scientific cognition, the test report may not cover all genes or loci that related to the vitamin C nutritional requirement.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. An individual's nutritional metabolism is the result of the interaction of genes, environment, and living habits.

3. This test only evaluates your vitamin C nutritional requirement at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status. If you have nutritional supplement related needs, you can consult an authoritative nutritionist, clinician or professional for professional nutritional advice based on the results of this genetic test if necessary.

 Test content

The test includes 4 polymorphic loci related to vitamin C nutritional requirement, such as rs6133175 and rs6053005.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to vitamin C nutritional requirement.

Vitamin K Nutritional Requirement

Vitamin K is a type of vitamin that has blood coagulation function and participates in bone metabolism.

My Vitamin K Nutritional Requirement

High, consider supplementary
Vitamin K intake

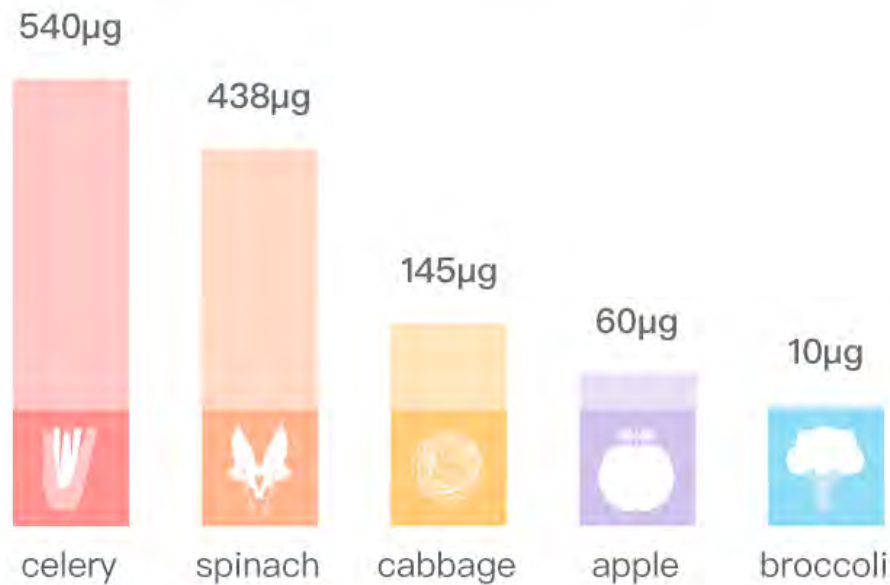
Intake recommendations

- Your need for vitamin K is **high**. It is recommended to Normally take in Foods rich in vitamin K such as green leafy vegetables, or supplement additional vitamin K1 or K2.
- Vitamin K1 in vegetables can be converted into K2 in the intestine, which is easily absorbed by the human body
- There is a homozygous mutation in your *VKORC1* gene, the Formation efficiency of coagulation factor is low, and you need to increase the requirement for vitamin K, and appropriately reduce the dosage of **Warfarin**.
- If there are Symptoms of vitamin K deficiency such as congestion, hematoma and nosebleed, pay attention to vitamin K supplementation.
- Newborns are more prone to vitamin K deficiency, which can be prevented by additional injection of vitamin K.
- When you choose to supplement additional vitamin K1/K2, you should pay attention to the Symptoms of vitamin K overdose such as allergy. If it occurs, you should reduce the use.
- People with liver function problems and pregnant women who supplement vitamin K may have corresponding risks.
- Vitamin K supplementation may affect the efficacy of Anticoagulants.

* If you need additional vitamin K supplement, please consult your doctor or professional nutrition consultant for specific dosage.

Vitamin K rich foods

nutrient content per 100g edible part



Knowledge

Problems caused by deficiency

Vitamin K is not easily deficient because it widely exists in food and can be synthesized by intestinal microorganisms. vitamin K deficiency may occur in people with impaired liver function, bowel disease, taking antibiotics and preterm infants. vitamin K deficiency may cause anemia, bruises, nosebleed and gum bleeding. Women may have excessive menstruation.

Problems caused by excessive intake

Excessive intake of vitamin K in natural form (including K1 and K2) has no obvious side effects and may cause allergic reaction. In contrast, excessive intake of vitamin K3 is obviously toxic, which may cause allergic reaction, hemolytic anemia and damage to hepatocytes. In addition, excessive intake of vitamin K may affect the efficacy of anticoagulants such as farwarrin.

Metabolism

Vitamin K mainly exists in the form of K1, K2, K3 and K4. It is closely related to the synthesis of four coagulation factors (prothrombin, coagulation factors VII, IX and X) in the liver and plays an important role in coagulation function. vitamin K can act on osteoblasts and promote bone tissue calcification, especially in the elderly. There is a positive correlation between bone mineral density and vitamin K. vitamin K is not easily deficient because it widely exists in food and can be synthesized by intestinal microorganisms. But newborns may be prone to vitamin K deficiency.

It should be noticed that vitamin K supplementation may have related risks for people with liver function problems and pregnant women, and may affect the efficacy of anti blood coagulants.

Therefore, it is recommended to formulate a supplement scheme after consulting with qualified doctors.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2108622	CYP4F2	CC	moderate vitamin K plasma concentration
RS9923231	VKORC1	TT	high vitamin K nutritional requirement

Test details

Applicable situation

The vitamin K nutritional requirement item uses 2 polymorphic loci on and between genes such as *CYP4F2*. The test result shows the vitamin K nutritional requirement represented by the genotype combination at the two polymorphic loci tested by the user, and the test cannot represent the user's real vitamin K nutritional requirement.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technology and the level of scientific cognition, the test report may not cover all genes or loci that related to the vitamin K nutritional requirement.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. An individual's nutritional metabolism is the result of the interaction of genes, environment, and living habits.
3. This test only evaluates your vitamin K nutritional requirement at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status. If you have nutritional supplement needs, you can consult an authoritative nutritionist, clinician or

professional for professional nutritional advice based on the results of this genetic test if necessary.

 Test content

The test includes 2 polymorphic loci related to vitamin K nutritional requirement, thus, rs2108622 and rs9923231.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to vitamin K nutritional requirement.

Vitamin B6 Nutritional Requirement

Vitamin B6 is a kind of water-soluble vitamin related to the metabolism of various nutrients such as amino acid metabolism.

My Vitamin B6 Nutritional Requirement

Slightly High

Intake recommendations

- Your vitamin B6 requirement is **slightly higher**, it is recommended to consume Food rich in vitamin B6 such as soybeans and animal liver and so on, or Additional supplement of vitamin B6.
- There are polymorphic loci in your *NBPF3* gene and *ALPL* gene, which affect the concentration of vitamin B6.
- The use of certain drugs, alcoholism, high temperature environments, and excessive stress may lead to vitamin B6 deficiency.
- If you have Symptoms of vitamin B6 deficiency such as paleness and oral ulcers, you should pay attention to vitamin B6 supplement.
- Vitamin B6, **vitamin B12 and vitamin B2** participate in the Folate metabolism.
- Pregnant women and lactating women have a high requirement for **folic acid**. If you need to supplement folic acid, you should also Appropriate increase the intake of vitamin B6.
- If you have Symptoms of vitamin B6 overdose such as limb pain and numbness, the dosage should be reduced.

* If you need additional vitamin B6, please consult your doctor or professional nutrition consultant for the specific dosage.

Vitamin B6 rich foods

nutrient content per 100g edible part



Knowledge

Problems caused by deficiency

Long-term insufficient intake of Vitamins, poor absorption, or long-term use of specific drugs can cause vitamin B6 deficiency. In addition, alcoholism, living or working in high temperature environments, and excessive stress can also cause vitamin B6 deficiency. Typical symptoms include:

1. Seborrheic dermatitis-like rash, conjunctivitis, stomatitis, and glossitis.
2. Neuropathy, drowsiness, neurological disorders; anemia; nausea and vomiting, lack of appetite, etc.

Problems caused by excessive intake

Generally, vitamin B6 supplementation through food rarely causes excessive problems, because it will be excreted in urine when kidney function is normal. However, long-term excessive supplementation may cause side effects, including pain, peripheral neuropathy, symptoms such as pain and numbness in limbs. High intake of pregnant women may lead to vitamin B6 dependence in neonates, so it should be taken under the guidance of a doctor.

Metabolism

Pyridoxal 5'-phosphate (PLP) is the active form of vitamin B6 in the body. It can participate in more than 100 physiological reactions as a coenzyme. It is mainly involved in amino acid metabolism and is of great significance to protein metabolism. At the same time, it is also involved in sugar and fatty acid metabolism and neurotransmitter synthesis. vitamin B6 cannot be stored in the body for a long

time, and it mainly depends on supplementation from food. Foods rich in vitamin B6 include yeast, fish, meat, eggs, milk, grains, and vegetables.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4654748	NBPF3	CT	slightly high vitamin B6 nutritional requirement
RS1256335	ALPL	AA	normal high vitamin B6 nutritional requirement

Test details

Applicable situation

The vitamin B6 nutritional requirement item uses 2 polymorphic loci on *ALPL* and *NBPF3* gene. The test results indicate the vitamin B6 nutritional requirement represented by the genotype combination at the two polymorphic loci you tested, and the test does not mean your true vitamin B6 nutritional requirement.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technological means and the level of scientific cognition, the test may not cover all genes or loci that relate to the vitamin B6 nutritional requirement.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. An individual's nutritional metabolism is the result of the interaction of genes, environment, and living habits.
3. This test only evaluates your vitamin B6 nutritional requirement at the genetic level, since the test does not involve consideration of other factors, it does not mean your true situation. If you have

relevant nutritional requirement, you can consult nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutritional advice.

Test content

The test includes 2 polymorphic loci related to the vitamin B6 nutritional requirement, rs1256335 and rs4654748.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to vitamin B6 nutritional requirement.

Vitamin D Nutritional Requirement

Vitamin D is a type of fat-soluble vitamin that can maintain the stability of calcium and phosphorus and many other biological effects.

My Vitamin D Nutritional Requirement

Slightly High

Intake recommendations

- Your vitamin D requirement is **slightly higher**, it is recommended to consume Food rich in vitamin D such as fish oil, liver, egg yolk and so on, or to consume Additional supplement of vitamin D and to increase sun exposure.
- There are variations in the genes associated with your plasma vitamin D concentration, so vitamin D should be supplemented appropriately.
- You should pay attention to the serum **25-hydroxyvitamin D**, bone mineral density and other indicators in the physical examination indicators to guide the intake of Vitamin D and Calcium.
- If you have Symptoms of vitamin D deficiency such as muscle pain, muscle weakness, fatigue, you should pay attention to vitamin D supplement.
- The elderly, people with insufficient sun exposure, patients with kidney or liver disease, and dark-skinned people (especially blacks) have higher requirement, and they should pay attention to vitamin D intake.
- If you have Symptoms of vitamin D toxicity such as loss of appetite, constipation, you should pay attention to reducing vitamin D intake.
- Patients with hypercalcemia, kidney stones and arteriosclerosis should be cautiously supplemented with vitamin D, which may cause Related diseases.

* If you need additional vitamin D supplement, please consult your doctor or professional nutrition consultant for the specific dosage.

Vitamin D rich foods

nutrient content per 100g edible part



Knowledge

Problems caused by deficiency

A vitamin D-deficient diet and insufficient sun exposure can cause vitamin D deficiency, which may lead to osteomalacia in adults, rickets in children, and neurological symptoms such as hyperhidrosis and irritability.

Problems caused by excessive intake

Generally, the content of vitamin D in food is not high, and there is generally no problem of excessive intake. Long-term excessive use of vitamin D may cause side effects such as hypercalcemia, constipation, headache, loss of appetite, and musculoskeletal pain. Vitamin D toxicity caused by excessive intake can lead to weight loss, itch, soft tissue calcification, and calcification of cardiac and renal tissues.

Metabolism

Vitamin D is also known as the anti-rickets Vitamin. The main members are VD2 (ergocalciferol) and VD3 (cholecalciferol). Vitamin D can be absorbed together with fat in the intestine, and the human body can also synthesize vitamin D autonomously through sunlight. Vitamin D can improve the body's absorption of calcium and phosphorus, promote growth and bone calcification, and also has a certain effect on the prevention of heart disease and diabetes. The content of vitamin D in general food is low. Natural vitamin D mainly comes from animal foods, such as fish oil, liver, and egg yolk.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS10877012	METTL1	GG	high vitamin D nutritional requirement
RS1155563	GC	CT	high vitamin D nutritional requirement
RS12512631	intergenic	TT	slightly high vitamin D nutritional requirement
RS12794714	CYP2R1	GG	normal vitamin D nutritional requirement
RS2282679	GC	TT	normal vitamin D nutritional requirement

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The vitamin D nutritional requirement item uses 8 polymorphic loci on genes such as *GC*. The test results indicate the vitamin D nutritional requirement represented by the genotype combination at the 8 polymorphic loci you tested, and the test does not mean your true vitamin D nutritional requirement.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technological means and the level of scientific cognition, the test may not cover all genes or loci that relate to the vitamin D nutritional requirement.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. An individual's nutritional metabolism is the result of the interaction of genes, environment, and living habits.

3. This test only evaluates your vitamin D nutritional requirement at the genetic level, since the test does not involve consideration of other factors, it does not mean your true situation. If you have relevant nutritional requirement, you can consult nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutritional advice.

 Test content

The test includes 8 polymorphic loci related to the vitamin D nutritional requirement, such as rs1155563, rs12512631.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to vitamin D nutritional requirement.

Zinc Nutritional Requirement

Zinc is one of the essential trace elements for the human body. It participates in the completion of many normal physiological functions, and plays an extremely important role in the growth and intellectual development of children.

My Zinc Nutritional Requirement

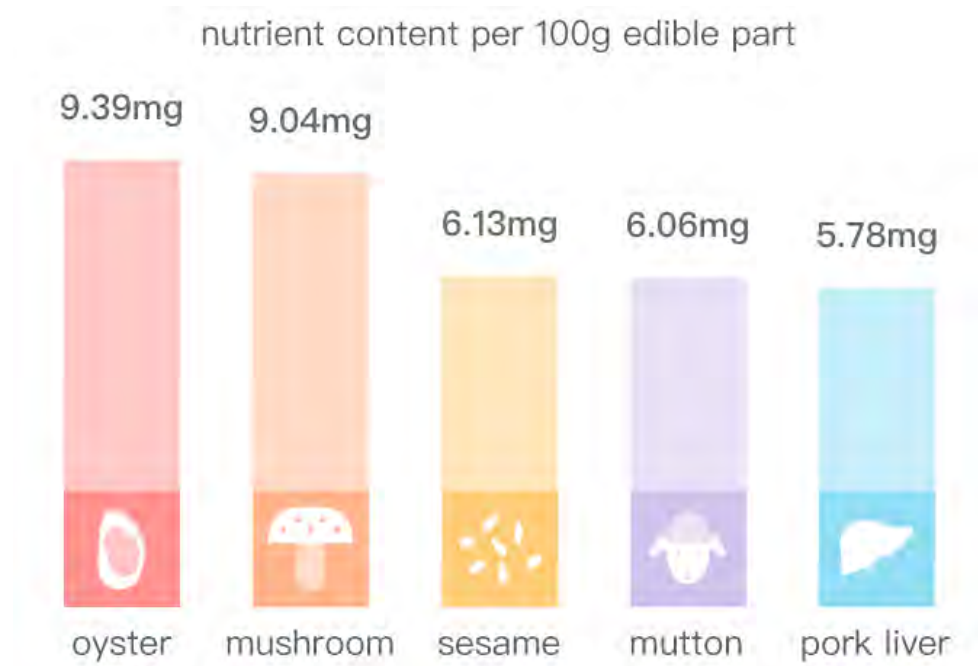
High

Intake recommendations

- Your zinc requirement is **high**, it is recommended to consume Food rich in zinc such as seafood, nuts and so on, or to consume Additional supplement of zinc.
- There are mutations in your Zinc metabolism gene, which may lead low concentration of zinc in your blood.
- You should pay attention to indicators such as **blood zinc** and **hair zinc** in the physical examination indicators to guide zinc intake.
- If you have Symptoms of zinc deficiency such as loss of appetite and growth retardation, you should pay attention to zinc supplement.
- Pay attention to the Zinc requirement and intake of children and pregnant women.
- Vegetarians, people with a high-fiber diet, people with a high intake of calcium supplements and iron supplements, should supplement zinc 15 mg per day additionally.
- If you have Symptoms of excessive zinc intake such as nausea and vomiting, the dosage should be reduced.

* If you need additional zinc supplement, please consult your doctor or professional nutrition consultant for the specific dosage.

Zinc rich foods



Knowledge

What is zinc

Zinc is a trace element, which cannot be synthesized in the body and can only be provided by external food. Zinc is a component of many important enzymes in the human body and an essential element for the synthesis of insulin. It plays an important role in the synthesis of protein and nucleic acid, in maintaining the integrity of red blood cells, and in the process of hematopoiesis. It is a key element to promote growth and development, especially for the development of children's brain and nervous system.

Functions of zinc

Zinc has three basic functions: catalytic function, structural function and regulation function. Through these three functions, zinc plays an important role in human development, cognitive behavior, wound healing, taste and immune regulation.

Symptoms of deficiency

1. Anorexia: Taste buds function diminished, taste function is reduced, the appetite is lost, and digestive ability is also weakened when zinc is lacking.
2. Growth retardation: Zinc deficiency hinders the synthesis of protein and reduces food intake, affecting the growth and development of children. In severe cases, dwarfism may occur.
3. Pica: Children with zinc deficiency may like to eat soil, walls, paper, cinder or other foreign objects, etc. Adults with zinc deficiency also have reports of liking to eat soil.

- 4. Prone to infection: Zinc-deficient people have reduced immune function and are susceptible to various infectious diseases, including diarrhea.
- 5. Skin mucosal symptoms: When zinc deficiency is severe, the whole skin may have rash, bullous dermatitis, and recurrent oral ulcer.
- 6. Fetal growth retardation: Pregnant women with severe zinc deficiency can lead to fetal growth retardation and various malformations, including neural tube malformations.
- 7. Others: Due to vitamin A metabolism disorder, serum vitamin A is reduced, the dark adaptation time is prolonged, and night blindness occurs.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS11126936	SLC30A3	GT	slightly high zinc nutritional requirement
RS13266634	SLC30A8	CC	high zinc nutritional requirement
RS233804	SLC39A8	CC	high zinc nutritional requirement
RS4872479	SLC39A14	GT	slightly high zinc nutritional requirement

Test details

Applicable situation

The zinc nutritional requirement item uses 4 polymorphic loci on genes such as *SLC30A8*, *SLC39A8*. The test results indicate the zinc nutritional requirement represented by the genotype combination at the 4 polymorphic loci you tested, and the test does not mean your true zinc nutritional requirement.

Notification

- 1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
- 2. Limited by the current technological means and the level of scientific cognition, the test may not cover all genes or loci that relate to the zinc nutritional requirement.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. An individual's nutritional metabolism is the result of the interaction of genes, environment, and living habits.
3. This test only evaluates your vitamin zinc nutritional requirement at the genetic level, since the test does not involve consideration of other factors, it does not mean your true situation. If you have relevant nutritional requirement, you can consult nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutritional advice.

Test content

The test includes 4 polymorphic loci related to the zinc nutritional requirement, such as rs11126936, rs233804.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to zinc nutritional requirement.

Vitamin B2 Nutritional Requirement

Vitamin B2 is an important water-soluble vitamin that regulates the metabolism of amino acids, fatty acids and carbohydrates.

My Vitamin B2 Nutritional Requirement

Normal

Intake recommendations

- Your requirement for vitamin B2 is **normal**, it is recommended to take in Normal supplement of Foods rich in vitamin B2 such as soybeans, animal liver.
- The use of specific drugs and alcohol abuse may lead to vitamin B2 deficiency.
- If you have Symptoms of vitamin B2 deficiency such as oral ulcers, skin pimples, you should pay attention to vitamin B2 supplementation.
- Vitamin B2 and **vitamin B12 and B6** participate in Folate metabolism together.
- Pregnant women and lactating women have a high requirement for **folic acid**. If folic acid is supplemented, the intake of vitamin B2 should also be Appropriately increased.

* If you need additional vitamin B2 supplements, please consult your doctor or professional nutrition consultant for the specific dosage.

Vitamin B2 rich foods

nutrient content per 100g edible part



Knowledge

Problems caused by deficiency

Vitamin B2 cannot be stored in the body for a long time. Insufficient intake, alcoholism and some drugs can all lead to vitamin B2 deficiency. Vitamin B2 deficiency can cause a variety of symptoms, mainly including:

1. Oral-genital syndrome: Mucous membrane ulcers throughout the mouth, cracking, glossitis, etc.; Eyes are prone to fatigue, conjunctival congestion and inflammation; skin papules, scrotal inflammation.
2. Children with long-term vitamin B2 deficiency have growth retardation, which is easy to cause iron deficiency anemia.
3. Extreme vitamin B2 deficiency can cause deficiencies of other B vitamins.

Problems caused by excessive intake

It is generally believed that vitamin B2 is relatively safe. The ability of human body to store vitamin B2 is limited, and excessive vitamin B2 will be excreted in free form. However, when the content of vitamin B2 in the body is very high, it can also cause symptoms such as itching, sensitivity to light, and yellowing of urine.

Metabolism

Vitamin B2 (riboflavin) plays an important role in the metabolism of protein, fat, carbohydrates, etc. Generally, it participates in energy metabolism in the form of flavin adenine dinucleotide (FAD) and

flavin mononucleotide (FMN). In addition to participating in metabolism, vitamin B2 is also related to cell growth, repair, iron absorption and storage, and mobilization. It also has an impact on nerve cells, retinal metabolism, the release of corticotropin from the pituitary gland, and the growth and development of the fetus.

Vitamin B2 is a water-soluble vitamin that is widely present in various foods, such as animal liver, eggs, lean meat, yeast, spinach, etc. Generally, the content of meat is higher than that of plant foods. In addition, it is generally recommended to use compound vitamin B when supplementing through drugs to maintain a balance with other B vitamins.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1801133	MTHFR	GG	normal vitamin B2 nutritional requirement

Test details

Applicable situation

The vitamin B2 nutritional requirement item uses one polymorphic locus on *MTHFR* gene. The test results showed the vitamin B2 nutritional requirement represented by the genotype combination of the user at one polymorphic locus tested. The test cannot represent the user's real vitamin B2 nutritional requirement.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technological means and the level of scientific cognition, this test may not cover all genes or loci related to vitamin B2 nutritional requirement.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.

-
2. An individual's nutritional metabolism is the result of the interaction of genes, environment, and living habits.
 3. The test result only predicts your vitamin B2 nutritional requirement at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status. If you have nutritional supplement related needs, you can consult authoritative nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutritional advice.

Test content

One polymorphic locus related to vitamin B2 nutritional requirement was detected, including rs1801133.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to vitamin B2 nutritional requirement.

Magnesium Nutritional Requirement

Magnesium is an essential macro-mineral nutrient for the human body. It is particularly important for metabolism, the functioning of muscle cells, and the health of bones.

My Magnesium Nutritional Requirement

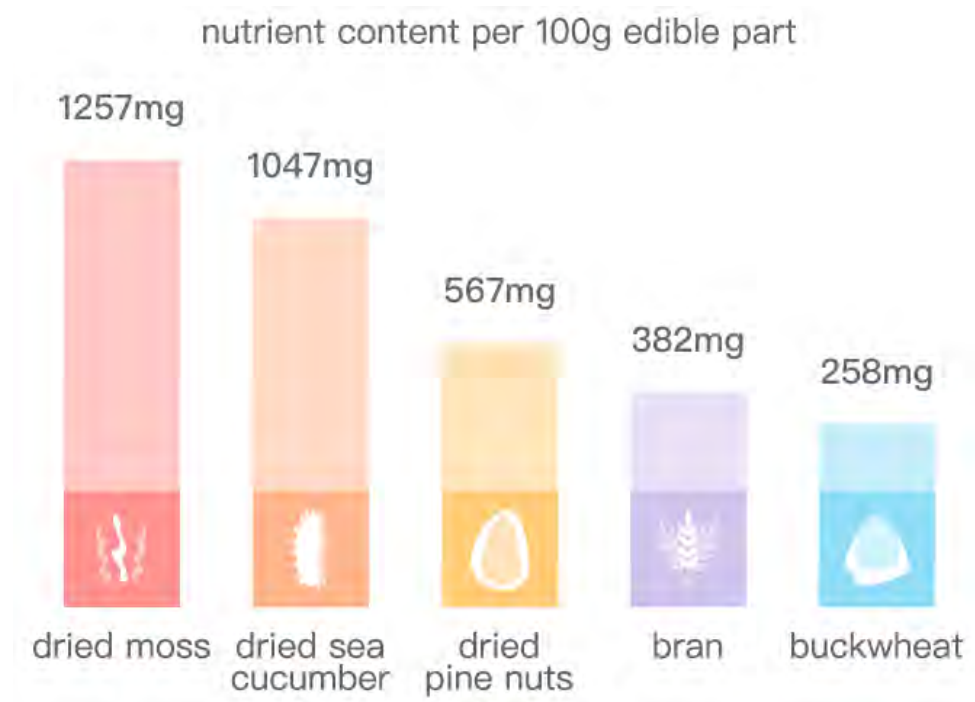
Slightly High

Intake recommendations

- Your requirement of magnesium is **slightly higher**, it is recommended to take in Additional supplement of Foods rich in magnesium such as green leafy vegetables, nuts, etc.
- If there are Manifestations of magnesium deficiency such as muscle cramps, fatigue, etc., pay attention to supplement **magnesium**.
- The magnesium requirement of the elderly is higher, so attention should be paid to magnesium intake.
- Excessive calcium and vitamin D supplementation will deplete magnesium, so attention should be paid to magnesium intake.
- If you consume too much sugar and often drink soda or other sugary drinks, there may be a risk of magnesium deficiency.
- Magnesium may also be lost through stress, lack of sleep, alcohol consumption and the use of Prescription drugs.
- Digestive system diseases such as Crohn's disease affect the body's absorption of magnesium, so attention should be paid to magnesium intake.
- If you have Symptoms of excessive magnesium intake such as nausea and vomiting, you should consider reducing your intake.

* If you need additional magnesium supplements, please consult your doctor or professional nutrition consultant for the specific dosage.

Magnesium rich foods



Knowledge

What is magnesium

Magnesium is a macronutrient needed in all organs of the human body, especially the heart, muscles and kidneys. Magnesium is the main cation in human cells, which is concentrated in mitochondria, second only to potassium and phosphorus.

The effect of magnesium on the human body

1. Magnesium is an activator of enzymes: Magnesium activates a variety of enzymes, such as hexokinase, and participates in many important metabolic processes in the body, including the metabolism of proteins, fats and carbohydrates, oxidative phosphorylation, ion transport, the generation and transmission of nerve impulses, muscle contraction, etc.
2. The effect of magnesium on bones: Magnesium is an element necessary for the structure and function of bone cells, allowing bones to grow and maintain. Magnesium can affect bone resorption.
3. The effect of magnesium on the cardiovascular system: Magnesium mainly acts on the peripheral vascular system and causes vasodilation. Low dose application can cause flushing, sweating and warmth, which is related to body temperature regulation.
4. The effect of magnesium on the gastrointestinal tract: Alkaline magnesium salt can neutralize gastric acid. Magnesium ions are absorbed slowly in the intestinal cavity, promote water retention, and cause catharsis.

Deficiency or excessiveness

1. Deficiency: When magnesium is deficient, symptoms such as decreased appetite, growth stagnation, hair loss, skin damage, weakness, edema, neuromuscular hyperexcitability, irregular heartbeat, organ calcification, and degeneration will be manifested.
2. Excessiveness: When there is too much magnesium in the serum, nausea, vomiting, peripheral vasodilation, decreased blood pressure, fever and thirst may occur.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS13146355	SHROOM3	GG	high magnesium nutritional requirement
RS3925584	intergenic	CT	slightly high magnesium nutritional requirement
RS4072037	MUC1	TT	normal magnesium nutritional requirement
RS7965584	intergenic	AA	normal magnesium nutritional requirement

Test details

Applicable situation

The magnesium nutritional requirement item uses four polymorphic loci on and between genes such as *SHROOM3* and *MUC1*. The test results showed the magnesium nutritional requirement represented by the genotype combination of the user at four polymorphic loci tested. The test cannot represent the user's real magnesium nutritional requirement.

Notification

1. Limited by the existing sample size, there may be some errors in this test. With the expansion of sample size, the test results will be continuously optimized.
2. Limited by the current technological means and the level of scientific cognition, this test may not cover all genes or loci related to magnesium nutritional requirement.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. An individual's nutritional metabolism is the result of the interaction of genes, environment, and living habits.
3. The test result only predicts your magnesium nutritional requirement at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status. If you have nutritional supplement-related needs, you can consult authoritative nutritionists, clinicians or professionals based on the results of this genetic test to obtain professional nutritional advice.

Test content

Four polymorphic loci related to magnesium nutritional requirement were detected, including rs13146355 and rs3925584, etc.

Study population

The basis of the test is mainly from the research of Caucasian.

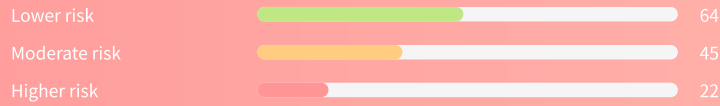
Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to magnesium nutritional requirement.



Health Risk

131 Reports



Item

Genetic risk

Late-Onset Alzheimer's Disease

1.14 times



Atrial Fibrillation

0.29 times



Age-related Macular Degeneration (AMD)

0.01 times



Colorectal Cancer

1.13 times



Restless Leg Syndrome (RLS)

1.07 times



Parkinson's Disease (PD)

0.32 times



Esophageal Cancer

0.87 times



Stomach Cancer

0.68 times



Primary Biliary Cholangitis (PBC)

0.37 times



Scleroderma

0.46 times



Alcohol Dependence

2.05 times



Asthma

0.16 times



Brain Aneurysm

0.51 times



Vitiligo

0.31 times



Allergic Rhinitis

1.42 times



Keloid

1.74 times



Kidney Diseases

0.36 times



Amyotrophic Lateral Sclerosis (ALS)

0.74 times



Meningioma

1.11 times



Nasopharyngeal Carcinoma

0.01 times



Paget's Disease of Bone

1.32 times



Sjögren's Syndrome

1.46 times



Type 2 Diabetes (T2D)

0.11 times



Exfoliative Glaucoma

0.96 times



Melanoma

0.74 times



Multiple Sclerosis (MS)

80.1 times



Type 1 Diabetes (T1D)

0.01 times



Behçet's Syndrome

0.09 times



Chronic Obstructive Pulmonary Disease (COPD)

0.62 times



Follicular Lymphoma

0.3 times



Renal Cancer

0.67 times



Renal Calculi

0.64 times



Neuroblastoma

0.48 times



Nicotine Dependence

1.27 times



Pancreatic Cancer

0.72 times



Peripheral Artery Disease (PAD)

0.81 times



Idiopathic Pulmonary Fibrosis (IPF)

0.68 times



Thyroid Cancer

0.23 times



Tardive Dyskinesia

0.42 times



Obsessive-Compulsive Disorder (OCD)

0.95 times



Obesity

0.48 times



Coronary Heart Disease (CHD)

0.09 times



Gout

0.1 times



Venous Thromboembolism (VTE)

1.27 times



Psoriasis

0.01 times



Lung Cancer

1.5 times



Gallstones

2.41 times



Rheumatoid Arthritis (RA)

0.01 times



Ulcerative Colitis

1.08 times



Crohn's Disease (CD)

1.1 times



Bipolar Disorder

1.33 times



Breast Cancer

2.35 times



Systemic Lupus Erythematosus (SLE)

21.23 times



Alopecia Areata

0.11 times



Ankylosing Spondylitis (AS)

0.56 times



Atopic Dermatitis

0.38 times



Chronic Lymphocytic Leukemia (CLL)

1.03 times



Dupuytren's Disease

1.35 times



Hypertension

0.79 times



Hypothyroidism

1.15 times



Migraines

1.01 times



Progressive Supranuclear Palsy (PSP)

1.37 times



Abdominal Aortic Aneurysm (AAA)

0.62 times



Narcolepsy

0.39 times



Osteoarthritis (OA)

2.82 times



Sarcoidosis

2.03 times



Scoliosis

0.46 times



Selective IgA Deficiency

0.71 times



Stroke

0.26 times



Endometriosis

0.67 times



Gestation Diabetes Mellitus (GDM)

1.03 times



Ovarian Cancer

0.97 times



Polycystic Ovarian Syndrome (PCOS)

0.39 times



Uterine Fibroids

0.9 times



Attention Deficit Hyperactivity Disorder (ADHD)

0.92 times



Back Pain

1.39 times



Cleft Lip and Cleft Palate

0.49 times



Developmental Dyslexia

0.92 times



Essential Tremor

0.89 times



Hashimoto's Thyroiditis

0.66 times



Hypertriglyceridemia

0.25 times



Myeloproliferative Neoplasm (MPN)

0.6 times



Non-Alcoholic Fatty Liver Disease (NAFLD)

0.34 times



Neural Tube Defects (NTDs)

0.49 times



Placental Abruption

0.99 times



Preeclampsia

0.87 times



Intrahepatic Cholestasis of Pregnancy (ICP)

0.42 times



Sudden Cardiac Death (SCD)

0.53 times



Depression

0.1 times



Schizophrenia

0.07 times



Celiac Disease

0.94 times



Otosclerosis

0.8 times



Allergic Diseases

1.09 times



Hyperemesis Gravidarum

0.79 times



Open-Angle Glaucoma

0.32 times



Gingivitis

0.97 times



Mouth Ulcers

1.93 times



Periodontitis

1.03 times



Dental Retardation

0.98 times



Mandibular Protrusion

1.7 times



Dental Caries

1.02 times



Endometrial Cancer

1.04 times



Osteoporosis

1.12 times



Diverticulosis

0.93 times



Autism

1.01 times



Cervical Cancer

3.38 times



Liver Cancer

1.04 times



Alcoholic Fatty Liver Disease (AFLD)

1.23 times



Oral Cancer

0.47 times



Laryngocarcinoma

0.86 times



Lumbar Disc Herniation

0.69 times



Anorexia Nervosa

0.88 times



Aortic Dissection

0.37 times



Kawasaki Disease

0.85 times



Epilepsy

0.77 times



Primary Adrenal Hypofunction

0.56 times



Primary Sclerosing Cholangitis (PSC)

0.37 times



Autoimmune Hepatitis

0.2 times



Vascular Dementia

0.3 times



Obstructive Sleep Apnea

0.15 times



Myopia

1.31 times



Astigmatism

0.97 times



Cataract

1.12 times



Post-Traumatic Stress Disorder (PTSD)

0.69 times



Autoimmune Addison's Disease

0.82 times



Haemorrhoids

1.01 times



Bladder Cancer

0.94 times



Gastroesophageal reflux disease

1.01 times



Vertigo

1.01 times



Spontaneous Urticaria

0.52 times



Insomnia

0.98 times



Multiple Sclerosis (MS)

Multiple sclerosis is an autoimmune disease of the central nervous system primarily characterized by inflammatory demyelinating lesions of the white matter.

My Genetic Risk Is Average For The Population

80.10 times

Average incidence rate is 4 per 100,000 in the population

Suggestions

1. If you have viral infections such as measles virus, Epstein-Barr virus, human herpes virus type 6, etc., you should be treated promptly to prevent the condition from getting worse.
 2. Establish good living habits, avoid overwork, and maintain a positive and optimistic mood.
-

Influencing factors

Controllable factors

1. Viral infection: Studies have shown that viral infections such as measles virus, Epstein-Barr virus, and human herpes virus type 6 can induce this disease.
2. Studies have shown that overwork can induce this disease.

Uncontrollable factors

1. Genetic factors: there is a family genetic tendency.
 2. Ethnic factors: The incidence rate in the west is higher than that in the East, and the incidence rate of Caucasian is significantly higher than that of Chinese.
 3. Latitude factors: Studies have shown that the prevalence of this disease increases with geographic latitude. The prevalence is smaller in tropical areas, while patients in temperate and frigid zones increase significantly.
 4. Discontinuation of hormone therapy can also induce this disease.
-

Knowledge



Time of onset

The peak age of onset is 10 to 60 years old, and the incidence rate is very low after 60 years of age.



Prevalence

According to statistics, there are about 4 cases of the disease for every 100,000 people in the average population.



Typical symptoms

1. **Limb weakness:** Limb weakness is the most common, and about 50% of patients' first symptoms include one or more limb weakness.
2. **Paresthesias:** Superficial sensory disturbances are manifested as numbness of limbs, trunk or face with acupuncture, abnormal limb chills, ant walking sensation, itching sensation, and sharp, burning pain and unclearly located paresthesias. There may also be deep sensory disturbances.
3. **Ocular symptoms:** often manifested as acute optic neuritis or retrobulbar optic neuritis, mostly acute onset of monocular vision loss, and sometimes both eyes are affected at the same time. Early fundus examination showed edema or normal optic papilla, and later optic nerve atrophy. About 30% of cases have ophthalmoplegia and diplopia. Nystagmus is mostly horizontal or horizontal plus rotation.
4. **Ataxia:** 30% to 40% of patients have varying degrees of ataxia dyskinesia.
5. **Paroxysmal symptoms:** Refers to sensory or motor abnormalities that have a short duration and can be induced by special factors. Paroxysmal neurological dysfunction lasts for several seconds to several minutes at a time, and can be induced by frequent, hyperventilation, anxiety or maintaining a certain posture of the limbs. It is one of the characteristic symptoms of multiple sclerosis. Tonic cramps, paresthesias, dysarthria, ataxia, epilepsy, and pain and discomfort are the more common symptoms of multiple sclerosis. Among them, tonic spasm confined to the limbs or face, often accompanied by radioactive abnormal pain, also known as cramps, generally unconscious loss and abnormal electroencephalogram during the attack. Passive neck flexion induces a tingling or lightning-like sensation, which spreads from the neck to the thighs or feet along the spine, which is called the Lhermitte sign.
6. **Psychiatric symptoms:** more common in patients with multiple sclerosis, most of which are depression and irritability. Some patients appear euphoria, excitement, or apathy, lethargy, strong crying and strong laughter, unresponsiveness, and intelligence. Inferiority, repetitive language, suspicion and persecution delusions, etc. There may be memory loss and cognitive impairment.

7. Other symptoms: Bladder dysfunction is one of the main pains of patients with multiple sclerosis. In addition, male patients with multiple sclerosis can also have primary or secondary sexual dysfunction.

 Treatment

The treatment goals of multiple sclerosis are to reduce the severity of the disease, reduce the recurrence rate, and reduce or delay the occurrence of functional disability. Corticosteroids, intravenous immunoglobulin, and plasma exchange can be used for acute treatment of multiple sclerosis; β -interferon, glatiramer acetate, natalizumab, etc. can be used for remission treatment; methotrexate, etc. can be used for secondary progression Treatment; Azathioprine can be used for the treatment of relapsed type.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS3129934	C6orf10	TT	11.04	0.91
RS3135391	HLA-DRA	AG	3.04	0.98
RS1327328	intergenic	AG	2.83	0.96
RS3135388	HLA-DRA	AG	1.99	0.99
RS117822168	intergenic	GG	1.89	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to multiple sclerosis. These literatures have found some polymorphic loci related to the risk of the disease by studying multiple sclerosis patients and normal people. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of multiple sclerosis item uses the 205 polymorphic loci in and between *KIF1B*, *CLEC16A*, *C6orf10*, *IL7R*, *BRINP1*. The test results indicate whether the user has a higher prevalence of multiple sclerosis in the combination of genotypes at the 205 polymorphic loci tested compared to all users. This test does not represent the user's true risk of multiple sclerosis. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of multiple sclerosis.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of multiple sclerosis at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of multiple sclerosis, or you think that you are likely to suffer from multiple sclerosis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 205 polymorphic loci associated with multiple sclerosis risk, including rs10492972, rs725613, rs3129934, rs68979932 and rs10984447.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to multiple sclerosis.

Systemic Lupus Erythematosus (SLE)

Systemic lupus erythematosus is an autoimmune-mediated diffuse connective tissue disease with immunoinflammation as its main manifestation.

My Genetic Risk Is Average For The Population

21.23 times

Average incidence rate is 144 per 100,000 in the population

Suggestions

1. Prevent UV exposure and take sun protection measures; prevent viral infections and vaccinate as soon as possible.
2. Maintain good living habits, quit smoking, limit alcohol, and eat lightly.
3. Maintain a relaxed and happy mood and avoid excessive mental stress or excessive psychological pressure.

Influencing factors

Controllable factors

1. Both ultraviolet radiation and viral infection can induce systemic lupus erythematosus.
2. Food and tobacco: Certain allergenic foods such as mushrooms, figs, smoked foods, etc. can also induce disease. In addition, smoking can induce disease.
3. Psychological stress: Excessive psychological stress can cause disorders of the immune system in the body, which may induce disease.

Uncontrollable factors

1. Genetic factors: there is a family genetic tendency.
2. Drug factors: drugs such as hydralazine and isoniazid may induce the disease.
3. Gender: related to the level of estrogen. It is more common in women than men.

4. Age: It is more common in the age group of 15 to 45 years old.

Knowledge



Time of onset

It is more common in women of childbearing age, and is more common in the age group of 15 to 45 years old.



Prevalence

According to statistics, the average population of 100,000 people have about 144 cases of the disease.



Typical symptoms

At the beginning, it can manifest as fever, fatigue, weight loss, hair loss, skin rash, white or purple hands and feet after being cold, repeated oral ulcers, superficial lymphadenopathy, continuous bleeding during menstruation, purpura of the skin, decreased platelet count, occult Nephritis, anorexia, nausea, vomiting and so on. As the disease progresses, patients gradually appear damage to multiple systems, most of which are manifested as a process of remission and aggravation alternately.



Treatment

At present, there is no cure, which emphasizes early diagnosis and early treatment to avoid or delay the pathological damage of tissues and organs. In terms of drugs, non-steroidal anti-inflammatory drugs, antimalarials, glucocorticoids, immunosuppressants such as cyclophosphamide, etc. can all be used for the treatment of this disease.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS9270970	intergenic	CC	3.35	0.94
RS3093061	CRP	TT	2.75	0.88
RS11889341	STAT4	TT	2.53	0.95
RS3095870	intergenic	CT	1.74	0.98

RS2736345

intergenic

GG

1.59

0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to systemic lupus erythematosus. By studying the genotype differences between patients with systemic lupus erythematosus and normal people, these literatures have found some polymorphic sites associated with the risk of systemic lupus erythematosus. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of systemic lupus erythematosus item uses 70 polymorphic loci on the *STAT4*, *TNPO3*, *ITGAM* and etc. genes. The results show whether the user had a higher prevalence of systemic lupus erythematosus than all users in the genotype combination of the 70 polymorphic loci tested, which does not represent the user's real risk of systemic lupus erythematosus. The test is based mainly on Caucasian, Asian and African studies, and other populations are for informational purposes only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of systemic lupus erythematosus.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

-
1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing systemic lupus erythematosus at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
 3. If you have a family history of systemic lupus erythematosus, or if you feel you have a risk of developing systemic lupus erythematosus, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 70 polymorphic loci associated with the risk of systemic lupus erythematosus, such as rs10488631, rs1143679, rs2004640 and rs2280714.

Population samples

The basis of the test is mainly from the research of Caucasian, Asian and African.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with systemic lupus erythematosus.

Cervical Cancer

Cervical carcinoma is cancer that occurs in the cervix, and is the most common gynecological malignant tumor.

My Genetic Risk Is Average For The Population

3.38 times

Average incidence rate is 12 per 100,000 in the population

Suggestions

1. Timely vaccinate against human papillomavirus (HPV).
2. Have safe sex and avoid multiple sexual partners.
3. No smoking.

Influencing factors

Controllable factors

1. HPV infection: more than 90% of cervical carcinoma patients have been infected with HPV. HPV infection is the most important factor inducing cervical carcinoma.
2. Sexual behavior related: unsafe sexual behavior, multiple sexual partners, first sexual life less than 16 years old and oral contraceptives can increase the risk of cervical carcinoma.
3. Smoking: smoking is more likely to induce cervical carcinoma.

Uncontrollable factors

1. Age: the older the age, the greater the risk of cervical carcinoma.
 2. Heredity: genetic factors affect the susceptibility to HPV and the response in the immune system.
 3. Immunity: low immunity is more likely to induce cervical carcinoma.
-

Knowledge

Time of onset

Carcinoma in situ is common in women aged 30 ~ 35, and invasive carcinoma is common in women aged 45 ~ 55.

Prevalence

Cervical carcinoma is the most common gynecological malignancy, with an incidence of about 12 per 100,000.

Typical symptoms

Early-stage cervical cancer may show no obvious symptoms and signs, and may show vaginal bleeding, contact bleeding or even mass. Vaginal discharge is common in advanced cervical carcinoma, or different secondary symptoms appear with the range of cancer focus. Such as frequent urination, urgent urination, constipation, swelling and pain of lower limbs, etc.

Treatment

According to the specific situation of the patient, surgery, radiotherapy, chemotherapy or combination of multiple means are generally available.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS6558578	intergenic	GG	5.34	0.92
RS4782151	intergenic	TT	3.65	0.90
RS7780883	intergenic	AG	3.28	0.84
RS9272143	intergenic	TT	2.22	0.97
RS8088832	intergenic	CT	1.94	0.90

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to cervical carcinoma. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between subjects with cervical carcinoma and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The cervical carcinoma item uses 13 polymorphic loci in and between *EXOC1*, *HLA-DPB2* and *ZNF804B* genes. The test results indicate whether the user has a higher risk of cervical carcinoma for the genotype combination of 13 polymorphic loci detected compared to all users. The test cannot represent the user's real risk of cervical carcinoma. The basis of the test is mainly from the research of Han Chinese, Japanese and Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that contribute to the risk of cervical carcinoma.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of diseases are jointly determined by multiple factors such as genes and environment. This study only predicts your risk of cervical carcinoma at the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
 3. If there is a family history of cervical carcinoma in your family, or if you think you are at risk of developing cervical carcinoma, you are recommended to consult an authoritative health professional or clinician for help.
-

Test content

Thirteen polymorphic loci associated with the risk of cervical carcinoma were detected, including rs13117307, rs4282438 and rs8067378.

Population samples

The basis of the test is mainly from the research of Han Chinese, Japanese and Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect cervical carcinoma.

Osteoarthritis (OA)

Osteoarthritis, also known as degenerative joint disease, bone hyperplasia and osteoarthropathy, is a group of heterogenous diseases in which joint symptoms and signs are caused by damaged articular cartilage integrity and articular subchondral bone plate lesions.

My Genetic Risk Is Average For The Population

2.82 times

Average incidence rate is 15000 per 100,000 in the population

Suggestions

- Red flags: joint pain when going up and down the stairs, pain when pressing the knee
- High risk population: middle-aged and elderly people, people with a higher body weight or engaged in strenuous physical activities, patients with rheumatoid Some diseases
- Typical symptoms: joint pain, morning stiffness and stickiness, etc.
- How to reduce the risk:
 - Maintain a healthy weight: Maintain BMI between 18.5 and 23.9
 - Exercise moderately, exercise the muscles around the knee joint, which can increase the synovial fluid in the knee joint
 - Avoid trauma, avoid hard concrete floors, choose plastic sports fields, and reduce the impact of exercise on your knees
 - Choose soft-soled or air-cushioned sports shoes to reduce the impact of sports
 - Some studies have shown that the intake of Glucosamine helps repair damaged cartilage and relieve joint pain
- Treatment methods: early use of muscle training, methylsulfonylmethane relieve pain, physical therapy, etc. Conservative treatment, or Surgical treatment
- The Heritability of osteoarthritis is about 50%

* If you have a family history of osteoarthritis, or feel that you have the possibility of suffering from osteoarthritis, or need to use dietary supplements or take treatment, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors

Controllable factors

1. Obesity and stout body type: High body weight will increase joint load, poor posture and gait change the biomechanics of joints, which may lead to joint degenerative changes.
2. Mechanical factors: such as trauma, abnormal joint morphology, long-term occupations that repeatedly use certain joints or strenuous cultural and sports activities, excessive exercise, etc., will increase the risk of osteoarthritis.

Uncontrollable factors

1. Age: Osteoarthritis mostly occurs in middle-aged and elderly people (over 40 years old). The older you are, the more likely you are to suffer from osteoarthritis.
 2. Family history: People with a family history of osteoarthritis are at higher risk of disease.
 3. Disease factors: Immune deficiency diseases, inflammation (rheumatoid arthritis, gout, etc.), bone diseases (Paget's disease) and other diseases will increase the risk of secondary osteoarthritis.
 4. Joint problems: Joint deformities (scoliosis and kyphosis) and joint injuries can cause osteoarthritis.
-

Knowledge

Time of onset

The disease can occur at any age, and it is more common in middle-aged and elderly people (over 40 years old).

Prevalence

A research on prevalence trends of site-specific osteoarthritis from 1990 to 2019 shows that the prevalent cases of osteoarthritis increased globally by 113.25% in nearly 3 decades, from 247.51 million in 1990 to 527.81 million in 2019. The prevalence and risk of osteoarthritis in women are significantly higher than that in men.

Typical symptoms

- Pain: The main symptom of this disease and the main cause of dysfunction. It is characterized by insidious attacks and persistent dull pain. It usually occurs after activities and can be relieved by rest. As the disease progresses, joint movement may be restricted due to pain, and pain may even occur during rest. During sleep, the muscles around the joints are damaged, and the joint protection function is reduced, and the pain-causing activities cannot be restricted as when waking, and the patient may wake up with pain. Because cartilage lacks pain-sensitive nerve fibers, the pain is mostly caused by intra-articular high pressure stimulating painful nerve fibers in the joint capsule, or intraosseous high pressure stimulating the periosteum or peripheral bone nerve fibers, or subchondral microfractures, or osteophytes, peri-articular muscles, and slippage Prostaglandins and other cytokines in the fluid stimulate the synovial sensory nerve endings.

- Morning stiffness and stickiness: Morning stiffness suggests the presence of synovitis, but unlike rheumatoid arthritis, the time is relatively short, generally no more than 30 minutes. Adhesive sensation refers to the stiffness of the joints when they start to move after a period of rest. If they are stuck, they can be relieved by a little movement. The above situation is more common in the joints of the lower limbs of the elderly.
- Other symptoms: As the disease progresses, joint contractures, instability, rest pain, and pain aggravate during weight bearing. Dysfunction may occur due to poor joint surface anastomosis, muscle spasm and contraction, joint capsule contraction, and mechanical atresia caused by bone spurs or joint mice.

Treatment

1. Conservative treatment: Conservative treatment should be used in the early stage, which can effectively reduce symptoms and delay the disease. Conservative treatment methods include protecting articular cartilage, strengthening muscles around the joints, pain relief, and physical therapy.
2. Surgical treatment: If none of the above treatments can alleviate the impact of pain on daily life, consider knee arthroplasty or unicondylar resurfacing, using artificial cartilage to replace the existing damaged cartilage, so that the patient can resume normal activities.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS551471509	intergenic	TT	0.18	1.00
RS4140564	intergenic	AA	2.53	1.00
RS9864422	COL6A4P1	TT	2.10	0.98
RS7639618	COL6A4P1	TT	1.96	0.98
RS16944	IL1B	AG	1.50	0.98

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to osteoarthritis. These literatures found some polymorphic sites associated with the risk of the disease

by studying the genetic differences between patients with osteoarthritis and normal people. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk item of osteoarthritis disease uses the 120 polymorphic loci on genes such as *BTNL2*, *MCF2L*, *GDF5*, *IL1B*, *COG5* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 120 polymorphic loci detected by this test increases the risk of osteoarthritis disease. It does not mean the user's true risk of osteoarthritis disease. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of osteoarthritis.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of osteoarthritis at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
 3. If you have a family history of osteoarthritis, or you think that you are likely to suffer from osteoarthritis, it is recommended that you consult an authoritative health professional or clinician for help.
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Test content

The test includes 120 polymorphic loci related to the risk of osteoarthritis, including rs10947262, rs11718863 and rs7639618.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to osteoarthritis.

Gallstones

Gallstones, including those occurring in the gallbladder and bile duct, are common and frequently-occurring diseases.

My Genetic Risk Is Average For The Population

2.41 times

Average incidence rate is 10000 per 100,000 in the population

Suggestions

1. Diet regulation is the best method to prevent gallstones. Diversified diet is recommended. Cold, greasy, high-protein, irritating foods and spirits can easily cause cholestasis, so you should eat less of those. You can eat more vegetables and fruits, fish and seafood that are rich in vitamin A and vitamin C.
2. Adjust the gastrointestinal flora and actively treat bacterial infections.
3. Maintain good living habits, eat regularly, eat breakfast on time, avoid gaining weight, and do not lose weight blindly.
4. Develop the habit of regular exercise and maintain a healthy body.
5. Some drugs can promote the occurrence of stones, such as ceftriaxone, dipyridamole, non-steroidal anti-inflammatory drugs (NSAIDs), oral contraceptives, etc. So the medication time needs to be controlled.
6. Reduce the number of pregnancies can reduce the risk of gallstones.

Influencing factors



Controllable factors

1. Dietary factors: High-calorie, high animal fat, high-protein diet are risk factors for gallstones. Moreover, gallstones appear to be a common side effect in very low-calorie diet, especially during weight loss.

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2. Infection factors: Bacterial infection can cause cholecystitis, its colonies, shedding epithelial cells, etc., can promote the formation of gallstones.
 3. Bad habits: Studies have found that long-term skip of breakfast will increase the concentration of bile, conducive to bacterial reproduction, easy to promote the formation of gallstones. In addition, prolonged fasting or rapid weight loss can also lead to stones.
 4. People who do not exercise will have a decreased contraction of the gallbladder muscle, which can easily cause bile siltation to induce stones.
 5. Drug factors: Some drugs can promote the occurrence of stones, such as ceftriaxone, dipyridamole, non-steroidal anti-inflammatory drugs (NSAIDs), oral contraceptives, etc.
 6. Pregnancy: Pregnancy is a high-risk factor for gallstones, and the number of pregnancies is positively correlated with the incidence of gallstones.

Uncontrollable factors

1. Inheritance: There is a family genetic tendency.
2. Race: The incidence among different races varies. For example, the incidence of gallstones in indigenous Indians is higher.

Knowledge

Time of onset

This disease can occur at any age. Generally, the incidence rate increases with age.

Prevalence

According to statistics, there are about 10,000 cases of the disease per 100,000 people in the average population. There are more female patients than males.

Typical symptoms

According to the location of occurrence in the biliary tract, the symptoms of gallstones may include:

- The main clinical manifestations of gallbladder stones: Biliary colic or epigastric pain, nausea and vomiting, indigestion, chills, fever, jaundice, etc.
- The main clinical manifestations of extrahepatic bile duct stones: Biliary colic, chills, high fever and jaundice.
- The main clinical manifestations of intrahepatic bile duct stones: Pain in the upper abdomen, jaundice, tenderness in the upper abdomen, etc.

Treatment

The treatment methods for gallstones include non-surgical methods such as extracorporeal shock wave lithotripsy and oral litholytic therapy, as well as surgical methods such as laparoscopic

cholecystectomy. The selection of treatment methods needs to consider the patient's objective conditions (such as symptoms, gallstone characteristics, gallbladder function) and subjective willingness.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS9514089	SLC10A2	CC	1.78	0.95
RS708686	intergenic	TT	1.59	0.99
RS1025447	DYNC2LI1	TT	1.39	1.00
RS10426201	intergenic	AA	1.37	1.00
RS686030	TTC39B	AA	1.30	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to gallstones. By studying the genetic differences between gallstones patients and normal people, these literatures have identified some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

 Applicable situation

The gallstones health risk item uses 56 polymorphic loci on genes such as the *SLC10A2*, *UGT1A1*, *ABCG8* and other intergenic regions. The results showed whether the user had a higher prevalence of gallstones than all users in the genotype combination of the 56 polymorphic loci tested, and that the test did not represent the user's real risk of gallstones. The basis of the test is mainly from the research of Caucasian, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of gallstones.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of gallstones at a genetic level and does not represent your true health status, nor does it involve your judgment of other health conditions, lifestyles, etc.
3. If you have a family history of gallstones, or if you feel you have a risk of gallstones, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 56 polymorphic loci related to the risk of gallstones, including rs6742078, rs9514089 and rs11887534.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with gallstones.

Breast Cancer

Breast cancer refers to the canceration of the gland or ductal epithelial cells of the breast.

My Genetic Risk Is Average For The Population

2.35 times

Average incidence rate is 39 per 100,000 in the population

Suggestions

1. Maintain good living habits: balanced nutrition, eat less high-fat, high-sugar and high-calorie foods, and eat more high-fiber foods. Keep exercising, at least 4 hours a week; avoid staying up late.
2. Maintain a healthy weight: Keep BMI between 18.5 and 23.9.
3. Reject active and passive smoking, and avoid exposure to tobacco; limit alcohol intake. The limit value for moderate drinking recommended by the Chinese Nutrition Society is that the amount of alcohol consumed by adult men does not exceed 25g/day; the amount of alcohol consumed by adult women does not exceed 15g/day.
4. The recommended childbearing age for women is 25-35 years old, and to avoid artificial abortion, it is best to insist on breastfeeding.
5. Avoid unnecessary X-ray and CT inspections. If you need to receive combined hormone therapy or oral contraceptives, please evaluate the risks and benefits with your doctor in advance before using it.

Influencing factors



Controllable factors

1. Living habits: Those who prefer high-fat, high-sugar and high-calorie foods have a higher incidence of breast cancer. Staying up late and lack of exercise can also increase the risk of breast cancer.

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2. Obesity: The risk of breast cancer for obese people is twice that of normal people.
 3. Tobacco and alcohol intake: Drinking alcohol can increase the risk of breast cancer. Smoking can cause the incidence to be 2/3 higher, and women who start smoking during adolescence have a 70% increase in the risk of the disease.
 4. Female childbearing experience: Women who have never given birth, gave birth until 35 years old, or do not breastfeed after giving birth have a higher incidence of breast cancer.
 5. Treatment experience: People who have been exposed to hormone-containing drugs or received radiation therapy on the chest are at higher risk of breast cancer.

Uncontrollable factors

1. Age: Breast cancer mostly occurs in women between 40 and 60 years old. The older you are, the more likely you are to develop breast cancer.
2. Endocrine problems: Women whose age of menarche (first menstruation) is less than 12 years old or menopause age over 55 years old have a higher risk of breast cancer.
3. Family history: People with a family history of breast cancer have a higher prevalence.

Knowledge

Time of onset

Breast cancer mostly occurs in women between 40 and 60 years old (before and after menopause), and the incidence increases with age, but the incidence of postmenopausal women is significantly lower than that of premenopausal women.

Prevalence

Prevalence rate: 100/100,000. The distribution of breast cancer incidence is different in different countries and regions. Most Asian and African countries are low-incidence areas, southern Europe and South America are middle-incidence areas, and North America and North Europe are high-incidence areas. China is an area with a relatively low incidence of breast cancer, and there are also regional differences. The general characteristics are: coastal cities are higher than inland areas, and cities with developed economies and high population densities are higher than those with backward economies and low population densities.

Typical symptoms

Early breast cancer often does not have typical symptoms and signs, and is not easy to attract attention. It is often found through physical examination or breast cancer screening. The following are typical signs of breast cancer. 1. Breast lumps: 80% of breast cancer patients are first diagnosed with breast lumps. Patients often find breast lumps inadvertently, which are mostly single, hard, with

irregular edges and a less smooth surface. Most breast cancers are painless masses, and only a few have varying degrees of dull pain or tingling. 2. Nipple discharge: Nipple discharge is called nipple discharge when blood, serous, milk, and pus are discharged from the nipple during non-pregnancy, or milk is discharged after stopping breast-feeding for more than half a year. There are many reasons for nipple discharge. Common diseases include intraductal papilloma, breast hyperplasia, mammary duct dilatation and breast cancer. Unilateral single-port bloody discharge should be further examined, if accompanied by breast masses, more attention should be paid. 3. Skin changes: Breast cancer can cause skin changes with various signs. The most common is that the tumor invades the breast skin and the deep pectoralis fascia ligament, which shortens and loses its elasticity, pulls the skin of the corresponding part, and causes "dimples". Sign, that is, a small depression in the breast skin, like a small dimple. If cancer cells block the lymphatic vessels, there will be "orange peel-like changes", that is, many small dot-like depressions appear on the breast skin, just like orange peels. In advanced breast cancer, cancer cells infiltrate into the skin and grow along the lymphatic vessels, glands, or fibrous tissues, forming scattered hard nodules in the skin around the main cancer focus, the so-called "skin satellite nodules." 4. Abnormal nipple and areola: The tumor is located in or close to the deep part of the nipple, which can cause the nipple to retract. When the tumor is far from the nipple and the large duct in the breast is invaded and shortened, it can also cause the nipple to retract or elevate. Eczema-like carcinoma of the nipple is manifested by itching, erosion, ulceration, crusting, scaling, and burning pain of the nipple skin, resulting in nipple retraction. 5. Axillary lymph node swelling: More than one third of breast cancer patients admitted to a large hospital have axillary lymph node metastasis. In the early stage, there may be swelling of the ipsilateral axillary lymph nodes, and the swollen lymph nodes are hard, scattered, and pushable. As the disease progresses, the lymph nodes gradually fuse, and adhere to and fixate with the skin and surrounding tissues. In the late stage, the metastatic lymph nodes can be felt on the clavicle and the contralateral armpit.



Treatment

1. Tumorectomy: Surgical removal of the tumor and its adjacent tissues, leaving most of the breast. For limited breast cancer, this method is the best choice.
2. Complete mastectomy: If more than one tumor is found, or the tumor is quite large, consider removing the entire breast (usually including the lymph nodes on the same side). Most women whose breasts have been removed can consider breast reconstruction. The patient should discuss with the doctor whether it is suitable for reconstruction and which method to use to reconstruct the breast.
3. Radiotherapy: Use precise radiation to illuminate the tumor to kill cancer cells and shrink the tumor.
4. Drug therapy: use one or more drugs to inhibit cancer cells.
5. Radiation therapy and drug therapy are generally used as adjuvant treatments after surgery. In the early stage of breast cancer, doctors will assess the patient's condition and may give anti-estrogen therapy. According to statistics, about one-third of breast cancer patients are effective after receiving anti-estrogen therapy.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1801157	CXCL12	CT	1.64	0.97
RS2981578	FGFR2	CT	0.58	0.97
RS1501299	ADIPOQ-AS1	GT	1.59	0.99
RS3218005	CDKN2B-AS1	TT	1.55	0.99
RS2268578	LUM	AG	1.40	0.97

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to breast cancer. By studying the genotype differences between patients with breast cancer and normal people, these literatures have found some polymorphic sites associated with the risk of breast cancer. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of breast cancer item uses 80 genetic and intergenic polymorphic loci of genes such as *PALB2*, *BRCA1*, *BRCA2*, *FGFR2*, and *ATM*. The results show whether the user had a higher prevalence of breast cancer than all users in the genotype combination of the 80 polymorphic loci tested, which

does not represent the user's real risk of breast cancer. The test is based mainly on Caucasian and Asian studies, and other populations are for informational purposes only.

 Notification

1. The test may not cover all genes or loci that could contribute to the risk of breast cancer.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

 Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing breast cancer at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of breast cancer, or if you feel you have a risk of developing breast cancer, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 80 polymorphic loci associated with the risk of breast cancer, including rs10801935, rs7895676 and rs29815782.

 Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with breast cancer.

Alcohol Dependence

Alcohol dependence exists if excessive drinking has already caused physical or mental damage and brought about adverse social consequences. If the duration and amount of drinking reaches a certain extent, drinkers become unable to control their drinking behavior, and somatization and withdrawal symptoms occur.

My Genetic Risk Is Average For The Population

2.05 times

Average incidence rate is 9000 per 100,000 in the population

Suggestions

1. Limit alcohol intake: the limit recommended by the Chinese academy of nutrition for adults to drink alcohol in moderation is no more than 25g for adult men and no more than 15g for adult women. If your risk is high, you should consume a lower amount of alcohol than the above standard as much as possible to prevent the formation of alcohol dependence.
2. Maintain a good mental state and cope with stress in a positive and healthy way.

Influencing factors



Controllable factors

1. Alcohol intake: For drinkers, if the alcohol intake reaches a certain time or amount, it will increase the risk of alcohol dependence.
2. Psychological factors: psychological dependence on alcohol and using alcohol as a way to relieve stress will also increase the risk of alcohol dependence.



Uncontrollable factors

Family history: People with a family history of alcohol dependence have a higher prevalence.

Knowledge

Time of onset

In the case of long-term excessive drinking, some people will develop symptoms of alcohol dependence. Under this premise, people of any age may be affected.

Prevalence

The Lancet magazine published the ranking of the total global disease burden in 2010, showing that in the 20 years from 1990 to 2010, among all disease risk factors, alcohol consumption has rapidly risen from sixth to third, second only to hypertension and smoking. According to the latest statistics, the lifetime prevalence rate of adult alcohol use disorder worldwide has reached 16%, and the prevalence rate is even higher in Eastern European countries.

Typical symptoms

Physical dependence on alcohol, with withdrawal syndrome typical of tremor appears after withdrawal. Acute psychiatric symptoms can occur after a heavy drinking. Mental abnormalities appear after long-term drinking, such as hallucinations, auditory hallucinations, tremors, delusions, and personality decline.

Main hazard

1. Physical damage: Excessive drinking can cause serious physical, psychological, and social damages, with obvious damage to the internal organs and nervous system. Common such as epilepsy, liver cirrhosis, gastrointestinal diseases and so on.
2. Psychological damage: Alcohol dependence can lead to personality changes, anxiety and depression, sexual dysfunction, Suicide, etc.
3. Social damage: traffic accidents, domestic violence, personal assaults, etc.

Treatment

Professional treatment needs to be treated in stages. In the early stage, alcohol-dependent people should be drug-free to eliminate the physical symptoms of alcohol-dependent people in the withdrawal state. Afterwards, treatment in the rehabilitation period is required, requiring joint intervention of psychotherapy and drug therapy, and strengthening psychological and social intervention and support.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS27072	SLC6A3	CC	5.11	0.91
RS1229984	ADH1B	CT	0.80	0.99
RS13382553	intergenic	AA	0.95	1.00
RS9937709	intergenic	AA	1.05	1.00
RS492602	FUT2	AA	0.96	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to alcohol dependence. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotypes difference between alcohol dependent patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The alcohol dependence health risk project uses 22 polymorphic loci in genes such as *SLC6A3* and *ADH1B*. The test result shows whether the user has a higher prevalence of alcohol dependence disease in the genotype results at the 2 polymorphic loci detected compared to all users. This test does not represent the user's real risk of alcohol dependence.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of alcohol dependence.
2. The test results have no clinical significance.

3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by multiple factors such as genes and environment. This study only predicts your risk of alcohol dependence at a genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc
3. If you have a family history of alcohol dependence, or you think that you are likely to suffer from alcohol dependence, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 22 polymorphic loci associated with scleroderma risk, including rs27072 and rs7590720.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that associated with alcohol dependence.

Sarcoidosis

Sarcoidosis is a multisystem granulomatous disease of unknown etiology. Clinically, the main manifestations are bilateral hilar lymph node enlargement, infiltration of the lungs, and skin and eye damage. Granulomatous nodules can also invade every organ in the body.

My Genetic Risk Is Average For The Population

2.03 times

Average incidence rate is 10 per 100,000 in the population

Suggestions

Before using hormone drugs, you should consult your doctor in detail to assess whether there is a risk of causing sarcoidosis based on your own situation.

Influencing factors



Controllable factors

Endocrine factors: Increased concentrations of prolactin and estrogen may increase the risk of sarcoidosis.



Uncontrollable factors

1. Age: Young adults are more likely to develop sarcoidosis.
2. Autoimmune diseases: Autoimmune diseases and sarcoidosis often coexist. Diseases in immune function may increase the risk of sarcoidosis, but the exact mechanism is still unclear.
3. Family history: People with a family history of sarcoidosis have a higher prevalence.
4. Infectious factors: Studies have found that some infectious pathogens (such as *Propionibacterium acnes*, *Mycobacterium*, etc.) may be closely related to the occurrence of sarcoidosis.

Knowledge

Time of onset

It is common in young adults, children and the elderly can also suffer.

Prevalence

Prevalence rate: 10/100,000. The incidence of sarcoidosis can be seen in all races and nationalities, and the incidence rate of women is slightly higher. More in cold regions and countries, less in tropical regions. Race and ethnicity are quite different from each other. The incidence of sarcoidosis in African Americans is 10 to 17 times higher than that of Whites. Sweden has the highest annual incidence rate of 64/100,000, and the actual prevalence of autopsy can reach 267/100,000 to 641/100,000.

Typical symptoms

The clinical manifestations of sarcoidosis are diverse and lack specificity. About 40% of cases have no clinical symptoms, and abnormalities are only found during physical examination. Most patients have a slow onset and mild symptoms. Clinically, more than 90% of patients have lung changes, which are characterized by hilar lymphadenopathy, lung infiltration, eye and skin damage. The liver, spleen, lymph nodes, salivary glands, heart, nervous system, muscles, bones and other organs can also be affected. Common symptoms are:

1. Non-specific systemic symptoms: fatigue, weakness, low fever, night sweats, loss of appetite, weight loss, muscle and joint pain, etc. Individual patients have a rapid onset, manifested as fever, erythema nodosa, and joint pain, that is, Lofgren syndrome, which is more common in young female patients during pregnancy or lactation.
2. Respiratory system symptoms: mainly dry cough and chest tightness. Individuals have chest pain, blood in sputum or even hemoptysis. Shortness of breath after activity is usually caused by fibrosis or granulomatous pulmonary parenchymal lesions. When extensive pulmonary fibrosis develops, shortness of breath worsens or even becomes cyanotic, and respiratory failure occurs.
3. Skin: About 11% of people with sarcoidosis have skin damage. Lupus nodosa is the most common, and frostbite-like lupus is also more common, manifested as purple patches on the nose, cheeks, and ears.
4. Eye: Ocular sarcoidosis accounts for about 21%. Accompanied by eye discomfort, pain and visual impairment. Chronic disease can progress to blindness. Mainly manifested as iridocyclitis, but can also be manifested as acute conjunctivitis and keratoconjunctivitis sicca, and later can be complicated by cataracts and secondary glaucoma, anterior uveitis, parotitis and facial nerve palsy, collectively known as Heerfordt's syndrome .
5. Nervous system: Sarcoidosis can affect almost any part of the nervous system, accounting for about 8%. There may be cranial nerve paralysis, neuromuscular disease, intracranial space-occupying lesions, granulomatous meningitis, etc. Facial nerve palsy is the most common. Most

facial nerve palsy is unilateral, accounting for about 65%. Involving the hypothalamus or posterior pituitary gland can cause diabetes insipidus.

6. Heart: Sarcoidosis can directly invade the myocardium, forming cardiac granulomas or fibrotic lesions, but usually does not show symptoms. If there are symptoms, it is mostly manifested as tachycardia, conduction block, and congestive heart failure. It can also manifest as arrhythmia (mostly ventricular), angina pectoris, ventricular aneurysm, repeated pericardial effusion or sudden death. Those with hypercalcemia can cause fatal ventricular arrhythmia. Sarcoidosis causes pulmonary heart disease, which is seen in severe pulmonary fibrosis.
7. Kidney: Sarcoidosis can cause calcium nephropathy and eventually lead to renal failure. Generally, hypercalcemia occurs in severe cases and cases with a wide range of lesions.
8. Peripheral lymph nodes: sarcoidosis invades peripheral lymph nodes accounting for about 75%, and can be in any part of the body, except for hilar and mediastinal lymph nodes, the most common place is the right scalene muscle.
9. Musculoskeletal system: there may be osteoporosis, cyst-like or deformed hands (foot) and finger (toe) nail dystrophy, arthritis, muscle granuloma with the same change and appearance. Sarcoidosis can also be accompanied by gout.

Treatment

30% to 70% of patients relieve themselves without treatment. The respiratory dysfunction caused by patients with pulmonary symptoms can be treated with symptomatic treatment for two to three months. If the inflammation does not subside on its own, further treatment can be carried out. Glucocorticoid therapy is the most commonly used method in treatment. For some mild cases, such as skin damage, anterior uveitis or cough, only topical corticosteroids are needed. Systemic sarcoidosis usually requires oral corticosteroids. Cardiac and nervous system diseases, hypercalcemia, and ocular diseases that are not effective for local treatment are indications for oral corticosteroids. Systemic corticosteroid therapy for lung and other extrapulmonary sarcoidosis is still controversial, but most scholars believe that systemic corticosteroid therapy should be considered when symptomatic and progressive development. For patients who require long-term corticosteroid therapy, the use of antimalarials and cytotoxic drugs should be considered, and lung transplantation can be considered for rare refractory cases.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2789679	intergenic	AA	2.02	0.97

RS1964995	intergenic	TT	1.00	0.94
RS2076530	BTNL2	TT	1.00	0.96
RS117633859	intergenic	AA	1.00	0.96
RS112463197	intergenic	CC	1.00	0.98

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to sarcoidosis. By studying the genotype differences between patients with sarcoidosis and normal people, these literatures have found some polymorphic sites associated with the risk of sarcoidosis. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

A health risk item for sarcoidosis uses 8 polymorphic loci of the *BTNL2*, *ANXA11* gene. The results show whether the user had a higher prevalence of sarcoidosis than all users in the genotype combination of the 8 polymorphic loci tested, which does not represent the user's real risk of sarcoidosis. The test is based mainly on Caucasian and East Asian, other populations are for informational purposes only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of sarcoidosis.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing sarcoidosis at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of sarcoidosis, or if you feel you have a risk of developing sarcoidosis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes rs2076530, rs1049550, rs2302006, and rs112463197, 8 polymorphic loci associated with the risk of sarcoidosis.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with sarcoidosis.

Mouth Ulcers

Mouth ulcers are ulcerative lesions of the soft tissue mucosa such as the inner lip of the mouth, tongue, cheek, and palate. They usually begin in childhood and recur frequently.

My Genetic Risk Is Average For The Population

1.93 times

Average incidence rate is 20000 per 100,000 in the population

Suggestions

- Typical symptoms: repeated round ulcers, with oral mucous membrane swelling, congestion, hyperemia and other symptoms. During eating, drinking, brushing teeth and even talking when touching ulcers, they will feel a clear **burning pain**.
- Susceptible people: children are more likely to have mouth ulcers.
- Prevention methods:
 - Supplementing with **vitamins B** can repair the mucous membranes of the mouth and reduce the risk of mouth ulcers.
 - Avoid contact with sharp objects in the mouth to avoid trauma.
 - Eat less chocolate, coffee, spicy food, etc. Foods that induce mouth ulcers.
 - Wash your mouth frequently and reduce the amount of time food stays in your mouth.
 - Avoid mental stress, excessive pressure, and maintain a happy mood.
- Treatment: use mouthwashes (e.g. Compound Chlorhexidine Gargle) or use **low semise acetic acid**, etc. Corticosteroids
- Long-term smoking to a certain extent, it will reduce the occurrence of mouth ulcers, but it is not conducive to the maintenance of oral health.
- A few Genetic diseases family history can also cause susceptibility to mouth ulcers.
- Recurrent oral ulcers Heritability about 75%.

* If you are suffering from mouth ulcers or need to use dietary supplements or treatment options, it is recommended that you can consult an authoritative health professional or clinician for assistance.

Influencing factors



Controllable factors

1. Physical damage: trauma can cause mouth ulcers.
2. Mental factors: mental stress and anxiety can cause mouth ulcers.
3. Food: chocolate, coffee, peanuts, eggs, corn, almonds, strawberries, cheese and tomatoes can induce mouth ulcers.



Uncontrollable factors

1. Family history: there is a strong family tendency.
2. Diseases: diseases such as Behcet syndrome, Systemic lupus erythematosus, Stevens-Johnson syndrome, Celiac Disease, Lichen planus, Pemphigus vulgaris and Bullous pemphigoid can all cause mouth ulcers.

Knowledge



Onset time

The disease can occur at any age, with higher prevalence among people under the age of 30.



Prevalence

It occurs in 20%-30% of adults and a higher proportion of children.



Typical symptoms

Generally manifested as recurrent round ulcers, with oral mucosa swelling, congestion, secretions and other characteristics, when eating, drinking, brushing teeth or even talking when touching the ulcer will feel obvious burning pain.



Treatment

The main purpose of treatment is pain relief (analgesic), and doctors often recommend the use of mouthwash (e.g., oral tartar gargle) or corticosteroids (e.g. prednisone tablets, dexamethasone rinsing solution, fluocinonide ointment or clobetasol propionate unguent).



The relationship between smoking and mouth ulcers

Long-term or heavy smoking can reduce the occurrence of oral ulcers to a certain extent, mainly through two ways: (1) smoking will produce oral mucous membrane local horny; (2) Nicotine in tobacco can induce T cell inactivation, inhibit the production of pro-inflammatory cytokines, activate the release of adrenocorticotrophic hormone and cortisol, and inhibit the inflammatory pathway. However, dry mouth, high temperature in the mouth, changes in oral acidity, or reduced resistance to viral, bacterial and fungal infections caused by smoking are not conducive to oral health

maintenance. Taken together, it is recommended to refuse active smoking or quit smoking as soon as possible.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS3806265	NLRP3	TT	14.98	0.77
RS1800795	intergenic	GG	14.59	0.99
RS5361	SELE	TT	10.90	1.00
RS10759931	intergenic	AA	3.89	0.92
RS1800796	intergenic	CG	2.06	0.94

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to Mouth Ulcers. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between the patients with Mouth Ulcers and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

 Applicable situation

The Mouth Ulcers risk item uses 74 polymorphic loci on *MTHFR*, *IL1B*, *NLRP3*, and etc. The test result indicates whether the user has a higher possibility of Mouth Ulcers in genotype combination of the 74 polymorphic loci detected compared with all users. The test does not represent the user's real risk of Mouth Ulcers. The test is mainly based on the research of Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of Mouth Ulcers.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Mouth Ulcers from the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If you have a family history of Mouth Ulcers, or you think that you are likely to suffer from Mouth Ulcers, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 74 polymorphic loci associated with Mouth Ulcers risk, such as rs1801133, rs1143634, rs1800896 and so on.

Population samples

The basis of the test is mainly from the research of Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with mouth ulcers.

Keloid

Keloid, also known as keloidal scar or claw-like tumor, occurs in scar tissue resulting from various levels of external skin trauma.

My Genetic Risk Is Average For The Population

1.74 times

Average incidence rate is 1 per 100,000 in the population

Suggestions

After skin injury, attention should be paid to thorough debridement and suture to prevent infection and reduce the chance of keloid formation.

Influencing factors



Controllable factors

Infection: Co-infection after skin injury can easily lead to abnormal growth of scar tissue.



Uncontrollable factors

1. Family history: The occurrence of keloids has a certain family genetic tendency.
2. Race: Keloids are easily affected by race. The incidence of colored people is significantly higher than that of whites, especially blacks, which is 5-15 times higher than that of whites.
3. Age: Keloids mainly occur in adolescents aged 10-30.

Knowledge



Time of onset

Keloids mainly occur in adolescents aged 10-30.

There are about 1 case of the disease for every 100,000 people in the average population.

Red or dark red, accompanied by varying degrees of itching and tingling, it will affect the appearance when it occurs on the exposed part, and the large scar and when it occurs near the joints, neck and waist will affect the function of movement.

In most cases, injecting glucocorticoid into the affected area is the preferred treatment, and cryoresection, surgical resection, and laser resection are also common treatment methods.

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS873549	intergenic	TT	4.20	0.90
RS1511412	intergenic	GG	0.28	1.00
RS8032158	NEDD4	CT	1.51	0.98
RS940187	BPESC1	CC	1.48	1.00

The risk value of this test is calculated based on published academic literatures related to keloid. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between keloid patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-

genetic factors are not taken into account.

Test details

Applicable situation

The keloid health risk item uses 4 polymorphic loci on *NEDD4*, *BPESC1* genes. The test results indicate that compared with all users, whether this user has a higher prevalence of keloid in the combination of genotypes of the 4 polymorphic loci tested. It does not mean the user's true risk of keloid. The basis of the test is mainly from the research of Chinese, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that cause the risk of keloid.
 2. The test result has no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of the disease is determined by multiple factors such as genes and environment. This study only predicts your risk of keloid from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of keloid, or you think that you are likely to suffer from keloid, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 4 polymorphic loci related to the risk of keloid, including rs8032158 and rs940187.

Population samples

The basis of the test is mainly from the research of Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to keloid.

Mandibular Protrusion

Mandibular protrusion is a kind of malocclusion of facial bone, which is one of the common developmental malformations of teeth and jaws.

My Genetic Risk Is Average For The Population

1.70 times

Average incidence rate is 480 per 100,000 in the population

Suggestions

1. Breastfeeding mothers should pay attention to correct poor breastfeeding posture and avoid excessive frontal extension of the baby's jaw.
2. Help children correct bad oral habits. For example, babies who bite their upper lip or extend their jaw should be intervened as early as possible to get rid of bad habits.

Influencing factors



Controllable factors

1. Improper feeding: Improper posture during breastfeeding (such as holding the bottle too high and sucking the jaw forward forcefully) may affect the child's overextended jaw.
2. Bad oral habits: bad habits such as biting the upper lip, spitting out the tongue and extending the jaw may lead to jaw protrusion.



Uncontrollable factors

1. Race: The prevalence rate of East Asian and African population is higher than that of white European.
2. Local obstacles in the replacement period: retention or early loss of deciduous teeth may cause individual anterior teeth to reverse occlusion and mandibular protrusion.
3. Diseases: Studies have shown that acromegaly may cause the body to produce too much growth hormone (GH), which makes the mandibular enlargement and jaw projection. In addition, basal

cell nevus syndrome may cause abnormal facial features.

Knowledge

Onset time

It is usually found in early childhood or adolescence.

Prevalence

The prevalence is higher among east Asians, such as Koreans, Chinese and Japanese (8-15%), and is relatively rare among people of European American descent (0.48-4%).

Typical symptoms

The facial features of anterior teeth were reversed occlusion, partial combination of posterior teeth with mesial malocclusion, mandibular protrusion and concave lateral shape of maxillary underdevelopment.

Treatment

1. Orthodontic therapy: mainly adjust the facial bones, teeth and jaw facial nerve and muscle coordination through a variety of corrective devices.
2. Orthodontic surgery: it's a kind of surgical treatment according to malformation and treatment requirements, reconstructing the three-dimensional spatial relationship and function of normal jaw structure, and achieving reasonable maxillofacial beauty effect.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS229038	intergenic	GG	1.00	0.96
RS1793953	COL2A1	GG	1.00	0.98
RS4654388	intergenic	AA	1.00	0.98

Risk calculation

The risk value of this test is calculated based on published academic literatures related to Mandibular protrusion. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between the patients with Mandibular protrusion and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Mandibular protrusion risk item uses three polymorphic loci on *EPB41*, *COL2A1* and between them. The test result indicates whether the user has a higher possibility of Mandibular protrusion in genotype combination of the three polymorphic loci detected compared with all users, and the test does not represent the user's real risk of Mandibular protrusion. The test is mainly based on the research of East Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to Mandibular protrusion.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Mandibular protrusion from the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
 3. If you have a family history of Mandibular protrusion, or you think that you are likely to suffer from Mandibular protrusion, it is recommended that you consult an authoritative health professional or clinician for help.
-

Test content

The test includes rs229038, rs4654388 and rs1793953, three polymorphic loci associated with the risk of Mandibular protrusion.

Population samples

The basis of the test is mainly from the research of East Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with Mandibular protrusion.

Lung Cancer

Lung cancer refers to primary malignant tumors in the lungs and bronchi.

My Genetic Risk Is Average For The Population

1.50 times

Average incidence rate is 49 per 100,000 in the population

Suggestions

- Red flags: In the physical examination report, **carcinoembryonic antigen (CEA)**, **carbohydrate antigen (CA125)**, etc. Serum tumor markers are too high
- High-risk population: smoking, over 50 years of age, drinking, air-polluted areas, excessive intake of β -carotene or vitamin E, use of hormones such as estrogen or progesterone, family history of lung cancer
- How to reduce the risk:
 - Quit smoking: refuse to take the initiative to smoke, stay away from second-hand smoke
 - Balanced diet: avoid excessive intake of β -carotene or vitamin E
 - Reduce the frequency of drinking, control Drinking amount
 - Weight control: Keep BMI between 18.5 and 23.9, avoid sitting for a long time, and exercise at least 4 hours a week
 - Have a history of smoking, and after 50 years old, do low-dose chest CT screening once a year
- The Heritability of lung cancer is about 12%~21%

* If you have a family history of lung cancer, or if you feel you are at risk of developing lung cancer, or need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors



Controllable factors

1. Smoking: Cigarettes cigarettes in particularly are the main cause of lung cancer so far. Cigarette smoke contains at least 73 known carcinogens, and lung cancer patients caused by smoking account for 80 to 90% of lung cancer patients.

-
2. Dietary factors: A large amount of β -carotene intake or excessive vitamin E intake may increase the risk of lung cancer.
 3. Severe alcohol abuse: Excessive intake of alcohol will increase the risk of lung cancer.
 4. Hormone replacement therapy: The use of hormone therapy such as estrogen and progesterone may increase the risk of lung cancer.
 5. Air pollution: Nitrogen dioxide, fine particles (PM_{2.5}) and sulfate aerosols contained in car exhaust, indoor soot and other air pollutants may increase the risk of lung cancer; radon is a colorless and odorless radioactive gases, which may be released from foundation soil, building materials and decoration materials, are the second most common cause of lung cancer in the United States; asbestos can cause a variety of lung diseases, including lung cancer.
 6. Other lung carcinogens: certain metals (aluminum products, cadmium and its compounds, etc.), some combustion products (incomplete combustion products, coal gas, etc.), ionizing radiation (X-rays, gamma rays, etc.), some harmful gas (methyl ether, sulfur mustard, etc.), rubber products and crystalline silica dust are all carcinogens associated with lung cancer.

Uncontrollable factors

Family history: People with a family history of lung cancer have a higher prevalence.

Knowledge

Time of onset

The disease can occur at any age, and people over 50 years of age who have a history of smoking may be the most susceptible to lung cancer.

Prevalence

Prevalence: 49 per 100,000 people. The cancer with the highest incidence and mortality rate among men worldwide is lung cancer. Among women, the incidence of lung cancer ranks third, and the mortality rate ranks second, second only to breast cancer. In 2012, there were 1.82 million new lung cancer patients worldwide, and 1.56 million eventually died of lung cancer, accounting for 19.4% of deaths due to cancer. The highest incidence of lung cancer is in North America, Europe and East Asia. In 2012, more than one-third of new patients appeared in China, while the incidence of lung cancer in Africa and South Asia was much lower.

Typical symptoms

The clinical manifestations of lung cancer are more complicated. The presence, severity, and time of symptoms and signs depend on the tumor site, pathological type, metastasis, and complications, as well as differences in patient response and tolerance. The early symptoms of lung cancer are often mild and may even be uncomfortable. The symptoms of central lung cancer appear early and severe,

and the symptoms of peripheral lung cancer appear late and mild, even asymptomatic, and are often found during physical examination. The symptoms of lung cancer are roughly divided into: local symptoms, systemic symptoms, extrapulmonary symptoms, infiltration and metastasis symptoms.

1. Local symptoms, including cough, blood or hemoptysis in sputum, chest pain, chest tightness, shortness of breath and hoarseness.
2. Systemic symptoms, including fever, weight loss and cachexia.
3. Extrapulmonary symptoms. Due to some special active substances (including hormones, antigens, enzymes, etc.) produced by lung cancer, patients may have one or more extrapulmonary symptoms, which often appear before other symptoms, and may follow the tumor Pulmonary osteoarticular hyperplasia is more common in clinical practice.
4. Symptoms of external invasion and metastasis, including lymph node metastasis, pleural invasion and metastasis, superior vena cava syndrome, kidney metastasis, digestive tract metastasis, bone metastasis, central nervous system symptoms, heart invasion, metastasis and peripheral nervous system symptoms .

Treatment

The treatment of lung cancer includes surgical treatment, radiotherapy, chemotherapy and immunotherapy. Surgical treatment has been recognized as the first choice for the treatment of lung cancer, and the treatment plan should be selected based on the clinical stage of lung cancer. Radical resection is so far the only possible treatment for lung cancer patients to be cured and return to normal life. Before surgery, it is necessary to assess whether the patient tolerates the surgery. These tests usually include: clinical physical examination, pulmonary ventilation function, blood test and so on. For marginal patients, lung perfusion scans should be used to more accurately assess lung function, or endurance tests and coronary angiography should be used to assess heart function.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS17576	MMP9	AG	1.68	0.99
RS664143	ATM	AG	1.60	0.99
RS11614913	MIR196A2	CT	1.54	0.98
RS1169300	HNF1A	AG	1.51	0.97
RS2352028	GPC5	CT	1.46	0.97

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to lung cancer. These literatures have found some polymorphic loci related to the risk of the disease by studying lung cancer patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of lung cancer item uses the 18 polymorphic loci in and between *MMP9*, *HNF1A*, *EGFR*, *GPC5*, *MPO*. The test results indicate whether the user has a higher prevalence of lung cancer in the combination of genotypes at the 18 polymorphic loci tested compared to all users. This test does not represent the user's true risk of lung cancer. The basis of the test is mainly from the research of Caucasian and Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of lung cancer.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of lung cancer at a genetic level, it does

not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.

3. If you have a family history of lung cancer, or you think that you are likely to suffer from lung cancer, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 18 polymorphic loci related to the risk of lung cancer, including rs2352028, rs2464196 and rs2293347.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to lung cancer.

Sjögren's Syndrome

Sjögren's syndrome is also known as autoimmune exocrine gland epithelial cell inflammatory disease or autoimmune exocrine disease, as this disease invades the lacrimal glands, salivary glands and other exocrine glands.

My Genetic Risk Is Average For The Population

1.46 times

Average incidence rate is 770 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Gender: Sjogren's syndrome tends to occur in women, and the prevalence rate of women is about 9-10 times that of men.
2. Age: the onset age of Sjogren's syndrome is mostly 40-50 years old.
3. Family history: Sjogren's syndrome has a certain family genetic tendency.
4. Infection: some viral infections (e.g., EB virus, Cytomegalovirus, HIV) are associated with Sjogren's syndrome.

Knowledge



Time of onset

The age of onset is mostly 40-50 years old.



Prevalence

The prevalence of Sjogren's syndrome in China is about 0.29%-0.77%.



Typical symptoms

Most patients show manifestations such as dry mouth and eyes, and some may also have dry skin, nasal cavity, and vaginal dryness.

Treatment

There is no cure for Sjogren's syndrome, which is mainly symptomatic treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS7574865	STAT4	TT	2.10	0.96
RS10168266	STAT4	TT	2.07	0.97
RS2736345	intergenic	GG	1.69	0.99
RS2729935	intergenic	AA	1.69	0.99
RS583911	IL12A-AS1	GG	1.61	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to Sjogren's syndrome. By studying the genotype differences between patients with Sjogren's syndrome and normal people, these literatures found some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Sjogren's syndrome health risk item uses 17 polymorphic loci on genes such as *STAT4*, *TNPO3*, *GTF2I*, *TNFAIP3*, *HLA-DRA* and other genes. The results show whether the user had a higher incidence of Sjogren's syndrome than all users in the combination of genotypes at the 17 polymorphic loci tested, which does not represent the user's true risk of Sjogren's syndrome. The basis of the test is mainly from the research of Caucasian and Chinese, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of developing Sjogren's syndrome.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. this study only predicts your risk of developing dry syndrome at a genetic level and does not represent your true health status or judgment of your other health conditions, lifestyle habits, etc.
3. If you have a family history of Sjogren's syndrome, or if you feel you have a risk of developing Sjogren's syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 17 polymorphic loci associated with the risk of Sjogren's syndrome, including rs10488631, rs7574865, rs117026326.

Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with Sjogren's syndrome.

Allergic Rhinitis

Allergic rhinitis, or hay fever, is a common otorhinolaryngological allergic disease of the respiratory tract.

My Genetic Risk Is Average For The Population

1.42 times

Average incidence rate is 20000 per 100,000 in the population

Suggestions

1. Keep the room clean, and wash the sheets and bedding at least once every 1 month; close the windows as much as possible during the flowering season and stay away from trees that are prone to blooming, so as to keep away from potential allergens.
2. If the air pollution in the city where you live is serious, it is best to wear a mask when you go out and move to a place with good air quality if you can.

Influencing factors



Controllable factors

1. Allergens: Allergens are mainly divided into inhaled allergens and food allergens. Inhaled allergens are the main cause of allergic rhinitis, such as dust mites, pollen, animal dander, mold and cockroaches. Food allergens include food (such as fish, eggs, milk) and drugs.
2. Air pollution: Air pollution (SO₂, NO) will increase the permeability of the nasal mucosa and enhance the response to allergens.
3. Cold air irritation: Cold air easily irritates the mucous membrane of the respiratory tract and induces rhinitis.
4. Mental factors: Mental stress, tension and excessive fatigue can easily induce nasal sensitivity.



Uncontrollable factors

1. Age: The majority of patients with this disease are young adults, and most of them are 15-40 years old.
2. History of atopic diseases: Patients with eczema and asthma are susceptible to allergic rhinitis.

Knowledge

Time of onset

Allergic rhinitis can occur at any age, but it mainly occurs in young adults, and it is now found to be more common in children. This disease is more common in people aged 15 to 40.

Prevalence

The incidence of this disease has increased significantly in the past 20 years, especially in developed countries. The incidence rate in Western countries is 10%-30%.

Typical symptoms

The typical symptoms of allergic rhinitis are mainly paroxysmal sneezing, clear watery nasal discharge, nasal congestion and nasal itching, and some are accompanied by hyposmia. The specific symptoms are:

1. Sneezes: Paroxysmal seizures several times a day, more than 3 at a time, mostly in the morning or at night or immediately after exposure to allergens.
2. Clear nasal discharge: A lot of clear watery nasal discharge, sometimes dripping from the nostril unconsciously.
3. Nasal congestion: Intermittent or continuous, unilateral or bilateral, with varying degrees of severity.
4. Nasal itching: Most patients have itchy inside the nose, and hay fever patients may be accompanied by itchy eyes, ears and throat.

Treatment

There is no cure for allergic rhinitis, but the symptoms can be alleviated by staying away from allergens, drugs and desensitization treatments.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS1059513	STAT6	CT	1.30	1.00
RS2155219	intergenic	GT	1.18	1.00
RS148505069	intergenic	GG	1.14	1.00
RS34004019	intergenic	AG	0.89	1.00
RS2070902	FCER1G	TT	1.10	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to allergic rhinitis. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between patients with allergic rhinitis and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The allergic rhinitis health risk item uses 32 polymorphic loci on and between genes such as *STAT6*, *RERE*, *LPP*, *JAZF1*, etc. The test results indicate that compared with all users, whether this user has a higher prevalence of allergic rhinitis in the combination of genotypes of the 32 polymorphic loci tested. It does not mean the user's true risk of allergic rhinitis. The basis of the test is mainly from the research of Caucasian, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that cause the risk of allergic rhinitis.
2. The test result has no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of the disease is determined by multiple factors such as genes and environment. This study only predicts your risk of allergic rhinitis from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of allergic rhinitis, or you think that you are likely to suffer from allergic rhinitis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 32 polymorphic loci related to the risk of allergic rhinitis, including rs2155219, rs7775228, and rs1059513.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to allergic rhinitis.

Back Pain

Back pain refers to pain in the back, waist, lumbosacral and sacroiliac areas, sometimes accompanied by lower limb pain or radiating pain.

My Genetic Risk Is Average For The Population

1.39 times

Average incidence rate is 22000 per 100,000 in the population

Suggestions

1. Do 75 minutes of high-intensity (running and jumping) exercises at least or 150 minutes of medium-intensity (brisk walking) exercises every week. You can choose exercises that use the waist, abdomen and back muscles, such as swimming, planks, abdominal crunches, ect.
2. Maintain a good standing and sitting posture, avoid bending over and hunchback; when sitting for a long time or driving, you can put a pillow on the lower back to support, and try to move your body every hour.
3. Maintain good living habits, eat a reasonable diet, do not stay up late, do not smoke, and do not abuse alcohol.

Influencing factors



Controllable factors

1. Posture: sitting for a long time, maintaining a fixed posture for a long time, or lacking a good standing and sitting posture may increase the risk of back pain.
2. Exercise frequency: those who lack exercise are at higher risk of back pain.
3. Life habits: back pain can be affected by many factors. If poor life habits lead to poor health, the risk of back pain will also increase.



Uncontrollable factors

Age: Middle-aged and elderly people are more likely to have back pain.

Knowledge

Time of onset

The disease can occur at any age, and the probability of back pain is higher in middle-aged and elderly people.

Prevalence

Back pain is one of the most common diseases. In the United States, acute back pain is the fifth most frequently diagnosed disease. On average, 9 out of 10 adults will experience back pain at least once in their lifetime. Half of the workers suffer from back pain every year.

Typical symptoms

The pain is mostly dull pain, tingling, local tenderness or with radiating pain, inability to move, inconvenience in pitching, inability to hold weight, difficulty in walking, fatigue and weakness of the limbs, and even symptoms such as lumbar flexion, extension, scoliosis, etc. Obstacles, severe spinal deformities appear. Back pain worsens when coughing and increasing abdominal pressure.

Treatment

Hyperthermia can relieve the symptoms of acute and subacute back pain. In some cases, cold compresses can also effectively relieve back pain. Massage and physical exercise are also effective ways to relieve pain, and are more suitable for chronic back pain. Psycho-cognitive therapy can also be useful. In all other treatment options have been tried or in emergency situations, surgical treatment will be used.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1934268	PTPRD	TT	2.52	0.98
RS2073711	CILP	AG	1.46	0.99
RS7833174	intergenic	CT	1.05	1.00
RS4384683	DCC	AG	0.97	1.00

Risk calculation

The risk value of the test is calculated based on published academic literature on back pain. By studying the genotype differences between back pain patients and normal people, these literatures found some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The back pain health risk item uses *CILP*, *PTPRD* genes, and 4 polymorphic loci between genes. The results show whether the user had a higher rate of back pain than the genotype combination of the 4 polymorphic loci tested compared to all users, and that the test did not represent the user's real risk of back pain. The basis of the test is mainly from the research of Asian and European, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of back pain.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of back pain at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
-

3. If you have a family history of back pain, or if you feel you have a risk of back pain, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 4 polymorphic loci related to the risk of back pain, including rs2073711, rs1934268 and rs7833174.

 Population samples

The study used in this test mainly Asian and European.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with back pain.

Progressive Supranuclear Palsy (PSP)

Progressive supranuclear palsy is an idiopathic neurodegenerative disease, a common Parkinson's syndrome, which progresses rapidly after onset.

My Genetic Risk Is Average For The Population

1.37 times

Average incidence rate is 7 per 100,000 in the population

Influencing factors



Uncontrollable factors

Age: The risk factor for progressive supranuclear palsy is age, which usually affects people around 60 years old.

Knowledge



Time of onset

The average age of onset is 63 years old. The onset is mostly insidious and gradually worsens.



Prevalence

The incidence of progressive supranuclear palsy is about 6.5/100000.



Typical symptoms

1. Extrapyrarnidal symptoms: early gait instability, balance disorder, and cervical dystonia.
2. Mental symptoms: memory loss and cognitive impairment gradually appear.
3. Eye movement disorder: mainly manifested as the vertical movement of the eyeball, that is, the inability to move up and down, and sometimes it can affect the activities in other directions as the disease develops.
4. Pseudobulbar palsy: manifested as dysarthria and difficulty swallowing.

Treatment

There is currently no specific treatment, and medications can alleviate symptoms.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS242557	MAPT	AG	0.51	0.89
RS1768208	MOBP	CT	1.71	0.99
RS1411478	STX6	GG	1.00	0.99
RS6687758	intergenic	AA	1.00	0.99
RS8070723	MAPT	AA	1.00	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to progressive supranuclear palsy. These literatures found some polymorphic sites associated with the risk of the disease by studying the genetic differences between patients with progressive supranuclear palsy and normal people. We uses the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of progressive supranuclear palsy item uses the 6 polymorphic loci in *MOBP*, *MAPT*, *STX6*, and *SLCO1A2*. The test results indicate whether the user has a higher prevalence of progressive supranuclear palsy in the combination of genotypes at the 6 polymorphic loci tested compared to all users. This test does not represent the user's true risk of progressive supranuclear palsy. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of progressive supranuclear palsy.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of progressive supranuclear palsy at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of progressive supranuclear palsy, or you think that you are likely to suffer from progressive supranuclear palsy, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 6 polymorphic loci related to the risk of progressive supranuclear palsy, including rs1768208 and rs8070723.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to progressive supranuclear palsy.

Dupuytren's Disease

Dupuytren's disease is a progressive disease characterized by the formation of numerous nodules and cord-like structures in the palmar fascia and finger fascia caused by proliferative fibrosis, thus leading to secondary flexion contracture of the finger joints.

My Genetic Risk Is Average For The Population

1.35 times

Average incidence rate is 3 per 100,000 in the population

Suggestions

1. Control alcohol intake: the limit value of moderate drinking for adults recommended by the Chinese Nutrition Society is that the amount of alcohol consumed by adult men should not exceed 25g per day; the amount of alcohol consumed by adult women should not exceed 15g per day.
2. Reject active and passive smoking, and avoid exposure to tobacco.
3. Maintain a healthy weight: BMI is maintained between 18.5-23.9.

Influencing factors



Controllable factors

1. Long-term alcohol intake may increase the risk of the disease.
2. Smoking may increase the risk of the disease.
3. Too low body weight and lower than average BMI index will increase the risk of disease.



Uncontrollable factors

1. Age: most of the disease is over 50 years old, and the risk increases with age.
2. Gender: the incidence of men is about 8-10 times higher than women.

- 3. Ethnic groups: the overall incidence of European and American populations is higher than Asian populations.
- 4. Other diseases: hypertension, alcoholism, diabetes (especially Type I), hyperlipidemia, ischemic heart disease, chronic obstructive pulmonary disease (COPD), tuberculosis, epilepsy and rheumatoid arthritis (RA), can lead to an increase in the risk of Dupuytren's disease.

Knowledge



Time of onset

Most of the onset time is after the age of 50.



Prevalence

In 2020, worldwide prevalence of Dupuytren's disease is 8.2%. Africa has the highest prevalence, which is 17.2%; And America has the lowest prevalence, which is 2.3%.



Typical symptoms

The disease generally progresses slowly and can last for several years and decades. Early symptoms are usually small nodules under the skin of the palms. These nodules can gradually form longitudinal cord-like lumps, leading to bending contractures of the fingers, and there is generally no obvious tenderness.



Treatment

If the disease is mild and the hand function has not caused a disorder, you can leave it out for the time being, and focus on observation. When treatment is needed, medication can be used; when necessary, surgery can be performed for correction.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS16879765	EPDR1	CT	1.98	0.96
RS11672517	DUXA	AA	1.80	0.98
RS611744	EIF3E	GG	0.59	0.98

RS2598107	EPDR1	CT	1.48	0.96
RS1509406	NEDD4	GG	1.35	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is calculated based on published academic literatures related to Dupuytren's disease. By studying the genotype differences between Dupuytren's disease patients and normal people, these literatures have found some polymorphic sites associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of Dupuytren's disease item uses the 27 polymorphic loci in and between genes, such as *EPDR1*, *WNT2*, *NEDD4*. The test results indicate whether the user has a higher prevalence of Dupuytren's disease in the combination of genotypes at the 27 polymorphic loci tested compared to all users. This test does not represent the user's true risk of Dupuytren's disease. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of Dupuytren's disease.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

-
1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Dupuytren's disease at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
 3. If you have a family history of Dupuytren's disease, or you think that you are likely to suffer from Dupuytren's disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 27 polymorphic loci related to the risk of Dupuytren's disease, including rs16879765, rs4730775 and rs6519955.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to Dupuytren's disease.

Bipolar Disorder

It is a type of mood disorder and belongs to mental illness. Bipolar refers to the depressive phase and the manic phase, which alternately appear repeatedly.

My Genetic Risk Is Average For The Population

1.33 times

Average incidence rate is 3700 per 100,000 in the population

Suggestions

1. Limit alcohol intake. The limit value for moderate drinking recommended by the Chinese Nutrition Society is that the amount of alcohol consumed by adult men should not exceed 25g per day; the amount of alcohol consumed by adult women should not exceed 15g per day.
2. Avoid contact with marijuana, cocaine and amphetamines.
3. Choose a social environment with high tolerance and low life pressure for work and life, and maintain harmonious and harmonious relations with relatives, friends and colleagues. Improve your ability to cope with psychological pressure and burden of life, so as not to induce disease.

Influencing factors



Controllable factors

1. Alcohol abuse: alcohol may cause chronic mental disorders caused by active substances through induced mechanisms, increasing the risk of bipolar disorder.
2. Marijuana and drugs: marijuana and some drugs that act on the nervous system (including amphetamines, cocaine, etc.) can lead to mental disorders and increase the risk of bipolar disorder.
3. Social environment: people who have been under high stress for a long time have an increased risk of developing the disease in the future.



Uncontrollable factors

-
1. Age: the high incidence of bipolar disorder is in young adults.
 2. Family history: people with family history have a higher prevalence.
 3. Nerve damage: some neurological impairment disorders may be closely related to the onset of bipolar disorder, including stroke, traumatic brain injury, HIV infection, multiple sclerosis, porphyria and temporal lobe epilepsy.
 4. Childhood experiences: people who suffer from mental trauma as children have an increased risk of developing the disease later on.
-

Knowledge

Time of onset

Generally speaking, bipolar disorder usually occurs around the age of 25, and there is no difference in the incidence between different sexes. It also occurs in children and adolescents. Compared with adults, the symptoms are more vague and the onset time is shorter.

Prevalence

Incidence rate: 3770 per 100,000 people. It is a relatively common mental illness. According to statistics, 1%-3% of adults worldwide suffer from bipolar disorder at some stage in their lives.

Typical symptoms

Bipolar disorder is a type of mood disorder, which refers to a type of disease that has both manic episodes and depressive episodes. The clinical manifestations of bipolar disorder can be divided into depressive episodes, manic episodes or mixed episodes according to the characteristics of the episodes.

1. Depressive episodes: Compared with unipolar depression, bipolar depression has a more rapid onset, shorter duration, and more frequent recurrent episodes. The characteristics of bipolar depression that are different from unipolar depression include emotional instability, irritability, psychomotor agitation, thinking competition/crowding, increased sleep, obesity/weight gain, inattention, and more Suicidal conceptions, comorbid anxiety and substance abuse (tobacco, alcohol, drugs, etc.).
2. Manic episode: People with milder clinical manifestations of manic episodes are called hypomania. Patients may have elevated mood, energetic, increased activity, significant self-feeling, inattention, and also lasting for at least a few days. Inability to last, mild profligacy, increased social activities, increased libido, and reduced sleep needs. Sometimes manifested as irritability, arrogance, and reckless behavior, but not accompanied by hallucinations, delusions and other psychotic symptoms. It has a slight impact on the social function of patients, and some patients sometimes fail to reach the level of affecting social function. It is often difficult for ordinary people to notice.

3. Mixed episodes: refers to the simultaneous occurrence of manic symptoms and depressive symptoms in one episode, which is relatively rare in clinical practice. It usually occurs when mania and depression change rapidly. For example, a patient with a manic episode suddenly becomes depressed and then reverts to mania a few hours later, giving the impression of "mixed". However, this mixed state generally lasts for a short period of time, and most of them quickly turn into a manic or depressive phase. Both manic symptoms and depressive symptoms in mixed episodes are atypical, and it is easy to be misdiagnosed as schizophrenia or schizophrenia.

 Treatment

The treatment of manic episodes of bipolar disorder mainly includes drug therapy, psychotherapy, physical therapy (MECT, commonly known as electrotherapy), and crisis intervention. Among them, the main treatment is medication, with mood stabilizers as the mainstay, and antipsychotic drugs mainly used to treat schizophrenia, hypnotic drugs mainly used to treat insomnia, and even depression Antidepressants. The treatment of depressive episodes in bipolar disorder is similar to the treatment of depression, but it is especially necessary to prevent suicide and prevent the symptoms from depressive episodes to manic episodes. Antidepressant drugs are often used in conjunction with mood stabilizers.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS10994336	ANK3	CT	1.45	0.98
RS17418283	MCTP1	TT	1.46	1.00
RS7757037	FKBP5	AG	0.68	0.99
RS4027132	intergenic	AG	1.39	0.99
RS1006737	CACNA1C	GG	1.39	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to bipolar disorder. By studying the genetic differences between bipolar disorder patients and normal people,

these literatures have identified some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The bipolar disorder health risk item uses 71 polymorphic loci on the *SORCS2*, *DGKH*, *FKBP5*, *ANK3* and *DPP10* genes. The results showed whether the user had a higher prevalence of bipolar disorder than all users in the genotype combination of the 71 polymorphic loci tested, and that the test did not represent the user's real risk of bipolar disorder. The basis of the test is mainly from the research of Caucasian, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of bipolar disorder.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of bipolar disorder at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you have a family history of bipolar disorder, or if you feel you have a risk of developing bipolar disorder, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 71 polymorphic loci related to the risk of bipolar disorder, including rs10937823, rs4027132 and rs1064395.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with bipolar disorder.

Paget's Disease of Bone

Paget's disease of bone refers to a chronic bone disease of unknown etiology mainly characterized by progressive rheumatoid arthritis, deformities of the spine and limbs, pathological fractures, and brain and spinal cord compressive symptoms.

My Genetic Risk Is Average For The Population

1.32 times

Average incidence rate is 1000 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Viral infection: studies have found that paramyxovirus infection, measles virus and canine distemper virus infection may increase the risk of deformed osteoarthritis. But there is no definitive basis.
2. Family history: people with a family history of deformed osteoarthritis have a higher prevalence.

Knowledge



Time of onset

Cases of under-55s are rare. With the increase of age, the incidence of deformed osteoarthritis increases.



Prevalence

Prevalence: 1000/100,000. The disease is common in the United States, Australia, New Zealand and so on, the incidence rate of people over 40 years of age is 1% to 3%, to 80 years old incidence rate as high as 10%. Deformed osteoarthritis is rare in Asian countries.



Typical symptoms

- 1. Pain, bone deformities and fractures: nearly 80 percent of cases show pain, and nearly half of them show joint pain. The disease can affect any bone in the human body, the most commonly affected bones are the pelvis, lumbar spine, femur, thoracic spine, sacrum, skull, tibia and humerus, etc. The bone becomes brittle at the lesion, which can cause fractures after spontaneous or minor trauma, especially in the long bones of limbs, which are often transverse fractures.
- 2. Vascular proliferation: excessive proliferation of blood vessels in the bone and skin at the lesions increases local temperature. Over-formed blood vessels at cranial lesions can obtain blood from the external carotid artery, making the superficial temporal artery coarse and curvy, resulting in insufficient blood supply to the brain (vascular steal syndrome), which can be manifested as lethargy, loneliness and indifference. Even lower limb paresis, lateral paralysis and congestive heart failure can occur.



Treatment

- 1. Medication: including calcitonin, bisphosphonate complex, nonsteroidal anti-inflammatory drugs, pramycin and so on.
- 2. Surgical treatment: surgical treatment may be considered when deformed osteoarthritis causes severe bone malformations (arched leg, pelvic malformations, etc.), or when nerve pressure causes severe pain that the drug cannot control, or when high collateral circulation leads to circulatory failure.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS484959	intergenic	CT	1.82	0.96
RS3018362	intergenic	AA	1.52	0.99
RS1561570	OPTN	CC	0.65	0.98

Risk calculation

The risk value of this test is calculated based on published academic literature related to paget disease of bone 3. By studying the genotype differences between patients with paget disease of bone 3 and normal people, these literatures have found some polymorphic sites associated with the risk of

osteoarthritis. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The paget disease of bone 3 health risk item uses three polymorphic loci on and between *OPTN* genes. The results show whether the user has a greater risk of developing paget disease of bone 3 than all users, but does not represent the user's risk of paget disease of bone 3 from all dimensions. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of paget disease of bone 3.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing paget disease of bone 3 at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you have a family history of paget disease of bone 3, or if you feel you have a risk of paget disease of bone 3, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes rs484959, rs1561570, and rs3018362, three polymorphic loci associated with the risk of paget disease of bone 3.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with paget disease of bone.

Myopia

Myopia is a type of refractive diseases in which near objects appear clear, but objects farther away look blurry.

My Genetic Risk Is Average For The Population

1.31 times

Average incidence rate is 45000 per 100,000 in the population

Suggestions

1. Avoid insufficient exercise: ensure adequate outdoor exercise every day, and ensure more than one hour of exercise time per day.
2. Avoid malnutrition: ensure adequate intake of trace elements, ensure balanced nutrients, eat more foods rich in calcium, zinc, chromium and vitamin D, and reduce sugar intake.
3. Avoid overuse of eyes: during adolescence, you should ensure that you use close eyes for no more than 8 hours a day, and pay attention to rest during eye use; Avoid eye fatigue caused by overuse.
4. Avoid using eyes in bad light: Make sure that there is enough light during eye use, and avoid reading in low light conditions. Avoid bad eye habits such as playing mobile phones after turning off the lights, reading books with a flashlight, etc.
5. Avoid bad hygienic habits: ensure eye hygiene, do not rub your eyes with your hands because of itching. At the same time, avoid foreign objects entering the eyes. If the foreign objects enter the eyes accidentally, please seek medical treatment immediately after washing.
6. Avoid contact with polluted air.

Influencing factors



Controllable factors

1. Exercise factors: School-age people who do not exercise enough have a higher risk of myopia than school-age people who maintain adequate exercise.

2. Nutritional factors: Lack of trace elements and imbalanced nutrients are the predisposing factors for myopia.
3. Factors of how to use eyes: Using eyes at close range for more than 8 hours a day or maintaining the same visual distance for a long time will increase the risk of myopia.
4. Light factor: Using eyes in a poorly lit environment will increase the risk of myopia.
5. Hygiene factors: Some eye diseases caused by unhygienic use of eyes can increase the risk of myopia.
6. Air pollution: Exposure to polluted air is also a predisposing factor for myopia.

Uncontrollable factors

1. Age: It is common in adolescence. People with incomplete eye development is more prone to myopia.
2. Family history: People with family history of myopia (especially high myopia) have a higher risk of myopia.

Knowledge

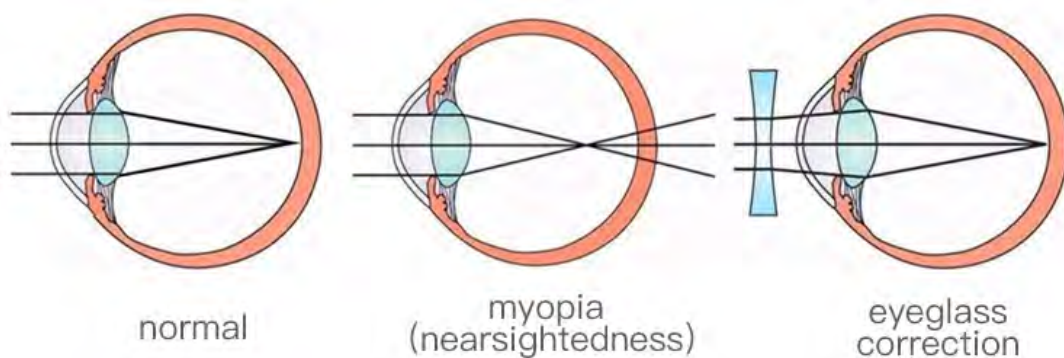
Time of onset

The disease usually occurs in adolescence or early adulthood, and the disease will linger for a lifetime.

Prevalence

According to a research report in 2019, globally, there are approximately 1.95 billion (28.3% of the global population) with myopia and 277 million (4.0% of the global population) with high myopia.

Pathogenesis



The light emitted by the normal eyeball to distant objects is focused on the retina after the refractive action of the cornea and lens, and the image is clear. In myopia patients, because of the decrease in the ability of the ciliary muscle to adjust the curvature of the lens, the light of distant objects cannot

be well focused on the retina after entering the eye, but can only focus on the front of the retina, so when looking at distant objects, they will feel unclear. Patients with myopia can focus the light on the retina again after the lens is corrected, so as to regain a clear vision.

Typical symptoms

- Distal vision is reduced, but near vision is normal. The patients often squint eyes unconsciously when looking at a distant target.
- Affected overuse of the eyes, there will appear some symptoms, such as double shadow, photophobia, dry eyes, itchy eyes, foreign body sensation, eye swelling, headache, etc.
- The collection function of the eyes is correspondingly weakened, which can easily cause exotropia or exotropia.
- When the degree of myopia is different in both eyes, the 3D imaging ability will be affected, and things will not look three-dimensional.
- The eyeball is protruding, the posterior pole of the eyeball is expanded, and the posterior scleral staphyma is formed.
- Degenerative changes in the fundus mostly occur in high myopia, and these patients will have symptoms of varying degrees.

Treatment

Including wearing frame glasses, corneal contact lens (contact lenses) and refractive surgery, myopia correction surgery, etc. There is no definitive conclusion on whether the drug can treat myopia, but there have been clinical studies on drugs such as low-concentration atropine eye drops. Except for surgery, other methods of correcting vision need to be performed for life, but generally speaking, it does not affect normal life and work.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS74764079	BMP3	TT	0.51	1.00
RS5442	GNB3	GG	0.59	1.00
RS698047	intergenic	GG	1.59	1.00
RS74633073	intergenic	CT	1.55	1.00
RS7839488	SNTB1	GG	1.51	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to myopia. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotypes difference between patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The myopia health risk project uses 107 polymorphic loci in genes such as *PRSS56*, *RGR* and *GNB3*. The test result shows whether the user has a higher prevalence of myopia in the genotype results at the 107 polymorphic loci detected compared to all users. This test does not represent the user's real risk of myopia. The basis of the test is mainly from the research of Caucasian and Asian.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of myopia.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease are determined by multiple factors such as genes and environment. This study only predicts your risk of myopia at a genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc
-

3. If you have a family history of myopia, or you think that you are likely to suffer from myopia, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 107 polymorphic loci associated with myopia risk, such as rs10880855, rs1649068 and rs1994840.

 Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that associated with myopia.

Nicotine Dependence

Also known as tobacco dependence, is a neuropsychiatric disease.

My Genetic Risk Is Average For The Population

1.27 times

Average incidence rate is 28100 per 100,000 in the population

Suggestions

1. Reject tobacco, refrain from imitating family smoking, refrain from smoking with peers, refrain from smoking and respecting cigarettes as social means.
2. Try to distract yourself, participate in meaningful activities, and engage in hobbies that are better than smoking.

Influencing factors



Controllable factors

1. Sociological factors: availability, tobacco is easily available everywhere; family factors, imitating family smoking; peer influence, poor juvenile discriminating ability, susceptible to peer influence and smoking; cultural background and social environment, tobacco as a gift, smoking or passing cigarettes as a social means.
2. Psychological factors: personality research, extroverted or easily anxious, sensitive people; psychological strengthening effect, after the formation of dependence, withdrawal disorder makes smokers unable to extricate themselves.



Uncontrollable factors

1. Family history: nicotine dependence has a certain heredity.

Knowledge

Prevalence

In 2016, the smoking rate of the population was 27.7%. Among them, the smoking rate of men was 52.1% and that of women was 2.7%.

Typical symptoms

It is characterized by the uncontrollable impulse to seek for nicotine, and the compulsive and continuous use of nicotine to experience the euphoria and pleasure it brings, and to avoid possible withdrawal symptoms.

Treatment

1. Medication: including nicotine replacement therapy, bupropion, varenicline, etc.
2. Combination therapy: the combined use of nicotine preparations, the combined use of nicotine preparations and bupropion, the combined use of drugs and psychotherapy.
3. Psychological intervention: psychological education intervention, behavioral skill training and cognitive behavioral intervention.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS4648317	DRD2	AG	2.62	0.95
RS806368	CNR1	TT	1.25	1.00
RS1044396	CHRNA4	GG	1.25	1.00
RS151176846	intergenic	TT	0.88	1.00
RS910083	intergenic	CC	1.12	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to nicotine dependence. By studying the genetic differences between nicotine-dependent patients and normal people, these literatures have found some polymorphic sites associated with the risk of the disease.

We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of nicotine-dependent item uses 11 polymorphic loci in genes of *CHRNA4*, *OPRM1*, *CHRNA3*, *CHRNA5*, *DRD2*, and *CNR1*. The test results indicate whether the user has a higher prevalence of nicotine dependence in the combination of genotypes at the 11 polymorphic loci tested compared to all users. This test does not represent the user's true risk of nicotine dependence. The basis of the test is mainly from the research of Asian and Caucasian, Asian, and African American, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of nicotine dependence.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. this study only predicts your risk of nicotine dependence at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
3. If you have a family history of nicotine dependence, or you think that you are likely to suffer from nicotine dependence, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 11 polymorphic loci related to the risk of nicotine dependence, including rs1044396, rs10485057, and rs1051730.



Population samples

The basis of the test is mainly from the research of Caucasian, Asian, and African American.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to nicotine dependence.

Venous Thromboembolism (VTE)

It refers to the abnormal coagulation of blood in the veins, causing the blood vessels to be completely or incompletely blocked. It is a disease of venous return disorder.

My Genetic Risk Is Average For The Population

1.27 times

Average incidence rate is 18 per 100,000 in the population

Suggestions

1. Obesity can easily lead to a series of cardiovascular diseases, and a healthy body weight needs to be controlled: BMI is maintained between 18.5 and 23.9.
 2. Oral contraceptives and estrogen therapy will increase the risk of thrombosis. It is recommended to control the medication time and not use it for a long time.
-

Influencing factors



Controllable factors

1. Obesity: Obese people have a higher probability of venous thromboembolism.
2. Oral contraceptives and estrogen therapy can increase the risk of thrombosis.



Uncontrollable factors

1. Genetic factors: there is a family genetic tendency.
2. Surgical factors: orthopedic surgery, especially bone and joint surgery, is an important cause of venous thromboembolism.
3. Age factor: Generally, people who are over 40 years old are regarded as high-risk factors, and the risk will double for every 10 years old in the future.
4. Malignant tumors, especially pancreatic cancer, gynecological tumors, and brain tumors can increase thrombosis. The toxic effects of chemotherapy on the endothelium may also increase the

formation of thrombosis.

Knowledge

Time of onset

The disease can occur at any age, and the peak age of onset in China is 40 to 60 years old.

Prevalence

An American Heart Association report from 2021 estimated that approximately 1,220,000 total cases of venous thromboembolism occur in the USA annually. A modelling study estimated that the annual venous thromboembolism incidence in six countries in Europe (total population 310.4 million) was 296,000 cases of pulmonary embolism.

Typical symptoms

According to the location and cause of thrombosis, it is divided into deep vein thrombosis (DVT) and pulmonary embolism (PE). Deep vein thrombosis often occurs in the deep veins of the calf, thigh, and buttocks. The clinical manifestations are mainly swelling and pain of the affected limb, sometimes accompanied by superficial varicose veins, skin pigmentation and even ulcer formation. The clinical manifestations of pulmonary embolism are diverse, including sudden breathing difficulties, chest pain, hemoptysis, chills, cold sweats, elevated jugular venous pressure, and hypotension (even circulatory failure and cardiac arrest).

Treatment

The treatment of deep vein thrombosis is based on anticoagulant therapy. Commonly used drugs are heparin, low molecular weight heparin and warfarin. The treatment of pulmonary embolism should be prioritized. Generally, low-risk patients can be treated with anticoagulant thrombolytic therapy to control the progression of thrombosis, and place an inferior vena cava filter as appropriate to prevent further larger thrombosis from returning to the pulmonary artery through the circulation. For patients with a large area of pulmonary embolism in the acute stage, the principle of treatment should be to save lives quickly, restore circulation, and perform thrombolytic therapy at the same time.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS2227624	SERPINC1	AA	0.47	1.00
RS687289	ABO	AA	1.80	0.98
RS78707713	intergenic	TT	1.61	1.00
RS8176749	ABO	TT	1.51	0.99
RS77542162	intergenic	AA	0.77	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to venous thromboembolism. These literatures have found some polymorphic loci related to the risk of the disease by studying venous thromboembolism patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of venous thromboembolism item uses the 108 polymorphic loci in and between genes of *PROCR*, *F2*, *F9*, *LPL*. The test results indicate whether the user has a higher prevalence of venous thromboembolism in the combination of genotypes at the 108 polymorphic loci tested compared to all users. This test does not represent the user's true risk of venous thromboembolism. The basis of the test is mainly from the research of Caucasian, African American, Latino American and Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of venous thromboembolism.
2. The test results have no clinical significance.

3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of venous thromboembolism at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of venous thromboembolism, or you think that you are likely to suffer from venous thromboembolism, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 108 polymorphic loci related to the risk of venous thromboembolism, including rs1799963, rs6025, rs6048 and rs268c

Population samples

The basis of the test is mainly from the research of Caucasian, African American, Latino American and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to venous thromboembolism.

Alcoholic Fatty Liver Disease (AFLD)

Alcoholic fatty liver is hepatocyte steatosis caused by long-term heavy drinking.

My Genetic Risk Is Average For The Population

1.23 times

Average incidence rate is 5100 per 100,000 in the population

Suggestions

Drinking less alcohol can significantly reduce the risk of suffering from alcoholic fatty liver.

Influencing factors



Controllable factors

Alcohol intake: excessive alcohol intake can cause steatosis of hepatocytes.



Uncontrollable factors

Heredity: the polymorphism of some loci in the genome affects the risk of alcoholic fatty liver.

Knowledge



Time of onset

Patients generally have a drinking history of more than 5 years, daily intake of 80 grams of alcohol for more than 10 years is prone to alcoholic fatty liver, but there are great differences among individuals.



Prevalence

The standardized prevalence of alcoholic fatty liver was 0.51% in the total population and 2.98% in the alcoholic population.

Typical symptoms

The most common manifestation of alcoholic fatty liver is enlargement of the liver, followed by pain and tenderness in the liver region, and mild jaundice in a few patients. Severe patients have ascites, lower limb edema, occasionally splenomegaly.

Treatment

Abstinence is the most important measure for the treatment of alcoholic fatty liver. In addition, high-protein diet, low-fat diet and appropriate supplementation of vitamin B, vitamin C, vitamin K and folic acid can be used as adjuvant treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS738409	PNPLA3	CC	3.06	0.94
RS58542926	TM6SF2	CC	2.89	0.99
RS626283	intergenic	CG	1.35	0.99

Risk calculation

The risk value of this test is calculated based on published academic literatures related to alcoholic fatty liver. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between subjects with alcoholic fatty liver and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The alcoholic fatty liver item uses 3 polymorphic loci in and between *PNPLA3* and *TM6SF2* genes. The test results indicate whether the user has a higher risk of alcoholic fatty liver for the genotype combination of 3 polymorphic loci detected compared to all users. The test cannot represent the user's real risk of alcoholic fatty liver. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that contribute to the risk of alcoholic fatty liver.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are jointly determined by multiple factors such as genes and environment. This study only predicts your risk of alcoholic fatty liver at the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If there is a family history of alcoholic fatty liver in your family, or if you think you are at risk of developing alcoholic fatty liver, you are recommended to consult an authoritative health professional or clinician for help.

Test content

Three polymorphic loci associated with the risk of alcoholic fatty liver were detected, thus, rs738409, rs58542926 and rs626283.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect alcoholic fatty liver.

Hypothyroidism

Hypothyroidism is systemic low metabolic syndrome resulting from low thyroid hormone levels or thyroid hormone resistance caused by a variety of factors. The pathology of the disease is characterized by accumulation of mucopolysaccharides in the tissue or skin, which manifests as mucinous edema.

My Genetic Risk Is Average For The Population

1.15 times

Average incidence rate is 2000 per 100,000 in the population

Suggestions

1. According to the recommendations of the World Health Organization, the daily intake of iodine for adults is 150µg, and it is recommended to add iodized salt (about 6g) when cooking.
2. The World Health Organization recommends that women during pregnancy and breastfeeding consume 250µg of iodine per day. It is recommended that iodine supplements be used rationally under the guidance of a doctor.

Influencing factors



Controllable factors

1. Diet: the lack of iodine in food is the main cause of hypothyroidism.
2. During pregnancy: women during pregnancy and lactation have 66% more iodine requirements than other women.



Uncontrollable factors

1. Family history: hypothyroidism tends to cluster in families. If relatives have autoimmune thyroid disease, the risk of illness will increase.
2. Autoimmune diseases: people with autoimmune thyroiditis (such as Hashimoto's thyroiditis, atrophic thyroiditis, postpartum thyroiditis, etc.) have a higher chance of concurrent

hypothyroidism.

3. Thyroid destruction: the incidence of hypothyroidism will increase for those who have received thyroid surgery and radioactive iodine treatment in the past.
4. Gender: hypothyroidism is more common in women, and the incidence rate of men and women is 1:4-1:5.

Knowledge

Time of onset

It can occur at all ages, from the newborn to the elderly, and it is more common in the elderly.

Prevalence

The prevalence rate of hypothyroidism in non-iodine-deficient areas is 0.3%-1.0%, and it can reach 2% over 60 years old. The prevalence rate of neonatal hypothyroidism is 1:7000-1:3000.

Typical symptoms

Fear of cold, weight gain, tiredness, indigestion, irregular menstruation, dry and cold skin, edema on the face and hands, hoarseness, thinning of hair, especially the loss of 1/3 of the eyebrows.

Treatment

Thyroid hormone is mainly supplemented by oral administration. If hypothyroidism is caused by other diseases, attention should be paid to the treatment of the primary disease.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2476601	PTPN22	GG	0.54	1.00
RS7850258	intergenic	GG	1.80	0.99
RS3184504	SH2B3	CC	0.71	1.00
RS4915077	VAV3	CT	1.30	0.99
RS2517532	intergenic	AG	1.16	0.99

Risk calculation

The risk value of the test is calculated based on published academic literatures related to hypothyroidism. By studying the genotype differences between hypothyroidism patients and normal people, these literatures have found some polymorphic sites associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of hypothyroidism item uses the 5 polymorphic loci in genes of *PTPN22*, *SH2B3*, *VAV3*. The test results indicate whether the user has a higher prevalence of hypothyroidism in the combination of genotypes at the 5 polymorphic loci tested compared to all users. This test does not represent the user's true risk of hypothyroidism. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of hypothyroidism.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of hypothyroidism at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
-

3. If you have a family history of hypothyroidism, or you think that you are likely to suffer from hypothyroidism, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 5 polymorphic loci related to the risk of hypothyroidism, including rs4915077, rs3184504 and rs2476601.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to hypothyroidism.

Late-Onset Alzheimer's Disease

It is a slow onset and brain function degradation (neurodegenerative) disease, that is not easy to identify. About 95% of Alzheimer's disease is late-onset Alzheimer's disease, often after the age of 65.

My Genetic Risk Is Average For The Population

1.14 times

Average incidence rate is 4900 per 100,000 in the population

Suggestions

- Regular testing of homocysteine (Hcy): A comprehensive evidence-based guideline for Alzheimer's disease published in 2020 recommended that the level of blood Hcy should be monitored regularly. Vitamin B and/or folic acid should be applied to patients with hyperhomocysteinemia, with close monitoring of their cognitive function.
- Prevention:
 1. Weight management is important, especially those over 65 years old should keep their body mass index (BMI) within a certain range (18.5-23.9), and should not be too thin.
 2. Dietary intake or additional vitamin C supplementation.
 3. Refuse active and passive smoking and avoid exposure to the tobacco environment.
 4. Ensure adequate and good sleep, and timely diagnose and treat sleep disorders.
 5. Encourage more participation in daily social activities, including mental and physical activity, to improve mood, calm the mood, and reduce anxiety.
 6. Challenge memory and intelligence on a regular basis. Chess and board games, use these games to stimulate the brain and make the mind work. Use your brain more, keep your enthusiasm and curiosity for learning at all times.
 7. Take good care of cardiovascular and cerebrovascular health. For stroke patients, especially patients with cerebral microbleeds, close monitoring of their cognitive function changes, and effective preventive measures are vital; To avoid orthostatic hypotension, the cognitive function of patients with orthostatic hypotension should be closely monitored.
 8. Be careful to avoid high-risk factors, such as avoiding head trauma, avoiding viral infections.

Influencing factors



Controllable factors

1. Weight: overweight or underweight will increase the risk of late-onset Alzheimer's disease.
2. Diet: the study found that compared with the control group, the risk of Alzheimer's disease in the population supplemented with vitamin C decreased by 16%.
3. Smoking: having a history of smoking or smoking in later life increases the risk of late-onset Alzheimer's disease.
4. Sleep: circadian rhythm disorder is related to the development of late-onset Alzheimer's disease, and sleep disorder may be an early warning signal of the disease.
5. Psychosocial factors: studies have shown that depression, especially in old age, increases the risk of late-onset Alzheimer's disease. Childhood shadows, anxiety, sleep disorders, and psychological distress may also be associated with late-onset Alzheimer's disease.
6. Education level: studies found that more education can significantly reduce the risk of late-onset Alzheimer's disease, and everyone is more or less able to change their education status.
7. Cardiovascular factors: The state of blood circulation in the body is related to the occurrence of late-onset Alzheimer's disease.
8. Other high-risk factors: studies have shown that moderate brain trauma doubles the risk of late-onset Alzheimer's disease and a 4.5-fold increased risk of severe brain trauma; viral infections may also contribute to the disease.



Uncontrollable factors

1. Age: symptoms mostly manifest after age 65, according to statistics, for every 5-year increase in age among older people over the age of 65, the risk increases 1.85 times.
2. Gender: both men and women are at risk of developing late-onset Alzheimer's disease, but women are at higher risk of late-onset Alzheimer's disease.
3. Family history: people with a family history of late-onset Alzheimer's disease are at higher risk. About 5% of patients with late-onset Alzheimer's disease have a relative with the disease (family history).
4. Mother's childbearing age: maternal childbearing too early or too late increases the risk of late-onset Alzheimer's disease.
5. History of hypertension: studies have shown that suffering from hypertension in middle age can significantly increase the risk of late-onset Alzheimer's disease.
6. History of diabetes: patients with diabetes should be closely monitored for cognitive decline.

Knowledge

Time of onset

Late-onset Alzheimer's disease is mostly found in people over 65 (or 60 years old).

Prevalence

Prevalence: 4900/100,000. The incidence of the disease increased with age, and the incidence rate was 5% to 10% over the age of 65. The elderly population aged 85 or above could reach 30 to 40%, and the number of female patients was more than that of men.

Typical symptoms

1. Prophase: Patients with hidden symptoms are easy to ignore, often with near memory loss as the main manifestation. For example, just done things forget. Time-directed obstacles can also occur, and you may not even find your way home.
2. Progress: Memory disorders and directional disorders gradually increase. Obvious signs of dementia include repeated questions about the same question, poor hygiene habits, and decreased judgment. It is difficult to recall the names of family and friends, relatives and colleagues and face recognition difficulties, self-care ability is beginning to have problems.
3. Advanced: Patients is deprived of self-care ability, time, place, character recognition ability also further decreased. Emotions can also gradually change, depression is negative or restless. When a patient's condition deteriorates, he often becomes detached from family and social relations and gradually loses his or her bodily function.

Treatment

Treatment for late-onset Alzheimer's disease consists of two main categories: (1) Control of psychopathic symptoms, such as taking anti-anxiety, antidepressant, antipsychotic drugs to improve symptoms. (2) Improve cognitive function and slow the progression of disease, such as taking drugs that improve the function of brain nerve transfer or drugs that improve brain blood circulation and brain cell metabolism.

Late-onset Alzheimer's disease does not necessarily require hospitalization, but there are often some acute symptoms that require urgent treatment. hospitalization is better for patients, such as severe mental disorders or concurrent infections, strokes. However, neither drug treatment nor physiotherapy nor psychological treatment, may deter the progression of Alzheimer's disease. This disease has no complete cure and can only be delayed in terms of disease development.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS429358	APOE	TT	0.32	1.00
RS5882	CETP	AG	0.52	0.92
RS7101429	GAB2	GG	0.58	0.98
RS928771	KCNJ15	GT	1.59	0.97
RS892086	DNM2	AA	1.55	0.98

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is based on published academic literature related to late-onset Alzheimer's disease. The literature identified some polymorphic sites associated with the risk of developing Alzheimer's disease by studying the genetic differences between patients with late-onset and normal people. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The late-onset Alzheimer's disease item uses 64 polymorphic loci of between the *SORL1*, *PSEN1*, *ABCA2*, *APOE*, *GCH1*, *KCNJ15*, etc. The results showed whether the user had a higher risk of late-onset Alzheimer's disease than all users, compared to the genotype combination of the 64 polymorphic loci tested, which did not represent the user's true risk of late-onset Alzheimer's disease. The basis of the test is mainly from the research of Caucasian, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of Alzheimer's disease.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not applicable to clinical practice.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of late-onset Alzheimer's disease at the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of late-onset Alzheimer's disease, or you think that you are likely to suffer from late-onset Alzheimer's disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 64 polymorphic loci associated with the risk of late-onset Alzheimer's disease, including rs908832, rs7412, and rs5848.

Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci related to late-onset Alzheimer's disease.

Colorectal Cancer

Colorectal cancer is a common malignant tumor of the gastrointestinal tract. Symptoms are not obvious in the early stage, but as the tumor enlarges, changes in bowel habits, blood in stool, diarrhea, alternations between diarrhea and constipation, local abdominal pain and other symptoms become apparent.

My Genetic Risk Is Average For The Population

1.13 times

Average incidence rate is 24 per 100,000 in the population

Suggestions

- Red flags: In the physical examination report, serum Glycosyl Antigen CA199 is too high, irregular bowel movements, changes in stool characteristics (thinning/bloody/mucinous), abdominal pain, intestinal obstruction
- High-risk population: high-fat diet, smoking, drinking, obesity, middle-aged and elderly people, family history of colorectal cancer
- How to reduce the risk:
 - Pay attention to your diet: eat more fruits and vegetables, control fat intake, eat less processed meats, and drink less alcohol.
 - Quit smoking: refuse to take the initiative to smoke, stay away from second-hand smoke.
 - Weight control: Keep BMI between 18.5 and 23.9, avoid sitting for a long time, and exercise at least 4 hours a week.
 - Colonoscopy was performed every 10 years after the age of 50.
- The Heritability of colorectal cancer is about 7%~35%

* If you have a family history of colorectal cancer, or if you think you are likely to develop colorectal cancer, or need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors



Controllable factors

1. Poor diet: High-fat diet, excessive intake of animal protein, especially red meat, low cellulose diet, are the main risk factor for colorectal cancer.

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2. Lifestyle factors: Poor habits, including long-term smoking, drinking alcohol, inactivity, etc., can increase the risk of colorectal cancer.
 3. Overweight and obesity: In recent years, overweight and obesity have been identified as risk factors for colorectal cancer.

Uncontrollable factors

1. Age: Middle-aged and elderly people are more likely to develop colorectal cancer.
2. Disease history: Studies have shown that the history of chronic diarrhea, mucosanguineous feces, colorectal polyps, constipation and appendicitis are the risk factors of colorectal cancer.
3. Family history: People with a family history of colorectal cancer have a higher prevalence.

Knowledge

Time of onset

Colorectal cancer mostly occurs between the ages of 50 and 70.

Prevalence

The incidence and mortality rate of colorectal cancer in China are higher than the world average and the level of developing countries, and lower than that of developed countries. The incidence of colorectal cancer in China is 28.08 per 100,000, accounting for 10.56% of the total incidence of malignant tumors.

Typical symptoms

Early colorectal cancer may have no obvious symptoms, the disease developed to a certain extent before the following symptoms: defecation habits change, stool traits change (thinning, blood stool, mucus stool, etc.), abdominal pain or abdominal discomfort, abdominal lumps, intestinal obstruction, anemia and systemic symptoms: such as wasting, fatigue, low fever. Its incidence and fatality rate are second only to stomach cancer, esophageal cancer and primary liver cancer in digestive malignant tumors.

Treatment

1. Surgical treatment: At present, the treatment of colorectal cancer, clinically generally take the principle of comprehensive treatment based on surgery. Treatment options are determined according to the patient's general condition and the function of each organ, the location of the tumor, clinical staging, pathological type and biological behavior. The basic principle of colorectal cancer surgery is consistent with the basic principle of tumor surgery. In summary, they are the three principles of radical, safety and function. Among them, when the tumor can be removed, the principle of radical treatment is first required, and the principle of radical treatment is considered

second. When it comes to safety, functional principles are considered as much as possible at the end.

2. Chemotherapy: Adjuvant chemotherapy is generally required after colon cancer. Patients with colorectal cancer may have tiny metastases or tumor cells after surgery, which can be killed as much as possible by adjuvant chemotherapy. However, not all patients need and are suitable for chemotherapy. For example, early stage (stage I) patients do not need chemotherapy. In addition, for patients diagnosed with large tumors or local lymph node metastasis, in order to improve the rate of surgical cure and reduce the scope of removal, doctors usually recommend patients should be treated with radiotherapy and chemotherapy first, which can shrink the tumor or reduce the stage, which is conducive to the operation.
3. Radiotherapy: Generally used as an ancillary therapy for surgery, treatment before or after surgery.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2306536	CHFR	CT	0.53	0.98
RS1049174	intergenic	CG	1.60	0.97
RS719725	intergenic	AA	1.42	0.99
RS1078643	intergenic	AA	1.28	1.00
RS12953717	SMAD7	CT	1.25	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to colorectal cancer. These literatures have found some polymorphic loci related to the risk of the disease by studying colorectal cancer. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of colorectal cancer item uses the 30 polymorphic loci of *SMAD7*, *RHPN2*, *APC*, *CHFR*, *ADIPOQ* genes. The test results indicate whether the user has a higher prevalence of colorectal cancer in the combination of genotypes at the 30 polymorphic loci tested compared to all users. It does not mean the user's true risk of colorectal cancer. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that can lead to the risk of colorectal cancer.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only predicts your risk of colorectal cancer from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of colorectal cancer, or you think that you are likely to suffer from colorectal cancer, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 30 polymorphic loci related to colorectal cancer risk, such as rs10411210, rs1295371, rs4464148 and so on..

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to colorectal cancer.

Osteoporosis

Due to the decrease of bone mineral density and the destruction of bone microstructure, bone fragility increases, which is prone to systemic bone disease of fracture.

My Genetic Risk Is Average For The Population

1.12 times

Average incidence rate is 15700 per 100,000 in the population

Suggestions

- Red flags: in physical examination report, **Bone mineral density** declines
- Common symptoms: back pain, spinal deformation, easy to fracture
- High-risk population: middle-aged and elderly, women have a high risk of osteoporosis
- High-risk lifestyles that can lead to osteoporosis include: Insufficient sun exposure, **Vitamin D** and insufficient calcium intake; smoking, drinking alcohol, drinking too much Coffee, and excessive sugar intake can lead to calcium loss; lack of exercise , etc.
- How to reduce risk: :
 - Get more sunlight to ensure the synthesis of vitamin D, and promote the absorption of calcium
 - Exercise more and increase the weight-bearing of bones appropriately to help improve bone density
 - Increase calcium and vitamin D intake; eat more Calcium-rich foods such as dairy, beans, etc. or use dietary supplements
 - Avoid smoking, alcohol, coffee and excessive sugar intake
- Clinical treatment of osteoporosis mainly uses anti-resorption drugs, bone formation promoting drugs or other drugs
- The Heritability of bone density is about 50% to 85%

* If you have a family history of osteoporosis, or have a decreased bone density, or if you feel you are at risk of osteoporosis, or need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for assistance.

Influencing factors



Controllable factors

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1. Insufficient calcium supplementation: people who lack vitamin D or normally consume less calcium, such as those who are vegetarian or not drinking milk, are more likely to develop osteoporosis.
 2. Lack of outdoor exercise: leads to increased load of bones and joints or reduced sun exposure.
 3. Unhealthy lifestyle: people who smoke, drink alcohol or excessively drink coffee are more likely to develop osteoporosis.

Uncontrollable factors

1. Race: Caucasians and Asians have a higher incidence of osteoporosis than Africans.
2. Gender: women have a higher risk of osteoporosis than men.
3. Age: reduced osteoblast activity in women over the age of 70 or men over the age of 80, resulting in reduced synthesis of new bone mass.
4. Family history: descendants of female patients in the family are more likely to develop the disease.

Knowledge

Onset time

Postmenopausal osteoporosis usually occurs within 5 to 10 years after menopause in women, senile osteoporosis generally refers to osteoporosis that occurs in the elderly aged over 70, and idiopathic osteoporosis mainly occurs in adolescence.

Prevalence

The total prevalence of osteoporosis among people over 50 years old in China is 15.7%, and osteoporosis will worsen with age, and is more common in female than in male patients.

Typical symptoms

1. Back pain: low back pain is more common, and the aggravated pain or limited mobility may occur when the load increases. In severe cases, there are difficulties in turning, sitting and walking.
2. Spinal deformation: severe osteoporosis can lead to height shortening and hunchbacks. Vertebral compression fracture can lead to thoracic deformation, abdominal compression, and impairment of cardiopulmonary function, etc.
3. Fragile fractures: fractures that occur from non-trauma or minor trauma are fragility fracture. Bone parts that are prone to fracture due to osteoporosis include the spine, forearm bone, and hip bone.

Treatment

1. There are three main types of drugs for clinical treatment of osteoporosis:

- Anti-resorptive drugs
- Drugs to promote bone formation
- Other drugs

2. Patients with typical osteoporosis such as low back pain should choose drugs like calcitonin, diphosphate to reduce pain and relieve symptoms, and should adopt a healthy lifestyle and increase intake of calcium and vitamin D. It's difficult to activate vitamin D of the elderly because of their liver and kidney function is weak, so it may also make the intake of calcium difficult to be really absorbed by the body. Therefore, patients should take osteolytic triol and other active vitamin D to promote calcium absorption, and increase muscle strength to prevent falls, prevent osteoporosis and bone pine fractures.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS3770748	QPCT	AG	1.45	0.98
RS2273061	JAG1	AG	0.70	0.98
RS6696981	intergenic	GG	1.32	1.00
RS34240317	intergenic	DD	0.76	1.00
RS7524102	intergenic	AA	1.25	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to Osteoporosis. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between the patients with Osteoporosis and normal subjects. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Osteoporosis risk item uses 53 polymorphic loci on *RSPO3*, *ESR1*, *LRP5*, *FUBP3*, *QPCT*. The test result indicates whether the user has a higher possibility of Osteoporosis in genotype combination of the 53 polymorphic loci detected compared with all users, and the test does not represent the user's true risk of Osteoporosis. The test is mainly based on the research of Caucasian and Chinese, other populations are for reference only.

Notification

1. The test may not cover all disease-causing genes or sites of Osteoporosis.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable to clinical diagnosis.
2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Osteoporosis from the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If you have a family history of Osteoporosis, or you think that you are likely to suffer from Osteoporosis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 53 polymorphic loci associated with osteoporosis risk, such as rs10457487, rs11003047 and rs3736228.

Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with Osteoporosis.

Cataract

Cataract is a kind of crystal turbidity that causes vision loss and even blindness.

My Genetic Risk Is Average For The Population

1.12 times

Average incidence rate is 59950 per 100,000 in the population

Suggestions

- How to reduce risk:
 - Do Eye protection well in bright light, avoid using excessively strong indoor light sources, avoid looking directly at strong light sources, pay attention to eye hygiene
 - Protective food: Proper supplementation of vitamin E, vitamin C, vitamin A and β -carotene is helpful for eye health
 - Healthy living: Maintain at least 4 hours of exercise a week and take care to avoid excessive sugar intake
 - Avoid environments containing fluorine and other harmful substances to reduce the impact of toxins on the eyes

* If you have a family history of cataracts, or if you feel you are at risk of developing cataracts, or if you need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for assistance.

Influencing factors



Controllable factors

1. Lighting conditions: People who are exposed to high-intensity light for a long time are more likely to develop cataracts.
2. Nutritional elements: People who lack essential nutrients such as vitamin E, vitamin C, vitamin A, and β -carotene are more likely to develop cataracts.
3. Lifestyle: Excessive intake of sugar will change the osmotic pressure of the eye and increase the risk of cataracts; lack of exercise will also increase the risk.
4. Toxic substances: People who are exposed to fluorine compounds for a long time are more likely to develop cataracts.

Uncontrollable factors

1. Age: It is common in middle-aged and old age, with increasing age, the risk of cataracts increases.
2. Gender: The incidence of cataracts among women in the population is slightly higher than that of men.
3. Diseases: Eye infections, trauma, high myopia, high blood pressure, diabetes, etc. may increase the risk of cataracts.
4. Family history: If there is cataract in the family, especially the incidence of cataract in individuals with early-onset cataract is higher than that of the general population.

Knowledge

Time of onset

It usually develops after middle-aged and old age. With age, the incidence rate gradually increases.

Prevalence

According to a study on cataracts in Beijing, 59.95% of people will suffer from cataracts of varying degrees before the age of 80, compared with 83.02% after the age of 80. Studies elsewhere in the country have shown similar incidences.

Typical symptoms

- In the early stage, spot-like or flake-like shadows with fixed positions and unchanged shapes can appear in front of the eyes.
- Loss of vision and blurred vision, as well as worsening vision loss.
- Eye fatigue is easy, and objects in the field of view are deformed or distorted.
- What you see may be glare or double-shadow, especially in the daytime.
- There may be day blindness or night blindness.

Treatment

When it has little effect on eyesight in the early stage, you can add necessary nutrients to slow down the occurrence of cataracts, such as vitamin E, vitamin C, vitamin A and β -carotene. Some drugs can be used in the early and mid-term to delay the progression of the disease. Usually, for some mid-term cataract patients, vision and lens opacity can also be improved to some extent after medication. Cataracts in mature stage can only be treated by surgery, and should be treated as early as possible due to the principle of surgery.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS7548209	intergenic	CG	1.49	1.00
RS79721202	intergenic	TT	0.71	1.00
RS1136410	PARP1	GG	1.42	1.00
RS1800407	OCA2	CC	0.81	1.00
RS7543472	intergenic	TT	1.23	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to cataracts. These literatures have found some polymorphic loci related to the risk of the disease by studying the genetic differences between patients with cataract and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The cataract health risk item uses the 49 polymorphic loci on genes such as *GSTP1*, *CASC15*, *KCNAB1* and other genes. The test results indicate that compared with all users, whether this user has a higher prevalence of cataracts in the combination of genotypes at the 49 polymorphic loci tested. It does not mean the user's true cataract risk. The basis of the test is mainly from the research of Caucasian, Asian, Latino, and African-American, other populations are for reference only.

Notification

-
1. The test may not cover all genes or loci that cause cataract.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of cataracts at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of cataracts, or you think that you are likely to suffer from cataracts, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 49 cataract-related polymorphic loci, including rs7366355, rs3176459, and rs7543472.

Population samples

The basis of the test is mainly from the research of Caucasian, Asian, Latino, and African American.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that cause cataract.

Meningioma

Meningioma is one of the most common intracranial tumors. It is benign tumor with slow growth and a long course of disease. Since the tumor swells as it grows, patients often experience headaches and seizures as their first symptoms.

My Genetic Risk Is Average For The Population

1.11 times

Average incidence rate is 6 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Age: meningiomas occur mostly around the age of 45.
 2. Gender: the prevalence of meningioma in women is twice that of men.
 3. Trauma and treatment factors: cranial brain trauma, radiation therapy, hormone replacement therapy, etc., may lead to an increased risk of meningioma.
 4. Family history: people with a family history of meningioma have a higher prevalence.
-

Knowledge



Time of onset

This disease can occur at any age, peaking at around 45 years of age, with more women than men, about 2:1.



Prevalence

Meningioma is a common primary intracranial tumor. The incidence of intracranial tumors is second only to gliomas, the incidence rate is about 6 per 100,000 people.



Typical symptoms

Meningiomas fall into four categories: tumors of meningothelial cells, mesenchymal non-meningothelial tumors, primary melanocytic lesions, and tumors of uncertain histogenesis. Meningiomas are mostly benign tumors with slow growth and long course of disease. Because the tumor is expanding, patients often have headaches and epilepsy as the first symptoms. Depending on the location of the tumor. Vision, vision, olfactory or auditory impairments, and limb movement disorders can also occur. Elderly patients mostly take epilepsy as the first symptom, cranial pressure increase symptoms are not obvious. In the context of the increasing popularity of CT tests, many patients have only minor headaches and have even been stumbled upon as meningiomas by CT scans. Because the tumor grows slowly, the tumor tends to grow very large, and the clinical symptoms are not serious. Meningiomas near the skull often cause bone changes.

Treatment

1. Surgery: Surgery is the preferred treatment for meningioma. Stereotactic radiotherapy (including γ , X, and proton knife) can be used for tumor with diameters ≤ 3 cm, tumor residues or recurrences after surgery, intracranial and sponge sinus tumors.
2. Embolism therapy: embolism therapy is only an auxiliary treatment for surgery, the main purpose is to reduce the blood supply of meningioma, but only to the extra-cervical artery blood supply-based meningioma.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS4968451	BRIP1	AC	1.61	0.98
RS1801394	MTRR	AG	1.08	1.00
RS12770228	CASC10	GG	1.00	1.00
RS11012732	MLLT10	AA	1.00	1.00

Risk calculation

The risk value of the test is based on published academic literature on meningioma. By studying the genotype differences between meningioma patients and normal people, these literatures found some polymorphic sites associated with the risk of meningioma. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By

comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The meningioma health risk item uses four polymorphic loci on the *MLLT10*, *CASC10*, *MTRR*, and *BRIP1*. The results showed whether the user had a greater risk of meningioma than all users, but did not represent the risk of meningioma in all dimensions compared to all users. The basis of the test is mainly from the research of Caucasian, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of meningioma.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. this study only predicts your risk of meningioma at a genetic level and does not represent your true health status, nor does it involve your judgment of other health conditions, lifestyles, etc.
3. If you have a family history of meningioma, or if you feel you have a risk of developing meningioma, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes four polymorphic loci associated with meningioma risk, including rs11012732, rs12770228, and rs1801394.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with meningioma.

Crohn's Disease (CD)

It is a chronic, recurrent inflammatory bowel disease that damages the digestive tract.

My Genetic Risk Is Average For The Population

1.10 times

Average incidence rate is 1 per 100,000 in the population

Suggestions

1. Smoking is an important risk factor for Crohn's disease, and it is recommended to quit smoking and avoid secondhand smoke.
2. Bacterial infections may increase the risk of Crohn's disease, so avoiding unclean foods and washing your hands before meals.

Influencing factors



Controllable factors

1. Smoking: Smoking is a risk factor for Crohn's disease, and smokers are 2-5 times more likely to develop Crohn's disease than non-smokers.
2. Infections: Some microorganisms may increase the risk of Crohn's disease, such as *Mycobacterium paratuberculosis*, *Listeria monocytogenes*, *Chlamydia trachomatis*, *Escherichia coli*, *Cytomegalovirus*, etc.



Uncontrollable factors

Family history: Crohn's disease can be clustered in families, especially when parents or siblings suffer from this disease, which increases the risk of Crohn's disease.

Knowledge

Time of onset

The disease can occur at any age, with a higher incidence in young and middle-aged adults.

Prevalence

It is estimated that the incidence of this disease is 3 to 20 cases per 100000. It is more common in the industrialized world, particularly in North America and Western Europe, though the incidence is rising in Asia and South America.

Typical symptoms

The course of the disease is chronic, and most patients have recurrent attacks and prolonged non-healing.

1. Digestive system symptoms: Abdominal pain, diarrhea, abdominal mass, fistula formation and intestinal obstruction.
2. Systemic manifestations: Fever, nutritional disorders, water, electrolyte, and acid-base balance disturbances during acute attacks.
3. Extraintestinal manifestations: Joints, skin, eyes, oral mucosa and other diseases.

Treatment

At present, there is no cure. Treatment (e.g., drugs and surgery) aims to alleviate the condition and prevent recurrence.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1800972	DEFB1	GG	0.30	0.85
RS13361189	intergenic	CC	1.90	0.97
RS4958847	IRGM	AA	1.85	0.98
RS10761659	intergenic	GG	1.78	0.99
RS6908425	CDKAL1	CT	1.63	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to Crohn's disease. By studying the genetic differences between Crohn's disease patients and normal people, these literatures have identified some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Crohn's disease health risk item uses 33 polymorphic loci on genes such as *IL23R*, *NOD2*, *NELL1*, *ATG16L1* and *PTPN2* and within other intergenic regions. The results showed whether the user had a higher prevalence of Crohn's disease than all users in the genotype combination of the 33 polymorphic loci tested, and that the test did not represent the user's real risk of Crohn's disease. The basis of the test is mainly from the research of Caucasian, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of Crohn's disease.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Crohn's disease at a genetic level and does not represent your true health status, nor does it involve your judgment of other health conditions, lifestyles, etc.

3. If you have a family history of Crohn's disease, or if you feel you have a risk of Crohn's disease, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 33 polymorphic loci related to the risk of Crohn's disease, including rs1004819, rs2201841, rs1793004, rs11209026 and rs1800972.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with Crohn's disease.

Allergic Diseases

Allergic diseases mainly include asthma, hay fever (allergic rhinitis), eczema (atopic dermatitis) and other symptoms.

My Genetic Risk Is Average For The Population

1.09 times

Average incidence rate is 22000 per 100,000 in the population

Suggestions

1. Avoid topical skin irritation, such as the use of irritating drug, etc.
2. Be careful with food that may trigger allergic diseases, such as seafood, eggs, milk, etc.
3. Keep indoor air circulating, and avoid house dust, pollen, dust mites, etc.; quit smoking and try to stay away from second-hand smoke. During the flu season, try to minimize the visits to crowded places and adjust clothing according to the change of seasons in order to reduce respiratory infections. Try to maintain the indoor hygiene and wash the beddings at least once every month; try to close the windows during the flowering season and stay away from trees that easily raise flowers, so as to avoid potential allergens. If living in a seriously polluted city, it is best to wear a mask during outdoor activities, and moving to a place with good air quality is recommended if possible.
4. Take care of the rest and avoid mental tension and emotional excitement.
5. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and efficacy of treatment methods vary from person to person and should always be undertaken under the advice and supervision of a medical professional, avoiding self-experimentation.

Influencing factors



Controllable factors

1. Skin irritation may induce atopic dermatitis.
2. Food: Foods that often cause allergies include eggs, milk, seafood, and to a lesser extent, fruit juices and nuts.
3. Inhalants: Pollen, animal hair, fungi, house dust, dust mites and other inhalants may induce atopic dermatitis. Air pollution (SO₂, NO) can cause bronchoconstriction, increased transient airway responsiveness as well as the increased permeability of the nasal mucosa, enhancing the response to allergens. In addition, cigarette smoke (including passive smoking) is an important asthma promoter, and induces asthma attack more often in children with asthma whose parents smoke.
4. Exertion, mental stress, and emotional stress increase the risk of allergic disease.



Uncontrollable factors

1. Family history: A family history of allergies can increase the risk of allergic disease.
2. Age: Among allergic diseases, the incidence of asthma is higher in children than in young adults, with children aged 1-6 years being the most prevalent; allergic rhinitis is predominant in young adults, with those aged 15-40 years being more common; atopic dermatitis is overwhelmingly initialized in infancy and early childhood, and some can occur in childhood and adulthood.
3. Diseases: Asthma, pollinosis (allergic rhinitis) and eczema (atopic dermatitis) are significantly correlated.

Knowledge



Time of onset

Allergic diseases can occur at all ages from newborns to the elderly.



Morbidity

World Allergy Organization (WAO) statistics show that the global prevalence of allergic diseases has reached 22%, and the prevalence in China has exceeded 20%.



Typical symptoms

1) Asthma

It is manifested by cough, skin rash and slight fever. After the allergic substance is agitated, mucus accumulates in the fine bronchial tubes and the tracheal tube diameter is contracted, resulting in difficulty in breathing, often at night.

2) Pollinosis (allergic rhinitis)

It manifests as runny nose, sneezing, nasal congestion, itching around the eyes, and itching of the palate, and worsens when the weather becomes cold.

3) Eczema (atopic dermatitis)

Prevalent on the knees, elbows, extremities, and the whole body, etc. It often manifests as symmetrical episodes of itchy skin with red patches, eczema, and urticaria, often recurring.

Treatment

Keep away from allergens, symptomatic treatment with medication (to relieve allergy symptoms), and specific immunotherapy (desensitization treatment). Among them, staying away from allergens requires patients to take better care of themselves, such as staying away from pollen and using some anti-mite tools. As for the various symptoms of allergic diseases, the symptoms are immediately relieved with anti-allergy medications, but desensitization is needed to achieve a real cure.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS10865050	IL1RL1	GG	1.28	1.00
RS10414065	intergenic	CC	1.21	1.00
RS28895016	intergenic	CC	1.20	1.00
RS12413578	intergenic	CC	1.20	1.00
RS11255968	intergenic	CC	1.19	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to allergic diseases (asthma, hay fever, eczema). These literatures have found some polymorphic loci related to the risk of the disease by studying allergic diseases (asthma, hay fever, eczema). We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our

products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The allergic disease health risk item uses 119 polymorphic loci on the genes of *FLG*, *IL3*, and *STAT6*, etc.,. The test results indicate/only indicate whether the user has a higher preference of developing allergic disease compared to all users based on the combination of genotypes at the 119 polymorphic loci tested. It does not mean the user's true risk of acquiring allergic disease. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of developing allergic disease.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The development of disease are determined by multiple factors such as genes and environment. This study only predicts your risk of developing allergic diseases at the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of allergic diseases, or you think that you are likely to suffer from allergic diseases, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 119 polymorphic loci associated with the risk of allergic diseases (including asthma, pollinosis, eczema) including rs10910095, rs301806, rs760805, rs76167968.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect allergic diseases.

Ulcerative Colitis

It is an unexplained inflammatory bowel disease. The lesions are located in the large intestine and are distributed continuously and diffusely. The lesions are mainly limited to the large intestinal mucosa and submucosa.

My Genetic Risk Is Average For The Population

1.08 times

Average incidence rate is 12 per 100,000 in the population

Suggestions

1. Maintain a relaxed and happy mood and avoid excessive mental stress or excessive psychological pressure.
2. If infected with Escherichia coli, rotavirus, cytomegalovirus, etc., active treatment is required to avoid deterioration.
3. Pay attention to light diet, quit smoking and alcohol.

Influencing factors

Controllable factors

1. Mental factors: studies have shown that negative emotions such as depression and anxiety can lead to an increased risk of illness.
2. Infection factors: it is not yet possible to determine the direct relationship between bacteria, viral infections and disease, but E.coli, Rotavirus, Cytocell viral infections seem to increase the risk of disease.
3. Appendectomy: studies have shown that people who have an appendectomy before the age of 20 have a significantly reduced risk of ulcerative colitis.
4. living habits: drinking alcohol is prone to recurrence of festering, but smoking seems to reduce the occurrence of festering. However, taking into account the overall health considerations, it is recommended to quit smoking and alcohol.



Uncontrollable factors

1. Genetic factors: there is a family genetic tendency.
2. Immune factors: the study found that the incidence of ulcerative colitis is closely related to the abnormality of the body's immune function, often coexists with thyroid gland, diabetes mellitus and other diseases, body fluid immunity plays an important role in the onset of ulcerative colitis, and cell immunity is second.

Knowledge



Time of onset

The disease can occur at any age, and it is more common in people between 20 and 30 years old.



Prevalence

According to statistics, there are about 12 cases of the disease per 100,000 people in the average population.



Typical symptoms

The clinical manifestations are mainly diarrhea, mucus pus and blood in the stool and abdominal pain. Among them, mucus pus and blood in the stool is an important manifestation of ulcerative colitis. Patients with mild symptoms can defecate 2-4 times a day, and severe cases can reach 10 times a day.



Treatment

At present, there is no specific medicine or method that can completely cure ulcerative colitis, but standard treatment can be used to reduce recurrence, prolong the course of remission, and improve the quality of life.

The main treatment methods include medication and surgical treatment. Drug therapy mainly includes aminosalicylic acids, corticosteroids, immunosuppressants, antibiotics, probiotics, and anti-TNF preparations. Among them, aminosalicylic acids are often used as the first choice and maintenance therapy drugs. Corticosteroids are usually said to be hormones. For rapid relief of symptoms, it is mostly used for acute attacks, while immunosuppressive agents can reduce or eliminate dependence on hormones, and can be used to maintain and relieve ulcers when aminosalicylic acid maintenance therapy fails. When medical treatment is ineffective and there are indications for surgery, appropriate surgical treatment can be used to remove the diseased intestinal segment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS9271366	intergenic	AG	2.10	0.92
RS9263739	CCHCR1	CC	1.00	0.89
RS117506082	intergenic	AA	1.00	0.95
RS1801274	FCGR2A	AA	1.00	0.96
RS4654903	intergenic	AA	1.00	0.96

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to ulcerative colitis. By studying the genetic differences between ulcerative colitis patients and normal people, these literatures have identified some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The ulcerative colitis health risk item uses 11 polymorphic loci on the *BSN*, *IL23R*, and *FCGR2A* genes. The results showed whether the user had a higher prevalence of ulcerative colitis than all users in the genotype combination of the 11 polymorphic loci tested, and that the test did not represent the user's real risk of ulcerative colitis. The basis of the test is mainly from the research of Caucasian, Japanese, and Korean, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of ulcerative colitis.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of ulcerative colitis at the genetic level and does not represent your true health status, nor does it involve your judgment of other health conditions, lifestyles, etc.
3. If you have a family history of ulcerative colitis, or if you feel you have a risk of developing ulcerative colitis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 11 polymorphic loci related to the risk of ulcerative colitis, including rs10883365, rs11209026, rs2395185, and rs9858542.

Population samples

The basis of the test is mainly from the research of Caucasian, Japanese, and Korean.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with ulcerative colitis.

Restless Leg Syndrome (RLS)

Restless leg syndrome is a common neurological disease, characterized by a strong desire to move affected limbs (mostly legs).

My Genetic Risk Is Average For The Population

1.07 times

Average incidence rate is 5000 per 100,000 in the population

Suggestions

Iron deficiency may cause restless legs syndrome, so iron intake should be maintained. Diet is the best source of iron, such as oysters and lean red meat is rich in hemoglobin iron, eggs, dairy products, spinach, and beans contain non-hemoglobin iron.

Influencing factors



Controllable factors

Iron deficiency: The study found that iron deficiency anemia is closely related to restless leg syndrome, and reducing iron storage in the body may also aggravate the symptoms.



Uncontrollable factors

1. Pregnancy: Symptoms usually appear in the third trimester of pregnancy, but usually disappear within 4 weeks of postpartum period.
 2. Neurological injury: Restless legs syndrome is associated with spinal cord and peripheral nerve damage.
 3. Family history: familial restless legs syndrome has a high incidence, but the exact genetic pattern is still unclear.
 4. Uremia: Up to 50% of patients with end-stage renal failure are found to have restless legs syndrome, and their symptoms improve after renal transplantation.
-

Knowledge

Time of onset

There are mainly the following two types. One is early onset (before 45 years of age) that worsens over time. The other is onset after the age of 45, which occurring suddenly but will not persistently with deterioration.

Prevalence

The prevalence of this disease is 0.1%~11.5%, which is frequently seen in Western population. The incidence of Asians is rare. There is no relevant epidemiological information in China.

Typical symptoms

Sensory abnormalities of double lower limbs occasionally involve the upper limbs. Sensory abnormalities include aching and numbness, ant walking, tingling, and even indescribable discomfort. This discomfort is reduced after limb activity, aggravated at rest, alleviated during the day and aggravated at night.

Treatment

Non-drug treatment should be used for mild and moderate patients, while severe patients should be treated with medication, and if secondary sleep foot disease patients, the primary disease needs to be actively treated.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS3923809	BTBD9	AG	1.58	0.96
RS1975197	PTPRD	AG	1.29	0.99
RS1820989	intergenic	CC	0.77	1.00
RS61192259	intergenic	AC	1.26	1.00
RS45544231	intergenic	CG	0.83	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to restless leg syndrome. These literatures have found some polymorphic loci related to the risk of the disease by studying restless leg syndrome. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of restless leg syndrome item uses the 21 polymorphic loci of *BTBD9*, *PTPRD*, and *CCDC148* gene. The test results show whether the user has a higher prevalence of restless leg syndrome than all users. The test cannot represent the user's real risk of restless leg syndrome. The test is mainly based on the research of Caucasian, and other populations are only for reference.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of restless leg syndrome.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not applicable to clinical practice.
 2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of restless legs syndrome at the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
 3. If you have a family history of restless legs syndrome, or you think that you are likely to suffer from restless legs syndrome, it is recommended that you consult an authoritative health professional or clinician for help.
-

Test content

The test includes 21 polymorphic loci associated with the risk of restless legs syndrome, including rs3923809, rs1975197, and rs80319144.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci related to restless legs syndrome.

Endometrial Cancer

Endometrial cancer is a common malignant tumor of the female reproductive tract. The most common initial symptom is non-menstrual vaginal bleeding.

My Genetic Risk Is Average For The Population

1.04 times

Average incidence rate is 26 per 100,000 in the population

Suggestions

1. Control your diet, work out your body: avoid eating a lot of high-fat and high-sugar food for a long time; Instead of sitting for long periods of time, get into the habit of regular exercise, such as at least 150 minutes of moderate-intensity exercise (such as brisk walking and dancing) or at least 75 minutes of high-intensity activity (such as running and swimming) per week for adults.
2. Age appropriate marriage and childbearing, the best childbearing age is 25-35 years old.

Influencing factors



Controllable factors

1. Obesity, diabetes and hypertension: this disease is more common in obese, diabetic or abnormal glucose tolerance and hypertension women.
2. Unmarried, infertile and childless: this disease occurs in unmarried, infertile and childless people, which may be related to the endometrium receiving estrogen stimulation for a long time.



Uncontrollable factors

1. Age: Common after menopause.
2. Past medical history: This disease is mostly seen in delayed menopause, functional uterine bleeding, polycystic ovary, ovarian gonadal stromal tumors such as granulosa cell tumor, and women who take estrogen for a long time after menopause.

3. Family history: Having a family member with Lynch syndrome (a common form of inherited colorectal cancer, caused by mutations in the MMR gene that can also lead to endometrial cancer) may increase the risk of the disease.

Knowledge

Onset time

It is common in women between the ages of 50 and 65, with relatively few patients under 40.

Prevalence

The number of new cases of Endometrial Cancer is about 26/100,000 women per year. The number of deaths from endometrial cancer per 100,000 women is about 4.6 per year.

Typical symptoms

1. Anovulatory vaginal bleeding occurs in postmenopausal women.
2. Abnormal uterine bleeding, menstrual disorders.
3. Vaginal bleeding in menopausal women over 40 years of age that is very long, heavy, or frequent may indicate precancerous changes.
4. Mild abdominal pain or pelvic cramps.

Treatment

1. Surgery: Surgical interventions should include cytological sampling of peritoneal fluid, exploration of the abdominal cavity, palpation and biopsy of suspicious lymphatic vessels, total abdominal hysterectomy, and bilateral salpingo-oophorectomy.
2. Radiotherapy: For stage I and II patients, the hospital may use surgery combined with radiotherapy to reduce the risk of tumor recurrence.
3. Chemotherapy: For stage III and IV patients, physicians may consider chemotherapy, usually chemotherapy alone or in combination with adriamycin, carboplatin, cisplatin, 5-fluorouracil, adriamycin, etc.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS148261157	intergenic	AG	1.26	1.00
RS35286446	intergenic	DD	1.21	1.00
RS882380	intergenic	AA	1.21	1.00
RS2747716	intergenic	AA	1.21	1.00
RS3184504	SH2B3	CC	1.21	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to Endometrial Cancer. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between the pregnant woman with Endometrial Cancer and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The endometrial Cancer risk item uses 11 polymorphic loci on *SH2B3*, *EIF2AK4*, and between these genes. The test result indicates whether the user has a higher possibility of endometrial Cancer in genotype combination of the 11 polymorphic loci detected compared with all users, and the test does not represent the user's real risk of endometrial Cancer. The test is mainly based on the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all disease-causing genes or sites of Endometrial Cancer.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Endometrial Cancer from the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If you have a family history of Endometrial Cancer, or you think that you are likely to suffer from Endometrial Cancer, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 11 polymorphic loci associated with Endometrial Cancer risk, such as rs148261157, rs1679014.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with Endometrial Cancer.

Liver Cancer

Liver cancer refers to malignant tumors that occur in or start from the liver.

My Genetic Risk Is Average For The Population

1.04 times

Average incidence rate is 25 per 100,000 in the population

Suggestions

- **Danger signal:** in the physical examination report, serum tumor markers such as alpha fetoprotein (AFP) and Golgi protein 73 (GP73) are too high
- **High-risk population:** hepatitis B/C virus infection, parasitic infection, alcohol consumption, obesity, middle-aged and elderly people, patients with liver cirrhosis, and family history of liver cancer.
- **How to reduce risk:**
 - Hepatitis B vaccination
 - Avoid fresh water sashimi to prevent parasitic infection
 - Avoid moldy and deteriorated food and control Alcohol consumption
 - **Weight control:** keep BMI between 18.5 and 23.9, avoid sitting for long periods of time, and exercise at least 4 hours a week

* If you have a family history of liver cancer, or if you think you are at risk of developing liver cancer, or if you need to use dietary supplements, you are advised to consult a leading health professional or clinician for help.

Influencing factors



Controllable factors

1. **Virus infection:** hepatitis B virus (HBV) and hepatitis C virus (HCV) infection can greatly increase the risk of liver cancer.
2. **Parasitic infection:** the infection of parasites such as Schistosoma and liver fluke can induce the occurrence of liver cancer.
3. **Dietary factors:** alcohol, aflatoxin, nitrosamines and other substances in food can increase the risk of liver cancer.

4. Obesity: obesity can lead to nonalcoholic fatty liver, and then induce liver cancer.

Uncontrollable factors

1. Age: The older you are, the more likely you are to develop liver cancer.
2. Gender: Men have a higher risk of liver cancer than women.
3. Family history: People with a family history of liver cancer are more likely to develop liver cancer.
4. Cirrhosis: Cirrhosis may progress to liver cancer.

Knowledge

Time of onset

Liver cancer occurs most frequently after the age of 40, with 50 to 60 years old being the most common age group.

Prevalence

According to the report of the World Health Organization, the incidence of liver cancer in China is 25.7/100,000, the highest in the world, and the new cases and deaths of patients account for about half of the global total every year.

Typical symptoms

Early stage liver cancer is often asymptomatic. Liver pain, fatigue, weight loss, progressive hepatomegaly or epigastric mass, jaundice, ascites and so on are common in middle and late stage liver cancer; Some patients have low fever, jaundice, diarrhea and upper gastrointestinal bleeding; Acute abdomen after rupture of liver cancer. There are also symptoms that are not obvious or only metastases.

Treatment

According to the stage of liver cancer and liver function, a reasonable treatment scheme is comprehensively considered: the options include surgery, chemotherapy, radiotherapy, targeted therapy, radiofrequency ablation, interventional embolization, liver transplantation, etc.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS4678680	intergenic	TT	5.15	0.97
RS9267673	C2	CC	3.88	0.96
RS17401966	KIF1B	AA	2.69	0.95
RS9275319	intergenic	AA	2.22	0.98
RS12100561	EFCAB11	AG	1.52	0.96

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to liver cancer. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between subjects with liver cancer and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The liver cancer item uses 8 polymorphic loci in and between *KIF1B*, *STAT4* and *C2* genes. The test results indicate whether the user has a higher risk of liver cancer for the genotype combination of 8 polymorphic loci detected compared to all users. The test cannot represent the user's real risk of liver cancer. The basis of the test is mainly from the research of East Asian population, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that contribute to the risk of liver cancer.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are jointly determined by multiple factors such as genes and environment. This study only predicts your risk of liver cancer at the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If there is a family history of liver cancer in your family, or if you think you are at risk of developing liver cancer, you are recommended to consult an authoritative health professional or clinician for help.

Test content

Eight polymorphic loci associated with the risk of liver cancer were detected, including rs7574865, rs9275319 and rs17401966.

Population samples

The basis of the test is mainly from the research of East Asian population.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect liver cancer.

Chronic Lymphocytic Leukemia (CLL)

Chronic lymphoblastic leukemia is a neoplastic disease of B-lymphocyte clonal proliferation. It is characterized by an accumulation of lymphocytes in the body, so that the number of lymphocytes in the blood and bone marrow increase, causing enlargement of the lymph nodes, liver and spleen.

My Genetic Risk Is Average For The Population

1.03 times

Average incidence rate is 1 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Age: The risk increases with age.
2. Gender: men are more likely than women to have chronic lymphoblastic leukemia, with a ratio of about 1.2:1-1.7:1.
3. Ethnic groups: the incidence of the disease is higher among Jews, and the incidence of the disease is higher among the overall European and American populations than among the Asian population.

Knowledge



Time of onset

Chronic lymphocytic leukemia is mostly in the elderly, and the onset age of this disease in Europe and America is generally 70-75 years old.



Prevalence

Chronic lymphocytic leukemia is the most common type of leukemia in the West, accounting for 25%-35% of all leukemias. The middle-aged incidence rate of European and American people reaches 4-5/100000. The proportion of male and female is 1.2:1-1.7:1. The incidence rate of Chronic

lymphocytic leukemia in Asian population is significantly lower than that in Europe and the United States. According to a population registration data, the incidence rate of Japan (2010), South Korea (2018) and Taiwan, China (2021) is about 1/10 of that of Europe and the United States.

Typical symptoms

The disease is often referred to as painless lymphadenopathy or an unexplained increase in the number of lymphocytes. Common symptoms such as systemic lymphadenopathy, hepatosplenomegaly, weight loss, repeated infections, bleeding and anemia. Symptoms can occur when certain organs and tissues are accompanied by leukemia cell infiltration, such as exophthalmos, upper respiratory tract obstruction, scalp nodules, and urethral obstruction.

Common complications include:

1. Infection: Infection is the main cause of death and deterioration of patients. About 40% of patients will have serious infections.
2. Secondary tumors: 9%-20% of patients may have secondary tumors, the most common ones are secondary soft tissue sarcoma and lung cancer.

Treatment

The disease is still difficult to be completely cured, but the prognosis is good, and the five-year survival rate is generally about 50%. The main treatment methods include chemotherapy, radiotherapy, glucocorticoids, splenectomy, bone marrow transplantation and so on.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS4987855	BCL2	CC	2.10	1.00
RS7176508	intergenic	AA	1.88	0.98
RS10936599	MYNN	CC	1.59	0.99
RS17246404	POT1	CC	1.49	0.99
RS8024033	intergenic	CC	1.49	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is calculated based on published academic literatures related to chronic lymphocytic leukemia. By studying the genotype differences between chronic lymphocytic leukemia patients and normal people, these literatures have found some polymorphic sites associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of chronic lymphocytic leukemia item uses the 34 polymorphic loci in and between genes of *PRKD2*, *SP140*, *BCL2* and *IRF4*. The test results indicate whether the user has a higher prevalence of chronic lymphocytic leukemia in the combination of genotypes at the 34 polymorphic loci tested compared to all users. This test does not represent the user's true risk of chronic lymphocytic leukemia. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of chronic lymphocytic leukaemia.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of chronic lymphocytic leukemia at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
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3. If you have a family history of chronic lymphocytic leukemia, or you think that you are likely to suffer from chronic lymphocytic leukemia, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 34 polymorphic loci related to the risk of chronic lymphocytic leukemia, including rs7176508, rs872071 and rs4987855.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to chronic lymphocytic leukemia.

Gestation Diabetes Mellitus (GDM)

It refers to the first diagnosis of glucose intolerance during pregnancy in women without diabetes or impaired glucose tolerance in the past, which usually occurs in the middle and late stages of pregnancy.

My Genetic Risk Is Average For The Population

1.03 times

Average incidence rate is 9160 per 100,000 in the population

Suggestions

1. Early marriage and early childbirth, pregnant women over 30 years old, the incidence of gestational diabetes has increased significantly.
2. Maintain a healthy weight: BMI before pregnancy is maintained between 18.5 and 23.9.
3. Eat a reasonable diet during pregnancy, avoid excessive nutrition, and prevent excessive weight gain. Suggested pregnancy weight gain: the first three months of pregnancy, the weight gain is about 2 kg, the second trimester (3-6 months of pregnancy) and the third trimester (7-9 months of pregnancy) each increase by about 5 kg, and the weight gain during the whole pregnancy is about 12 kg.
4. Reducing the number of pregnancies can reduce the risk of disease.

Influencing factors



Controllable factors

1. Age: Studies have shown that the incidence of gestational diabetes is significantly increased when pregnant women are ≥ 30 years old.
2. Obesity before pregnancy: BMI ≥ 24.0 , the incidence of gestational diabetes has increased significantly.
3. Unreasonable diet during pregnancy (for example, excessive intake of carbohydrates), and those with excessive weight gain during pregnancy have an increased risk of disease.

4. Pregnant women with multiple pregnancies have a higher risk of gestational diabetes.

Uncontrollable factors

1. Race: There are great differences in the incidence of different races. Foreign studies have shown that Spanish or African Americans have the highest incidence of gestational diabetes, followed by Asians, and Caucasians have a lower incidence.
2. Family inheritance: Pregnant women who have a family history of diabetes or whose mother is a patient with gestational diabetes are more susceptible to the disease.
3. Past obstetric medical history: pregnant women who have given birth to congenital malformations, giant babies, fetal abortion, cesarean section, and gestational diabetes are more susceptible to the disease.
4. Physical factors: pregnant women with short stature, patients with polycystic ovary syndrome, α -thalassemia, and hepatitis B carriers are more susceptible to this disease.
5. Pregnant women with multiple pregnancies have a higher risk of gestational diabetes.

Knowledge

Time of onset

Pregnancy (usually in the middle and late stages). Routine obstetrics are generally screened for gestational diabetes at 24 to 28 weeks of gestation.

Prevalence

According to statistics, an average of 9160 cases of the disease occur per 100,000 people.

Typical symptoms

People who have symptoms of polydipsia, polyphagia, polyuria during pregnancy, or repeated bacterial infections, and this pregnancy is complicated by polyhydramnios or a giant fetus.

Treatment

The key to treatment is blood sugar control, and the key measure is diet control. Patients whose blood sugar cannot be controlled by diet need to use insulin. Dietary control principles: the recommended caloric intake for pregnant women with a BMI index of 22.0-25.0 is 30 kcal/kg; the recommended caloric intake for pregnant women with a BMI index of 26.0-29.0 is 24 kcal/kg; the recommended caloric intake for pregnant women with a BMI index >30.0 is 12 ~15 kcal/kg. The total dietary ratio is 33%-40% carbohydrates, 35%-40% fat, and 20% protein.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS7754840	CDKAL1	CG	1.52	0.97
RS10830962	intergenic	CG	1.45	0.97
RS10830963	MTNR1B	CG	1.18	1.00
RS7903146	TCF7L2	CC	1.00	0.98

Risk calculation

The risk value of this test is calculated based on published academic literatures related to gestational diabetes. These literatures have found some polymorphic loci related to the risk of the disease by studying patients with gestational diabetes and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of gestational diabetes item uses 4 polymorphic loci in and between genes of *MTNR1B*, *CDKAL1* and *TCF7L2*. The test results indicate whether the user has a higher prevalence of gestational diabetes in the combination of genotypes at the 4 polymorphic loci tested compared to all users. This test does not represent the user's true risk of gestational diabetes. The basis of the test is mainly from the research of Caucasian and Asian (including Chinese), other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could lead to a risk of gestational diabetes.
2. The test results have no clinical significance.

3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of gestational diabetes at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of gestational diabetes, or you think that you are likely to suffer from gestational diabetes, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 4 polymorphic loci related to the risk of gestational diabetes, including rs10830962, rs10830963, rs7754840 and rs7903146.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian (including Chinese).

Limit of detection

The test may not cover all genes or loci related to gestational diabetes.

Periodontitis

Periodontitis is an inflammation of periodontal tissue (periodontal ligament, gingiva, cementum and alveolar bone). It is a further development of gingivitis, mainly caused by the accumulation of dental plaque and calculus between the teeth and the gums. It is one of the main causes of tooth loss in adults.

My Genetic Risk Is Average For The Population

1.03 times

Average incidence rate is 80000 per 100,000 in the population

Suggestions

1. Develop good oral hygiene habits: brush your teeth at least twice a day, brush your teeth with a horizontal quiver brush, use fluorine toothpaste, floss and mouthwash, change your toothbrush for about three months, and gargle after meals. Regularly visit the hospital to check the mouth (twice a year) and perform cleaning (tooth washing) and under-gum scraping (deep cleaning) to effectively remove plaid and plaque.
2. Smoking can easily exacerbate periodontitis, refuse to take the initiative to smoke, or quit smoking as soon as possible.
3. To ensure a balanced nutrition, eat vitamin C-rich fruits and vegetables or supplements.
4. Excessive stress can cause periodontitis, when the pressure is too great, it is recommended to talk to friends and family, through sports, music and other ways to relax, or consult a psychologist.
5. Obesity leads to periodontitis susceptibility and the need to control a healthy weight: BMI is maintained between 18.5-23.9.

Influencing factors

Controllable factors

1. Oral hygiene: periodontitis is caused by the long-term accumulation of plaque and platones between teeth and gums.

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2. Smoking: smoking reduces saliva secretion and can easily exacerbate periodontitis.
 3. Vitamin: vitamin C deficiency can cause periodontitis.
 4. Mental factors: excessive stress can cause periodontitis.
 5. Obesity: obesity can lead to changes in the body's insulin tolerance and low inflammation of the body, which can lead to periodontitis.

Uncontrollable factors

1. Age: The incidence increases with age.
2. Disease: The incidence of periodontitis in diabetics is significantly higher than that of healthy people, and the disease is more serious.

Knowledge

Onset time

Chronic periodontitis occurs in adults older than 35 years old, and invasive periodontitis usually occurs in children or young adults.

Prevalence

The prevalence of adult periodontitis in China is as high as 80%-90%, and the prevalence of periodontitis increases with age.

Typical symptoms

Gum redness, gums that bleed easily, bad breath odor (halitosis), periodontal bag formation, tooth slot bone absorption, periodontal pus, loose teeth, etc.

Treatment

Repeated professional care is generally required, including cleaning and under-gum scraping to remove plaque and deposited dental stones, and deeper pockets in your gums usually require surgical cleaning or removal. If deep periodontal bags are present, consider using antibiotics such as amoxicillin, tetracycline, and metronidazole, or placing small sticks or gels containing antibiotics in deep periodontal bags. If necessary, perform periodontal surgery and loosen teeth to secure.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1537415	GLT6D1	GG	2.53	1.02
RS3826782	ADGRE1	AG	2.01	1.06
RS6596473	intergenic	CC	1.82	1.01
RS2521634	intergenic	AG	1.49	1.01
RS1333048	intergenic	AA	1.00	1.03

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to Periodontitis. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between the patients with Periodontitis and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Periodontitis risk item uses eight polymorphic loci on *IL1B*, *SLC23A1*, *GLT6D1*, and etc. The test result indicates whether the user has a higher possibility of Periodontiti in genotype combination of the seven polymorphic loci detected compared with all users, and the test does not represent the user's real risk of Periodontiti. The test is mainly based on the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of Periodontitis.

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2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Periodontitis from the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If you have a family history of Periodontitis, or you think that you are likely to suffer from Periodontitis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 8 polymorphic loci associated with Periodontitis risk, such as rs2521634, rs1143634, and rs6596473, etc.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with Periodontitis.

Dental Caries

Dental caries, commonly known as tooth decay or decayed tooth, refers to the phenomenon that the teeth are corroded due to bacterial activities, which goes deep into the teeth and causes tooth pain.

My Genetic Risk Is Average For The Population

1.02 times

Average incidence rate is 60000 per 100,000 in the population

Suggestions

1. Develop good oral hygiene habits: Brush your teeth at least twice a day; Brush your teeth with horizontal quiver brushing technique; Use floss and mouthwash; Change your toothbrush for about three months; Gargle after meals; Regularly visit the hospital to check the mouth cavity (twice a year) and perform cleaning (tooth washing) and under-gum scraping (deep cleaning) in order to effectively remove plaid and plaque.
2. Eat less carbohydrate, especially high-sugar content food that can promote the formation of dental plaque food.
3. Use fluorine toothpaste and apply fluorine coating to the teeth can enhance the anti-acid resistance of tooth enamel, reducing the occurrence of caries.
4. When teeth are sprouting, one should have pit and fissure sealant treatment, which can reduce the occurrence of caries.
5. Do not eat too much hard food, so as not to damage teeth. If tooth damage is occurred, one should repair in time.

Influencing factors



Controllable factors

1. Oral hygiene: dental caries is caused by acids produced by bacteria in dental plaques.

2. Food: frequent consumption of carbohydrates and sugars promotes the formation of plaque, which can easily cause dental caries.
3. Fluoridation treatment: if the teeth are not exposed to enough fluoride environment, the surface is prone to decay.
4. Pit and fissure sealant treatment: it is easy to accumulate bacteria and food residues at the pit and fissure of the teeth. Sealing the pit and fissure with special materials can prevent dental caries.
5. Physical damage: defects formed by tooth damage may lead to dental caries.



Uncontrollable factors

Age: the occurrence of dental caries increases with age.

Knowledge



Onset time

It can occur at any age.



Prevalence

According to the world health organization (WHO), there are 60-90% school-age children and nearly 100% adults worldwide suffer from dental caries.



Typical symptoms

In early caries, pain does not occur when the cavity is shallow, but begins when the cavity penetrates into the dentin. Sometimes you only feel pain when you eat hot food, cold drinks or sweets.



Treatment

If the caries have not progressed to dentin, fluoride treatment (remineralization treatment) can be carried out through fluoride mouthwash, high-fluoride toothpaste, etc. Once caries has reached the dentin, decaying materials need to be removed and filled (amalgam, composite resin materials, etc). When the tooth is too advanced to permanently damage the pulp, root canal treatment, crown repair or extraction of the affected tooth may be required.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS2046315	intergenic	AG	1.28	1.00
RS10486722	INHBA-AS1	CT	1.18	1.00
RS72748935	intergenic	TT	0.94	1.00
RS11672900	intergenic	AA	0.96	1.00
RS10048146	intergenic	AG	0.97	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to dental caries. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between the patients with dental caries and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The dental caries risk item uses 8 polymorphic loci on *INHBA-AS1*, *AAMP*, *PBX3* etc. The test result indicates whether the user has a higher possibility of dental caries in genotype combination of the 8 polymorphic loci detected compared with all users, and the test does not represent the user's real risk of dental caries. The test is mainly based on the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of dental caries.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of dental caries from the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If you have a family history of dental caries, or you think that you are likely to suffer from dental caries, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 8 polymorphic loci associated with dental caries risk, such as rs2046315, rs10486722, rs1079204.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with dental caries.

Migraines

Migraine is a kind of common chronic neurovascular disease. The attack is often unilateral pulsatile headache, which usually lasts for 4-72 hours.

My Genetic Risk Is Average For The Population

1.01 times

Average incidence rate is 9300 per 100,000 in the population

Suggestions

1. Reduce or avoid the use of Drugs that may cause migraine, and give priority to drugs that will not cause migraine under the guidance of doctors.
2. Actively regulate emotions to prevent migraine caused by emotional or psychological factors.
3. Pay attention to rest and keep enough but not excessive sleep time.
4. Avoid taking Food that may cause migraine; Make sure of adequate Magnesium and perform fasting in a scientific way.
5. Avoid other External stimuli.

Influencing factors



Controllable factors

1. Typical migraine attacks may occur after taking some drugs, such as oral contraceptives, nifedipine (nifedipine), isosorbide (isosorbide), nitroglycerin, etc.
2. Emotional or psychological factors, such as stress, depression and anxiety, will increase the risk of migraine.
3. Too much or too little sleep can cause migraine.
4. Some foods will increase the risk of migraine, such as caffeine, citrus fruits, artificial sweeteners (aspartame, saccharin), and meat containing nitrite; Magnesium deficiency and fasting may also increase the risk.

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5. Other external stimuli, such as alcohol, tobacco, strong light, noise, strong smell, strong cold stimulus, crowded environment will cause migraine.



Uncontrollable factors

1. Endocrine: hormonal changes can cause migraines, such as pregnancy and menstruation.
 2. Gender: women are more likely to suffer from migraines than men.
 3. Age: migraines are more likely to occur during adolescence or early adulthood.
 4. Minor head movements: It can also cause migraines, such as riding in a car, climbing stairs, sports, etc.
 5. Other external stimuli, such as weather changes can also cause migraines.
-

Knowledge



Time of onset

Migraines usually start in adolescence or early adulthood. About 25% of patients occur before the age of 10, 55% of the patients occur before the age of 20, and more than 90% of the patients occur before the age of 40.



Prevalence

The annual prevalence rate of migraine in Chinese population is 9.3% according to a 2011 statistics. This disease is more common in women than in men.



Typical symptoms

1. Aura: about 20% of migraine patients will have aura symptoms. The most common are visual auras, such as lightning, dark spots, black eyes, distortion of vision, blank space outside the field of vision, etc. Other auras include paralysis and aphasia. Generally, the aura lasts for 5 to 20 minutes.
2. Headache: it can appear in any part of the head and neck, most common are unilateral, often pulsating headache or drilling pain. The degree of headache is moderate to severe and unbearable. The headache lasts for about 4-72 hours. During the headache period, it will be accompanied by nausea, blurred vision, photophobia, sound fear and like living alone.
3. Other symptoms: there will be mental and psychological changes before and after the headache, such as irritability, elevated mood or depression, but also physical discomfort such as fatigue, nausea, urination or oliguria. The specific symptoms vary greatly among individuals.



Treatment

It mainly includes symptomatic treatment and preventive treatment. At present, migraine cannot be completely cured. Adult migraine often recurs for decades, while about half of children no longer experience migraine after 6 years of onset, and about 1/3 of migraine can be improved.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS75002882	intergenic	GG	1.37	1.00
RS13078967	intergenic	AA	1.34	1.00
RS138556413	intergenic	CC	1.30	1.00
RS12260159	HPSE2	GG	1.18	1.00
RS9349379	PHACTR1	GG	0.86	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is calculated based on published academic literatures related to migraine. By studying the genotype differences between migraine patients and normal people, these literatures have found some polymorphic sites associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

 Applicable situation

The health risks of migraine item uses the 102 polymorphic loci in and between genes of *TRPM8*, *PRDM16*. The test results indicate whether the user has a higher prevalence of migraine in the combination of genotypes at the 102 polymorphic loci tested compared to all users. This test does not represent the user's true risk of migraine. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of migraine.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of migraine at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
3. If you have a family history of migraine, or you think that you are likely to suffer from migraine, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 102 polymorphic loci related to the risk of migraine, including rs10166942 and rs2651899.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to migraine.

Autism

Autism is a disease caused by brain development disorders, which is characterized by difficulties in emotional, verbal and non-verbal expression and social interaction disorders.

My Genetic Risk Is Average For The Population

1.01 times

Average incidence rate is 1000 per 100,000 in the population

Suggestions

1. Pay attention to health during pregnancy: stay away from pathogens and other high-risk factors during pregnancy, do not smoke or drink alcohol, pay attention to the use of banned drugs for pregnant women, and maintain a healthy lifestyle.
2. Early childbearing age: try to get married and have children as early as possible, and the age gap between childbearing couples should not be too large.

Influencing factors

Controllable factors

1. Infection or habit during pregnancy: If pregnant women are infected with viruses, the probability of children getting sick will increase. Pathogens include chickenpox, rubella virus, *Toxoplasma gondii*, etc. These pathogens may cause the development of children's brain nerves, leading to the occurrence of autism. The risk of children suffering from autism was also increased if the mother drank heavily, smoked cigarettes or took banned drugs during pregnancy.
2. Childbearing age: Studies have shown that the age gap between parents increases the risk of children suffering from autism, especially if the father is older.

Uncontrollable factors

-
1. Age: Onset usually begins in childhood, before age three, and often continues into adolescence and adulthood.
 2. Gender: Male has a relatively higher incidence rate than female.
 3. Family history: Studies have shown that 20% of autistic patients have a family history of autism.
-

Knowledge

Time of onset

The age of onset is generally less than three years old, and most children show abnormalities at the age of seven or eight months.

Prevalence

At present, the prevalence of autism in China is similar to that of other countries in the world, about 1%. The number of autistic patients has exceeded 10 million, and the number of children aged 0-14 years has reached more than 2 million. The incidence rate of the disease is significantly different between men and women, and the prevalence rate of men and women in China is 4:1.

Typical symptoms

1. Social difficulties: children have no attachment to their parents, no emotional contact with people, and do not communicate with others; Have no fear of strangers; Without eye-to-eye contact with people, they will not express their feelings or needs with their eyes.
2. Speech retardation: accompanied by some special speech abnormalities, especially it is difficult to use words to communicate with people. Poor speech comprehension, sometimes imitative speech and stereotyped speech, sometimes pronoun misuse, etc.
3. Stereotyped behavior: often do things or play with toys in the same way, require items to be placed in a fixed position, take the same route when going out, and only eat a few kinds of food for a long time.

Treatment

1. Medication: the most common classes of medications are antidepressants, stimulants and antipsychotics.
 2. Behavioral therapy: the principle is to improve and remove autistic symptoms and maladaptive behaviors through the use of individual spontaneous reactive behaviors and so-called operational constraints.
-

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2217262	DOCK4	AA	5.62	0.97
RS12518194	intergenic	AA	1.72	0.99
RS7088729	intergenic	CT	1.23	0.99
RS73416724	intergenic	AA	1.23	1.00
RS3740304	DIP2C	AG	1.21	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to autism. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between subjects with autism and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The autism item uses 28 polymorphic loci in genes such as *FHIT*, *FOXP1*, *ATP2B2* and between other genes. The test results indicate whether the user has a higher risk of autism for the genotype combination of 28 polymorphic loci detected compared to all users. The test cannot represent the user's real risk of autism. The basis of the test is mainly from the research of Caucasian and Asian, other populations are for reference only.

Notification

1. The test does not cover all genes or loci that contribute to the risk of autism.

-
2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are jointly determined by multiple factors such as genes and environment. This study only predicts your risk of autism at the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If there is a family history of autism in your family, or if you think you are at risk of developing autism, you are recommended to consult an authoritative health professional or clinician for help.

Test content

28 polymorphic loci associated with the risk of autism were detected, including rs1353545, rs2828478 and rs10258862.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect autism.

Haemorrhoids

Haemorrhoids are swollen veins in the anus and rectum, similar to varicose veins. It can be divided into internal hemorrhoids, external hemorrhoids and mixed hemorrhoids.

My Genetic Risk Is Average For The Population

1.01 times

Average incidence rate is 49140 per 100,000 in the population

Suggestions

1. Eat more fiber-rich foods, such as fruits, vegetables, beans and whole grains; avoid spicy and other irritating foods; drink more water; and abstain from alcohol.
2. Go to the toilet as soon as you feel the urge to defecate; avoid excessive force and hold your breath during defecation, relax your mind and defecate naturally.
3. Avoiding sitting or standing for a long time, avoiding frequent lifting of heavy objects, and adhering to regular exercise habits can help reduce the occurrence of hemorrhoids.
4. The use of side lying posture in pregnant women can reduce the risk of hemorrhoids.

Influencing factors



Controllable factors

1. Food: Alcohol, spicy, irritating food can easily induce hemorrhoids.
2. Defecation habits: Habits such as not defecation in time when have desire to defecate, excessive force during defecation, holding your breath during defecation, reading or playing with mobile phone during defecation, etc. are easy to induce hemorrhoids.
3. Other lifestyle habits: Standing for a long time, sitting for a long time, lifting heavy objects frequently, and lack of exercise habits can easily induce hemorrhoids.
4. Sleeping position: Side lying posture can reduce the risk of hemorrhoids in pregnant women.

Uncontrollable factors

1. Age: Commonly between 45-65 years old.
-

Knowledge

Time of onset

The disease can occur in all age groups, and it is more common between 45-65 years of age. It is more prone to disease with age.

Prevalence

It is a common anorectal disease. A 2015 survey of the Chinese population indicated an incidence rate of 49.14% (68,906 people). In the American population, there are even reports of an incidence rate as high as 86%.

Typical symptoms

External hemorrhoids:

- Pain or itching: Mainly manifested as pain or itching in the anal area.
- Anus swelling: Swelling occurs around the anus due to inflammation.
- Bleeding: After a hard bowel movement, accompanied by bleeding.

Internal hemorrhoids:

- Painless bleeding during defecation: After a normal defecation, there is no discomfort in the anus, but bright red blood is found on the toilet paper or in the toilet.
- Itching: When internal hemorrhoids ooze mucus, they can irritate the skin of the anal canal and cause itching.
- Discomfort: The patient may still have the urge to defecate after defecation. This discomfort is caused by the bulging of hemorrhoid tissue at the end of the large intestine.
- Pain: Most internal hemorrhoids do not cause pain, but if large hemorrhoids bulge from the anal opening and are squeezed by the anal sphincter, it may cause pain. Severe pain may indicate that the blood supply of hemorrhoids is blocked, and emergency treatment should be given at this time.

Mixed hemorrhoids:

- There may be symptoms of both external and internal hemorrhoids.

Thrombotic hemorrhoids:

- When blood accumulates on the external hemorrhoids and forms a thrombus, it is a more serious situation, with severe pain, obvious swelling and hard lumps around the anus.

Treatment

Most new-onset hemorrhoids can be cured by fine-tuning dietary habits, such as eating fiber-rich foods, drinking more water, and bowel habits. Some common home treatments: hemorrhoid cream

or suppository, hydrocortisone ointment, sitz bath, over-the-counter pain relievers. If hemorrhoids are very large and painful, surgery or other treatments are required.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS676996	ABO	GG	0.88	1.00
RS1333047	intergenic	AA	1.08	1.00
RS13017210	intergenic	TT	0.94	1.00
RS2327426	intergenic	CC	0.94	1.00
RS2180811	intergenic	AA	1.06	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to haemorrhoids. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences in the risk of haemorrhoids among drinking and non-drinking people. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

 Applicable situation

The health risks of haemorrhoids item uses 68 polymorphic loci on genes such as *ANO1*, *SRPX*, *ABO*, *GDF7* and other genes. The test results indicate whether the user has a higher possibility of haemorrhoids in the combination of genotypes at the 68 polymorphic loci tested compared to all users. It does not represent the user's real risk of haemorrhoids. The basis of the test is mainly from the research of European, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that can lead to the risk of haemorrhoids.
2. The test result has no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of haemorrhoids from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of haemorrhoids, or you think that you are likely to suffer from haemorrhoids, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 68 polymorphic loci related to hemorrhoids, such as rs2186797, rs35318931, and rs676996.

Population samples

The basis of the test is mainly from the research of European.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to haemorrhoids.

Gastroesophageal reflux disease

Gastroesophageal reflux disease is a common chronic disease of digestive tract system, which refers to a series of symptoms, end organ effects and complications caused by the reflux of gastric contents to esophagus, throat, mouth, lungs, etc.

My Genetic Risk Is Average For The Population

1.01 times

Average incidence rate is 12500 per 100,000 in the population

Suggestions

1. Develop healthy dietary habits: Reduce the intake of high-fat foods and fried foods; reduce the intake of foods that aggravate the disease, such as chocolate, coffee, carbonated drinks, garlic, mint, etc.; eat slowly and avoid binge eating; it is not advisable to eat 2-3 hours before bedtime; it is not advisable to lie down immediately after eating during the day.
2. Change unhealthy living habits: Quit smoking and drinking; don't wear tight clothes; avoid strenuous exercise.
3. Maintain a good state of mind: Find timely and effective adjustment measures to deal with negative emotions such as worry and anxiety.
4. Control your weight: Keep your BMI between 18.5 and 23.9, avoid sitting for a long time, and exercise at least 4 hours a week.

Influencing factors

Controllable factors

1. Dietary habits: Carbonated drinks, coffee, chocolate, mint, oily foods; eating within three hours before going to bed; lying down immediately after eating may induce gastroesophageal reflux disease.
2. Living habits: Smoking, drinking alcohol, wearing tight-fitting clothes frequently, and strenuous exercise may induce gastroesophageal reflux disease.

3. Mental factors: Anxiety and depression may induce gastroesophageal reflux disease.
4. Obesity: Obesity can easily lead to the occurrence of this disease.

Uncontrollable factors

1. Age: 40-60 years old has a higher incidence.
2. Diseases that cause gastric outlet blockage: Such as duodenal ulcer, duodenal cancer, pyloric obstruction, etc.
3. Diseases that cause decreased saliva and mucus secretion: Such as Sjogren's syndrome, scleroderma, etc.

Knowledge

Time of onset

The disease has a tendency to increase with age, and the peak age of onset is 40-60 years old.

Prevalence

In 2015, studies reported that the incidence of gastroesophageal reflux disease in North America was as high as 27.8%, while that in Europe was as high as 25.9%. In 2015, a meta-analysis showed that the incidence of gastroesophageal reflux disease in China was approximately 12.5%.

Typical symptoms

- "Heartburn": is a discomfort or burning pain behind the breastbone. It may occur after eating, shortly after lying down, or when leaning forward. It is one of the most common symptoms of this disease.
- Reflux: refers to the sensation of stomach contents flowing towards the pharynx or oral cavity.
- Chest pain: manifested as a dull, heavy discomfort that permeates the entire chest. Chest pain may co-exist with a burning sensation in the upper abdomen and may be confused with the pain during a heart attack.
- Discomfort in the upper abdomen: Burning sensation in the upper abdomen.
- Throat discomfort: belching; hoarse voice; feeling something stuck in the throat; coughing; excessive saliva; nausea.
- Difficulty swallowing: After swallowing food, it feels that the food is stagnant in a certain position, not entering the stomach, or even spit out from the mouth; it is more common in advanced gastroesophageal reflux disease.

Treatment

1. Drug therapy: acid inhibitor and prokinetic therapy. Acid inhibitors achieve the purpose of treatment by inhibiting the secretion of gastric acid. They are the main treatment drugs for gastroesophageal reflux disease. Commonly used drugs include omeprazole, rabeprazole, ranitidine, famotidine and so on. The role of prokinetic agents is to accelerate gastrointestinal

motility, reduce the residence time of acidic substances in the stomach, thereby reducing reflux. Commonly used drugs include mosapride and domperidone.

- 2. Surgical treatment: fundoplication is the most commonly used operation for the treatment of gastroesophageal reflux disease, which can relieve the symptoms of gastroesophageal reflux disease and inflammation of the esophagus (esophagitis); gastric fixation refers to the connection and fixation of the stomach and the diaphragm. The goal is to prevent the stomach from entering the thoracic cavity through the diaphragm hiatus.
- 3. Psychotherapy: It can alleviate the anxiety of patients and avoid aggravation of the condition.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS13107325	SLC39A8	CC	0.72	1.00
RS7241572	intergenic	GG	0.84	1.00
RS3766823	ADGRB2	GG	0.84	1.00
RS205262	C6orf106	AA	0.85	1.00
RS12357321	intergenic	GG	0.86	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to gastroesophageal reflux disease. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype difference between gastroesophageal reflux disease patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk item of vertigo uses the 57 polymorphic loci on genes such as *DRD2*, *FTO*, *ERBB4*, *FOXP2*, *DPYD* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 57 polymorphic loci detected by this test increases the risk of vertigo. It does not mean the user's true risk of vertigo. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that cause the risk of gastroesophageal reflux disease.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only evaluates your risk of gastroesophageal reflux disease at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of gastroesophageal reflux disease, or you think that you are likely to suffer from gastroesophageal reflux disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 57 polymorphic loci associated with gastroesophageal reflux disease, including rs2734839, rs9940128, and rs7600261.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to gastroesophageal reflux disease.

Vertigo

Vertigo is a disturbing illusion of motion. The most common is the rotational motion of oneself or environment.

My Genetic Risk Is Average For The Population

1.01 times

Average incidence rate is 3400 per 100,000 in the population

Suggestions

1. Control alcohol intake: For people who have not developed alcohol dependence, it is recommended to reduce their alcohol intake to prevent alcohol dependence and avoid the occurrence of alcohol withdrawal syndrome; for people who are alcohol dependent, it is recommended to take a step-by-step approach to reduce alcohol intake, and consult a clinician if necessary.

Influencing factors



Controllable factors

1. Alcohol withdrawal syndrome: People who are dependent on alcohol suddenly reduce or interrupt their alcohol intake can cause vertigo.



Uncontrollable factors

1. Drugs: Certain drugs with vestibular or cerebellar toxicity, such as aminoglycoside drugs (such as gentamicin 9) and antiepileptic drugs can cause vertigo.
2. Disease: Vertigo is a typical complication of peripheral vestibular diseases such as Meniere's disease, benign paroxysmal positional vertigo, vestibular neuritis and central diseases such as vestibular migraine anemia; other diseases such as tumors, heart vascular diseases, mental diseases, etc. can also cause vertigo.
3. Age: The incidence rate increases with age.

4. Gender: The incidence of females is higher in males.

Knowledge

Time of onset

With age, the incidence rate gradually increases. The high-incidence period is between 40 and 59 years old, followed by people 60 years and older, and adolescent patients are the least.

Prevalence

Vertigo accounts for 5% of China's domestic outpatient clinics and 15% of ENT clinics. A survey in 2006 showed that the national standardized prevalence rate of vertigo in China was 3.4%.

Typical symptoms

- Feeling of yourself or the surrounding environment rotating, swaying, tilting, up and down, jumping up and down or sliding.
- Distorted sense of self movement during normal head movement.
- Accompanied by systemic sensations, such as dizziness, nausea, vomiting, and sweating.
- The attack can last from a few seconds to several hours. In severe cases, it can last for many days or months, causing great inconvenience to life.
- It is the main risk factor for falls and fractures.

Distinguish vertigo from dizziness

When you feel lightheaded or disoriented, this is dizziness; When you feel yourself or your room spinning, this is vertigo.

Treatment

1. Etiology treatment: Determine the cause of vertigo, and take treatment for the cause.
2. Relieve symptoms: In order to relieve the pain and discomfort caused by symptoms, anti-dizziness, antiemetic drugs and vestibular rehabilitation training can be used.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS428549	intergenic	AG	1.10	1.00

RS2272744	intergenic	GG	1.00	1.00
RS10862089	OTOGL	GG	1.00	1.00
RS612969	TECTA	AA	1.00	1.00
RS6753393	intergenic	GG	1.00	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to vertigo. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype difference between vertigo patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk item of vertigo disease uses the 6 polymorphic loci on genes such as *OTOG*, *OTOGL*, *TECTA* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 6 polymorphic loci detected by this test increases the risk of vertigo disease. It does not mean the user's true risk of vertigo disease. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that cause the risk of vertigo.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only evaluates your risk of vertigo at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of vertigo, or you think that you are likely to suffer from vertigo, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 6 polymorphic loci associated with vertigo, including rs428549, rs7130190, and rs10862089.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to vertigo.

Placental Abruption

Placental abruption is after 20 weeks of pregnancy or during childbirth, the placenta in its normal position is partially or completely peeled from the uterine wall before the fetus is delivered.

My Genetic Risk Is Average For The Population

0.99 times

Average incidence rate is 2000 per 100,000 in the population

Suggestions

1. It is recommended to wear non-slip shoes during pregnancy, walk carefully when going up and down the stairs, and avoid crowded places to prevent the abdomen from being hit or squeezed.
 2. During pregnancy, you should quit smoking, drug abuse and other bad habits.
 3. Pregnancy-induced hypertension syndrome is prone to occur in the middle and late stages of pregnancy. Once pregnant women develop symptoms of hypertension, edema and proteinuria, they should actively go to the hospital for early treatment.
 4. Placental abruption is related to body position, so during pregnancy, especially in the third trimester, avoid prolonged supine position. It is recommended to take a side lying position to rest.
 5. Choose the best childbearing age: 25 to 35 years old, older women (over 35 years old) will increase the risk of placental abruption.
-

Influencing factors



Controllable factors

1. Trauma: a pregnant woman has a trauma, especially a direct impact or squeeze on the abdomen (such as a fall or a car accident).
 2. Bad habits: smoking, drug abuse and other bad habits will increase the risk of placental abruption.
-

3. Vascular disease: women with hypertension during pregnancy (especially severe preeclampsia), chronic hypertension, chronic kidney disease or systemic vascular disease, the placenta is easily peeled from the uterine wall.
4. Position: in the third trimester of pregnancy or after delivery, if the pregnant woman lies supine for a long time, the enlarged uterus will compress the inferior vena cava, difficult to return, and may cause uterine vein congestion or rupture, leading to placental dissection.
5. Older-age women: women over 35 years of age are more likely to have placental abruption.

Uncontrollable factors

1. Umbilical cord factor: when the umbilical cord is too short (<30 cm) or because the umbilical cord wraps around the neck or the body is relatively short, the fetus drops and pulls the umbilical cord during delivery, causing placental abruption.
2. Sudden drop in intrauterine pressure: premature rupture of the membranes before full-term pregnancy; during twin pregnancy, the first fetus is delivered too quickly; or too much amniotic fluid, which can cause the uterine cavity to flow out too quickly after artificial rupture of the membrane. The pressure drops sharply, the uterus shrinks suddenly, and the placenta and the uterine wall are dislocated and peeled off.
3. Amniocentesis: during operation, if the blood vessel attached to the placenta is punctured, the formation of a hematoma after the placenta may cause the placenta to peel off.
4. History of placental abruption: pregnant women with a history of placental abruption have a 10 times higher risk of recurring placental abruption than those without a history of placental abruption.

Knowledge

Time of onset

Placental abruption occurs after 20 weeks of pregnancy or during childbirth.

Prevalence

The incidence rate is 1%-2% abroad and 0.46%-2.1% domestically.

Typical symptoms

The main symptom of mild placental abruption is vaginal bleeding, which may be accompanied by mild or insignificant abdominal pain, and the signs of anemia are not significant. Severe placental abruption is manifested by sudden onset of persistent abdominal pain and/or backache and low back pain. The degree of this varies with the size of the stripped surface and the amount of blood accumulated after the placenta. The more blood accumulated, the more severe the pain.

Treatment

Once placental abruption is diagnosed, the pregnancy should be terminated promptly by vaginal delivery or cesarean section.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS6025	F5	CC	1.00	0.99

Risk calculation

The risk value of the test is calculated based on published academic literatures related to placental abruption. By studying the genotype differences between placental abruption patients and normal people, these literatures have found some polymorphic sites associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of placental abruption item uses a polymorphic locus in the *F5* gene. The test results indicate whether the user has a higher prevalence of placental abruption compared to all users. This test does not represent the user's true risk of placental abruption. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of placental abruption.
2. The test results have no clinical significance.

3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of placental abruption at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
3. If you have a family history of placental abruption, or you think that you are likely to suffer from placental abruption, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus rs6025 which related to the risk of placental abruption.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to placental abruption.

Dental Retardation

Dental retardation, which refers to the slow germination of deciduous and permanent teeth, is generally assessed by the time of first tooth germination or the number of teeth at a given age.

My Genetic Risk Is Average For The Population

0.98 times

Average incidence rate is 10000 per 100,000 in the population

Suggestions

Pay attention to nutrition during pregnancy: the tooth embryo of fetal deciduous teeth begins to form at 6-7 weeks. Pregnant women should ensure balanced nutrition and adequate supplementation of calcium and vitamin D. Lactation period to infant period should ensure sufficient amount of protein, calcium, vitamins and other intake, conducive to the normal development of teeth.

Influencing factors

Controllable factors

Nutrition: insufficient vitamin D and calcium during pregnancy may affect the germination of your baby's deciduous teeth. Food nutrition from lactation to early childhood can affect the calcification of teeth.

Uncontrollable factors

1. Gender: Girls typically develop their baby and permanent teeth earlier than boys.
2. Diseases: Some disease may delay tooth development, such as Rickets, Stupid disease, Down syndrome, and etc.

Knowledge

Onset time

Deciduous teeth retardation occurred before 3 years old, and permanent teeth retardation occurred between 6-13 years old.

Prevalence

Dental abnormalities are common, with defects due to hypoplasia affecting approximately 10% of the population and accounting for 6% of all dental care.

Typical symptoms

Dental retardation usually manifests itself as the loss or slow germination of teeth in a certain age. Usually there are 20 deciduous teeth and 28-32 permanent teeth.

Treatment

Dental abnormalities is prone to induce dental caries, we should pay attention to periodontal health, prevention of dental caries. When more teeth are missing, denture repair should be done, or more complicated orthodontics, prostheses and surgical treatment should be done.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS17101923	HMGA2	GG	1.78	0.99
RS4937076	CDON	AA	1.50	0.99
RS1799922	OPN1SW	TT	1.46	1.00
RS997154	C14orf93	GG	1.46	1.00
RS12229918	MSRB3	CG	1.35	0.98

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to Dental retardation. These literatures have found some polymorphic loci related to the risk of the disease by

studying the genotype differences between the patients with Dental retardation and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Dental retardation risk item uses 19 polymorphic loci on *MSRB3*, *EDA*, *RAD51B*, and etc. The test result indicates whether the user has a higher possibility of Dental retardation in genotype combination of the 19 polymorphic loci detected compared with all users, and the test does not represent the user's real risk of Dental retardation. The test is mainly based on the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of Dental retardation.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Dental retardation from the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If you have a family history of Dental retardation, or you think that you are likely to suffer from Dental retardation, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 19 polymorphic loci associated with the risk of dental stunting, such as rs12424086, rs2281845, and rs4491709.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with Dental retardation.

Insomnia

It is a sleep disorder characterized by frequent and persistent difficulties in falling asleep and/or maintaining sleep status, resulting in insufficient sleep satisfaction, and affecting daytime functions such as fatigue, drowsiness, and delayed response.

My Genetic Risk Is Average For The Population

0.98 times

Average incidence rate is 23500 per 100,000 in the population

Suggestions

1. Arrange meals reasonably: Avoid excessive consumption of Caffeinated beverages; Avoid overeating at dinner; Avoid eating 2-3 hours before bedtime.
2. Create a sleep environment: Try to avoid light and noise in the sleep environment. Use eye masks, earplugs, and other aids to sleep; Adjust the indoor temperature, keep the environment cool; Use Comfortable bedding.
3. Adjust sleep habits: Try to go to bed and get up on time every day to ensure a regular biological clock; A few hours before going to bed, avoid Electronic devices with luminescent screens; Avoid vigorous exercise before bedtime.
4. Adjust emotions: Actively adjust your mindset daily. Simple activities like yoga, stretching, meditation may help reduce stress and relax, relieve your body and mind, and promote sleep.

Influencing factors



Controllable factors

1. Eating habits: Excessive consumption of coffee, tea, alcohol, and other beverages at night may cause insomnia.
2. Environment: Inappropriate temperature, noise, and excessive brightness in the sleeping environment may cause insomnia.

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3. Sleep habits: Irregular sleep, vigorous exercise before bedtime, use electronic devices with luminescent screens before bedtime can interfere with the sleep cycle and cause insomnia.
 4. Psychological factors: When encountering stressful events, it may lead to temporary insomnia, and long-term stress may also lead to chronic insomnia. In addition, emotions such as anxiety, nervousness, and overexcitement can also lead to insomnia.

Uncontrollable factors

1. Age: Insomnia is more likely to occur with increasing age.
2. Gender: Women (especially during menstruation, pregnancy, and menopause) may experience insomnia due to changes in hormone levels.
3. Heredity: People with a family history of insomnia are more likely to experience insomnia.
4. Disease: Patients with anxiety, depression, post-traumatic stress disorder (PTSD), Parkinson's disease, pulmonary disease, hypertension, diabetes, cancer, chronic pain, heart failure and other diseases are prone to insomnia.
5. Drugs: Taking central nervous system stimulants, respiratory stimulants, appetite inhibitors, antidepressants, β -blockers, glucocorticoids and other drugs may cause insomnia. Insomnia may also occur after sudden withdrawal of hypnotic and sedative drugs for a long period of time.

Knowledge

Time of onset

Insomnia can occur at any age, especially 60-year-old age group. According to NSF (National Sleep Foundation), around 30-48% of elderly people experience insomnia.

Prevalence

More than 20% of the general adult population in the U.S. and Canada have reported experiencing insomnia (Sleep Research Society, 2012). Each year, 30-40% of adults in the U.S. report symptoms of insomnia at some point annually (American Journal of Managed Care, 2020). The lifetime risk of getting insomnia may be up to 40% higher for women than for men (Philosophical Transactions of the Royal Society, 2016).

Typical symptoms

Symptoms of insomnia may include:

- Difficulty in falling asleep at night (≥ 30 minutes before falling asleep)
- Difficulty in maintaining sleep (awake time at night ≥ 30 minutes)
- Waking up early (sleep termination time is at least 30 minutes earlier than expected)

At the same time, it is often accompanied by daytime fatigue or drowsiness, irritability, depression or anxiety, difficulty in concentrating, poor memory, increased errors or accidents and persistent worry about sleep.

Treatment

The basic goal of treating insomnia is to improve sleep quality, increase effective sleep time, eliminate daytime functional damage caused by insomnia, and ultimately improve the quality of life.

- Short-term insomnia caused by stress (<3 months) is mainly alleviated through psychological counseling. If insomnia is severe or accompanied by significant daytime dysfunction, medication can be used to intervene. The choice of medication varies from person to person.
- Chronic insomnia (insomnia symptoms occurring at least 3 times a week for at least 3 months) can be intervened with sleep cognitive behavioral therapy (CBTI) and medication.
- Other intervention methods include light therapy, biofeedback therapy, electrotherapy, ultrasound therapy, physical therapy, etc.

Adjuvant therapy

- Nutritional supplements: melatonin, 5-hydroxy-L-tryptophan, γ -aminobutyric acid, Omega-3 fatty acids, and other nutrients can help alleviate emotions and improve insomnia. The above nutrients can be consumed through Natural food intake or nutritional supplements under the guidance of a professional dietitian.
- Aromatherapy: Aromatherapy is one of the non-drug therapies for improving insomnia. Applying natural aromatic extracts to the human body through aerosol inhalation, massage, bathing, and other methods can help alleviate stress, improve sleep disorders, and reduce anxiety and depression. Natural aromatic extracts often use plants such as lavender, chamomile, orange peel, roses, and herbs as raw materials, and can also be selected according to your preferences. The specific treatment should be carried out under the guidance of a professional therapist.

My genetic result details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS79693059	intergenic	CC	0.86	1.00
RS4304930	intergenic	AA	1.18	1.00
RS67588220	intergenic	TT	0.86	1.00
RS11897824	intergenic	AA	0.87	1.00
RS10957292	intergenic	AA	0.87	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to insomnia. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between the patients with insomnia and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of insomnia uses 167 polymorphic loci on *PPP1R10*, *SLC39A8*, *DPYD*, *MYO1A* genes and among other genes. The test result indicates whether the user has a higher possibility of insomnia in genotype combination of the 167 polymorphic loci detected compared with all users, and the test does not represent the user's real risk of insomnia. The test is mainly based on the research of Caucasian , other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to insomnia.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of insomnia from the genetic level, and

does not represent your real health status, nor does it involve the judgment of your other health status and living habits.

3. If you have a family history of insomnia, or you think that you are likely to suffer from insomnia, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 167 polymorphic loci associated with insomnia, such as rs55777621, rs13107325, rs12039093 and rs703848.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to insomnia.

Ovarian Cancer

Ovarian cancer is a type of malignant tumour develops from germinal epithelium or ovarian stroma. It is one of the three most commonly found tumor in female reproductive system.

My Genetic Risk Is Average For The Population

0.97 times

Average incidence rate is 5 per 100,000 in the population

Suggestions

1. Dietary factors: high animal fat, high protein, high calorie diet can increase the risk of ovarian cancer; and eating more vegetables, grains, vitamins, etc. can reduce the risk of disease.
2. Women with a history of chronic pelvic inflammatory disease and other gynecological diseases are at increased risk.
3. Infertility patients who use ovulation-stimulating drugs: Studies have shown that patients who use ovulation-stimulating drugs to treat infertility have an increased risk of disease compared with untreated patients.

Influencing factors



Controllable factors

1. Dietary factors: high animal fat, high protein, high calorie diet can increase the risk of ovarian cancer; and eating more vegetables, grains, vitamins, etc. can reduce the risk of disease.
2. Women with a history of chronic pelvic inflammatory disease and other gynecological diseases are at increased risk.
3. Infertility patients who use ovulation-stimulating drugs: Studies have shown that patients who use ovulation-stimulating drugs to treat infertility have an increased risk of disease compared with untreated patients.

Uncontrollable factors

1. Genetic factors: there is a family genetic tendency.
2. Age: The incidence of diseases in women over 50 years old has increased significantly.

Knowledge

Time of onset

It can occur in women of all ages, most commonly in menopausal and menopausal women (40 to 60 years old), and the incidence is lesser under 20 years old.

Prevalence

According to statistics, an average of 72 cases of the disease occur per 100,000 people.

Typical symptoms

Early symptoms are not obvious. Clinical manifestations include pain (persistent pain), irregular menstruation, weight loss, masses in the lower abdomen, and effusion in the abdominal cavity, etc.

Treatment

Diagnose and treat as early as possible, and the 5-year survival rate for advanced ovarian cancer is only 35%. Treatment methods include: surgical treatment, radiotherapy and chemotherapy, targeted therapy (such as bevacizumab, PARP-1 inhibitor), etc.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS854560	PON1	AA	0.67	1.00
RS4320932	INS-IGF2	CT	1.34	0.99
RS662	PON1	CC	1.00	0.98
RS523349	SRD5A2	CC	1.00	0.98
RS3814113	intergenic	TT	1.00	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to ovarian cancer. These literatures have found some polymorphic loci related to the risk of the disease by studying patients with ovarian cancer and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of ovarian cancer item uses 16 polymorphic loci in genes of *PGR*, *GALNT1*, *HAGLR*, *SYNE1* and *TIPARP*. The test results indicate whether the user has a higher prevalence of ovarian cancer in the combination of genotypes at the 16 polymorphic loci tested compared to all users. This test does not represent the user's true risk of ovarian cancer. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of ovarian cancer.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of ovarian cancer at a genetic level, it

does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.

3. If you have a family history of ovarian cancer, or you think that you are likely to suffer from ovarian cancer, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 16 polymorphic loci related to the risk of ovarian cancer, including rs1042838, rs17647532, rs2072590, rs2295190 and rs2665390.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to ovarian cancer.

Gingivitis

Gingivitis is an inflammation of the gum tissue, usually caused by plaque deposits at the gum line.

My Genetic Risk Is Average For The Population

0.97 times

Average incidence rate is 60000 per 100,000 in the population

Suggestions

1. Develop good oral hygiene habits: brush your teeth at least twice a day, brush your teeth with a horizontal quiver brush, use fluorine toothpaste, floss and mouthwash, change your toothbrush about three months, and gargle after meals.
2. Go to the hospital to check the oral cavity regularly (twice a year) and perform cleansing (washing teeth). Cleansing effectively removes dental plaque or tartar deposited on and below the gums around the gum edge.
3. When the condition requires the use of drugs that cause excessive gum hyperplasia, the hyperplasiad gums can be surgically removed.
4. Smoking is prone to gingivitis and does not actively smoke or chew cigarettes.
5. To ensure a balanced nutrition, eat fruits and vegetables rich in vitamin C and niacin or supplements.

Influencing factors



Controllable factors

1. Oral hygiene: Bacterial groups (plaques) at the edge of the gums are the main cause of Gingivitis.
2. Other oral problems: calculus, defective prosthesis, crowded teeth, food impaction, etc. May increase inflammation of the gums.

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3. Drugs: some drugs (e.g. Phenytoin sodium, cyclosporine, nifedipine) can cause the growth of gum tissue, and gingivitis often occurs in combination.
 4. Smoking: smoking or chewing cigarettes can easily cause gingivitis.
 5. Vitamins: vitamin C or niacin deficiency can cause gingivitis.

Uncontrollable factors

1. Age: The prevalence and severity increased with age, peaked at adolescence, and decreased after 17 years of age.
2. Hormonal changes: Changes in hormone levels during pregnancy, menopause, puberty and menopause may increase inflammation of the gums.
3. Diseases: diabetes, aids, cancer, vitamin deficiency, leukemia, leukopenia, acute herpes simplex stomatitis, thrush and other diseases can affect the body's response to infection, thus causing gingivitis.

Knowledge

Onset time

It can occur from three years old to the elderly.

Prevalence

The prevalence of Chronic Gingivitis in the population ranges from 60%-90%, especially among children and adolescents, with prevalence and severity increased with age.

Typical symptoms

Swollen gums, bright red or reddish-purple gums, bleeding gums (when brushing or flossing), malodour, gingival recession, loose teeth, gum softening, etc.

Treatment

Regular oral hygiene, professional cleansing, adequate nutritional supply and the use of mouthwash can help treat gingivitis. Regular dental check-ups remove plaque that has accumulated and can harden into tartar, and cure the disease before the initial stage of gingivitis deteriorates into serious symptoms (resorption of alveolar bone, loosening of teeth, etc.).

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1800896	intergenic	TT	3.24	1.00
RS3918242	intergenic	CC	1.00	1.02
RS1800795	intergenic	GG	1.00	1.00

Risk calculation

The risk value of this test is calculated based on published academic literatures related to Gingivitis. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between the patients with Gingivitis and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Gingivitis risk item uses three polymorphic loci between genes, such as rs1800795, rs1800896, rs3918242. The test result indicates whether the user has a higher possibility of Gingivitis in genotype combination of the three polymorphic loci detected compared with all users, and the test does not represent the user's real risk of Gingivitis. The test is mainly based on the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of Gingivitis.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Gingivitis from the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If you have a family history of Gingivitis, or you think that you are likely to suffer from Gingivitis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes rs1800795, rs1800896, rs3918242, three polymorphic loci associated with the Gingivitis

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect Gingivitis.

Astigmatism

Astigmatism is a kind of eye refractive error disease that cannot see far and near objects.

My Genetic Risk Is Average For The Population

0.97 times

Average incidence rate is 58800 per 100,000 in the population

Suggestions

- How to reduce the risk:
 - Avoid squeezing the eyes to ensure that the cornea is not deformed due to external forces
 - Keep your eyes moist and increase blinking frequency. You can also use artificial tears to moisturize your eyes when they are dry
 - Protective food: Appropriate supplementation of vitamin A, vitamin B12, vitamin C, vitamin D, Omega-3, etc. contributes to eye health
 - When reading, you should ensure that you sit upright, do not squint, and do not read on your side. At the same time avoid reading in small print
 - Reduce blue light exposure: In the evening, you can turn on the eye protection mode of mobile phones or other electronic products, and you can wear anti-blue glasses at the same time
 - Avoid eye infections. Do not rub your eyes with your hands because of itching. At the same time, avoid foreign objects entering the eyes. If the foreign objects enter the eyes accidentally, please seek medical treatment immediately after washing.

* If you have a family history of astigmatism, or if you think you are likely to suffer from astigmatism, or need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors



Controllable factors

1. Squeezing factor: People who have bad habits that cause their eyes to be squeezed are more likely to suffer from astigmatism.
2. Eye factors: Excessive use of eyes, tired eyes, incorrect sitting posture for reading, and reading with small fonts can easily lead to astigmatism.

-
3. Light factor: Too much blue light received by the eyes will increase the risk of astigmatism.
 4. Infectious factors: Eye diseases caused by infection will increase the risk of astigmatism.

Uncontrollable factors

1. Age: Astigmatism is more common in middle-aged and elderly people. With age, the risk of astigmatism increases.
2. Family history: If there is a family history of astigmatism, the incidence of astigmatism in individuals with high astigmatism is higher than that of the general population.

Knowledge

Time of onset

With age, the incidence rate gradually increases. The incidence rate in the age group over 70 years old is more than 87%.

Prevalence

According to a survey on astigmatism in Singapore, 58.8% of people have varying degrees of astigmatism. The incidence rate increases with age. But in recent years, the incidence of astigmatism is increasing year by year.

Typical symptoms

- Visual acuity is decreased, and patients with moderate to high astigmatism have poor near and far vision.
- Prone to visual fatigue, eye pain, tearing, and headache after excessive use of the eyes, especially on the front forehead, and double vision.
- Like to squint or look sideways.

Treatment

Different cylindrical lenses can be used to correct astigmatism according to the type and degree of astigmatism. Convex cylindrical lens for hyperopic astigmatism and concave cylindrical lens for myopic astigmatism. Mild astigmatism can be corrected by wearing corrective glasses. For patients with high astigmatism, irregular astigmatism or those who cannot adapt to frame glasses, contact lenses can be used for correction. Patients with high astigmatism can also be treated with excimer laser refractive corneal surgery, laser thermal keratoplasty, keratotomy and other surgical methods.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS62075722	intergenic	GG	1.16	1.00
RS72629670	intergenic	AG	0.93	1.00
RS196052	intergenic	AA	0.96	1.00
RS12032649	intergenic	GT	1.02	1.00
RS7677751	PDGFRA	CC	1.00	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to astigmatism. These literatures have found some polymorphic loci related to the risk of the disease by studying the genetic differences between patients with astigmatism and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk item of astigmatism uses the 9 polymorphic loci on *HERC2*, *CASC15*, *PDGFRA* and other genes. The test results indicate that compared with all users, whether this user has a higher prevalence of astigmatism in the combination of genotypes of the 9 polymorphic loci tested. It does not mean the user's true risk of astigmatism. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Notification

-
1. The test may not cover all genes or loci that cause astigmatism.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of the disease is determined by multiple factors such as genes and environment.
This study only predicts your risk of astigmatism from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of astigmatism, or you think that you are likely to suffer from astigmatism, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 9 polymorphic loci related to the risk of astigmatism, including rs12913832, rs10946507, and rs7677751.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, and the test may not cover all genes or loci related to astigmatism.

Exfoliative Glaucoma

It develops from exfoliation syndrome (also known as pseudoexfoliation syndrome), and is an age-related systemic disease, with the features of fibrous stripped content widely deposit on the eye and other systemic organizations. It often leads to secondary open-angle glaucoma.

My Genetic Risk Is Average For The Population

0.96 times

Average incidence rate is 1000 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Age: The incidence of exfoliative glaucoma tends to increase with age.
2. Family history: Exfoliative glaucoma has a certain genetic tendency.

Knowledge



Time of onset

This disease is most common in people over the age of 45.



Prevalence

The incidence of the total population is 1000/100000, and the incidence of people over the age of 45 is 2000/100000.



Typical symptoms

In the early stage, there are generally no obvious symptoms, sometimes accompanied by eye swelling, eye pain; With the aggravation of the disease, visual field defects may occur..



Treatment

Oral medication, laser or surgical treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS3825942	LOXL1	GG	24.38	0.98
RS2107856	intergenic	GT	1.42	0.97
RS4926244	CACNA1A	TT	1.00	0.99

Risk calculation

The risk value of this test is calculated based on published academic literatures related to exfoliative glaucoma. These literatures have found some polymorphic sites associated with the risk of the disease by studying the genetic differences between patients with exfoliative glaucoma patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of exfoliative glaucoma item uses the 3 polymorphic loci on the *LOXL1*, *CACNA1A* and *CNTNAP2* genes. The test results indicate whether the user has a higher incidence of exfoliative glaucoma than all users in the genotype combination of the 3 polymorphic loci tested. This test does not represent the user's true risk of exfoliative glaucoma. The basis of the test is mainly from the research of Caucasian and Asian, other populations are for reference only.

Notification

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1. The test may not cover all genes or loci that could contribute to a risk of exfoliative glaucoma.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of exfoliative glaucoma at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of exfoliative glaucoma, or you think that you are likely to suffer from exfoliative glaucoma, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 3 polymorphic loci associated with exfoliative glaucoma risk, including rs3825942, rs4926244 and rs2107856.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to exfoliative glaucoma.

Obsessive-Compulsive Disorder (OCD)

It is a common chronic mental disorder characterized by recurring painful obsessive thoughts and compulsive behaviors.

My Genetic Risk Is Average For The Population

0.95 times

Average incidence rate is 1800 per 100,000 in the population

Suggestions

1. Choose a social environment with high tolerance and low life pressure for work and life, and maintain harmonious relations with relatives, friends and colleagues. Improve your ability to cope with psychological pressure and burden of life, so as not to induce disease.
2. If you have a compulsive personality tendency, you should learn to understand and strengthen cognitive correction, and work hard to correct your own personality defects on the premise of proficiency in psychological adjustment.

Influencing factors

Controllable factors

1. Psychosocial factors: Some behaviorists believe that the onset of obsessive-compulsive disorder is caused by excessive repetitive stimulation of psychosocial factors, which leads to anxiety and discomfort, which makes the central nervous system's excitability inhibition imbalance, leading to the formation of abnormal habits. These psychosocial factors include domestic violence, family environment intimacy, and parental rearing styles.
2. Personality factors: obsessive-compulsive personality or mental weakness are more likely to develop obsessive-compulsive disorder. Compulsive personality or mental weakness is mainly manifested as: trying to maintain close control of themselves and the environment, they pay attention to details, and strive to be accurate and perfect in everything, but even so, there are still "imperfect", "insecure" and "uncertain" feeling. Personality is often manifested as following the rules, lack of decision, indecision, and reliance on obedience; or manifested as stubbornness,

sticking in the mud, preferring to bend and quick temper, which leads to the occurrence of obsessive-compulsive disorder.

Uncontrollable factors

1. Age: Obsessive-compulsive disorder usually develops in early childhood or adolescence.
2. Neuro-endocrine factors: imbalance of the neurotransmitters serotonin and dopamine may lead to the occurrence of obsessive-compulsive disorder.
3. Family history: People with a family history of obsessive-compulsive disorder have a higher prevalence.

Knowledge

Time of onset

Obsessive-compulsive disorder usually develops in early childhood or adolescence, and about two-thirds of obsessive-compulsive disorder cases occur before the age of 25.

Prevalence

Prevalence rate: 1800 per 100,000 people. A global disease survey conducted by the World Health Organization (WHO) found that obsessive-compulsive disorder has become one of the 20 diseases that cause the heaviest burden of disease among young and middle-aged people aged 15-44.

Typical symptoms

Obsessive-compulsive disorder is a common chronic disorder, which is characterized by recurring painful obsessions and compulsions. Obsessive-compulsive disorder consists of two components: obsessive thinking and compulsive behavior. Obsessive thinking is defined as periodic continued thoughts, images or impulses, not just cognitive events, including not help thinking again of some unnecessary problems, have some to do things against their will impulses; compulsive behavior is repetitive behaviors or mental rituals, therefore, compulsive behavior may be cognitive events. The key difference between thinking and compulsive behavior is compulsive obsessions cause anxiety or pain, compulsive behavior and the purpose is to prevent or reduce the pain or some terrible things.

Treatment

1. Psychotherapy: Psychotherapy is necessary and beneficial, including cognitive behavioral therapy (such as exposure response therapy (ERP) and cognitive therapy, etc.), psychodynamic therapy, supportive psychotherapy, and Morita therapy. However, each type of psychotherapy has its own limitations. The treatment of obsessive-compulsive disorder can only improve the symptoms, but cannot solve the problem fundamentally.

2. Medication: If the condition is serious, scientific medication can be carried out as soon as possible. The first-line anti-compulsive drugs currently used are the same as antidepressants, and their characteristic is that they can regulate the function of neurotransmitters such as serotonin in the brain. Including paroxetine, fluvoxamine, fluoxetine, citalan, sertraline, etc., generally need to be taken for at least 1 to 2 years to consolidate the efficacy.
3. Physiotherapy: Physiotherapy is also a candidate for the treatment of obsessive-compulsive disorder. For patients with refractory obsessive-compulsive disorder, when neither drug therapy nor psychotherapy can achieve satisfactory results, modified electrical therapy can be selectively used according to the specific situation. Shock therapy or transcranial magnetic stimulation therapy, these two common physical therapy methods in psychiatry, also have a certain effect on refractory obsessive-compulsive disorder. Neurosurgery is regarded as the last option for the treatment of obsessive-compulsive disorder. Because of its serious and irreversible side effects such as seizures and loss of sensation, it is necessary to strictly grasp the indications for surgery. It should be consulted by three psychiatric chief physicians to consider whether to implement surgical treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS4565946	TPH2	CT	1.78	0.95
RS25531	intergenic	TT	1.00	0.93

Risk calculation

The risk value of this test is calculated based on published academic literatures related to obsessive-compulsive disorder. These literatures have found some polymorphic loci related to the risk of the disease by studying patients with obsessive-compulsive disorder and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of obsessive-compulsive disorder item uses the 2 polymorphic loci in *TPH2* and *SLC6A4*. The test results indicate whether the user has a higher prevalence of obsessive-compulsive disorder in the combination of genotypes at the 2 polymorphic loci tested compared to all users. This test does not represent the user's true risk of obsessive-compulsive disorder. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of obsessive-compulsive disorder.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of obsessive-compulsive disorder at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of obsessive-compulsive disorder, or you think that you are likely to suffer from obsessive-compulsive disorder, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 2 polymorphic loci related to the risk of obsessive-compulsive disorder, including rs4565946 and rs25531.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to obsessive-compulsive disorder.

Celiac Disease

It is an immune-mediated systemic disease triggered by the ingestion of gluten in individuals with genetic susceptibility.

My Genetic Risk Is Average For The Population

0.94 times

Average incidence rate is 1000 per 100,000 in the population

Suggestions

Celiac disease can be avoided by avoiding foods that contain gluten.

Influencing factors



Controllable factors

1. Gluten intake: Gluten is a protein found in wheat, barley and rye that is essential for celiac disease.



Uncontrollable factors

1. Age: Celiac disease peaks in children and young adults, but in recent years it has also increased in older adults.
2. Gender: Celiac disease is slightly more common in women than men.
3. Family history: At the time of diagnosis, both first degree relatives and second degree relatives had a higher than average prevalence of celiac disease.
4. Other autoimmune diseases: People with autoimmune diseases, such as type I diabetes and autoimmune thyroid disease and so on, are at greater risk of developing other autoimmune diseases. People with celiac disease have an increased risk of developing other autoimmune diseases.

Knowledge

Time of onset

Any age can be affected, mainly in children and teenagers, the incidence of the elderly has a rising trend.

Prevalence

It affects about 1% of the world's population.

Typical symptoms

Recurrent abdominal distention, abdominal pain, chronic diarrhea, and fecal matter is often pale and smelly; Weight loss, fatigue, early childhood onset can lead to dysplasia or microsomia, delayed puberty; Elevated transaminase, joint pain, osteoporosis; A few people also have neuropathy, symptoms of mental disorders such as depression and anxiety.

Treatment

At present, the only effective treatment has been a lifelong gluten-free diet. There are no other medicines that can prevent intestinal damage if gluten is consumed. A strict gluten-free diet completely alleviates intestinal symptoms in most cases, and may also alleviate other symptoms such as osteoporosis.

Celiac disease, gluten allergy and non-celiac gluten sensitivity (NCGS)

- Celiac disease is a systemic autoimmune disease, in which the body's immune response to its own antigens causes systemic damage to its own tissues.
- Gluten allergy is an allergic reaction, in which the body's immune system reacts to a foreign substance (an allergen) without causing damage to the body's own tissue.
- NCGS does not involve an allergy or an autoimmune response, but its symptoms are similar to those of celiac disease and can be an inflammation of the gut caused by other substances in wheat products.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS917997	intergenic	TT	1.42	0.99
RS1738074	TAGAP	TT	1.35	0.99
RS1464510	LPP	AC	1.29	0.99

RS2187668	HLA-DQA1	CC	1.00	0.76
RS13098911	intergenic	CT	1.30	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to celiac disease. These literatures have found some polymorphic loci associated with the risk of celiac disease by examining genotypic differences between patients with and without celiac disease. We comprehensively score the polymorphic loci involved in this test through the original data in these literatures and obtains the risk values of all subjects. By comparing with the average risk of all subjects tested in our products, we can give the risk multiple of subjects relative to the average risk of the population, and obtain the distribution of the risk of the population. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The celiac disease health risk item uses 34 polymorphic loci on *HLA-DQA1*, *LPP*, *MYO9B*, *PUS10*, *BACH2* and other genes. The test results indicated whether the subject had a higher prevalence of celiac disease compared to all subjects at 34 polymorphic loci tested. The test does not represent a subject's true risk for celiac disease. The basis of the test is mainly from the research of Caucasian and Argentinean, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of celiac disease.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The development of the disease is determined by a combination of genetic and environmental factors. This test only predicts the risk of celiac disease from the genetic level, and it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc..
3. If you have a family history of celiac disease, or you think that you are likely to suffer from celiac disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 34 mutation related to celiac disease, including rs2187668, rs9851967 and rs2305767.

Population samples

The basis of the test is mainly from the research of Caucasian and Argentinean.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect celiac disease.

Bladder Cancer

Bladder cancer is a malignant tumor on the bladder mucosa, which is formed due to the uncontrolled growth of bladder cells.

My Genetic Risk Is Average For The Population

0.94 times

Average incidence rate is 4 per 100,000 in the population

Suggestions

1. Develop healthy dietary habits: Drink more water, eat more vegetables and fruits. Studies have shown that it can reduce the risk of many types of cancer and is beneficial to health.
2. Control unhealthy living habits: Quit smoking and alcohol.
3. Avoid contact with toxic substances: Employees in the rubber, leather, and printing industries, as well as those in the textile and paint industries, pay attention to occupational protection and avoid contact with toxic substances.

Influencing factors



Controllable factors

1. Dietary habits: Insufficient drinking water and insufficient intake of fruits and vegetables may induce bladder cancer.
2. Living habits: Smoking and drinking alcohol may induce bladder cancer.
3. Toxic substances: Frequent contact with rubber and leather may induce bladder cancer; practitioners in printing, textile, paint and other industries are at high risk of bladder cancer.



Uncontrollable factors

1. Age: Common in 50 years and above.
2. Gender: Mostly male.

Knowledge

Time of onset

The onset is more common in the age of 50 and above, and it is more likely to be onset with age.

Prevalence

In 2017, there were approximately 79,030 cases of bladder cancer in the United States. In 2015, there were approximately 80,500 cases of bladder cancer in China. The overall incidence rate in China from 2003 to 2007 was 6.69 per 100,000 people. There are obvious gender differences in the incidence of bladder cancer. The global incidence of bladder cancer in men is about 4 times that of women.

Typical symptoms

- Hematuria: Usually, the first symptom of bladder cancer is blood in the urine. Blood in the urine may be pink or crimson urine, or the color of the urine under the naked eye is normal, but routine urine tests show an increase in red blood cells. Early bladder cancer with hematuria occurs, usually without other discomforts such as painful urination.
- Changes in urination habits: Including frequent urination (significant increase in the frequency of urination), urgency (repeated wanting to urinate), painful urination (pain when urinating), poor urination, increased nocturia, etc.
- Symptoms caused by cancer cell metastasis: Inability to urinate, unilateral low back pain, poor appetite, significant weight loss, fatigue, bone pain, etc.

Treatment

1. Surgical treatment: The vast majority of bladder cancer can be treated with surgery, and the choice of surgery depends on the different stages of bladder cancer. Commonly used types of surgery include: transurethral resection of bladder tumor, cystectomy.
2. Infusion therapy: The doctor injects the drug into the bladder through a catheter, and the drug will stay in the bladder for about 2 hours. This therapy is not suitable for advanced bladder cancer.
3. Radiotherapy: Use high-energy rays to kill cancer cells.
4. Chemotherapy: The use of drugs to kill cancer cells or prevent the growth of cancer cells. Commonly used drugs include: cisplatin, fluorouracil, docetaxel, paclitaxel, methotrexate, vincristine and so on.
5. Immunotherapy: By influencing the patient's own immune system, it can more effectively identify and kill cancer cells. Commonly used immunotherapy includes: BCG, immune checkpoint inhibitors, etc.
6. Targeted therapy: Targeting cancer-causing mutations, such as the targeted drug Erdafitinib and so on.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2294008	PSCA	CT	1.30	0.99
RS1495741	intergenic	GG	0.76	1.00
RS710521	intergenic	CT	1.18	1.00
RS1058396	SLC14A1	AG	1.14	1.00
RS9642880	CASC11	GG	1.00	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to bladder cancer. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype difference between bladder cancer patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk item of bladder cancer uses the 11 polymorphic loci on genes such as *SLC14A1*, *TACC3*, *PSCA*, *MYNN*, *CASC11*. The test results indicate that compared with all users, whether the combination of genotypes at the 11 polymorphic loci detected by this test increases the risk of bladder cancer. It does not mean the user's true risk of bladder cancer. The basis of the test is mainly from the research of European, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that cause the risk of bladder cancer.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only evaluates your risk of bladder cancer at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of bladder cancer, or you think that you are likely to suffer from bladder cancer, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 11 polymorphic loci associated with bladder cancer, including rs10775480, rs1014971, and rs8102137.

Population samples

The basis of the test is mainly from the research of European.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to bladder cancer.

Diverticulosis

Diverticulosis refers to the outward cystic protrusion of any part of the gastrointestinal tract. It usually occurs in the large intestine, especially the sigmoid colon.

My Genetic Risk Is Average For The Population

0.93 times

Average incidence rate is 5000 per 100,000 in the population

Suggestions

1. Increase dietary fiber intake: Eating a diet high in fiber can prevent diverticulosis. Food supplements, such as flaxseed, can be used to increase fiber in food, promote intestinal movement, and reduce intestinal pressure.
2. Improve lifestyle habits: Eat regularly, actively participate in exercise, rest fully, and control stress.
3. Smoking and drinking: Avoid smoking and drinking.

Influencing factors



Controllable factors

1. Lack of fiber in the diet: Lack of fiber in the diet can cause high pressure in the intestinal cavity, which can cause the mucosal protrusion and diverticulosis.
2. Poor living habits: Lack of exercise, irregular work and rest schedules, and excessive mental stress are also related to the occurrence of diverticulosis.
3. Smoking and drinking: Research has shown that smoking and drinking affect colon motility and pressure in the colon, which can induce the formation of diverticulosis.



Uncontrollable factors

1. Age: This disease is more common in people over 60 years old, and the incidence increases with age.

2. Race: The incidence of diverticulosis is high in Europe and North America, and is relatively lower in Africa and Asia.

Knowledge

 Onset time

This disease is more common in people over 60 years old, with only 2-5% of diverticulosis occurring in people under 40 years old.

 Prevalence

Diverticulosis has always been considered a more prevalent disease in Western countries, with the highest incidence in the Americas, Europe, and Australia. Traditionally, diverticulosis is considered a disease that affects the elderly. By the age of 85, the incidence of diverticulosis can reach as high as 65%, while it can be as low as around 5% in people aged 40 and below.

 Typical symptoms

Most of people with diverticulosis have no symptoms. Symptoms that may occur in some patients include abdominal pain, diarrhea, and abnormal bowel movement.

 Treatment

- 1. Medication: Infection, inflammation, need to be treated with antibiotics.
- 2. Surgical treatment: Patients with bleeding should use endoscopy to find the bleeding location, and stop the bleeding in a timely manner. Severe bleeding requires urgent surgery. The occurrence of a large diverticular infection and perforation requires surgical treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS111316530	intergenic	II	1.35	1.00
RS4333882	SLC35F3	GG	1.27	1.00
RS61814883	intergenic	GG	0.79	1.00
RS6734367	ARHGAP15	GG	0.80	1.00

RS9520339

FAM155A

CC

0.81

1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to diverticulosis. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between the patients with diverticulosis and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details



Applicable situation

The diverticulosis risk item uses 49 polymorphic loci on genes such as *BMPRI1B*, *BABAM2* and *FBXL13*. The test result indicates whether the user has a higher possibility of diverticulosis in genotype combination of the 49 polymorphic loci detected compared with all users, and the test does not represent the user's real risk of diverticulosis. The test is mainly based on the research of Caucasian, other populations are for reference only.



Notification

1. The test may not cover all disease-causing genes or sites of diverticulosis.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results



Notification

1. The test is not suitable for clinical diagnosis.

-
2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of diverticulosis from the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
 3. If you have a family history of diverticulosis, or you think that you are likely to suffer from diverticulosis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 49 polymorphic loci associated with diverticulosis risk, such as rs101203333, rs10173528.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with diverticulosis.

Attention Deficit Hyperactivity Disorder (ADHD)

The main clinical manifestations of attention deficit hyperactivity disorder are obvious inattention and a short duration of attention, excessive activity and impulsiveness, which affect learning efficiency and interpersonal communication.

My Genetic Risk Is Average For The Population

0.92 times

Average incidence rate is 10000 per 100,000 in the population

Suggestions

1. Create a good family atmosphere, avoid quarreling in front of your children, don't insult or beat your children, and communicate with them more often.
2. In the early pregnancy, avoid go to crowded places, keep warm; during pregnancy, do not smoke, drink alcohol or take drugs.

Influencing factors



Controllable factors

1. Family: parental discord, improper parenting style, poor parental personality (such as depression in the mother, impulsivity in the father, anti-social behavior, etc.), separation from the parents in childhood, and abuse, all of which may be the cause of the disease or persistence of symptoms.
2. Pregnancy factors: early pregnancy infection, smoking, drinking and drug use during pregnancy.



Uncontrollable factors

1. Biological factors: Nervous system development disorders, neurotransmitter system disorders.
2. Physical diseases: epilepsy, thyroid abnormalities.
3. Age: The age of onset of most attention deficit hyperactivity disorder is 3-4 years old.

Knowledge

Time of onset

The age of onset of most attention deficit hyperactivity disorder is 3 to 4 years old, but there is a lot of evidence that this disease is continuous, and about 10% to 60% of patients will continue into adolescence and adulthood.

Prevalence

The international prevalence of attention deficit hyperactivity disorder is 6-8% for children and 3-5% for adults. According to the latest 2016 epidemiological survey in mainland China, the prevalence of attention deficit hyperactivity disorder among children and adolescents in mainland China is 6.26%.

Typical symptoms

1. Inability to concentrate: Loss of concentration, especially inability to concentrate on boring things.
2. Hyperactivity: Like to walk around, talkative, and look energetic.
3. Impulsive: Likes to provoke others and is nosy.

Treatment

Attention deficit hyperactivity disorder treatment methods include behavior therapy, drug therapy, education therapy and other methods.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1801260	intergenic	AA	2.34	0.99
RS9677504	intergenic	GG	1.26	1.00
RS11591402	intergenic	TT	0.83	1.00
RS6032660	intergenic	AG	0.92	1.00
RS6686722	intergenic	CC	1.00	0.97

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to attention deficit hyperactivity disorder. These literatures have found some polymorphic loci related to the risk of the disease by studying patients with attention deficit hyperactivity disorder and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of attention deficit hyperactivity disorder item uses 14 genetic and intergenic polymorphic loci of genes such as *FOXP2*, *IP6K1*. The test results indicate whether the user has a higher prevalence of attention deficit hyperactivity disorder in the combination of genotypes at the 14 polymorphic loci tested compared to all users. This test does not represent the user's true risk of attention deficit hyperactivity disorder. The basis of the test is mainly from the research of European and Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of attention deficit hyperactivity disorder.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of attention deficit hyperactivity disorder at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
-

3. If you have a family history of attention deficit hyperactivity disorder, or you think that you are likely to suffer from attention deficit hyperactivity disorder, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 14 polymorphic loci related to the risk of attention deficit hyperactivity disorder, including rs1801260, rs1427829, rs11420276, etc.

 Population samples

The basis of the test is mainly from the research of European and Asian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to attention deficit hyperactivity disorder.

Developmental Dyslexia

Developmental dyslexia refers to the obvious developmental impairment of children's word recognition skills and reading comprehension from the early stage of development. This disorder is not caused by lack of learning opportunities, mental retardation, acquired brain injury or disease.

My Genetic Risk Is Average For The Population

0.92 times

Average incidence rate is 8000 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Age: usually onset in infancy or childhood.
2. Gender: boys are more likely to develop developmental dyslexia.
3. Writing system: the type of text presentation in the writing system has a certain impact on dyslexia. Children who use ideographic characters as their mother tongue (such as English) have a higher incidence of developmental dyslexia than school-age children who use ideograms as their mother tongue (such as Chinese).
4. Social stratification: research has found that children with low social stratification have a higher prevalence of developmental dyslexia.
5. Family medical history: studies have found that 40% of children with dyslexia have parents who also have dyslexia. And fathers have a higher rate of dyslexia. The study of twins also shows that the same disease rate of identical twins is higher than that of fraternal twins, with a reported ratio of 87/24.

Knowledge



Time of onset

It usually starts in infancy or childhood, and it is obvious at 6 to 7 years of age. Most dyslexia is only discovered when learning problems occur in kindergarten or first grade.

 Prevalence

According to a systematic evaluation and meta-analysis study on the prevalence of developmental dyslexia among primary school students in 2022, the prevalence of developmental dyslexia among primary school students worldwide is 7.10%, and boys are more prevalent than girls.

 Typical symptoms

Preschool children with developmental dyslexia can manifest as: language development delay, dysarthria, difficulty in remembering letters, obstacles in counting and recognizing colors. Delays and hesitation in choosing words (disorders of word discovery), alternative words, naming letters and pictures are often early symptoms of dyslexia. Common short-term (working) auditory memory and auditory sorting problems. Many people with dyslexia confuse similar letters and words or visual choices, font recognition, and recognition of consonants and vowels in vocabulary. Reversal or visual confusion often occurs in children in lower grades of elementary school. Most of the reversal of reading and writing occurs because people with dyslexia forget or confuse the names of similarly structured letters and words when recalling.

 Treatment

The following interventions are generally adopted: through direct guidance or indirect guidance (including basic phonetic teaching, word recognition guidance, decoding or reading comprehension) patients reading, and some compensatory methods (such as the use of recorded texts, reader and scribe) to help patients improve their reading ability. In mild cases, reading will gradually catch up after treatment, and there will be no signs of dyslexia in adulthood. In severe cases, despite treatment, many signs of the disorder will continue for life.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS16928927	intergenic	CC	0.45	0.99
RS17663182	MIR924HG	GG	2.03	1.00
RS2274305	DCDC2	CT	0.75	0.99
RS2255526	intergenic	AG	1.30	0.99

RS761100

KIAA0319

AC

1.25

1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is calculated based on published academic literature on developmental dyslexia. By studying the genotype differences between developmental dyslexia patients and normal people, these literatures found some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of developmental dyslexia item uses 34 polymorphic loci on genes such as *KIAA0319*, *DCDC2*, *DRD2* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 34 polymorphic loci detected by this test increases the risk of developmental dyslexia. It does not mean the user's true risk of developmental dyslexia. The basis of the test is mainly from the research of Chinese and Caucasian, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of developmental dyslexia.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

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1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developmental dyslexia at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
 3. If you have a family history of developmental dyslexia, or if you feel you have a risk of developmental dyslexia, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 34 polymorphic loci associated with the risk of developmental dyslexia, including rs761100, rs2255526, etc.

Population samples

The study used in this test is mainly Chinese and Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with developmental dyslexia.

Uterine Fibroids

It is a benign tumor formed by proliferating uterine smooth tissue and a small amount of connective tissue. It is the most common tumor in female reproductive organs.

My Genetic Risk Is Average For The Population

0.90 times

Average incidence rate is 30000 per 100,000 in the population

Suggestions

1. Eat a balanced diet, eat more vegetables and fruits in the diet, and maintain a normal vitamin D intake (10 µg/day).
2. Try to avoid drugs or health products containing hormones, such as contraceptives.

Influencing factors

Controllable factors

1. Diet: Ingesting more fruits and vegetables in your diet can reduce the risk of uterine fibroids. The role of fruits is more obvious than that of vegetables, and research has found that the protective effect of citrus is more obvious. Studies have shown that vitamin D in the daily diet can reduce the risk of uterine fibroids.
2. Hormonal factors: estrogen is the main factor that promotes the growth of fibroids. Fibroids rarely occur before puberty. Fibroids grow up with pregnancy and shrink after menopause; progesterone, progesterone and progesterone receptors contribute to the formation of fibroids and promote the proliferation of fibroids. If there are uterine fibroids, fibroids will increase during pregnancy; growth hormone may promote and maintain the growth of fibroids. In patients with acromegaly, the incidence of uterine fibroids is higher.

Uncontrollable factors

-
1. Age: The incidence of uterine fibroids increases with age.
 2. Family history: People with a family history of uterine fibroids have a higher prevalence.
-

Knowledge

Time of onset

Clinically, fibroids rarely occur before puberty, and generally occur after women reach the reproductive age, and are more common in women between 30 and 50 years old. As women who give birth increase with age, the incidence of uterine fibroids is higher. After reaching the age of menopause, the incidence of uterine fibroids decreases.

Prevalence

Prevalence rate: 30,000/100,000. The incidence of uterine fibroids is different in different regions and races. Women of African origin have 3 to 9 times higher incidence of uterine fibroids than women of other races, and Blacks are also earlier than Whites at the age of peak incidence.

Typical symptoms

Some fibroids are small, slow-growing, and asymptomatic, and can go undetected for life. In recent years, due to the widespread use of B-mode ultrasound examination, many patients have been found to have uterine fibroids by B-ultrasound due to routine physical examinations, but they have no symptoms. Among the symptoms of uterine fibroids, uterine bleeding, abdominal masses, vaginal discharge, and compression symptoms are common. If uterine fibroids compress to the bladder, frequent urination may occur; other symptoms include pain during intercourse or lower back pain, and the patient may experience one or more symptoms. In rare cases, uterine fibroids may cause infertility.

Treatment

Asymptomatic uterine fibroids generally do not require treatment. If you have mild symptoms, you can treat it with ibuprofen or acetamidophenol. If you have menstrual periods, you need to add iron. Drugs such as gonadal hormone releasing hormone inhibitors may shrink fibroids, but they are expensive and have side effects. If you have more obvious symptoms, you can consider removing uterine fibroids by hysterectomy or total hysterectomy. It can also be treated with uterine artery embolization or high-intensity focusing technology (also known as HIFU).

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2280543	BET1L	CT	0.88	1.00
RS12484776	TNRC6B	AA	1.00	1.00
RS7913069	intergenic	CC	1.00	1.00

Risk calculation

The risk value of this test is calculated based on published academic literatures related to uterine fibroids. These literatures have found some polymorphic loci related to the risk of the disease by studying patients with uterine fibroids and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of uterine fibroids item uses 3 polymorphic loci in and between genes of *TNRC6B* and *BET1L*. The test results indicate whether the user has a higher prevalence of uterine fibroids in the combination of genotypes at the 3 polymorphic loci tested compared to all users. This test does not represent the user's true risk of uterine fibroids. The basis of the test is mainly from the research of African and African descent, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of uterine fibroids.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of uterine fibroids at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of uterine fibroids, or you think that you are likely to suffer from uterine fibroids, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 3 polymorphic loci related to the risk of uterine fibroids, including rs12484776, rs2280543 and rs7913069.

Population samples

The basis of the test is mainly from the research of African and African descent.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to uterine fibroids.

Essential Tremor

Essential tremor is one of the most common movement disorders, which mainly manifests as tremors of the hands, head and other postures and movements.

My Genetic Risk Is Average For The Population

0.89 times

Average incidence rate is 900 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Family history: essential tremor is also called familial tremor. About 30-70% of patients have a family history.
2. Age: the incidence rate increases with age. The incidence rate in people over 65 years old is 4.6%.

Knowledge



Time of onset

Essential tremor can develop at any age.



Prevalence

The incidence of essential tremor in the general population is 0.9%, and it increases with age.



Typical symptoms

The only symptom is tremor, with occasional reports of intonation and slight gait abnormalities. The patient usually starts with the upper limbs and mainly affects the upper limbs. It can also affect the head, legs, trunk, vocalization and facial muscles. It is manifested as postural tremor, which can contain components of exercise, intentional, or static tremor at the same time.



Treatment

Usually drugs can effectively control symptoms; if the drug treatment is not effective, surgery can be tried.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS9652490	LINGO1	AG	2.13	0.99
RS1127215	PTGFRN	CC	1.19	1.00
RS7903491	intergenic	AG	1.16	0.99
RS1945016	intergenic	GT	1.10	1.00
RS1143633	IL1B	TT	1.00	0.92

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is calculated based on published academic literature on essential tremor. By studying the genotype differences between essential tremor patients and normal people, these literatures found some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details



Applicable situation

The essential tremor item uses 8 polymorphic loci of between the *LINGO1*, *IL1B*, *PPARGC1A*, *STK32B*, *CTNNA3*, etc. gene. The results show whether the user had a higher rate of essential tremor than the

genotype combination of the 8 polymorphic locus tested compared to all users, and that the test did not represent the user's real risk of essential tremor. The basis of the test is mainly from the research of Caucasian, Asian, and North American, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of essential tremor.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of essential tremor at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
3. If you have a family history of essential tremor, or if you feel you have a risk of essential tremor, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 8 polymorphic loci which related to the risk of essential tremor including rs9652490, rs10937625, and rs7903491.

Population samples

The study used in this test is mainly Caucasian, Asian, and North American.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with essential tremor.

Anorexia Nervosa

Anorexia nervosa is a chronic eating disorder. Psychological factors and genetic susceptibility may increase the risk of anorexia nervosa in some people.

My Genetic Risk Is Average For The Population

0.88 times

Average incidence rate is 500 per 100,000 in the population

Suggestions

1. Reshape the correct values: improve the cognition of one's body image, reduce the drive to pursue "beauty with thinness", and increase the acceptance of one's body image.
2. Improve the level of self-esteem and self-confidence: you can cultivate hobbies and learn new skills. New hobbies and skills can divert attention from food and increase self-confidence. Learning new skills, developing new hobbies, volunteering, raising a small animal, etc. can all be effective.
3. Improve family relationship: If the family has children with anorexia nervosa, parents need to work hard to improve the family atmosphere, to tolerate and accept the children. Parents should give children a strong family support system to resist bad mental and spiritual problems.

Influencing factors



Controllable factors

1. Psychological factors: Misunderstandings about food, dieting and weight lead to patients' dissatisfaction with their own body. If this long-term psychological distress cannot be relieved, it may lead to abnormal eating behaviors.
2. Personality traits: Studies have found that the common personality characteristics of patients with anorexia nervosa are high neuroticism, low self-orientation, and perfectionism.
3. Family factors: family communication styles, member relationships, parental harmony in marriage, parental attitudes and methods of disciplining their children, and parents' perceptions

of their own body will all have an impact on the formation of children's anorexia.

Uncontrollable factors

1. Age: Commonly seen in teens.
2. Gender: Most patients are women.
3. Family history: The incidence of depression, anxiety disorder, substance abuse disorder and dependence, eating disorder and obesity in anorexia nervosa family is higher than that in the general population.
4. Social factors: more common in westernized, modernized and urbanized societies.

Knowledge

Time of onset

Eating disorders frequently appear during the teen years or young adulthood, and people rarely develop them during childhood or later in life.

Prevalence

Western countries estimate that 0.9-4.3% of women and 0.2-0.3% of men have had anorexia nervosa at some point in their lives. There is still a lack of nationwide epidemiological surveys on eating disorders in China; many surveys use self-assessment questionnaires for estimation, the data may be biased.

Typical symptoms

-Restriction of food intake leads to significant weight loss. In severe cases, it may cause symptoms such as malnutrition and water and electrolyte disorders. -Although they are already very thin, they are still worried about getting fat. -Denies being underweight. -Passionate about researching food and analyzing the calories of food. -Hoarding, hiding or discarding food, preparing fine food for others. -Pretend to eat or deceive the real food intake. -Exercising overload, wearing loose or multi-layered clothing. -Weigh the weight several times a day. -Self-esteem is hit by not being thin enough.

Treatment

Including restoring a healthy weight, treating potential psychological problems, and solving the inertial behaviors that cause anorexia. Although drugs cannot help gain weight, they can help treat related anxiety or depression. Effective treatments include: letting parents be responsible for feeding their children and cognitive behavioral therapy. Some patients need to be hospitalized to regain their weight.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS370838138	intergenic	GG	1.17	1.00
RS2287348	ERLEC1	CT	1.11	1.00
RS9821797	intergenic	TT	1.00	1.00
RS13100344	intergenic	AA	1.00	1.00
RS10747478	intergenic	GG	1.00	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to anorexia nervosa. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between subjects with anorexia nervosa and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of anorexia nervosa item uses the 6 polymorphic loci on and between genes such as *MGMT*, *EERLEC1*, ect. The test results indicate that compared with all users, whether the genotype combination at the 6 polymorphic loci tested has a greater risk of suffering from anorexia nervosa, but it does not represent the user's risk of anorexia nervosa from all dimensions. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect anorexia nervosa.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This test only evaluates your risk of anorexia nervosa at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If your family has a history of anorexia nervosa, or you feel that you are at risk of suffering from anorexia nervosa, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 6 polymorphic loci related to anorexia nervosa, such as rs10747478.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect anorexia nervosa.

Esophageal Cancer

Esophageal cancer is a common cancer of the gastrointestinal tract.

My Genetic Risk Is Average For The Population

0.87 times

Average incidence rate is 16 per 100,000 in the population

Suggestions

1. Nitrosamine is closely related to esophageal cancer. Eat less or no pickled foods such as salted fish and pickles.
 2. Mold can enhance the carcinogenic effect of nitrosamines, so avoid eating moldy food, such as rice, peanuts, etc.
 3. Malnutrition is related to esophageal cancer. It is recommended to eat 200-350g fresh fruits and 300-500g vegetables every day to supplement enough vitamins and trace elements.
 4. Smoking and drinking can induce esophageal cancer. It is recommended to quit smoking and drinking.
 5. Avoid chewing rough and hard food, such as betel nut and cut tobacco; It is better to drink tea and porridge below 50 °C.
-

Influencing factors



Controllable factors

1. Nitrosamine: Eating salted fish, pickles and other high nitrite foods will increase the risk of esophageal cancer.
2. Fungi: A variety of fungi can be isolated from the upper gastrointestinal tract of patients with esophageal cancer or resected esophageal cancer specimens. The investigation shows that there is a certain relationship between eating moldy food and the incidence of esophageal cancer.

-
3. Nutritional deficiency: Epidemiological evidence shows that the lack of multiple vitamins (such as vitamins A, B2 and C) or some trace elements (including molybdenum, zinc and selenium) in the diet is related to the incidence of esophageal cancer.
 4. Smoking and drinking: long term smoking can induce esophageal cancer. At the same time, the incidence of esophageal cancer in regular heavy drinkers is significantly higher than that in the general population.
 5. Eating habits: Long-term consumption of rough and hard food or eating too fast or too hot, etc., is easy to cause mechanical damage to esophageal mucosa. Repeated stimulation can lead to mucosal epithelial hyperplasia and eventually cancer.

Uncontrollable factors

1. Family history: The occurrence of esophageal cancer has a more obvious phenomenon of family aggregation.
2. Age: Most of the age of onset is over 40 years old.
3. Gender: Male majority.

Knowledge

Time of onset

The age of onset is more than 40 years old.

Prevalence

The incidence rate of esophageal cancer varies greatly among countries. China is a high incidence area of esophageal cancer. Henan has the highest incidence in the world.

Typical symptoms

1. Early symptoms: The main characteristics are burning sensation behind the sternum, acupuncture like or traction like pain, slow food passage, retention feeling or slight choking feeling. Early symptoms may be mild or severe, or even asymptomatic.
2. Middle and late stage symptoms: progressive dysphagia (first difficult to swallow dry food, then semi liquid food, and finally difficult to swallow water and saliva), food reflux, swallowing pain, accompanied by weight loss, enlarged lymph nodes, hoarseness, etc.

Treatment

Surgical treatment, radiation therapy, chemotherapy.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2274223	PLCE1	AG	1.29	0.99
RS2074356	HECTD4	GG	1.00	0.98
RS130079	CCHCR1	CC	1.00	0.99
RS1041981	LTA	CC	1.00	0.99
RS204900	TNXB	AA	1.00	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to esophageal cancer. These literatures have found some polymorphic loci related to the risk of the disease by esophageal cancer. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of esophageal cancer item uses the 8 polymorphic loci of *PLCE1*, *SLC39A6*, *HECTD4*, *CCHCR1*, *TCN2*, *TNXB*, *LTA*, *CYP26B1* gene. The test results show whether the user has a higher prevalence of esophageal cancer than all users. The test cannot represent the user's real risk of esophageal cancer. The test is mainly based on the research of Chinese, and other populations are only for reference.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of esophageal cancer.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of esophageal cancer from the genetic level, which does not represent your real health status, nor does it involve the judgment of your other health status, living habits, etc.
3. If you have a family history of esophageal cancer, or you think that you are likely to suffer from esophageal cancer, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 8 polymorphic loci related to esophageal cancer risk, including rs1050631, rs2274223, rs2074356, rs130079 and so on.

Population samples

The basis of the test is mainly from the research of Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect esophageal cancer.

Preeclampsia

It is a syndrome unique to pregnancy and is the main obstetric complication leading to perinatal death. It is often accompanied by high blood pressure, proteinuria, edema and other characteristics.

My Genetic Risk Is Average For The Population

0.87 times

Average incidence rate is 8000 per 100,000 in the population

Suggestions

1. Early marriage and early childbearing, the childbearing age should not exceed 40 years old as far as possible.
2. Maintain a healthy weight: keep BMI between 18.5 and 23.9.
3. Pay attention to personal health and nutritional supplements. Studies have shown that a daily intake of no less than 85 mg of vitamin C can reduce the occurrence of preeclampsia.

Influencing factors

Controllable factors

1. Age: pregnant women aged ≥ 40 years old have an increased risk of disease.
2. Overweight pregnant women with a BMI of 35.0 or more at the initial check-up have an increased risk of disease.
3. Pregnant women with malnutrition have an increased risk of illness, such as anemia, hypoproteinemia, etc.

Uncontrollable factors

1. People with positive antiphospholipid antibodies have an increased risk of disease.
2. People with a history of disease are at increased risk, such as hypertension, chronic nephritis, diabetes, autoimmune diseases, etc.

- 3. Genetic factors: people who have a history of preeclampsia or a family history (mother or sister had a history of the disease) are at increased risk.
- 4. This pregnancy is the first pregnancy, multiple pregnancy, polyhydramnios, and the pregnancy interval ≥ 10 years, the risk of disease is increased.

Knowledge

Time of onset

It usually occurs after 20 weeks of pregnancy.

Prevalence

According to statistics, there are about 8,000 cases of the disease per 100,000 people.

Typical symptoms

On the basis of hypertension and proteinuria, a series of clinical symptoms are developed such as headache, vertigo, edema, epigastric discomfort, nausea and vomiting.

Treatment

Antispasmodic lowering blood pressure, prevention of convulsions and timely termination of pregnancy are the key to treatment, and termination of pregnancy is the most effective treatment. Commonly used therapeutic drugs are magnesium sulfate and antihypertensive drugs. In addition, it is recommended that pregnant women eat less salt, rest in bed, and avoid getting angry.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS4762	AGT	GG	1.00	0.97
RS10509305	STOX1	AA	1.00	1.00

Risk calculation

The risk value of the test is calculated based on published academic literatures related to preeclampsia. By studying the genotype differences between preeclampsia patients and normal people, these literatures have found some polymorphic sites associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of preeclampsia item uses 2 polymorphic loci in the *STOX1*, *AGT* gene. The test results indicate whether the user has a higher prevalence of preeclampsia compared to all users. This test does not represent the user's true risk of preeclampsia. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could lead to a risk of preeclampsia.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of preeclampsia at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
3. If you have a family history of preeclampsia, or you think that you are likely to suffer from preeclampsia, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 2 polymorphic loci related to the risk of preeclampsia, including rs10509305 and rs4762.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to preeclampsia.

Laryngocarcinoma

Laryngocarcinoma is a malignant tumor that occurs in the larynx or other parts spreading to the larynx.

My Genetic Risk Is Average For The Population

0.86 times

Average incidence rate is 2 per 100,000 in the population

Suggestions

1. Maintain a healthy lifestyle. Don't smoke, drink alcohol in moderation.
2. Take appropriate protective measures. Wear a mask when going out in polluted cities. Take necessary protective measures when contacting toxic chemicals and radioactive substances.
3. Pay attention to sexual hygiene. Maintain a single sexual relationship and use condoms correctly.

Influencing factors



Controllable factors

1. Lifestyle factors: bad lifestyle, including long-term smoking and alcohol consumption, can increase the risk of laryngocarcinoma.
2. Environmental factors: air pollution, such as dust, sulfur dioxide, chromium and arsenic in the air, long-term occupational exposure to toxic chemicals, such as mustard gas, asbestos and nickel, and long-term exposure to radionuclides, such as radium, uranium and radon, can increase the risk of laryngocarcinoma.
3. Virus infection: HPV can cause laryngeal papilloma, which is currently considered to be a precancerous lesion of laryngocarcinoma.



Uncontrollable factors

1. Age: Laryngocarcinoma is more common in people over 50.
2. Gender: Men are more likely to develop laryngocarcinoma.

3. Genetic factors: Genetic variations can cause laryngocarcinoma.

Knowledge

Time of onset

Laryngocarcinoma mostly occurs after the age of 50.

Prevalence

The incidence rate of laryngocarcinoma in China is lower than the world average level. The incidence rate of laryngocarcinoma is 2.04/100,000 in China.

Typical symptoms

Laryngocarcinoma has the following clinical symptoms: persistent hoarseness, dyspnea, sore throat, dysphagia and pain, cough and hemoptysis, cervical lymphatic metastasis.

Treatment

1. Surgical treatment: surgical treatment can be divided into the following types: resection under a support laryngoscope, partial laryngectomy, supraglottic laryngectomy and total laryngectomy.
2. Radiotherapy: cobalt 60 and linear accelerator are the main means of radiotherapy at present. For early-stage laryngocarcinoma, the cure rate and 5-year survival rate of radiotherapy are comparable to that of surgical treatment. The disadvantage is that the treatment cycle is long, and there may be symptoms such as loss of taste and smell and dry mouth.
3. Chemotherapy: it can be divided into induction chemotherapy, adjuvant chemotherapy, palliative chemotherapy, etc. Induction chemotherapy is giving drugs before surgery or radiotherapy. At this time, the tumor blood supply is abundant, which is conducive to the effect of drugs. Adjuvant chemotherapy refers to the addition of chemotherapy after surgery or radiotherapy to kill possible residual tumor cells. Palliative chemotherapy refers to palliative treatment for patients with recurrence or systemic metastasis who cannot be operated on.
4. Biotherapy: including recombinant cytokines, adoptive metastatic immune cells, monoclonal antibodies, tumor molecular vaccines, etc., but the efficacy has not been confirmed.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS4975616	intergenic	AA	0.27	0.93
RS1056836	CYP1B1	CG	0.56	0.96
RS1056827	CYP1B1	CC	1.00	0.95

Risk calculation

The risk value of this test is calculated based on published academic literatures related to laryngocarcinoma. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between subjects with laryngocarcinoma and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The laryngocarcinoma item uses 3 polymorphic loci in and between *CYP1B1* gene. The test results indicate whether the user has a higher risk of laryngocarcinoma for the genotype combination of 3 polymorphic loci detected compared to all users. The test cannot represent the user's real risk of laryngocarcinoma. The basis of the test is mainly from the research of Chinese and American, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that contribute to the risk of laryngocarcinoma.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are jointly determined by multiple factors such as genes and environment. This study only predicts your risk of laryngocarcinoma at the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If there is a family history of laryngocarcinoma in your family, or if you think you are at risk of developing laryngocarcinoma, you are recommended to consult an authoritative health professional or clinician for help.

Test content

Three polymorphic loci associated with the risk of laryngocarcinoma were detected, including rs1056827 and rs4975616.

Population samples

The basis of the test is mainly from the research of Chinese and American.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect laryngocarcinoma.

Kawasaki Disease

Kawasaki disease is a childhood vasculitis syndrome, with acute fever and rash as the main symptoms.

My Genetic Risk Is Average For The Population

0.85 times

Average incidence rate is 69 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Age: Kawasaki disease usually affects children under the age of 8, and 90% of the onset populations are younger than 5.
2. Gender: The incidence of boys is about twice that of girls.
3. Population: Those with Asian or Pacific Islander descent are more likely to get it than that in Europe and America.

Knowledge



Time of onset

Kawasaki disease usually affects children under the age of 8.



Prevalence

In the United States and Europe, Kawasaki disease occurs in approximately 1 in 10,000 children under 5. The incidence rate in East Asia is much higher than that in United States and Europe. Kawasaki disease occurs in approximately 69 in 100,000 children under 5 in Taiwan.



Typical symptoms

- High fever lasting at least five days.
- A rash, urticaria-like, measles-like or scarlet fever-like rash.

- Oral mucosal lesions.
- Swollen lymph nodes.
- Severe complications involve the heart, including acute myocarditis with heart failure, arrhythmia, and coronary aneurysms.

Treatment

Kawasaki disease is usually treated in the hospital with an intravenous dose of immunoglobulin and aspirin which can quickly control inflammation and prevent complications such as coronary artery aneurysm and myocardial inflammation.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2254546	intergenic	GG	3.42	0.94
RS2618476	BLK	CC	2.31	0.97
RS1801274	FCGR2A	AA	1.74	0.98
RS28493229	ITPKC	CG	1.52	0.99
RS1569723	intergenic	AC	1.42	0.97

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to Kawasaki disease. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between subjects with Kawasaki disease and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of Kawasaki disease item uses the 12 polymorphic loci on and between genes such as *BLK*, *PELI1*, etc. The test results indicate that compared with all users, whether the genotype combination at the 12 polymorphic loci tested has a greater risk of suffering from Kawasaki disease, but it does not represent the user's risk of Kawasaki disease from all dimensions. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect Kawasaki disease.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This test only evaluates your risk of Kawasaki disease at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If your family has a history of Kawasaki disease, or you feel that you are at risk of suffering from Kawasaki disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 11 polymorphic loci related to Kawasaki disease, such as rs2736340.

Study population

The basis of the test is mainly from the research of Caucasian and East Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect Kawasaki disease.

Autoimmune Addison's Disease

Autoimmune Addison's disease is a systemic disease caused by autoimmunity and insufficient secretion of adrenocortical hormone caused by structural or functional defects of adrenal cortex.

My Genetic Risk Is Average For The Population

0.82 times

Average incidence rate is 20 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Age: Commonly between 30-50 years old.

Knowledge



Time of onset

The disease can occur at any age, and it is more common between 30-50 years of age. There are no special symptoms at first, and symptoms gradually develop over months or years.



Prevalence

It is an uncommon disease. In 2015, in the Nordic population, about 200 out of every million people became ill. There is still a lack of epidemiological survey data of autoimmune addison's disease in China.



Typical symptoms

- Fatigue: It is usually conscious fatigue and weakness, poor mental state, and cannot be relieved after rest.
- Dizziness: Often have the feeling that the head is dizzy, spinning and afraid to open your eyes.
- Orthostatic syncope: Manifested as a sudden transient loss of consciousness and fainting.
- Nausea and vomiting: Often feel discomfort in the upper abdomen after eating, and then vomit.

- Diarrhea: Defecation varies from 3-8 times a day, and the stool is mushy.
- Loss of appetite: Loss of appetite, decreased appetite.
- Weight loss: Due to loss of appetite and increased consumption, the weight lost more than 4 kg in 1 month.
- Female body hair reduction: Mainly axillary hair and pubic hair reduction.
- Menstrual disorders: Manifested as abnormal menstrual cycles and menstrual blood volume.
- Skin pigmentation: Lighter is brownish-black, brownish-yellow, bronze, darker pigmented is coking coal. Pigmentation occurs mainly on the palm side of the limbs, armpits, genitals, around the nipples, and lower breasts. There may also be pigmentation on exposed parts such as the face and neck. The oral mucosa and lips may also be affected, causing bluish-black spots.
- Shock: Pale and cold skin, shallow and fast breathing, even purple skin, confusion.

Treatment

Drug therapy is the mainstay, and most patients need long-term drug therapy. The purpose is to supplement the corticosteroids that the body lacks. Commonly used drugs: hydrocortisone, prednisone, deoxycorticosterone, 9a-fluoxycortisone.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2075876	AIRE	AG	2.17	0.87
RS10806425	BACH2	AC	1.69	0.98
RS11571303	intergenic	AG	1.39	0.98
RS1464510	LPP	AC	1.37	0.98
RS11203203	UBASH3A	GG	1.00	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to addison's disease. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between addison's disease patients and normal people. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this

test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of Addison's disease item uses 9 polymorphic loci on genes such as *PTPN22*, *BACH2*, *AIRE* and other genes. The test results indicate whether the user has a higher possibility of Addison's disease in the combination of genotypes at the 9 polymorphic loci tested compared to all users. It does not represent the user's real risk of Addison's disease. The basis of the test is mainly from the research of Caucasian, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that can lead to the risk of Addison's disease.
2. The test result has no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of Addison's disease from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of Addison's disease, or you think that you are likely to suffer from Addison's disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes rs2476601, rs8112143, rs74203920 and other 9 polymorphic loci related to Addison's disease.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to Addison's disease.

Peripheral Artery Disease (PAD)

Also known as peripheral vascular disease, peripheral arterial obstructive disease, and peripheral occlusive arterial disease, is a narrowing of the arteries that supply the brain and heart.

My Genetic Risk Is Average For The Population

0.81 times

Average incidence rate is 6000 per 100,000 in the population

Suggestions

1. Reject tobacco: If you already smoke, quit smoking.
2. Maintain a healthy weight: BMI is maintained between 18.5-23.9.

Influencing factors



Controllable factors

1. Smoking: Smoking is the main risk factor.
2. Obesity: Obesity can easily lead to the occurrence of the disease.



Uncontrollable factors

1. Gender: In developing countries, this disease is more prevalent in women.
2. Age: It can occur at any age, with a high incidence over the age of 50.
3. Disease: Diabetes, high blood pressure and high cholesterol may lead to this disease.
4. Family history: If there is a genetic history of vascular disease in the family, it is more likely to cause the disease.

Knowledge

Time of onset

The disease can occur at any age, with a high incidence over 50 years of age.

Prevalence

In developed countries, peripheral artery disease accounts for 5.3% of people aged 45 to 50, and 18.6% of people aged 85 to 90 years old. The proportions of diseased men and women are equal. In developing countries, peripheral artery disease accounts for 4.6% of people aged 45 to 50, and 15% of people aged 85 to 90 years old. Women have higher prevalence.

Typical symptoms

- Pain: This is the first symptom.
- Paresthesias: The pain in the affected limb is accompanied by numbness and chills.
- Motor dysfunction: Embolization leads to acute ischemia of limb muscles, fatigue and weakness during activity, weakened muscle strength, paralysis in severe cases, pain when passively moving limbs, and loss of deep reflexes.
- Disappearance or weakening of artery pulsation of limbs: The artery pulsation at the distal end of the embolization disappears due to the obstruction of blood flow, artery spasm, and secondary thrombosis.
- Skin changes: Generally, ischemic skin changes appear about 10cm below the embolization site, which manifests as pale, variegated or cyanotic skin.

Treatment

1. Non-surgical treatment: Including general treatment, thrombolytic treatment, anticoagulation treatment, percutaneous transluminal angioplasty and other medications.
2. Surgical treatment: Most patients with thromboembolism require surgical treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS138294113	intergenic	CC	1.19	1.00
RS7528419	CELSR2	AA	1.14	1.00
RS505922	ABO	CC	1.12	1.00
RS62084752	intergenic	CG	1.07	1.00
RS55784307	intergenic	AC	1.06	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to peripheral artery disease. By studying the genotype differences between patients with peripheral artery disease and normal people, literatures have found some polymorphic sites associated with the risk of peripheral artery disease. We uses the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

A health risk item for peripheral artery disease uses 21 genetic and intergenic polymorphic loci of genes such as *CHRNA3*, *F5*, *LPL*, *MMP3* and *PTPN11*. The results show whether the user had a higher prevalence of peripheral artery disease than all users in the genotype combination of the 21 polymorphic loci tested, which does not represent the user's real risk of peripheral artery disease. The test is based mainly on Caucasian studies, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of peripheral artery disease.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.

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2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing peripheral artery disease at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
 3. If your family has a family history of peripheral artery disease, or if you feel you have a risk of developing peripheral artery disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 21 polymorphic loci associated with the risk of peripheral artery disease, including rs1051730, rs6025 and rs322.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with peripheral artery disease.

Otosclerosis

Otosclerosis is a focal lesion of the bone labyrinth characterized by progressive hearing loss and associated with abnormal bone metabolism of the bone labyrinth.

My Genetic Risk Is Average For The Population

0.80 times

Average incidence rate is 713 per 100,000 in the population

Suggestions

Otosclerosis is associated with the measles virus, so measles vaccination is recommended for those who have never had an infectious skin disease, who have not been vaccinated, and those who have failed vaccination.

Influencing factors



Controllable factors

Viral infections: Study links measles virus to otosclerosis.



Uncontrollable factors

1. Family history: Otosclerosis has obvious genetic tendency, 60% of clinical otosclerosis patients have a family history, if one of the parents with otosclerosis, the children of otosclerosis increased the chance.
2. Age: Most of the patients were middle-young people, mostly between 20 and 40 years old.
3. Race: The incidence is higher in Caucasians and lower in Asians and Africans.

Knowledge

Time of onset

The onset age is 20-40 years old.

Prevalence

The incidence of otosclerosis is closely related to race, with Caucasian populations having a higher incidence and Asian and African populations having a lower incidence.

Typical symptoms

Otosclerosis begins with progressive loss of hearing in one or both ears. More than half of patients are accompanied by tinnitus, and a small number of patients will go dizzy when moving their heads.

Treatment

The cause of this disease is unknown, the purpose of treatment is only to restore hearing, can not be cured. Surgical treatment is the most effective measure to improve hearing, and those who are not suitable for surgery can be fitted with hearing AIDS.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS3914132	RELN	TT	1.63	1.00
RS4917	AHSG	CC	0.71	0.99
RS80339979	STX17-AS1	DD	1.32	1.00
RS553652	MAML2	AG	1.30	0.99
RS10592836	intergenic	DI	1.25	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to otosclerosis. These studies identified some polymorphic loci associated with the risk of celiac disease by examining genotypic differences between patients with and without otosclerosis. We

comprehensively score the polymorphic loci involved in this test through the original data in these literatures and obtains the risk values of all subjects. By comparing with the average risk of all subjects tested in our projects, we can give the risk multiple of subjects relative to the average risk of the population, and obtain the distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The otosclerosis health risk item uses 17 polymorphic loci on *RELN*, *SUPT3H*, *AHSG*, *STX17-AS1* and other genes. The test results indicated whether the subject had a higher prevalence of otosclerosis compared to all subjects at 17 polymorphic loci tested. The test does not represent a subject's true risk for otosclerosis. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of otosclerosis.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The development of the disease is determined by a combination of genetic and environmental factors. This test only predicts the risk of otosclerosis from the genetic level, and it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc..
3. If you have a family history of otosclerosis, or you think that you are likely to suffer from otosclerosis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 17 polymorphic loci related to the risk of otosclerosis, including rs791903, rs553652 and rs4877080.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect otosclerosis.

Hypertension

Hypertension is a clinical syndrome that mainly manifests as systemic increased arterial blood pressure.

My Genetic Risk Is Average For The Population

0.79 times

Average incidence rate is 11880 per 100,000 in the population

Suggestions

- Red flags: Systolic pressure or diastolic pressure is too high
- Common symptoms: no obvious symptoms, most people don't know or don't realize they are sick until complications (stroke, heart disease, etc.) appear
- High-risk population: middle-aged and elderly people, obesity, drinking, smoking, high-oil and high-salt diet, long-term stress, kidney disease or diabetes, and family history of hypertension
- How to reduce the risk:
 - Pay attention to your diet: eat more vegetables, drink less alcohol, and eat less high-salt, high-fat, and high-calorie foods
 - Weight control: keep BMI between 18.5 and 23.9, avoid sitting for a long time, and exercise at least 4 hours a week
 - Relieve stress: avoid being in a state of high pressure for a long time, and resolve negative emotions in time
 - Quit smoking: refuse to take the initiative to smoke, stay away from second-hand smoke
 - Measure your blood pressure frequently, and master your own blood pressure changes
 - Protective food: consider supplementing with folic acid, niacin, arginine, resveratrol and other supplements, such as garlic.
- Hypertension Heritability is about 20%~60%

* If you have a family history of hypertension, or if you think you are likely to suffer from hypertension, or need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors



Controllable factors

-
1. Dietary factors: a high-sugar and high-fat diet, eating more meat and less vegetables, excessive sodium intake, and low potassium intake will increase the risk of hypertension.
 2. Tobacco and alcohol intake: smokers or passive smokers have an increased risk of hypertension. Alcohol raises blood pressure and promotes an increase in the content of certain fatty acids in the blood.
 3. Poor living habits: sitting for a long time and lack of exercise may increase the risk of hypertension.
 4. Psychological and emotional factors: people who have been under excessive stress, anxiety, irritability, and emotional agitation for a long time have a higher risk of hypertension.
 5. Overweight and obesity: with the increase of BMI and waist circumference, the mean blood pressure showed a significant increase trend, and the prevalence of hypertension also increased significantly.

Uncontrollable factors

1. Age: the prevalence of hypertension increases with age.
2. Family history: hypertension has obvious familial clustering and is obviously related to immediate family members. People with a family history of hypertension have a higher prevalence.
3. Blood glucose and blood lipid levels: epidemiological data show that hypertension is often accompanied by many risk factors related to metabolism, including impaired glucose tolerance and dyslipidemia, which are related to hypertension.

Knowledge

Time of onset

It can occur at any age, but the prevalence of hypertension increases with age.

Prevalence

According to data released by the Ministry of Health: The average incidence rate in China is 11.88%, and the number of patients is over 160 million. In some European and American countries, the incidence rate is as high as 20%.

Typical symptoms

Hypertension is a clinical syndrome characterized by increased systemic arterial blood pressure. The cause of most patients is unknown. Studies have shown that patients with hypertension are more likely to have other cardiovascular and cerebrovascular diseases than those with normal blood pressure, such as atherosclerosis. It also affects the functions of important organs such as the heart, brain, and kidneys, and ultimately leads to the failure of these organs. Some hypertensive patients

have headaches (especially back pain and headaches in morning), dizziness, tinnitus (buzzing or sizzling in the ears), visual changes, or fainting episodes.

Treatment

The cause of most hypertension patients is not clear, and most patients need long-term medication to control their blood pressure. For patients with mild and early hypertension, blood pressure can be controlled within the normal range through weight loss, diet improvement, exercise, etc. And it is important to maintain a healthy lifestyle. If the blood pressure level drops after taking the medicine, the patient should not stop the medicine by himself, otherwise the blood pressure rebounds or is more dangerous. Although high blood pressure cannot be cured completely, by insisting on taking medication, blood pressure can be effectively controlled and the risk of various adverse consequences can be minimized.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS5370	EDN1	GT	2.35	0.92
RS3781719	CALCA	AG	1.94	0.96
RS4961	ADD1	TT	1.80	0.98
RS16998073	intergenic	AT	1.30	0.99
RS1805762	M6PR	CG	1.23	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is calculated based on published academic literatures related to hypertension. By studying the genotype differences between hypertension patients and normal people, these literatures have found some polymorphic sites associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account

the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of hypertension item uses the 18 polymorphic loci in and between genes of *ADD1*, *ADD2*, *AGT*, *EDN1* and *CALCA*. The test results indicate whether the user has a higher prevalence of hypertension in the combination of genotypes at the 18 polymorphic loci tested compared to all users. This test does not represent the user's true risk of hypertension. The basis of the test is mainly from the research of Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of hypertension.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of hypertension at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
3. If you have a family history of hypertension, or you think that you are likely to suffer from hypertension, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 18 polymorphic loci related to the risk of hypertension, including rs3755351, rs9739493 and rs1126742.

Population samples

The basis of the test is mainly from the research of Asian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to hypertension.

Hyperemesis Gravidarum

About 0.3-2% of women will experience severe morning sickness during pregnancy, which is called hyperemesis gravidarum.

My Genetic Risk Is Average For The Population

0.79 times

Average incidence rate is 470 per 100,000 in the population

Suggestions

1. Keep your mood happy, don't be too nervous, and correctly understand the physiological phenomenon of morning sickness. Appropriate to participate in some gentle activities, such as outdoor walking, do maternal health exercises. Appropriate to participate in some gentle activities, such as outdoor walking, maternity aerobics, etc.
2. Food should be light, easy to digest, and nutrient-rich, such as rice soup, gruel, soybean milk, all kinds of broth, etc. Avoid fishy, greasy, too sweet, too cold, too hot food. Pay attention to food hygiene, do not eat corrupt, expired food, etc.
3. Excessive vitamin loss during severe vomiting. Patients with vomiting are often accompanied by loss of appetite and inadequate vitamin intake, which requires vitamin supplementation, especially vitamin B6 supplementation.
4. Keep the indoor air well ventilated and try not to go to crowded places. The room should be as clean, quiet and comfortable as possible. Avoid the odor of tobacco, alcohol, paint, coatings, pesticides and other chemicals. Vomit should be removed immediately to avoid malignant irritation, and gargle with warm boiled water to keep oral cavity clean.

Influencing factors



Controllable factors

1. Psychological factors: excessive mental stress, fear of pregnancy, etc.

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2. Dietary factors: Improper diet during pregnancy (such as greasy or unhealthy food) will increase the risk of vomiting during pregnancy.
 3. Nutritional factors: deficiency of vitamins, especially vitamin B6, can aggravate hyperemesis gravidarum.
 4. Other inducing factors: tobacco, alcohol, kitchen lampblack stimulation; Or avoid the odor of paints, coatings, pesticides and other chemicals.

Uncontrollable factors

1. Age: The younger the mother is, the more likely it is to occur, mostly in primipara.
2. Medical history: complicated with hyperthyroidism, mental illness, past history of glucose, diabetes, gastrointestinal disease and asthma.
3. Pregnant women with multiple pregnancies are at higher risk of Hyperemesis gravidarum.
4. Infectious factors: Increased helicobacter pylori infection: Compared with asymptomatic pregnant women, hyperemesis gravidarum patients had increased serum IgG concentration against Helicobacter pylori.

Knowledge

Onset time

Most of Hyperemesis gravidarum occurs in early pregnancy, which is a kind of early pregnancy reaction, with an incidence of 50%-90%. It begins to appear about 6 weeks after menopause, mostly disappears spontaneously around 12 weeks after menopause, and a few continue through the whole pregnancy.

Prevalence

About 0.3-2% of women experience severe morning sickness during pregnancy.

Typical symptoms

Hyperemesis gravidarum can cause a range of symptoms including weight loss, dehydration, ketonuria, nutritional deficiencies, electrolytes imbalances and the health of the baby can also be affected in some cases.

Treatment

1. They are usually given intravenous fluids to replenish electrolytes and water, and for those with poor nutrition, they also need intravenous supplements of nutrients such as amino acids and fat milk. Antiemetic and acidosis remedies may also be added as appropriate. Generally, through systematic treatment, pregnant women can alleviate after 2-3 days.

2. If after active treatment, vomiting still cannot be alleviated, or even persistent jaundice, proteinuria, fever (temperature $\geq 38^{\circ}\text{C}$), accelerated heart rate (heart rate > 120 beats/min), Wernicke syndrome, etc., pregnancy may need to be terminated to ensure the life safety of the pregnant woman.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS16982345	GDF15	AG	1.50	0.97
RS4865234	intergenic	AG	1.33	0.98

Risk calculation

The risk value of this test is calculated based on published academic literatures related to Hyperemesis gravidarum. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between pregnant women with hyperemesis gravidarum and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Hyperemesis gravidarum risk item uses two polymorphic loci between genes. The test result indicates whether the user has a higher possibility of hyperemesis gravidarum in genotype combination of the two polymorphic loci detected compared with all users, and the test does not represent the user's real risk of Hyperemesis gravidarum. The test is mainly based on the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of Hyperemesis gravidarum.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Hyperemesis gravidarum from the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If you have a family history of Hyperemesis gravidarum, or you think that you are likely to suffer from Hyperemesis gravidarum, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes rs16982345, rs4865234, two polymorphic loci associated with the total Hyperemesis gravidarum.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci.

Epilepsy

Epilepsy, commonly known as epilepsy crazy or sheep epilepsy, is a chronic non communicable disease caused by abnormal electrical signals of the brain. The symptoms are convulsions, loss of consciousness and so on.

My Genetic Risk Is Average For The Population

0.77 times

Average incidence rate is 700 per 100,000 in the population

Suggestions

1. Eliminate parasites in the environment and reduce risk factors such as cerebral cysticercosis caused by infection.
2. Prevent and control hypertension, diabetes, and obesity. It is recommended to eat healthily, exercise actively, and avoid smoking and excessive drinking.

Influencing factors



Controllable factors

1. Environment: Parasites may cause brain infections and increase the risk of epilepsy.
2. Cardiovascular disease: Hypertension, diabetes, obesity are risk factors of cardiovascular disease, which can increase the risk of epilepsy.



Uncontrollable factors

1. Genetic factors: The incidence of direct relatives of epileptic patients is higher than that of the general population.
2. Brain diseases: Brain malformations caused by congenital abnormalities or genetic diseases, strokes and tumors are common causes.
3. Inflammation and infection: Brain infections such as meningitis, encephalitis, and cysticercosis may cause epilepsy.

4. Brain trauma: Brain trauma is one of the factors that affect adults.

Knowledge



Time of onset

It often starts in early childhood or late adulthood.



Prevalence

The incidence rate is 49 per 100,000 in high-income countries and 139 per 100,000 in low-income and middle-income countries.



Typical symptoms

- Recurrent seizures, involuntary convulsions of a part of the body or the whole body.
- Numbness, stiffness or twitching of the limbs.
- Sometimes accompanied by loss of consciousness and urinary incontinence.



Treatment

1. Drug treatment: Phenytoin sodium, phenobarbital, valproic acid, carbamazepine, lamotrigine, topiramate, etc. In the process of taking medicine, pay attention to eliminate incentives, such as drinking, overwork, insomnia, etc.
2. Non-drug treatment: surgery, vagus nerve stimulation, ketogenic diet, etc.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2292096	CAMSAP2	GG	0.40	0.97
RS1939012	MMP8	TT	1.25	1.00
RS6732655	SCN1A	TT	1.25	1.00
RS2947349	intergenic	AC	1.23	0.99
RS11890028	intergenic	TT	1.19	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to epilepsy. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype difference between patients with epilepsy and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details



Applicable situation

The epilepsy health risk item uses 11 polymorphic loci on genes such as *CAMSAP2*, *MMP8* and other genes. The test result shows whether the user has a higher possibility of epilepsy in the combination of genotypes at the 11 polymorphic loci detected compared to all users. The test cannot represent the user's real risk of epilepsy. The basis of the test is mainly from the research of European, East Asian and African American, other populations are for reference only.



Notification

1. The test may not cover all the pathogenic genes or loci of that affect epilepsy.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results



Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only predicts your risk of epilepsy at the genetic level, it does not

mean your true health status, nor does it involve the assessments on your other health conditions, living habits, etc.

3. If you have a family history of epilepsy, or you think that you are likely to suffer from epilepsy, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 11 polymorphic loci related to the risk of epilepsy, including rs2292096, rs1939012 and rs6732655.

Study population

The basis of the test is mainly from the research of European, East Asian and African American.

Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all genes or loci that affect epilepsy.

Amyotrophic Lateral Sclerosis (ALS)

It is a neurodegenerative disease that mainly affects motor neurons in the cerebral cortex, brainstem, and spinal cord. It can cause gradual muscle weakness and atrophy in the limbs, trunk, and body, ultimately leading to death due to respiratory failure.

My Genetic Risk Is Average For The Population

0.74 times

Average incidence rate is 4 per 100,000 in the population

Suggestions

1. Avoid contact with toxic substances, especially heavy metals, pesticides, etc.
2. Avoid external injuries, especially protect the head.
3. Avoid strenuous physical activity, ensure adequate nutrition and rest.

Influencing factors



Controllable factors

1. Toxic substances: Exposure to plant toxins (e.g., cassava), heavy metals (e.g., lead, cadmium, chromium), pesticides, organic solvents may induce the disease.
2. Trauma/injury: Research has shown that past injuries (e.g., head injuries) are associated with an increased risk of the disease.
3. Excessive physical labor: Excessive physical activity or professional sportsman (e.g., professional football players) increases the risk of developing this disease.



Uncontrollable factors

1. Genetic factors: There is a family genetic tendency.
2. Immune factors: Patients with autoimmune diseases are at high risk.

Knowledge

Time of onset

Most patients develop the disease over the age of 50, and the incidence increases every 10 years thereafter, and it is rare for patients to develop the disease before the age of 30.

Prevalence

According to statistics, there are about 4 cases of the disease for every 100,000 people in the average population.

Typical symptoms

The first symptom is often poor finger movement and weak strength, with inaccurate precise manipulation. As a result, small muscles such as the greater and lesser thenar muscles, interosseous muscles, and vermiform muscles in the hand significantly atrophy, and grooves appear between the flexor tendons of the palm. The hands are in the shape of eagle claws, gradually developing towards the muscles of the forearm, upper arm, and scapular girdle. Lower limb stiffness, weakness, uncoordinated movements, and spasmodic paraplegia can occur simultaneously or sequentially with upper limb symptoms. As the disease progresses, muscle weakness and atrophy can extend to the trunk and neck, and patients cannot rotate their necks, lift their heads, or be forced to lie in bed.

Treatment

Currently, this disease cannot be cured. Riluzole is the first drug approved by the US Food and Drug Administration (FDA) and the European Union for the treatment of amyotrophic lateral sclerosis, and is currently the only drug recognized to be effective in alleviating the progression of the disease, improving patient survival.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS6539137	TXNRD1	TT	0.13	0.99
RS12608932	UNC13A	CC	1.46	0.99
RS3825776	intergenic	CT	1.34	0.98

RS6700125	intergenic	CT	0.80	0.99
RS10463311	intergenic	CC	1.17	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to amyotrophic lateral sclerosis. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between patients with amyotrophic lateral sclerosis and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The amyotrophic lateral sclerosis health risk item uses 20 polymorphic loci on genes such as *DPP6*, *UNC13A*, *TXNRD1*, *ANG* and other intergenic regions. The test results indicate that compared with all users, whether this user has a higher prevalence of amyotrophic lateral sclerosis in the combination of genotypes of the 20 polymorphic loci tested. It does not mean the user's true risk of amyotrophic lateral sclerosis. The basis of the test is mainly from the research of Caucasian and Asian, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that cause the risk of amyotrophic lateral sclerosis.
2. The test result has no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of the disease is determined by multiple factors such as genes and environment.
This study only predicts your risk of amyotrophic lateral sclerosis from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of amyotrophic lateral sclerosis, or you think that you are likely to suffer from amyotrophic lateral sclerosis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 20 polymorphic loci related to the risk of amyotrophic lateral sclerosis, including rs10239794, rs12608932, rs10260404, rs6539137, and rs11701.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to amyotrophic lateral sclerosis.

Melanoma

Clinically, melanoma is a common malignant neoplasm of the skin mucosa and pigmented membranes, and also has one of the fastest growing incidence rates of all malignant neoplasms.

My Genetic Risk Is Average For The Population

0.74 times

Average incidence rate is 1 per 100,000 in the population

Suggestions

1. Avoiding UV exposure, such as the use of sunscreen, UV-protection clothing, etc., can effectively prevent the production of melanoma.
2. Usually avoid deliberately picking and scraping moles to prevent malignant transformation of benign pigmented moles.
3. To increase physical activity, adults perform at least 150 minutes of moderate-intensity exercise (e.g., fast walking, jogging) per week to improve the body's immunity.

Influencing factors

Controllable factors

1. UV exposure: excessive UV exposure is the main cause of melanoma, UVA and UVB in UV can induce melanoma.
2. Artificial irritation: Too much artificial stimulation of benign pigmented moles (such as repeated scraping and spotting) may cause benign pigmented moles to become malignant.
3. Immune factors: People with weakened immunity have a higher risk of melanoma.

Uncontrollable factors

1. Race: The incidence of white people is much higher than that of black people.
2. Family history: The onset of melanoma has a more obvious family genetic tendency.

- 3. Age: It is more common in middle-aged people over 30 years old.
- 4. Gender: There are more men than women.

Knowledge

Time of onset

The age of onset is mostly over 30 years old.

Prevalence

The incidence of melanoma varies from race, region, and ethnicity. The incidence of whites is much higher than that of blacks. The incidence of whites living in Queensland, Australia, as high as 44/100,000. Compared with European and American countries, the incidence of melanoma in Asian countries such as China and Japan is relatively low, with a prevalence of 0.6 per 100,000.

Typical symptoms

Melanoma mostly develops from the transformation of moles. Changes in the appearance of moles include changes in shape, irregular edges, changes in color, increased size, itching or ulcers, etc.

Treatment

Surgical treatment, radiotherapy, chemotherapy and immunotherapy.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS149617956	MITF	GG	0.15	1.00
RS1805007	MC1R	CC	0.32	1.00
RS117132860	intergenic	GG	0.50	1.00
RS35407	SLC45A2	AG	0.48	0.94
RS78378222	TP53	TT	0.58	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is calculated based on published academic literature on melanoma. By studying the genetic differences between melanoma patients and normal people, these literatures have identified some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of melanoma item uses 52 polymorphic loci in *SLC45A2*, *PIGU*, and *AFG3L1P*. The results show whether the user had a higher incidence of melanoma than all users in the genotype combination of the 52 polymorphic loci tested, which does not represent the user's real melanoma risk. The test is based mainly on studies from American, Oceaniaan and Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of melanoma.
2. The test results have not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of melanoma at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.

3. If you have a family history of melanoma, or you think that you are likely to suffer from melanoma, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 52 polymorphic loci associated with melanoma risk in and between genes such as rs4785763, rs16891982, rs910873, and so on.

 Population samples

The basis of the test is mainly from the research of American, Oceanian, and Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to melanoma.

Pancreatic Cancer

Pancreatic cancer is a malignant tumor of digestive tract caused by canceration of pancreatic cells.

My Genetic Risk Is Average For The Population

0.72 times

Average incidence rate is 5 per 100,000 in the population

Suggestions

1. Reject tobacco: If you already smoke, quit smoking.
2. Maintain a healthy weight: BMI is maintained between 18.5 and 23.9.
3. Limit alcohol intake: the limit value of moderate drinking for adults recommended by the Chinese Nutrition Society is that the amount of alcohol consumed by adult men should not exceed 25g per day; the amount of alcohol consumed by adult women should not exceed 15g per day.

Influencing factors



Controllable factors

1. Smoking: for the most thoroughly studied and avoidable risk factor for pancreatic cancer, the risk of long-term smoking is approximately twice as high, and the risk increases with smoking age. After quitting smoking, the risk will slowly decrease, and it will take about 20 years to return to the same level as non-smokers.
2. Obesity: The risk of people with a BMI greater than 35.0 is about 1.5 times that of the average person.
3. Alcoholism: It is the main cause of chronic pancreatitis. Patients with chronic pancreatitis are also more likely to get pancreatic cancer.



Uncontrollable factors

1. Gender: more male patients than female.

- 2. Age: more common among middle-aged and elderly people.
- 3. Heredity: 5-10% of pancreatic cancer patients also have a family history. if more than one first-class relative suffers from the disease, the risk increases significantly and the disease usually occurs before the age of 50. Hereditary pancreatic cancer will increase the risk of developing the disease by about 30-40% before the age of 70.

Knowledge

Time of onset

This disease is more common in the elderly.

Prevalence

The incidence of pancreatic cancer has been increasing in recent years around the world. According to population statistics, its incidence is about 10/100,000 per year, and it can be as high as 100/100,000 in the elderly.

Typical symptoms

- 1. Pain: it is the main symptom of pancreatic cancer, regardless of whether the cancer is located in the head or tail of the pancreas.
- 2. Jaundice: in the past, in the diagnosis of pancreatic cancer, painless jaundice was often the first or mandatory symptom of pancreatic cancer, and jaundice was used as an important basis for diagnosing pancreatic cancer. Therefore, early diagnosis and surgery opportunities were often lost.
- 3. Gastrointestinal symptoms: the most common is loss of appetite, followed by nausea, vomiting, diarrhea or constipation or even melena. Diarrhea is often steatorrhea.
- 4. Weight loss and fatigue: pancreatic cancer is different from other cancers. It often has weight loss and fatigue in the early stage.

Treatment

Pancreatic cancer is mainly treated by surgery combined with radiotherapy and chemotherapy.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS505922	ABO	CC	1.66	0.98
RS2941471	HNF4G	GG	0.79	1.00
RS1517037	intergenic	CT	0.86	1.00
RS3790844	NR5A2	GG	1.00	0.98
RS13303010	NOC2L	AA	1.00	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to pancreatic cancer. By studying the genetic differences between pancreatic cancer patients and normal people, these literatures have found some polymorphic sites associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of pancreatic cancer item uses 8 polymorphic loci in and between genes of *ATM*, *NR5A2*, *ABO*, *NOC2L*, *HNF4G*, *HNF1B* and *GRP*. The test results indicate whether the user has a higher prevalence of pancreatic cancer in the combination of genotypes at the 8 polymorphic loci tested compared to all users. This test does not represent the user's true risk of pancreatic cancer. The basis of the test is mainly from the research of Asian and Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of pancreatic cancer.
2. The test results have no clinical significance.

3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of pancreatic cancer at a genetic level and does not represent your true health status, nor does it involve your judgment of other health conditions, lifestyles, etc.
3. If you have a family history of pancreatic cancer, or you think that you are likely to suffer from pancreatic cancer, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 8 polymorphic loci related to the risk of pancreatic cancer, including rs1801516, rs3790844, rs9543325, rs505922, rs13303010, rs2941471, rs4795218 and rs1517037.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to pancreatic cancer.

Selective IgA Deficiency

This may be caused by a genetic abnormality in the non-IgA gene region rather than an IgA deficiency. Patients often also have autoimmune diseases and hypersensitivity diseases.

My Genetic Risk Is Average For The Population

0.71 times

Average incidence rate is 10 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Family history: The family genetic incidence of selective IgA defects is about 20%.
2. Ethnicity: Studies have shown that 1 out of every 500 Caucasians suffers from selective IgA deficiency, which has a higher prevalence than other ethnicities.
3. Gender: The incidence of selective IgA deficiency in men is higher than in women.

Knowledge



Time of onset

It can be onset at any age.



Prevalence

Selective IgA deficiencies are high in Caucasians, with an incidence rate of 1/500 to 700, African descent for 1/6000, and Asians for 1/14840 to 18500.



Typical symptoms

Most patients are asymptomatic, and a few may have repeated respiratory infections, chronic diarrhea, allergic reactions or autoimmune diseases.

Treatment

Most patients do not need treatment, and those with symptoms receive targeted treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS9271366	intergenic	AG	7.69	0.97
RS2187668	HLA-DQA1	CC	0.16	0.93
RS11299600	intergenic	DI	0.73	0.98
RS1990760	IFIH1	CC	1.00	0.98
RS7773987	intergenic	TT	1.00	0.98

Risk calculation

The risk value of this test is calculated based on published academic literature related to selective IgA deficiency. By studying the genotype differences between patients with selective IgA deficiency and normal people, these literatures have found some polymorphic sites associated with the risk of selective IgA deficiency. We uses the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

A health risk item for selective IgA deficiency uses 5 polymorphic loci of the *IFIH1*, *HLA-DQA1*, *AHI1*, etc. genes. The results show whether the user had a higher prevalence of selective IgA deficiency than

all users in the genotype combination of the 5 polymorphic loci tested, which does not represent the user's real risk of selective IgA deficiency. The test is based mainly on Caucasian studies, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of selective IgA deficiency.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing selective IgA deficiency at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of selective IgA deficiency, or if you feel you have a risk of developing selective IgA deficiency, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes rs1990760, rs2187668 and rs9271366, 5 polymorphic loci associated with the risk of selective IgA deficiency.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with selective IgA deficiency.

Lumbar Disc Herniation

Lumbar disc herniation is one of the more common diseases, mainly because the various parts of the lumbar intervertebral disc have different degrees of degenerative changes. Under the action of external force factors, the spinal nerve roots are stimulated or compressed, resulting in lumbar pain, lower extremity pain or numbness etc.

My Genetic Risk Is Average For The Population

0.69 times

Average incidence rate is 15385 per 100,000 in the population

Suggestions

1. Protect your waist during activities, and seek medical attention if you are injured or feel unwell.
2. Pay attention to the waist posture. Avoid using the same posture for a long time and avoid overwork. Strengthen low back muscle training.

Influencing factors

Controllable factors

1. Injury: Long-term repeated external forces cause slight damage and aggravate the degree of degeneration.
2. Inducing factors: common inducing factors include increased abdominal pressure, irregular waist posture, sudden weight bearing, pregnancy, cold and damp.

Uncontrollable factors

1. Age: mostly occurs in young adults.
2. Gender: most patients are men.
3. Genetic factors: familial incidences of lumbar disc herniation have been reported.

-
4. Lumbosacral congenital abnormalities: including lumbar sacralization, sacral lumbarization, hemivertebral deformities, facet joint deformities and asymmetry of articular processes.
-

Knowledge

Time of onset

Disc herniation occurs mainly between the 20 years and 40 years.

Prevalence

The number of patients with lumbar spondylosis in China has exceeded 200 millions, and patients with lumbar disc herniation accounted for 15.2% of the total number of people in China.

Typical symptoms

Lumbar disc herniation will have the following symptoms: low back pain and restricted lumbar movement, conductive pain in the lower extremities, urinary and bowel disorders caused by compression of the cauda equina, intermittent claudication, and changes in spine posture.

Treatment

1. Conservative treatment: Including absolute bed rest, traction therapy, physiotherapy and massage, massage, supportive therapy with glucosamine sulfate and chondroitin sulfate, corticosteroid epidural injection, chemical lysis of nucleus pulposus, etc.
2. Percutaneous nucleotomy/nucleus pulposus laser vaporization: Enter the intervertebral space with special equipment under X-ray monitoring, and squeeze out part of the nucleus pulposus or laser vaporize, thereby reducing the pressure in the intervertebral disc to relieve symptoms. It is suitable for patients with bulging or mild herniation, not suitable for the combined side. Patients with crypts stenosis or prominent protrusion, and those with nucleus pulposus prolapsed into the spinal canal.
3. Surgical treatment: Partial lamina and articular processes are removed through a posterior lumbar back incision, or intervertebral disc resection through the interlaminar space. Central disc herniation, after laminectomy, epidural or intradural disc resection. Patients with instability of the lumbar spine and stenosis of the lumbar spinal canal require simultaneous spinal fusion.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS143383	GDF5	AA	3.28	0.95
RS16924573	KIAA1217	GG	1.80	0.99
RS9406328	THBS2	AG	1.43	0.98
RS77681114	GSDMC	AG	0.81	0.99
RS6651255	intergenic	CT	0.81	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to lumbar disc herniation. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between subjects with lumbar disc herniation and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of lumbar disc herniation item uses the 10 polymorphic loci on and between genes such as *COL11A1*, *GDF5*, *THBS2*, etc. The test results indicate that compared with all users, whether the genotype combination at the 10 polymorphic loci tested has a greater risk of suffering from lumbar disc herniation, but it does not represent the user's risk of lumbar disc herniation from all dimensions. The basis of the test is mainly from the research of Japanese, Chinese, European, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect lumbar disc herniation.
2. The test results have no clinical significance.

3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This test only evaluates your risk of lumbar disc herniation at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If your family has a history of lumbar disc herniation, or you feel that you are at risk of suffering from lumbar disc herniation, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 10 polymorphic loci related to lumbar disc herniation, such as rs1676486, rs143383, rs9406328.

Population samples

The basis of the test is mainly from the research of Japanese, Chinese and European.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect lumbar disc herniation.

Post-Traumatic Stress Disorder (PTSD)

Post-traumatic stress disorder (PTSD) is a type of mental disorder caused by severe psychological trauma.

My Genetic Risk Is Average For The Population

0.69 times

Average incidence rate is 6750 per 100,000 in the population

Suggestions

If you have experienced a traumatic event that may lead to post-traumatic stress disorder, please receive psychological guidance and treatment as soon as possible.

Influencing factors



Controllable factors

Psychological intervention: If the group suffering from psychological trauma does not receive interventions such as psychological counseling in time, the incidence rate will increase.



Uncontrollable factors

1. Gender: The incidence of females in the population is slightly higher than that of men.
2. Severe trauma: People with severe trauma are more likely to suffer from post-traumatic stress disorder.
3. Disease factors: Patients with severe depression are more likely to suffer from traumatic stress disorder.

Knowledge



Disease triggers

Psychological trauma that can induce post-traumatic stress disorder is divided into three main categories, situational trauma, continuous trauma, and trauma caused by the experience of others:

1. Situational trauma: such trauma is primarily caused by events caused by man-made, technical or natural disasters, including war, abuse, violence, mechanized accidents (car, train or plane crashes, etc.) or medical emergencies (medical resource runs, etc.).
2. Continuous trauma: divided into environmental-induced trauma and self-inflicted trauma. Environments such as long-term stressful environments, such as political factors, work environments, and epidemics, cause psychological trauma due to high environmental stress, and their own circumstances lead to excessive recall after events such as accidents, such as mechanized accidents, excessive self-blame of soldiers after the war, etc.
3. Trauma caused by witnessing or hearing that someone else has experienced a serious traumatic event (or even death).

Sometimes trauma is not necessarily caused by a single classification, but may also be caused by a mixture of factors.

Time of onset

No specific age of onset. It usually occurs after experiencing an event that causes severe psychological trauma, and if you do not receive good psychological guidance, you may still get sick more than ten years after the event.

Prevalence

The lifetime prevalence of post-traumatic stress disorder ranges from 6.1 to 9.2 percent in national samples of the general adult population in the US and Canada, with one-year prevalence rates of 3.5 to 4.7 percent. The epidemiological prevalence data of trauma and stress-related disorders vary greatly. The incidence of acute stress disorder among survivors of violent crimes is 19% to 33%, and it is 1.6% to 41.1% after traffic accidents; The prevalence of post-traumatic stress disorder related to war among American veterans ranges from 2% to 17%, and the lifetime prevalence is approximately 6% to 31%. One to two months after the 9/11 terrorist attacks in the United States, the incidence of post-traumatic stress disorder among survivors ranged from 7.5% to 11.2%.

Typical symptoms

1. Will produce Intrusive thoughts or Over reaction.
2. Continuity avoidance something and the scene related to causing trauma.
3. Negative emotions, such as self-blame or avoiding social interaction, can occur.

Treatment

1. Psychotherapy: treatment through psychological guidance, including Psychodynamic therapy, Cognitive behavioral therapy, Stress prevention training and Eye movement desensitization and reprocessing, etc.

- 2. Drug therapy: by use Antidepressants and Anticonvulsants etc. for treatment.
- 3. Combined therapy: psychotherapy combined drug therapy is more effective than a single treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS34465541	intergenic	AA	0.24	0.95
RS599550	intergenic	AA	2.23	1.00
RS3800872	intergenic	CC	2.11	0.98
RS4129585	TSNARE1	AA	1.75	0.99
RS8015578	intergenic	CT	0.77	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to post-traumatic stress disorder. These literatures have found some polymorphic loci related to the risk of the disease by studying the genetic differences between patients with post-traumatic stress disorder and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

 Applicable situation

The post-traumatic stress disorder health risk item uses the 14 polymorphic loci on genes such as *PRTFDC1*, *NOS1AP*, *FOXP2* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 14 polymorphic loci detected by this test increases the risk of post-traumatic stress disorder. It does not mean the user's true post-traumatic stress disorder risk. The basis of the test is mainly from the research of Caucasian, African-American, Hispanic and Latino, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect post-traumatic stress disorder.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of post-traumatic stress disorder at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of post-traumatic stress disorder, or you think that you are likely to suffer from post-traumatic stress disorder, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 14 polymorphic loci associated with post-traumatic stress disorder, including rs34517852, rs1033962, rs34465541, and rs386231.

Population samples

The basis of the test is mainly from the research of Caucasian, African American, Hispanic and Latino.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect post-traumatic stress disorder.

Stomach Cancer

Stomach cancer is a malignant tumor occurring in gastric mucosa epithelium, accounting for the first incidence of gastrointestinal malignancy.

My Genetic Risk Is Average For The Population

0.68 times

Average incidence rate is 21 per 100,000 in the population

Suggestions

- Red flags: In the physical examination report, Stomach cancer antigen (CA724) is too high.
- High risk population: high-salt, high-fat diet, smoking, obesity, **helicobacter pylori** infection, stomach disease patients, middle-aged and elderly people, family history of stomach cancer
- How to reduce risk:
 - Balanced diet: eat more fresh fruits and vegetables, eat less pickled, smoked, high-fat foods, drink less alcohol
 - Quit smoking: refuse to take the initiative to smoke and stay away from second-hand smoke
 - Weight control: keep BMI between 18.5 and 23.9, avoid sedentary, and exercise at least 4 hours a week
 - People over 40 years old should have gastroscopy every 3 ~ 5 years
 - Check and eradicate Helicobacter pylori

* if you have a family history of stomach cancer, or you think you are likely to suffer from stomach cancer, or you need to use dietary supplements, it is recommended that you consult an authoritative health expert or clinician for help.

Influencing factors



Controllable factors

1. Poor eating patterns: Studies have shown that poor eating patterns can increase the risk of stomach cancer. Common risk factors are not eating breakfast, irregular meals, liking dry and hard food, liking hot food, eating fast, liking to eat smoked, roasted and fried food, etc. These bad habits and physical stimulation of food can cause mechanical gastric mucosal damage and gastric juice secretion disorders, etc. resulting in the occurrence of chronic stomach disease and eventually carcinogenesis.

-
2. Living habits factors: Poor living habits, including long-term smoking, drinking, inactivity, obesity, etc. can increase the risk of stomach cancer.
 3. Infection factors: A large number of epidemiological studies show that *Helicobacter pylori* infection is positively correlated with the prevalence of stomach cancer. Other studies have shown that *Helicobacter pylori* can promote cell proliferation and lead to the mutation of key oncogenes. Persistent *Helicobacter pylori* infection can induce chronic gastritis and atrophic gastritis, and then cause the metabolic changes of gastric mucosal cells, leading to intestinal metaplasia, gastric epithelial dysplasia and other pre-cancerous lesions.
 4. Stomach disease experience: Some stomach diseases can significantly increase the risk of stomach cancer, including chronic atrophic gastritis, gastric ulcer, gastric polyps and superficial gastritis. Among them, gastric ulcer, chronic atrophic gastritis are most closely related to stomach cancer. Atrophic gastritis is widely regarded as a precursor of stomach cancer. The further development of inflammation may lead to cancer.



Uncontrollable factors

1. Age: Middle-aged and elderly people are more likely to develop stomach cancer.
2. Gender: There is an obvious gender imbalance in the incidence of stomach cancer. The incidence of stomach cancer in men is much higher than that in women.
3. Geographical environmental factors: The studies show that there are significant differences in the incidence of stomach cancer in different regions and races. Some data indicate that stomach cancer is more common in high latitudes. The farther the country is from the equator, the higher the incidence of stomach cancer. Some data also indicate that its incidence is related to coastal factors. There are actors of different eating habits. Geographical and chemical factors and the possibility of carcinogens in the environment should also be considered.
4. Family history: People with a family history of stomach cancer have a higher prevalence.

Knowledge



Time of onset

Most of them occur after middle age, mostly in 40 ~ 60 years old.



Prevalence

Among the tumors of the digestive system in China, stomach cancer has the highest incidence. According to a report published by the World Health Organization, the annual incidence of stomach cancer in the worldwide is 14 per 100,000 people. The incidence of stomach cancer varies greatly worldwide. South Korea, Japan, and East Asia have the highest incidence. Other high-incidence areas

include parts of Eastern Europe and Latin America, while Western Europe and the United States have lower incidences.

Typical symptoms

Early stomach cancer is mostly asymptomatic or only mild symptoms. When the clinical symptoms are obvious, the disease is already at an advanced stage. Therefore, we must be very alert to the early symptoms of stomach cancer to avoid delay in diagnosis and treatment.

1. Early manifestations: Epigastric discomfort is the most common initial symptom in stomach cancer. About 80% of patients have this manifestation, which is similar to dyspepsia. If abdominal pain occurs, it is generally mild and irregular, and can not be relieved after eating. Nearly 50% of patients with stomach cancer have obvious symptoms of anorexia or anorexia. Some patients restrict their eating because eating too much will cause abdominal bloating or abdominal pain. Unexplained anorexia and weight loss are probably to be the initial symptoms of early stomach cancer, which should be paid attention to. Patients with early stomach cancer generally have no obvious positive signs. Most patients only have deep tenderness in the upper abdomen, except for the weak general condition.
2. Late manifestations: When stomach cancer develops and expands, especially when the infiltration penetrates the serosa and invades the pancreas, persistent severe pain can occur and radiate to the waist and back. The absorption of cancer toxin can make patients lose weight, fatigue and anemia, and finally show cachexia. After the cancer grows up, obstruction symptoms may appear. Cardia or fundus of stomach carcinoma may cause dysphagia, stomach cancer in the antrum may cause pyloric obstruction, and a lump may be palpable in the abdomen. When an ulcer is formed on the surface of the cancer, hematemesis and melena. As for the appearance of metastasis, such as pre rectal mass, umbilical mass, supraclavicular lymph node enlargement and ascites, it is also the evidence of advanced stomach cancer.

Treatment

1. Surgical treatment: Surgical resection is still the only option for radical treatment of early stomach cancer, and it is also the main method to treat stomach cancer. For a long time, due to the late discovery of stomach cancer, most of them belong to advanced tumors, the surgical effect is poor, and the 5-year survival rate has been maintained at about 30%. Therefore, it is necessary to strengthen the attention to the symptoms of early stomach cancer and the monitoring of high-risk groups, so as to improve the detection rate of early stomach cancer.
2. Chemotherapy: The surgical resection rate of stomach cancer is 50% ~ 60%, while the cure rate is only 20% ~ 40%. Most of them can relapse and often die of local recurrence and distant metastasis. Because the admitted cases are relatively late, and in order to improve the curative effect of operation, the comprehensive therapy of radical operation and chemotherapy can be used. For palliative resection cases, more long-term chemotherapy is needed after surgery. About 40% of patients can not be resected with tumors. For advanced cases that can not be operated, chemotherapy is the main treatment.

3. Immunotherapy: Immunotherapy combined with chemotherapy is considered to prolong life, and freeze-dried Bacille Calmette-Guerin (BCG) is the most commonly used.
4. Intraoperative radiation: Intraoperative radiotherapy can improve the 5-year survival rate.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2294008	PSCA	CT	1.46	0.97
RS10509671	intergenic	GT	1.34	0.99
RS2070600	AGER	CT	1.27	0.99
RS2274223	PLCE1	AG	0.79	0.99
RS7624041	intergenic	AG	1.21	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to stomach cancer. These literatures have found some polymorphic loci related to the risk of the disease by stomach cancer. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

 Applicable situation

The health risks of stomach cancer item uses the 8 polymorphic loci of *AGER*, *SERPINE1*, *PLCE1*, *IL17A*, *PSCA*, *MPO* gene. The test results show whether the user has a higher prevalence of stomach cancer than all users. The test cannot represent the user's real risk of stomach cancer. The test is mainly based on the research of Asian, and other populations are only for reference.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of stomach cancer.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of stomach cancer from the genetic level, which does not represent your real health status, nor does it involve the judgment of your other health status, living habits, etc.
3. If you have a family history of stomach cancer, or you think that you are likely to suffer from stomach cancer, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 13 polymorphic loci related to stomach cancer risk, including rs2070600, rs2227692, rs2274223 and so on.

Population samples

The basis of the test is mainly from the research of Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect stomach cancer.

Idiopathic Pulmonary Fibrosis (IPF)

Also known as cryptogenic fibrosing alveolitis and diffuse fibrosing alveolitis, is an unexplained pulmonary interstitial inflammatory disease.

My Genetic Risk Is Average For The Population

0.68 times

Average incidence rate is 10 per 100,000 in the population

Suggestions

1. Reject tobacco: If you already smoke, quit smoking.
2. Take protective measures: avoid exposure to metal powder, sawdust, coal powder, silica and hay ash.

Influencing factors



Controllable factors

1. Smoking: currently considered the most recognized and accepted risk factor.
2. Environmental exposure: such as exposure to metal powder, wood chips, coal powder, silica, hay ash, etc., will also increase the risk.



Uncontrollable factors

1. Gender: the incidence rate of men are slightly higher than women.
2. Age: Most patients are over 50 years old, and the peak incidence is 70 years old. Later as the age increases, the incidence rate increases.

Knowledge

Time of onset

Most patients are over 50 years old, and the peak incidence is 70 years old.

Prevalence

The estimated incidence rate is 3 to 6 per 100,000, and it is 27 to 29 per 100,000 in some countries.

Typical symptoms

1. Symptoms: The disease is often insidious onset, with dry cough and difficulty breathing at first. For most patients, progressive dyspnea is the main and most prominent symptom. Another common symptom is an irritating dry cough, which is often ineffective against antitussives. Generally, there are no extrapulmonary manifestations, and there may also be weight loss, discomfort, fatigue, etc., fever is rare, and high fever may occur when combined with infection. Sometimes joint soreness and chest pain are rare.
2. Signs: Breathing rate increases faster at rest, and severely ill patients have cyanosis and hypoxemia. On auscultation of both lungs, small inspiratory crackles can be heard, usually located at the base of the lungs. As the disease progresses in 80% of patients, the lung rales can spread to the apex of the lung; bronchial breath sounds indicate lung consolidation; 25% to 50% of patients have clubbing.

Treatment

1. Medication: corticosteroids.
2. Adjuvant treatment: oxygen inhalation can reduce hypoxemia caused by exercise and improve exercise capacity.
3. Lung transplantation: Single lung transplantation is an important treatment option for certain medical treatments of relapsed end-stage pulmonary fibrosis.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS112087793	intergenic	CC	1.80	1.00
RS2736100	TERT	AA	1.45	0.99
RS2034650	IVD	AA	1.00	0.69
RS2076295	DSP	TT	1.43	0.99

RS1981997

MAPT

GG

1.41

1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to idiopathic pulmonary fibrosis. By studying the genotype differences between patients with idiopathic pulmonary fibrosis and normal people, these literatures have found some polymorphic sites associated with the risk of idiopathic pulmonary fibrosis. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details



Applicable situation

A health risk item for idiopathic pulmonary fibrosis uses 17 genetic and intergenic polymorphic loci of genes such as *DPP9*, *ATP11A*. The results show whether the user had a higher prevalence of idiopathic pulmonary fibrosis than all users in the genotype combination of the 17 polymorphic loci tested, which does not represent the user's real risk of idiopathic pulmonary fibrosis. The test is based mainly on Caucasian and Asian studies, and other populations are for reference only.



Notification

1. The test may not cover all genes or loci that could contribute to the risk of idiopathic pulmonary fibrosis.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results



Notification

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1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing idiopathic pulmonary fibrosis at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
 3. If your family has a family history of idiopathic pulmonary fibrosis, or if you feel you have a risk of developing idiopathic pulmonary fibrosis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 17 polymorphic loci associated with the risk of idiopathic pulmonary fibrosis, including rs121610495, rs1278769 and rs2076295.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with idiopathic pulmonary fibrosis.

Renal Cancer

Renal cancer is a pathologically heterogeneous disease. It originates in renal tubular epithelial cells and can occur in any part of the renal parenchyma.

My Genetic Risk Is Average For The Population

0.67 times

Average incidence rate is 3 per 100,000 in the population

Suggestions

1. Precautions: avoid exposure to toxic substances such as aromatic amines, aromatic hydrocarbons, aflatoxins, nitrosyl compounds, alkyl compounds, hydrazine, lead, cadmium, etc.
2. Safe medication: use anti-cancer drugs under the guidance of a doctor.
3. Control food additives: reasonable intake of food additives.
4. Reject tobacco: quit smoking if you already smoke.

Influencing factors



Controllable factors

1. Toxic substances: aromatic amines, aromatic hydrocarbons, aflatoxins, nitros compounds, alkyl compounds, hydrazine, lead, cadmium, etc.
2. Anti-cancer drugs: phenacetins, amphetamines, diuretics, etc.
3. Food additives: coffee and food additives such as potassium bromate, etc.
4. Smoking: dimethylnitrosamine in tobacco induces kidney cancer.



Uncontrollable factors

1. Gender: the ratio of male to female incidence is 2:1.
2. Age: the main age of onset is 40 to 70 years old, sometimes in younger groups.

3. Have a history of atopic diseases: For example, families with retinal angiomas have a higher incidence.

Knowledge

 Time of onset

The main age of onset of kidney cancer is 40 to 70 years old, sometimes occurs in younger groups.

 Prevalence

The annual incidence of the disease is 0.037% to 0.01%.

 Typical symptoms

The clinical manifestations of renal cell carcinoma range from typical triad, hematuria, pain and possible kidney lumps to more insidious peritumoral syndrome. Triad often occurs in the late stage, usually only 10% of patients develop typical symptoms, most of which are accidental.

 Treatment

The most effective treatment for kidney cancer is radical nephrectomy. The scope of resection includes the perirenal fascia and fat, upper ureter and hilar lymph nodes. For upper pole tumors, the ipsilateral adrenal gland should be removed.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS699947	intergenic	CC	1.00	0.99
RS7105934	intergenic	GG	1.00	0.99
RS1049380	ITPR2	TT	1.00	0.99
RS12105918	ZEB2	TT	1.00	0.99
RS3025039	VEGFA	CC	1.00	1.00

Risk calculation

The risk value of the test is calculated based on published academic literatures related to kidney cancer. By studying the genotype differences between kidney cancer patients and normal people, these literatures have found some polymorphic sites associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of kidney cancer item uses 5 polymorphic loci in and between genes of *ITPR2*, *ZEB2*, *VEGFA*. The test results indicate whether the user has a higher prevalence of kidney cancer in the combination of genotypes at the 5 polymorphic loci tested compared to all users. This test does not represent the user's true risk of kidney cancer. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of kidney cancer.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of kidney cancer at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
 3. If you have a family history of kidney cancer, or you think that you are likely to suffer from kidney cancer, it is recommended that you consult an authoritative health professional or clinician for
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help.

 Test content

The test includes 5 polymorphic loci related to the risk of kidney cancer, including rs1049380, rs12105918, rs3025039, rs699947, and rs7105934.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to kidney cancer.

Endometriosis

Endometriosis refers to the appearance of the endometrium outside the uterus.

My Genetic Risk Is Average For The Population

0.67 times

Average incidence rate is 10000 per 100,000 in the population

Suggestions

1. Avoid multiple uterine cavity operations, including abortion and curettage, and reduce the inevitable endometrial implantation during the operation.
2. Timely discover and treat diseases that cause menstrual blood retention, such as congenital genital malformations, atresia, stenosis and secondary cervical adhesions, vaginal stenosis, etc., to prevent menstrual blood reflux.
3. Right-age marriage and childbirth, the best childbearing age is 25 to 35 years old.

Influencing factors

Controllable factors

1. Uterine cavity surgery: curettage, artificial abortion, etc. in the early and mid-term pregnancy may cause endometrium transplantation to certain parts.
2. Menstrual blood retention: congenital malformations of the genital tract, atresia, stenosis and secondary cervical adhesions, vaginal stenosis and other diseases can cause menstrual blood retention, leading to ectopic endometrial tissue growth in the pelvic cavity.
3. Childbearing age: The incidence of women who give birth late is significantly higher than those who give birth early.

Uncontrollable factors

Family history: Endometriosis has a certain genetic predisposition and family clustering. Most people with family history suffer from this disease.

Knowledge

Time of onset

Childbearing age is the age at which endometriosis occurs, most of which are between 25 and 45 years old.

Prevalence

In recent years, the incidence of endometriosis has been on the rise, and it is about 10% in women of childbearing age.

Typical symptoms

1. Dysmenorrhea: It presents secondary sex with progressive aggravation, which often occurs when menstrual cramps occur.
2. Menstrual abnormalities: manifested as prolonged menstrual periods and increased menstrual flow.
3. Infertility: The infertility rate is as high as 40%.
4. Discomfort during intercourse: it can cause deep intercourse pain.

Treatment

Endometriosis emphasizes individualized treatment, and the treatment methods mainly include expectant treatment, drug treatment, and surgical treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1800871	IL10	AA	0.27	0.92
RS9340799	ESR1	AG	2.67	0.90
RS2235529	WNT4	CT	1.29	0.99
RS9434741	CHD5	AA	1.00	0.94
RS10965235	CDKN2B-AS1	CC	1.00	0.98

Risk calculation

The risk value of this test is calculated based on published academic literature related to endometriosis. By studying the genotype differences between patients with endometriosis and normal people, these literatures have found some polymorphic sites associated with the risk of endometriosis. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

A health risk item for endometriosis uses 6 polymorphic loci of the *CDKN2B-AS1*, *WNT4*, *ESR1* gene. The results show whether the user had a higher prevalence of endometriosis than all users in the genotype combination of the 6 polymorphic loci tested, which does not represent the user's real risk of endometriosis. The test is based mainly on Caucasian and Asian studies, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of endometriosis.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing endometriosis at a genetic

level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.

3. If you have a family history of endometriosis, or if you feel you have a risk of developing endometriosis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 6 polymorphic loci associated with the risk of endometriosis, including rs10965235, rs2235529, and rs9340799.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with endometriosis.

Hashimoto's Thyroiditis

Also known as chronic lymphocytic thyroiditis, is an autoimmune disease and is the most common cause of goiter with hypothyroidism.

My Genetic Risk Is Average For The Population

0.66 times

Average incidence rate is 1000 per 100,000 in the population

Suggestions

1. According to the recommendations of the World Health Organization, the daily intake of iodine for adults is 150µg. Avoid excessive consumption of dried kelp (36240µg/100g iodine content), dried seaweed (4323µg/100g iodine content) and other seafood.
2. During the flu season, try to avoid crowded places as much as possible. It is recommended to get flu vaccine.

Influencing factors



Controllable factors

1. High iodine intake: epidemiological studies have shown that the disease is more common in iodine-sufficient areas than iodine-deficient areas, and it occurs more frequently as iodine intake increases.
2. Infection: viral or bacterial infection may induce an autoimmune response through various mechanisms and increase the risk of Hashimoto's thyroiditis.



Uncontrollable factors

1. Family history: the family of a patient with Hashimoto's thyroiditis usually has a history of thyroid disorders or autoimmune diseases.
2. Gender: the disease is more common in women, and the probability of women suffering from Hashimoto's thyroiditis is about 8-15 times than men.

Knowledge

Time of onset

It can occur at any age, and most commonly occurs in women aged 30-60.

Prevalence

There is no exact statistics on the incidence rate, but in recent years, domestic and foreign data indicate that the incidence has increased, with an average incidence rate of 1%.

Typical symptoms

Slowly progressive bilateral diffuse thyroid enlargement, tough texture, can also be confined to one side. In addition, it may be accompanied by changes in thyroid function, manifested as hyperthyroidism, normal or hypothyroidism.

Treatment

There is no effective treatment currently, medical treatment of hyperthyroidism or hypothyroidism caused by this disease, surgical treatment of oppressive symptoms caused by goiter, or possible malignant thyroid lesions.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS5912838	intergenic	AA	1.59	0.98
RS1521	intergenic	CT	1.55	0.98
RS1024161	intergenic	CT	1.31	0.98
RS1265883	SLAMF6	AA	1.00	0.98
RS2476601	PTPN22	GG	1.00	1.00

Risk calculation

The risk value of the test is calculated based on published academic literature on Hashimoto's thyroiditis. By studying the genotype differences between Hashimoto's thyroiditis patients and normal people, these literatures found some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details



Applicable situation

The Hashimoto's thyroiditis item uses *PTPN22*, *SLAMF6* genes, and 5 polymorphic loci between genes. The results show whether the user had a higher rate of Hashimoto's thyroiditis than the genotype combination of the 5 polymorphic loci tested compared to all users, and that the test did not represent the user's real risk of Hashimoto's thyroiditis. The basis of the test is mainly from the research of Caucasian and Chinese, other races are for reference only.



Notification

1. The test may not cover all genes or loci that could contribute to a risk of Hashimoto's thyroiditis.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results



Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Hashimoto's thyroiditis at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
3. If you have a family history of Hashimoto's thyroiditis, or if you feel you have a risk of Hashimoto's thyroiditis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 5 polymorphic loci related to the risk of Hashimoto's thyroiditis, including rs2476601, rs1265883 and rs1024161.

Population samples

The study used in this test mainly Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with Hashimoto's thyroiditis.

Renal Calculi

Renal calculi refer to stones that occur in the renal calyx, renal pelvis, and the junction between the renal pelvis and the ureter.

My Genetic Risk Is Average For The Population

0.64 times

Average incidence rate is 10000 per 100,000 in the population

Suggestions

- Red flags: **Small crystals on the kidney** found during a B-ultrasound examination, frequent urination, low back pain
- Common symptoms: Dull waist pain, renal colic, hematuria, urinary tract obstruction, etc.
- High-risk population: Males, young adults, and hot area inhabitants
- Dangerous lifestyle: Inadequate drinking of water, drinking hardened water, taking too much spinach, coffee, etc. Food that induces calculus, supplementing calcium, taking too much protein and sugar, etc.
- Hyperuricemia and other Diseases and some Drug use can also cause renal calculi
- How to reduce the risk:
 - Drink plenty of water, exercise moderately, drink about 2-2.5 liters of water per day
 - Avoid eating a lot of foods that can induce stones (such as spinach, coffee, etc.), and reduce sugar intake
 - **Lemon juice** (containing **Citrate ion**), orange juice, wine, etc. are effective in preventing renal calculi
 - If you need calcium supplements, use calcium citrate products to reduce the risk of renal calculi
 - People with high uric acid should limit protein intake
- Treatment of renal calculi: Generally, kidney stones smaller than 5 mm can be discharged spontaneously in the urine. Larger stones may cause severe obstruction, or have hematuria, infection, etc., requiring lithotripsy
- The Heritability of renal calculi is approximately 56%

* If you have a family history of renal calculi, or you already have renal calculi, or you think you are likely to have renal calculi, or need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors

Controllable factors

1. Drinking too little water leads to increased excretion of urine liquid crystal substances, e.g., hypercalciuria, hyperoxaluria, hyperuricemia, homocystinuria, xanthinuria.
2. Effects of diet and medication on renal calculi: Drinking hardened water, malnutrition, lack of vitamin A can cause urothelial to fall off and form the core of the stone, and taking triamterene and acetazolamide may cause renal calculi.
3. Effects of urinary tract infection on renal calculi: Persistent or repeated urinary tract infections can cause infectious stones.

Uncontrollable factors

1. Gender: More male patients.
2. Age: It can occur at any age, most of which occur in young adults, accounting for about 80% of 21-50 years old.
3. Regional: It is more common in tropical and subtropical regions, indicating that hot climate and excessive sun can increase the incidence of renal calculi.

Knowledge

Time of onset

Renal calculi can occur at any age, most of which occur in young adults, accounting for about 80% of 21-50 years old.

Prevalence

The incidence of renal calculi is regional, and more common in the tropics and subtropics, indicating that hot weather and excessive sun exposure can increase the incidence of renal calculi.

Typical symptoms

1. Asymptomatic: Most of them are calculus of the kidney. It is usually found during a B-ultrasound examination, and also urine test is negative or there are a small amount of red and white blood cells.
2. Dull pain in the waist: mostly large stones such as cast stones in the renal pelvis, hematuria may occur after strenuous exercise.
3. Renal colic: usually small stones, microscopic or gross hematuria, obvious percussion pain in the kidney area. When the pain occurs, the patient is pale, with cold sweat, rapid weak pulse or even blood pressure drop, and is often accompanied by gastrointestinal symptoms such as nausea, vomiting, and abdominal distension.

- 4. History of stone discharge: When pain and hematuria occur, sand or small stones may be discharged with urine. When the stone passes through the urethra, the urine flow is blocked and the urinary tract feels tingling. After the stone is discharged, the urine flow is immediately restored and the patient feels relaxed and comfortable.
- 5. Symptoms of infection: pyuria may occur in co-infection, and symptoms of chills, fever, low back pain, frequent urination, urgency, and dysuria may occur in acute attacks.
- 6. Renal insufficiency: Obstruction caused by a kidney stone on one side can cause hydronephrosis and progressive renal dysfunction on that side; bilateral kidney stones or solitary kidney stones can cause obstruction and can develop into uremia.
- 7. Urinary obstruction: Bilateral kidney stones cause urinary tract obstruction on both sides, solitary kidney or only functional renal stone obstruction can cause urinary obstruction, one kidney stone obstruction, and the opposite side can have reflex urinary obstruction.
- 8. Waist mass: When the stone obstruction causes severe hydronephrosis, the mass can be palpated in the waist or upper abdomen.

 Treatment

- 1. General treatment: Kidney stones smaller than 5 mm can often be discharged spontaneously in the urine. Sometimes the stones cause blockage and severe pain. Analgesics such as diclofenac, or smooth muscle antispasmodics such as hyoscine butylbromide, can be used to dilate the ureter and accelerate the discharge of the stone.
- 2. Surgical treatment: Larger stones may cause severe blockage, or lead to hematuria, infection and other conditions. Lithotripsy may be required.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS219780	CLDN14	CC	1.56	1.00
RS219781	CLDN14	GG	1.51	1.00
RS17216707	intergenic	TT	1.42	1.00
RS4142110	DGKH	TT	1.30	1.00
RS71606723	intergenic	AA	0.81	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is calculated based on published academic literatures related to renal calculi. By studying the genotype differences between renal calculi patients and normal people, these literatures have found some polymorphic sites associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of renal calculi item uses 40 polymorphic loci in and between genes of *RGS14*, *DGKH*, *UMOD*, *CLDN14*. The test results indicate whether the user has a higher prevalence of renal calculi in the combination of genotypes at the 40 polymorphic loci tested compared to all users. This test does not represent the user's true risk of renal calculi. The basis of the test is mainly from the research of Asian and Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of renal calculi.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of renal calculi at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.

3. If you have a family history of renal calculi, or you think that you are likely to suffer from renal calculi, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 40 polymorphic loci related to the risk of renal calculi, including rs11746443, rs1000597, rs4142110, rs4293393, and rs219781.

 Population samples

The basis of the test is mainly from the research of Asian and Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to renal calculi.

Chronic Obstructive Pulmonary Disease (COPD)

Chronic obstructive pulmonary disease is characterized by airflow limitation that is not fully reversible and is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases, particularly tobacco smoke.

My Genetic Risk Is Average For The Population

0.62 times

Average incidence rate is 10000 per 100,000 in the population

Suggestions

1. Smoking is the main risk factor leading to chronic obstructive pulmonary disease, so we should actively quit smoking and avoid second-hand smoke.
2. Avoid burning biofuels in poorly ventilated spaces, such as burning wood for cooking indoors, or starting a stove for heating, to reduce indoor air pollution.
3. Reduce occupational dust and chemical substance inhalation, and labor protection should be done for people who are engaged in exposure to occupational dust (such as coal mines, metal mines, cotton textile industry, chemical industry, etc.).
4. Actively prevent and treat upper respiratory tract infections, such as injecting influenza vaccine in autumn and winter, and avoid crowded places.

Influencing factors



Controllable factors

1. Smoking: Smoking, especially active smoking, is considered to be the most important risk factor for chronic obstructive pulmonary disease. At least 95% of patients with chronic obstructive pulmonary disease are smokers.
2. Air pollution: Long-term exposure to air pollution (SO₂, NO₂) can damage the airway mucosal epithelium and cause inflammation.

-
3. Chemical substances: exposure to occupational dust and chemical substances will increase the risk of chronic obstructive pulmonary disease, such as smoke, industrial waste gas, etc.
 4. Infection: Upper respiratory tract infection is one of the important factors in the occurrence and development of chronic obstructive pulmonary disease.

Uncontrollable factors

Family history: Chronic obstructive pulmonary disease has a certain tendency of family gathering.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of chronic obstructive pulmonary disease at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of chronic obstructive pulmonary disease, or you think that you are likely to suffer from chronic obstructive pulmonary disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 6 polymorphic loci related to the risk of chronic obstructive pulmonary disease, including rs17576, rs3918396, and rs7671167.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to chronic obstructive pulmonary disease.

Knowledge

Time of onset

Most of the onset after middle age.

Prevalence

The global incidence rate over 40 years old has reached 9-10%.

Typical symptoms

1. Chronic cough: Cough is often obvious in the morning.
2. Sputum expectoration: usually white mucus or serous foamy sputum, occasionally bloodshot. The amount of sputum increases during the acute attack, and there may be purulent sputum.
3. Shortness of breath or dyspnea: It appears in the early stage of exertion, and then gradually worsens, so that shortness of breath is felt during daily activities or even at rest. It is a hallmark symptom of chronic obstructive pulmonary disease (COPD).

Treatment

Medication, home oxygen therapy.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS3918396	ADAM33	CC	2.42	1.00
RS3736309	AQP5	AG	0.44	0.94
RS503464	intergenic	AA	0.72	0.99
RS1800469	TGFB1, B9D2	AG	1.32	0.98
RS7671167	FAM13A	CT	0.76	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to chronic obstructive pulmonary disease. These literatures have found some polymorphic loci related to the risk of the disease by studying chronic obstructive pulmonary patients and normal people. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our

products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of chronic obstructive pulmonary disease item uses the 12 polymorphic loci in *AQP5*, *MMP9*, *ADAM33*, *FAM13A* and *TGFB1*. The test results indicate whether the user has a higher prevalence of chronic obstructive pulmonary disease in the combination of genotypes at the 12 polymorphic loci tested compared to all users. This test does not represent the user's true risk of chronic obstructive pulmonary disease. The basis of the test is mainly from the research of Caucasian, Asian, African American, Afro-Caribbean, and Latino American, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of chronic obstructive pulmonary disease.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not applicable to clinical practice.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of chronic obstructive pulmonary disease at the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of chronic obstructive pulmonary disease, or you think that you are likely to suffer from chronic obstructive pulmonary disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 12 polymorphic loci associated with the risk of chronic obstructive pulmonary disease, including rs17576, rs3918396, and rs7671167.

 Limit of detection

The basis of the test is mainly from the research of Caucasian, Asian, African American, Afro-Caribbean, and Latino American.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci related to chronic obstructive pulmonary disease.

Abdominal Aortic Aneurysm (AAA)

It is an irreversible aortic dilation and bulging disease, which manifests as the local diameter of the artery greater than 3 cm or more than 50% of the normal artery diameter. Infrarenal abdominal aortic aneurysm is the most common type of all aortic aneurysms.

My Genetic Risk Is Average For The Population

0.62 times

Average incidence rate is 30 per 100,000 in the population

Suggestions

1. Control the amount of fat, cholesterol and salt in the diet, and eat more high fiber foods such as bamboo shoots, carrots, celery, etc. It is helpful to maintain normal blood glucose, blood lipid and blood pressure levels; Reduce the damage and load of blood vessels; Prevent arteriosclerosis.
2. Refuse active smoking and passive smoking, and avoid exposure to tobacco environment; Limit alcohol intake.
3. Strike a balance between work and rest, and avoid excessive stress level; Maintain communication with relatives and friends; Exercise actively and moderately; Avoid getting angry and impatient.

Influencing factors



Controllable factors

1. Dietary factors: Unbalanced diet, abnormal blood glucose, lipid and blood pressure levels will increase the risk of abdominal aortic aneurysm.
2. Tobacco and alcohol intake: Smokers or passive smokers have an increased risk of abdominal aortic aneurysm. Alcohol raises blood pressure and promotes the increase of certain fatty acids in the blood, which may lead to arteriosclerosis and even abdominal aortic aneurysm.
3. Psychological and emotional factors: People who have prolonged exposure to excessive stress, anxiety, irritability, and emotional agitation have a higher risk of abdominal aortic aneurysm.

Uncontrollable factors

1. Age: The elderly are more likely to develop abdominal aortic aneurysm.
2. Gender: Male has a higher prevalence compared to female.
3. Disease: Hypertension and atherosclerosis can cause abdominal aortic aneurysm.
4. Family history: People with a family history of abdominal aortic aneurysm have a higher prevalence.

Knowledge

Time of onset

Abdominal aortic aneurysm mostly occurs in the elderly, and the incidence has gradually increased since the age of 50.

Prevalence

Studies have shown that the prevalence of abdominal aortic aneurysm is 30 cases per 100,000. The incidence in men is 3-4 times higher than that in women.

Typical symptoms

- Most non-ruptured abdominal aortic aneurysms have occult onset and no obvious symptoms. When the aneurysm is large, it can compress the intestine and cause gastrointestinal symptoms such as abdominal distension, vomiting or discomfort in defecation; The compression of inferior vena cava can cause lower limb swelling and other symptoms of lower limb venous hypertension.
- Threatened rupture or ruptured abdominal aortic aneurysm usually has pain symptoms. The pain site is generally located in the middle abdomen or low back, mostly dull pain, which can last for hours or even days; In severe cases, severe abdominal and back pain, hypotension and pulsatile abdominal mass may occur.

Treatment

- The treatment of abdominal aortic aneurysm is mainly surgical treatment. When the diameter of abdominal aortic aneurysm in men is more than 5.5 cm, or the diameter of abdominal aortic aneurysm in women is more than 5 cm, surgery is recommended.
- The types of surgery were divided into open aortic repair (OAR) or endovascular aortic repair (EVAR). For patients who are in good general condition and can tolerate surgery, OAR is the standard surgical treatment; EVAR has the advantages of minimally invasive and safe, but OAR is still needed when necessary.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS10455872	LPA	AA	0.62	1.00
RS4977574	CDKN2B-AS1	AA	0.69	0.99
RS73015011	intergenic	TT	1.39	1.00
RS7994761	intergenic	AA	1.31	1.00
RS7025486	DAB2IP	AG	1.21	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to abdominal aortic aneurysm. These literatures found some polymorphic sites associated with the risk of the disease by studying the genetic differences between patients with abdominal aortic aneurysm and normal people. We uses the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of abdominal aortic aneurysm item the 30 polymorphic loci on genes such as *DAB2IP*, *PCSK9*, *LPA*, *LPL* and *ERG* and within other intergenic regions. The test results indicate whether the user has a higher prevalence of abdominal aortic aneurysm in the combination of genotypes at the 30 polymorphic loci tested compared to all users. This test does not represent the user's true risk of abdominal aortic aneurysm. The basis of the test is mainly from the research of Caucasian and African, other populations are for reference only.

Notification

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1. The test may not cover all genes or loci that could contribute to a risk of abdominal aortic aneurysm.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of abdominal aortic aneurysm at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of abdominal aortic aneurysm, or you think that you are likely to suffer from abdominal aortic aneurysm, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 30 polymorphic loci related to the risk of abdominal aortic aneurysm, including rs7025486, rs10757278 and rs11591147.

Population samples

The basis of the test is mainly from the research of Caucasian and African.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to abdominal aortic aneurysm.

Myeloproliferative Neoplasm (MPN)

Myeloproliferative neoplasm (MPN) is a group of clonal hematopoietic stem cell diseases characterized by persistent proliferation of one or more lines of myeloid cells in the bone marrow. It includes chronic myeloid leukemia (CML), polycythemia vera (PV), essential thrombocytosis (ET) and primary myelofibrosis (PMF).

My Genetic Risk Is Average For The Population

0.60 times

Average incidence rate is 9 per 100,000 in the population

Suggestions

1. Reject active and passive smoking, and avoid exposure to tobacco.
2. Avoid contact with high-risk carcinogens such as chemical fertilizers, pesticides, stone, rubber, plastic and glass fiber.

Influencing factors

Controllable factors

1. Smoking: A history of smoking or smoking in old age will increase the risk of myeloproliferative neoplasms.
2. Environmental factors: People who have long-term exposure to high-risk pathogens such as benzene and benzene analogs, fertilizers, pesticides, stone, rubber, plastics and glass filaments have a higher prevalence.

Uncontrollable factors

1. Age: Middle-aged and elderly people have a higher risk of myeloproliferative neoplasms.
2. Treatment history: People who have received chemotherapy and radiotherapy in the past have an increased risk of developing myeloproliferative neoplasms.

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3. Family history: People with a family history of myeloproliferative neoplasms have a higher prevalence.
-

Knowledge

Time of onset

It is most common between 60 and 70 years old, and young cases account for about 20% of diagnosed cases.

Prevalence

Myeloproliferative neoplasms is a relatively rare disease with an incidence rate of 9/100,000.

Typical symptoms

Myeloproliferative neoplasms (MPN) are a group of clonal hematopoietic stem cell diseases characterized by the continuous proliferation of one or more lines of myeloid cells in the bone marrow, including chronic myeloid leukemia (CML), polycythemia vera (PV), and essential thrombocythemia (ET) and primary myelofibrosis (PMF) and multiple myeloma (MM).

1. Chronic myeloid leukemia is characterized by the production of a large number of immature white blood cells. These white blood cells accumulate in the bone marrow and inhibit the normal hematopoiesis of the bone marrow, and can spread throughout the body through the blood, leading to anemia, easy bleeding, infection, splenomegaly, unexplained weight loss, night sweats and organ infiltration, etc.
2. Polycythemia vera is characterized by a significant increase in the number of total red blood cells and total blood volume, usually accompanied by an increase in granulocytes and thrombocytosis, increased blood viscosity, and bone marrow hyperplasia. In the late stage, there is often bone marrow fibrosis, extramedullary hematopoiesis, enlarged spleen, distinctive reddish purple skin and mucous membranes, and various vascular and nervous system symptoms.
3. Essential thrombocytosis, characterized by bleeding, thrombosis, and persistent thrombocytosis, accompanied by mild proliferation of other hematopoietic cell lines. This disease is more common in adults over middle age, and occasionally children.
4. Primary bone marrow fibrosis is characterized by anemia, bone marrow fibrous tissue hyperplasia and extramedullary hematopoiesis. Teardrop-like red blood cells can be seen in the peripheral blood, and there is hepatosplenomegaly. Primary bone marrow fibrosis can be accompanied by a series of complications: portal hypertension, splenic infarction, gastrointestinal hemorrhage, spinal cord compression, seizures, hemoptysis, pleural effusion, infectious complications, bone hyperplasia, hypertrophic osteoarthropathy and hyperuricemia.
5. For multiple myeloma, early patients may not have any specific symptoms. Usually, the symptoms are caused by the accumulation of bone marrow cancer cells in the bone marrow, bone erosion

and excessive production of abnormal antibody proteins, which cause a series of symptoms. The four main symptoms are hypercalcemia, renal failure, anemia, and bone lesions. In addition, 34% of patients with multiple myeloma will have amyloidosis.

 Treatment

1. Chronic myelogenous leukemia: Tyrosine kinase inhibitors are generally used to improve the control prognosis of patients.
2. Polycythemia vera: The treatment for this condition is to prevent acute bleeding syndrome. Low-dose aspirin is effective for essential thrombocythemia and polycythemia.
3. Essential thrombocytosis: The treatment method is the same as that of polycythemia vera.
4. Primary myelofibrosis: The methods for myelofibrosis include alleviating anemia, splenomegaly and other symptoms.
5. Multiple myeloma: The main drugs for the treatment of this disease can be divided into two categories, one is the target drug (such as bortezomib), and the other is the immunomodulatory drug (such as thalidomide). In addition, the treatment of multiple myeloma focuses on plasma cell proliferation. Regular use of bisphosphonates can prevent fractures, and erythropoietin can be used to prevent anemia.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS4487645	DNAH11	CC	1.54	0.99
RS139402	intergenic	CC	1.49	0.99
RS10936600	LRRC34	AA	1.44	0.99
RS6599192	intergenic	AG	1.26	0.99
RS17507636	intergenic	CC	1.25	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to myeloproliferative neoplasms. By studying the genotype differences between patients with myeloproliferative neoplasms and normal people, these literatures have found some polymorphic sites associated with the risk of myeloproliferative neoplasms. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk item for myeloproliferative neoplasms uses 18 genetic and intergenic polymorphic loci of genes such as *JAK2*. The results show whether the user had a higher prevalence of myeloproliferative neoplasms than all users in the genotype combination of the 18 polymorphic loci tested, which does not represent the user's real risk of myeloproliferative neoplasms. The test is based mainly on Caucasian studies, and other populations are for informational purposes only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of myeloproliferative neoplasms.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing myeloproliferative neoplasms at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of myeloproliferative neoplasms, or if you feel you have a risk of developing myeloproliferative neoplasms, it is recommended that you consult an authoritative

health professional or clinician for help.

 Test content

The test includes 18 polymorphic loci associated with the risk of myeloproliferative neoplasms, including rs12340895 and rs10936600.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with myeloproliferative neoplasms.

Ankylosing Spondylitis (AS)

It is a chronic progressive disease. Inflammatory back pain and lower back stiffness commonly occur in the early stage of the disease. Disease progression may lead to swelling and pain in waist, back, neck, hip, and joint(s), even spinal deformity and ankylosis in severe cases.

My Genetic Risk Is Average For The Population

0.56 times

Average incidence rate is 300 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Gender: Men have a higher risk of disease than women.
2. Age: It is more likely to occur in adolescence.

Knowledge



Time of onset

It is more likely to occur in adolescence, with a peak of 20-30 years old.



Prevalence

The incidence of this disease in different ethnic groups in the world is different, generally 0.1%-1.4%. Men have a higher risk than women.



Typical symptoms

Early onset is generally mild systemic symptoms, such as fatigue, weight loss, anorexia, etc. Early stage mainly includes intermittent pain and stiffness of the sacroiliac joint, especially after getting up in the morning or after exercise. The pain will generally worsen further, involving thoracic vertebrae, cervical vertebrae and other parts. Other symptoms may include joint swelling, redness of eyes,

angina pectoris, etc. In the late severe stage, the whole spine and lower limbs become rigid and arched, and the body flexes forward.

Treatment

There is currently no cure for this disease, but there are ways to relieve the symptoms. For example, drugs can be used to relieve pain and delay disease progression; Therapeutic exercises can relieve stiffness and pain. In late stages, surgery can be used to correct deformities.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS10050860	ERAP1	CC	22.94	0.93
RS6759298	intergenic	CC	1.66	0.99
RS10865331	intergenic	GG	1.59	0.99
RS7115956	intergenic	CC	1.52	0.99
RS2910686	intergenic	CC	1.47	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to ankylosing spondylitis. By studying the genotype differences between patients with ankylosing spondylitis and normal people, these literatures have found some polymorphic sites associated with the risk of ankylosing spondylitis. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of ankylosing spondylitis item the 43 polymorphic loci on genes such as *ERAP1*, *ERAP2*, *LNPEP*, *IL23R* and *TNFRSF1A* and within other intergenic regions. The results show whether the user had a higher prevalence of ankylosing spondylitis than all users in the genotype combination of the 43 polymorphic loci tested, which does not represent the user's real risk of ankylosing spondylitis. The basis of the test is mainly from the research of Caucasians, Asians and Latinos, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of ankylosing spondylitis.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing ankylosing spondylitis at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of ankylosing spondylitis, or if you feel you have a risk of developing ankylosing spondylitis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 43 polymorphic loci associated with the risk of ankylosing spondylitis, including rs2303138, rs10050860 and rs30187.

Population samples

The basis of the test is mainly from the research of Caucasians, Asians and Latinos.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with ankylosing spondylitis.

Primary Adrenal Hypofunction

Primary adrenal hypofunction is a disease in which adrenal cortex hormones are insufficiently secreted due to the destruction of bilateral adrenal glands.

My Genetic Risk Is Average For The Population

0.56 times

Average incidence rate is 12 per 100,000 in the population

Suggestions

1. Avoid infections such as tuberculosis and HIV.
2. Some patients with Addison's disease can benefit from a high-sodium diet. Please consult medical and nutrition experts for specific sources and dosage of sodium. At the same time, treatment with glucocorticoids can increase the risk of osteoporosis. For patients using high-dose glucocorticoids, supplementation of calcium and vitamin D can reduce the risk of osteoporosis.

Influencing factors



Controllable factors

1. Infection: Specific infections (such as tuberculosis) can cause Addison's disease. Patients with HIV/AIDS have a higher risk of developing the disease.
2. Diet: The symptoms of Addison's disease may include hyponatremia, hyperkalemia, and hypotension. Diet has an impact on the condition.



Uncontrollable factors

1. Gender: The incidence of Addison's disease is higher in women.
2. Age: Addison's disease usually occurs between 30 and 50 years old.
3. Autoimmune diseases: In developed countries, 8-9 of every 10 cases of Addison's disease are caused by autoimmune diseases.
4. Family history: Addison's disease has a certain family genetic predisposition.

Knowledge



Time of onset

It can occur at any age, and most commonly occurs in women aged 30-50.



Prevalence

In developed countries, the incidence is about 100 to 140 per million.



Typical symptoms

1. Chronic or long-term fatigue, muscle weakness, loss of appetite, weight loss, abdominal pain.
2. People with Addison's disease may also have darkening of their skin. This darkening is most visible on scars, skin folds, pressure points (such as the elbows, knees, knuckles, and toes), lips and mucous membranes.



Treatment

Adrenal hypofunction mainly uses hormone replacement therapy. Replace cortisol produced by the adrenal gland with steroids. Commonly used drugs are hydrocortisone, and less commonly used are prednisone and dexamethasone.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS12150220	NLRP1	AA	2.96	1.00
RS231806	intergenic	CG	0.74	0.99
RS7565213	intergenic	AG	0.77	0.99
RS12917716	intergenic	CG	0.78	0.99
RS3757247	BACH2	CT	1.20	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to Addison's disease. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype difference between patients with Addison's disease and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Addison's disease health risk item uses 8 polymorphic loci on and between genes such as *BACH2*, *PTPN22*, *CTLA4*, *NLRP1*. The test results indicate whether the user has a higher prevalence of primary hypoadrenocorticism in the genotype results at the 8 polymorphic loci tested compared to all users. The test cannot represent the user's real risk of Addison's disease. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of Addison's disease.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only predicts your risk of Addison's disease at the genetic level, it does not mean your true health status, nor does it involve the assessments on your other health conditions, living habits, etc.
-

3. If you have a family history of Addison's disease, or you think that you are likely to suffer from Addison's disease, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 8 polymorphic loci related to the risk of Addison's disease, including rs12150220.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all genes or loci that affect Addison's disease.

Sudden Cardiac Death (SCD)

Sudden cardiac death refers to unexpected death of the heart. Regardless of whether the patient has a past history of heart disease, cardiac arrest occurs within 1 hour of acute onset of symptoms and causes sudden interruption of cerebral blood flow and loss of consciousness. If the patient is not treated in time, biological death will occur.

My Genetic Risk Is Average For The Population

0.53 times

Average incidence rate is 27 per 100,000 in the population

Suggestions

- Common symptoms: sudden loss of consciousness without aura, fainting
- High-risk population: Long-term staying up late, obesity, being excessively emotional, smoking, drinking, elderly people, patients with heart disease, having a family history of sudden cardiac death
- How to reduce the risk:
 - Strike a proper balance between work and rest to avoid overwork
 - Relieve stress: Avoid being in a state of high pressure for a long time, and resolve negative emotions in time
 - Maintain normal blood pressure, blood glucose and blood lipid levels
 - Weight control: Keep BMI between 18.5~23.9, avoid sitting for a long time, and exercise moderately
 - Balanced diet: Eat more fruits and vegetables, drink less alcohol, and eat less high salt, high fat, high calorie foods
 - Quit smoking: Refuse active smoking, stay away from second-hand smoke
 - Protective food: Consider supplementing **Coenzyme Q10**

* If you have a family history of sudden cardiac death, or you think that you are likely to suffer from sudden cardiac death, or need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors

Controllable factors

1. Unhealthy lifestyle habits: smoking, alcoholism, staying up late, overwork, etc.
2. Being excessively emotional, such as the extreme grief and happiness, the rage, etc.
3. Studies have shown that exertive defecation can increase sympathetic-adrenaline nerve tension, cause tachycardia, vasoconstriction, hypertension, etc., increase the burden on the heart, and induce sudden death.
4. Unfamiliar with or pay no attention to the aura symptoms before sudden death; patients with coronary heart disease and other heart-related primary diseases do not pay attention to treatment and maintenance.

Uncontrollable factors

1. Gender: Men have a higher risk of disease than women.
2. Age: The elderly are more likely to get sick.
3. Season and time: Studies have shown that most sudden cardiac deaths occur in winter and spring, and less in summer and autumn. Sudden death occurs more in the morning and less at night.

Knowledge

Time of onset

The disease can occur at any time, especially in the elderly.

Prevalence

According to statistics, the incidence is about 27 cases per 100,000 people.

Typical symptoms

The typical symptoms of sudden cardiac death are short course of disease and rapid death. Part of sudden death patients will have symptoms such as asthenia, panting, dizziness, palpitation, chest distress, chest or upper abdomen pain, transient loss of consciousness, convulsions, nausea, and vomiting, etc.; some sudden death patients may die suddenly without any symptoms.

Treatment

On-site cardiopulmonary resuscitation is the best treatment for sudden cardiac death.

For some patients with heart disease, besides the standardized treatment of existing heart disease, it is currently recognized that there are four major measures that can significantly reduce the incidence of sudden cardiac death. They are subcutaneous implantable cardioverter-defibrillator (ICD), the

public automated external defibrillator (AED), the β -blockers (such as metoprolol), and timely cardiopulmonary resuscitation at the scene of sudden death.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS16847548	intergenic	CT	1.31	0.98
RS174230	BAZ2B	CC	1.00	0.91
RS4665058	BAZ2B	CC	1.00	0.94

Risk calculation

The risk value of this test is calculated based on published academic literatures related to sudden cardiac death. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences between the genotypes of patients with sudden cardiac death and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Risk Assessment Item for sudden cardiac death uses 3 polymorphic loci on the *BAZ2B* gene and the intergenic regions. The test results, genotype combinations at the 3 polymorphic loci detected, only indicate whether the user is more prone to sudden cardiac death. The test cannot represent the user's real risk of sudden cardiac death. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test does not cover all genes or loci that increase the risk of sudden cardiac death.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The onset and development of the disease are determined by multiple factors such as genes and environment. This study only screens whether you are carrying the disease-causing gene or locus.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have glucose-6-phosphate dehydrogenase deficiency.
4. If you have a family history of sudden cardiac death, or you think that you are likely to suffer from sudden cardiac death, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 3 polymorphic loci associated with sudden cardiac death, such as rs16847548, rs174230 and rs4665058.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test does not cover all genes or loci associated with sudden cardiac death.

Spontaneous Urticaria

It is a localized edema reaction due to the expansion of small blood vessels in the skin and mucosa and the increase of permeability without obvious external stimulation, which is manifested by the appearance of red patches (also called hives or wheals) of different sizes on the skin surface, often accompanied by pruritus and redness.

My Genetic Risk Is Average For The Population

0.52 times

Average incidence rate is 1400 per 100,000 in the population

Influencing factors



Uncontrollable factors

1. Diseases: Rheumatic fever, rheumatoid arthritis, systemic lupus erythematosus, malignant tumors, metabolic disorders, endocrine disorders, autoimmune thyroiditis, abnormal blood coagulation and other diseases.
2. Allergic history: Having suffered from urticaria, vascular edema or other allergic reactions (due to food, drugs, vaccination, etc.) may increase the risk of disease.
3. Genetic factors: A family history of urticaria, angioedema or hereditary angioedema may increase the risk of disease.

Knowledge



Time of onset

The disease can occur at any age, with 20-49 years of age being the most common.



Prevalence

A large meta-analysis in 2019 showed that the prevalence of chronic urticaria in Asia was about 1.4%, higher than that in Europe (0.5%) and North America (0.1%).

Typical symptoms

The typical symptoms of spontaneous urticaria may be limited to the skin, but some patients have vascular edema or systemic symptoms.

- **Skin symptoms:** Urticaria may present red and swollen plaques (also called hives or wheals) of different sizes and shapes. It can occur in any skin area of the body. The skin area under the pressure of clothing (such as the skin at the belt) or the area where the skin rubs against each other (such as the armpit and thigh root) is sometimes more serious. When the wind mass appears, it is often accompanied by pruritus. Within a few minutes or hours, the edema and erythema are reduced, and gradually return to normal without leaving traces.
- **Vascular edema:** Local redness and swelling may occur in the lips, eyelids, tongue, limbs and other parts. The onset usually lasts for several minutes to several hours, and then gradually subsides within 1-3 days. The inflamed area is often accompanied by slight pain, numbness or tingling.
- **Systemic symptoms:** Some patients have systemic symptoms, including headache, fatigue, joint pain/swelling, chest tightness, flushing, vomiting, abdominal pain, palpitation, etc., and severe patients may have shock.

Treatment

At present, most of the existing treatments can not cure spontaneous urticaria, but can help alleviate and eliminate symptoms. Clinically, a step-wise treatment is advocated:

- At the initial treatment, a second-generation antihistamine was given at the standard treatment dose (adult dose) to control the disease within 1-2 weeks.
- If the symptoms cannot be fully controlled in the first stage of treatment within 1-2 weeks, the doctor shall adjust the dosage, change the medication or use different drugs in stages, and gradually reduce the medication or extend the medication period after the condition is controlled, so as to maintain the treatment at the lowest dose.
- If the symptoms are not controlled after multi-stage treatment, the doctor can diagnose whether it is refractory chronic spontaneous urticaria or induced urticaria, and consider using other special therapies or various anti-inflammatory and immunosuppression drugs for treatment.

Factors that may aggravate urticaria

At present, no clear cause(s) has been identified to explain the onset of spontaneous urticaria, but some factors may aggravate its symptoms. Especially for patients with spontaneous urticaria, it is recommended to avoid exposure to the following:

- **Food:** Intake of some Particular food.
- **Infection:** Virus, bacteria, fungi, parasites infection.
- **Inhalation or contact:** Allergen that is easy to trigger hypersensitivity after inhalation by respiratory tract or contact with skin.
- **Mental factors:** Excessive tension, anxiety and fatigue.
- **Physical stimulation:** Thermal stimulation or cold stimulation, ultraviolet ray, skin compression, moist environment.
- **Drugs:** Vaccines, NSAIDs, serum preparations, Antibiotics, Drugs containing histamine release, and Drugs containing some additives.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS708777	intergenic	AG	1.44	0.97
RS453755	intergenic	GG	1.00	0.91
RS34141382	intergenic	TT	1.00	0.94

Risk calculation

The risk value of this test is calculated based on published academic literatures related to spontaneous urticaria. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype difference between spontaneous urticaria patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk item of spontaneous urticaria disease uses the 3 polymorphic loci on *HLA-DQA1* gene and in other intergenic regions. The test results indicate that compared with all users, whether the combination of genotypes at the 3 polymorphic loci detected by this test increases the risk of spontaneous urticaria disease. It does not mean the user's true risk of spontaneous urticaria disease. The basis of the test is mainly from the research of Caucasian and Chinese, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that cause the risk of spontaneous urticaria.
2. The test results have no clinical significance.

3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only evaluates your risk of spontaneous urticaria at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of spontaneous urticaria, or you think that you are likely to suffer from spontaneous urticaria, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 3 polymorphic loci associated with spontaneous urticaria, including rs453755, rs708777, and rs34141382.

Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to spontaneous urticaria.

Brain Aneurysm

Brain aneurysm refers to abnormal swelling of the intracranial arterial wall. An aneurysm is formed as a result of changes in the structure of the intracranial arterial wall and hemodynamics due to numerous factors.

My Genetic Risk Is Average For The Population

0.51 times

Average incidence rate is 12 per 100,000 in the population

Suggestions

1. Control the intake of fat, cholesterol and salt in your diet, and eat more high-fiber foods such as bamboo shoots, carrots, and celery. Maintain normal blood sugar, blood lipid and blood pressure levels, reduce blood vessel damage and load, and prevent arteriosclerosis.
2. Refuse active and passive smoking, avoid exposure to tobacco; limit alcohol intake, the limit value of moderate alcohol consumption recommended by the Chinese Nutrition Society is that the amount of alcohol consumed by adult men does not exceed 25g a day; adult women drink alcohol a day The amount of alcohol does not exceed 15g.
3. In life, you should pay attention to balance work and rest, while avoiding excessive stress; maintain communication with friends, actively and moderately exercise, and avoid irritability.

Influencing factors



Controllable factors

1. Dietary factors: A balanced diet and maintaining normal blood sugar, blood lipid, and blood pressure levels can reduce the risk of cerebral aneurysms.
2. Tobacco and alcohol intake: Smokers or passive smokers have an increased risk of developing cerebral aneurysms. Alcohol can raise blood pressure and increase the content of certain fatty acids in the blood, which may lead to arteriosclerosis and even cerebral aneurysms.

3. Staying up late: staying up late will cause changes in vascular tension, and then form intracranial aneurysms under the impact of blood.
4. Psychological and emotional factors: People who have been under excessive stress, anxiety, irritability, and emotional agitation for a long time have a higher risk of illness.



Uncontrollable factors

1. Age: The elderly are more likely to develop cerebral aneurysms.
2. Disease factors: Hypertension and atherosclerosis can cause cerebral aneurysms.
3. Family history: People with a family history of cerebral aneurysms have a higher prevalence.
4. Infectious factors: After the bacterial infection spreads through the blood, it may cause cerebral aneurysms.

Knowledge



Time of onset

Cerebral aneurysms mostly occur in middle-aged and elderly people between 40 and 60 years old.



Prevalence

Prevalence: 12 per 100,000 people. Due to different definitions of aneurysm size and different inspection methods, the incidence of cerebral aneurysms is not uniform. The incidence of women is slightly higher than that of men, with unruptured aneurysms accounting for 50%. Childhood aneurysms account for only 2% of all aneurysms.



Typical symptoms

Cerebral aneurysm is an abnormal bulge on the wall of the intracranial artery. Its formation is due to changes in the structure and hemodynamics of the intracranial artery wall caused by many factors. Subarachnoid bleed is the most common pathogenesis. Cerebral aneurysms usually occur at the branches of cerebral arteries and main trunks. It is also the place that is the congenital developmental defect of the muscular layer of the artery wall and most impacted by the blood. So under the action of the pressure and impact of the long-term blood flow, the weak points of the artery wall here protrude outwards and gradually expand, thus forming the skull Internal aneurysm. Patients often have typical manifestations such as headache, nausea, vomiting, disturbance of consciousness, epileptic seizures, cranial nerve palsy and meningeal irritation.



Treatment

1. Non operative treatment of intracranial aneurysms. The main purpose is to prevent rebleeding and control arterial spasm. It is applicable to the following situations: ①the patient's condition is not suitable for operation or the general condition cannot tolerate craniotomy; ②The diagnosis is

unclear and needs further examination; ③The patient refused or failed the operation; ④As an adjuvant treatment before and after operation.

2. Surgical treatment of intracranial aneurysms. Patients with intracranial aneurysms who have subarachnoid hemorrhage should be operated early (clamping the aneurysm pedicle or embolizing the aneurysm), take measures to protect the brain during operation (mannitol, barbiturates, isoflurane), and expand the volume after operation.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1537373	intergenic	TT	0.70	0.99
RS3742321	STARD13	TT	0.75	1.00
RS700651	BOLL	AG	0.76	0.99
RS1429412	intergenic	AG	1.27	0.99
RS2280543	BET1L	CT	1.27	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to cerebral aneurysms. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotypes difference between patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The cerebral aneurysms health risk project uses more than 20 polymorphic loci in genes such as *CDKN2B-AS1*, *BOLL*. The test result shows whether the user has a higher prevalence of cerebral aneurysms in the genotype results at the more than 20 polymorphic loci detected compared to all users. This test does not represent the user's real risk of cerebral aneurysms. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of cerebral aneurysms.
2. The test results have no clinical significance.
3. If you have clinical needs, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by multiple factors such as genes and environment. This study only predicts your risk of cerebral aneurysms at a genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc
3. If you have a family history of cerebral aneurysms, or you think that you are likely to suffer from cerebral aneurysms, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes more than 20 polymorphic loci associated with cerebral aneurysms risk, such as rs1429412, rs9298506 and rs1333040.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that associated with cerebral aneurysms.

Cleft Lip and Cleft Palate

Cleft lip and cleft palate are the most common fetal facial deformities. Cleft lip and cleft palate not only affect the appearance of children, but also affect patients' ability to swallow, suck, enunciate and other functions.

My Genetic Risk Is Average For The Population

0.49 times

Average incidence rate is 150 per 100,000 in the population

Suggestions

1. Women during pregnancy should pay attention to the reasonable ration of nutrients, and timely supplement vitamin A, B2, B6, C, E, calcium, phosphorus, iron and other minerals. Studies have shown that taking folic acid before and during pregnancy can reduce the occurrence of cleft lip and cleft palate.
2. During pregnancy, especially the first trimester, avoid exposure to radiation and microwaves; avoid fatigue and trauma.
3. Women of childbearing age, especially women who are planning to become pregnant, maintain good living habits and quit smoking and drinking.
4. If you are infected with a virus or have an acute infection, such as rubella virus, you need to be treated and recovered before becoming pregnant.
5. Women who are planning to become pregnant or pregnant women should not use drugs that may cause fetal malformations.

Influencing factors

Controllable factors

1. Nutritional factors: the mother is malnourished, such as vitamin deficiency.
2. Bad lifestyle habits, such as mother smoking, alcohol abuse, excessive coffee consumption, etc.

3. Occupational factors: such as the mother's occupational exposure to certain chemical or radioactive substances.
4. Viral factors: rubella virus, measles virus, smallpox virus and other viruses can cause fetal cleft lip and palate deformity.
5. Drug factors: during pregnancy, mothers suffering from diabetes or taking phenytoin sodium, corticosteroids, cyclophosphamide and other teratogenic drugs due to illness can cause fetal cleft lip and palate.

Uncontrollable factors

1. Genetic factors: Parents with a family history of cleft lip and cleft palate are at higher risk of giving birth to children with cleft lip and cleft palate.
2. Pregnant mothers with epilepsy are at high risk of giving birth to children with cleft lip and cleft palate.
3. Environmental factors: The mother's living environment is seriously polluted, and heavy metals such as mercury and arsenic are seriously exceeded, and the risk of giving birth to children is high.

Knowledge

Time of onset

Congenital malformations occur immediately during the fetal period.

Prevalence

According to statistics, there are about 150 cases of the disease per 100,000 people in the average population.

Typical symptoms

Cleft lip is divided into unilateral (left or right), bilateral and median cleft (upper or lower lip) according to the location of the cleft. Cleft palate is divided into unilateral (left or right) and bilateral according to the location of the cleft.

Treatment

Surgical repair of cleft lip and cleft palate is effective. Because it is a complex developmental defect, the treatment needs to be integrated with multidisciplinary experts (such as maxillofacial plastic surgery, pediatrics, speech correction department, etc.) for sequential treatment.

Under normal circumstances, unilateral cleft lip is repaired 2-6 months after birth, and bilateral cleft lip can be postponed to 6 months later, not more than one year old. Cleft palate is generally recommended to be repaired at the age of 2-4 years. For severe soft and hard cleft palate, especially

complete cleft palate, repair cleft lip and cleft palate at the bottom of the nostril at 5-6 months after birth. Cleft palate surgery is postponed to 4-5 years old; the restoration of alveolar process deformity is best done at 10-12 years old.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS7967428	TMEM19	GG	0.66	0.99
RS957448	KIAA1429	GG	0.66	0.99
RS1880646	NTN1	AG	1.22	0.99
RS1243573	intergenic	AC	1.20	0.99
RS987525	intergenic	CC	1.00	0.97

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is calculated based on published academic literature on cleft lip and cleft palate. By studying the genotype differences between cleft lip and cleft palate patients and normal people, these literatures found some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

 Applicable situation

The cleft lip and cleft palate health risk item uses *IRF6*, *NTN1* genes, and 12 polymorphic loci between genes. The results show whether the user had a higher rate of cleft lip and cleft palate than the genotype combination of the 12 polymorphic loci tested compared to all users, and that the test did not represent the user's real risk of cleft lip and cleft palate. The basis of the test is mainly from the research of Caucasian and Asian, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of cleft lip and cleft palate.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of cleft lip and cleft palate at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
3. If you have a family history of cleft lip and cleft palate, or if you feel you have a risk of cleft lip and cleft palate, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 12 polymorphic loci related to the risk of cleft lip and cleft palate, including rs12543318, rs227731, rs861020, rs987525 and rs287980.

Population samples

The study used in this test mainly Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with cleft lip and cleft palate.

Neural Tube Defects (NTDs)

Neural tube defects (NTDs) are a group of birth defects in which the closure of the neural tube is affected during embryonic development, forming a deformity of the brain or spine.

My Genetic Risk Is Average For The Population

0.49 times

Average incidence rate is 274 per 100,000 in the population

Suggestions

1. Women who take 400 µg of folic acid every day during the first month of pregnancy to the third month of pregnancy can reduce the incidence of neural tube defects by 70%.
2. Insufficient intake of trace elements can induce neural tube defects. Therefore, in the early stages of pregnancy, pregnant women should supplement appropriate amounts of trace elements under the guidance of a doctor.
3. Severe pregnancy reactions can lead to the loss of vitamins and trace elements. Therefore, when severe pregnancy vomiting occurs, it is necessary to go to the hospital for rehydration treatment as soon as possible.
4. During the flu period, avoid going to crowded public places as much as possible. It is best to wear a mask when going out to reduce the chance of virus infection.
5. Toxoplasma infection may cause neural tube defects, so avoid contact with cats and their feces in early pregnancy, and do not eat immature meat, eggs, and milk.
6. In early pregnancy, you should take drugs under the guidance of a doctor to avoid drug abuse.
7. In early pregnancy, try to avoid radiation exposure to the abdomen or pelvis.

Influencing factors



Controllable factors

1. Folic acid deficiency: Folic acid is a nutrient essential for early fetal neurodevelopment. Folic acid deficiency in women's early pregnancy is the main cause of neural tube defects.
2. Zinc deficiency: Inadequate intake of zinc and other trace elements such as copper, calcium, selenium, etc. during the first trimester can induce neural tube defects.
3. Severe pregnancy reaction: Severe vomiting causes water loss and temporary dehydration, resulting in a lack of trace elements (such as zinc) or vitamins (such as folic acid), leading to neural tube defects.
4. Viral infection: Infection of cytomegalovirus or influenza A virus in early pregnancy can cause developmental disorders of the fetal central nervous system and lead to neural tube malformations.
5. Toxoplasma infection: Toxoplasma infection in early pregnancy may also cause neural tube defects.
6. Drugs: anti-epileptics, contraceptives, anti-tumor drugs, or large or continuous use of hormones, may interfere with folate metabolism and induce neural tube defects.
7. Radiation exposure: If the abdomen or pelvis is irradiated with radiation in the first trimester, the development of the fetal central nervous system will be affected, which may lead to neural tube malformations.

Uncontrollable factors

Family history: As long as one parent has a history of neural tube defects, the incidence of their offspring is 3%, which is significantly higher than that of the general population.

Knowledge

Time of onset

Neural tube malformations generally occur within 3 months of pregnancy.

Prevalence

The incidence of neural tube defects in many countries in the world (excluding China) has fluctuated between 0.3‰ and 2.1‰ in the past 20 years. The incidence rate in China is 2.74‰, and the incidence rate is higher in the north than in the south, in rural areas than in cities, and in summer and autumn than in winter and spring.

Typical symptoms

Neural tube malformations are a group of congenital malformations with many different clinical phenotypes, including anencephaly, encephalocele, and spina bifida.

Treatment

Cerebral bulge generally requires surgical treatment; for lower limb paralysis and muscle atrophy caused by spina bifida, rehabilitation and symptomatic treatment can be tried.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1801133	MTHFR	GG	1.00	0.92

Risk calculation

The risk value of this test is calculated based on published academic literature related to neural tube defects. By studying the genotype differences between patients with neural tube defects and normal people, these literatures have found some polymorphic sites associated with the risk of neural tube defects. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk item for neural tube defects uses 1 polymorphic locus of the *MTHFR* gene. The results show whether the user had a higher prevalence of neural tube defects than all users in the genotype combination of the 1 polymorphic locus tested, which does not represent the user's real risk of neural tube defects. The test is based mainly on Caucasian studies, and other populations are for informational purposes only.

Notification

-
1. The test may not cover all genes or loci that could contribute to the risk of neural tube defects.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing neural tube defects at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of neural tube defects, or if you feel you have a risk of developing neural tube defects, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes rs1801133, 1 polymorphic locus associated with the risk of neural tube defects.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with neural tube defects.

Neuroblastoma

It is a malignant tumor of the sympathetic nervous system and the most common extracranial solid tumor in childhood. The common involved sites include the adrenal gland, abdomen, and chest.

My Genetic Risk Is Average For The Population

0.48 times

Average incidence rate is 1 per 100,000 in the population

Suggestions

1. Couples at childbearing age and developing children should avoid exposure to harmful substances (e.g., tobacco, alcohol, paint, solvents, radioactive sources).
 2. Improve body immunity through healthy diet, physical exercise, maintaining a good attitude and sufficient sleep.
-

Influencing factors



Controllable factors

1. Before the patient was born, their parents had long-term exposure to certain chemicals, such as paint, petroleum products, solvents, pesticides; Or there may be a history of exposure to hazardous radiation by pregnant women.
2. The weakened immune system of the body leads to tumor formation.



Uncontrollable factors

1. Gender: Male is slightly more risky than female.
 2. Age: The high incidence age is 0-5 years old.
 3. Family history: People with a family history of tumor have a higher risk.
-

Knowledge

Time of onset

About 90% of the cases occurred in children under 5 years old, and very few cases occurred after 10 years old.

Prevalence

According to Children's Oncology Group (COG) statistics, there are more than 65/1,000,000 new cases of neuroblastoma occur every year. The ratio of male to female incidence was 1.2:1.

Typical symptoms

1. Body parts:

- Originating in the abdomen: It is more common in the sympathetic nerve chain on both sides of the adrenal gland and spine. Symptoms usually appear when the tumor is large. There may be abdominal pain, enlarged abdomen circumference, gastrointestinal symptoms, etc.
- Originating in the thoracic cavity: Symptoms related to mediastinal compression and respiratory symptoms, such as shortness of breath, cough, etc.

2. Late-stage manifestations: Patients often have limb pain, anemia, fever, weight loss, and orbital metastasis. Orbital metastasis forms characteristic panda eyes, which are characterized by prominent eyeballs and periorbital bruising. Other symptoms may include hypertension and compression related to the tumor, such as dyskinesia and incontinence when infiltrated and compressed in the spinal canal.

3. The route(s) of metastasis: The main route of metastasis is lymph and blood.

Treatment

Surgery, chemotherapy, and radiotherapy are still the three main treatments for neuroblastoma, and treatment programs of different intensities are used according to their clinical prognostic factors. Generally, it is advisable for localized tumors to be surgically removed first, and then chemotherapy. For those who cannot be resected by surgery, the strategy of chemotherapy, surgery, chemotherapy or radiotherapy is adopted.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS6939340	CASC15	GG	1.97	0.98
RS2229571	BARD1	CG	0.58	0.93

RS7585356	BARD1	AG	0.63	0.95
RS110419	LMO1	AG	1.46	0.97
RS1048108	BARD1	AG	0.64	0.96

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is calculated based on published academic literatures related to neuroblastoma. By studying the genotype differences between patients with neuroblastoma and normal people, these literatures have found some polymorphic sites associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of neuroblastoma item uses 7 polymorphic loci in genes of *CASC15*, *BARD1* and *LMO1*. The test results indicate whether the user has a higher prevalence of neuroblastoma in the combination of genotypes at the 7 polymorphic loci tested compared to all users. This test does not represent the user's true risk of neuroblastoma. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of neuroblastoma.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing neuroblastoma at a genetic level and does not represent your true health status, nor does it involve your judgment of other health conditions, lifestyles, etc.
3. If you have a family history of neuroblastoma, or you think that you are likely to suffer from neuroblastoma, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 7 polymorphic loci related to the risk of neuroblastoma, including rs6939340, rs6435862 and rs110419.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to neuroblastoma.

Obesity

Obesity refers to excessive accumulation and abnormal distribution of body fat and weight gain.

My Genetic Risk Is Average For The Population

0.48 times

Average incidence rate is 7100 per 100,000 in the population

Suggestions

- Focus on indicators: BMI, Waist circumference
- Red flags: High-calorie diet, Eating abnormality, Snoring etc.
- Pay special attention to the crowd: Children and Elderly
- How to reduce the risk:
 - Healthy diet, limit calorie intake, eat more fruits and vegetables, reduce refined sugar intake
 - Physical exercise, adults should have no less than 150 minutes of moderate-intensity exercise per week (such as brisk walking, etc.)
 - Ensure Sufficient sleep and develop good living habits
 - Relieve psychological pressure and avoid overeating
 - If necessary, follow the doctor's advice, supplemented by medication or surgery
- Obesity Heritability is about 40%-70%

* If you suffer from obesity, or you feel that you are likely to suffer from obesity, or need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors



Controllable factors

1. Food intake: Excessive intake of total calories is related to the occurrence of obesity.
2. Exercise consumption: The reduction in the demand for physical activity will affect the body's energy consumption and cause obesity.



Uncontrollable factors

1. Family history: obesity tends to gather in families.
2. Endocrine diseases: diseases such as hypothyroidism, Cushing's syndrome, growth hormone deficiency and their treatment drugs, will increase the risk of obesity in patients.
3. Drugs: Certain drugs can cause weight gain and changes in physical composition, such as insulin, antidepressants, glucocorticoids, certain antiepileptic drugs, and hormonal contraceptives.

Knowledge

Time of onset

Obesity can be seen at any age, and about 1/2 adult obese people have a history of juvenile obesity.

Prevalence

The incidence of obesity is increasing in both adults and children, and it occurs more frequently in women than in men. In 2014, 600 million adults (13%) and 42 million children under 5 years old had obesity problems worldwide. On average, there are about 7100 cases per 100,000 people.

Typical symptoms

Excessive accumulation of fat in the body and/or abnormal fat distribution are usually associated with weight gain.

Treatment

Behavior therapy for diet control and strengthening exercise, supplemented by medication or surgery when necessary.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS988712	BDNF-AS	GG	1.99	0.99
RS7647305	intergenic	CC	1.36	1.00
RS2116830	intergenic	GG	1.30	1.00
RS4788102	SH2B1	AG	1.25	1.00

RS8004796

intergenic

CC

0.85

1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to obesity. These literatures have found some polymorphic loci related to the risk of the disease by studying obesity patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of obesity item uses 306 polymorphic loci in FTO, PCSK1, SH2B1 and BDNF-AS. The test results indicate whether the user has a higher prevalence of obesity in the combination of genotypes at the 306 polymorphic loci tested compared to all users. This test does not represent the user's true risk of obesity. The basis of the test is mainly from the research of Caucasian and Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of obesity.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.

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2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of obesity at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
 3. If you have a family history of obesity, or you think that you are likely to suffer from obesity, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 306 polymorphic loci related to the risk of obesity, including rs1121980, rs6232, rs7566605, rs13130484 and rs10871777.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to obesity.

Oral Cancer

Oral cancer is a general term for malignant tumors that occur in the oral cavity, and is one of the more common malignant tumors in the head and neck.

My Genetic Risk Is Average For The Population

0.47 times

Average incidence rate is 3 per 100,000 in the population

Suggestions

1. Maintain a healthy lifestyle. Do not smoke, drink less alcohol, maintain good oral hygiene, do not chew betel nut.
2. Avoid long-term stimulation of foreign bodies. Wear good dentures without stimulating tissues. If you find inflammation and pain in oral tissue, get medical attention in time.
3. Eat a balanced diet with balanced nutrition.
4. Pay attention to sexual hygiene and maintain a single sexual relationship.

Influencing factors



Controllable factors

1. Lifestyle factors: bad lifestyle, including long-term smoking and drinking, and poor oral hygiene habits, can increase the risk of oral cancer.
2. Long-term stimulation of foreign bodies: tooth roots or sharp tooth tips, inappropriate dentures and other long-term stimulation of oral mucosa, resulting in chronic ulcer and even canceration.
3. Malnutrition: long-term vitamin A deficiency can lead to thickening and hyperkeratosis of oral mucosal epithelium, resulting in oral cancer. In addition, Zinc deficiency may lead to mucosal epithelial damage and increase the risk of oral cancer.
4. HPV virus: Oral HPV infection increases the risk of oral cancer by about 15 times.



Uncontrollable factors

1. Age: Oral cancer is more common in people over 40.
2. Genetic factors: Mutations in genes can lead to oral cancer.

Knowledge

Time of onset

The onset age of oral cancer is mostly after the age of 40.

Prevalence

The incidence rate of oral cancer in China is lower than that of the world average level and developing countries, which is similar to that of developed countries. The incidence of oral cancer in China is 3.28/100,000.

Typical symptoms

Oral cancer will appear the following symptoms: there are masses and nodules in the mouth; there are white and smooth squamous plaques; there are red patches, ulcers, inflammatory areas and other symptoms which can not be cured for a long time; repeated bleeding without obvious cause in the mouth; numbness, burning or dryness in the mouth without obvious cause; difficulty or abnormality in speaking or swallowing.

Treatment

1. Surgical treatment: at present, the treatment of oral cancer generally adopts the principle of comprehensive treatment based on surgery. Surgical treatment of early-stage oral cancer only requires extensive resection of a small area of cancer foci and neck dissection if necessary.
2. Radiotherapy: generally used as adjuvant therapy after surgery. Postoperative adjuvant radiotherapy should be used in the following cases: cervical lymph node metastasis; The size of the primary tumor was large, with the maximum diameter exceeding 4cm; Histopathological examination revealed extensive spread of tumor tissue along nerves or blood vessels, etc.
3. Chemotherapy: currently in the stage of research and observation, it is mostly used for palliative treatment of advanced oral cancer cases with no hope of radical cure.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS6547741	intergenic	AA	0.71	1.00
RS2517452	intergenic	CT	0.69	0.97
RS818912	intergenic	AC	1.21	0.99
RS8181047	intergenic	AG	1.19	1.00
RS5953210	MAOA	AA	1.00	0.90

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to oral cancer. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between subjects with oral cancer and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The oral cancer item uses 6 polymorphic loci in and between *MAOA* gene. The test results indicate whether the user has a higher risk of oral cancer for the genotype combination of 6 polymorphic loci detected compared to all users. The test cannot represent the user's real risk of oral cancer. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that contribute to the risk of oral cancer.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are jointly determined by multiple factors such as genes and environment. This study only predicts your risk of oral cancer at the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If there is a family history of oral cancer in your family, or if you think you are at risk of developing oral cancer, you are recommended to consult an authoritative health professional or clinician for help.

Test content

Six polymorphic loci associated with the risk of oral cancer were detected, including rs8181047, rs6547741 and rs928674.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect oral cancer.

Scleroderma

Also known as systemic sclerosis, it is a type of unexplained connective tissue disease characterized by fibrosis of the skin, blood vessels and internal organs (including the gastrointestinal tract, lungs, heart, kidneys, etc.).

My Genetic Risk Is Average For The Population

0.46 times

Average incidence rate is 3 per 100,000 in the population

Suggestions

Avoid long-term exposure to chemicals (e.g., trichloroethylene) and take precautions if necessary to avoid scleroderma.

Influencing factors



Controllable factors

Chemicals: long-term exposure to certain chemicals (e.g., trichloroethylene) may result in skin changes like scleroderma.



Uncontrollable factors

1. Family history: Scleroderma has a certain genetic tendency, if there are relatives with scleroderma, the risk of disease is significantly higher than the general population.
2. Gender: The ratio of male to female in patients with scleroderma is about 1:3. Women are more likely to suffer from scleroderma, and it is more common in women of childbearing age.

Knowledge



Time of onset

80% of cases occur between the ages of 11 and 50.

Prevalence

On average, there are 3 cases of scleroderma per 100,000 people. Women usually have a higher incidence than men.

Typical symptoms

1. Scleroderma circumscriptum: lesions mainly affect the skin, according to skin damage can be divided into drip-type, plaque-like, linear and general scleroderma. Generally, no self-conscious symptoms, occasionally reduced sensory function.
2. Systemic scleroderma: The lesion not only invades the skin, but can also affect the multiple internal organs. Clinically, it is divided into acral type and diffuse type. The acral type accounts for about 95% of systemic scleroderma. Raynaud's phenomenon usually occurs first. Skin sclerosis usually starts from the hands and face, and the course of the disease progresses slowly; The diffuse type accounts for only 5 %, without Raynaud's phenomenon and acral sclerosis, it is diffuse sclerosis throughout the body at the beginning, the disease progresses rapidly, and extensive sclerosis of the skin and internal organs occurs mostly within 2 years.

Treatment

There is currently no specific remedy for the treatment of scleroderma, and early treatment is aimed primarily at preventing new skin and organs from being affected, while late-stage treatment is aimed at improving existing symptoms.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS415929	NOTCH4	TT	1.80	0.99
RS11117420	intergenic	GG	0.66	1.00
RS2056626	CD247	TT	0.66	0.99
RS9884090	ARHGAP31	GG	0.69	0.99
RS11217020	intergenic	GG	0.71	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to scleroderma. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotypes difference between scleroderma patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of scleroderma item uses the 40 polymorphic loci on genes such as *STAT4*, *DNASE1L3*, *NFKB1* and *NOTCH4* and within other intergenic regions. The test results indicate whether the user had a higher incidence of scleroderma than all users in the genotype combination of the 40 polymorphic loci tested. It does not mean the user's true risk of suffering from scleroderma. The basis of the test is mainly from the research of Oceanian, white American, European, and Japanese, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of scleroderma.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by multiple factors such as genes and environment. This study only predicts your risk of scleroderma at a genetic level. Since the test

does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.

3. If you have a family history of scleroderma, or you think that you are likely to suffer from scleroderma, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 40 polymorphic loci associated with scleroderma risk, including rs443198, rs35677470, and rs230534.

Population samples

The basis of the test is mainly from the research of Oceanian, white American, European, and Japanese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that associated with scleroderma.

Scoliosis

Scoliosis is a three-dimensional deformity of the spine, which includes coronal, sagittal and axial sequence abnormalities.

My Genetic Risk Is Average For The Population

0.46 times

Average incidence rate is 2000 per 100,000 in the population

Suggestions

1. Pay attention to maintaining a good reading and writing posture, such as punching from the edge of the table with your eyes, and your eyes are 30 cm away from the book, which helps to maintain a good body shape.
2. Keep the weight of the backpack below 10% of the backpacker's weight, and place the items in the schoolbag reasonably. The heaviest items should be placed near the back. Bring a backpack as much as possible to help disperse the weight of the backpack and reduce the possibility of body deformation.
3. Idiopathic scoliosis mostly occurs in adolescence. It is recommended that school-age children take about 1 hour of physical exercise every day. Participate more in single parallel bars, balance beams and other projects to increase the strength of the back muscles and have a good effect on preventing the curvature of the spine.

Influencing factors



Controllable factors

Bad posture: Long-term bad standing and sitting posture can aggravate the degree of scoliosis.



Uncontrollable factors

1. Neurological or muscular diseases: cerebral palsy, poliomyelitis (poliomyelitis), progressive muscular atrophy, etc.

- 2. Age: idiopathic scoliosis usually begins in children aged 10-13 years, and tends to occur in adolescence with rapid physical development.
- 3. Gender: the incidence rate of female idiopathic scoliosis is significantly higher than that of men, with a male to female ratio of 1:10.

Knowledge

Time of onset

Idiopathic scoliosis usually begins in children aged 10-13 years, and often occurs in adolescence with rapid physical development. The incidence rate of degenerative scoliosis increased with age, most of which occurred at the age of 60 years.

Prevalence

The most common scoliosis is in the developed countries, accounting for about 80% of all scoliosis, with an incidence rate of 0.5%-2%. The incidence rate of adolescent idiopathic scoliosis in China is 1%-2%.

Typical symptoms

The shoulders are uneven, the spine deviates from the midline, the shoulder blades are one high and one low, the skin folds on both sides of the waist are asymmetric, and the razor back will appear after bending down (the height of the back is asymmetric).

Treatment

Mild scoliosis can be observed regularly, and exercise therapy and brace correction can be carried out. Severe scoliosis needs surgical treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS12149832	FTO	GG	1.35	1.00
RS482012	NT5DC1	TT	1.30	1.00
RS9389985	intergenic	AG	1.21	0.99

RS17011903	intergenic	AC	1.20	1.00
RS11205303	MTMR11	CT	1.17	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to scoliosis. By studying the genotype differences between patients with scoliosis and normal people, these literatures have found some polymorphic sites associated with the risk of scoliosis. We uses the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

A health risk item for scoliosis uses 11 polymorphic loci. The results show whether the user had a higher prevalence of scoliosis than all users in the genotype combination of the 11 polymorphic loci tested, which does not represent the user's real risk of scoliosis. The test is based mainly on Chinese and Japanese studies, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of scoliosis.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.

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2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing scoliosis at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
 3. If you have a family history of scoliosis, or if you feel you have a risk of developing scoliosis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 11 polymorphic loci associated with the risk of scoliosis.

Population samples

The basis of the test is mainly from the research of Chinese and Japanese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with scoliosis.

Tardive Dyskinesia

Tardive dyskinesia is a specific type of muscular tension disorder, of which main symptoms are involuntary body movements at lips, tongue, or choreic movement.

My Genetic Risk Is Average For The Population

0.42 times

Average incidence rate is 16600 per 100,000 in the population

Suggestions

1. Reject active and passive smoking, and avoid exposure to tobacco.
2. For patients who need to take antipsychotic drugs for a long time, if it is feasible after comprehensive evaluation by doctors, drugs with low risk of tardive dyskinesia can be considered.

Influencing factors

Controllable factors

1. Smoking: Studies have found that smoking increases the risk of tardive dyskinesia.
2. Medication: Tardive dyskinesia is more common in patients who take long-term (more than 1 year) high-dose antipsychotics that block dopaminergic receptors or bind dopaminergic receptors.

Uncontrollable factors

1. Age: The elderly are more likely to develop tardive dyskinesias and are not easy to recover.
2. Gender: The incidence of tardive dyskinesia in women is higher than that in men.
3. Race: African and African American have a higher rate of tardive dyskinesia after exposure to antipsychotic drugs.
4. Other diseases: Diabetes, brain structural damage, schizophrenia, and patients with acute extrapyramidal reactions related to dopamine receptor blockers are at higher risk of tardive dyskinesia.

Knowledge

Time of onset

It can occur at any age, and it mostly occurs in elderly patients, especially women.

Prevalence

Tardive dyskinesia most often occurs in patients with psychosis who have been treated with antipsychotic drugs for many years. For example, patients receiving schizophrenia drugs have an average prevalence of about 30%. The incidence is higher in patients older than 50 years.

Typical symptoms

It is clinically characterized by involuntary, rhythmic and stereotyped movement. Most of the manifestations are "mouth-tongue-cheek triad", that is, the involuntary movement of the mouth, lips, tongue, and face, such as the involuntary tongue turning in the mouth and involuntary chewing. Another common manifestation is dystonia with abnormal posture, often involving the neck (spasmodic torticollis), and can be extended to segmental or generalized. The coexistence of chorea and dystonia is common.

Treatment

For new-onset patients with tardive dyskinesia, antipsychotic drugs that are suspected of causing tardive dyskinesia should be stopped first. Given that most patients with schizophrenia require long-term antipsychotic treatment, it is recommended to switch to drugs with a lower risk of tardive dyskinesia. If there are clinical indications, switching to clozapine may be a better choice. For patients with chronic, well-diagnosed tardive dyskinesia, VMAT2 inhibitors can be used to treat adult tardive dyskinesia.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1800497	ANKK1	GG	1.00	0.88

Risk calculation

The risk value of this test is calculated based on published academic literature related to tardive dyskinesia. By studying the genotype differences between patients with tardive dyskinesia and normal people, these literatures have found some polymorphic sites associated with the risk of tardive dyskinesia. We uses the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

A health risk item for tardive dyskinesia uses a polymorphic locus on the *ANKK1* gene. The results show whether the user had a higher prevalence of tardive dyskinesia than all users in the genotype combination of the polymorphic locus tested, which does not represent the user's real risk of tardive dyskinesia. The test is based mainly on Asian studies, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of tardive dyskinesia.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing tardive dyskinesia at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If your family has a family history of tardive dyskinesia, or if you feel you have a risk of developing tardive dyskinesia, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes rs1800497, a polymorphic locus associated with the risk of tardive dyskinesia.

Population samples

The basis of the test is mainly from the research of Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with tardive dyskinesia.

Intrahepatic Cholestasis of Pregnancy (ICP)

It is a specific complication of pregnancy with elevated bile acid in the blood, characterized by pruritus clinically and cholestasis biochemistry.

My Genetic Risk Is Average For The Population

0.42 times

Average incidence rate is 6000 per 100,000 in the population

Suggestions

Try to get married and have early childbirth as early as possible, and do not be pregnant women over 35 years old.

Influencing factors



Controllable factors

Pregnant woman's age: The age of pregnant women > 35 years old, the risk of disease is increased.



Uncontrollable factors

1. Genetic factors: there is a family genetic tendency.
2. Multiple pregnancy: the risk of multiple pregnancy is significantly higher than that of single pregnancy.
3. Artificial insemination: pregnant women with artificial insemination have an increased risk of disease.
4. Seasonal factors: studies have shown that the incidence rate in winter is higher than that in summer, which may be related to the lower blood selenium content in winter than in summer.
5. Suffering from chronic hepatobiliary diseases: some disease can increase the risk, such as viral hepatitis, cirrhosis, cholecystitis, gallstones, etc.
6. Those who suffered from the previous pregnancy have a high recurrence rate during the second pregnancy.

Knowledge

Time of onset

The onset is mostly in the third trimester, and more than 70% occur after 28 weeks of gestation. There are also a small number of pregnant women who have the disease in the second trimester.

Prevalence

According to statistics, there are about 6000 cases per 100,000 people of the disease in the average population.

Typical symptoms

Itching is often the first symptom, and some patients may develop jaundice within 2-4 weeks after itching occurs. Severe itching can cause insomnia and mood changes, fatigue, loss of appetite, nausea and even vomiting.

Treatment

Treatment is mainly to relieve symptoms, such as reducing skin itching, improving liver function, lowering bile acid levels, monitoring the condition of the fetus, and waiting until 37 weeks of pregnancy to give birth as much as possible. The main medication is ursodeoxycholic acid.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2287622	ABCB11	AG	2.40	0.85
RS3740066	ABCC2	CC	1.00	0.93

Risk calculation

The risk value of the test is calculated based on published academic literatures related to intrahepatic cholestasis of pregnancy. By studying the genotype differences between intrahepatic cholestasis of pregnancy patients and normal people, these literatures have found some polymorphic sites

associated with the risk of the disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of intrahepatic cholestasis item uses 2 polymorphic loci in the *ABCB11*, *ABCC2* gene. The test results indicate whether the user has a higher prevalence of intrahepatic cholestasis compared to all users. This test does not represent the user's true risk of intrahepatic cholestasis. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of intrahepatic cholestasis of pregnancy.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of intrahepatic cholestasis of pregnancy at a genetic level and does not represent your true health status, nor does it involve your other health conditions, lifestyle, etc.
3. If you have a family history of intrahepatic cholestasis of pregnancy, or you think that you are likely to suffer from intrahepatic cholestasis of pregnancy, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 2 polymorphic loci related to the risk of intrahepatic cholestasis of pregnancy, including rs2287622 and rs3740066.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to intrahepatic cholestasis of pregnancy.

Narcolepsy

Narcolepsy is a sleep disorder characterized by excessive sleep and an irresistible urge to sleep during the day.

My Genetic Risk Is Average For The Population

0.39 times

Average incidence rate is 3 per 100,000 in the population

Suggestions

Refuse active and passive smoking and avoid exposure to the tobacco environment.

Influencing factors



Controllable factors

Tobacco intake: Smoking is very common in patients with narcolepsy and is related to the clinical features of narcolepsy. Narcolepsy is a chronic neurological disease with autoimmune pathogenesis. Nicotine stimulates the central nervous system, and smoking increases the risk of autoimmune diseases.



Uncontrollable factors

1. **Family history:** When a patient is diagnosed, the risk of illness from relatives is 20-40 times higher than that of the normal population.
2. **Birth order:** The study found that the incidence of narcolepsy is related to the birth order, and the lower the birth order, the higher the prevalence of children. This may be related to the infection of *Streptococcus agalactiae*.
3. **Infectious factors:** It has been reported that the incidence of narcolepsy will increase significantly after H1N1 infection or H1N1 vaccine injection. The non-specific immunity caused by avian influenza virus infection and the strong autoimmunity stimulated by the adjuvant arsenous acid in the vaccine may be the key to the effect.

Knowledge

Time of onset

the onset of the disease often begins in childhood and adolescence, but is delayed by an average of 15 years between onset and diagnosis.

Prevalence

Prevalence: 3/100,000. The prevalence of narcolepsy varies greatly in different regions and different races. Studies have found that the prevalence of narcolepsy in the United States is higher than that in Europe and Asia. There is no obvious difference in the incidence of the disease between different sexes.

Typical symptoms

The clinical manifestations of narcolepsy include excessive sleep, daytime sleepiness, and sudden lethargy or cataplexy, and the appearance of pathological rapid eye movement sleep. Under strong emotional stimulation (mostly positive emotions), cataplexy is more likely to occur, but at this time the patient's consciousness is generally not lost. In addition, there are nocturnal sleep disorders, such as nightmares, interrupted sleep at night, sleep paralysis, hallucinations before going to bed, etc. Having narcolepsy is often accompanied by other diseases. Such as obesity, the more severe the sleepiness of patients, the more significant the weight gain may be, which may be related to the low energy consumption of patients with narcolepsy. In addition, complications such as obstructive sleep apnea syndrome and migraine may also occur.

Treatment

It is generally considered that narcolepsy is a lifelong disease, but reasonable treatment of narcolepsy can also relieve the symptoms of patients to a greater extent, mainly through behavioral intervention and drug treatment.

- Behavior intervention: Patients with narcolepsy can take regular naps every day for 15-20 minutes; they also need to maintain good sleep habits; in addition, change their life and behavior habits and try to adapt to narcolepsy so as not to induce narcolepsy anxiety and depression are equally important. Patients with narcolepsy should not drive or work at heights. However, some mentally uplifting jobs, such as teachers or trainers, are competent for patients with narcolepsy; if they are tedious or require high precision, they may be prone to errors.
- Medication: Medication is mainly through central nervous system stimulants to reduce daytime sleepiness, cataplexy and increase the quality of night sleep, so as to minimize the impact on study and work during the day. Commonly used drugs are modafinil, mazindole and methylphenidate.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS198811	intergenic	CT	1.69	0.97
RS2305795	P2RY11	AA	1.69	0.99
RS2834188	intergenic	AG	0.77	0.99
RS1154155	intergenic	TT	1.00	0.96
RS1551570	PPAN	TT	1.00	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to narcolepsy. These literatures found some polymorphic sites associated with the risk of the disease by studying the genetic differences between patients with narcolepsy and normal people. We uses the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of narcolepsy item uses the 9 polymorphic loci in *P2RY11*, *CHKB-CPT1B* and *HCRT*. The test results indicate whether the user has a higher prevalence of male narcolepsy in the combination of genotypes at the 9 polymorphic loci tested compared to all users. This test does not represent the user's true risk of narcolepsy. The basis of the test is mainly from the research of Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of narcolepsy.

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2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of narcolepsy at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of narcolepsy, or you think that you are likely to suffer from narcolepsy, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 9 polymorphic loci related to the risk of narcolepsy, including rs1154155, rs2305795 and rs7553711.

Population samples

The basis of the test is mainly from the research of Asian, other populations are for reference only.

Limit of detection

The test may not cover all genes or variants related to narcolepsy.

Polycystic Ovarian Syndrome (PCOS)

It is one of the most common endocrine disorders in women of childbearing age and the main cause of anovulatory infertility.

My Genetic Risk Is Average For The Population

0.39 times

Average incidence rate is 6000 per 100,000 in the population

Suggestions

1. For obese women, weight loss is very effective, not only can reduce hyperinsulinemia and hyperandrogenemia, but also improve menstruation, restore ovulation, and even get pregnant.
2. Regular physical examination: a physical examination once a year to check blood sugar, blood lipids, gynecological examination and B-ultrasound. If hyperinsulinemia is found, it should be treated actively.

Influencing factors



Controllable factors

1. Obesity: Adolescent girls with BMI ≥ 25.0 and women of childbearing age are more susceptible to the disease.
2. Female patients with severe insulin resistance and hyperinsulinemia are more likely to develop polycystic ovary syndrome.



Uncontrollable factors

1. Genetic factors: there is a family genetic tendency.
2. Age: Occurs in adolescent girls and women of childbearing age.
3. Gender: Only females are affected.
4. Exposure to high levels of androgen before birth can induce this disease.

Knowledge



Time of onset

Most of them occur in adolescence, onset before or after menarche, and some patients only show symptoms in their 20s.



Prevalence

According to reports, an average of 6,000 cases of the disease occur per 100,000 people.



Typical symptoms

With chronic anovulation, hyperandrogen and polycystic ovarian changes (PCO) as the main features, common clinical symptoms include infertility, hirsutism, obesity, menstrual disorders, insulin resistance and hyperinsulinemia.



Treatment

1. Genetic factors: People who have family genetic tendencies and whose mothers or siblings are patients with polycystic ovary syndrome are more susceptible to the disease.
2. Age: Occurs in adolescent girls and women of childbearing age.
3. Gender: Only females are affected.
4. Exposure to high levels of androgen before birth can induce this disease.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS12468394	THADA	AC	0.72	0.98
RS12478601	THADA	CT	0.72	0.98
RS804279	intergenic	TT	1.30	1.00
RS4784165	intergenic	GT	1.15	1.00
RS1784692	ZBTB16	CT	1.15	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to polycystic ovary syndrome. These literatures have found some polymorphic loci related to the risk of the disease by studying patients with polycystic ovary syndrome and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of polycystic ovary syndrome item uses 28 polymorphic loci in genes of *LHCGR*, *THADA* and *DENND1A*. The test results indicate whether the user has a higher prevalence of polycystic ovary syndrome in the combination of genotypes at the 28 polymorphic loci tested compared to all users. This test does not represent the user's true risk of polycystic ovary syndrome. The basis of the test is mainly from the research of Caucasian and Chinese, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could lead to a risk of polycystic ovary syndrome.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of polycystic ovary syndrome at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
-

3. If you have a family history of polycystic ovary syndrome, or you think that you are likely to suffer from polycystic ovary syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 28 polymorphic loci related to the risk of polycystic ovary syndrome, including rs13405728, rs13429458 and rs2479106.

 Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

 Limit of detection

The test may not cover all genes or loci related to polycystic ovary syndrome.

Atopic Dermatitis

Atopic dermatitis is a chronic, recurrent, inflammatory disease of the skin. The most basic characteristics are dry skin, chronic eczema-like dermatitis and severe itching.

My Genetic Risk Is Average For The Population

0.38 times

Average incidence rate is 690 per 100,000 in the population

Suggestions

1. Avoid local skin irritation, such as the use of irritating drugs.
 2. Do not eat foods that may induce the disease, such as seafood, eggs, milk, etc.
 3. Keep indoor air circulating and fresh, and avoid house dust, pollen, dust mites, etc.
 4. Pay attention to rest and avoid nervousness and emotional excitement.
-

Influencing factors



Controllable factors

1. Skin irritation may induce atopic dermatitis.
2. Food: Foods that often cause allergies include eggs, milk, seafood, and a small amount of those that are allergic to fruit juices and nuts.
3. Inhalation: Pollen, animal hair, fungus, house dust, dust mites and other inhalants may induce atopic dermatitis.
4. Tiredness, nervousness, emotional agitation, etc. will increase the risk of atopic dermatitis.



Uncontrollable factors

1. Age: The vast majority of cases first occur in infants and toddlers, and some can occur in childhood and adulthood.

2. Disease: Bronchial asthma or allergic rhinitis is significantly related to the disease.

Knowledge

Time of onset

Generally, the age of onset is early, and 60% of patients have the onset within 1 to 6 months, and some have the onset as early as 1 week. About 90% of the disease occurs within 5 years of age. No more than 5% of those who are older than 35 years of age have the disease.

Prevalence

The global prevalence of this disease is about 2.3%. The incidence of atopic dermatitis is constantly increasing.

Typical symptoms

The most basic symptoms are chronic recurrent episodes and intense itching. There are different manifestations in different stages of disease.

1. Infancy: Acute erythema and papules that occur early on the face. In severe cases, there may be blisters, pustules, serous or pus, and yellow scab; it can cause secondary infection and lymphadenopathy. Mild symptoms can be relieved after half a year of age, redness and swelling gradually disappear, discharge decreases, the damage gradually becomes dry, there is no longer thick scabs, but only thin scabs and scales, or only scales, and can be cured by the age of 1 year. The heavier ones are cured by 2 years of age, and the heavier ones continue to develop into childhood.
2. Childhood: mostly evolved from infancy, and can also occur without going through infancy. It mostly occurs in the elbow fossa, rouge fossa and the extended side of the calf, with subacute and chronic dermatitis as the main manifestations. The rash is often dry and thick, with obvious lichenoid changes.
3. Adolescence and adulthood: similar to late childhood damage, it mainly occurs in the elbow and popliteal fossa, but the scope is more extensive, sometimes involving the face, neck and hands. The distribution is symmetrical. The skin is dry and thick, accompanied by lichenoid changes. Some patients may develop subacute eczema-like patches on the extension side of the limbs, the base is reddish, and there are a lot of scales and scabs on it, which will not heal for a long time. The skin lesions showed disseminated neurodermatitis changes, the course of the disease was chronic, recurring, it could spread throughout the body, consciously itching, or there may be a longer period of remission, and individual patients may be prolonged to old age.

Treatment

The disease increases with age, and most people can be significantly relieved and cured. With treatment, the healing process can be accelerated. Treatment methods include cause-relief therapy, internal use of related drugs, desensitization therapy, glucocorticoid therapy, and phototherapy.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS176095	PBX2, GPSM3	AA	1.96	0.98
RS848	IL13	CC	0.51	0.98
RS13015714	IL18R1	GG	1.61	0.99
RS10738626	intergenic	TT	1.51	0.99
RS16999165	intergenic	AA	1.42	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to atopic dermatitis. By studying the genotype differences between patients with atopic dermatitis and normal people, these literatures have found some polymorphic sites associated with the risk of atopic dermatitis. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

 Applicable situation

The health risk of atopic dermatitis item uses 85 polymorphic loci on genes such as *IL13*, *ZNF365* and *PUS10* and within other intergenic regions. The results show whether the user had a higher prevalence of atopic dermatitis than all users in the genotype combination of the 85 polymorphic loci tested, which does not represent the user's real risk of atopic dermatitis. The test is based mainly on Caucasian and Asian studies, and other populations are for informational purposes only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of atopic dermatitis.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing atopic dermatitis at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of atopic dermatitis, or if you feel you have a risk of developing atopic dermatitis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 85 polymorphic loci associated with the risk of atopic dermatitis, including rs848, rs479844 and rs7927894.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with atopic dermatitis.

Primary Biliary Cholangitis (PBC)

Also known as primary biliary cirrhosis, it is a chronic autoimmune disease, the cause of which is unknown. It is characterized by progressive, nonsuppurative, inflammatory damage in the liver and small bile duct and manifests clinically as intrahepatic cholestasis.

My Genetic Risk Is Average For The Population

0.37 times

Average incidence rate is 492 per 100,000 in the population

Suggestions

1. Use safe, non-toxic, and qualified nail polish and food. If the toxic waste disposal station near the residence, consider moving.
2. Maintain good living habits, quit smoking and drinking.

Influencing factors



Controllable factors

1. Environmental factors: Studies have shown that the incidence of primary biliary cholangitis is significantly higher for those who use nail polish and cosmetics and who live around toxic waste disposal stations.
2. Bad habits: The incidence rate of long-term smoking is high. Alcoholism can also induce liver injury.



Uncontrollable factors

1. Genetic factors: There is a tendency for family inheritance.
2. Disease factors: Studies have shown that the incidence of infections (especially urinary tract infections) and people who use sex hormone replacement therapy are high.

Knowledge

Time of onset

It is more common in middle-aged women. 85%~90% of patients have the onset of the disease at the age of 40~60. The incidence ratio of male to female is 1:9.

Prevalence

According to statistics, there are about 492 cases of the disease per 100,000 people in the average population.

Typical symptoms

Combined with the liver function and symptoms of the patient, it can be divided into 4 stages.

Asymptomatic period with normal liver function: In this period, the patient has no symptoms and normal liver function, but the serum anti-mitochondrial antibody (AMA) is positive.

Asymptomatic period of abnormal liver function: Patients in this period have no symptoms of primary biliary cholangitis. However, liver function has appeared abnormal, and AMA is positive in the circulation.

Clinical symptom period: In this period, patients often experience symptoms such as fatigue, pruritus, and jaundice.

Decompensation period: Decompensation of liver function occurs during this period, such as ascites, esophageal varices, etc.

Treatment

Clinically, symptomatic and supportive therapies are generally adopted. For patients with severe pruritus, cholestyramine can be used for treatment. Liver transplantation is the only effective treatment for end-stage primary biliary cholangitis.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS574808	IL12A-AS1	TT	2.37	0.97
RS3745516	SPIB	AA	0.47	0.97
RS4979462	TNFSF15	CT	1.57	0.96

RS7574865	STAT4	TT	1.51	0.99
RS3790567	IL12RB2	AG	1.51	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to PBC. These literatures have found some polymorphic loci related to the risk of the disease by PBC. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of PBC item uses the 12 polymorphic loci of *TNFSF15*, *SPIB*, *IL12A-AS1*, *IL12RB2*, *STAT4* gene. The test results show whether the user has a higher prevalence of PBC than all users. The test cannot represent the user's real risk of PBC. The test is mainly based on the research of Caucasian and Asian, and other populations are only for reference.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of PBC.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.

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2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of PBC from the genetic level, which does not represent your real health status, nor does it involve the judgment of your other health status, living habits, etc.
 3. If you have a family history of PBC, or you think that you are likely to suffer from PBC, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 12 polymorphic loci related to PBC risk, including rs4979462, rs3745516, rs574808, rs3790565, rs7574865 and so on.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect PBC.

Aortic Dissection

Aortic dissection is a disease in which the inner layer of the aorta is torn, the walls of the arteries of different layers are separated from each other, and blood flows into them and forms a false cavity.

My Genetic Risk Is Average For The Population

0.37 times

Average incidence rate is 6 per 100,000 in the population

Suggestions

1. Eat a healthy diet and take appropriate physical exercises, control blood pressure, blood lipids and blood sugar, quit smoking and limit alcohol. Reasonably arrange work and rest time to ensure adequate sleep.
 2. Avoid strenuous exercise, adjust mood and blood pressure, and perform surgery in time if an aortic dissecting aneurysm is diagnosed.
 3. Avoid using cocaine.
-

Influencing factors



Controllable factors

1. Cardiovascular disease: Hypertension, atherosclerosis, aortic valve stenosis, aortic aneurysm, etc. are risk factors that contribute to the occurrence of aortic dissecting aneurysms.
2. Excessive exercise: Blood pressure rises during resistance training such as high-intensity weightlifting and bench press, which increases the risk of aortic dissecting aneurysm.
3. Cocaine: The use of cocaine will increase blood pressure and may be a risk factor for aortic dissecting aneurysm.



Uncontrollable factors

1. Genetic factors: Marfan syndrome, Ehlers-Danlos syndrome, Turner syndrome, congenital heart disease (such as bicuspid aortic valve malformation) may lead to aortic dissecting aneurysm.
2. Age: It is common in people aged 60 to 80.
3. Gender: The incidence of men is about twice that of women.
4. Accidents: Accidental injuries such as car accidents and falls that cause a violent impact on the chest may cause aortic dissecting aneurysm.

Knowledge



Time of onset

It usually affects people over 60 years old.



Prevalence

In the United States and Europe , aortic dissection occurs in 6 in 100,000 people. There is no systematic epidemiological survey data in China. In recent years, the literature estimates that the incidence rate in China is higher than that in European and American countries, and the incidence is younger.



Typical symptoms

1. It is felt below the chest bone, and then moves under the shoulder blades or to the back. It is often sudden onset, slight tearing or severe pain like splitting.
2. Symptoms such as heart failure, stroke, small bowel infarction, myocardial infarction, and lower abdominal pain may occur.



Treatment

1. Medicines: The main purpose of medication is to lower blood pressure. Patients with aortic disease often have diseases such as coronary artery disease, chronic kidney disease, diabetes, and lipid metabolism.
2. Surgery: Acute aortic dissection will be treated with surgery, which can significantly reduce the mortality rate, but surgery still has a certain mortality rate and neurological complications.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS2272007	ULK4	CT	1.35	0.99
RS11172113	LRP1	CT	0.82	0.99
RS2118181	FBN1	TT	1.00	0.91
RS10519177	FBN1	AA	1.00	0.93

Risk calculation

The risk value of this test is calculated based on published academic literatures related to aortic dissection. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between subjects with aortic dissection and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of aortic dissection item uses the 4 polymorphic loci on genes such as *FBN1*, *LRP1*, etc. The test results indicate that compared with all users, whether the genotype combination at the 4 polymorphic loci tested has a greater risk of suffering from aortic dissection, but it does not represent the user's risk of aortic dissection from all dimensions. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect aortic dissection.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This test only evaluates your risk of aortic dissection at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If your family has a history of aortic dissection, or you feel that you are at risk of suffering from aortic dissection, it is recommended that you consult an authoritative health professional or clinician for help.

Test results

The test includes 4 polymorphic loci related to aortic dissection, such as rs10519177.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect Aortic dissection.

Primary Sclerosing Cholangitis (PSC)

It is a cholestatic liver disease characterised by multi-focal bile duct strictures and progressive liver disease.

My Genetic Risk Is Average For The Population

0.37 times

Average incidence rate is 11 per 100,000 in the population

Suggestions

1. Eat breakfast on time to avoid cholestasis. If the content of fat-soluble vitamins in the body is low, you can supplement foods rich in vitamins A, D, E and K.
2. Pay attention to a balanced diet to build healthy gut microbial.
3. Avoid enteritis and ulcerative bowel disease.

Influencing factors



Controllable factors

1. Bile duct injury: The bile duct is damaged by bile acid.
2. Gut microbiota: Changes in the microbiota of the digestive tract, especially the gut microbiota, may affect the pathogenesis of this disease.
3. Enteritis: Patients with inflammatory bowel disease (IBD), especially ulcerative colitis (UC), have a high probability of suffering from the disease.



Uncontrollable factors

1. Immune system issues.
2. Family history: Primary sclerosing cholangitis has a certain family genetic predisposition.
3. Gender: The incidence is higher in males.
4. Age: The incidence is higher in adults aged 30-50.

Knowledge

Time of onset

The disease has a higher incidence in men aged 30-50.

Prevalence

The reported prevalence rate in Europe and America is 3.85-16.2/100000, and that in Asia is 0.95-3.85/100000. However, since people who may have the disease are not diagnosed, the number may be underestimated. The disease is more common in males.

Typical symptoms

The main symptoms of primary sclerosing cholangitis are feeling tired or weak, skin pruritus. Other symptoms may include weight loss, loss of appetite, fever, abdominal pain, etc. As the disease gets worse, you may get symptoms of cirrhosis and liver failure.

Treatment

- For bile duct stricture or obstruction: Endoscopic retrograde cholangiopancreatography and percutaneous transhepatic cholangiography.
- For skin pruritus: Skin creams or lotions containing camphor, menthol, promocaine, capsaicin; And antihistamines.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS80060485	FOXP1	CT	1.44	0.98
RS725613	CLEC16A	TT	1.44	0.99
RS663743	CCDC88B	GG	1.44	1.00
RS1452787	TCF4	AG	0.75	0.98
RS3099844	intergenic	CC	1.00	0.77

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to primary sclerosing cholangitis. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype difference between patients with primary sclerosing cholangitis and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The primary sclerosing cholangitis health risk item uses 32 polymorphic loci on and between genes such as *GPR35*, *MST1*, *FUT2*, *BCL2L11*. The test results indicate whether the user has a higher prevalence of primary sclerosing cholangitis in the genotype results at the 32 polymorphic loci tested compared to all users. The test cannot represent the user's real risk of primary sclerosing cholangitis. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of primary sclerosing cholangitis.
 2. The test result has no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only predicts your risk of primary sclerosing cholangitis at the

genetic level, it does not mean your true health status, nor does it involve the assessments on your other health conditions, living habits, etc.

3. If you have a family history of primary sclerosing cholangitis, or you think that you are likely to suffer from primary sclerosing cholangitis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 32 polymorphic loci related to the risk of primary sclerosing cholangitis, including rs4676410, rs3197999 and rs602662.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all genes or loci that affect primary sclerosing cholangitis.

Kidney Diseases

Kidney diseases refer to abnormal renal structure and function caused by various reasons, and the history of renal damage is generally more than 3 months.

My Genetic Risk Is Average For The Population

0.36 times

Average incidence rate is 10800 per 100,000 in the population

Suggestions

1. Avoid infection, timely detection and treatment of infectious diseases that may induce kidney damage: Skin infections, pharyngitis, tonsillitis and other diseases need to be cured immediately, and antibiotics should be used thoroughly and not halfway. If swelling of the eyelids or face is found after 1 to 2 weeks of suffering from these diseases, the urine should be tested immediately and the changes in urine should be tracked. People with allergic diseases such as urticaria, allergic purpura, etc., should track changes in urine.
2. Good urination habits. Drink plenty of water every day, urinating in time without holding back when you have a desire to urinate, then the urinary tract is not easy to be infected, and it is not easy to get stones.
3. Pay attention to the regularity and rationality of diet. Do not overeating, eat moderately as you grow older, and avoid over-satisfaction and excessive protein.
4. Enhance self-immunity. Keep warm and add clothes in time. Avoid contact with susceptible people to prevent colds and infections. Pay attention to exercise, choose a suitable exercise method and persist in exercise to enhance physical fitness and improve immunity and disease resistance. Do not smoke or drink alcohol.
5. Use medication carefully. Do not take it without a doctor's prescription, especially the use of antibiotics and analgesics. Follow the doctor's advice and try to avoid using drugs that may damage the kidneys. Do not eat herbal remedies indiscriminately to avoid delaying treatment and damaging the kidneys.

Influencing factors



Controllable factors

1. Infectious factors: Certain infections such as pharynx, intestinal tract, urinary tract or hepatitis B virus infection may lead to the occurrence of kidney diseases.
2. Urinating habits: Drinking less water is prone to urinary stones, which increases the risk of kidney diseases. The habit of holding back urine may cause damage to the kidneys.
3. Diet: Irregular diet, especially high-protein diet, will increase the burden on the kidneys and may increase the risk of diseases.
4. Physical fitness: Lack of exercise for those with weak physical fitness will reduce the ability to resist infection and may also increase the risk of kidney diseases.



Uncontrollable factors

1. Age: As age increases, the rate of glomerular filtration rate decreases, and the risk of developing kidney diseases increases.
2. Disease factors: These people are at higher risk of developing kidney diseases: Patients with systemic diseases such as hypertension, diabetes, gout, and autoimmune diseases such as systemic lupus erythematosus, and patients have a history of acute kidney diseases (such as acute nephritis, acute renal tubular necrosis, and acute interstitial nephritis) and tumor patients.
3. Family history: People with a family history of kidney diseases have a higher prevalence.
4. Drug effects: Some Chinese and Western medicines may have renal toxicity and cause damage to the kidneys.

Knowledge



Time of onset

The disease can occur at any age, and the elderly have a higher risk.



Prevalence

The incidence of this disease is increasing rapidly worldwide. In 2022, it is estimated that this disease affects more than 10% of the general population worldwide, amounting to more than 800 million individuals.



Typical symptoms

In different stages of kidney diseases, its clinical manifestations are also different. Before the third stage of kidney diseases, the patient may have no symptoms or only mild discomfort such as fatigue, backache, and nocturia; a small number of patients may have loss of appetite, metabolic acidosis,

and mild anemia. After the third stage of kidney diseases, the above symptoms become more obvious, and after entering the renal failure stage, the symptoms are further aggravated. Sometimes acute heart failure, severe hyperkalemia, gastrointestinal bleeding, central nervous system disorders, etc. may occur, and even life-threatening may occur.

1. Gastrointestinal symptoms: The most common symptoms are gastrointestinal symptoms, which are mainly manifested as loss of appetite, nausea, vomiting, and a smell of urine in the mouth.
2. Stomach and duodenal inflammation, ulcers, bleeding: More common, and their incidence is higher than normal.
3. Cardiovascular diseases: Cardiovascular diseases is one of the main complications and the most common cause of death in patients with kidney diseases. With the continuous deterioration of kidney function, the prevalence of heart failure has increased significantly, reaching 65% to 70% in the uremia stage.
4. Neuromuscular system symptoms: In the early stage of kidney diseases, neuromuscular system symptoms may include insomnia, inattention, and memory loss. As the disease progresses, there are often apathy, convulsions, hallucinations, lethargy, coma, mental disorders, etc. Peripheral neuropathy is also common.
5. Endocrine dysfunction: Patients with kidney diseases often have endocrine dysfunction, and the kidney itself has endocrine dysfunction.

Treatment

1. Etiology treatment: Find the culprit leading to kidney diseases and treat the primary diseases to reduce the damage to the kidneys.
2. Risk factor control: If the cause of the diseases cannot be eliminated, other factors that may affect kidney function need to be reduced to avoid worsening the situation. The currently recognized risk factors include: long-term hypertension; diabetes; persistent proteinuria; persistent hyperuricemia; use of nephrotoxic drugs; use of contrast agents.
3. Prevention and treatment of complications: In the process of gradual loss of kidney function, various systems of the body will be affected and abnormalities occur, that is, complications. Need to intervene for these abnormalities to help the body maintain stability.
4. Renal replacement therapy for renal failure: When the kidney function has completely failed and is not enough to maintain survival, it is necessary to find a way to replace one's own kidney function, that is, renal replacement therapy.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS873549	intergenic	TT	0.24	0.91
RS76762104	intergenic	TT	0.53	1.00
RS187355703	intergenic	CC	0.67	1.00
RS3782886	BRAP	TT	0.67	0.99
RS80282103	intergenic	AA	0.83	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to kidney diseases. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between patients with kidney diseases and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The kidney diseases health risk item uses 108 polymorphic loci on genes such as *HLA-DQA1*, *PLA2R1*, *MTHFS* and within other intergenic regions. The test results indicate that compared with all users, whether this user has a higher prevalence of kidney diseases in the combination of genotypes of the 108 polymorphic loci tested. It does not mean the user's true risk of kidney diseases. The basis of the test is mainly from the research of Caucasian, East Asian, Latino, Native Hawaiian and American Indian and other populations are for reference only.

Notification

-
1. The test may not cover all genes or loci that cause the risk of kidney diseases.
 2. The test result has no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of the disease is determined by multiple factors such as genes and environment.
This study only predicts your risk of kidney diseases from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of kidney diseases, or you think that you are likely to suffer from kidney diseases, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 108 polymorphic loci related to the risk of kidney diseases, including rs2187668, rs4664308, rs6495446 and rs873549.

Population samples

The basis of the test is mainly from the research of Caucasian, East Asian, Latino, Native Hawaiian and American Indian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to kidney diseases.

Non-Alcoholic Fatty Liver Disease (NAFLD)

Non-alcoholic fatty liver disease refers to a clinical and pathological syndrome characterized by diffuse hepatocyte bullous steatosis from causes other than alcohol or other obvious factors of liver damage. It includes simple fatty liver disease and steatohepatitis and cirrhosis resulting from its evolution.

My Genetic Risk Is Average For The Population

0.34 times

Average incidence rate is 13500 per 100,000 in the population

Suggestions

1. Obesity is related to abnormal fat metabolism, so a healthy weight should be maintained: BMI should be maintained between 18.5 and 23.9.
2. Long-term malnutrition may also cause fatty liver. Therefore, if BMI<18.5kg/m², or weight loss (compared with usual weight, weight loss at any time>10%; or weight loss>5% within 3 months), you should pay attention to adjusting diet to strengthen nutrition. The method of weight loss must be scientific and reasonable, and the necessary energy intake must not be given up.
3. For long-term use of drugs that have side effects on the liver (such as anti-tumor drugs, anti-tuberculosis drugs), liver function should be reviewed regularly, and if abnormalities occur, the drug regimen should be adjusted under the guidance of the doctor.

Influencing factors



Controllable factors

1. Obesity: About 39% to 100% of patients with non-alcoholic fatty liver disease are overweight. Obesity, especially abdominal obesity, is closely related to abnormal fat metabolism.
2. Malnutrition: The lack of protein in the body of long-term malnourished people affects the synthesis of lipoproteins by the liver, which prevents the liver from transporting excess fat, leading

to fatty liver. Therefore, people with chronic malnutrition and excessive dieting to lose weight may have fatty liver.

3. Drugs: anti-tumor drugs, anti-tuberculosis drugs, corticosteroids and other drugs are risk factors for aggravating non-alcoholic fatty liver disease.



Uncontrollable factors

Insulin resistance: Insulin resistance is the most likely cause of non-alcoholic fatty liver disease. About 28~39% of diabetic patients have abnormal liver function, and most of them have fatty liver.

Knowledge



Time of onset

The age of onset of non-alcoholic fatty liver disease is usually 40-50 years old, and with the improvement of living standards, the age of onset tends to be younger.



Prevalence

The incidence of non-alcoholic fatty liver disease in European and American countries is close to 20%~30%.



Typical symptoms

Most patients have no symptoms, some patients may have fatigue, indigestion, liver pain, hepatosplenomegaly, etc.



Treatment

This disease can be controlled by adjusting the lifestyle, such as a reasonable diet, regular exercise, and drug treatment for liver disease.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2290602	PPARGC1A	GT	2.62	0.97
RS3772622	AGTR1	CT	1.17	1.00

RS738409	PNPLA3	CC	1.00	0.94
RS8107974	intergenic	AA	1.00	0.99
RS13118664	intergenic	AA	1.00	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to non-alcoholic fatty liver disease. By studying the genotype differences between patients with non-alcoholic fatty liver disease and normal people, these literatures have found some polymorphic sites associated with the risk of non-alcoholic fatty liver disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk item for non-alcoholic fatty liver disease uses 9 polymorphic loci of the *PPARGC1A*, *AGTR1* gene. The results show whether the user had a higher prevalence of non-alcoholic fatty liver disease than all users in the genotype combination of the 9 polymorphic loci tested, which does not represent the user's real risk of non-alcoholic fatty liver disease. The test is based mainly on Asian studies, and other populations are for informational purposes only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of non-alcoholic fatty liver disease.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing non-alcoholic fatty liver disease at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of non-alcoholic fatty liver disease, or if you feel you have a risk of developing non-alcoholic fatty liver disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 9 polymorphic loci associated with the risk of non-alcoholic fatty liver disease, including rs3772622, rs2290602, etc.

Population samples

The basis of the test is mainly from the research of Asian and European.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with non-alcoholic fatty liver disease.

Parkinson's Disease (PD)

Parkinson's disease is a type of chronic degenerative disorder of the central nervous system that compromises the patient's motor skills, language ability and other functions. It is a common neurodegenerative disease among the middle-aged and elderly that presents clinically as resting tremors, bradykinesia, muscle rigidity and postural gait disorder.

My Genetic Risk Is Average For The Population

0.32 times

Average incidence rate is 1000 per 100,000 in the population

Suggestions

1. Keep exercising for at least 4 hours a week.
2. Pay attention to avoid high-risk factors, such as head trauma, exposure to insecticides or pesticides environments, etc.

Influencing factors

Controllable factors

1. Lifestyle: Studies have found that keeping exercise helps reduce the risk of Parkinson's disease.
2. Environmental factors: Some environmental factors may increase the risk of Parkinson's disease, including pesticides, head trauma, farming or living in the countryside. Suburban environment and drinking water are more likely to be exposed to pesticides, so they may indirectly affect people's risk of disease.

Uncontrollable factors

1. Family history: People with a family history of Parkinson's disease have a high prevalence. At present, it is considered that about 10% of patients have a family history.

2. Nervous system aging: Parkinson's disease mainly occurs in the middle-aged and elderly, and rarely occurs before the age of 40, suggesting that aging is related to the disease. Some data show that after the age of 30, dopaminergic neurons in substantia nigra begin to degenerate and dopaminergic neurons gradually decrease with age. Nevertheless, its degree is not enough to cause the disease, and only a few of the elderly people suffer from the disease. Therefore, aging is only a contributing factor to Parkinson's disease.

Knowledge

Time of onset

The disease occurs more than 60 years old, occasionally under the age of 30. At present, the youngest patient is only 18 years old. Occult onset and slow progress.

Prevalence

Prevalence: 1,000/100,000. According to statistics, more than 1% of the elderly over the age of 60 in China suffer from Parkinson's disease, about 1.7% of the elderly population over the age of 65 suffer from Parkinson's disease, and the prevalence in over the age of 70 is 3% ~ 5%. It is the "third killer" of the middle-aged after tumors and cardiovascular and cerebrovascular diseases, and there are nearly 100,000 new cases every year. Currently, Parkinson's patients are becoming younger, and "juvenile Parkinson's disease" patients account for 10% of the total number.

Typical symptoms

Clinically, it is mainly characterized by rest tremor, bradykinesia, myotonia and postural gait disorder.

1. Static tremor: This is the most obvious symptom. Most of the symptoms begin at the distal end of one upper limb, gradually spread to the ipsilateral lower limb, and then spread to the contralateral upper limb and lower limb. It appears or becomes apparent in the static position, reduces or stops when moving at will, intensifies when nervous, and disappears after falling asleep.
2. Myotonia: It refers to the increase of resistance when passively moving joints. Its characteristic is that the resistance is always the same when passively moving the joint, and the resistance is basically not affected by the speed and force of passive movement, which is similar to the feeling of bending soft lead pipe, so it is called "leadpipe rigidity". In patients with static tremor, intermittent pauses in uniform resistance can be felt, like the sense of rotating gear, which is called "cog-wheel rigidity". The limb stiffness of patients with Parkinson's disease in the early stage is often asymmetric, and often occurs in the neck and shoulders, then extends to the face and limbs, and finally spreads to the whole body with the progress of the disease, making the patients gradually lose their motor ability.

3. Slow movement: It refers to the reduction of random movement, slow and clumsy movement. In the early stage, the fine finger movements, such as unbuttoning and tying shoelaces, were slow, and gradually developed into comprehensive random movement, which was reduced and slow. In the late Dynasty, it was difficult to get up and turn over due to the increase of muscle tension. There are also symptoms such as stiff face, dyskinesia of oropharyngeal and palatine muscles, dysgraphia and so on.
4. Postural and gait disorder: It refers to postural and gait instability, easy to fall and unstable posture and gait caused by the disappearance of postural reflexeasy. This symptom is an important sign of disease progression, poor response to treatment and an important cause of disability.
5. Other symptoms: Autonomic symptoms are common, such as constipation, abnormal sweating, decreased sexual function and seborrheic dermatitis (fat face). Reduced swallowing activity can lead to excessive salivation and salivation. Nearly half of the patients were accompanied by depression and/or sleep disorders. About 15% to 30% of patients develop dementia in the late stage of the disease.

Treatment

At present, no method has been developed to completely cure Parkinson's disease, but there are a number of drugs, surgery and cross domain integrated treatment can alleviate the symptoms. Drugs for the treatment of motor symptoms include L-dopa, dopamine receptor agonists and monoamine oxidase inhibitors. The specific drug selection depends on the state of the disease. Generally speaking, the latter two drugs mentioned above will be used for treatment at the initial stage of symptoms to avoid L-dopa related side effects. Only when the symptoms are strong in the later stage of symptoms will L-dopa be used. During this period, pay attention to the harm of dose fluctuation to patients, and the increase or decrease of dose can only be carried out under the prescription of doctors. When drug treatment can no longer control symptoms, deep brain stimulation surgery can be considered. At the end of the disease, palliative therapy is recommended to maintain the patient's quality of life.

My Genetic Result Details

● Low Risk

● Average Risk

● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS9652490	LINGO1	AG	1.80	0.95
RS356203	SNCA	TT	0.61	0.99
RS75859381	intergenic	TT	0.64	1.00

RS10513789	MCCC1	GG	0.64	0.99
RS356220	intergenic	CC	1.48	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to Parkinson's disease. These literatures have found some polymorphic loci related to the risk of the disease by Parkinson's disease. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of Parkinson's disease item uses the 109 polymorphic loci of *MCCC1*, *EIF4G1*, *PHACTR2*, *SREBF1*, *SLC45A3* gene. The test results show whether the user has a higher prevalence of Parkinson's disease than all users. The test cannot represent the user's real risk of Parkinson's disease. The test is mainly based on the research of Caucasian, and other populations are only for reference.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of Parkinson's disease.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

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1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of Parkinson's disease from the genetic level, which does not represent your real health status, nor does it involve the judgment of your other health status, living habits, etc.
 3. If you have a family history of Parkinson's disease, or you think that you are likely to suffer from Parkinson's disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 109 polymorphic loci related to Parkinson's disease risk, including rs10513789, rs111290936, rs112019125 and so on.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect Parkinson's disease.

Open-Angle Glaucoma

It is a chronic progressive eye disease characterized by elevated intraocular pressure, resulting in optic nerve damage and visual field defect, and may lead to blindness.

My Genetic Risk Is Average For The Population

0.32 times

Average incidence rate is 570 per 100,000 in the population

Suggestions

1. Try to avoid facing the computer, TV for a long time every day. Prolonged use of eyes in dark conditions will cause the pupil to disperse for a long time, blocking the circulation of liquid in the eye, and then our eyes may lead to glaucoma.
2. Mental stress may induce open-angle glaucoma, so when stressed out, it is recommended to talk to friends and family, relax through sports, music and other means, or consult a psychologist.
3. If you have hypertension, you should pay attention to blood pressure control and regular physical examination.

Influencing factors



Controllable factors

1. Eye time: Using eyes excessively can induce glaucoma.
2. Mental factors: long-term stress, anxiety and other mental pressure, can induce this disease.
3. Hypertension: having a history of hypertension increases the risk of primary open-angle glaucoma.



Uncontrollable factors

1. Increased eye pressure: the average eye pressure range is 10-21 mmhg. The higher the pressure, the higher the risk of glaucoma.

- 2. Age: glaucoma becomes more common as you age.
- 3. Race: people of African descent are more likely to develop glaucoma and are younger than Caucasian.
- 4. Family history: people with a history of glaucoma family disease have a higher prevalence.

Knowledge

Onset time

Common in middle and old age, the prevalence increases with age.

Prevalence

The worldwide overall prevalence of primary open-angle glaucoma was 2.4% , primary open-angle glaucoma is responsible for 12.3% of blindness worldwide. In people older than 40 years old, the prevalence of primary open-angle glaucoma is 1.86%.

Typical symptoms

There are no obvious symptoms in the early or mild stages of primary open-angle glaucoma. Until the advanced stage of the disease, it was found that eye pressure increased, optic nerve pressure, ischemic optic neuropathy, vision defects and other symptoms.

Treatment

- 1. Medical treatment: appropriate medication is given to those with progressive vision impairment, and drugs are added before peak through a 24-hour circadian eye pressure curve to avoid hidden visual impairment.
- 2. Surgical treatment: surgical treatment should be considered for those who cannot control eye pressure with medication, or who are intolerant to the drug, or whose visual function continues to decline under medication.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS10819187	intergenic	GG	1.46	1.00
RS1381486	intergenic	TT	0.68	1.00

RS2472494	intergenic	TT	1.43	0.99
RS10281637	intergenic	TT	0.73	1.00
RS4074961	RSPO1	TT	1.35	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to open-angle glaucoma. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences between the patients with open-angle glaucoma and normal subjects. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The open-angle glaucoma risk item uses 140 polymorphic loci on *TMCO1* gene, *RSPO1* gene, *RARB* gene, and between these genes. The test result indicates whether the user has a higher possibility of open-angle glaucoma in genotype combination of the 140 polymorphic loci detected compared with all users, and the test does not represent the user's real risk of open-angle glaucoma. The test is mainly based on the research of Caucasian and Japanese, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to open-angle glaucoma.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of open-angle glaucoma from the genetic level, and does not represent your real health status, nor does it involve the judgment of your other health status and living habits.
3. If you have a family history of open-angle glaucoma, or you think that you are likely to suffer from open-angle glaucoma, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 140 polymorphic loci associated with open-angle glaucoma, such as rs10918274, rs12139208.

Population samples

The basis of the test is mainly from the research of Caucasian and Japanese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect open-angle glaucoma.

Vitiligo

Vitiligo is a common skin pigment disease. This disease is characterized by localized or generalized loss of skin pigment, which forms white spots. It is an acquired localized or generalized skin pigment disorder and a common skin disease that affects appearance.

My Genetic Risk Is Average For The Population

0.31 times

Average incidence rate is 72 per 100,000 in the population

Suggestions

1. Maintain a relaxed and happy mood and avoid excessive mental stress or excessive psychological pressure.
2. When taking photosensitizing drugs (such as sulfonamides, thiazides), pay attention to sun protection.
3. Avoid excessive sun exposure to avoid sunburn.
4. Balance nutrition and pay attention to supplementing trace elements.

Influencing factors



Controllable factors

1. Mental factors: Studies have shown that negative emotions such as depression and anxiety can increase the risk of this disease.
2. Drug factors: Some drugs have photosensitization, such as sulfonamides and thiazides, which can prevent the formation of melanin and induce leukoplakia.
3. UV factor: Exposure to the sun will damage melanocytes and increase the risk of disease.
4. Trace element deficiency: Studies have shown that the lack of trace elements such as copper may also lead to this disease.

Uncontrollable factors

1. Genetic factors: Family genetic tendency.
2. Immune factors: People with immune system diseases such as systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) are at increased risk. In addition, the presence of anti-melanocyte antibodies in the blood is also related to the pathogenesis of generalized vitiligo.

Knowledge

Time of onset

The disease can occur at any age, and it is more common in young adults. About 50% of patients have the disease before the age of 20.

Prevalence

According to statistics, there are about 72 cases of the disease per 100,000 people in the average population.

Typical symptoms

It manifests as bleaching leukoplakia of various sizes and shapes, and the surrounding color is normal or there is increased pigmentation. The number of skin lesions can be one or more, which can be merged into pieces; the white spots are of different sizes and irregular shapes; the hair on the affected area can turn white; generally there are no symptoms.

Treatment

Treatment is difficult. Generally, comprehensive treatment with a course of at least 3 months is used. The treatment effect is often good if the skin lesion area is small, or occurs in the exposed part, or the course of the disease is short. The drugs that can be used for treatment include methoxsalen, high-dose vitamins, immunomodulators, corticosteroids and so on.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS4822024	intergenic	GG	0.45	0.98
RS10774624	intergenic	AA	0.62	1.00

RS1635168	HERC2	AC	1.37	0.98
RS229527	C1QTNF6	AC	1.38	1.00
RS5952553	intergenic	CC	0.74	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to vitiligo. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotypes difference between patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The vitiligo health risk report uses 50 polymorphic loci in genes such as *UBASH3A*, *LPP*, *SMOC2*, *TYR*, *RNASET2*. The test result shows whether the user has a higher prevalence of vitiligo in the genotype results at the 50 polymorphic loci detected compared to all users. This test does not represent the user's real risk of vitiligo. The basis of the test is mainly from the research of Caucasian and Chinese, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of vitiligo.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by multiple factors such as genes and environment. This study only predicts your risk of vitiligo at a genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc
3. If you have a family history of vitiligo, or you think that you are likely to suffer from vitiligo, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes more than 13 polymorphic loci associated with vitiligo risk, such as rs11203203, rs13076312, rs13208776, rs1393350, rs2236313.

Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that associated with vitiligo.

Follicular Lymphoma

It is a heterogeneous disease of abnormal lymphocyte proliferation, which originates from B cells in the follicular germinal center. Generally, the disease develops slowly.

My Genetic Risk Is Average For The Population

0.30 times

Average incidence rate is 20 per 100,000 in the population

Suggestions

1. Maintain a healthy weight: Keep BMI between 18.5 and 23.9.
2. Hold a positive attitude towards life; Pay attention to proper resting and avoid staying up late; At least 4 hours exercise per week is recommended.
3. Healthy diet: Eat less high-fat, high-sugar and high-calorie foods and eat more high-fiber foods.
4. Reject active and passive smoking, and avoid exposure to tobacco.
5. Stay away from radiation and harmful substances that may reduce immunity, and maintain a healthy body.
6. Avoid unprotected sex, pay attention to safety and hygiene, and prevent virus infection.

Influencing factors



Controllable factors

1. Obesity: Obese people have a higher risk of follicular lymphoma.
2. Lifestyle: Staying up often and lack of exercise will increase the risk of follicular lymphoma.
3. Eating habits: Those who prefer high-fat, high-sugar and high-calorie foods have a higher incidence of follicular lymphoma.
4. Smoking: Smoking increases the risk of follicular lymphoma.

-
5. Body immunity: Long-term fatigue, excessive mental stress, exposure to rays and harmful substances may cause immune system dysfunction and increase the risk of follicular lymphoma.
 6. Viral infection: Epstein-Barr virus, human T cell virus type 1, HIV and human herpes virus type 8 can directly infect lymphocytes and cause cancer.

Uncontrollable factors

1. Age: Follicular lymphoma mostly occurs in adults over the age of 40. The older you are, the more likely you are to develop follicular lymphoma.
2. Family history: People with a family history of follicular lymphoma have a higher prevalence.
3. Autoimmune diseases: rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), dry syndrome, celiac disease and other autoimmune diseases, the treatment of some diseases using immunosuppressants, resulting in a decline in the body's immunity, the risk of follicle lymphoma increased.

Knowledge

Time of onset

Follicular lymphoma mostly occurs in adults over 40 years old. The median age of onset is about 60 years old. The incidence rate of men and women is similar, but women are slightly higher.

Prevalence

Prevalence: 20 per 100,000. Follicular lymphoma is one of the common pathological types of non-Hodgkin's lymphoma, second only to diffuse large B-cell lymphoma. The incidence of follicular lymphoma in western countries accounted for 22% to 35% of non-Hodgkin's lymphoma, while the incidence in Asia decreased significantly, and the incidence of follicular lymphoma in China accounted for 8.1% to 23.5%, its incidence has been increasing in recent years.

Typical symptoms

Follicular lymphoma is derived from centrocytes and centroblasts in the center of follicle growth, which is morphologically manifested as the tumor partially retaining the pattern of follicle growth, and is a group of malignant lymphocytosis diseases that contain the filter center cells (small crack cells) and the filter center parent cells (large fission cells). Follicular lymphoma manifests itself as a slow accumulation of low-proliferative tumor cells, which belongs to low-malignant lymphoma. Clinically, painless and slow peripheral lymph node swelling is commonly seen, some patients may have lymph nodes of constant size variations. Early primary lymph node aggression, systemic symptoms such as fever, wasting and night sweating are rare, but are common symptoms at an advanced stage and often invade the bone marrow and spleen. In addition, about one-third of follicle lymphomas can be converted to diffuse, but further biopsy is required.

Treatment

- Radiotherapy is the first choice for patients with early-stage follicular lymphoma.
- For patients with high tumor burden and Follicular Lymphoma International Prognostic Index (FLIPI) scores in the middle and high-risk groups, combined immunochemotherapy, radioimmunotherapy, hematopoietic stem cell transplantation, and clinical trials of some new drugs can be considered.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS17749561	intergenic	GG	1.80	1.00
RS6457327	intergenic	AC	0.59	0.96
RS4937362	intergenic	CT	1.19	0.99
RS12195582	intergenic	CC	1.00	0.92
RS10484561	intergenic	TT	1.00	0.98

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to follicular lymphoma. These literatures have found some polymorphic loci related to the risk of the disease by studying follicular lymphoma patients and normal people. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk item of follicular lymphoma disease uses the 8 polymorphic loci on genes such as *LPP* and *PVT1*. The test results indicate that compared with all users, whether the combination of genotypes at the 8 polymorphic loci detected by this test increases the risk of follicular lymphoma disease. It does not mean the user's true risk of follicular lymphoma disease. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of follicular lymphoma.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of follicular lymphoma at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of follicular lymphoma, or you think that you are likely to suffer from follicular lymphoma, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 8 polymorphic loci related to the risk of follicular lymphoma, including rs6457327 , rs12195582 and rs10484561.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to follicular lymphoma.

Vascular Dementia

Vascular dementia refers to severe cognitive dysfunction syndrome caused by ischemic stroke and hemorrhagic stroke moderate cerebrovascular disease.

My Genetic Risk Is Average For The Population

0.30 times

Average incidence rate is 313 per 100,000 in the population

Suggestions

1. Control the intake of fat, cholesterol and salt in the diet, maintain normal blood glucose, blood lipid, and blood pressure levels, and reduce blood vessel damage and load.
2. Maintain a healthy weight: BMI is maintained between 18.5-23.9.
3. Maintain a healthy habits, do not stay up late, maintain active and moderate exercise, and exercise should not be too intense.

Influencing factors

Controllable factors

1. Dietary factors: A high-sugar and high-fat diet, eating more meat and less vegetables, high sodium intake, and low potassium intake will all increase the risk of vascular dementia.
2. Overweight and obesity: With the increase of BMI and waist circumference, the mean blood pressure showed a significant increase trend, and the prevalence of vascular dementia also increased significantly.
3. Poor living habits: Sitting for a long time and lack of exercise may increase the risk of vascular dementia.

Uncontrollable factors

1. Age: Common in the middle-aged and elderly.
2. Gender: There are more male patients than female patients.

3. Heredity: Some single gene hereditary cerebrovascular diseases can lead to vascular dementia.
 4. Disease: Cardiovascular diseases such as hypertension and hyperlipidemia will increase the probability of suffering from the disease.
-

Knowledge

Time of onset

It usually starts after 60 years of age. In recent years, the age of onset tends to be younger, with more men than women. The duration of the disease is as short as 2 months and as long as more than 20 years.

Prevalence

Foreign studies show that the incidence of vascular dementia in people over 60 years old is 242 per 100,000 people, and the incidence of people over 65 is 313 per 100,000 people.

Clinical manifestations

Patients with vascular dementia can show different forms of cognitive decline, which varies according to the damaged brain functional areas. Its early manifestations are mainly headache, dizziness, acroanaesthesia, sleep disorder, tinnitus, etc. It may have mild memory impairment, inattention and some emotional changes in the near future. However, with the disease progresses, neuropsychiatric symptoms will appear, such as unclear pronunciation, dysphagia, muscle paralysis, agnosia, urinary incontinence, hearing sounds out of thin air (auditory hallucination), seeing things that do not actually exist (visual hallucination), or emotional vulnerability, irritability, impermanence of crying and laughing, etc.

Treatment

The general treatment is the same as that of all dementia, including improving safety and supporting treatment. Controlling blood glucose, lowering blood pressure and lowering blood lipid can prevent or delay the occurrence and development of vascular dementia. It is recommended to quit smoking.

There is no special treatment for vascular dementia. Because most patients with vascular dementia have Alzheimer's disease at the same time, some acetylcholinesterase inhibitors and memantine used to treat Alzheimer's disease can be used. For stroke patients, doctors may recommend may recommend them to take aspirin, which can reduce the risk of stroke again. Patients with atrial fibrillation taking warfarin (an anticoagulant) can help reduce the risk of stroke again.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS290227	SYK	AG	2.20	0.86
RS16952362	intergenic	GG	1.00	0.79
RS1801133	MTHFR	GG	1.00	0.98
RS429358	APOE	TT	1.00	0.98
RS1333049	intergenic	GG	1.00	0.98

Risk calculation

The risk value of this test is calculated based on published academic literatures related to vascular dementia. These literatures have found some polymorphic loci related to the risk of the disease by vascular dementia. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of vascular dementia item uses the 5 polymorphic loci of *APOE*, *MTHFR* gene. The test results show whether the user has a higher prevalence of vascular dementia than all users. The test cannot represent the user's real risk of vascular dementia. The test is mainly based on the research of Caucasian, and other populations are only for reference.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of vascular dementia.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only predicts your risk of vascular dementia from the genetic level, which does not represent your real health status, nor does it involve the judgment of your other health status, living habits, etc.
3. If you have a family history of vascular dementia, or you think that you are likely to suffer from vascular dementia, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 5 polymorphic loci related to vascular dementia risk, including rs16952362 and so on.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect vascular dementia.

Atrial Fibrillation

Atrial fibrillation is a kind of abnormal rhythm of the heart, which is characterized by rapid and irregular beating of the heart. It can occur regardless of sex or age, and with or without organic heart disease.

My Genetic Risk Is Average For The Population

0.29 times

Average incidence rate is 5000 per 100,000 in the population

Suggestions

1. Maintain a good lifestyle: exercise moderately, avoid overwork, avoid excessive intake of alcohol and coffee, etc. The limit value of moderate drinking for adults recommended by the Chinese Nutrition Society is that the amount of alcohol consumed by adult men should not exceed 25g per day; the amount of alcohol consumed by adult women should not exceed 15g per day.
 2. Maintain an optimistic attitude, control your emotions, and not be happy or sad.
-

Influencing factors

Controllable factors

1. Unhealthy lifestyle: Studies have shown that fatigue, excessive intake of alcohol and coffee, etc. can induce the occurrence of atrial fibrillation.
2. Negative emotions such as emotional agitation and psychentonia can also lead to the occurrence of atrial fibrillation.

Uncontrollable factors

1. Genetic factors: There is a family genetic tendency.
2. Age factor: Common in the elderly, the incidence doubles for every 10 years old after 60.
3. Disease factors: Patients with coronary heart disease, hypertensive heart disease, various types of cardiomyopathy, congenital heart disease, constrictive pericarditis, pulmonary heart disease, and

hyperthyroidism are more likely to develop atrial fibrillation.

Knowledge

Time of onset

The disease can occur at any age, and it is common in elderly people over 60 years old.

Prevalence

According to statistics, there are about 5,000 cases of the disease per 100,000 people in the average population.

Typical Symptoms

Mild cases have palpitation, palpitations, chest tightness, shortness of breath, fatigue and other symptoms; severe cases of rapid heartbeat can induce angina, pulmonary edema, blood pressure reduction, and even cardiogenic shock, syncope, etc., requiring emergency treatment.

Treatment

There are a large proportion of clinical patients cannot completely eliminate atrial fibrillation. Generally speaking, treatment has three main goals: (1) control ventricular rate; (2) control heart rhythm; (3) prevent thromboembolism.

There are many kinds of drugs for the treatment of atrial fibrillation, such as β -blockers, calcium channel antagonists, digitalis, amiodarone and so on. Anticoagulant therapy is a necessary means to prevent thrombosis and embolism in patients with atrial fibrillation. The main drugs are warfarin, aspirin or/and clopidogrel.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS67249485	intergenic	AT	1.44	0.98
RS17461925	PPFIA4	AA	1.44	0.99
RS2359171	intergenic	AA	1.42	0.99
RS16997168	intergenic	CT	1.39	0.98

RS10033464

intergenic

GT

1.39

0.98

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to atrial fibrillation. These literatures have found some polymorphic loci related to the risk of the disease by studying atrial fibrillation. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of atrial fibrillation item uses 131 polymorphic loci of *PRRX1*, *HAND2*, *WNT8A*, *CAV1*, *RPL3L*, etc genes. The test results indicate whether the user has a higher prevalence of atrial fibrillation in the combination of genotypes at the 131 polymorphic loci tested compared to all users. It does not mean the user's true risk of atrial fibrillation. The basis of the test is mainly from the research of Caucasian, African American, Brazilian, Japanese, and Korean, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that can lead to the risk of atrial fibrillation.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.

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2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only predicts your risk of atrial fibrillation from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
 3. If you have a family history of atrial fibrillation, or you think that you are likely to suffer from atrial fibrillation, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 131 polymorphisms related to the risk of atrial fibrillation, including rs10033464, rs2200733, and rs3903239.

Population samples

The basis of the test is mainly from the research of Caucasian, African American, Brazilian, Japanese, and Korean.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to atrial fibrillation.

Stroke

Also known as cerebrovascular accident, it is a group of diseases with symptoms of brain ischemia and hemorrhagic injury as its main clinical manifestations. It has an extremely high rate of mortality and morbidity.

My Genetic Risk Is Average For The Population

0.26 times

Average incidence rate is 445 per 100,000 in the population

Suggestions

- Common symptoms: Unconsciousness in one limb, loss of vision on one or both sides, headache, dizziness, slurred speech
- High-risk population: Obesity, drinking, elderly people, smoking, hypertension or diabetes, and family history of stroke
- How to reduce the risk:
 - Maintain normal blood pressure, blood sugar and blood lipid levels
 - Pay attention to diet: Reduce alcohol consumption, high-fat food, high-calorie food, and high-salt food; Increase intake of fruits and vegetables
 - Quit smoking: Avoid active smoking; Stay away from second-hand smoking
 - Weight control: Keep BMI within 18.5-23.9; Avoid sitting for a long time, and exercise at least 4 hours per week
 - Protective food: Consider **folic acid**, **Omega-3** and other supplements
- The Heritability of stroke is about 16%-73%

* If you have a family history of stroke, or if you think you are likely to have a stroke, or need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors



Controllable factors

1. Dietary factors: A balanced diet to maintain normal blood sugar, blood lipid, and blood pressure levels can reduce the risk of stroke.

-
2. Tobacco and alcohol intake: Data show that the risk of stroke in smokers or passive smokers is twice that of non-smokers. Alcohol raises blood pressure and increases the content of certain fatty acids in the blood, which may cause arteriosclerosis and even stroke.
 3. Living habit factors: Hard urination and bowel movements, emotional agitation, overeating, excessive exercise, hot baths in winter, etc. may cause stroke.

Uncontrollable factors

1. Age: The elderly are more prone to stroke.
 2. Disease factors: Hypertension, atherosclerosis, heart disease, aneurysms caused by abnormal intracranial vascular development, arteriovenous malformations, cerebral arteritis, various inflammations that invade cerebral blood vessels, rheumatism, leukemia, diabetes, etc., may cause stroke.
 3. Trauma and treatment factors: All kinds of trauma, poisoning, or radiation therapy for brain tumors may cause stroke.
 4. Family history: People with a family history of stroke have a higher prevalence.
 5. Medication factors: Improper use of anticoagulant drugs, contraceptives, and postmenopausal hormone replacement therapy may increase the risk of stroke.
-

Knowledge

Time of onset

The incidence increases with age, and about 72% of first strokes occur in elderly over 65 years of age.

Prevalence

Statistics have shown that there are approximately 445 strokes per 100,000 people in the general population.

Typical symptoms

There are two types of stroke:

- Ischemic stroke caused by blood vessel obstruction.
- Hemorrhagic stroke caused by hemorrhage.

Both ischemic and hemorrhagic stroke can cause abnormal brain function. Common symptoms of stroke include inability to move one limb, loss of sensation on one side, inability to understand what others are saying, inability to speak, dizziness, and loss of vision on one side. The symptoms of a stroke usually appear shortly after its onset. If the symptoms disappear within 24 hours, it is often referred to as transient ischemic attack (TIA), or mini-stroke. The symptoms of a stroke may become permanent sequelae; Stroke patients may also have long-term sequelae such as pneumonia and

urinary incontinence. Patients with hemorrhagic stroke may experience sudden and severe headaches.

Treatment

Early identification of stroke is very important. Only through detailed physical examination and medical imaging can a stroke be diagnosed, as well as the type and severity of the stroke. Therefore, even for general signs of stroke, it is important to seek medical attention promptly.

- Ischemic stroke: At the onset of the disease, antiplatelet or anticoagulant drugs can be given to the patient according to the situation; Whether to implement thrombolytic therapy requires examination and evaluation. Other methods to protect the brain during a stroke include ensuring normal blood sugar levels as much as possible, ensuring adequate oxygen supply, and having the patient's head flat on a stretcher instead of sitting straight. If the examination shows stenosis of the internal carotid artery in the patient and residual function is still present in the affected area, carotid endarterectomy should be performed promptly after stroke. If ischemic stroke is caused by arrhythmia-induced clot, anticoagulation with warfarin or high-dose aspirin can be used to prevent embolism.
- Hemorrhagic stroke: Patients with intracerebral hemorrhage or pericerebral hemorrhage (subarachnoid hemorrhage) require neurosurgical examination to identify and treat the cause of bleeding.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS3783799	PRKCH	CT	1.69	0.97
RS966221	PDE4D	AG	1.51	0.97
RS12932445	ZFH3	CC	1.44	0.99
RS13143308	intergenic	GT	1.32	0.99
RS17612742	EDNRA	CT	1.19	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to stroke. By studying the genotype differences between patients with stroke and normal people, these literatures

have found some polymorphic sites associated with the risk of stroke. We uses the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

A health risk item for stroke uses 43 genetic and intergenic polymorphic loci of genes such as *PRKCH*, *PDE4D*, *ABCG2*, and *ALOX5AP*. The results show whether the user had a higher prevalence of stroke than all users in the genotype combination of the 43 polymorphic loci tested, which does not represent the user's real risk of stroke. The test is based mainly on Caucasian, Asian, and African American studies, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of stroke.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing stroke at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of stroke, or if you feel you have a risk of developing stroke, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 43 polymorphic loci associated with the risk of stroke, including rs12425791, rs3783799, and rs966221.



Population samples

The basis of the test is mainly from the research of Caucasian, Asian, and African American.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with stroke.

Hypertriglyceridemia

Hypertriglyceridemia is a an interracial triglyceride protein synthesis or degradation disorder. It is an important risk factor for the development of metabolic syndrome-related diseases, such as coronary artery disease, hypertension, diabetes, etc.

My Genetic Risk Is Average For The Population

0.25 times

Average incidence rate is 11900 per 100,000 in the population

Suggestions

- Red flags: serum Triglycerides is too high
- Common symptoms: generally no obvious symptoms, can cause fatty liver, Eyelid xanthoma
- High-risk population: Obesity, drinking, smoking, high-fat diet, diabetes and hypothyroidism disease patients, long-term beta-blockers and thiazide diuretic drug users
- How to reduce the risk:
 - Weight control: Keep BMI between 18.5 and 23.9, avoid sitting for a long time, and exercise at least 4 hours a week
 - Pay attention to your diet: drink less alcohol and eat less high-fat, high-calorie, high-salt foods
 - Quit smoking: refuse to take the initiative to smoke, stay away from second-hand smoke
 - Protective food: Consider eating protective foods or supplements such as garlic and Omega-3 fatty acids
- The Heritability of hypertriglyceridemia is about 37%

* If you have a family history of hypertriglyceridemia, or if you feel you are likely to suffer from hypertriglyceridemia, or need to use dietary supplements, it is recommended that you consult an authoritative health professional Or the clinician asks for help.

Influencing factors



Controllable factors

1. Obesity: Studies have shown that people who are overweight and obese have a significantly higher chance of getting the disease than the normal population.

2. Poor lifestyle: high-carbohydrate diet, smoking, alcoholism, etc. can induce illness.
3. Hormones: Decreased estrogen levels can cause lipid metabolism disorders, which may induce illness.
4. Drug factors: Long-term use of drugs such as β -blockers, thiazide diuretics can induce illness.

Uncontrollable factors

Disease factors: Many diseases, such as diabetes, kidney disease, hypothyroidism, glycogen accumulation syndrome, etc., can induce hypertriglyceridemia.

Knowledge

Time of onset

There is no specific time of onset, mostly adults, middle-aged and elderly people.

Prevalence

According to statistics, there are about 11,900 cases of the disease per 100,000 people.

Typical symptoms

Generally, there are no obvious symptoms, which can cause fatty liver and eyelid xanthoma (small yellow grains on the eyelids). The consequence of high triglycerides is that it is easy to cause "blood thick", easy to deposit on the blood vessel wall, and gradually form small plaques, that is, atherosclerosis.

Treatment

1. Boundary increase: diet control, weight loss.
2. Mild to moderate increase: drug intervention should be given, which can be used alone or in combination with statins, fibrates or niacin drugs.
3. Severe increase: Use fibrates or niacin lipid-lowering drugs to reduce triglyceride levels, and be alert to high levels of triglycerides that induce acute pancreatitis.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average

RS261342	intergenic	CG	2.13	0.97
RS9686661	C5orf67	CC	1.00	0.93
RS1260326	GCKR	CC	1.00	0.94
RS2068888	intergenic	AA	1.00	0.95
RS964184	ZPR1	CC	1.00	0.96

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to hypertriglyceridemia. By studying the genotype differences between patients with hypertriglyceridemia and normal people, these literatures have found some polymorphic sites associated with the risk of hypertriglyceridemia. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk item for hypertriglyceridemia uses 6 genetic and intergenic polymorphic loci of genes such as *ZPR1*, *C5orf67*, and *GCKR*. The results show whether the user had a higher prevalence of hypertriglyceridemia than all users in the genotype combination of the 6 polymorphic loci tested, which does not represent the user's real risk of hypertriglyceridemia. The test is based mainly on Caucasian studies, and other populations are for informational purposes only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of hypertriglyceridemia.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing hypertriglyceridemia at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of hypertriglyceridemia, or if you feel you have a risk of developing hypertriglyceridemia, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 6 polymorphic loci associated with the risk of hypertriglyceridemia, including rs964184, rs9686661 and rs1260326.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with hypertriglyceridemia.

Thyroid Cancer

Thyroid cancer's clinical manifestations are nonspecific, and it is often accompanied by thyroid adenoma, nodular goiter, hyperthyroidism and other benign thyroid diseases.

My Genetic Risk Is Average For The Population

0.23 times

Average incidence rate is 11 per 100,000 in the population

Suggestions

1. Take protective measures to avoid exposure to radiation.
2. Strengthen exercise, enhance physique, improve autoimmunity and prevent autoimmune diseases.

Influencing factors



Controllable factors

1. Environmental exposure: Researchers suspect that exposure to natural or artificial ionizing radiation may play a major role in the mutation of thyroid cells.
2. Decreased immunity: Decreased immunity can easily lead to disease.



Uncontrollable factors

1. Gender: The incidence of women is three times that of men.
2. Age: It mostly occurs in people over 50 years old.
3. Disease: Inflammation of the thyroid gland and other thyroid symptoms can easily develop into thyroid cancer.

Knowledge



Time of onset

Benign lumps are more common before the age of 40 and may not progress in 20 to 30 years. In the late stage, patients over 50-60 years of age may progress faster.



Prevalence

Thyroid cancer accounts for about 1.3%-1.5% of systemic malignant tumors, which has an upward trend in recent years.



Typical symptoms

1. Early clinical manifestations are not obvious. Patients or family members and doctors accidentally find a thyroid mass in the neck. Most of them have no symptoms. Neck masses are often asymmetrical lumps.
2. After a period of time, the thyroid nodules can gradually increase, move up and down with swallowing, and can invade the trachea and be fixed, accompanied by hoarseness, poor breathing, difficulty swallowing, or local tenderness and other compression symptoms. When the jugular vein is compressed, physical signs such as ventilated veins on the affected side and facial edema may appear.
3. Cervical lymph node metastasis and distant metastasis can also occur, such as lung metastasis and bone metastasis, and even pathological fractures.
4. The thyroid function may be normal, but in the late stage, there may be multiple hypothyroidism.



Treatment

1. Surgical treatment: Once thyroid cancer is diagnosed or a patient with a high level of suspicion for thyroid cancer, surgical treatment is generally required as soon as possible.
2. Radioactive Iodine (I-131) Therapy: Iodine 131 scan can show residual cancer tissues or distant metastases after surgery.
3. External radiotherapy: Undifferentiated cancer has a certain degree of radiosensitivity, and radiotherapy can be used.
4. Chemotherapy: Thyroid cancer is not sensitive to chemotherapy and can be used for comprehensive palliative treatment of thyroid cancer.
5. Percutaneous ethanol injection treatment: mainly used for the treatment of solid small to medium nodules.
6. Symptomatic treatment: When hypoparathyroidism occurs after thyroid cancer surgery, calcium and vitamin D can be supplemented.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1867277	FOXE1	AG	1.48	0.98
RS2145418	intergenic	AA	1.00	0.70
RS965513	intergenic	GG	1.00	0.95
RS699947	intergenic	CC	1.00	0.99
RS944289	intergenic	CC	1.00	0.99

Risk calculation

The risk value of this test is calculated based on published academic literature related to thyroid cancer. By studying the genotype differences between patients with thyroid cancer and normal people, these literatures have found some polymorphic sites associated with the risk of thyroid cancer. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

A health risk item for thyroid cancer uses 5 polymorphic loci on the *FOXE1* gene. The results show whether the user had a higher prevalence of thyroid cancer than all users in the genotype combination of the 5 polymorphic loci tested, which does not represent the user's real risk of thyroid cancer. The test is based mainly on Caucasian studies, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of thyroid cancer.

-
2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing thyroid cancer at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If your family has a family history of thyroid cancer, or if you feel you have a risk of developing thyroid cancer, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 5 polymorphic loci associated with the risk of thyroid cancer, including rs1867277, rs2145418 and rs699947.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with thyroid cancer.

Autoimmune Hepatitis

It is a chronic disease in which the immune system attacks the liver, causing inflammation and liver damage.

My Genetic Risk Is Average For The Population

0.20 times

Average incidence rate is 17 per 100,000 in the population

Suggestions

1. Maintain good living habits: Pay attention to diet, avoid drinking alcohol, have Physical exercise, have sufficient rest, etc.
2. Be mindful of medication use and infection status.

Influencing factors



Controllable factors

1. Bad living habits: Regular drinking, eating too greasy food, lack of exercise, etc., may be related to the occurrence of autoimmune hepatitis.



Uncontrollable factors

1. Drugs: Minocycline, methyldopa, meloxicam, etc. may cause autoimmune hepatitis.
2. Infection: Measles virus, hepatitis virus, cytomegalovirus and other infections may be involved in the occurrence of the disease.
3. Gender: The incidence of the disease is higher in women.
4. Family history: The disease has a certain family genetic tendency.

Knowledge

Time of onset

Autoimmune hepatitis can occur at any age.

Prevalence

This disease has an estimated prevalence of 10-24 per 100,000 individuals. The incidence is higher in women.

Typical symptoms

Most patients had no obvious symptoms or only non-specific symptoms such as fatigue. About 1/3 of the patients were diagnosed with liver cirrhosis.

Treatment

Without clinical intervention, patients may rapidly progress to cirrhosis or end-stage liver disease. Therefore, the treatment goal is mainly to prevent the progression to cirrhosis and/or liver failure, thereby improving the survival time and quality of life of patients. At present, the first-line treatment mainly adopts non-specific immunosuppression: prednisone/prednisolone combined with azathioprine, or prednisone/prednisolone monotherapy.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS6932730	intergenic	TT	1.00	0.89
RS3891175	HLA-DQB1	CC	1.00	0.90
RS2187668	HLA-DQA1	CC	1.00	0.93
RS6809477	SYNPR	CC	1.00	0.99
RS72929257	intergenic	CC	1.00	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to autoimmune hepatitis. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype difference between patients with autoimmune hepatitis and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The autoimmune hepatitis health risk item uses the 6 polymorphic loci on genes such as *HLA-DQA1* and *SYNPR* and within other intergenic regions. The test results indicate whether the user has a higher prevalence of autoimmune hepatitis in the genotype results at the 6 polymorphic loci tested compared to all users. The test cannot represent the user's real risk of autoimmune hepatitis. The basis of the test is mainly from the research of Caucasian and Chinese, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of autoimmune hepatitis.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by multiple factors such as genes and environment. This study only predicts your risk of autoimmune hepatitis at a genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of autoimmune hepatitis, or you think that you are likely to suffer from autoimmune hepatitis, it is recommended that you consult an authoritative health professional or

clinician for help.

 Test content

The test includes 6 polymorphic loci related to the risk of autoimmune hepatitis, including rs2187668, rs6000782, rs6809477, and rs6932730.

 Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

 Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all genes or loci that affect autoimmune hepatitis.

Asthma

Asthma is a common chronic inflammatory disease of the airways. The main features of asthma are changing and recurring symptoms, reversible obstruction of airflow, and bronchial spasms.

My Genetic Risk Is Average For The Population

0.16 times

Average incidence rate is 1240 per 100,000 in the population

Suggestions

1. Keep the room clean, and wash and dry the sheets and bedding at least once every 1 month; In the flowering season, try to close the windows and stay away from trees that are easy to bloom, so as to stay away from potential allergens.
2. If the air pollution in the living city is serious, it is best to wear a mask when going out, and those with conditions move to a place with good air quality.
3. Quit smoking and try to stay away from second-hand smoke.
4. In the flu season, try to avoid crowded places, and pay attention to increasing or decreasing clothes with the season to reduce respiratory tract infection.
5. Asthma is associated with obesity, so we should maintain a healthy weight: BMI should be maintained between 18.5 and 23.9.
6. The new house shall be ventilated for at least 3 months after decoration, so as to avoid inhaling formaldehyde which can cause asthma.
7. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and efficacy of treatment methods vary from person to person and should always

be undertaken under the advice and supervision of a medical professional, avoiding self-experimentation.

Influencing factors

Controllable factors

1. Allergens: the main motivator for asthma is inhalation of allergens. Common indoor allergens include dust mites, cockroaches, animal dander and mold, and outdoor allergens are mainly pollen. There are also certain medications (such as aspirin) and foods (tree nuts, milk, peanuts, seafood, etc.) that may cause allergies.
2. Air pollution: air pollution (SO₂, NO) can cause bronchial contraction, increased airway reactivity and increased response to allergens.
3. Smoking: cigarette smoke (including passive smoking) is an important asthma booster, especially in children with asthma whose parents smoke, often causing asthma attacks due to smoke.
4. Respiratory virus infection: closely related to asthma attacks, more pronounced in winter and spring or when the climate is variable.
5. Obesity: obesity can lead to decreased respiratory function, and adipose tissue can cause pro-inflammatory conditions
6. Volatile organic exposure: inhalation of formaldehyde, for example, has a positive association with asthma attacks.
7. Overwork: intense and prolonged physical labor, or intense competitive exercise can trigger asthma.
8. Mental factors: mood swings can be a trigger, such as anxiety, sadness, overexcitement, and even laughter, which can lead to asthma attacks.

Uncontrollable factors

1. Age: the incidence rate of children is higher than that of young adults, and children aged 1 to 6 years are the highest incidence group.
2. Have a history of specific diseases: patients with eczema and allergic rhinitis have a higher risk of developing asthma.

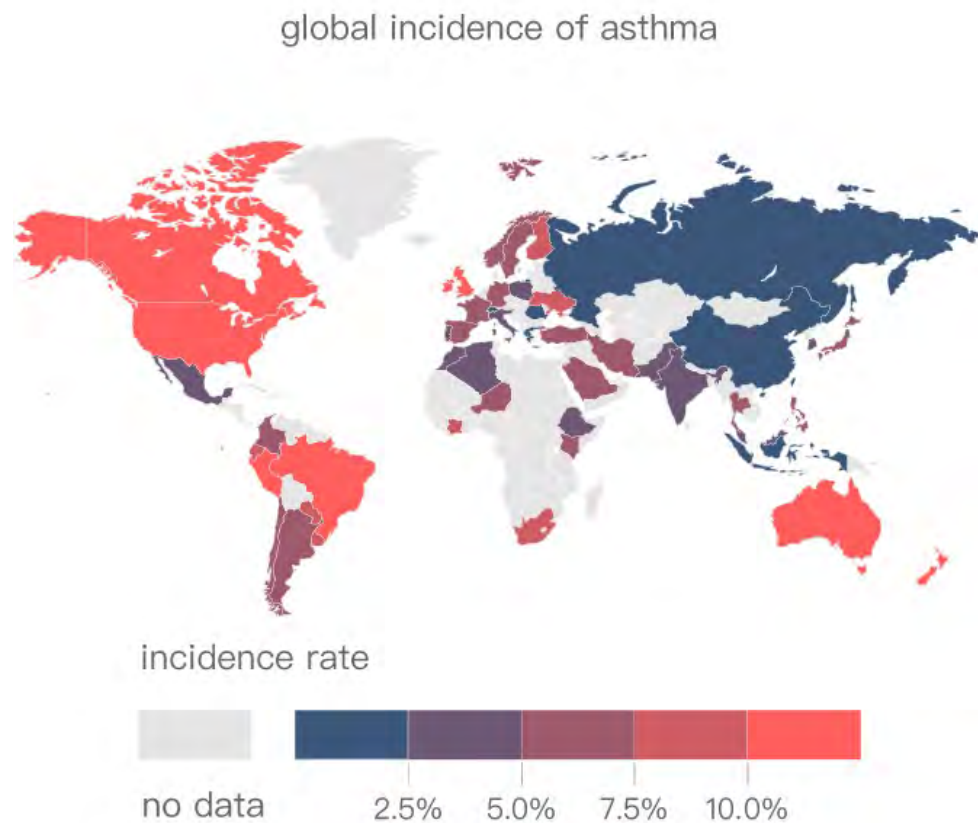
Knowledge

Time of onset

Asthma can occur at any age, children are higher than young adults, children aged 1 to 6 years are high incidence of the population; and the prevalence of the elderly is also increasing.

Prevalence

The prevalence rate of asthma among people over 14 years of age in China is 1240 million/100,000, and the prevalence rate of people under 14 years of age is 3020 million/100,000.



Typical symptoms

Asthma is typically characterized by recurrent coughing, chest tightness, wheezing and breathing difficulties, often before the attack of nasal congestion, sneezing, itching and other precursor symptoms, often at night or in the early morning attacks and aggravation. Symptoms are characterized by reversibility, that is, it can be relieved in a short time after treatment.

Treatment

Asthma is currently incurable, but can be prevented and alleviated in a number of ways, such as avoiding exposure to allergens, improving lifestyle, and using inhaled drugs.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1695	GSTP1	AG	0.32	0.85
RS4794067	intergenic	CC	2.15	0.94
RS7216389	GSDMB	TT	1.71	0.99
RS7918084	intergenic	TT	1.31	1.00
RS7523907	CD247	TT	1.30	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to asthma. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotypes difference between patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The asthma health risk project uses 62 polymorphic loci in genes such as *SLC6A7*, *TLR2*, *GSTP1*. The test result shows whether the user has a higher prevalence of asthma disease in the genotype results at the 62 polymorphic loci detected compared to all users. This test does not represent the user's real risk of asthma. The basis of the test is mainly from the research of Caucasian and Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of asthma.

-
2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by multiple factors such as genes and environment. This study only predicts your risk of asthma at a genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc
3. If you have a family history of asthma, or you think that you are likely to suffer from asthma, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 62 polymorphic loci associated with asthma risk, rs13153971, rs1695, rs3804100, rs4794067, rs7216389.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that associated with asthma.

Obstructive Sleep Apnea

Obstructive sleep apnea is a sleep apnea disease, which is often accompanied by abnormal noise and special snoring. Overweight and obesity may increase the risk of disease.

My Genetic Risk Is Average For The Population

0.15 times

Average incidence rate is 4500 per 100,000 in the population

Suggestions

1. Control your weight: keep your BMI between 18.5 and 23.9, avoid sitting for a long time, and exercise at least 4 hours a week
2. Improve sleeping posture: You can raise the head of the bed appropriately or switch to sleeping on the side. Improve airway patency through sleeping position and reduce the impact of obstructive sleep apnea.
3. Reduce the impact of lifestyle habits: It is recommended to quit smoking and drinking, use sedative hypnotic drugs and other drugs that may cause obstructive sleep apnea with caution.
4. Reduce the impact of stress: It is recommended to avoid over-stressed work and slow down the pace of life. At the same time, avoid overwork during the day and avoid staying up late.

Influencing factors



Controllable factors

1. Obesity: Research finds that exceeding standard weight increases the risk of obstructive sleep apnea.
2. Sleep quality: Low pillows may affect obstructive sleep apnea. Sleeping on your back has a higher risk of sleep apnea than lying on your side.
3. Lifestyle: Drinking alcohol, taking tranquilizers or sleeping pills can increase the risk of obstructive sleep apnea by reducing the muscle tone of the upper respiratory tract.

4. Social factors: more common in modern cities with high pressure and fast-paced life.

Uncontrollable factors

1. Age: common in middle and old age.
2. Gender: Men are twice as likely to get the disease as women. But after menopause, women have the same probability of getting the disease as men.
3. Physiological features: The following physiological characteristics also increase the risk of disease, such as too small space behind the tongue or posterior hanging urn, too large tongue, tonsil hypertrophy, excessive soft tissues of the mouth and nasopharynx, too high soft jaw, receding mandible, too small mandible or facial deformities, etc.

Knowledge

Time of onset

Any age group may suffer from this disease, but it is common between 55 and 60 years of age.

Prevalence

According to the survey, about 4.5% of people in China have obvious obstructive sleep apnea, of which about 6% are men and about 2.5% are women.

Typical symptoms

- The loud snoring sound can be heard by people (even neighbors) in several rooms.
- The snoring pattern is special. The snoring sound will be interrupted due to the stop of breathing, and then gasp, which means that the person's breathing has stopped and then started breathing.
- Sleepiness during the day.
- Inattention, forgetfulness, irritability, anxiety or depression.
- Headache in the morning, frequent urination at night, and decreased libido.
- Decreased blood oxygen levels during apnea (<90%).
- Children with this disease are also accompanied by symptoms of attention deficit hyperactivity disorder.
- Impotence occurs occasionally in men and menstrual disorders may occur in women.

Treatment

Treatment methods include restoring a healthy weight, using respiratory assist devices, and surgical treatment. Overweight and obese patients can start by reducing their weight. Restoring a healthy weight can help reduce the occurrence of obstructive sleep apnea. More severe patients need to be evaluated by a doctor to use respiratory assist devices or undergo surgical treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS12415421	ST8SIA6-AS1	CC	1.00	0.67
RS10097555	NRG1	AG	0.84	0.99
RS2975100	GDNF-AS1	GG	1.00	0.91
RS10928560	intergenic	CT	0.92	1.00
RS2808630	intergenic	TT	1.00	0.94

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to obstructive sleep apnea. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotypes difference between patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The obstructive sleep apnea health risk project uses 11 polymorphic loci in genes such as *NRG1*, *ST8SIA6-AS1*. The test result shows whether the user has a higher prevalence of obstructive sleep apnea disease in the genotype results at the 11 polymorphic loci detected compared to all users. This test does not represent the user's real risk of obstructive sleep apnea disease. The basis of the test is

mainly from the research of Caucasian, East Asian and African American, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of obstructive sleep apnea.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by multiple factors such as genes and environment. This study only predicts your risk of obstructive sleep apnea at a genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc
3. If you have a family history of obstructive sleep apnea, or you think that you are likely to suffer from obstructive sleep apnea, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 11 polymorphic loci associated with obstructive sleep apnea risk, such as rs10097555, rs12415421 and rs9526240.

Population samples

The basis of the test is mainly from the research of Caucasian, East Asian and African American.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that associated with obstructive sleep apnea.

Type 2 Diabetes (T2D)

Type 2 diabetes is a metabolic disorder that is characterized by high blood sugar. It is mainly caused by insulin resistance and a relative lack of insulin.

My Genetic Risk Is Average For The Population

0.11 times

Average incidence rate is 9700 per 100,000 in the population

Suggestions

- Red flags: Excessive blood glucose
- Common symptoms: "three more and one less", polydipsia, polyphagia, polyuria, weight loss, and be prone to fatigue in the early stage.
- High risk population: Overweight and obesity (especially central obesity), family history of type 2 diabetes, sedentary and less active, polycystic ovary syndrome, history of hypertension or dyslipidemia, most commonly seen in patients over 45 years old.
- How to reduce risk:
 - Control weight, limit calorie intake, cultivate exercise habits and develop Healthy lifestyle
 - Low glycemic index (GI) diets, such as Coarse grain and milk, help control blood glucose.
 - Regular physical examination, monitor blood glucose level.
 - Relax.
- The Heritability of type 2 diabetes is about 25%-80%.

* If your family has a family history of type 2 diabetes, or you feel that you are suffering from type 2 diabetes, or you need to use dietary supplements, it is recommended that you consult an authoritative health expert or a clinician for help.

Influencing factors



Controllable factors

1. Obesity: People who are overweight ($\text{BMI} \geq 24 \text{ kg/m}^2$), obese ($\text{BMI} \geq 28 \text{ kg/m}^2$), or central (male waist circumference $\geq 90 \text{ cm}$ and female waist circumference $\geq 85 \text{ cm}$) are more likely to have type 2 diabetes.

-
2. Eat more and move less: people who eat too much high-fat, high-sugar foods for long periods of time, and lack physical activity, have an increased risk of type 2 diabetes.
 3. Mental factors: long-term stress, anxiety and other mental pressure, can induce type 2 diabetes.
 4. Hypertension, hyperlipidemia: A history of hypertension and hyperlipidemia will increase the risk of type 2 diabetes.

Uncontrollable factors

1. Age: The prevalence of type 2 diabetes increases with age, and it is more common in people over 55 years of age.
2. Family history: Type 2 diabetes has a relatively obvious tendency to cluster disease in the family. If there are diabetic patients among parents, children, or brothers and sisters, the risk of developing the disease will be greatly increased.
3. Race: Type 2 diabetes has a higher risk of developing the disease in ethnic groups such as South Asians, Pacific Islanders, Hispanics, and Native Americans.

Knowledge

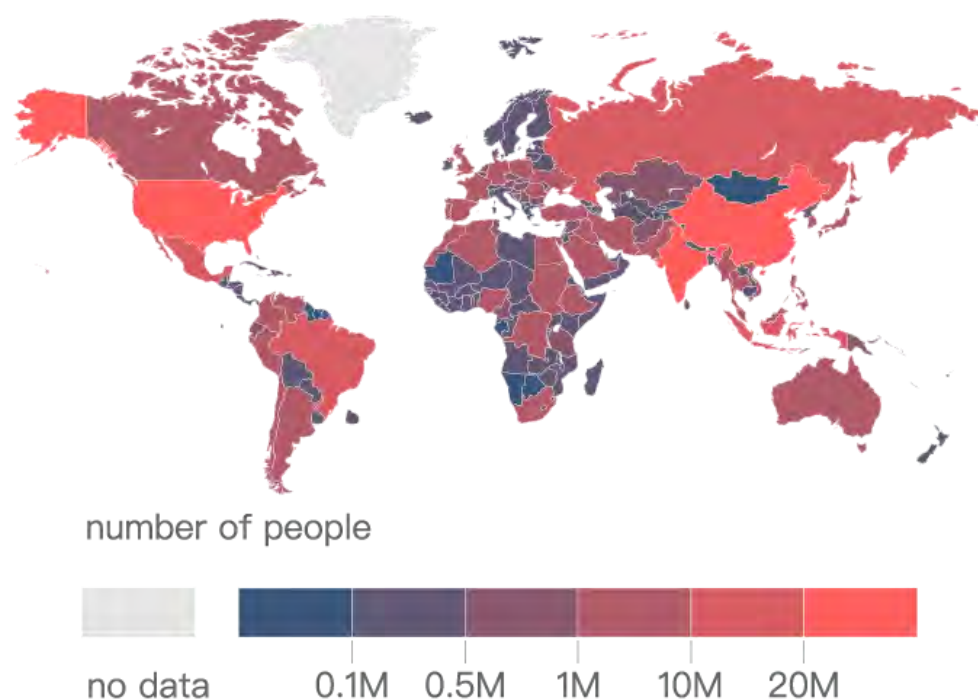
Time of onset

It is more common in middle-aged and elderly people, 35-55 years old is the main age group of onset.

Prevalence

Diabetes has become a global epidemic. There are currently 40 million diabetic patients in China, of which the incidence of type 2 diabetes is about 9.7%.

global distribution of diabetes



Typical symptoms

There is no obvious discomfort in the early stage. As the disease progresses, symptoms of "three more and one less" appear: polydipsia, polyphagia, polyuria, weight loss.

Treatment

Comprehensive treatment for type 2 diabetes includes diet control, exercise therapy, oral hypoglycemic drugs, insulin, blood glucose monitoring, diabetes education, etc.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2334499	intergenic	TT	1.82	0.99
RS5945326	intergenic	AA	1.61	0.98
RS2943641	intergenic	CC	1.62	1.00

RS148316037	intergenic	TT	1.59	1.00
RS9552911	SGCG	AG	1.49	0.98

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of the test is based on published academic literature related to type 2 diabetes. By studying the genotype differences between patients with type 2 diabetes and normal people, these literatures found some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The type 2 diabetes health risk item uses 213 polymorphic loci on genes such as *WFS1*, *MTNR1B*, *TCF7L2*, *SLC30A8*, and *KCNJ11*. The results showed whether the user had a higher risk of type 2 diabetes than all users in the genotype combination of the 213 polymorphic loci tested, which did not represent the user's real risk of type 2 diabetes. The test was based mainly on Caucasian and Asian, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of developing type 2 diabetes.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. this study only predicts your risk of developing type 2 diabetes at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of type 2 diabetes, or if you feel you have a risk of developing type 2 diabetes, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 213 polymorphic loci associated with type 2 diabetes risk, including rs10012946, rs136634, rs10830963, rs3802177, rs117767867.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with type 2 diabetes.

Alopecia Areata

Alopecia areata is a non-scarring inflammatory hair loss disease. Common clinical manifestations are hair loss in a circular plaque with clear boundaries.

My Genetic Risk Is Average For The Population

0.11 times

Average incidence rate is 100 per 100,000 in the population

Suggestions

The most common cause of alopecia areata is mental factors. When stressed out, it is recommended to talk to friends and family, relax through exercise, music, etc., or consult a psychologist.

Influencing factors

Controllable factors

Mental factors: Mental factors are considered to be an important factor. Many cases have mental trauma before the onset, such as long-term anxiety, sadness, mental tension, and emotional upset.

Uncontrollable factors

1. **Family history:** About 10% to 20% of cases have a family history. Studies have found that families with two or more members of alopecia areata has an increased genetic risk.
2. **Immune factors:** It is clinically found that patients with alopecia areata are often accompanied by autoimmune diseases such as rheumatoid arthritis and type 1 diabetes. Therefore, the occurrence of alopecia areata may be closely related to autoimmunity.

Knowledge

Time of onset

Alopecia areata can occur at any age. It is common in young and middle-aged people, and there is no significant sex predominance.

Prevalence

In 2015, statistics showed that the lifetime incidence rate of alopecia areata worldwide was about 2%. In 2009, statistics showed that the prevalence of alopecia areata in China was 0.27%.

Typical symptoms

Round or oval patchy hair loss with clear boundaries appears on the head. In a few severe cases, the hair loss can affect the terminal hair of the entire head (total baldness). Alopecia areata has a serious impact on patients' psychology, work and social activities.

Treatment

There is currently no cure for this disease, and it can be relieved by oral or topical corticosteroids, but the therapeutic effect is difficult to predict.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS3118470	IL2RA	CC	3.18	0.89
RS9275524	intergenic	CT	1.91	0.93
RS9275572	intergenic	AG	1.71	0.95
RS574087	intergenic	AA	1.74	0.99
RS998592	CLEC16A	CC	1.64	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to alopecia areata. By studying the genotype differences between patients with alopecia areata and normal people, these literatures have found some polymorphic sites associated with the risk of alopecia areata. We use the raw data in these literatures to score the polymorphic sites involved in this test

and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of alopecia areata item uses 17 polymorphic loci on genes such as *IL2RA*, *CTLA4*, *ACOXL* and other gene. The results show whether the user had a higher prevalence of alopecia areata than all users in the genotype combination of the 17 polymorphic loci tested, which does not represent the user's real risk of alopecia areata. The test is based mainly on Caucasian studies, and other populations are for informational purposes only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of alopecia areata.
 2. The test results have no clinical significance.
 3. If you have a clinical need, contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of developing alopecia areata at a genetic level and does not represent your true health status or your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of alopecia areata, or if you feel you have a risk of developing alopecia areata, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 17 polymorphic loci associated with the risk of alopecia areata, such as 3118470, rs1024161 and rs9275572.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with alopecia areata.

Gout

Gout is a disease caused by the accumulation of uric acid due to impaired purine metabolism. It is a kind of arthritis, also known as metabolic arthritis.

My Genetic Risk Is Average For The Population

0.10 times

Average incidence rate is 1140 per 100,000 in the population

Suggestions

- Red flags: Uric acid is too high
- Common symptoms: Feeling of foot pain, which usually occurs at night or has lasted for several days to several weeks
- High-risk population: Over 40 years old, obese, male, frequent alcohol consumption, family history of gout
- How to Reduce the risk:
 - Pay attention to diet: Drink more water, drink less alcohol, and eat less **high-purine** foods
 - Weight control: Keep BMI between 18.5-23.9, at least Exercise 4 hours a week
 - Keep warm in winter and avoid direct cold wind in summer
 - Maintain a positive attitude, avoid staying up late and overwork
 - Appropriate intake Vitamin C, coffee and dairy products
- The Heritability of gout is about 17%-53%

* If you have a family history of gout, or if you think you are likely to suffer from gout, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors



Controllable factors

1. Drinking: Alcoholic beverages contain purines, which increase the risk of gout. The purine content of beer and rice wine is much higher than that of other alcoholic beverages, and has a higher risk of disease than other alcoholic beverages.

2. Eating habits: High-purine foods can increase the risk of gout. High-purine foods include animal offal, seafood, fish eggs, and thick soup gravy. Recent studies have pointed out that high sugar (such as carbonated drinks and fructose) can also cause gout. And vitamin C, coffee and dairy products may help prevent the occurrence of gout.
3. Obesity: Obesity can lead to an increase in uric acid concentration and induce gout.
4. Exercise: Exercise can promote the excretion of uric acid, but strenuous exercise will increase the concentration of uric acid and increase the risk of gout.
5. Uric acid concentration: Lack of water in the body can lead to high uric acid concentration and increase the risk of gout.
6. Mental factors: Increased mental stress and fatigue may also lead to endocrine disorders, causing abnormal metabolism of uric acid, increasing the concentration of uric acid, and inducing gout.
7. Low temperature environment: Urate is more likely to precipitate as crystals in a low temperature environment and deposit around joints, causing arthritis. This is one of the reasons why gout tends to attack at night.



Uncontrollable factors

1. Age: Gout usually develops after the age of 40, and as the age increases, the risk of gout increases.
2. Gender: Men usually have a higher incidence than women. Women generally do not have gout before the age of 50, because estrogen has an inhibitory effect on the formation of uric acid. However, the incidence will increase after menopause.
3. Family history: People with a family history of gout have a higher prevalence.
4. Other diseases: Kidney disease or decreased uric acid excretion caused by certain drugs, myeloproliferative diseases and increased uric acid production caused by tumor chemotherapy, etc., may induce gout.

Knowledge



Time of onset

The prevalence of gout increases with age. Clinical gout before puberty is very rare, and men with primary gout risk will show hyperuricemia at this stage. Gout patients are usually older than 40 years old, 95% of them are male, and most of them had family genetic history. Gout in women usually occurs several years after menopause.



Prevalence

The prevalence of gout ranged 1-4% worldwide and incidence ranged 0.1%-0.3%. Men usually have a higher incidence than women.

Typical symptoms

Gout presents many symptoms, the most common of which is the periodic onset of acute irritating arthritis (joint redness, soft, heat, and edema), and the metatarsophalangeal joint at the base of the big toe is most commonly affected, accounting for half of the case. The reason may be that the temperature of toes is lower, and the fluid near the toes will be absorbed by the body and the concentration of uric acid will increase. Other joints, such as ankles, knees, wrists and fingers may also be affected. Joint pain usually occurs at night and lasts for 2-4 hours, which may be caused by a low body temperature. When sleeping at night, the human body's anti-inflammatory substance corticotropin secretes the least in the middle of the night. In addition, the acid-base value of the body tends to be acidic due to the accumulation of carbon dioxide during sleep, so gout is most likely to appear in the middle of the night. Other rare symptoms including fatigue and high fever may also along with joint pain.

Treatment

There is currently no cure for primary gout, but controlling hyperuricemia can reverse the condition. The main goal of treatment is to relieve the symptoms of acute gout.

1. General handling: Protein intake should be limited to about $1\text{g}/(\text{kg} \cdot \text{day})$. Do not eat high-purine foods (animal heart, liver, kidney, sardines, etc.), strictly abstain from alcohol, and avoid predisposing factors. Encourage drinking more water. At the same time, drugs that inhibit uric acid excretion, such as hydrochlorothiazide, furosemide, etc., should not be used.
2. Treatment of acute arthritis: Bed rest should be absolutely necessary, the affected limb should be raised, and the weight of the affected joint should be avoided. It can be gradually moved until about 72h after the joint pain is relieved. Non-steroidal anti-inflammatory analgesics (NSAIDs), colchicine and steroids should be used as soon as possible to control arthritis and relieve symptoms.
3. Intermittent and chronic treatment: Despite the above treatment, the symptoms are still difficult to control and recurring. The author can use small doses of colchicine to maintain the treatment, and pay close attention to the possible inhibitory effect of colchicine on bone marrow during the medication. Regular review of liver and kidney function. Reasonably use drugs that inhibit uric acid synthesis and promote uric acid excretion to control hyperuricemia.
4. Treatment of complicated acute renal failure: Patients with gout caused by uric acid nephropathy should be given acetazolamide immediately, while supplementing enough water intravenously to increase urine output. If blood urea nitrogen and creatinine are significantly elevated, hemodialysis or peritoneal dialysis can be used.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS7927466	CNTN5	TT	3.42	0.99
RS2188380	intergenic	TT	3.06	0.96
RS4073582	CNIH2	GG	2.76	0.99
RS4766566	CUX2	TT	2.28	0.97
RS671	ALDH2	GG	2.16	0.98

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to gout. These literatures have found some polymorphic loci related to the risk of the disease by studying gout patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of gout item uses the 49 polymorphic loci on genes such as *SLC17A3*, *SLC2A9*, *ALDH16A1* and *ABCG2* and within other intergenic regions. The test results indicate whether the user has a higher prevalence of gout in the combination of genotypes at the 49 polymorphic loci tested compared to all users. This test does not represent the user's true risk of gout. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of gout.
2. The test results have no clinical significance.

3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of gout at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of gout, or you think that you are likely to suffer from gout, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 49 polymorphic loci related to the risk of gout, including rs1165205, rs12498742 and rs16890979.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to gout.

Depression

Depression is also called depressive disorder. Clinically, it is mainly characterized by significant persistent feelings of sadness and is the main type of mood disorder.

My Genetic Risk Is Average For The Population

0.10 times

Average incidence rate is 5000 per 100,000 in the population

Suggestions

- Danger signal: If The typical depressive symptoms lasting more than two weeks cannot be relieved, it may be depression.
- Common symptoms: Low mood, Mental retardation, Hypobulia, Cognitive impairment and specific Physical symptoms.
- High-risk population: between the ages of 20 and 30, women, people who have experienced abuse or major physical and mental trauma in childhood, people who live in high latitudes, some diseases and drug users, people who are under high social pressure and have poor psychological adjustment ability.
- How to reduce risk:
 - Pay attention to rest and maintain good exercise habits. Exercise helps to improve brain function. Outdoor exercise increases the sun exposure can also reduce the risk of depression.
 - Protective food: appropriate supplements such as **Omega-3** and **vitamin B12** may help reduce the risk of depression.
 - Actively adjust yourself psychologically, be aware of your own emotions and identify with your own emotions; If there are negative emotions, you can express and release emotions by talking to friends and keeping a diary.
 - Choose a social environment with a high tolerance for work and life, and maintain a harmonious relationship with relatives, friends and colleagues.
- The Heritability of depression is about 40%.
- Major depression has a high risk of suicide, so if someone needs help, call the Toll-free helpline.

*If you have a family history of depression, or if you think you are likely to suffer from depression, or need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors



Controllable factors

1. Psychological factor: individuals cannot find timely and effective adjustment measures for their own worries, anxiety and other negative emotions, which may increase the risk of depression.
2. Social factor: in an environment that lacks tolerance and understanding, the risk of depression is higher.
3. Geographical factor: Areas with cold climate and lack of sunshine have a higher incidence of depression.



Uncontrollable factors

1. Age: this disease that occurs between the ages of 20 and 30 is most common.
2. Gender: the incidence is higher in women than men.
3. Special health condition: ①Patients with other mental problems: patients with alcohol dependence, personality disorder, and attention deficit hyperactivity disorder (ADHD) are more likely to have concurrent depression. ②Patients with pain and cardiovascular disease are at higher risk of depression. ③AIDS, asthma and other diseases are more likely to have secondary depression. ④Postpartum depression may occur due to hormonal changes associated with pregnancy. ⑤Patients with other diseases who are receiving medication may cause depression due to medication. Medications related to depression include interferon therapy, beta-blockers, isotretinoin, contraceptives, and heart medications, anticonvulsants, anti-migraine drugs, antipsychotics, hormones and other gonadotropin releasing hormone agonists.
4. Past experience: people who experienced childhood abuse or major trauma have an increased risk of depression.

Knowledge



Time of onset

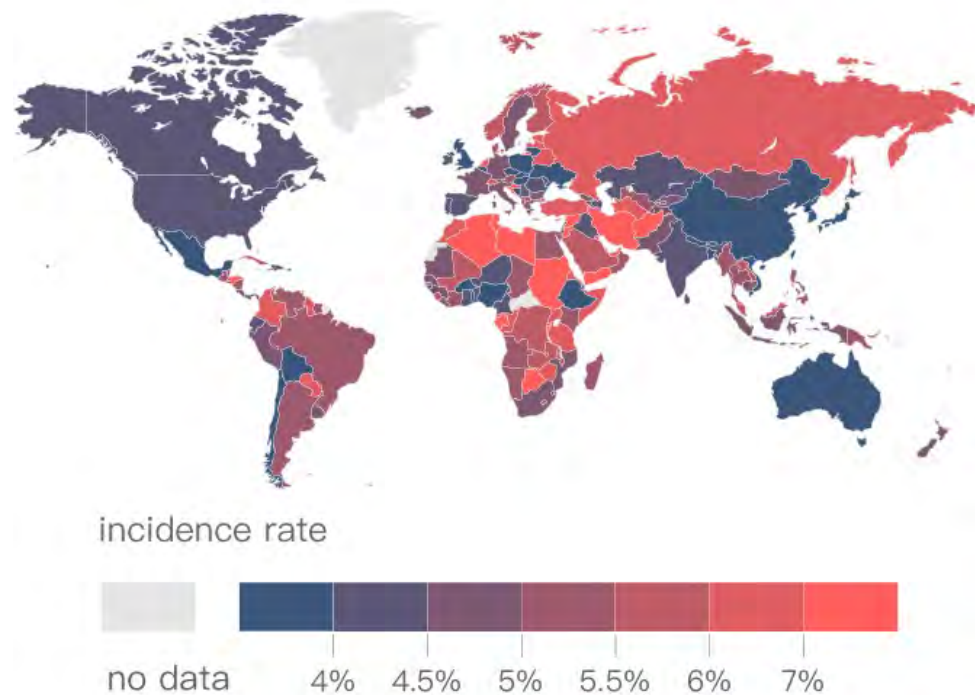
Onset can occur at any age, with most common cases occurring between the ages of 20 and 30.



Prevalence

Incidence: 5000/100,000. The prevalence of depression varies from region to region. The "World Map of Depression" drawn by scholars from the University of Queensland, Australia in 2013 showed that Japan and Southeast Asia, Southern Europe, and Australia, which are sunny and warm in climate, belong to the regions with low incidence of depression. While Northern Europe, Russia, Afghanistan and other regions, which are cold, lack of sunlight, belong to the regions with a high incidence of depression.

global incidence of depression



Typical symptoms

Depression can be shown as a single or repeated multiple depressive episodes. The following are the main manifestations of depressive episodes.

1. Low mood: mainly manifested as significant and lasting emotional depression and pessimism. On the basis of low mood, patients will experience reduced self-evaluation, feelings of uselessness, hopelessness, helplessness and worthlessness, often accompanied by self-blame and guilt. In severe cases, guilt and hypochondriac delusions may occur. Some patients may have hallucinations.
2. Thought retardation: the patient's thinking speed is slow, the reaction is slow, the train of thought is occluded. Clinically, it can be seen that the active speech is reduced, the speed of speech is obviously slowed down, the voice is low, the answer is difficult, and the communication can not be carried out smoothly in serious cases.
3. Hypobulia: the patient's volitional activity showed significant and lasting inhibition. Clinical manifestations of slow behavior, passive life, laziness, do not want to do things, unwilling to interact with people around, often sit alone, or stay in bed all day, live alone behind closed doors, alienate relatives and friends, avoid social interaction.
4. Cognitive impairment: research suggests that patients with depression have cognitive impairment. The main symptoms are: short-term memory, attention disorder, prolonged reaction time, increased alertness, poor abstract thinking ability, learning difficulty, poor language fluency,

spatial perception, eye-hand coordination and thinking flexibility to decline their all abilities. Cognitive impairment leads to social dysfunction and affects long-term prognosis.

5. Physical symptoms: there are sleep disorder, fatigue, anorexia, weight loss, constipation, pain in any part of the body, hypaphrodisia, impotence, amenorrhea and so on.

Treatment

Treatments for depression include medication, psychotherapy, exercise, electroconvulsive therapy (ECT), transcranial magnetic stimulation (TMS), or light therapy. Treatment can be customized based on symptom severity, other complications, the patient's past treatment experience, and patient preferences. Psychotherapy should be used for depression patients under 18 years old.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2267012	BCR	AG	1.62	0.94
RS1545843	intergenic	AA	1.44	0.99
RS1031681	intergenic	TT	1.39	1.00
RS2267015	BCR	TT	1.00	0.91
RS200949	intergenic	AA	1.09	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to depression. These literatures have found some polymorphic loci related to the risk of the disease by studying genotype differences between depression patients and normal controls. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: the risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The depression health risk item uses 126 polymorphic loci in and between genes such as the *BCR* and *FKBP5*. This test result indicates whether the user has a higher risk of depression due to the genotype combination of the 126 polymorphic loci detected compared to all users, but does not represent the user's risk of depression from all dimensions. The basis of the test is mainly from the research of Asian and Caucasian. Other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of depression.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The onset and development of the disease are determined by multiple factors such as genes and environment. This study only predicts whether you are carrying the disease-causing gene or locus at the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of depression, or you think that you are likely to suffer from depression, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 126 polymorphic loci associated with the risk of depression, such as rs1360780, rs2156921 and rs2267012.

Population samples

The basis of the test is mainly from the research of Asian and Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect depression.

Behçet's Syndrome

Also known as behcet disease, it is a chronic, recurrent autoimmune/inflammatory disease with pathological changes based on vasculitis.

My Genetic Risk Is Average For The Population

0.09 times

Average incidence rate is 14 per 100,000 in the population

Suggestions

Pay attention to cleanliness and hygiene to prevent infection by various germs.

Influencing factors



Controllable factors

Infection: studies have found that pathogenic microbial infections such as streptococcus, staphylococcus, and herpes simplex virus are closely related to behçet's syndrome.



Uncontrollable factors

1. Geography: there are significant regional differences in the prevalence rate worldwide, with higher rates in the Middle East, Far East, and Mediterranean regions, hence it is known as the "Silk Road disease".
2. Race: the prevalence rate of Western Caucasians is lower than that of Middle Eastern and Yellow people; The prevalence of Türkiye in Iran is significantly higher than that of other ethnic groups in the country.
3. Immunity: the patient's body produces antibodies targeting the oral mucosa and some blood vessel walls, causing lesions in the oral mucosa and blood vessel walls.
4. Genetics: the increased risk of behçet's syndrome is associated with carrying certain *HLA* genotypes, especially HLA-B * 51.

Knowledge

Time of onset

The age of onset was mostly 15-50 years, and the median age of onset was 34 years.

Prevalence

Behçet syndrome is more common along the ancient silk road, which extends from eastern Asia to the Mediterranean. It is most common in Turkey (80 to 370 cases per 100,000), while the prevalence ranges from 13.5 to 35 per 100,000 in Japan, Korea, China, Iran, Iraq, and Saudi Arabia. The combined global prevalence was 10.3/100,000 individuals.

Typical symptoms

1. Mouth ulcer: it is usually the first symptom and the most common symptom, with an incidence of more than 95%, it can occur in any part of the mouth, such as the tongue, buccal, lip, gingiva, pharynx, hard palate, etc., it can appear at multiple sites simultaneously, pain is intense, and recurrent.
2. Genital ulcers: patients of both sexes may present with ulcers in the genital area, but rarely as the first manifestation, with an incidence of 51.7% - 93%. genital ulcers occur less frequently and in fewer numbers than oral ulcers, but usually the wound is deeper and larger with intense pain, and scars are often retained after ulcers heal.
3. Ocular impairment: the eye is also the most commonly affected organ, which can be found in 26.8% - 93% of patients, and about 15% of patients show ocular lesions as the first manifestation, with symptoms such as redness, pain, photophobia or decreased visual acuity and unclear vision, which can lead to blindness if left untreated.
4. Skin lesions: about 39.4% - 87.1% of patients can present with skin lesions such as pseudofolliculitis, erythema nodosum, pyoderma gangrenosum, thrombophlebitis, etc.
5. Joint damage: 5.3% - 93% of patients will experience joint pain or swelling, which can appear in single or multiple joints, is more common in the joints of the lower extremities, is often accompanied by red, swelling, heat, pain, and in severe cases, joint effusion, synovitis.
6. Gastrointestinal damage: the incidence is 4% - 38%, and all the digestive tract from the esophagus to the anus can be damaged, and the common symptoms are abdominal pain, abdominal mass, diarrhea, bloating, dysphagia, eructation, vomiting, hematochezia, constipation, etc., retrosternal pain can occur when ulcers involve the esophagus, and severe patients may experience gastrointestinal ulceration, bleeding, intestinal perforation, intestinal obstruction, etc.
7. Vascular injury: 2.2% - 50% of patients can have vascular injury, with a male predominance. Vascular involvement is one of the leading causes of death in this disease, with venous involvement more common compared with arterial, including thrombotic superficial phlebitis

and deep vein thrombosis, which can also be complicated by pulmonary embolism in severe cases. There are also patients who can present with aneurysms that cause local embolization, ischemia, and, after rupture of the aneurysm, can cause major bleeding and be life-threatening.

8. Neurological impairment: neurological involvement is one of the most serious complications, with an incidence of 2.3% - 44%, occurring mostly in those aged 30-40 years. Manifesting as headache, cranial nerve palsy, inability to pronounce normally, ataxia, and hemiplegia, it is a major disabling, lethal cause.
9. Other: the disease may also involve the heart, lungs, kidneys, blood system and may cause myocardial infarction, pulmonary thrombosis, nephrotic syndrome, aplastic anemia and other diseases.

Treatment

At present, there is no recognized effective radical drug for this disease, and the main treatment goals are to rapidly inhibit inflammation, prevent recurrence, prevent irreversible organ damage, and delay disease progression. Treatment of behçet’ s syndrome requires close cooperation from multiple disciplines such as internal medicine, ophthalmology and stomatology, different conditions have their focus again, treatment options need to be highly individualized and often topical treatment is combined with systemic medications.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS41546114	MICA	CT	6.51	0.77
RS9266406	intergenic	AG	2.80	0.77
RS2245961	HCG9	CC	0.27	0.90
RS6910516	intergenic	AG	2.64	0.79
RS6920323	intergenic	CT	2.68	0.84

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to behçet' s syndrome. These literatures have found some polymorphic loci related to the risk of the disease by studying behçet' s syndrome patients and normal people. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of behçet' s syndrome item uses 37 polymorphic loci of *IL10*, *FUT2*, *TFCP2L1*, etc. genes. The test results indicate whether the user has a higher prevalence of behçet' s syndrome in the combination of genotypes at the 37 polymorphic loci tested compared to all users. This test does not represent the user' s true risk of behçet' s syndrome. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of behçet' s Syndrome.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of behçet' s syndrome at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of behçet' s syndrome, or you think that you are likely to suffer from behçet' st syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 37 polymorphic loci associated with the risk of behçet' s syndrome, including rs1800871, rs1518111 and rs1495965.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to behçet' s syndrome.

Coronary Heart Disease (CHD)

It is a heart disease caused by atherosclerotic lesions of coronary arteries that cause vascular lumen stenosis or obstruction, resulting in myocardial ischemia, hypoxia, or necrosis.

My Genetic Risk Is Average For The Population

0.09 times

Average incidence rate is 1020 per 100,000 in the population

Suggestions

- Red flags: abnormal coronary angiography, serum troponin, electrocardiogram, etc.
- Common symptoms: Chest discomfort or pain, which can spread to the shoulders, back, jaw, abdomen, etc.
- High-risk population: smoking, high blood pressure, high cholesterol, diabetic patients, obesity, drinking, family history of coronary heart disease
- How to reduce the risk:
 - Quit smoking: refuse to take the initiative to smoke, stay away from second-hand smoke
 - Weight control: Keep BMI between 18.5 and 23.9, avoid sitting for a long time, and exercise at least 4 hours a week
 - Pay attention to your diet: drink less alcohol and eat less high-fat, high-calorie, high-salt foods
 - Pay attention to chronic diseases: control high blood pressure, high cholesterol, high blood sugar/diabetes and other chronic diseases
 - Protective food: Consider eating protective foods or supplements such as **garlic, coenzyme, Omega-3** fatty acids, etc.
- The Heritability of coronary heart disease is about 40%~60%

* If you have a family history of coronary heart disease, or if you think you have the possibility of suffering from coronary heart disease, or need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors



Controllable factors

1. Obese, overweight people have a high risk of disease.

2. Dietary factors: unhealthy diets such as high fat, high cholesterol, and high calories can easily lead to coronary heart disease.
3. Unhealthy living habits: such as lack of physical activity, excessive drinking, etc. can also induce this disease.

Uncontrollable factors

1. Genetic factors: there is a family genetic tendency.
2. Disease factors: high blood pressure, high blood lipids, high blood sugar and diabetes patients with high incidence of coronary heart disease.

Knowledge

Time of onset

It often occurs in people over the age of 35. In developed countries, its annual deaths can account for 1/3 of the total deaths in middle-aged and elderly people.

Prevalence

According to statistics, there are about 1020 cases of the disease per 100,000 people in the average population.

Typical symptoms

The typical chest pain is caused by physical activity, emotional agitation, etc., sudden feeling of pain in the precordial area, mostly paroxysmal colic or squeezing pain, or a feeling of depression. The pain starts from the back of the breastbone or the front of the heart, and radiates upward to the left shoulder, arm, and even the little finger and ring finger. The part where chest pain disperses can also involve the neck, jaw, teeth, abdomen, etc. In addition, symptoms of gastrointestinal diseases such as upper abdominal pain, nausea, and vomiting may also occur.

Treatment

Although coronary heart disease, especially myocardial infarction, is very dangerous, the first thing to do is to solve the stenosis and infarction and restore myocardial blood supply as soon as possible, so as to quickly control the symptoms and minimize the scope and extent of myocardial necrosis. Drug treatment includes sublingual administration of nitroglycerin and other nitrate drugs, metoprolol and other β -receptor blockers, and nifedipine calcium channel blockers. Surgical procedures include heart bypass.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS6903956	ADTRP	AG	1.44	0.97
RS4766566	CUX2	TT	0.72	0.99
RS9349379	PHACTR1	GG	1.30	1.00
RS56289821	intergenic	GG	1.30	1.00
RS6511720	LDLR	GG	1.29	1.00

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to coronary heart disease. These literatures have found some polymorphic loci related to the risk of the disease by studying patients with coronary heart disease and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of coronary heart disease item uses the 183 polymorphic loci in and between genes of *BRAP*, *SNX19*, *ADTRP*, *PALLD*, *PON1*. The test results indicate whether the user has a higher prevalence of coronary heart disease in the combination of genotypes at the 183 polymorphic loci tested compared to all users. This test does not represent the user's true risk of coronary heart disease. The basis of the test is mainly from the research of Caucasian and Asian, other populations are for reference only.

Notification

-
1. The test may not cover all genes or loci that could contribute to a risk of coronary heart disease.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of coronary heart disease at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of coronary heart disease, or you think that you are likely to suffer from coronary heart disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 183 polymorphic loci related to the risk of coronary heart disease, including rs3782886, rs229856, rs6903956, rs7439293, rs662.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to coronary heart disease.

Schizophrenia

Schizophrenia is a chronic persistent disease with a high rate of psychiatric relapse and high morbidity, which usually onsets in young adulthood, generally manifested as sensory disorders and abnormal social behavior.

My Genetic Risk Is Average For The Population

0.07 times

Average incidence rate is 10 per 100,000 in the population

Suggestions

1. Limit alcohol intake. The limit value for moderate drinking recommended by the Chinese Nutrition Society is no more than 25g of alcohol a day for adult men. Adult women drink no more than 15g of alcohol a day.
2. Avoid exposure to marijuana, cocaine, amphetamines, etc.
3. Choose a social environment with high tolerance and low life pressure to work and live, and maintain a harmonious relationship with friends, relatives and colleagues. Improve their ability to cope with psychological pressure and burden of life, so as not to induce disease.

Influencing factors



Controllable factors

1. Alcohol abuse: alcohol may increase the risk of schizophrenia by inducing mechanisms that cause chronic mental disorders caused by active substances.
2. Marijuana and drugs: Marijuana may be one of the predisposing factors of schizophrenia, which may cause high-risk individuals to develop schizophrenia. Amphetamines and cocaine can cause temporary irritant psychosis.
3. Social environment: studies have found that people who grow up or live in cities are at higher risk of developing schizophrenia. The prevalence of schizophrenia is also closely related to social class. The study found that the prevalence of schizophrenia is highest among people of lower

economic level, and this correlation is more obvious in cities. Other important factors include social isolation, migration related to social adversity, racial discrimination, family difficulties, unemployment, and poor housing conditions.

Uncontrollable factors

1. Gender: the incidence rate of men is about 1.4 times higher than that of women, and men usually get onset earlier.
2. Age: the high period incidence period of schizophrenia is young and middle-aged.
3. Family history: patients with acute and transient psychotic disorder who have a family history of schizophrenia have a 20% to 40% chance of being diagnosed with schizophrenia one year later.
4. Fetal developmental factor: exposure to hypoxia during fetal development may increase the risk of schizophrenia. If the fetus is infected by pathogens such as toxoplasma during pregnancy or at birth, the chance of getting the disease will increase in the future.
5. Childhood experience: people who experience trauma as children are at increased risk for disease later in life.

Knowledge

Time of onset

The onset of schizophrenia in young adults, late adolescence to early adulthood is the peak of the onset of schizophrenia. 40% of men and 23% of women diagnosed with schizophrenia show symptoms of the disease before the age of 19.

Prevalence

Incidence: 10/100,000. Approximately 0.3% to 0.7% of the world's population will experience schizophrenia during their lifetime. In 2016, there were estimated to be more than 21 million people living with schizophrenia worldwide.

Typical symptoms

The clinical symptoms of schizophrenia are complex and varied, which can involve sensory perception, thinking, emotion, volitional behavior and cognitive function. The symptoms vary greatly between individuals, even if the same patient may show different symptoms at different stages of the disease.

1. Sensory impairment: schizophrenia can have a variety of sensory disturbances. The most prominent sensory barrier is hallucinations, including acousma, heteroptics, pseudosmia, pseudogeusia and tactile hallucination, among which acousma are the most common.

2. **Thought disorder:** thought disorder is the core symptom of schizophrenia, mainly including formal thought disorder and content-thought disorder. Formal thought disorder is mainly manifested as disorder of thinking association process, including the disorder of thinking association activity process (quantity, speed and form), the coherence and logic. Delusion is the most common and important content-thought disorder. The most common delusions are delusion of persecution, delusion of reference, delusion of physical influence, delusions of jealousy, delusion of grandeur, delusion of non-consanguinity, etc. It is estimated that up to 80% of schizophrenia patients have delusions of persecution, which can be expressed as varying degrees of insecurity, such as being monitored, rejected, the fear of being drugged or murdered. Under the influence of delusions, patients will behave defensively or aggressively. In addition, passive experience is also prominent in some patients, affecting their thinking, emotion and behavior.
3. **Affective disorder:** apathy and uncoordinated emotional responses are the most common emotional symptoms of schizophrenics. In addition, emotional symptoms such as incongruous excitement, irritability, depression and anxiety are also more common.
4. **Will and behavior disorders:** most patients have decreased or even lack of will, manifested by reduced activities, solitude, passive behavior, lack of enthusiasm and initiative, decreased interest in work and study, not caring about the future, and no clear plan for the future. Some patients may have plans and intentions, but rarely carry them out.
5. **Cognition impairment:** the incidence of cognitive deficits in patients with schizophrenia is high. About 85% of patients suffer from cognitive dysfunction, such as information processing and selective attention, working memory, short-term memory and learning, executive function and other cognitive deficits. There is a certain correlation between the symptoms of cognitive deficits and other psychotic symptoms. For example, patients with obvious mental disorder have more obvious symptoms of cognitive deficits, and patients with obvious negative symptoms have more obvious symptoms of cognitive deficits. Cognitive deficits may be related to certain positive symptoms. Cognitive deficits may occur before the psychiatric symptoms become clear (such as the prodromal phase), it is preliminarily believed that chronic schizophrenia patients than the first-episode schizophrenia patients with cognitive deficits more obvious, or decline sharply with the onset of psychotic symptoms, or with the prolongation of the course of disease and gradually decline.

Treatment

1. **Pharmacotherapy:** first-line treatment for schizophrenia is the prescription of antipsychotic drugs, which can reduce the severity of acute symptoms in about 7 to 14 days. However, antipsychotic medications do not improve chronic symptoms and cognitive dysfunction significantly. If a patient continues to take medication, the risk of relapse can be reduced.
2. **Social psychotherapy:** many psychosocial interventions may help treat schizophrenia, including family therapy, positive community therapy, employment assistance, cognitive correction

therapy, skills training, token therapy, and psychosocial interventions for substance use and weight management.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2270954	DCC	AC	0.19	0.91
RS11246226	DRD4	AC	2.65	0.86
RS2283123	DBH	CT	0.30	0.88
RS1522305	PAH	GG	2.69	0.98
RS6277	DRD2	GG	2.18	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to schizophrenia. These literatures have found some polymorphic loci related to the risk of the disease by studying genotype differences between schizophrenic patients and normal controls. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: the risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

 Applicable situation

The schizophrenia health risk item uses 300 polymorphic loci in genes such as *DRD2*, *SLC6A3* and *ZNF804A*. This test result indicates whether the user has a higher risk of schizophrenia due to the genotype combination of the 300 polymorphic loci detected compared to all users, but does not represent the user's risk of depression from all dimensions. The basis of the test is mainly from the research of Caucasian and Asian. Other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of schizophrenia.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The onset and development of the disease are determined by multiple factors such as genes and environment. This study only predicts whether you are carrying the disease-causing gene or locus at the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of schizophrenia, or you think that you are likely to suffer from schizophrenia, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 300 polymorphic loci associated with the risk of schizophrenia, such as rs6277, rs1801028 and rs3131296.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect schizophrenia.

Age-related Macular Degeneration (AMD)

Age-related macular degeneration is characterized by senile changes in the central area of the retina (macula). It mainly begins around age 50 and the prevalence increases with age.

My Genetic Risk Is Average For The Population

0.01 times

Average incidence rate is 1500 per 100,000 in the population

Suggestions

- Red flags: sudden changes in vision
- Common symptoms: vision loss, blurred vision, visual impairment in the central field of vision.
- High-risk population: Most of them occur over the age of 45.
- How to reduce the risk:
 - Reduce intake of high-fat, high-cholesterol foods, and eat more eye-care foods such as Green leafy vegetables and Blueberries.
 - Smoking is an important factor in the development of age-related macular degeneration, so it is recommended to quit smoking.
 - Workers in strong light environment recommend wearing dark glasses to reduce Light damage.
 - The main components of the macula are vitamins A, C, E, and the mineral element zinc. These antioxidant nutrients protect retinal cells.
 - Appropriate intake of macular pigment Lutein and **Vitamin B12**, is suitable for the elderly to prevent visual deterioration and macular degeneration
- Age-related macular degeneration Heritability is approximately 23%

* If your family has a family history of Age-related macular degeneration, or you feel you have a risk of developing age-related macular degeneration, it is recommended that you consult an authoritative health professional or clinician for help.

Influencing factors



Controllable factors

1. Diet: High-fat and high-cholesterol diet can increase the incidence of age-related macular degeneration.

- 2. Smoking: Smoking is an important factor in inducing age-related macular degeneration. Studies have found that smokers are 2.8 times more likely to suffer from wet age-related macular degeneration.
- 3. Light damage: Long-term visible light (such as blue light) can damage the retinal pigment epithelium and induce this disease.

 Uncontrollable factors

- 1. Age: The prevalence of age-related macular degeneration increases with age.
- 2. Family history: There may be genetic factors in the pathogenesis of age-related macular degeneration.

Knowledge

 Time of onset

It mostly occurs over 45 years of age.

 Prevalence

The average population incidence rate is 1,500 per 100,000 people.

 Typical symptoms

There is vision loss, blurred vision, and visual disturbance in the central visual field, which may affect one or both eyes.

 Treatment

Oral medication, laser or surgical treatment.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS3750846	ARMS2	CT	2.81	0.78
RS3750848	ARMS2	GT	2.76	0.78
RS10490924	ARMS2	GT	2.76	0.78

RS3793917	intergenic	CG	2.67	0.79
RS1061147	CFH	AC	2.53	0.94

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to age related macular degeneration. These literatures have found some polymorphic loci related to the risk of the disease by studying age related macular degeneration. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of age related macular degeneration item uses the 56 polymorphic loci of genes, for example, *CFH*, *CST3*, *ARMS2*, *HTRA1*, *SERPINF1*. The test results indicate whether the user has a higher prevalence of age related macular degeneration in the combination of genotypes at the 56 polymorphic loci tested compared to all users. It does not mean the user's true risk of age related macular degeneration. The basis of the test is mainly from the research of Caucasian and few Asian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that can lead to the risk of age related macular degeneration.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only predicts your risk of age related macular degeneration from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of age related macular degeneration, or you think that you are likely to suffer from age related macular degeneration, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 56 polymorphic loci related to age related macular degeneration risk, such as rs1064039, rs10490924, rs11200638, rs2511989 and rs1136287.

Population samples

The basis of the test is mainly from the research of Caucasian and few Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that associated with age related macular degeneration.

Nasopharyngeal Carcinoma

This is a malignant tumor that occurs in the top and side walls of the nasopharynx.

My Genetic Risk Is Average For The Population

0.01 times

Average incidence rate is 3 per 100,000 in the population

Suggestions

1. Nitrites-rich food increase the risk of nasopharyngeal carcinoma, and it is recommended to avoid salted fish, pickles, bacon, cured meats and other preserved foods.
2. Smoking can induce nasopharyngeal carcinoma, so you should quit smoking and avoid secondhand smoke.
3. Actively treat inflammatory diseases in the nasal cavity and nasopharynx.

Influencing factors

Controllable factors

1. Diet: Food with high level of nitrites, such as salted fish and bacon, may increase the risk of nasopharyngeal carcinoma.
2. Smoking: Smoking is one of the predisposing factors of nasopharyngeal carcinoma.

Uncontrollable factors

1. History of rhinitis: People with a history of sinusitis have a higher risk of nasopharyngeal carcinoma than the general population.
2. Viral infection: Epstein-Barr virus (EBV, also called human herpesvirus 4) infection is closely related to nasopharyngeal carcinoma, and the detection rate of EB virus is higher in patients with this disease.
3. Gender: The incidence is higher in male compared to female.

4. Age: Nasopharyngeal carcinoma is common in 40-50 years old.
5. Family history: This disease has family aggregation, many patients with nasopharyngeal carcinoma have a family history, especially their immediate relatives.
6. Race: Nasopharyngeal carcinoma has a racial predisposition, which is mainly seen in the yellow race, but rare in European and American Caucasians. Ethnic groups with a high incidence migrate elsewhere (or expatriate abroad), and their descendants still have a higher incidence.

Knowledge

Time of onset

The age of onset started from more than 20 years old and gradually increased, with the peak at 40-50 years old.

Prevalence

Nasopharyngeal carcinoma occurs in all countries in the world, but there is an obvious geographical high incidence. The incidence in China and Southeast Asian countries is high, followed by North Africa, and less than 1/100000 in Europe, America and Oceania. The incidence of male is about 2-3 times that of female.

Typical symptoms

- Nasal symptoms: Stuffy nose, bloody nose, etc.
- Ear symptoms: Tinnitus, ear congestion, hearing loss, etc.
- It may also be accompanied by diplopia, headache and other symptoms.

Treatment

Nasopharyngeal carcinoma is mostly sensitive to radiotherapy, so radiotherapy is the preferred treatment. However, surgery and chemotherapy are also indispensable means for the cases of highly differentiated cancer, late course of disease and recurrence after radiotherapy.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS2517713	intergenic	GG	0.30	0.92

RS2975042	intergenic	GG	0.31	0.93
RS5009448	intergenic	TT	0.37	0.95
RS3131875	intergenic	CT	1.97	0.89
RS9260734	intergenic	AG	0.57	0.94

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to nasopharyngeal carcinoma. These literatures found some polymorphic loci associated with the risk of the disease by studying the genetic differences between patients with nasopharyngeal carcinoma and normal people. We uses the raw data in these literatures to score the polymorphic loci involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risk of nasopharyngeal carcinoma item the 21 polymorphic loci on genes such as *TNFRSF19*, *CLPTM1L*, *GABBR1*, *HLA-DRA* and *CDKN2B-AS1* and within other intergenic regions. The results show whether the user had a higher incidence of nasopharyngeal carcinoma than the genotype combination of the 21 polymorphic loci tested compared to all users, and that the test did not represent the user's real risk of nasopharyngeal carcinoma. The test is based mainly on Chinese, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of nasopharyngeal carcinoma.
2. The test results have no clinical significance.

3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. this study only predicts your risk of nasopharyngeal carcinoma at a genetic level and does not represent your true health status, nor does it involve your judgment of other health conditions, lifestyle habits, etc.
3. If you have a family history of nasopharyngeal carcinoma, or if you feel you have a risk of nasopharyngeal carcinoma, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 21 polymorphic loci related to the risk of nasopharyngeal carcinoma, including rs29232, rs9510787, rs31489, rs9268644 and rs1412829.

Population samples

The basis of the test is mainly from the research of Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci associated with nasopharyngeal carcinoma.

Type 1 Diabetes (T1D)

Type 1 diabetes, formerly known as insulin-dependent diabetes mellitus, mostly occurs in children and adolescents, but can also occur at any age.

My Genetic Risk Is Average For The Population

0.01 times

Average incidence rate is 1 per 100,000 in the population

Suggestions

1. Virus infection can induce type 1 diabetes, it is recommended to get vaccinated in time. Keep children warm in cold climate areas to reduce the chance of virus invasion.
2. Some certain diets may induce type 1 diabetes. It is recommended to eat less grain protein (mainly found in wheat, barley, oats, rye and other grains) in early infants.

Influencing factors



Controllable factors

1. Viral infection: Type 1 diabetes is often associated with certain infections or occurs after infection. Common infections include mumps virus, rubella virus, measles virus, influenza virus, cytomegalovirus, polio virus, Coxsackie virus, etc.
2. Diet: Dietary components (such as milk, gluten, etc.) may cause disorders of the autoimmune system and induce type 1 diabetes.



Uncontrollable factors

1. Family history: The family members of patients with type 1 diabetes have a higher risk of diabetes than the general population.
2. Age: mostly occurs in children and adolescents.
3. Region: The incidence varies from country to region. The countries with the lowest annual incidence rate are Japan and China, and the highest annual incidence rate is Finland.

Knowledge

Time of onset

Most of the onset occurs in childhood and adolescence.

Prevalence

The incidence of type 1 diabetes varies significantly in different countries and regions. The incidence rate is less than 1/100,000 in Japan, 0.9/100,000 in China, 8-17/100,000 in the United States, and 28.6/100,000 in Finland.

Typical symptoms

Symptoms: polydipsia, polyuria, polyphagia, and weight loss (thin).

Treatment

Comprehensive treatment: including diet control, exercise therapy, insulin therapy and blood sugar testing.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS5498	ICAM1	AA	2.46	0.98
RS725613	CLEC16A	TT	2.22	0.97
RS1800783	NOS3	TT	1.54	1.00
RS9273363	HLA-DQB1	CC	1.00	0.67
RS9272346	HLA-DQA1	GG	1.00	0.70

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to type 1 diabetes. These literatures have found some polymorphic loci related to the risk of the disease by studying type 1 diabetes patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of type 1 diabetes item uses the 10 polymorphic loci in *HLA-DQA1*, *HLA-DQB1*, *NOS3*, *TLR2*, and *ICAM1*. The test results indicate whether the user has a higher prevalence of type 1 diabetes in the combination of genotypes at the 10 polymorphic loci tested compared to all users. This test does not represent the user's true risk of type 1 diabetes. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of type 1 diabetes.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of type 1 diabetes at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of type 1 diabetes, or you think that you are likely to suffer from type 1 diabetes, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 10 polymorphic loci associated with the risk of type 1 diabetes, including rs9272346, rs9273363, rs7255613, rs7574865, and rs3804100.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to type 1 diabetes.

Psoriasis

Psoriasis is a common chronic and recurring inflammatory skin disease.

My Genetic Risk Is Average For The Population

0.01 times

Average incidence rate is 123 per 100,000 in the population

Suggestions

1. Bacterial infection is related to the onset of psoriasis, so the infected wound and inflammation should be actively treated to avoid all kinds of trauma.
 2. Mental stress may induce psoriasis, so when the pressure is too high, it is recommended to talk to friends and family, relax through exercise, music, etc., or consult a psychologist.
 3. Heavy drinking may induce or aggravate psoriasis. It is recommended to abstain from alcohol.
 4. Smoking is not conducive to the recovery of patients with psoriasis. It is recommended to quit smoking and avoid second-hand smoke.
-

Influencing factors



Controllable factors

1. Infections: bacterial infections such as streptococcus and staphylococcus aureus, is related to the onset and prolonged course of psoriasis.
2. Mental factors: stress, excessive stress may induce psoriasis.
3. Drinking alcohol: heavy drinking may induce or aggravate psoriasis.
4. Smoking: smoking is harmful to human health and is not conducive to the rehabilitation of psoriasis patients.



Uncontrollable factors

1. Family history: Psoriasis has an obvious genetic predisposition.

2. Season: The symptoms of psoriasis tend to get worse in winter.

Knowledge

 Time of onset

The disease can occur at any age, but mainly in young and middle-aged.

 Prevalence

The prevalence in Europe and the United States is 1 to 2%, and the prevalence in China is lower at 0.123%.

 Typical symptoms

- 1. Ordinary type: There are clear borders and erythema of different sizes on the skin, and the surface is covered with silvery white scales. Gently scrape off the surface scales to reveal a light red and shiny translucent film, and then scrape the film to see bleeding. Spots, accompanied by varying degrees of itching.
- 2. Pustular type: characterized by aseptic pustules.
- 3. Arthropathy: In addition to erythema and scales, there are also manifestations of arthritis.
- 4. Erythroderma: manifested as diffuse flushing, swelling and desquamation of the skin all over the body.

 Treatment

Currently, there is no radical cure. Treatment measures include oral medication, topical therapy and photochemotherapy.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1265181	intergenic	GG	1.00	0.37
RS10832027	ARNTL	GG	1.99	0.97
RS3212227	IL12B	GT	1.62	0.98

RS10484554	intergenic	CC	1.00	0.70
RS6887695	intergenic	CG	0.70	0.99

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to psoriasis. These literatures have found some polymorphic loci related to the risk of the disease by studying psoriasis patients and normal people. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk multiple relative to the population average, and get the population risk distribution. Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The health risks of psoriasis item uses the 29 polymorphic loci in and between genes of *IL12B*, *ERAP1*, *HCP5*. The test results indicate whether the user has a higher prevalence of psoriasis in the combination of genotypes at the 29 polymorphic loci tested compared to all users. This test does not represent the user's true risk of psoriasis. The basis of the test is mainly from the research of Caucasian, Chinese and Egyptian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to a risk of psoriasis.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

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1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of psoriasis at a genetic level, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
 3. If you have a family history of psoriasis, or you think that you are likely to suffer from psoriasis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 29 polymorphic loci related to the risk of psoriasis, including rs3212227, rs2395029, rs1576 and rs27044.

Population samples

The basis of the test is mainly from the research of Caucasian, Chinese and Egyptian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to psoriasis.

Rheumatoid Arthritis (RA)

It is a long-term chronic inflammation caused by autoimmune disorders that causes the immune system to attack the joints. This inflammation can cause joint deformation and disability, and partial loss of mobility due to joint pain and wear.

My Genetic Risk Is Average For The Population

0.01 times

Average incidence rate is 300 per 100,000 in the population

Suggestions

Reject active and passive smoking, and avoid exposure to tobacco.

Influencing factors



Controllable factors

Smoking: studies have found that smokers have four times the incidence of rheumatoid arthritis than nonsmokers.



Uncontrollable factors

1. Gender: the incidence rate among female is 2 to 4 times higher than male.
 2. Age: the disease is most common in people aged 35 to 50 years, the incidence rate generally increases with age.
 3. Family history: people with a family history of rheumatoid arthritis have a higher prevalence. The prevalence rate of first-degree relatives in patients with rheumatoid arthritis was 2 to 3%, while the prevalence of identical twins was 15 to 20%.
 4. Infection: studies have found that EB virus infection can increase the incidence of rheumatoid arthritis.
-

Knowledge

Time of onset

The disease can occur at any age, and is more common in people aged 35 to 50. The incidence generally increases with age.

Prevalence

The incidence of rheumatoid arthritis is 300/100,000. Rheumatoid arthritis is a common disease distributed in all races and ethnic groups. The patients are spread all over the world. It is more common in temperate, subtropical and frigid regions, but rare in tropical regions. Among them, the incidence of women is 2 to 4 times than men, and the incidence of smokers is 4 times than non-smokers.

Typical symptoms

1. Pain: There is local joint pain in the early stage of the disease, especially in the active phase, accompanied by tenderness and tenderness. This is the earliest and most sensitive sign of the patient.
2. Stiffness: Stiffness of the affected joints, especially when starting activities in the morning, but will gradually improve after a period of activity.
3. Swelling: The soft tissues around the affected joints are diffusely swollen, and the surface temperature is slightly higher than that of normal joints.
4. Deformity: Metacarpophalangeal joint flexion and ulnar deformity are generally present in later cases; if it occurs on the toes, it will show the appearance of claw-like toe deformity.
5. Subcutaneous nodules: 30%-40% of patients may have subcutaneous nodules.
6. Elevated body temperature: some patients in the acute phase may have fever, mostly low-grade fever below 38°C.

Treatment

1. Rest: Especially when the disease is in the acute phase, the patient should rest completely to relieve pain; in the non-acute phase, it is not recommended that the patient be excessively active and do strenuous exercise.
2. Physiotherapy: During the recovery period, you can choose effective physiotherapy as appropriate to help joint movement and improve the inflammatory response of diseased joints, while also preventing excessive loss of functionality.
3. Drugs: mainly include nonsteroidal antiinflammatory drugs, slow-acting anti-rheumatic drugs, glucocorticoids, botanical preparations, etc.
4. Surgical treatment: The deformity caused by rheumatoid lesions can be treated with surgery in the stationary phase. Commonly used surgical methods include synovectomy, arthroscopic

synovectomy, arthroplasty, and artificial joint replacement.

My Genetic Result Details

● Low Risk ● Average Risk ● High Risk

Gene locus	Gene name	My genotype	My risk	Average
RS1830035	ZNF679	TT	9.57	0.99
RS2493291	PRDM16	CC	8.61	0.99
RS6018432	intergenic	CC	7.51	0.99
RS4918037	SH3PXD2A	GG	6.86	1.00
RS9378212	intergenic	CC	0.13	0.77

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literature related to rheumatoid arthritis. By studying the genetic differences between rheumatoid arthritis patients and normal people, these literatures have identified some polymorphic sites associated with the risk of the disease. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the user's risk multiple relative to the population average and get a distribution of the risk of the population.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The rheumatoid arthritis health risk item uses 115 polymorphic loci on the *AIF1*, *STAT4*, *PHF19*, *MMEL1* and *TRAF1* genes. The results showed whether the user had a higher prevalence of

rheumatoid arthritis than all users in the genotype combination of the 115 polymorphic loci tested, and that the test did not represent the user's real risk of rheumatoid arthritis. The basis of the test is mainly from the research of Caucasian, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to the risk of rheumatoid arthritis.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of rheumatoid arthritis at a genetic level and does not represent your true health status, nor does it involve your judgment of other health conditions, lifestyles, etc.
3. If you have a family history of rheumatoid arthritis, or if you feel you have a risk of developing rheumatoid arthritis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 115 polymorphic loci related to the risk of rheumatoid arthritis, including rs2837960, rs660895 and rs2269475.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with rheumatoid arthritis.



Psychological Traits

26 Reports



Personality Characteristics

Emotional Stability

Strong



Extraversion

Weak



Agreeableness

Strong



Conscientiousness

Weak



Openness

Moderate



Decision-Making Tendency

Risk Tolerance

Weak



Delayed Gratification

Moderate



Anti-Emotional Eating Ability

Strong



Anti-Compulsive Hoarding

Moderate



Cognitive Ability

Executive Capacity

Moderate



Cognitive Ability

Moderate



Digit Memory Ability

Moderate



Spatial Memory Ability

Moderate



Episodic Memory Ability

Moderate



Verbal Memory Ability

Weak



Positive Emotions

Subjective Well-being

Moderate



Resistance to Job Burnout

Moderate



Anti-Anxiety Ability

Moderate



Positive Emotion

Moderate



Stress Handling Ability

Moderate



Interpersonal Psychology

Empathy

Moderate



Anti-Aggression

Strong



Social Communication Ability

Weak



Nonconformity

Weak



Anti-Loneliness

Moderate



Anti-Social Anxiety Disorder Ability

Strong



Subjective Well-being

Subjective well-being (SWB) refers to how people experience and evaluate their lives and specific domains and activities in their lives, it is a subjective feeling of whether an individual is happy or not.

My Subjective Well-being

Moderate

25.8% of We users are similar to me

Suggestions

How to improve subjective well-being?

1. Maintain good interpersonal relationships: Happy people are more likely to be chosen as friends and trusted ones by others. To help others more, to reduce negative emotions, and to build a relationship of mutual trust to meet people's need of belonging, will make people feel happy and satisfied.
2. Create a satisfactory living environment: we should choose a place to live that is physically safe, comfortable for ourselves and our families, achievable with beautiful natural scenery, comfortable climate, and music and art.
3. Actively increase physical activity: The short-term effects of exercise lead to the release of endorphins and morphine-like peptides in the brain; long-term regular exercise reduces depression and anxiety, improving the accuracy and speed of our work, enhancing our self-concept, and promoting cardiovascular health and function. In addition, people who exercise regularly often exercise in pairs with others and benefit from this additional social support, so we should maintain good health and participate in regular physical activity.
4. Moderate leisure time activities: Some studies have shown that participation in leisure or sports organizations, especially those that include dance, music, volunteer work or sports, can lead to higher subjective well-being.

Knowledge

What is subjective well-being

Subjective well-being includes two basic components: life satisfaction and emotional experience. The former is an individual's cognitive evaluation of the overall quality of life, that is, the degree of satisfaction judgment on personal life in general; the latter refers to the emotional experience in an individual's life, including both positive emotions (pleasant, relaxed, etc.) and negative emotions (depression, anxiety, tension, etc.). Subjective well-being consists of satisfaction with life, the experience of positive emotions and the lack of negative emotions. The higher the degree of satisfaction with overall life, the more positive emotions and the less negative emotions experienced, the stronger the individual's experience of well-being, and it is an important comprehensive psychological indicator to measure the quality of an individual's life.

Characteristics of subjective well-being

1. **Subjectivity:** The assessment of subjective well-being mainly relies on the criteria set by the individual himself, rather than the criteria of others or the outside world. Only one's personal feeling of happiness is the most authentic and accurate assessment of well-being, therefore, happiness is highly subjective, and the subjective reporting method is mostly used in research.
2. **Holistic:** Subjective well-being includes life satisfaction, positive emotions and negative emotions, reflecting the overall subjective quality of life of individuals, and is a comprehensive psychological indicator, so it has the characteristics of holistic and comprehensive.

Factors affecting subjective well-being

1. **Genetic:** According to a famous twin study, identical twins raised in different family environments were much closer in their subjective well-being levels than heterozygotic twins raised in the same family.
2. **Age:** Some studies have found that people's lifetime happiness changes with age in a U-shaped curve, namely, people's happiness is higher when they are young, reaches its lowest around the age of 40, and then gradually rises again as their happiness increases with age.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS324420	FAAH	CC	weak subjective well-being sensitivity
RS1585897	intergenic	AA	weak subjective well-being sensitivity
RS348258	intergenic	CC	strong subjective well-being sensitivity

RS815721	intergenic	CT	moderate subjective well-being sensitivity
RS183290952	intergenic	GG	strong subjective well-being sensitivity

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The subjective well-being item uses 82 polymorphic loci on and between the genes *RAPGEF6*: rs4680、rs324420、rs3756290, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian populations, other populations are for reference only.
2. The test may not cover all genes or loci that affect the subjective well-being.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your subjective well-being at the genetic level. Since the test does not involve consideration of other factors, it does not represents your true status.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the subjective well-being.

Risk Tolerance

Willingness to take risk refers to an individual's attitude towards risk and risk-taking tendency in the face of uncertainty of choice. Willingness to take risks will affect social behaviors such as investment and career choice (whether to start a business or not), as well as habits related to physical health such as diet, smoking, drinking and exercise.

My Risk Tolerance

Weak

22.3% of We users are similar to me

Knowledge

Risk-taking willingness affecting Factors

1. Inheritance: In twin studies, it is found that risk-taking willingness has a moderate degree of heritability, about 20%-60%, that is, 20-60% of risk-taking willingness variation is determined by genetic factors.
2. Age: The willingness to take risks decreases with age, and this trend is non-linear. The general explanation is that young people have more time to recover from potential losses (such as investment failures) behind their choices and tend to take more risks. Studies have also found that with age, biological changes such as enzyme activity may also be the underlying reasons.
3. Gender: Generally men have a higher willingness to take risks than women. In a European survey of 86,000 people, 33.6% of men considered themselves "adventurous", while the proportion of women was only 18.3%.
4. Education level: Education can improve a person's ability to assess risks, so that they have a higher willingness to take risks.
5. Income level: Income level has a positive effect on the willingness to take risks.
6. Wealth level: Wealth level is related to risk-taking willingness, but the way of influence is uncertain. On the one hand, people with wealth can easily bear the losses caused by risky behaviors (such as investment), and the level of wealth may also be a manifestation of their willingness to take risks to obtain greater benefits; on the other hand, wealthy people may be

relatively more likely to have more wealth. Conservative, and the poor regard risk as a speculative behavior, hoping to bring high returns through high risk.

 The relationship between risk-taking willingness and disease

1. Risky behavior is an important factor in evaluating many mental illnesses (such as attention deficit hyperactivity disorder, bipolar disorder, schizophrenia, etc.) or problem behaviors (such as smoking, drug abuse, alcohol abuse).
2. Obesity and other physical health problems are also considered to be related to the increase in risk-taking, involving abnormal reward processes, response inhibition and psychological mechanisms related to decision-making processes. Studies have also found genetic associations between obesity and willingness to take risks.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1051920	ZBTB10	CC	weak risk tolerance
RS1438026	intergenic	CT	moderate risk tolerance
RS2409095	intergenic	AA	strong risk tolerance
RS2357023	intergenic	CT	moderate risk tolerance
RS2325036	CADM2	CC	weak risk tolerance

For display consideration, only some genetic details are displayed.

Test details

 Applicable situation

The risk-taking willingness item uses 92 polymorphic loci in and between *EPHA5*, including rs13084531 and rs9379971.

 Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. This test may not cover all genes or sites that related to risk-taking willingness.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your willingness to take risks at the genetic level, and does not represent your true situation because it does not involve consideration of other factors.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all genes or loci related to risk-taking willingness.

Emotional Stability

Emotional stability is the ability to maintain emotional stability and reduce mood swings, as opposed to neuroticism, and it is a basic personality trait in psychology.

My Emotional Stability

Strong

9.4% of We users are similar to me

Suggestions

How to improve emotional stability?

1. Perceive your emotions: To understand your own emotional changes, know what kind of physical, psychological or external factors will affect your emotions, and when you predict that you will fall into an emotional trough due to something, adjust your emotions in advance and leave the current affairs.
2. Relieve your emotions: You can choose to cry bitterly or talk to a trusted friend, you can also choose to go shopping, listen to music, take a walk or force yourself to do other things to avoid always thinking of unpleasant things.
3. Cultivate hobbies: Through some artistic hobbies that cultivate temperament, such as piano, chess, calligraphy and painting, and singing. Many artistic activities can give people space to vent their feelings. It is best if you are interested and can express your emotions.
4. Enhance physical exercise: Actively participate in physical exercise activities, such as fitness, playing ball, dancing, deep relaxation, and massage. Imagining bad emotions being beaten out like a ball, or swaying out with sweat, will give a feeling of joy.
5. Create a pleasant living environment: Create a pleasant living environment for yourself, such as playing music, incense, and soft lighting, or put yourself in a refreshing natural environment, physiologically to relieve your nervous tension.

Knowledge

What is emotional stability

Emotional stability refers to small, positive emotional fluctuations and good adjustment of emotions in the face of external stimuli; its opposite is neuroticism, that is, emotional instability, negative, and it is difficult to make reasonable adjustments to external stimuli.

Emotional stability, or neuroticism, occupies an important position in psychological study, and it is listed as the five major aspects of personality theory along with extraversion, agreeableness, conscientiousness and openness, which is the Five-Factor Model (FFM).

Behavioral performance of emotional stability

Emotional stability is generally divided into five sub-dimensions:

- Anti-anxiety: an individual's ability to overcome anxiety.
 - High score performance: Calm
 - Low score performance: Fidgeting
- Anti-depression: an individual's ability to overcome depression.
 - High score performance: Calm
 - Low score performance: Depressed
- Anti-oversensitiveness: an individual can reduce the ability to pay too much attention to their own behavior and to care about what others think of them.
 - High score performance: Calm, confident
 - Low score performance: Sensitive and suspicious
- Anti-vulnerability: the ability of an individual to overcome powerlessness and fragility in the face of crisis or difficulty.
 - High score performance: Strong
 - Low score performance: Incapable, vulnerable, evasive
- Anti-anger/hostility: the ability of an individual to cope with anger and related emotions.
 - High score performance: Calm
 - Low score performance: Irritable

Factors affecting emotional stability

1. Inheritance: Emotional stability is the result of the combined effect of heredity and environment. According to research, genetics contribute about 40% to 60% of emotional stability.
2. Gender: In a large-scale population study, it was found that women have a higher level of neuroticism than men. Even in different cultural environments, women are slightly higher than men.
3. Age: The level of neuroticism will decrease as a person matures, and will stabilize after the age of 40.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS12421026	intergenic	CT	moderate likelihood to be emotionally stable
RS6606710	MYO1H	TT	more likely to be emotionally stable
RS2273085	L3MBTL2	CT	moderate likelihood to be emotionally stable
RS6882046	intergenic	AA	more likely to be emotionally stable
RS9584850	intergenic	GG	less likely to be emotionally stable

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The emotional stability item uses 114 polymorphic loci on genes and between genes, including the *CACNA1E* gene, such as rs2921036, rs10097870, and rs2953805, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect emotional stability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. The test result only predicts your emotional stability from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to emotional stability.

Empathy

Empathy is the ability to put oneself in the position of others and understand or feel what others experience in their situation.

My Empathy

Moderate

24.7% of We users are similar to me

Suggestions

How to improve empathy?

1. Learn to empathize: Be able to find rationality for the other party's behavior from the other party's perspective, so as to understand the other party to the greatest extent.
 2. Learn to listen: Refers to the ability to listen to each other's expressions wholeheartedly. Listening not only refers to listening to the content of their spoken expressions, but also includes observing non-verbal behaviors, such as actions, expressions, voice, pronunciation, and intonation.
 3. Express respect: Respect each other's personality and abilities instead of acting on your own feelings. Accept the other's beliefs and choices or decisions made instead of commenting or trying to make decisions for them.
-

Knowledge

What is empathy?

It refers to being able to think from the position of the other person, to experience the emotions and thoughts of others in the process of interpersonal communication, to understand the positions and feelings of others, and to think and deal with problems from the perspective of others. It is mainly reflected in aspects related to emotional intelligence, such as emotional self-control, empathy, listening ability, and expression of respect.

Empathy is the basis of altruistic behavior because people with empathy characteristics can feel the needs and distress of others firsthand, and can provide support and help to others in a decent and

respectful manner when necessary. Therefore, it is very helpful for the establishment of healthy interpersonal relationships. In daily life, a person with empathy is less likely to have conflicts with others, because he (she) can always understand others to the maximum and get along with others in a peaceful state of mind.

 The difference between empathy and compassion

Compassion is the recognition of the suffering of others, which causes compassion. Empathy is the ability to empathize with one another, put yourself in consideration for others, and actually feel the pain of others. When hearing other people’ s misfortune, sympathetic people will say: "I really feel sorry for you." But people with empathy will say something like this: "I have encountered something like this, and I know what it feels like." Empathy is the process of interaction between two people. It must be listened carefully and understood. , Accept the other person's feelings, this is an attitude that recognizes the experience of others. Empathy is an art of care and love, and a manifestation of high emotional intelligence.

 Factors affecting empathy

- 1. Inheritance: Studies have shown that the influence of heredity in empathy may reach more than 30%.
- 2. Gender: A number of studies have confirmed that the empathy ability of women is generally higher than that of men.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS53576	OXTR	AG	moderate empathy
RS76891664	intergenic	AG	moderate empathy
RS11264567	intergenic	AG	moderate empathy
RS1141090	RASSF10	AC	moderate empathy
RS10265275	intergenic	CT	moderate empathy

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The empathy item uses 6 polymorphic loci on genes and between genes, including the *OXTR* gene, such as rs53576, rs76891664, and rs11264567, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect empathy.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. The test result only predicts your empathy from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to empathy.

Resistance to Job Burnout

Anti-job burnout ability refers to the ability to correctly overcome physical, emotional or mental exhaustion when occupational factors such as high work pressure appear.

My Resistance to Job Burnout

Moderate

33.6% of We users are similar to me

Suggestions

How to improve resistance to job burnout?

1. Through active communication and feedback, get emotional support from colleagues or superiors, and adjust various stressors from work.
2. Balance work and life time to ensure adequate sleep.

Knowledge



Common causes of job burnout

1. There is no decision-making power and lack of control over the work content, schedule, and workload.
2. The authority of the self or the expectations of the superiors is not clear.
3. Encountered bullying in the workplace or over-management of work details by superiors.
4. My own values do not match the company's business or working methods.
5. Not interested in the job content and required skills.
6. The job content is too monotonous and it is necessary to require constant consumption of energy to stay focused.
7. Feeling isolated and helpless in work and life, lack of emotional support.

8. There is an imbalance between work and life, and work takes up too much time with family and friends.

Potential signs of job burnout

Chronic fatigue, insomnia, low concentration, chest tightness, shortness of breath, loss of appetite, decreased immunity, negative emotions (anxiety, depression, apathy, irritability, pessimism), forgetfulness, lack of accomplishment, low work efficiency.

Factors affecting job burnout

Poor sleep quality, lack of exercise, lack of recreational activities and other lifestyles may aggravate job burnout.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS13219957	UST	GG	strong resistance to job burnout
RS6265	BDNF	CT	moderate resistance to job burnout
RS12506228	F11-AS1	AC	moderate resistance to job burnout

Test details

Applicable situation

The resistance to job burnout item uses 3 polymorphic loci on genes and between genes, including *F11-AS1*, *UST*, and *BDNF*.

Notification

1. The basis of the test is mainly from the research of Caucasian and Chinese, other populations are for reference only.
2. The test may not cover all genes or loci that affect resistance to job burnout.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. The test result only predicts your resistance to job burnout from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

Test content

The test includes rs13219957, rs12506228, and rs6265, a total of 3 polymorphic loci related to the risk of resistance to job burnout.

Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to resistance to job burnout.

Delayed Gratification

Delayed gratification refers to the tendency to give up the current short-term satisfaction for longer-term and more valuable goals, as well as the ability to self-control in the waiting period.

My Delayed Gratification

Moderate

24.9% of We users are similar to me

Knowledge

What is delayed gratification

In psychological research, it is found that when people evaluate the long-term value of things, people tend to underestimate future benefits (or losses) compared with current or near-term gains (or losses). The delayed gratification is the ability to resist this tendency, to be willing to achieve greater long-term goals, and to suppress temporary short-term impulses and needs, and to be able to control itself in the process of achieving the goals. This ability includes analysis of the status quo, prediction of the future, and self-control.

Delayed gratification performance

People with high delayed gratification will appear "long-sighted" in daily life, and often have better achievements in career, family, health, finance, etc. For example, in the famous "Marshmallow Experiment" hosted by psychologist Walter Mischel, children with low delayed gratification chose to eat marshmallows immediately, while children with high delayed gratification chose to wait 15 minutes before eating. Obtained an extra second piece of marshmallow according to the rules. People with low delayed gratification often only value immediate enjoyment and convenience, while ignoring long-term benefits. For example, the cost of preventive medical treatment is often lower than the cost of remedial medical treatment after illness. For example, weight loss can reduce the risk of many diseases. However, due to the current pain of losing weight, people with low delayed gratification often delay and delay, but it causes higher medical costs.

Factors affecting delayed gratification

1. Inheritance: The delayed gratification is highly influenced by heredity, and some studies have speculated that its heritability rate is 57%.
2. Age: In early childhood, ordinary children do not have the delayed gratification. Delayed gratification begins to appear after about 5 years old, and will increase to a certain extent with age.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS324420	FAAH	CC	weak delayed gratification
RS3756450	intergenic	GG	strong delayed gratification
RS3773678	DRD3	GG	weak delayed gratification
RS464049	SLC6A3	GG	strong delayed gratification
RS4680	COMT	GG	weak delayed gratification

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The delayed gratification item uses 10 polymorphic loci on genes and between genes, including the *COMT* gene, such as rs4680 and rs324420.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect delayed gratification.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. The test result only predicts your delayed gratification from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

Study population

The basis of the test is mainly from the research of Caucasians.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to delayed gratification.

Anti-Anxiety Ability

It refers to the ability to resist physical or emotional states such as rapid heartbeat, shortness of breath and difficulty in concentrating due to anxiety.

My Anti-Anxiety Ability

Moderate

34.2% of We users are similar to me

Suggestions

- Your anti-anxiety ability is **medium**, sometimes prone to anxiety, it is recommended to learn how to deal with anxiety properly
- In addition to genetic factors, these people are also more prone to anxiety: People with inferiority complex and pessimistic personality, People who smoke and are alcoholic, People who experience stress and trauma
- Appropriate anxiety can make us motivated to do something, but excessive anxiety can make us Emotionally distressful and also bring a lot of Physical discomfort
- Use the following methods to improve your anxiety state:
 - Take deep breaths, relax muscles and imagine positive images in a quiet place (such as a quiet natural landscape)
 - Supplementing **L-tyrosine** and **Vitamin B12** is good for soothing mood
 - Adjust to Healthy living habits

* If you need to use dietary supplements, or need psychological intervention, it is recommended that you consult an authoritative health professional or clinician for help.

Knowledge



What is anxiety?

Anxiety is a general term for emotions that include worry, tension, fear, embarrassment and other emotions. It also brings some physical arousal, such as rapid heartbeat, shortness of breath, sweaty palms, dizziness, gastrointestinal discomfort and other physical reaction.



Is anxiety entirely a bad thing?

Appropriate anxiety can make us motivated to do something. The inverted U-shaped curve derived from research on anxiety and performance also confirms this point. Appropriate anxiety can get the highest performance. However, excessive anxiety can bring emotional pain and a lot of physical discomfort, such as often feeling tired, muscle tension, neck soreness, decreased sleep quality, and decreased happiness. In short, excessive anxiety will make a person's quality of life drop a lot.

 Factors affecting anxiety

- 1. Inheritance: Genetically speaking, if one of the identical twins suffers from anxiety, the chance of the other will also increase. If the parents have anxiety disorders, the probability of their children getting the disease will rise to 50%~75%.
- 2. Personality: Some personality traits, such as low self-esteem, over-reliance on others, self-blame, pessimism, easy to be overwhelmed by pressure, will make you more prone to anxiety.
- 3. Smoking and alcohol abuse: Studies have shown that about 30% of patients with severe anxiety are related to alcohol and substance abuse. In addition, people with anxiety are about twice as likely to become addicted to nicotine.
- 4. Environment: Stressful life events and feelings of loss may induce anxiety, such as widowhood, divorce, unemployment, financial crisis, poor health.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS62353264	intergenic	AA	strong anti-anxiety ability
RS12637928	intergenic	AA	strong anti-anxiety ability
RS4653663	intergenic	TT	strong anti-anxiety ability
RS12378446	intergenic	TT	weak anti-anxiety ability
RS12682352	MFHAS1	CC	strong anti-anxiety ability

For display consideration, only some genetic details are displayed.

Test details

 Applicable situation

The anti-anxiety ability item uses 15 polymorphic loci on genes and between genes, including the *KSR2* gene, such as rs7973260 and rs62100776.

 Notification

1. The basis of the test is mainly from the research of Caucasians and Latinos, other populations are for reference only.
2. The test may not cover all genes or loci that affect anti-anxiety ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

 Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. The test result only predicts your anti-anxiety ability from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

 Study population

The basis of the test is mainly from the research of Caucasians and Latinos.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to anti-anxiety ability.

Executive Capacity

Execution ability refers to some advanced cognitive functions and processes that need to be used to complete a specific goal or task, such as concentration, working memory, control, and cognitive flexibility. Its essence is the ability to control and regulate the cognitive process, which is generally called executive function.

My Executive Capacity

Moderate

49.4% of We users are similar to me

Suggestions

How to improve executive capacity?

1. Studies have shown that professional targeted behavior training can improve executive capacity, including task switching training, memory update training, reaction inhibition training, etc.
2. Actively participate in aerobic exercise. Participating in aerobic exercise can effectively improve the function of the prefrontal cortex of the brain; at the same time, the human body secretes endorphins during aerobic exercise, which can increase the degree of pleasure. Aerobic exercise involving multiple people can also increase social connections, and negative emotions such as sadness and loneliness will reduce the executive capacity.

Knowledge

What is executive capacity

Executive capability, or executive function, is the conscious mental control ability that people need to perform tasks and complete specific goals. It is a very comprehensive and advanced cognitive function. Executive capability is mainly reflected in the following aspects:

1. Planning and decision-making capability: Be able to analyze the steps needed to complete the task, and build a complete plan.

- 2. Management capability: Be able to make reasonable arrangements for time and resources.
- 3. Working memory capability: When completing complex tasks, be able to remember the key information and use this information to formulate a problem-solving plan.
- 4. Control: It can restrain the instinctive impulse during action, take the completion of tasks as the goal, do not delay, control its own behavior and manage its own emotions in the process of completion, and do not be distracted by other things.
- 5. Adaptive capacity: In the face of new changes, obstacles, and mistakes, the plan can be revised in time, and the environment can be adequately adapted.

 Development process of executive capability

Executive capability is one of the latest psychological functions of maturity. It is in continuous development and change at all stages of life.

Early childhood: Attention, working memory and other abilities appear in early childhood. During the kindergarten period (3 to 5 years old), adaptability, planning and other abilities will also begin to form rapidly. Of course, in this period, there is no mature and complete executive capability, so mistakes are often made in early childhood. This is not caused by insufficient ability, but because the brain's decision-making ability and executive capability are not mature enough in this period.

Before puberty: During this period, executive ability will once again improve rapidly, especially language working memory, purposeful behavior, attention, control and other abilities will be significantly improved. However, similar to the early childhood, the overall executive ability is still in a relatively limited state.

Puberty: The abilities that appeared before puberty continue to grow at this stage, and the system is integrated. The ability to concentrate may have an explosive growth at this stage.

Adulthood: The most important change in adulthood is the continuous myelination of the prefrontal cortex. The age of 20-29 is the strongest period of execution, allowing most people to participate in very challenging tasks during this period. As the age gradually increases, the execution ability will gradually decline accordingly.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS3748348	RNASE13	GG	weak executive capacity
RS17518584	CADM2	TT	strong executive capacity
RS10771511	intergenic	CT	moderate executive capacity

RS16987794	intergenic	TT	weak executive capacity
RS68179743	MLH3	CC	weak executive capacity

Test details

Applicable situation

The executive capability item uses 5 polymorphic loci on and between genes in *CADM2* and *RNASE13*, including rs17518584 and rs3748348, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect executive capacity.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. The test result only predicts your emotional stability from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to executive capacity.

Anti-Aggression

Violent behavior, that is, aggressive behavior, refers to behavior that causes harm to people or things physically, verbally, or psychologically (such as hitting, kicking, biting, scratching, threatening, humiliating, blaming, sabotaging, etc.). The ability to resist violence refers to the ability to prevent or restrict aggressive behavior.

My Anti-Aggression

Strong

11.6% of We users are similar to me

Knowledge

Aggressive behavior in childhood

As children grow up, they will gradually learn to control and express their emotions and behaviors, and communicate with others in a positive way. Before learning, children may try to express themselves with aggressive behavior (hitting, kicking, biting, scratching, etc.). There are many reasons for aggressive behavior, such as expressing the need for attention, wanting to achieve their goal quickly, gaining a sense of power in the behavior, etc.

Factors influencing aggressive behavior

1. Biological factors: males have more physically aggressive behaviors than females, while females have more non-physically aggressive behaviors (such as verbal aggression). In addition to sex hormones, adrenocorticosteroid and pituitary adrenocorticotrophic hormone also play a role in regulating aggressive behavior.
2. Disease factors: epilepsy, dementia, autism, attention deficit hyperactivity disorder, schizophrenia and brain injury can all lead to increased aggressive behavior.
3. Other factors: drug and alcohol abuse, unhealthy family relationships, childhood witnessed aggressive behavior, and chronic stress can all contribute to the increase of aggressive behavior.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1611115	DBH	CC	strong anti-aggression
RS53576	OXTR	AG	moderate anti-aggression
RS11126630	intergenic	TT	strong anti-aggression
RS6770632	intergenic	CC	strong anti-aggression
RS35974940	NTM	GG	strong anti-aggression

Test details

Applicable situation

The anti-aggression item uses five polymorphic loci on and between three genes such as *OXTR* and *NTM*, etc., including rs11126630, rs1611115.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci affecting the anti-aggression.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your anti-aggression at the genetic level. Since the test does

not involve consideration of other factors, it does not mean your true health status.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to anti-aggression.

Social Communication Ability

Personality trait with weak emotional expression ability is also called alexithymia, which manifests as a lack of emotional awareness, difficulty in identifying and expressing feelings and emotions.

My Social Communication Ability

Weak

13.6% of We users are similar to me

Suggestions

How to improve the ability of social communication ability?

1. Increase your chances of emotional contact by writing diaries, reading stories, watching movies, and touching art works.
 2. Use language and words to express your inner feelings.
 3. Listen and empathize more, improve your understanding of others, and express your concern for them.
 4. If you feel that you are greatly affected by alexithymia, you can seek help from a psychological counselor.
-

Knowledge

what are the manifestations of alexithymia?

Alexithymia, that is, the social communication ability is weak, is characterized by a lack of sensitivity to emotions and may seem indifferent and showing a sense of distance in the eyes of others. People with alexithymia traits may be indifferent to positive emotions (such as joy and surprise) or extremely calm in the face of negative emotions (such as tension and anxiety). On the other hand, although the brain's ability to deal with emotions is weak, the body's senses will not disappear or even become more sensitive, such as shoulder stiffness in the face of pressure.

Factors affecting alexithymia

- 1. Socio-cultural differences: people from different cultural backgrounds will show different levels of alexithymia. For example, the level of alexithymia of Asian Americans is higher than that of European Americans.
- 2. Socioeconomic status: People with low education and poverty are more likely to have hyperalexithymia because their socioeconomic status requires them to struggle for survival and discourages the processing and expression of their emotions and feelings.
- 3. Family environment: bad family environment may affect children's early socialization and suppress their sense of attachment, which is a risk factor for the development of alexithymia.

 Diseases associated with alexithymia

Most of the patients with mental illness (autism, depression, schizophrenia, etc.) have alexithymia. Patients with diabetes, coronary heart disease, hypertension, stroke, psoriasis, asthma, duodenal ulcer, and breast cancer are more likely to be affected by alexithymia than the general population.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2352908	intergenic	GT	moderate social communication ability
RS3761168	intergenic	AC	moderate social communication ability
RS4453791	intergenic	TT	strong social communication ability
RS6295	HTR1A	GG	weak social communication ability
RS9257616	intergenic	AG	moderate social communication ability

Test details

 Applicable situation

The social communication ability item uses five polymorphic loci on and between *HTR1A* gene, including rs6295, rs9257616.

 Notification

- 1. The basis of the test is mainly from the research of Caucasian and Chinese, other populations are for reference only.

2. The test may not cover all genes or loci affecting the social communication ability ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your social communication ability ability at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to social communication ability.

Cognitive Ability

Cognitive ability refers to the comprehensive ability of the brain to process and process information from the outside world, and to use this information to solve problems.

My Cognitive Ability

Moderate

37.3% of We users are similar to me

Suggestions

How to improve cognitive ability?

1. Keep reading: reading is an activity that stimulates the brain, can stimulate our imagination and critical thinking, helps us perceive the world in new ways, and improve our cognitive ability.
2. Stress management: living and working in a high-pressure environment for a long time will reduce cognitive ability. Regular exercise, listening to music, meditation, etc. will help reduce stress.
3. Reasonable diet: many foods contain more DHA, vitamins, anthocyanins and other substances that help improve cognitive ability, such as green tea, salmon, nuts, cabbage and so on.
4. Pay attention to a good rest: Getting at least seven hours of sleep not only improves memory, but also improves your ability to focus on tasks the next day, all of which contribute to improve cognitive ability.
5. Keep exercising: exercise can improve the ability of oxygen supply to the brain, improve memory and reduce the possibility of dementia. At the same time, exercise can also relieve stress, release growth factors and promote brain function.
6. Good living habits: quitting smoking and drinking, including staying away from second-hand smoke, can avoid environmental factors harmful to cognitive ability.

Knowledge

 What is cognitive ability?

Cognitive ability refers to the comprehensive ability of the brain to process external information and use this information to solve problems. Cognitive ability does not refer to our knowledge reserve, but to more basic learning, memory, concentration and problem-solving ability. From the simplest to the most complex affairs in daily life, cognitive ability is needed. For example, when completing the topic of "reading pictures and telling stories" in the primary school examination, It requires perception (identifying objects in graphics), decision-making (deciding what topic to write), language ability (converting ideas into text information), and motion control ability (control hands to complete writing) and other complex cognitive abilities. People with strong cognitive ability have good learning ability and academic performance at school; they are good at communication and cooperation at work, are more competent for difficult work, do not give up easily, and can focus on completing complex tasks.

Cognitive ability is supported by the functions of multiple specific brain regions. For example, memory skills mainly depend on parts of the temporal lobe and hippocampus. Although the function of the brain has not been fully defined, cognitive ability is the most basic ability of biology and the greatest advantage of human beings over other species.

Factors affecting cognitive ability

1. Living habits: long term alcoholism will damage the brain and reduce cognitive ability; Smoking or second-hand smoke can lead to brain hypoxia and damage brain cells, as well as cognitive decline.
2. Pressure: long term exposure to high pressure will promote the release of cortisol. Cortisol is also known as "stress hormone". A small amount of cortisol can temporarily increase concentration ability and improve learning ability, but a large amount of cortisol secretion over a long period of time will reduce cognitive ability, and lead to emotional instability, metabolic disorder, decreased immune ability and other problems.
3. Learning atmosphere: good family atmosphere, correct guidance from teachers and mutual promotion among peers can help us learn better, focus on effective exercise and improve cognitive ability.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS10119	TOMM40	GG	strong cognitive ability
RS2352974	intergenic	CC	strong cognitive ability
RS62181012	intergenic	TT	strong cognitive ability

RS62198803	intergenic	GG	weak cognitive ability
RS7573001	intergenic	GG	strong cognitive ability

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The cognitive ability item uses 195 polymorphic loci on and between genes such as *TOMM40*, etc., including rs4731365, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci affecting the cognitive ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your cognitive ability at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to cognitive ability.

Digit Memory Ability

Digital memory ability refers to the ability to remember various forms of numbers.

My Digit Memory Ability

Moderate

45.3% of We users are similar to me

Suggestions

How to improve digit Memory ability?

1. Adopt reasonable memory strategies: there are many training methods and skills in digital memory. Through targeted practice, the efficiency of digital memory can be significantly improved.
2. Pay attention to sleep: in the sleep stage, the brain will process and consolidate the memory content, so timely sleep is conducive to memory. At the same time, adequate sleep can improve the concentration and efficiency of memory.
3. Use other levels of memory: for example, our ability to remember images is stronger than simple numbers. Therefore, it is easier to abstract information into graphics and images in the process of memory than to memorize numbers directly.
4. In daily life, you can use doggerel to help you to remember: for example, you can use "a temple and a pot of wine on the top of the mountain" to correspond to 3.14159 of PI.

Knowledge



What is digit Memory ability?

Digital memory refers to the ability to remember numbers in different forms, whether heard or seen. People with strong digital memory ability generally have a deep memory of digital content in their study and work, it is easy to remember various scientific constants (such as pi, natural logarithm, etc.) and various digital related contents such as telephone, certificate number and memorial day. It

will be more advantageous to engage in statistics, accounting and other digital related industries, and may also perform better in digital related games such as poker, chess and cards.

Factors affecting digit Memory ability

1. Language environment: some studies have shown that under the same conditions, Chinese speakers have stronger digital memory capacity than English speakers. It is speculated that the Chinese digital monosyllabic pronunciation method is more suitable for digital memory.
2. Disease: damage to the brain, especially the left side of the brain, can lead to a decline in digital memory capacity, and other mental disorders can also negatively affect digital memory capacity.
3. Dietary habits: a variety of Vitamins and trace elements in the diet can affect brain function, and the unsaturated fatty acids in fish also have a corresponding promotion effect on memory. Long-term intake of too much saturated fatty acid will reduce the digital memory capacity to a certain extent.
4. Living habits: long term alcoholism will damage the brain and further affect the digit Memory ability.
5. Education level: studies have shown that the longer you receive education, the slower the decline of the digit Memory ability with age.
6. Gender: the digit memory ability of men is slightly higher than that of women.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS6265	BDNF	CT	moderate digit memory ability
RS34087853	CAMK2A	AA	moderate digit memory ability

Test details

Applicable situation

The digit Memory ability item uses two polymorphic loci on and between *CAMK2A* gene and *BDNF* gene, including rs34087853, rs6265.

Notification

1. The basis of the test is mainly from the research of Caucasian and Chinese, other populations are for reference only.

2. The test may not cover all genes or loci affecting the digit Memory ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your digit Memory ability at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to digit Memory ability.

Extraversion

Extraversion is one of the five personality traits, the tendency of individuals to seek motivation through the company of others. It generally includes enthusiasm, sociality, decisiveness, activeness, adventurousness, optimism and other aspects.

My Extraversion

Weak

22.5% of We users are similar to me

Knowledge

What is extraversion?

Extraversion, also known as extroversion, is one of the five main factors of personality structure. extraversion (extroversion) refers to the tendency of individuals to seek encouragement through the company of others. Individuals with high scores are sociable, energetic, optimistic and confident, and like the company and cooperation of others. People with low scores are calm, introverted and implicit, and are more willing to be alone or think. Too much interpersonal communication will make them feel tired.

Extraversion behavior

Extraversion can generally be divided into three sub-dimensions:

- Gregariousness: Individuals are willing to stay with others, that is, the tendency to get close to the crowd.
 - High score performance: Sociable and gregarious
 - Low score performance: Personality closed, and solitude
- Warmth: An individual's attitude towards others.
 - High score performance: Hospitality
 - Low score performance: Indifference
- Domination: Individuals like to command, manipulate others and tend to lead others.
 - High score performance: Aggressive and bossy
 - Low score performance: Obedience and low profile

What are the effects of extraversion or introversion?

- Evolutionary aspect

In evolutionary history, extroverts are considered more beneficial because they tend to be happier and have more opportunities for sexual reproduction. But extroverts have more contact with others, so they have a greater chance of catching infectious diseases. Introverts tend to pursue peace, be single-minded to their spouse, and pursue less external stimulation, so that they can get a better and stable living environment, and their average life expectancy is longer.

- Work aspect

People with high extroversion tend to be attracted to motivators in the work environment. It is likely that people will prioritize incentives in their job choices and perform better in such an environment; People with high extraversion are more optimistic in the face of difficulties, so they experience less stress than people with low extraversion. In addition, people with high extroversion tend to have good social skills, which can help them get information and help relatively easily at work and reduce the pressure from work.

- Interpersonal communication

Extroverts are generally keen on interpersonal communication. They are enthusiastic, talkative, confident and like to make friends. They feel happy at social gatherings, such as banquets, community activities, etc. Performance, teaching, conducting, management and agent are the fields of more extroverts. Most extroverts prefer to be with others rather than alone. Introverts tend to think before speaking, and introverts prefer to think alone rather than talk to others.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS17147371	intergenic	AA	strong extraversion
RS11103814	intergenic	AA	weak extraversion
RS7922066	CRTAC1	AA	weak extraversion
RS7905091	intergenic	GG	weak extraversion
RS12419614	GRIA4	GG	weak extraversion

Test details

 Applicable situation

The extraversion item uses six polymorphic loci on and between genes such as *CRTAC1*, etc., including rs17147371, rs11103814, etc.

 Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci affecting the extraversion.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

 Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your extraversion at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to extraversion.

Nonconformity

Herd behavior refers to the phenomenon in which an individual's ideas and behaviors change in the same direction as the majority due to the influence of real or imagined group pressure. Nonconformity refers to the degree of being less susceptible to the actions of others.

My Nonconformity

Weak

27.2% of We users are similar to me

Knowledge



Why do we follow the crowd?

1. Behavioral reference: Unable to make a definite choice due to lack of personal knowledge and experience.
2. Fear of deviation: When deviating from the group, there will be strong group pressure (exclusion, rejection, disgust, etc.).
3. Interpersonal adaptation: Expect recognition from the group and maintain good interpersonal relationships so as to gain a sense of self-worth from social support.



Factors affecting conformity tendency

1. Difficulty of tasks: difficult tasks may lead to both increased and decreased conformity. When faced with a difficult task and do not know what to do, people tend to follow the crowd; However, increasing the difficulty of the task will make it easier for people to accept various results, resulting in less conformity.
2. Individual characteristics: people with strong initiative and leadership are more difficult to follow the crowd.
3. Group size: when the group size is 3 ~ 5 people, it is easier to follow the crowd.
4. Uncertain situations: people tend to follow the crowd when facing uncertain situations.
5. Cultural factors: people from collectivist cultural areas (such as Asia, Central America, etc.) are more likely to follow the crowd.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2619056	intergenic	GG	weak nonconformity
RS2056708	CCDC146	CT	moderate nonconformity
RS10062113	intergenic	TT	weak nonconformity
RS13170785	ARL10	AG	moderate nonconformity
RS56324903	intergenic	GG	strong nonconformity

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The nonconformity item uses six polymorphic loci on and between *CCDC146* gene and *ARL10* gene, including rs2619056, rs2056708, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian and Chinese, other populations are for reference only.
2. The test may not cover all genes or loci affecting the nonconformity.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your nonconformity at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to nonconformity.

Agreeableness

Agreeableness is one of the five personality traits, it refers to the tendency of individuals to treat others with compassion and cooperation. It generally includes traits such as trust, straightforwardness, altruism, compliance, modesty, and tender-mindedness etc.

My Agreeableness

Strong

19% of We users are similar to me

Knowledge



What is agreeableness?

Agreeableness is one of the five main factors of personality structure, which refers to whether an individual has compassion and willingness to cooperate with others. People with high scores of agreeableness are helpful and compassionate. People with low scores are more hostile and suspicious. The former focuses on cooperation rather than competition, the latter likes to fight for their own interests and beliefs.



Behavioral performance of agreeableness

Agreeableness can generally be divided into three sub-dimensions:

- Trust: The degree to which an individual believes in another person or/and the speech of another person.
 - High score performance: Trust others
 - Low score performance: Doubt
- Consideration: The degree to which an individual pays attention to the interests and needs of others.
 - High score performance: Considerate and gentle
 - Low score performance: Indifferent and unconcerned
- Sympathy: An individual's attitude towards a disadvantaged person or thing.
 - High score performance: Compassionate
 - Low score performance: Indifference



What are the effects of agreeableness?

- Interpersonal relationship

People with high scores of agreeableness tend to like others and look at them positively, while people with low scores of agreeableness tend to look at people negatively. Research also shows that people with high scores of agreeableness are more likely to control negative emotions such as anger in conflict situations. People with high scores of agreeableness are more likely to use constructive strategies in conflict with others, while people with low scores of agreeableness are more likely to use coercive means.

- Prosocial behavior

The core feature of agreeableness is that it is positively related to altruism and helping behavior. In various situations, people with high scores of agreeableness are more likely to be interested and participate in helping others. Experiments show that although most people may help their relatives or help others when sympathy is aroused, friendly people will help others even when these conditions do not exist.

- Mental illness

In terms of mental illness, people with low scores of agreeableness may be related to narcissism and antisocial tendencies.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS3204145	IKBKAP	AT	moderate agreeableness
RS6832769	intergenic	AA	weak agreeableness
RS2287826	intergenic	GG	strong agreeableness
RS13113475	intergenic	AG	moderate agreeableness
RS7042201	intergenic	CC	weak agreeableness

For display consideration, only some genetic details are displayed.

Test details



Applicable situation

The agreeableness item uses 9 polymorphic loci on and between *IKBKAP* gene, including rs2287826, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect the agreeableness.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your agreeableness at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to agreeableness.

Anti-Loneliness

Loneliness refers to a psychological feeling when social needs are not met. People with strong anti-loneliness are less likely to feel loneliness.

My Anti-Loneliness

Moderate

36.8% of We users are similar to me

Suggestions

How to reduce loneliness?

1. Get out of the house, feel the outdoor air and sunshine, and say hello to the people around you.
 2. Do some exercise, jogging, swimming, yoga, boxing are all good choices.
 3. Call family or friends, chat for a while, and make an appointment to meet up.
 4. Control the screen usage time of mobile phones, computers, TVs, etc.
 5. Try to keep a pet.
 6. Work and rest regularly and get a good night's sleep.
 7. Know yourself and know that loneliness is not a permanent state.
 8. If none of the above methods work, seek the help of a psychologist or counselor.
-

Knowledge

Is it normal to feel lonely often?

Everyone will feel lonely. Gender, age, appearance and socio-economic conditions can't protect you from loneliness. Don't worry too much about it. Loneliness and solitude cannot be completely equated. Loneliness may result from being alone for a long time, or simply feeling incomprehensible by others.

The possible impact of loneliness

Loneliness can have a series of psychological and physical effects: loneliness makes people more prone to depression, decreased sleep quality, impaired executive function, decreased cognitive ability, decreased immunity, decreased cardiovascular function. Loneliness is a risk factor for Alzheimer's disease, heart disease, type 2 diabetes, arthritis and other diseases. Loneliness can increase the "fight-or-flight response" (a series of physiological reactions when a person faces threats). Loneliness can also affect people's life expectancy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1044396	CHRNA4	GG	weak anti-loneliness
RS53576	OXTR	AG	moderate anti-loneliness
RS613872	TCF4	TT	weak anti-loneliness
RS11605348	intergenic	GG	weak anti-loneliness
RS2069117	intergenic	AA	strong anti-loneliness

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The anti-loneliness ability item uses 14 polymorphic loci on and between 5 genes such as *TCF4*, *OR1S1*, *MBD5*, including rs613872, rs2069117, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect the anti-loneliness ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your anti-loneliness ability at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the anti-loneliness ability.

Anti-Social Anxiety Disorder Ability

Anti-social anxiety disorder ability refers to the ability to resist anxiety and fear in social situations.

My Anti-Social Anxiety Disorder Ability

Strong

10.3% of We users are similar to me

Suggestions

How to overcome social anxiety disorder?

1. Find out the reasons and try to overcome: Find out the specific factors that trigger your fear or anxiety, and the scenes you are afraid of. Based on your analysis of triggers and fear scenarios, you can deal with these anxiety emotions in a more positive or targeted way by understanding what is the response that causes your anxiety.
2. Try to calm yourself: learn to recognize your own physical reactions to anxiety or fear, and then try those methods to calm yourself: for example, take a deep breath, or tighten your body for three seconds and then relax, listen to some songs that can make you feel happy, or calm down to meditate.
3. Improve your own thoughts: Use positive thoughts to replace the negative thoughts that are automatically generated by the brain, and cultivate more self-confidence.
4. Reduce attention to yourself: Observe the people and environment around you, and focus on what others are saying instead of focusing too much on yourself.
5. Try to communicate more with people who understand you: try to find those who have the same troubles with you and understand you, and communicate more with them. Maybe it can help you improve each other, or let you accumulate some confidence. Some of their successful experiences can also be used for reference.

Knowledge

What is social anxiety disorder

Social anxiety disorder is a psychological state of intense fear or anxiety for any social or public occasion.

Performance of social anxiety disorder

People with social anxiety disorder have a significant and persistent fear that their behavior or nervous performance will cause humiliation or embarrassment in front of strangers or in social or performance occasions that may be carefully observed by others. Even some people find it difficult to attend parties, call, shop, or ask authorities.

Causes of social anxiety disorder

1. Physiological reasons: Studies have shown that it may be caused by the imbalance of the chemical substance called "serotonin" in the human body. This substance is responsible for transmitting information to the nerve cells of the brain. Too much or too little of it may cause people's fear emotions.
2. Psychological reasons: People with social anxiety disorder generally have strong self-esteem, fear of being rejected by others, or have no confidence in their appearance.
3. Family reasons: Their personality is depressed since childhood, or their parents do not teach them social skills, or the family moves too frequently.
4. Social reasons: The social environment is relatively bad, and most of them suffer setbacks when communicating with others.
5. Way of thinking: Incorrect way of thinking has created social anxiety disorder, such as excessive self-reflection after being hit by excessive perfectionists. Every patient with social anxiety disorder should have a full understanding of "self".
6. Genetic factors: Studies have found that social anxiety disorders have family genetic tendencies, especially affecting female relatives.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS10994359	ANK3	CT	moderate anti-social anxiety disorder ability
RS12579350	ANO2	GG	strong anti-social anxiety disorder ability
RS1799971	OPRM1	AA	strong anti-social anxiety disorder ability

RS2710102	CNTNAP2	GG	strong anti-social anxiety disorder ability
RS2911968	MCPH1-AS1	CT	moderate anti-social anxiety disorder ability

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The anti-social anxiety disorder ability item uses 16 polymorphic loci on and between genes such as *ANK3*, *ANO2*, *GTF2I*, including rs10144552, rs12579350, rs1799971, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.
2. The test may not cover all genes or loci that affect the anti-social anxiety disorder ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your anti-social anxiety disorder ability at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian and East Asian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the anti-social anxiety disorder ability.

Spatial Memory Ability

Spatial memory ability refers to the ability to process and remember information about the surrounding environment and the relative position of objects.

My Spatial Memory Ability

Moderate

30.7% of We users are similar to me

Suggestions

How to improve spatial Memory ability?

1. Adopt a reasonable memory strategy: After the information enters the brain, it begins to be forgotten. According to the research of Ebbinghaus et al., the forgetting speed changes with time. Targeted recall based on the "forgetting curve" can improve our memory efficiency of verbal content. For example, the content that needs to be remembered can be improved by recalling the content one hour, one day and three days after memory.
2. Pay attention to sleep: The brain processes and consolidates memory content during sleep, so timely sleep is conducive to memory. At the same time, adequate sleep can improve the concentration and efficiency of memory.
3. Reduce dependence on navigation equipment: Establishing a complete spatial cognitive map through more observation of the surrounding environment is conducive to the improvement of spatial Memory ability and route planning ability.
4. Pay attention to developing good living habits: regular exercise, adequate nutrition, and higher blood glucose levels are conducive to improving spatial Memory ability. Intake of anti-aging foods with strong antioxidant capacity can also contribute to the improvement of spatial Memory ability.
5. Carry out targeted memory training: In daily life, consciously memorizing the relative relationship of things, or playing some games that depend on spatial Memory ability, will help improve spatial Memory ability.

Knowledge

What is spatial Memory ability?

Spatial memory refers to the ability to process and remember information about the surrounding environment and the relative position of objects. Such abilities mainly include the cognition and memory of the space and relative position of things, as well as the ability to plan different routes in a familiar environment. People with strong spatial memory ability and a general sense of direction are not easy to get lost, and it is unlikely that they can't remember where to put things in life. Specific occupations such as drivers, tour guides and architectural designers have relatively high requirements for spatial memory ability.

Spatial memory ability is related to the functions of hippocampus and medial entorhinal cortex. The hippocampus can store external spatial map information. At the same time, the grid cells found in the medial entorhinal cortex play a role in processing and integrating geometric relationships in the environment.

Factors affecting spatial memory ability

1. Diseases: The damage of the brain, especially the hippocampus, can lead to a decline in spatial memory ability. Other diseases such as Alzheimer's disease will lead to the atrophy of brain, including hippocampus, so getting lost is one of the typical symptoms of Alzheimer's disease. Others such as schizophrenia, strabismus, attention hyperactivity disorder and hypertension also increase the risk of decline in spatial memory ability.
2. Living habits: Studies have shown that people who regularly rely on GPS and other navigation devices will have a certain degree of decline in spatial memory ability. Long-term alcohol abuse can cause damage to the brain and further affect the ability of spatial memory ability.
3. Living environment: Hypoxia conditions will reduce the ability of spatial memory, so it is easy for ordinary people to go to high-altitude areas.
4. Education level: Some studies have shown that the longer you receive education, the slower the decline of spatial memory ability with age.
5. Gender: The spatial memory ability of men is slightly higher than that of women.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1799990	PRNP	AA	strong spatial memory ability
RS3824968	SORL1	AA	weak spatial memory ability

RS6265	BDNF	CT	moderate spatial memory ability
RS4941573	HTR2A	GG	strong spatial memory ability

Test details

Applicable situation

The spatial Memory ability item uses 4 polymorphic loci on and between genes such as *PRNP* and *SORL1*, including rs1799990, rs3824968, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian and Chinese, other populations are for reference only.
2. The test may not cover all genes or loci that affect the spatial Memory ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your spatial Memory ability at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the spatial Memory ability.

Positive Emotion

Positive emotions, also called positive moods, refers to the emotions accompanied by pleasant feelings due to internal and external stimuli and events meeting individual needs.

My Positive Emotion

Moderate

30.3% of We users are similar to me

Suggestions

How to promote positive emotion?

1. Record your positive emotions: Record the positive emotions you have experienced and are familiar with in your life. When you find a new positive emotion, pay attention first, and then record it when you can confirm it. When a certain number is recorded, look at the list and recall the activities, scenes, or characters when each emotion occurred.
 2. Pay attention to a positive emotion and strengthen it: Recall the situation when the positive emotion was generated, and try to add these scenes to daily life to strengthen this type of positive emotion.
 3. Create a treasure chest of positive emotions: Sometimes we forget the source of positive emotions, so the treasure chest of positive emotions becomes very meaningful. You can save items that generate positive emotions, such as photos, souvenirs, diaries, cards, etc. Collect them in your treasure chest, you can add, reduce and sort them out at any time. When you are happy or unhappy, open the treasure chests and let them promote your positive emotions.
-

Knowledge



Description of positive emotion

- Russell: "Positive emotions are the kind of good feelings you feel when you want to smile because of things going well."

- Fredrickson: "Positive emotion is a unique instant response to personally meaningful things, it is a temporary pleasure."
- Meng Zhaolan: "Positive emotions are related to the satisfaction of a certain need, usually accompanied by a pleasant subjective experience, and can improve people's enthusiasm and activity ability."

Ten forms of positive emotions most often felt by people

- Joy: When we feel joy, we are generally in a situation where everything develops in a predetermined way, and the result meets our expectations, even better than we expect. Joy is a brisk and bright feeling. When we feel joy, we feel relaxed all over, and even the things around us look more vivid and pleasing to the eye. We may want to join others in the conversation and are eager to try the next thing.
- Gratitude: When we realize what others have done to us, we experience gratitude. Sometimes the object of our gratitude is not necessarily a person, but it may also be a feeling that something brings us benefits. When we appreciate the preciousness of people, things, and objects, gratitude arises. Gratitude will give us the impulse to "want to give something in return." We will hope to do something good to those who have helped us, and we may also want to pass on the blessings we have received by helping others.
- Serenity: Serenity is a soft, low-key, relaxing version of joy, which usually occurs in an environment that feels safe and beautiful deep down. Serenity will make people feel that they need to be immersed in the present and savor the current feelings.
- Interest: Interest is the emotion we feel when we are attracted by some novel people, things, and objects in a safe environment. We will be led by interest to explore, try, dispel the mystery, and learn more.
- Hope: Compared with ordinary daily life, we are more likely to feel hope when things are unfavorable to us or there is uncertainty. The core of hope is our belief and desire that things can get better and those good things can happen. Because we believe that the future may be good, we are willing to continue to survive.
- Pride: Pride is a sense of satisfaction that arises because of what we are, what we have done, or what we have achieved. This is a very important emotion because it improves our self-esteem, makes us more confident in ourselves, and makes us stronger in the face of negative emotions.
- Amusement: Life is full of humorous stories or jokes, and those funny experiences will make you laugh and truly feel amused.
- Inspiration: When we see a perfect masterpiece or hear a great speech, we will feel inspired. It is a very strong driving force that can change the mood and let people pursue perfection.
- Awe: Awe is a feeling of surprise, fear, but love and admiration when faced with noble, fierce, and mysterious things. It may occur in the aesthetics of nature, society and art.
- Love: Love is the most complicated emotion. When we were just born babies, we first experienced such a wide range of strong emotions. The purest form is unconditional love, where you can only give without asking for anything in return.

The impact of positive emotion

- Positive emotions bring openness and enhance our creativity.
- Positive emotions bring positive interpersonal interaction experiences.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS322931	intergenic	AG	moderate positive emotion

Test details

Applicable situation

The positive emotion item uses one polymorphic locus between genes: rs322931.

Notification

1. The basis of the test is mainly from the research of African American, other populations are for reference only.
2. The test may not cover all genes or loci that affect the positive emotion.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This test only evaluates your positive emotion at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of African American.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the positive emotion.

Stress Handling Ability

Stress handling, also called psychological endurance, is an ability to withstand and regulate the psychological pressure and negative emotions caused by adversity. It is mainly the degree of adaptability, tolerance, endurance, and ability to overcome adversity.

My Stress Handling Ability

Moderate

23.5% of We users are similar to me

Suggestions

How to improve stress handling ability?

1. Make efforts that match your abilities: if you don't work hard enough, you will worry that you can actually do better and become anxious.
2. Be confident: you have to believe that you can be a good person.
3. Don't blame yourself: too much responsibility will put too much pressure on yourself. Sometimes an excuse can also be effective in resisting stress.
4. Physical exercise: exercise can decompress, improve one's physical function, enhance one's physical strength and then enhance psychological resistance to stress.
5. Be positive: When you're down, a lot of small pressure will suddenly gather on you and make you tired.
6. Don't pay too much attention to gains and losses: When you care about gains and losses, you will definitely have invisible or tangible pressure, and this pressure will often make you lose more than gain.

Knowledge



Learn about stress

"Pressure is harmful when you think it is harmful". What is terrible is not stress but avoiding stress which leads to a result harmful to ourselves. We can make stress beneficial by facing and dealing with stress. Stress is not all a scourge for us, and there are also good aspects, mainly in three:

- Stress can help us better cope with stressful events: when facing stress, our heartbeat will speed up and our breathing will deepen, so there will be more blood pressure and energy into the brain and body, so that we have more strong physical and mental power to deal with stressful events.
- Stress can increase our social frequency: our pro-social attributes will be activated under stressful situations. For example, when we are uncomfortable, we will find friends and family to complain, so we will have more social frequency.
- Stress can improve our learning ability: Appropriately experiencing stressful events can make the part of the brain that controls thinking and self-control more developed, easier to solve problems, and less prone to anxiety.

How to face stress

- Talk to someone: Don't deal with all the pressure by yourself, you can discuss with your trusted relatives and friends, and these friends should not add extra pressure to you. Even if the current situation cannot be changed, talking to others can ease tension.
- Social support: If you don't have the courage to talk about it for the time being, spend time with your relatives and friends, you don't need to say anything, the feeling of staying together can also relieve the pressure in your heart.
- Take action: Take up the courage to take on and change, no matter how small the impact is. Try to change the current pressure situation, or at least part of it. A small step forward can also make you more confident and reduce stress.
- Try to understand your body: If you know how your body responds to stress, you can use actions to relieve stress, such as yoga, meditation, and sports.
- Don't over-stress: If you feel overwhelmed, choose to give up some unnecessary pressure. Learn to say "no" without guilt, and to refuse less important things so that you won't breathe.
- Learn to be happy: Pay attention to the things that happen to you to make you feel happy, even if those things are so small, record those feelings, and you will find that life is better than you think.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1360780	FKBP5	CC	strong stress handling ability
RS3800373	FKBP5	AA	strong stress handling ability
RS1109089	intergenic	CC	weak stress handling ability
RS1884641	intergenic	AA	strong stress handling ability
RS2074997	intergenic	CC	weak stress handling ability

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The handle stress item uses the 8 polymorphic loci on and between *FKBP5* gene, *RHEB* gene and *CYB561A3* gene, such as rs3800373, rs1360780, rs1109089, rs1884641 and rs2074997.

Notification

1. The basis of the test is mainly from the research of Caucasian and African American, other populations are for reference only.
2. The test may not cover all genes or loci that affect handle stress ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This study only evaluates your handle stress ability at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian and African American.

Limit of detection

Limited by the current technology and the level of scientific cognition. The test may not cover all genes or loci that related to handle stress ability.

Anti-Emotional Eating Ability

Anti-emotional eating ability refers to the ability to resist overeating due to emotional reasons.

My Anti-Emotional Eating Ability

Strong

20.1% of We users are similar to me

Suggestions

How to overcome emotional eating?

1. Find the cause of emotional eating: If you want to stop emotional eating, you have to face it, and analyze the incentives for your to start eating. If this pattern is relatively clear, it is relatively easy to correct it.
2. Distract: Take your mind off eating. When you want to eat, try a 10-minute distraction, such as getting up and doing something else immediately (cleaning the house/exercising/washing your face and applying a face mask) or talking to a friend on the phone, to distract your mind from eating.
3. Prepare healthy food: prepare healthy food at hand, such as instant oatmeal, instant tomato soup, unsweetened whole wheat bread and other relatively healthy and full-feeling foods. When you want to eat something out of psychological need, take healthy food to meet the urgent need for emotional eating.
4. Cultivate the habit of coping with emotional problems: you can try some ways to substitute foods, such as taking a hot bath or getting a massage when you are stressed; if you feel depressed and lonely, try getting a pet or meeting with friends or online regularly; or develop a certain sport into your hobby, such as yoga.
5. Develop healthy living habits: go to bed early and wake up early and do not stay up late. Only when the quality of sleep is high, the body and mind are relaxed and the rest is adequate, can we better deal with the difficulties in life and cope with the changing environment; regular and moderate exercise. Regular stress reduction, relaxation, and active social activities at the same time can help improve emotional eating.

Knowledge



What is emotional eating?

Emotional eating is to satisfy emotions, not physical hunger. Stress will increase the level of cortisol in the human body, and high-carb, high-sugar and high-fat foods can reduce stress hormones. Eating high-calorie foods in times of depression and anxiety will reduce psychological stress and negative emotions. However, the pleasure of food intake can only last for 1 minute, and then the feelings of regret, shame, and self-blame occur and obesity will also produce. Therefore, emotional eating cannot solve the problems and emotions in people's real life, but will increase the emotional instability of people. If it is not controlled, emotional eating may develop into bulimia or bulimia nervosa.



Are you an emotional eater?

- Do you eat more when you are nervous?
- Do you eat when you are not hungry or already full?
- When you feel sad, angry, bored, or anxious, do you use food to soothe yourself, restore peace, and eat to make yourself feel better?
- Do you reward yourself with food?
- Have you ever eaten it too much?
- Do you feel powerless or out of control with food?



The difference between emotional hunger and physical hunger

1. Emotional hunger comes suddenly: Emotional hunger comes suddenly, it makes you feel irresistible and very urgent in an instant; physical hunger comes gradually, the desire to eat is not very urgent, and it does not require immediate satisfaction (Unless you haven't eaten for a long time).
2. Emotional hunger craves for specific foods for comfort: Emotional hunger craves for specific foods for comfort; when you are physically hungry, almost all foods sound delicious-including healthy foods such as vegetables.
3. Emotional hunger often leads to overeating: before you realize it, you have eaten a large bag of potato chips or a whole box of ice cream, but haven't really noticed anything or fully enjoyed the food. And when you eat because of physical hunger, you are often more aware of what you are doing.
4. Emotional hunger is not satisfied by eating full: you just want to eat more and more, often not stopping until you feel uncomfortable. On the other hand, physical hunger does not cause you to eat, and when your stomach is full, you feel satisfied.
5. Emotional hunger often leads to regret, guilt and shame: Emotional hunger often leads to regret, guilt and shame. When you eat something because of physical hunger, you are unlikely to feel guilty or ashamed, because you are just giving the body what is necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1514175	FPGT-TNNI3K	AG	moderate anti-emotional eating ability
RS1558902	FTO	TT	strong anti-emotional eating ability
RS2287019	QPCTL	CC	strong anti-emotional eating ability
RS3810291	ZC3H4	GG	weak anti-emotional eating ability
RS3817334	MTCH2	CC	strong anti-emotional eating ability

Test details

Applicable situation

The anti-emotional eating ability item uses the 5 polymorphic loci in genes such as *FPGT-TNNI3K*: rs1514175, rs1558902, rs2287019, rs3810291, rs3817334.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect anti-emotional eating ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This study only evaluates your anti-emotional eating ability at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition. The test may not cover all genes or loci that related to anti-emotional eating ability.

Conscientiousness

Conscientiousness is one of the five personality traits, it refers to the tendency of individual's behavior is organized and thoughtful. It generally includes traits such as competence, fairness, orderliness, due diligence, self-discipline, prudence and restraint and etc.

My Conscientiousness

Weak

10.9% of We users are similar to me

Knowledge



What is conscientiousness?

Conscientiousness is one of the five main factors of personality structure. It refers to the tendency of individuals to behave in a methodical and thoughtful manner. People with high conscientious scores are planned, organized, and persevering; people with low scores tend to be careless and lack planning and organization.



Conscientious behavior

Conscientiousness can generally be divided into five sub-dimensions:

- Responsibility: The individual takes responsibility for the task and others seriously, and keeps his own promises.
 - High score performance: a sense of responsibility, responsible for doing things.
 - Low score performance: shirk responsibility and avoid punishment.
- Self-control: The individual's ability to restrain himself and persistence from beginning to end.
 - High score performance: self-control, perseverance
 - Low score performance: reckless, sloppy
- Deliberation: The mental state of an individual before taking specific actions.
 - High score performance: cautious, careful
 - Low score performance: reckless, sloppy
- Orderliness: The order, orderliness and logic of individual handling of affairs and work.
 - High score performance: neat and organized
 - Low score performance: chaotic, sloppy

- Industriousness: The degree of individual work and study effort and the enterprising spirit shown in order to achieve the goal.
 - High score performance: diligent and assiduous
 - Low score performance: laziness

What are the effects of conscientiousness?

- Career satisfaction

Individuals with high conscientiousness score have strong achievement motivation, sense of responsibility, earnest and devotion to work, and are willing to continuously improve their own quality and work performance through efforts. Therefore, they are likely to get support from the organization and attention from leaders, and get more salary increases and promotion opportunities. Achievements at work encourage individuals to make positive evaluations of their careers, thus improving career satisfaction.

- Procrastination

Studies have shown that a low level of conscientiousness is closely related to procrastination. This behavior refers to postponing a planned task to a later time. Procrastinators usually feel anxious about starting or completing any task or decision, and use procrastination as a mechanism to cope with anxiety.

- Unemployment response

Although conscientiousness is generally regarded as a positive trait, recent research has shown that it may be disadvantageous in certain situations. A four-year study of 9,570 people showed that once a person with high conscientiousness becomes an unemployed, he will suffer more than twice as much pain as a person with low conscientiousness. The reason may be that conscientious people have different attributions for why they are unemployed, or have a stronger reaction after experiencing failure.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2576037	KATNAL2	TT	weak conscientiousness
RS13070781	intergenic	AA	weak conscientiousness

Test details

Applicable situation

The conscientiousness item uses the 2 polymorphic loci in the *KATNAL2*: rs13070781, rs2576037.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect conscientiousness.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This study only evaluates your conscientiousness at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition. The test may not cover all genes or loci that related to conscientiousness.

Openness

Openness is one of the five personality traits, it refers to the degree of openness to experience various activities. It generally includes traits such as active imagination, aesthetic sensitivity, attentiveness to inner feelings, and intellectual curiosity and etc.

My Openness

Moderate

25.3% of We users are similar to me

Knowledge



What is openness?

Openness is not only an openness in an interpersonal sense, but also refers to an open and exploratory attitude towards experience. It is one of the five main factors of personality structure. People with a high degree of openness are more curious, interested broadly, creative, imaginative, unconventional, and independent thinking; people with low scores tend to be more traditional and prefer familiar things to new things.



Openness performance

Openness can generally be divided into five sub-dimensions:

- Imagination: The level of an individual full of fantasy and imagination.
 - High score performance: Imaginative
 - Low score performance: lack of imagination
- Aesthetics: The degree of individual sensitivity and love for art and beauty.
 - High score performance: artistic
 - Low score performance: generally not sensitive to art
- Curiousness: The individual's attitude towards the unknown.
 - High score performance: broad interest, strong curiosity
 - Low score performance: little interest, no curiosity
- Adventure: Individual differences in willingness to try risky activities.
 - High score performance: risky

- Low score performance: conservative
- Attitude: the individual's attitude towards new things, new ideas, and weird ideas.
 - High score performance: open and accept new things calmly
 - Low score performance: conservative, not accepting new things

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS677035	intergenic	CC	strong openness
RS11685318	intergenic	CC	strong openness
RS10966354	intergenic	GT	moderate openness
RS8034376	intergenic	GG	strong openness

Test details

Applicable situation

The openness item uses the 4 polymorphic loci in genes such as rs11685318.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect openness.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This study only evaluates your openness at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition. The test may not cover all genes or loci that related to openness.

Episodic Memory Ability

Episodic memory ability refers to the ability to form unique memories of specific experienced by oneself.

My Episodic Memory Ability

Moderate

35.8% of We users are similar to me

Suggestions

How to improve episodic Memory ability?

1. Adopt a reasonable memory strategy: For specific scene memory, you can try a camera-like principle to help memory by taking snapshots in your mind. Regular review, such as writing a diary, can also help improve episodic memory.
2. Pay attention to sleep: The brain processes and consolidates memory content during sleep, so timely sleep is conducive to memory. At the same time, adequate sleep can improve the concentration of memory and improve memory efficiency.
3. Exercise your brain regularly: such as often trying to remember someone's clothes, the surrounding environment, etc. when meeting him.
4. Develop good living habits: Both abstinence from alcohol and exercise can improve episodic memory. Higher intensity exercise will stimulate the hippocampus, which is an important area related to episodic Memory ability.

Knowledge



What is episodic memory ability?

The episodic Memory ability refers to the ability to form a unique memory of a specific event that one has experienced, so even for the same event, different people have different episodic memories. The scene of going to the playground for the first time, the scene of a birthday party, the feeling of the first confession, etc., are all episodic memories. It should be noted that episodic memory is slightly

different from another concept of autobiographical memory, and the former is a part of the latter. Autobiographical memory can include parts of non-episodic memory, such as the date and place of birth, which have no episodic memory.

Characteristics of episodic memory ability

The formation of episodic memory includes different steps. First, it is necessary to collect and process the information of the experienced scene, and then consolidate the information in the brain, which is to transform these events into stable long-term memory. The hippocampus plays an important role in this process. Finally these memories can be recalled when needed. Our episodic memory is often triggered by something, for example, an image, a sentence or even a smell can evoke specific episodic memory content.

Factors affecting episodic memory ability

1. Disease: Brain trauma, hydrocephalus, and certain diseases such as Alzheimer's disease can reduce episodic Memory ability.
2. Living habits: Long-term alcohol abuse can cause damage to the brain and further affect episodic Memory ability.
3. Dietary habits: Metabolic state such as vitamin B1 deficiency will lead to a decline in episodic memory.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS17070145	WWC1	CT	moderate episodic memory ability
RS7594645	FASTKD2	AA	weak episodic memory ability

Test details

Applicable situation

The episodic Memory ability item uses the 2 polymorphic loci in genes such as *WWC1*: rs17070145, rs7594645.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

2. The test may not cover all genes or loci that affect episodic Memory abilities.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This study only evaluates your episodic Memory abilities at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition. The test may not cover all genes or loci that related to episodic Memory ability.

Verbal Memory Ability

Verbal memory ability refers to the ability to remember various forms of language, such as the ability to remember words or sentences that they are heard or seen.

My Verbal Memory Ability

Weak

7.5% of We users are similar to me

Suggestions

How to improve verbal Memory ability?

1. Adopt a reasonable memory strategy: after the information enters the brain, it begins to be forgotten. According to the research of Ebbinghaus et al., the forgetting speed changes with time. Targeted recall based on the "forgetting curve" can improve our memory efficiency of verbal content. For example, recall the content that needs to be memorized in one hour, one day, and three days after the memory can improve the memorized content.
2. Memorizing on the basis of understanding: Memorizing meaningless words requires more repetitions than memorizing complete poems. We will be more efficient to memorize on the basis of understanding. At the same time, for knowledge content, you can also ask yourself to answer, or explain to others and other ways to improve the understanding of the content, in order to facilitate memory.
3. Pay attention to sleep: The brain processes and consolidates memory content during sleep, so timely sleep is conducive to memory. At the same time, adequate sleep can improve the concentration of memory and improve memory efficiency.
4. Use other levels of memory: For example, our ability to remember images is stronger than words. Therefore, it is easier to abstract information into graphics and images in the memory process than to memorize words directly.
5. Use music memory: Studies have shown that lyrics combined with music are easier to remember than other textual content. For example, people often reduce the difficulty of memory by compiling the content into ballads and mantras, such as alphabet songs when learning English, and multiplication mantras when learning mathematics.

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6. Develop good living habits: Both abstinence from alcohol and exercise can improve speech and memory skills.
-

Knowledge

What is verbal memory ability?

Verbal memory refers to the ability to remember different forms of words, short sentences, paragraphs, etc. It is all related to verbal memory in learning foreign languages, reciting texts, and recalling other people's speeches. People with strong verbal memory skills generally have a deep memory of text content in their studies and work, and can easily recall the content of the teacher's lectures or the details of discussions with colleagues. For most people, the verbal memory ability is controlled by the left brain. Diseases and trauma of the left brain may cause the decline of verbal memory ability. At the same time, some mental illnesses will also affect verbal memory to a certain extent, such as attention deficit hyperactivity disorder, depression and post-traumatic stress disorder.

Characteristics of verbal memory ability

Verbal memory ability mainly includes the content of the memory, the duration of the memory and the memory on the basis of understanding. Generally speaking, as time goes by, we will lose more and more memory of specific speech content, but if we have a deeper understanding of the content, we can remember more and longer content. On the other hand, memorizing on the basis of understanding sometimes makes us have a wrong memory of paired words. For example, if the word "house" appears in a memory test, people often think of the word "building". In addition, we will have better memories of words that represent actual concepts, such as "moon", "water", "stone", etc., than abstract words such as "evil", "deception", and "sacred".

Factors affecting verbal memory ability

1. Diseases: Damage to the brain, especially the left brain, can lead to a decline in verbal memory, and other mental illnesses can also have a negative impact on verbal memory.
2. Living habits: Long-term alcoholism can cause damage to the brain and further affect verbal memory.
3. Music: Music training can stimulate the development of the auditory cortex and related brain regions. Studies have shown that the earlier and longer you start to receive music training, the greater the improvement of verbal memory.
4. Hormone levels: Whether for men or women, the hormone levels in the body will have a certain impact on verbal memory. At the same time, women are generally slightly better than men in verbal Memory ability.

5. Education level: Studies have shown that the longer you receive education, the slower your verbal Memory ability declines with age.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS17070145	WWC1	CT	moderate verbal memory ability
RS4420638	APOC1	AA	weak verbal memory ability
RS6265	BDNF	CT	moderate verbal memory ability
RS6439886	CLSTN2	AA	weak verbal memory ability
RS7594645	FASTKD2	AA	weak verbal memory ability

For display consideration, only some genetic details are displayed.

Test details

 Applicable situation

The verbal Memory ability item uses the 7 polymorphic loci on and between *WWC1* gene and *BDNF* gene, including rs17070145, rs283815.

 Notification

- 1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
- 2. The test may not cover all genes or loci that affect verbal Memory abilities.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This study only evaluates your verbal Memory ability at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition. The test may not cover all genes or loci that related to verbal Memory ability.

Anti-Compulsive Hoarding

Compulsive hoarding refers to the behavior of over-obtaining and not being able to discard items that are useless or valueless. People with strong ability to anti-compulsive hoarding are not easily to have this behavior tendency.

My Anti-Compulsive Hoarding

Moderate

38.2% of We users are similar to me

Suggestions

How to improve compulsive hoarding?

1. Record the consumption cycle of daily consumables: You can make a table, such as how long it takes to use up a bottle of shampoo. Generally you can only store items for three months or half a year. In this way, you have an overall grasp of the cycle of consumption of daily necessities in your own home.
 2. Fix the storage location of spare supplies: Blind shopping is one of the reasons why it is easy to hoard. Before going to the supermarket, you can check your own stocks and make a list and only buy the goods on the list to avoid excessive hoarding of items.
 3. Determine whether it is a must-buy item before shopping: you can ask before shopping and then decide whether to buy it. First, is this item useful to me? Second, is there a place to store it? Three, are there alternatives?
 4. Decline unneeded gifts and gifts: Direct refusal is the best way to solve the problem. You can politely refuse the gifts given by others if they are not your heart's desire. It is forbidden to take these items home from the source.
 5. Use shared resources: Instead of buying a treadmill or exercise machine, go to a fitness center when you need to exercise. Instead of buying books, borrow books from the library or read e-books when you need them.
-

Knowledge

What is hoarding

Hoarding, the repetitive collection of excessive quantities of poorly useable items of little or no value with failure to discard these items over time. Severe hoarding behavior will be regarded as a hoarding disorder, affecting the lives of oneself and others, and even impairing normal social functions.

Performance of hoarding

- Difficulty in discarding these items, no matter what other people think of their value.
- There is a strong desire to store items, and it is painful to discard them. -The accumulation of a large number of items makes the life or workplace messy and even makes it impossible for people to live and work in it.
- Severe hoarding behavior may affect and interfere with the oneself and other's daily life and work, and even cause major misfortune, such as causing fires and falls.

Influencing factors of hoarding

- Gender: Relevant epidemiological surveys have found that men's hoarding is more common than women's (this may be due to that female are more willing to participate in such research voluntarily). From the characteristics of hoarding behavior, female hoarders have more frequent over-purchasing behaviors than male hoarders, but male hoarders have more behaviors of over-acquiring free items than female hoarders.
- Age: Older people have more serious hoarding behavior problems than younger people and children. Other studies have shown that hoarding behavior's severity increases with age if it starts at an early age which usually manifests in childhood or adolescence.
- Traumatic events: Compared with patients with obsessive-compulsive disorder and normal subjects, people with hoarding report more traumatic events, and hoarding behaviors occur at the same time as the stressful life events they experience. This means that traumatic or stressful life events may be closely related to the appearance and development of hoarding behavior.

The difference between hoarding and collecting

-Collectors are usually proud of their collections and are happy to show them to others. Hoarders often feel embarrassed about their hoardings and do not want others to see them. -Collectors organize and categorize their collections reasonably; however, people with hoarding disorders often fail to effectively classify the hoardings and mess up their homes. -Collectors will make a reasonable budget for their collections; and those with severe hoarding may buy them in debt.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2388436	intergenic	GG	weak anti-compulsive hoarding
RS3747767	intergenic	CC	strong anti-compulsive hoarding

Test details

Applicable situation

The anti-compulsive hoarding item uses the 2 polymorphic loci in genes such as rs2388436.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect anti-compulsive hoarding.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's psychological traits are the common result of the interaction of genes, environment, and living habits. This study only evaluates your anti-compulsive hoarding at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition. The test may not cover all genes or loci that related to anti-compulsive hoarding.



Life Guidance

18 Reports

Effect of Smoking

Smoking x Colorectal Cancer

Moderate elevating risk



Smoking x Stomach Cancer

Slightly high elevating risk



Smoking x Esophagus Cancer

Slightly low elevating risk



Smoking x Breast Cancer

Slightly low elevating risk



Smoking x Bladder Cancer

Moderate elevating risk



Smoking x CHD

Moderate elevating risk



Smoking x Hypertension

Moderate elevating risk



Smoking x Lung Cancer

Slightly low elevating risk



Effect of Drinking

Drinking x Esophagus Cancer

Slightly low elevating risk



Drinking x Breast Cancer

Slightly low elevating risk



Drinking x Bladder Cancer

Moderate elevating risk



Drinking x Colorectal Cancer

Slightly high elevating risk



Drinking x CHD

Moderate elevating risk



Drinking x Hypertension

Moderate elevating risk



Drinking x Lung Cancer

Slightly high elevating risk



Effect of Physical Activity

Physical Activity x T2D

Moderate protective role



Physical Activity x Hypertension

Moderate protective role



Physical Activity x CHD

Slightly weak protective role



Smoking x Colorectal Cancer

In general, smoking can increase the risk of colorectal cancer. And to a certain extent, the increase of this risk is affected by genes.

My Smoking x Colorectal Cancer

Moderate elevating risk

Quit smoking knowledge

Tobacco is the largest controllable hazard affecting human health. Smoking cessation has health benefits, including a lower risk of cancer and death from cardiovascular disease. In addition to smoking cessation policies, environmental and social support, and personal determination and perseverance, appropriate smoking cessation methods should also be adopted, or a combination of multiple methods should be used. Common methods include:

- Quit smoking through willpower: Smoking cessation through willpower alone without any other supplementary means, but the success rate is very low, less than 3%.
- Drug intervention: Smoking cessation drugs can be effective in helping smokers quit their addiction. Different drugs have different efficacy, but all of them are significantly better than "Quit smoking through willpower".
- Behavioral intervention: Different levels of behavioral intervention, including speech intervention and telephone follow-up. A combination of drug interventions can greatly improve the success rate.
- E-cigarettes: E-cigarettes can be used as an alternative to tobacco, but their safety needs to be verified by further studies.
- TCM (Traditional Chinese Medicine) smoking cessation: Some studies have shown that TCM acupuncture can help smokers quit smoking by regulating endorphin levels, but this method has a high relapse rate.
- Other methods: such as Information-based smoking cessation, Aversion therapy, Hypnotherapy, etc., can be used as a second choice after drugs, behavioral intervention and other methods fail.

Knowledge



Smoking and colorectal cancer

Smoking is an important risk factor for the onset of colorectal cancer, and it has a certain dose-effect relationship with the number of years and total amount of smoking. The "Consensus on the

Prevention of Colorectal Cancer in China (2016)" pointed out: Compared with non-smokers, the relative risk of colorectal cancer among people with a history of smoking is 1.18, and the incidence of colorectal cancer among smokers is lower than that of non-smokers per 100,000 people. The number of cases is 10.8 more cases; each increase in smoking 10 cigarettes per day can increase the risk of colorectal cancer by 7.8%.

Different genotypes interact with smoking, leading to different risks of colorectal cancer. For example, the locus of rs1799931 on the N-acetyltransferase 2 (NAT2) gene can increase the risk of colorectal cancer from smoking. Smokers with the AG or AA genotype are more likely to develop colorectal cancer than smokers with the GG genotype.



Onset time of colorectal cancer

Colorectal cancer mostly occurs between the ages of 50 and 70.



Prevalence of colorectal cancer

The incidence and mortality of colorectal cancer in china are higher than the world average and the level of developing countries, and lower than the level of developed countries. The incidence of colorectal cancer in china is 28.08/100,000, accounting for 10.56% of the total incidence of all malignant tumors.



Typical symptoms of colorectal cancer

Early colorectal cancer may have no obvious symptoms, and the following symptoms do not appear until the disease has progressed to a certain extent: changes in bowel habits; changes in stool characteristics (thinning, bloody stools, mucous stools, etc.); abdominal pain or abdominal discomfort; abdominal masses; intestinal obstruction; anemia and Systemic symptoms: such as weight loss, fatigue, and low-grade fever. Its morbidity and fatality rate are second only to gastric cancer, esophageal cancer and primary liver cancer in malignant tumors of the digestive system.



Treatment of colorectal cancer

1. Surgical treatment: The current treatment of colorectal cancer generally adopts the principle of comprehensive treatment based on surgery. The treatment plan is determined according to the patient's general condition and the functional status of various organs, tumor location, clinical stage, pathological type and biological behavior. The basic principles of colorectal cancer surgery are consistent with the basic principles of tumor surgery. In summary, they are the three principles of radical cure, safety, and functionality. Among them, when the tumor can be removed, the principle of radical treatment is first required, and the principle of radical treatment is considered second. When it comes to safety, functional principles are considered as much as possible at the end.
2. Chemotherapy: Adjuvant chemotherapy is generally required after colon cancer surgery. Colorectal cancer patients may have micrometastases or tumor cells remaining after surgery. Adjuvant chemotherapy is used to kill these metastases or tumor cells as much as possible. However, not all patients need and are suitable for chemotherapy. For example, early stage (stage

l) patients do not need chemotherapy. In addition, for patients diagnosed with large tumors or local lymph node metastases, in order to improve the cure rate of surgery and reduce the scope of resection, doctors usually advise patients to receive radiotherapy and chemotherapy first, which can shrink the tumor or reduce the stage, which is conducive to the operation.

3. Radiotherapy: generally as an adjuvant therapy for surgery, it is treated before or after surgery.

Other influencing factors

Controllable factors

1. Poor diet: High-fat diet, excessive intake of animal protein, especially excessive red meat, and low-fiber diet are the main risk factors for colorectal cancer.
2. Living habits factors: Bad lifestyle habits, including long-term drinking and lack of exercise, can increase the risk of colorectal cancer.
3. Overweight and obesity: In recent years, it has been considered that overweight and obesity are risk factors for colorectal cancer.

Uncontrollable factors

1. Age: Middle-aged and elderly people are more likely to develop colorectal cancer.
2. Disease history: Studies have shown that the history of chronic diarrhea, mucus blood in the stool, colorectal polyps, constipation and appendicitis and other diseases and symptoms are risk factors for colorectal cancer.
3. Family history: People with a family history of colorectal cancer have a higher prevalence.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1799931	NAT2	GG	moderate risk caused by smoking x colorectal cancer
RS1800440	CYP1B1	TT	moderate risk caused by smoking x colorectal cancer
RS1800734	MLH1	GG	moderate risk caused by smoking x colorectal cancer
RS6478972	intergenic	AG	slightly low risk caused by smoking x colorectal cancer

RS1042028

intergenic

CC

moderate risk caused by smoking x colorectal cancer

Risk calculation

The risk value of this test is calculated based on published academic literatures on the risk of colorectal cancer from smoking. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences in the risk of colorectal cancer between smokers and non-smokers. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can show the changes in the risk of colorectal cancer for users if they smoke relative to those who do not smoke.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The risk of colorectal cancer from smoking item uses 5 polymorphic loci on genes such as *NAT2*, *CYP1B1*, *MLH1* and other genes. The test results indicate whether the user has a higher risk of colorectal cancer from smoking in the combination of genotypes at the 5 polymorphic loci tested compared to all users. It does not represent the user's real risk of colorectal cancer from smoking. The basis of the test is mainly from the research of American, Canadian, and Chinese, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that can affect the risk of colorectal cancer from smoking.
2. The test result has no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.

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2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only analyzes the impact of smoking on the risk of colorectal cancer from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
 3. If you have a family history of colorectal cancer, or you think that you are likely to suffer from colorectal cancer, it is recommended that you consult an authoritative health professional or clinician for help.
 4. Smoking as a single factor can significantly increase the risk of cancer and cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, smoking should be quit as soon as possible to reduce the risk of disease.

Test content

The test includes 5 polymorphisms related to the risk of colorectal cancer from smoking, including rs6478972, rs1042028, and rs1799931.

Population samples

The basis of the test is mainly from the research of American, Canadian, and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to smoking x colorectal cancer.

Drinking x Esophagus Cancer

In general, alcohol consumption can increase the risk of esophageal cancer. And to a certain extent, the increase of this risk is affected by genes.

My Drinking x Esophagus Cancer

Slightly low elevating risk

Quit drinking knowledge

Alcohol is a recognized addictive psychoactive substance. Excessive drinking and chronic drinking can cause harmful behavior problems, physical health problems and mental and psychological problems. Quit drinking is good for health. It can not only reduce the incidence of cancer, but also reduce the risk of death from cardiovascular disease. Quit drinking is not just a one-man battle, it also requires psychological, spiritual and behavioral support. Common methods include:

- Quit drinking through willpower: quit drinking only depends on willpower without other auxiliary means, but the success rate is low and the possibility of drinking again is high.
- Drug intervention: Quit drinking drugs are effective in helping drinkers to abstain from alcohol. Different drugs have different effects, but all are significantly better than "quit drinking through willpower".
- Physical intervention: Physiotherapy can control the bad behavior of the drinker during the onset of alcohol addiction.
- Psychological intervention: Psychological intervention emphasizes the attention to the psychological level of drinkers, which can meet the needs of drinkers at different stages of quit drinking, helps them establish a healthy attitude and way of life, and helps them to maintain the effect of quit drinking for a long time.
- Other methods: such as receiving Health education and seeking Social support.

Knowledge



Drinking and esophageal cancer

Single-factor and multi-factor analysis showed that alcohol consumption is significantly related to the occurrence of esophageal cancer. The study found that the risk of esophageal cancer among drinkers is 2.46 compared with non-drinkers; the risk of esophageal cancer increases with the length

of drinking and the increase in alcohol consumption; drinking is the main risk factor for esophageal cancer in men, and it has a significant dose reaction relationship.

Different genotypes interact with alcohol, leading to different risks of esophageal cancer. For example, the locus of rs1229984 on the alcohol dehydrogenase 1B (ADH1B) gene can increase the risk of esophageal cancer from drinking. Drinkers with the CC genotype are more likely to develop esophageal cancer than drinkers with the CT or TT genotype.

Onset time of esophageal cancer

The age of onset is mostly over 40 years old.

The prevalence of esophageal cancer

The incidence of esophageal cancer varies greatly among countries. China is a high incidence area of esophageal cancer. Henan has the highest incidence in the world.

Typical symptoms of esophageal cancer

1. Early symptoms: mainly post-sternal burning sensation, acupuncture-like or traction-like pain, slow passage of eating, and a sense of retention or mild choking. The early symptoms are mild and severe, and may even be asymptomatic.
2. Middle and late symptoms: Progressive dysphagia (first hard to swallow dry food, then semi-liquid food, and finally hard to swallow water and saliva), food reflux, painful swallowing, accompanied by weight loss, lymphadenopathy, Hoarse voice etc.

Treatment of esophageal cancer

Surgery, radiotherapy, and chemotherapy.

Other influencing factors

Controllable factors

1. Nitrosamine: Eating salted fish, pickled vegetables and other high nitrite foods will increase the risk of esophageal cancer.
2. Fungi: A variety of fungi can be isolated from the upper gastrointestinal tract of patients with esophageal cancer or from resected esophageal cancer specimens. Investigations have shown that eating moldy food has a certain relationship with the onset of esophageal cancer.
3. Nutritional deficiency: Epidemiology shows that the lack of a variety of vitamins (such as vitamin A, B2, C) in the diet or the lack of certain trace elements (including molybdenum, zinc, selenium, etc.) are related to the onset of esophageal cancer.
4. Smoking: Long-term smoking can induce esophageal cancer.

5. Eating habits: Long term consumption of rough and hard food or eating too fast or too hot is easy to cause mechanical damage to esophageal mucosa. Repeated stimulation can lead to mucosal epithelial hyperplasia and eventually cancer.

 Uncontrollable factors

- 1. Family history: The occurrence of esophageal cancer has obvious family clustering phenomenon.
- 2. Age: The age of onset is more than 40 years old.
- 3. Gender: Males predominantly.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1042026	ADH1B	CT	slightly low risk caused by drinking x esophagus cancer
RS1051740	EPHX1	CC	slightly high risk caused by drinking x esophagus cancer
RS1229984	ADH1B	CT	moderate risk caused by drinking x esophagus cancer
RS1614972	ADH1C	CT	slightly low risk caused by drinking x esophagus cancer
RS1789903	ADH1C	CG	slightly low risk caused by drinking x esophagus cancer

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures on the risk of esophageal cancer from drinking alcohol. These literatures have found some polymorphic loci related to the risk of the disease by studying the genotype differences in the risk of esophageal cancer between drinking and non-drinking populations. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can show the changes in the risk of esophageal cancer for users if they drink alcohol relative to those who do not drink alcohol.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-

genetic factors are not taken into account.

Test details

Applicable situation

The risk of esophageal cancer from drinking alcohol item uses 11 polymorphic loci on genes such as *ALDH2*, *ADH4*, *ADH1C* and other genes. The test results indicate whether the user has a higher possibility of the risk of esophageal cancer from drinking alcohol in the combination of genotypes at the 5 polymorphic loci tested compared to all users. It does not represent the user's real risk of esophageal cancer from drinking alcohol. The basis of the test is mainly from the research of East Asian, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that can affect the risk of esophageal cancer from drinking alcohol.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only analyze the impact of drinking on the risk of esophageal cancer from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of esophageal cancer, or you think that you are likely to suffer from esophageal cancer, it is recommended that you consult an authoritative health professional or clinician for help.
4. Drinking as a single factor can significantly increase the risk of cancer and cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, drinking should be quit as soon as possible to reduce the risk of disease.

Test content

The test includes 11 polymorphic loci related to the risk of esophageal cancer from drinking alcohol, including rs4646903, rs671, and rs3805322.



Population samples

The basis of the test is mainly from the research of East Asian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the risk of esophageal cancer from drinking alcohol.

Drinking x Breast Cancer

In general, alcohol consumption can increase the risk of breast cancer. And to a certain extent, the increase of this risk is affected by genes.

My Drinking x Breast Cancer

Slightly low elevating risk

Quit drinking knowledge

Alcohol is a recognized addictive psychoactive substance. Excessive drinking and chronic drinking can cause harmful behavior problems, physical health problems and mental and psychological problems. Quit drinking is good for health. It can not only reduce the incidence of cancer, but also reduce the risk of death from cardiovascular disease. Quit drinking is not just a one-man battle, it also requires psychological, spiritual and behavioral support. Common methods include:

- Quit drinking through willpower: quit drinking only depends on willpower without other auxiliary means, but the success rate is low and the possibility of drinking again is high.
- Drug intervention: Quit drinking drugs are effective in helping drinkers to abstain from alcohol. Different drugs have different effects, but all are significantly better than "quit drinking through willpower".
- Physical intervention: Physiotherapy can control the bad behavior of the drinker during the onset of alcohol addiction.
- Psychological intervention: Psychological intervention emphasizes the attention to the psychological level of drinkers, which can meet the needs of drinkers at different stages of quit drinking, helps them establish a healthy attitude and way of life, and helps them to maintain the effect of quit drinking for a long time.
- Other methods: such as receiving Health education and seeking Social support.

Knowledge



Drinking and breast cancer

Drinking alcohol is one of the risk factors for breast cancer. The American Cancer Society pointed out that drinking alcohol is more harmful to women than men. Women who drink excessively have a 20% higher risk of breast cancer than women who do not drink; women who drink 1 cup or more a day

have a 45% higher risk of breast cancer than women who rarely drink; this danger is most pronounced in premenopausal women.

Different genotypes interact with alcohol, leading to different risks of breast cancer. For example, the locus of rs1045485 on an aspartate-specific cysteine protease (CASP8) gene is associated with the risk of breast cancer from drinking. Drinkers with CG or GG genotype at this locus are more likely to develop breast cancer than drinkers with CC genotype.

Onset time of breast cancer

Breast cancer mostly occurs in women between 40 and 60 years old (before and after menopause), and the incidence increases with age, but the incidence of postmenopausal women is significantly lower than that of premenopausal women.

The prevalence of breast cancer

Prevalence rate: 100/100,000. The distribution of breast cancer incidence varies in different countries and regions. Most Asian and African countries are low-incidence areas, Southern Europe and South America are middle-incidence areas, and North America and North Europe are high-incidence areas. China is an area with a relatively low incidence of breast cancer, and there are also regional differences. The general characteristics are: coastal cities are higher than inland areas, and cities with developed economies and high population densities are higher than those with backward economies and low population densities.

Typical symptoms of breast cancer

Early breast cancer often does not have typical symptoms and signs that appear for attention. It is often found through physical examination or breast cancer screening. The following are typical signs of breast cancer.

1. Breast lumps: 80% of breast cancer patients are first diagnosed with breast lumps. Patients often find breast lumps inadvertently, which are mostly single, hard, with irregular edges and a less smooth surface. Most breast cancers are painless masses, and only a few have varying degrees of dull pain or tingling.
2. Nipple discharge: If blood, serous, milk, or pus is discharged from the nipple during non-pregnancy, or if breastfeeding is stopped for more than half a year, it is called nipple discharge. There are many reasons for nipple discharge, and the common diseases include intraductal papilloma, breast hyperplasia, mammary duct dilatation and breast cancer. Unilateral single-port bloody discharge should be further examined, if accompanied by breast masses, more attention should be paid.
3. Skin changes: Breast cancer can cause skin changes with various signs. The most common is that the tumor invades the breast skin and the deep pectoralis fascia ligament, which shortens and loses its elasticity, pulls the skin of the corresponding part, and causes "dimples", that is, a small depression in the breast skin, like a small dimple. If cancer cells block the lymphatic vessels, there will be "orange peel-like changes", that is, many small dot-like depressions appear on the breast

skin, just like orange peels. In advanced breast cancer, cancer cells infiltrate into the skin and grow along the lymphatic vessels, glands, or fibrous tissues, forming scattered hard nodules in the skin around the main cancer focus, the so-called "skin satellite nodules".

4. Abnormal nipple and areola: The tumor is located in or near the deep part of the nipple, which can cause inverted nipple. When the tumor is far away from the nipple, the large duct in the breast is invaded and shortened, which can also cause the nipple to invert or lift. Eczema-like carcinoma of the nipple is characterized by itching, erosion, ulceration, crusting, scaling, and burning of the nipple skin, leading to inverted nipple.
5. Axillary lymph node swelling: More than one third of breast cancer patients admitted to a large hospital have axillary lymph node metastasis. There may be swelling of the ipsilateral axillary lymph nodes in the initial stage, and the swollen lymph nodes are hard, scattered, and pushable. As the disease progresses, the lymph nodes gradually fuse, and adhere to and fixate with the skin and surrounding tissues. In the late stage, the metastatic lymph nodes can be felt on the clavicle and the contralateral armpit.



Treatment of breast cancer

1. Tumor resection: surgical removal of the tumor and its adjacent tissues, leaving most of the breast. For limited breast cancer, this method is the best choice.
2. Complete mastectomy: If more than one tumor is found, or the tumor is quite large, consider removing the entire breast (usually including the lymph nodes on the same side). Most women whose breasts have been removed can consider breast reconstruction. The patient should discuss with the doctor whether it is suitable for reconstruction and which method to use to reconstruct the breast.
3. Radiotherapy: Use precise radiation to illuminate the tumor to kill cancer cells and shrink the tumor.
4. Drug therapy: use one or more drugs to inhibit cancer cells.
5. Radiation therapy and drug therapy are generally used as adjuvant treatments after surgery. In the early stage of breast cancer, doctors will assess the patient's condition and may give anti-estrogen therapy. According to statistics, about one-third of breast cancer patients are effective after receiving anti-estrogen therapy.

Other influencing factors



Controllable factors

1. Lifestyle: People who like high-fat, high-sugar, and high-calorie foods have a higher incidence of breast cancer. Frequent staying up late and lack of exercise can also increase the risk of breast cancer.

- 2. Obesity: Obese people have twice the risk of breast cancer.
- 3. Smoking: Smoking causes the incidence rate to be higher by 2/3, and women who start smoking during adolescence will increase the risk of disease by 70%.
- 4. Female childbearing experience: The incidence of breast cancer is higher in women who has never given birth, gave birth at the age of 35s, and did not breastfeed after childbirth.
- 5. Treatment experience: People who have been exposed to hormone-containing drugs or received chest radiation therapy are at higher risk of breast cancer.

 Uncontrollable factors

- 1. Age: Breast cancer mostly occurs in women between 40 and 60 years old. The older you are, the more likely you are to develop breast cancer.
- 2. Endocrine problems: Women younger than 12 years old at menarche (first menstruation) or women who have menopause after 55 years old have a higher risk of breast cancer.
- 3. Family history: People with a family history of breast cancer have a higher prevalence.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1045485	CASP8	CG	slightly high risk caused by drinking x breast cancer
RS2046210	intergenic	AG	slightly high risk caused by drinking x breast cancer
RS941764	CCDC88C	AG	slightly low risk caused by drinking x breast cancer

Risk calculation

The risk value of this test is calculated based on published academic literatures on the risk of breast cancer from drinking alcohol. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of breast cancer risk between drinking and non-drinking populations. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can show the changes in the risk of breast cancer for users if they drink alcohol relative to those who do not drink alcohol.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The risk of breast cancer from drinking alcohol item uses the 3 polymorphic loci on and between genes such as *CASP8*. The test results indicate that compared with all users, whether this user has a higher risk of breast cancer from drinking alcohol in the combination of genotypes of the 3 polymorphic loci tested. It does not mean the user's true risk of breast cancer from drinking alcohol. The basis of the test is mainly from the research of European, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of breast cancer from drinking alcohol.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by multiple factors such as genes and environment. This study only analyzes the effect of your drinking on the risk of breast cancer from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of breast cancer, or you think that you are likely to suffer from breast cancer, it is recommended that you consult an authoritative health professional or clinician for help.
4. Drinking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop drinking as soon as possible to reduce the risk of disease.

Test content

The test includes 3 polymorphic loci related to the risk of breast cancer from drinking alcohol, including rs2046210, rs941764, and rs1045485.



Population samples

The basis of the test is mainly from the research of European.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to the risk of breast cancer from drinking alcohol.

Drinking x Bladder Cancer

In general, alcohol consumption can increase the risk of bladder cancer. And to a certain extent, the increase of this risk is affected by genes.

My Drinking x Bladder Cancer

Moderate elevating risk

Quit drinking knowledge

Alcohol is a recognized addictive psychoactive substance. Excessive drinking and chronic drinking can cause harmful behavior problems, physical health problems and mental and psychological problems. Quit drinking is good for health. It can not only reduce the incidence of cancer, but also reduce the risk of death from cardiovascular disease. Quit drinking is not just a one-man battle, it also requires psychological, spiritual and behavioral support. Common methods include:

- Quit drinking through willpower: quit drinking only depends on willpower without other auxiliary means, but the success rate is low and the possibility of drinking again is high.
- Drug intervention: Quit drinking drugs are effective in helping drinkers to abstain from alcohol. Different drugs have different effects, but all are significantly better than "quit drinking through willpower".
- Physical intervention: Physiotherapy can control the bad behavior of the drinker during the onset of alcohol addiction.
- Psychological intervention: Psychological intervention emphasizes the attention to the psychological level of drinkers, which can meet the needs of drinkers at different stages of quit drinking, helps them establish a healthy attitude and way of life, and helps them to maintain the effect of quit drinking for a long time.
- Other methods: such as receiving Health education and seeking Social support.

Knowledge



Drinking and bladder cancer

Drinking alcohol is one of the risk factors for bladder cancer. The analysis of the risk of drinking and bladder cancer showed that compared with non-drinkers, the risk of bladder cancer in moderate drinkers was 0.98, while the risk of bladder cancer in heavy drinkers was higher, with a risk value of 1.31.

Different genotypes interact with alcohol to cause different risks of bladder cancer. For example, the locus of rs671 on the acetaldehyde dehydrogenase 2 (ALDH2) gene is associated with the risk of bladder cancer from drinking alcohol. Drinkers with GA or AA genotype at this locus are more likely to develop bladder cancer than drinkers with GG genotype.

Onset time of bladder cancer

The onset time is more common in the age of 50 and above. Prone to disease is increasing with age.

The prevalence of bladder cancer

In 2014, there were 74,690 new cases of bladder cancer in the United States. The overall incidence rate in China from 2003 to 2007 was 6.69 per 100,000 people.

Typical symptoms of bladder cancer

1. Hematuria: Usually, the first symptom of bladder cancer is blood in the urine. Blood in the urine may be pink or crimson urine, or the color of the urine under the naked eye is normal, but routine urine tests show an increase in red blood cells. Early bladder cancer with hematuria occurs, usually without other discomforts such as painful urination.
2. Changes in urination habits: including frequent urination (significant increase in the frequency of urination), urgency (repeated wanting to urinate), painful urination (pain when urinating), poor urination, increased nocturia, etc.
3. Symptoms caused by cancer cell metastasis: inability to urinate, unilateral low back pain, poor appetite, significant weight loss, fatigue, bone pain, etc.

Treatment of bladder cancer

1. Surgical treatment: The vast majority of bladder cancer can be treated with surgery, and the choice of surgery depends on the different stages of bladder cancer. Commonly used types of surgery include: transurethral resection of bladder tumor, cystectomy.
2. Infusion therapy: The doctor injects the drug into the bladder through a catheter, and the drug will stay in the bladder for about 2 hours. This therapy is not suitable for advanced bladder cancer.
3. Radiotherapy: Use high-energy rays to kill cancer cells.
4. Chemotherapy: The use of drugs to kill cancer cells or prevent the growth of cancer cells. Commonly used drugs include: cisplatin, fluorouracil, docetaxel, paclitaxel, methotrexate, vincristine and so on.
5. Immunotherapy: By influencing the patient's own immune system, it can more effectively identify and kill cancer cells. Commonly used immunotherapy includes: BCG, immune checkpoint inhibitors, etc.
6. Targeted therapy: Targeting cancer-causing mutations with targeted drug such as erdafitinib.

Other influencing factors

Controllable factors

1. Toxic substances: Frequent contact with rubber and leather may induce bladder cancer; practitioners in printing, textile, paint and other industries are at high risk of bladder cancer.
2. Eating habits: Insufficient drinking water and insufficient intake of fruits and vegetables may induce bladder cancer.
3. Smoking: Smoking may induce bladder cancer.

Uncontrollable factors

1. Age: commonly in 50 years old and above.
2. Gender: mainly male.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS671	ALDH2	GG	moderate risk caused by drinking x bladder cancer
RS755622	MIF-AS1	GG	moderate risk caused by drinking x bladder cancer

Risk calculation

The risk value of this test is calculated based on published academic literatures on the risk of bladder cancer from drinking alcohol. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of bladder cancer risk between drinking and non-drinking populations. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can show the changes in the risk of bladder cancer for users if they drink alcohol relative to those who do not drink alcohol.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The risk of bladder cancer from drinking alcohol item uses the 2 polymorphic loci on and between genes such as *ALDH2* and *MIF*. The test results indicate that compared with all users, whether this user has a higher risk of bladder cancer from drinking alcohol in the combination of genotypes of the 2 polymorphic loci tested. It does not mean the user's true risk of bladder cancer from drinking alcohol. The basis of the test is mainly from the research of East Asian, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of bladder cancer from drinking alcohol.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by multiple factors such as genes and environment. This study only analyzes the effect of your drinking on the risk of bladder cancer from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of bladder cancer, or you think that you are likely to suffer from bladder cancer, it is recommended that you consult an authoritative health professional or clinician for help.
4. Drinking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop drinking as soon as possible to reduce the risk of disease.

Test content

The test includes 2 polymorphic loci related to the risk of bladder cancer from drinking alcohol, including rs671 and rs755622.

Population samples

The basis of the test is mainly from the research of East Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to the risk of bladder cancer from drinking alcohol.

Drinking x Colorectal Cancer

In general, alcohol consumption can increase the risk of colorectal cancer. And to a certain extent, the increase of this risk is affected by genes.

My Drinking x Colorectal Cancer

Slightly high elevating risk

Quit drinking knowledge

Alcohol is a recognized addictive psychoactive substance. Excessive drinking and chronic drinking can cause harmful behavior problems, physical health problems and mental and psychological problems. Quit drinking is good for health. It can not only reduce the incidence of cancer, but also reduce the risk of death from cardiovascular disease. Quit drinking is not just a one-man battle, it also requires psychological, spiritual and behavioral support. Common methods include:

- Quit drinking through willpower: quit drinking only depends on willpower without other auxiliary means, but the success rate is low and the possibility of drinking again is high.
- Drug intervention: Quit drinking drugs are effective in helping drinkers to abstain from alcohol. Different drugs have different effects, but all are significantly better than "quit drinking through willpower".
- Physical intervention: Physiotherapy can control the bad behavior of the drinker during the onset of alcohol addiction.
- Psychological intervention: Psychological intervention emphasizes the attention to the psychological level of drinkers, which can meet the needs of drinkers at different stages of quit drinking, helps them establish a healthy attitude and way of life, and helps them to maintain the effect of quit drinking for a long time.
- Other methods: such as receiving Health education and seeking Social support.

Knowledge



Drinking and colorectal cancer

Long-term heavy drinking is a high-risk factor for the onset of colorectal cancer. The "China Colorectal Cancer Prevention Consensus (2016)" pointed out that the study found that the risk of colorectal cancer in the highest alcohol intake group was 60% higher than that in the control group, and the relative risk of colorectal cancer increased with the increase in alcohol consumption. A meta-

analysis suggests that there is a linear positive correlation between the risk of colorectal cancer and alcohol.

Different genotypes interact with alcohol, leading to different risks of colorectal cancer. For example, the locus of rs671 on the acetaldehyde dehydrogenase 2 (ALDH2) gene can increase the risk of colorectal cancer from drinking. Drinkers with the GG genotype are more likely to develop colorectal cancer than drinkers with the AA and AG genotypes.

Onset time of colorectal cancer

Colorectal cancer mostly occurs between the ages of 50 and 70.

The prevalence of colorectal cancer

The incidence and mortality of colorectal cancer in China are higher than the world average and the level of developing countries, and lower than the level of developed countries. The incidence of colorectal cancer in China is 28.08/100,000, accounting for 10.56% of the total incidence of all malignant tumors.

Typical symptoms of colorectal cancer

Early colorectal cancer may have no obvious symptoms, and the following symptoms do not appear until the disease has progressed to a certain extent: changes in bowel habits; changes in stool characteristics (thinning, bloody stools, mucous stools, etc.); abdominal pain or abdominal discomfort; abdominal masses; intestinal obstruction; anemia and systemic symptoms: such as weight loss, fatigue, and low-grade fever. Its morbidity and fatality rate are second only to stomach cancer, esophageal cancer and primary liver cancer in malignant tumors of the digestive system.

Treatment of colorectal cancer

1. Surgical treatment: The current treatment of colorectal cancer generally adopts the principle of comprehensive treatment based on surgery. The treatment plan is determined according to the patient's general condition and the functional status of various organs, tumor location, clinical stage, pathological type and biological behavior. The basic principles of colorectal cancer surgery are consistent with the basic principles of tumor surgery. In summary, it is the three principles of radical cure, safety, and functionality. Among them, when the tumor can be removed, the principle of radical cure is required first, the safety is considered second, and the functional principle is considered as much as possible at the end.
2. Chemotherapy: Adjuvant chemotherapy is generally required after colon cancer surgery. Colorectal cancer patients may have micrometastases or tumor cells remaining after surgery. Adjuvant chemotherapy is used to kill these metastases or tumor cells as much as possible. However, not all patients need and are suitable for chemotherapy. For example, early stage (stage I) patients do not need chemotherapy. In addition, for patients diagnosed with large tumors or local lymph node metastases, in order to improve the cure rate of surgery and reduce the scope of resection, doctors usually advise patients to receive radiotherapy and chemotherapy first, which can shrink the tumor or reduce the stage, which is conducive to the operation.

3. Radiation therapy: Generally used as an adjuvant therapy to surgery, treatment before or after surgery.

Other influencing factors



Controllable factors

- 1. Poor diet: high-fat diet, excessive intake of animal protein, especially red meat, and low-fiber diet are the main risk factors for colorectal cancer.
- 2. Living habits factors: Bad living habits, including long-term smoking, lack of exercise, etc., can increase the risk of colorectal cancer.
- 3. Overweight and obesity: In recent years, it has been considered that overweight and obesity are risk factors for colorectal cancer.



Uncontrollable factors

- 1. Age: Middle-aged and elderly people are more likely to develop colorectal cancer.
- 2. Disease history: Studies have shown that the history of chronic diarrhea, mucus blood in the stool, colorectal polyps, constipation, and appendicitis and other diseases and symptoms are risk factors for colorectal cancer.
- 3. Family history: People with a family history of colorectal cancer have a higher prevalence.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1229984	ADH1B	CT	moderate risk caused by drinking x color ectal cancer
RS1800795	intergenic	GG	moderate risk caused by drinking x color ectal cancer
RS1801133	MTHFR	GG	moderate risk caused by drinking x color ectal cancer
RS1804197	APC	AC	slightly high risk caused by drinking x co lorectal cancer
RS671	ALDH2	GG	slightly high risk caused by drinking x co lorectal cancer

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures on the risk of colorectal cancer from drinking alcohol. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of colorectal cancer risk between drinking and non-drinking populations. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can show the changes in the risk of colorectal cancer for users if they drink alcohol relative to those who do not drink alcohol.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The risk of colorectal cancer from drinking alcohol item uses the 6 polymorphic loci on and between genes such as *APC*, *MTHFR*, and *ALDH2*. The test results indicate that compared with all users, whether this user has a higher risk of colorectal cancer from drinking alcohol in the combination of genotypes of the 6 polymorphic loci tested. It does not mean the user's true risk of colorectal cancer from drinking alcohol. The basis of the test is mainly from the research of American, Asian and European, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of colorectal cancer from drinking alcohol.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.

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2. The occurrence and development of the disease is determined by multiple factors such as genes and environment. This study only analyzes the effect of your drinking on the risk of colorectal cancer from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
 3. If you have a family history of colorectal cancer, or you think that you are likely to suffer from colorectal cancer, it is recommended that you consult an authoritative health professional or clinician for help.
 4. Drinking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop drinking as soon as possible to reduce the risk of disease.

Test content

The test includes 6 polymorphic loci related to the risk of colorectal cancer from drinking alcohol, including rs1804197, rs1801133, and rs671.

Population samples

The basis of the test is mainly from the research of American, Asian and European.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to the risk of colorectal cancer from drinking alcohol.

Drinking x CHD

In general, alcohol consumption can increase the risk of coronary heart disease (CHD). And to a certain extent, the increase of this risk is affected by genes.

My Drinking x CHD

Moderate elevating risk

Quit drinking knowledge

Alcohol is a recognized addictive psychoactive substance. Excessive drinking and chronic drinking can cause harmful behavior problems, physical health problems and mental and psychological problems. Quit drinking is good for health. It can not only reduce the incidence of cancer, but also reduce the risk of death from cardiovascular disease. Quit drinking is not just a one-man battle, it also requires psychological, spiritual and behavioral support. Common methods include:

- Quit drinking through willpower: quit drinking only depends on willpower without other auxiliary means, but the success rate is low and the possibility of drinking again is high.
- Drug intervention: Quit drinking drugs are effective in helping drinkers to abstain from alcohol. Different drugs have different effects, but all are significantly better than "quit drinking through willpower".
- Physical intervention: Physiotherapy can control the bad behavior of the drinker during the onset of alcohol addiction.
- Psychological intervention: Psychological intervention emphasizes the attention to the psychological level of drinkers, which can meet the needs of drinkers at different stages of quit drinking, helps them establish a healthy attitude and way of life, and helps them to maintain the effect of quit drinking for a long time.
- Other methods: such as receiving Health education and seeking Social support.

Knowledge



Drinking and coronary heart disease

Drinking alcohol is one of the risk factors for coronary heart disease (CHD). Epidemiological studies have found that drinking alcohol is associated with coronary heart disease, and the associated risk coefficient for coronary heart disease is 6.0. Excessive drinking promotes arteriosclerosis and reduces the efficiency of lipid metabolism. Especially for spirits, its higher concentration of alcohol promotes

the synthesis of triglycerides in the body. This process will reduce the body's level of removal of lipids, aggravate coronary atherosclerosis, and increase the risk of coronary heart disease.

Different genotypes interact with alcohol, leading to different risks of coronary heart disease. For example, the locus of rs36071027 on the Early B-cell factor 1 (EBF1) gene is associated with the risk of coronary heart disease from drinking. Drinkers with TT or CT genotype at this locus are more likely to develop coronary heart disease than drinkers with CC genotype.

Onset time of coronary heart disease

It occurs in people over 35 years old. In developed countries, its annual deaths can account for 1/3 of the total deaths in middle-aged and elderly people.

The prevalence of coronary heart disease

According to statistics, there are about 10 cases of the disease for every 100,000 people in the average population.

Typical symptoms of coronary heart disease

The typical chest pain is caused by physical activity, emotional excitement, etc., sudden feeling of pain in the precordial area, mostly paroxysmal colic or squeezing pain, or a feeling of depression. The pain starts from the back of the breastbone or the front of the heart and radiates upwards to the left shoulder, arm, and even the little finger and ring finger. The part where chest pain disperses can also involve the neck, jaw, teeth, abdomen, etc. In addition, symptoms of gastrointestinal diseases such as upper abdominal pain, nausea, and vomiting may also occur.

Treatment of coronary heart disease

Although coronary heart disease, especially myocardial infarction, is very dangerous, the first thing to do is to solve the stenosis and infarction and restore myocardial blood supply as soon as possible, so as to quickly control the symptoms and minimize the scope and extent of myocardial necrosis. Drug treatments include sublingual administration of nitroglycerin and other nitrate drugs, metoprolol and other β -receptor blockers, and nifedipine calcium channel blockers. Surgical procedures include heart bypass.

Other influencing factors

Controllable factors

1. Obese/overweight people have a higher risk than normal.
2. Dietary factors: unhealthy diets such as high fat, high cholesterol, and high calories can easily lead to coronary heart disease.
3. Unhealthy living habits: such as lack of physical activity can also induce this disease.

Uncontrollable factors

1. Genetic factors: there is a family genetic tendency.
2. Disease factors: high blood pressure, high blood lipids, high blood sugar/diabetics have a high incidence of coronary heart disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1800795	intergenic	GG	moderate risk caused by drinking x CHD
RS854560	PON1	AA	moderate risk caused by drinking x CHD
RS36071027	intergenic	CC	moderate risk caused by drinking x CHD

Risk calculation

The risk value of this test is calculated based on published academic literatures on the risk of coronary heart disease from drinking alcohol. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of coronary heart disease risk between drinking and non-drinking populations. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can show the changes in the risk of coronary heart disease for users if they drink alcohol relative to those who do not drink alcohol.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The risk of coronary heart disease from drinking alcohol item uses the 3 polymorphic loci on and between genes such as *PON1*. The test results indicate that compared with all users, whether this user has a higher risk of coronary heart disease from drinking alcohol in the combination of

genotypes of the 3 polymorphic loci tested. It does not mean the user's true risk of coronary heart disease from drinking alcohol. The basis of the test is mainly from the research of Chinese and African American, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of coronary heart disease from drinking alcohol.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by multiple factors such as genes and environment. This study only analyzes the effect of your drinking on the risk of coronary heart disease from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of coronary heart disease, or you think that you are likely to suffer from coronary heart disease, it is recommended that you consult an authoritative health professional or clinician for help.
4. Drinking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop drinking as soon as possible to reduce the risk of disease.

Test content

The test includes 3 polymorphic loci related to the risk of coronary heart disease from drinking alcohol, including rs854560, rs1800795, and rs36071027.

Population samples

The basis of the test is mainly from the research of Chinese and African American.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to the risk of coronary heart disease from drinking alcohol.

Drinking x Hypertension

In general, alcohol consumption can increase the risk of hypertension. And to a certain extent, the increase of this risk is affected by genes.

My Drinking x Hypertension

Moderate elevating risk

Quit drinking knowledge

Alcohol is a recognized addictive psychoactive substance. Excessive drinking and chronic drinking can cause harmful behavior problems, physical health problems and mental and psychological problems. Quit drinking is good for health. It can not only reduce the incidence of cancer, but also reduce the risk of death from cardiovascular disease. Quit drinking is not just a one-man battle, it also requires psychological, spiritual and behavioral support. Common methods include:

- Quit drinking through willpower: quit drinking only depends on willpower without other auxiliary means, but the success rate is low and the possibility of drinking again is high.
- Drug intervention: Quit drinking drugs are effective in helping drinkers to abstain from alcohol. Different drugs have different effects, but all are significantly better than "quit drinking through willpower".
- Physical intervention: Physiotherapy can control the bad behavior of the drinker during the onset of alcohol addiction.
- Psychological intervention: Psychological intervention emphasizes the attention to the psychological level of drinkers, which can meet the needs of drinkers at different stages of quit drinking, helps them establish a healthy attitude and way of life, and helps them to maintain the effect of quit drinking for a long time.
- Other methods: such as receiving Health education and seeking Social support.

Knowledge



Drinking and hypertension

Excessive drinking significantly increases the risk of hypertension, and its risk increases with the increase in alcohol consumption. The study found that alcohol intake decreased by 67% on average, systolic blood pressure decreased by 3.31 mmHg, and diastolic blood pressure decreased by 2.04 mmHg. At present, there is insufficient evidence that a small amount of alcohol is beneficial to

cardiovascular health, but even for people who drink small amounts of alcohol, reducing alcohol intake can reduce the risk of hypertension and improve cardiovascular health.

Different genotypes interact with alcohol, leading to different risks of hypertension. For example, the locus of rs4551304 on telomerase inhibitor 1 (PINX1) gene is associated with the risk of hypertension from drinking. Drinkers with AA or GA genotype at this locus are more likely to develop hypertension than drinkers with GG genotype.

Onset time of hypertension

It can occur at any age, but the prevalence of hypertension increases with age.

The prevalence of hypertension

According to data released by the Chinese Ministry of Health: Chinese average incidence rate is 11.88%, and the number of patients is over 160 million. In some European and American countries, the incidence rate is as high as 20%.

Typical symptoms of hypertension

Hypertension is a clinical syndrome characterized by increased systemic arterial blood pressure. The cause of most patients is unknown. Studies have shown that hypertensive patients are more likely to develop other cardiovascular and cerebrovascular diseases than those with normal blood pressure, such as atherosclerosis, and affect the functions of important organs such as the heart, brain, and kidneys, and ultimately lead to the failure of these organs. Some hypertensive patients have headaches (especially back pain and morning headaches), dizziness, tinnitus (buzzing or sizzling in the ears), visual changes or fainting episodes.

Treatment of hypertension

The cause of most hypertension patients is not clear, and most patients need long-term medication to control their blood pressure. For patients with mild and early hypertension, blood pressure can be controlled within the normal range through weight loss, diet improvement, exercise, etc., and at the same time, it is necessary to adhere to a healthy lifestyle. If the blood pressure level drops after taking the medicine, the patient should not stop the medicine by himself, otherwise, the blood pressure rebounds or is more dangerous. Although high blood pressure cannot be completely cured, blood pressure can be effectively controlled by insisting on taking medication and the risk of various adverse consequences can be minimized.

Other influencing factors

Controllable factors

1. Dietary factors: A high-sugar and high-fat diet, eating meat more than vegetable, excessive sodium intake, and low potassium intake will increase the risk of hypertension.

- 2. Smoking: Smokers or passive smokers have an increased risk of hypertension.
- 3. Poor living habits: Sitting for a long time and lack of exercise may increase the risk of hypertension.
- 4. Psychological and emotional factors: People who have been under excessive stress, anxiety, irritability, and emotional agitation for a long time have a higher risk of illness.
- 5. Overweight and obesity: With the increase of BMI and waist circumference, the mean blood pressure showed a significant increase trend, and the prevalence of hypertension also increased significantly.

 Uncontrollable factors

- 1. Age: The prevalence of hypertension increases with age.
- 2. Family history: Hypertension has obvious familial clustering and is obviously related to immediate family members. People with a family history of hypertension have a higher prevalence.
- 3. Blood glucose and blood lipid levels: Epidemiological data show that hypertension is often accompanied by many risk factors related to metabolism, including impaired glucose tolerance, dyslipidemia, etc., which are related to hypertension.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS10826334	intergenic	CC	moderate risk caused by drinking x hypertension
RS1799998	CYP11B2	AA	slightly high risk caused by drinking x hypertension
RS9928094	FTO	AA	slightly high risk caused by drinking x hypertension
RS3735814	GATA4	AA	slightly high risk caused by drinking x hypertension
RS4551304	intergenic	GG	moderate risk caused by drinking x hypertension

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures on the risk of hypertension from drinking alcohol. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of hypertension risk between drinking and non-drinking populations. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can show the changes in the risk of hypertension for users if they drink alcohol relative to those who do not drink alcohol.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The risk of hypertension from drinking alcohol item uses the 10 polymorphic loci on and between genes such as *BLK*, *PINX1*, and *GATA4*. The test results indicate that compared with all users, whether this user has a higher risk of hypertension from drinking alcohol in the combination of genotypes of the 10 polymorphic loci tested. It does not mean the user's true risk of hypertension from drinking alcohol. The basis of the test is mainly from the research of African, Asian, European and Brazilian, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of hypertension from drinking alcohol.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by multiple factors such as genes and environment. This study only analyzes the effect of your drinking on the risk of hypertension from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.

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3. If you have a family history of hypertension, or you think that you are likely to suffer from hypertension, it is recommended that you consult an authoritative health professional or clinician for help.
 4. Drinking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop drinking as soon as possible to reduce the risk of disease.

Test content

The test includes 10 polymorphic loci related to the risk of hypertension from drinking alcohol, including rs9286060, rs2409784, and rs1799998.

Population samples

The basis of the test is mainly from the research of African, Asian, European and Brazilian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to the risk of hypertension from drinking alcohol.

Drinking x Lung Cancer

In general, alcohol consumption can increase the risk of lung cancer. And to a certain extent, the increase of this risk is affected by genes.

My Drinking x Lung Cancer

Slightly high elevating risk

Quit drinking knowledge

Alcohol is a recognized addictive psychoactive substance. Excessive drinking and chronic drinking can cause harmful behavior problems, physical health problems and mental and psychological problems. Quit drinking is good for health. It can not only reduce the incidence of cancer, but also reduce the risk of death from cardiovascular disease. Quit drinking is not just a one-man battle, it also requires psychological, spiritual and behavioral support. Common methods include:

- Quit drinking through willpower: quit drinking only depends on willpower without other auxiliary means, but the success rate is low and the possibility of drinking again is high.
- Drug intervention: Quit drinking drugs are effective in helping drinkers to abstain from alcohol. Different drugs have different effects, but all are significantly better than "quit drinking through willpower".
- Physical intervention: Physiotherapy can control the bad behavior of the drinker during the onset of alcohol addiction.
- Psychological intervention: Psychological intervention emphasizes the attention to the psychological level of drinkers, which can meet the needs of drinkers at different stages of quit drinking, helps them establish a healthy attitude and way of life, and helps them to maintain the effect of quit drinking for a long time.
- Other methods: such as receiving Health education and seeking Social support.

Knowledge



Drinking and lung cancer

Drinking alcohol increases the risk of lung cancer. The study found that the risk of lung cancer among drinkers is 1.41 times more than non-drinkers; the longer they have been drinking, the greater the risk occur. Those who have been drinking for more than 30 years have 1.62 times risk of lung cancer.

Different genotypes interact with alcohol, leading to different risks of lung cancer. For example, the locus of rs1801133 on the methylenetetrahydrofolate reductase (MTHFR) gene is associated with the risk of lung cancer from drinking. Drinkers with AA genotype at this locus are more likely to develop lung cancer than drinkers with GG or AG genotypes.

Onset time of lung cancer

The disease can occur at any age, and people who are over 50 years old with a history of smoking may be the most susceptible to lung cancer.

The prevalence of lung cancer

Prevalence rate: 49 per 100,000 people. The cancer with the highest incidence and mortality rate among men worldwide is lung cancer. Among women, the incidence of lung cancer ranks third, and the mortality rate ranks second, second only to breast cancer. In 2012, there were 1.82 million new lung cancer patients worldwide, and 1.56 million eventually died of lung cancer, accounting for 19.4% of deaths due to cancer. The highest incidence of lung cancer is in North America, Europe and East Asia. In 2012, more than one-third of new patients appeared in China, while the incidence of lung cancer in Africa and South Asia was much lower.

Typical symptoms of lung cancer

The clinical manifestations of lung cancer are more complicated. The presence, severity, and time of symptoms and signs depend on the tumor site, pathological type, metastasis, and complications, as well as differences in patient response and tolerance. The early symptoms of lung cancer are often mild and may even be uncomfortable. The symptoms of central lung cancer appear early and severe, and the symptoms of peripheral lung cancer appear late and mild, even asymptomatic, and are often found during physical examination. The symptoms of lung cancer are roughly divided into: local symptoms, systemic symptoms, extrapulmonary symptoms, infiltration and metastasis symptoms.

1. Local symptoms, including cough, blood or hemoptysis in sputum, chest pain, chest tightness, shortness of breath and hoarseness.
2. Systemic symptoms, including fever, weight loss and cachexia.
3. Extrapulmonary symptoms. Due to some special active substances (including hormones, antigens, enzymes, etc.) produced by lung cancer, patients may have one or more extrapulmonary symptoms, which often appear before other symptoms, and it can disappear or appear with the growth and decline of tumors. Pulmonary osteoarticular hyperplasia is more common in clinical practice.
4. Symptoms of external invasion and metastasis, including lymph node metastasis, pleural invasion and/or metastasis, superior vena cava syndrome, kidney metastasis, digestive tract metastasis, bone metastasis, central nervous system symptoms, heart invasion, metastasis and peripheral nervous system symptoms.

Treatment of lung cancer

The treatment of lung cancer includes surgical treatment, radiotherapy, chemotherapy and immunotherapy. Surgical treatment has been recognized as the first choice for the treatment of lung cancer, and the treatment plan should be selected based on the clinical stage of lung cancer. Radical resection is so far the only possible treatment for lung cancer patients to be cured and return to normal life. Before surgery, it is necessary to assess whether the patient tolerates the surgery. These tests usually include: clinical physical examination, pulmonary ventilation function, blood test and so on. For marginal patients, lung perfusion scans should be used to more accurately assess lung function, or endurance tests and coronary angiography should be used to assess heart function.

Other influencing factors



Controllable factors

1. Smoking: Cigarettes in particular are the main cause of lung cancer so far. Cigarette smoke contains at least 73 known carcinogens, and lung cancer patients caused by smoking account for 80 to 90% of lung cancer patients.
2. Dietary factors: A large amount of β -carotene intake or excessive vitamin E intake may increase the risk of lung cancer.
3. Hormone replacement therapy: The use of hormone therapy such as estrogen and progesterone may increase the risk of lung cancer.
4. Air pollution: Nitrogen dioxide, fine particles (PM_{2.5}) and sulphate aerosols contained in car exhaust, indoor soot, etc. may all increase the risk of lung cancer; radon is a colorless and odorless radioactive gases, which may be released from foundation soil, building materials, and decoration materials, are the second most common cause of lung cancer in the United States; asbestos can cause a variety of lung diseases, including lung cancer.
5. Other lung carcinogens: certain metals (aluminum products, cadmium and its compounds, etc.), some combustion products (incomplete combustion products, coal gas, etc.), ionizing radiation (X-rays, gamma rays, etc.), some harmful gas (methyl ether, sulfur mustard, etc.), rubber products and crystalline silica dust are all carcinogens associated with lung cancer.



Uncontrollable factors

Family history: People with a family history of lung cancer have a higher prevalence.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS1801131	MTHFR	GT	moderate risk caused by drinking x lung cancer
RS1801133	MTHFR	GG	moderate risk caused by drinking x lung cancer
RS2031920	intergenic	CT	moderate risk caused by drinking x lung cancer
RS4680	COMT	GG	moderate risk caused by drinking x lung cancer

Risk calculation

The risk value of this test is calculated based on published academic literatures on the risk of lung cancer from drinking alcohol. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of lung cancer risk between drinking and non-drinking populations. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can show the changes in the risk of lung cancer for users if they drink alcohol relative to those who do not drink alcohol.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The risk of lung cancer from drinking alcohol item uses the 4 polymorphic loci on and between genes such as *COMT* and *MTHFR*. The test results indicate that compared with all users, whether this user has a higher risk of lung cancer from drinking alcohol in the combination of genotypes of the 4 polymorphic loci tested. It does not mean the user's true risk of lung cancer from drinking alcohol. The basis of the test is mainly from the research of Japanese, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of lung cancer from drinking alcohol.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by multiple factors such as genes and environment. This study only analyzes the effect of your drinking on the risk of lung cancer from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of lung cancer, or you think that you are likely to suffer from lung cancer, it is recommended that you consult an authoritative health professional or clinician for help.
4. Drinking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop drinking as soon as possible to reduce the risk of disease.

Test content

The test includes 4 polymorphic loci related to the risk of lung cancer from drinking alcohol, including rs4680, rs1801133, and rs2031920.

Population samples

The basis of the test is mainly from the research of Japanese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to the risk of lung cancer from drinking alcohol.

Smoking x Stomach Cancer

In general, smoking can increase the risk of gastric cancer. And to a certain extent, the increase of this risk is affected by genes.

My Smoking x Stomach Cancer

Slightly high elevating risk

Quit smoking knowledge

Tobacco is the largest controllable hazard affecting human health. Smoking cessation has health benefits, including a lower risk of cancer and death from cardiovascular disease. In addition to smoking cessation policies, environmental and social support, and personal determination and perseverance, appropriate smoking cessation methods should also be adopted, or a combination of multiple methods should be used. Common methods include:

- Quit smoking through willpower: Smoking cessation through willpower alone without any other supplementary means, but the success rate is very low, less than 3%.
- Drug intervention: Smoking cessation drugs can be effective in helping smokers quit their addiction. Different drugs have different efficacy, but all of them are significantly better than "Quit smoking through willpower".
- Behavioral intervention: Different levels of behavioral intervention, including speech intervention and telephone follow-up. A combination of drug interventions can greatly improve the success rate.
- E-cigarettes: E-cigarettes can be used as an alternative to tobacco, but their safety needs to be verified by further studies.
- TCM (Traditional Chinese Medicine) smoking cessation: Some studies have shown that TCM acupuncture can help smokers quit smoking by regulating endorphin levels, but this method has a high relapse rate.
- Other methods: such as Information-based smoking cessation, Aversion therapy, Hypnotherapy, etc., can be used as a second choice after drugs, behavioral intervention and other methods fail.

Knowledge



Smoking and stomach cancer

Smoking is an important risk factor for the onset of stomach cancer, and it has a dose-effect relationship with the total amount of smoking and the number of years of smoking. The study found

that compared with non-smokers, the relative risk of stomach cancer in people with a history of smoking was 1.6, and the younger they started smoking, the higher the risk of stomach cancer; the relative risk of stomach cancer for those who consumed nearly 20 cigarettes a day was 1.53 compared with those who had never smoked.

Different genotypes interact with smoking, leading to different risks of stomach cancer. For example, the locus of rs1047840 on the exonuclease 1 (EXO1) gene is associated with the risk of stomach cancer from smoking. Smokers with AG or AA genotype at this locus are more likely to develop stomach cancer than smokers with GG genotype.

Onset time of stomach cancer

Most occur after middle-aged, more common in ages between 40 to 60.

The prevalence of stomach cancer

Among the tumors of the digestive system in China, stomach cancer has the highest incidence. According to a report published by the World Health Organization, the annual incidence of stomach cancer in the world is 14 per 100,000 people. The incidence of stomach cancer varies greatly worldwide. South Korea, Japan, and East Asia have the highest incidence. Other high-incidence areas include parts of Eastern Europe and Latin America, while Western Europe and the United States have lower incidences.

Typical symptoms of stomach cancer

Early stomach cancer is mostly asymptomatic or only mild symptoms. When the clinical symptoms are obvious, the disease is already at an advanced stage. Therefore, we must be very alert to the early symptoms of stomach cancer to avoid delay in diagnosis and treatment.

1. Early manifestations: Upper abdominal discomfort is the most common initial symptom of stomach cancer. About 80% of patients have this manifestation, which is similar to indigestion. If abdominal pain occurs, it is generally mild and irregular, and cannot be relieved after eating. Nearly 50% of patients with stomach cancer have obvious symptoms of loss of appetite. Some patients restrict their eating due to excessive eating which can cause bloating or abdominal pain. Unexplained anorexia and weight loss are probably the initial symptoms of early stomach cancer and need to be paid attention to. Patients with early stomach cancer generally have no obvious positive signs. Most patients only have deep tenderness in the upper abdomen, except for the general condition.
2. Late-stage manifestations: When stomach cancer develops and expands, especially when it penetrates through the serosa and invades the pancreas, persistent severe pain may occur and radiate to the lower back. The absorption of cancer toxins can make patients lose weight, fatigue, anemia, and finally show cachexia. After the cancer grows, symptoms of obstruction may appear. Cardia or fundus cancer may cause dysphagia, gastric antrum cancer may cause pyloric obstruction symptoms, and a lump may be palpable in the abdomen. When an ulcer forms on the surface of the cancer, hematemesis and melena appear. As for the appearance of metastases such

as anterior rectal masses, umbilical masses, supraclavicular lymphadenopathy, and ascites, it is even more evidence of advanced stomach cancer.

Treatment of stomach cancer

1. Surgical treatment: Surgical resection is still the only option for radical treatment of early stomach cancer, and it is also the main method for the treatment of stomach cancer. For a long time, due to the late detection of stomach cancer, most of which are advanced tumors, the surgical effect is not good, and the 5-year survival rate after surgery has been maintained at about 30%. Therefore, it is necessary to strengthen the attention to the symptoms of early stomach cancer and the monitoring of high-risk groups to improve detection rate of early stomach cancer.
2. Chemotherapy: Surgical resection rate of stomach cancer is 50%-60%, while the cure rate is only 20%-40%. Most of them can relapse again, and often die from local recurrence and distant metastasis; because the admitted cases are relatively late in order to improve the efficacy of surgery, a comprehensive treatment of radical surgery and chemotherapy can be used; for palliative resection cases, longer-term chemotherapy is required after surgery; about 40% of patients cannot be resected with tumors, and for advanced cases that cannot be operated, chemotherapy It is the main treatment.
3. Immunotherapy: The combined use of immunotherapy and chemotherapy is believed to have the effect of prolonging life, and freeze-dried Bacille Calmette-Guerin (BCG) is the most commonly used.
4. Intraoperative radiation exposure: Intraoperative radiation therapy can improve the 5-year survival rate.

Other influencing factors

Controllable factors

1. Poor eating style: Studies have shown that poor eating style can increase the risk of stomach cancer. Common risk factors are skipping breakfast, irregular meals, preferring dry and hard food, preferring hot food, eating fast, preferring to eat smoked, grilled food and fried food, etc. These bad habits and physical stimulation of food can cause mechanical gastric mucosal damage and gastric juice secretion disorders, etc., leading to the occurrence of chronic gastric disease and ultimately carcinogenesis.
2. Living habits factors: Bad lifestyle habits, including long-term drinking, lack of exercise, obesity, etc., can increase the risk of stomach cancer.
3. Infectious factors: The results of a large number of epidemiological studies have shown that *Helicobacter pylori* infection is positively correlated with the prevalence of stomach cancer. Other studies have shown that *Helicobacter pylori* can promote cell proliferation and cause mutations in key oncogenes. Persistent *Helicobacter pylori* infection can induce chronic gastritis and atrophic

gastritis, and then cause changes in gastric mucosal cell metabolism, leading to intestinal metaplasia, gastric epithelial dysplasia and other precancerous lesions.

4. Gastric disease experience: Many gastric diseases can significantly increase the risk of stomach cancer, including chronic atrophic gastritis, gastric ulcer, gastric polyps and superficial gastritis. Among them, gastric ulcer, chronic atrophy and gastritis are most closely related to stomach cancer. Atrophic gastritis is widely regarded as a precursor to stomach cancer, and the further development of inflammation may lead to the occurrence of cancer.

Uncontrollable factors

1. Age: Middle-aged and elderly people are more likely to develop stomach cancer.
2. Gender: There is an obvious gender imbalance in the incidence of stomach cancer. The incidence of stomach cancer in men is much higher than that in women.
3. Geographical environmental factors: Studies have shown that there are significant differences in the incidence of stomach cancer in different regions and races. Some data indicate that stomach cancer is more common in high latitudes. The farther the country is from the equator, the higher the incidence of stomach cancer. There are also data that the incidence is related to coastal factors. Here are the factors of different eating habits. Geographical and chemical factors and the possibility of carcinogens in the environment should also be considered.
4. Family history: People with a family history of stomach cancer have a higher prevalence.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1047840	EXO1	AG	slightly high risk caused by smoking x stomach cancer
RS1799750	WTAPP1	DI	slightly high risk caused by smoking x stomach cancer
RS2031920	intergenic	CT	slightly high risk caused by smoking x stomach cancer
RS2736100	TERT	AA	moderate risk caused by smoking x stomach cancer
RS4646903	CYP1A1	AG	slightly high risk caused by smoking x stomach cancer

Risk calculation

The risk value of this test is calculated based on published academic literatures on the risk of stomach cancer from smoking. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of stomach cancer risk between smokers and non-smokers. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can show the changes in the risk of stomach cancer for users if they smoke relative to those who do not smoke.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The risk of stomach cancer from smoking item uses the 5 polymorphic loci on and between genes such as *TERT*, *EXO1* and *WTAPP1*. The test results indicate that compared with all users, whether this user has a higher risk of stomach cancer from smoking in the combination of genotypes of the 5 polymorphic loci tested. It does not mean the user's true risk of stomach cancer from smoking. The basis of the test is mainly from the research of Chinese, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of stomach cancer from smoking.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by multiple factors such as genes and environment. This study only analyzes the effect of your smoking on the risk of stomach cancer from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of stomach cancer, or you think that you are likely to suffer from stomach cancer, it is recommended that you consult an authoritative health professional or clinician for help.

4. Smoking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop drinking as soon as possible to reduce the risk of disease.

 Test content

The test includes 5 polymorphic loci related to the risk of stomach cancer from smoking, including rs2736100, rs4646903, and rs1047840.

 Population samples

The basis of the test is mainly from the research of Chinese.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to the risk of stomach cancer from smoking.

Smoking x Esophagus Cancer

In general, smoking can increase the risk of esophageal cancer. And to a certain extent, the increase of this risk is affected by genes.

My Smoking x Esophagus Cancer

Slightly low elevating risk

Quit smoking knowledge

Tobacco is the largest controllable hazard affecting human health. Smoking cessation has health benefits, including a lower risk of cancer and death from cardiovascular disease. In addition to smoking cessation policies, environmental and social support, and personal determination and perseverance, appropriate smoking cessation methods should also be adopted, or a combination of multiple methods should be used. Common methods include:

- Quit smoking through willpower: Smoking cessation through willpower alone without any other supplementary means, but the success rate is very low, less than 3%.
- Drug intervention: Smoking cessation drugs can be effective in helping smokers quit their addiction. Different drugs have different efficacy, but all of them are significantly better than "Quit smoking through willpower".
- Behavioral intervention: Different levels of behavioral intervention, including speech intervention and telephone follow-up. A combination of drug interventions can greatly improve the success rate.
- E-cigarettes: E-cigarettes can be used as an alternative to tobacco, but their safety needs to be verified by further studies.
- TCM (Traditional Chinese Medicine) smoking cessation: Some studies have shown that TCM acupuncture can help smokers quit smoking by regulating endorphin levels, but this method has a high relapse rate.
- Other methods: such as Information-based smoking cessation, Aversion therapy, Hypnotherapy, etc., can be used as a second choice after drugs, behavioral intervention and other methods fail.

Knowledge

Smoking and esophageal cancer

Smoking is one of the risk factors for the onset of esophageal cancer. The amount of smoking per day and the number of years of smoking have a dose-response relationship with the onset of esophageal

cancer. The study found that the risk of esophageal cancer in people with smoking behavior is 2.08 times that of non-smokers; the risk of esophageal cancer in people who have smoked for 20 years or more is 4.41 times that of non-smokers.

Different genotypes interact with smoking, leading to different risks of esophageal cancer. For example, the locus of rs1138272 on the glutathione S-transferase P1 (GSTP1) gene is associated with the risk of esophageal cancer from smoking. Smokers with CT or TT genotype at this locus are more likely to develop esophageal cancer than smokers with CC genotype.

Onset time of esophageal cancer

The age of onset is mostly over 40 years old.

The prevalence of esophageal cancer

The incidence of esophageal cancer varies greatly among countries. China is an area with a high incidence of esophageal cancer, and the incidence of esophageal cancer in Henan ranks first in the world.

Typical symptoms of esophageal cancer

1. Early symptoms: Mainly post-sternal burning sensation, acupuncture-like or stretch-like pain, slow passage of eating, and a feeling of retention or mild choking. The early symptoms are mild and severe, and may even be asymptomatic.
2. Middle and late symptoms: Progressive dysphagia (first hard to swallow dry food, then semi-liquid food, and finally hard to swallow water and saliva), food reflux, painful swallowing, accompanied by weight loss, lymphadenopathy, Hoarse voice etc.

Treatment of esophageal cancer

Surgery, radiation therapy, and chemotherapy.

Other influencing factors

Controllable factors

1. Nitrosamines: Eating salted fish, pickled vegetables and other high nitrite foods will increase the risk of esophageal cancer.
2. Fungi: A variety of fungi can be isolated from the upper gastrointestinal tract of patients with esophageal cancer or excised esophageal cancer specimens. Investigations have shown that eating moldy food has a certain relationship with the onset of esophageal cancer.
3. Nutritional deficiency: Epidemiology shows that the lack of a variety of vitamins (such as vitamin A, B2, C) in the diet, or the lack of certain trace elements (including molybdenum, zinc, selenium, etc.) are related to the onset of esophageal cancer.

- 4. Drinking: The incidence of esophageal cancer in people who often drink hard alcohol is significantly higher than that of the general population.
- 5. Eating habits: Long-term eating of hard food or eating too fast or too hot can easily cause mechanical damage to the esophageal mucosa. Repeated stimulation can lead to mucosal epithelial hyperplasia and eventually cancer.

 Uncontrollable factors

- 1. Family history: The occurrence of esophageal cancer has obvious family clustering phenomenon.
- 2. Age: The age of onset is more than 40 years old.
- 3. Gender: Males predominantly.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1051740	EPHX1	CC	slightly low risk caused by smoking x esophagus cancer
RS1138272	GSTP1	CC	moderate risk caused by smoking x esophagus cancer
RS1229984	ADH1B	CT	moderate risk caused by smoking x esophagus cancer
RS2031920	intergenic	CT	slightly high risk caused by smoking x esophagus cancer
RS4646903	CYP1A1	AG	slightly high risk caused by smoking x esophagus cancer

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures on the risk of esophageal cancer from smoking. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of esophageal cancer risk between smokers and non-smokers. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can show the changes in the risk of esophageal cancer for users if they smoke relative to those who do not smoke.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The risk of esophageal cancer from smoking item uses the 7 polymorphic loci on and between genes such as *ALDH2*, *EPHX1* and *ADH1B*. The test results indicate that compared with all users, whether this user has a higher risk of esophageal cancer from smoking in the combination of genotypes of the 7 polymorphic loci tested. It does not mean the user's true risk of esophageal cancer from smoking. The basis of the test is mainly from the research of East Asian and South African, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of esophageal cancer from smoking.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by multiple factors such as genes and environment. This study only analyzes the effect of your smoking on the risk of esophageal cancer from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of esophageal cancer, or you think that you are likely to suffer from esophageal cancer, it is recommended that you consult an authoritative health professional or clinician for help.
4. Smoking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop drinking as soon as possible to reduce the risk of disease.

Test content

The test includes 7 polymorphic loci related to the risk of esophageal cancer from smoking, including rs4646903, rs671, and rs1042028.

 Population samples

The basis of the test is mainly from the research of East Asian and South African.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to the risk of esophageal cancer from smoking.

Smoking x Breast Cancer

In general, smoking can increase the risk of breast cancer. And to a certain extent, the increase of this risk is affected by genes.

My Smoking x Breast Cancer

Slightly low elevating risk

Quit smoking knowledge

Tobacco is the largest controllable hazard affecting human health. Smoking cessation has health benefits, including a lower risk of cancer and death from cardiovascular disease. In addition to smoking cessation policies, environmental and social support, and personal determination and perseverance, appropriate smoking cessation methods should also be adopted, or a combination of multiple methods should be used. Common methods include:

- Quit smoking through willpower: Smoking cessation through willpower alone without any other supplementary means, but the success rate is very low, less than 3%.
- Drug intervention: Smoking cessation drugs can be effective in helping smokers quit their addiction. Different drugs have different efficacy, but all of them are significantly better than "Quit smoking through willpower".
- Behavioral intervention: Different levels of behavioral intervention, including speech intervention and telephone follow-up. A combination of drug interventions can greatly improve the success rate.
- E-cigarettes: E-cigarettes can be used as an alternative to tobacco, but their safety needs to be verified by further studies.
- TCM (Traditional Chinese Medicine) smoking cessation: Some studies have shown that TCM acupuncture can help smokers quit smoking by regulating endorphin levels, but this method has a high relapse rate.
- Other methods: such as Information-based smoking cessation, Aversion therapy, Hypnotherapy, etc., can be used as a second choice after drugs, behavioral intervention and other methods fail.

Knowledge



Smoking and breast cancer

Smoking is one of the risk factors for breast cancer. The "Guidelines for Breast Cancer Screening and Early Diagnosis and Treatment in Women in China (2021)" pointed out that smoking has increased

the risk of breast cancer by 10%; the risk of breast cancer in people with a history of smoking is 1.50 times that of people without a history of smoking. Long smoking time (20 years or more) and a lot of cigarettes per day (20 or more cigarettes) increase the risk of breast cancer by 13% to 16%.

Different genotypes interact with smoking, leading to different risks of breast cancer. For example, the locus of rs2234693 on the estrogen receptor 1 (ESR1) gene is associated with the risk of breast cancer from smoking. Smokers with TT genotype at this locus are more likely to develop breast cancer than smokers with CC or TC genotype.



Onset time of breast cancer

Breast cancer mostly occurs in women between 40 and 60 years old (before and after menopause), and the incidence increases with age, but the incidence of postmenopausal women is significantly lower than that of premenopausal women.



The prevalence of breast cancer

Prevalence rate: 100/100,000. The distribution of breast cancer incidence is different in different countries and regions. Most Asian and African countries are low-incidence areas, Southern Europe and South America are middle-incidence areas, and North America and North Europe are high-incidence areas. China is an area with a relatively low incidence of breast cancer, and there are also regional differences. The general characteristics are: coastal cities are higher than inland areas, and cities with developed economies and high population densities are higher than those with backward economies and low population densities.



Typical symptoms of breast cancer

Early breast cancer often does not have typical symptoms and signs, and is not easy to attract attention. It is often found through physical examination or breast cancer screening. The following are typical signs of breast cancer.

1. Breast lumps: 80% of breast cancer patients are first diagnosed with breast lumps. Patients often find breast lumps inadvertently, which are mostly single, hard, with irregular edges and a less smooth surface. Most breast cancers are painless masses, and only a few have varying degrees of dull pain or tingling.
2. Nipple discharge: If blood, serous, milk, or pus is discharged from the nipple during non-pregnancy, or if breastfeeding is stopped for more than half a year, it is called nipple discharge. There are many reasons for nipple discharge, and the common diseases include intraductal papilloma, breast hyperplasia, mammary duct dilatation and breast cancer. Unilateral single-port bloody discharge should be further examined, if accompanied by breast masses, more attention should be paid.
3. Skin changes: Breast cancer can cause skin changes with various signs. The most common is that the tumor invades the breast skin and the deep pectoralis fascia ligament, which shortens and loses its elasticity, pulls the skin of the corresponding part, and causes "dimples", that is, a small depression in the breast skin, like a small dimple. If cancer cells block the lymphatic vessels, there

will be "orange peel-like changes", that is, many small dot-like depressions appear on the breast skin, just like orange peels. In advanced breast cancer, cancer cells infiltrate into the skin and grow along the lymphatic vessels, glands, or fibrous tissues, forming scattered hard nodules in the skin around the main cancer focus, the so-called "skin satellite nodules".

4. Abnormal nipple and areola: The tumor is located in or near the deep part of the nipple, which can cause inverted nipple. When the tumor is far away from the nipple, the large duct in the breast is invaded and shortened, which can also cause the nipple to invert or lift. Eczema-like carcinoma of the nipple is characterized by itching, erosion, ulceration, crusting, scaling, and burning of the nipple skin, leading to inverted nipple.
5. Axillary lymph node swelling: More than one third of breast cancer patients admitted to a large hospital have axillary lymph node metastasis. There may be swelling of the ipsilateral axillary lymph nodes in the initial stage, and the swollen lymph nodes are hard, scattered, and pushable. As the disease progresses, the lymph nodes gradually fuse, and adhere to and fixate with the skin and surrounding tissues. In the late stage, the metastatic lymph nodes can be felt on the clavicle and the contralateral armpit.



Treatment of breast cancer

1. Tumor resection: surgical removal of the tumor and its adjacent tissues, leaving most of the breast. For limited breast cancer, this method is the best choice.
2. Complete mastectomy: If more than one tumor is found, or the tumor is quite large, consider removing the entire breast (usually including the lymph nodes on the same side). Most women whose breasts have been removed can consider breast reconstruction. The patient should discuss with the doctor whether it is suitable for reconstruction and which method to use to reconstruct the breast.
3. Radiotherapy: Use precise radiation to illuminate the tumor to kill cancer cells and shrink the tumor.
4. Drug therapy: use one or more drugs to inhibit cancer cells.
5. Radiation therapy and drug therapy are generally used as adjuvant treatments after surgery. In the early stage of breast cancer, doctors will assess the patient's condition and may give anti-estrogen therapy. According to statistics, about one-third of breast cancer patients are effective after receiving anti-estrogen therapy.

Other influencing factors



Controllable factors

1. Lifestyle: People who like high-fat, high-sugar, and high-calorie foods have a higher incidence of breast cancer. Frequent staying up late and lack of exercise can also increase the risk of breast cancer.

- 2. Obesity: Obese people have twice the risk of breast cancer.
- 3. Smoking: Smoking causes the incidence rate to be higher by 2/3, and women who start smoking during adolescence will increase the risk of disease by 70%.
- 4. Female childbearing experience: The incidence of breast cancer is increase with women who has never given birth, giving birth at the age of 30s, and did not breastfeed after childbirth.
- 5. Treatment experience: People who have been exposed to hormone-containing drugs or received chest radiation therapy are at higher risk of breast cancer.

 Uncontrollable factors

- 1. Age: Breast cancer mostly occurs in women between 40 and 60 years old. The older you are, the more likely you are to develop breast cancer.
- 2. Endocrine problems: Women younger than 12 years old at menarche (first menstruation) or women who have menopause after 55 years old have a higher risk of breast cancer.
- 3. Family history: People with a family history of breast cancer have a higher prevalence.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1056836	CYP1B1	CG	slightly high risk caused by smoking x breast cancer
RS1143627	IL1B	AG	slightly low risk caused by smoking x breast cancer
RS13181	ERCC2	TT	moderate risk caused by smoking x breast cancer
RS2234693	ESR1	CT	moderate risk caused by smoking x breast cancer
RS25487	XRCC1	CT	slightly low risk caused by smoking x breast cancer

Risk calculation

The risk value of this test is calculated based on published academic literatures on the risk of breast cancer from smoking. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of breast cancer risk between smokers and non-smokers. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all

users tested in our products, we can show the changes in the risk of breast cancer for users if they smoke relative to those who do not smoke.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The risk of breast cancer from smoking item uses the 5 polymorphic loci on and between genes such as *CYP1B1*, *XRCC1* and *ESR1*. The test results indicate that compared with all users, whether this user has a higher risk of breast cancer from smoking in the combination of genotypes of the 5 polymorphic loci tested. It does not mean the user's true risk of breast cancer from smoking. The basis of the test is mainly from the research of Chinese and Caucasian, and other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of breast cancer from smoking.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by multiple factors such as genes and environment. This study only analyzes the effect of your smoking on the risk of breast cancer from the genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of breast cancer, or you think that you are likely to suffer from breast cancer, it is recommended that you consult an authoritative health professional or clinician for help.
4. Smoking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop drinking as soon as possible to reduce the risk of disease.

Test content

The test includes 5 polymorphic loci related to the risk of breast cancer from smoking, including rs1143627, rs1056836, and rs25487.

Population samples

The basis of the test is mainly from the research of Chinese and Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to the risk of breast cancer from smoking.

Smoking x Bladder Cancer

In general, smoking can increase the risk of bladder cancer. And to a certain extent, the increase of this risk is affected by genes.

My Smoking x Bladder Cancer

Moderate elevating risk

Quit smoking knowledge

Tobacco is the largest controllable hazard affecting human health. Smoking cessation has health benefits, including a lower risk of cancer and death from cardiovascular disease. In addition to smoking cessation policies, environmental and social support, and personal determination and perseverance, appropriate smoking cessation methods should also be adopted, or a combination of multiple methods should be used. Common methods include:

- Quit smoking through willpower: Smoking cessation through willpower alone without any other supplementary means, but the success rate is very low, less than 3%.
- Drug intervention: Smoking cessation drugs can be effective in helping smokers quit their addiction. Different drugs have different efficacy, but all of them are significantly better than "Quit smoking through willpower".
- Behavioral intervention: Different levels of behavioral intervention, including speech intervention and telephone follow-up. A combination of drug interventions can greatly improve the success rate.
- E-cigarettes: E-cigarettes can be used as an alternative to tobacco, but their safety needs to be verified by further studies.
- TCM (Traditional Chinese Medicine) smoking cessation: Some studies have shown that TCM acupuncture can help smokers quit smoking by regulating endorphin levels, but this method has a high relapse rate.
- Other methods: such as Information-based smoking cessation, Aversion therapy, Hypnotherapy, etc., can be used as a second choice after drugs, behavioral intervention and other methods fail.

Knowledge

Smoking and bladder cancer

Smoking is the main cause of bladder cancer, and it is estimated that 50% of bladder cancer is related to smoking. The study found that the risk of bladder cancer in former smokers is 2.2 times that of

non-smokers, and current smokers are 4.1 times that of non-smokers.

Different genotypes interact with smoking, leading to different risks of bladder cancer. For example, the polymorphism of the rs2228001 locus of XPC gene is related to the risk of the onset of the bladder in a smoking environment. This locus is GT genotype or TT genotype of smoking compared with smokers of the TT genotype, they are more likely to develop bladder cancer.

Time of onset of bladder cancer

The onset is more common in the age of 50 and above. It is more prone to disease with age.

Prevalence of bladder cancer

In 2014, the number of new cases of bladder cancer in the United States was 74,690 cases. The overall incidence rate in China from 2003 to 2007 was 6.69 per 100,000 people.

Typical symptoms of bladder cancer

- Urine: Usually, the spontaneous symptoms of bladder cancer are carried in urine. The urine may be pink or deep red, or the color of the urine under the eyes is normal, but the urine routine test shows that there are more red blood cells. Early bladder cancer with non-urinary, usually without other discomforts such as painful urination.
- Changes in urination habits: including frequent urination (significant increase in the frequency of urination), urgency (repeated wanting to have a small bowel movement), painful urination (pain when urinating), poor urination, increased nocturia, etc.
- Symptoms caused by cancer cell metastasis: inability to use stool, unilateral low back pain, poor desire, significant weight loss, fatigue, pain, etc.

Treatment of bladder cancer

1. Surgery treatment: The vast majority of bladder cancer can be treated with surgery. The choice of surgical method depends on the different stages of bladder cancer. Common types of surgical procedures include: transurethral resection of bladder tumors and cystectomy.
2. Infusion therapy: The doctor infuse the drug into the bladder through a catheter, and the drug will stay in the bladder for about 2 hours. This therapy is not suitable for advanced bladder cancer.
3. Radiotherapy: Use high-energy rays to kill cancer cells.
4. Chemotherapy: Use drugs to kill or prevent cancer cells. Commonly used drugs include: cisplatin, fluorouracil, docetaxel, paclitaxel, methotrexate, cristine and soon.
5. Immunotherapy: By affecting the patient's own immune system, it can more effectively identify and kill cancer cells. Commonly used immunotherapy includes: BCG, immune checkpoint inhibitors, etc.
6. Targeted therapy: treatment for cancer-causing mutations, such as Erdafitinib, a targeted drug.

Other influencing factors

Controllable factors

1. Toxic substances: Frequent exposure to rubber and whites may induce bladder cancer; practitioners in printing, textile, paint and other industries belong to the high-risk group for bladder cancer.
2. Drinking habits: Inadequate drinking, inadequate intake of vegetables and fruits may induce bladder cancer.

Uncontrollable factors

1. Age: Common in 50 years and above.
2. Gender: Mostly male.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS13181	ERCC2	TT	moderate risk caused by smoking x bladder cancer
RS1801270	CDKN1A	AC	slightly low risk caused by smoking x bladder cancer
RS2228000	XPC	GG	moderate risk caused by smoking x bladder cancer
RS2228001	XPC	GG	slightly high risk caused by smoking x bladder cancer
RS2567206	intergenic	GG	moderate risk caused by smoking x bladder cancer

Risk calculation

The risk value of this test is calculated based on published academic literatures related to the risk of smoking on bladder cancer. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of bladder cancer among smokers and non-smokers. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk change of bladder cancer if the user smokes, compared with the non-smoker group.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-

genetic factors are not taken into account.

Test details

Applicable situation

The smoking risk item for bladder cancer uses the 5 polymorphic loci on genes such as *CDKN1A*, *XPC*, *ERCC2* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 5 polymorphic loci detected by this test increases the risk of smoking on bladder cancer. It does not mean the user's true smoking impact on the risk of bladder cancer. The basis of the test is mainly from the research of Chinese, Pakistani and American, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of smoking on bladder cancer.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only evaluates your risk of smoking on the risk of bladder cancer at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of bladder cancer, or you think that you are likely to suffer from bladder cancer, it is recommended that you consult an authoritative health professional or clinician for help.
4. Smoking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop smoking as soon as possible to reduce the risk of disease.

Test content

The test includes 5 polymorphic loci associated with the risk of smoking on bladder cancer, including rs1801270, rs2228000, rs2567206, etc.

Population samples

The basis of the test is mainly from the research of Chinese, Pakistani and American.

Limit of detection

Limited by the current technological means and the level of scientific cognition, the test may not cover all genes or loci that cause bladder cancer from smoking.

Smoking x CHD

In general, smoking can increase the risk of coronary heart disease (CHD). And to a certain extent, the increase of this risk is affected by genes.

My Smoking x CHD

Moderate elevating risk

Quit smoking knowledge

Tobacco is the largest controllable hazard affecting human health. Smoking cessation has health benefits, including a lower risk of cancer and death from cardiovascular disease. In addition to smoking cessation policies, environmental and social support, and personal determination and perseverance, appropriate smoking cessation methods should also be adopted, or a combination of multiple methods should be used. Common methods include:

- Quit smoking through willpower: Smoking cessation through willpower alone without any other supplementary means, but the success rate is very low, less than 3%.
- Drug intervention: Smoking cessation drugs can be effective in helping smokers quit their addiction. Different drugs have different efficacy, but all of them are significantly better than "Quit smoking through willpower".
- Behavioral intervention: Different levels of behavioral intervention, including speech intervention and telephone follow-up. A combination of drug interventions can greatly improve the success rate.
- E-cigarettes: E-cigarettes can be used as an alternative to tobacco, but their safety needs to be verified by further studies.
- TCM (Traditional Chinese Medicine) smoking cessation: Some studies have shown that TCM acupuncture can help smokers quit smoking by regulating endorphin levels, but this method has a high relapse rate.
- Other methods: such as Information-based smoking cessation, Aversion therapy, Hypnotherapy, etc., can be used as a second choice after drugs, behavioral intervention and other methods fail.

Knowledge



Smoking and coronary heart disease

Smoking is a risk factor for coronary heart disease (CHD). The study found that even a small amount of exposure to active or passive smoking (average 1 cigarette/day) can increase the risk of

cardiovascular disease by 30%; with the increase in the number of smoking cigarettes, from 5 cigarettes/day to 20 cigarettes/day, the risk of morbidity increases linearly to twice, while non-smokers exposed to second-hand smoke increase the risk of coronary heart disease by 25% to 30%.

Different genotypes interact with smoking, leading to different risks of coronary heart disease. For example, the rs1799983 polymorphism of NOS3 gene is associated with the risk of coronary heart disease in a smoking environment. Smokers with GT genotype or TT genotype at this locus are more susceptible to coronary heart disease than smokers with GG genotype.

Time of onset of coronary heart disease

It mostly occurs in people over the age of 35. In developed countries, its annual deaths can account for 1/3 of the total deaths in middle-aged and elderly people.

Prevalence of coronary heart disease

According to statistics, there are about 10 cases of the disease for every 100,000 people in the average population.

Typical symptoms of coronary heart disease

The typical chest pain is caused by physical activity, emotional agitation, etc., sudden feeling of pain in the precordial area, mostly paroxysmal colic or squeezing pain, or a feeling of depression. The pain starts from the back of the breastbone or the front of the heart, and radiates upward to the left shoulder, arm, and even the little finger and ring finger. The part where chest pain disperses can also involve the neck, jaw, teeth, abdomen, etc. In addition, symptoms of gastrointestinal diseases such as upper abdominal pain, nausea, and vomiting may also occur.

Treatment of coronary heart disease

Although coronary heart disease, especially myocardial infarction, is very dangerous, the first thing to do is to solve the stenosis and infarction and restore myocardial blood supply as soon as possible, so as to quickly control the symptoms and minimize the scope and extent of myocardial necrosis. Drug treatments include sublingual nitroglycerin and other nitrate drugs, metoprolol and other β -receptor blockers, and nifedipine calcium channel blockers. Surgical procedures include heart bypass.

Other influencing factors

Controllable factors

1. Obese/overweight people have a high risk of disease.
2. Dietary factors: Unhealthy diets such as high fat, high cholesterol, and high calories can easily lead to coronary heart disease.

3. Unhealthy living habits: Such as lack of physical activity, excessive drinking, etc. can also induce this disease.



Uncontrollable factors

- 1. Genetic factors: There is a family genetic tendency.
- 2. Disease factors: High blood pressure, high blood fat, high blood sugar and diabetic patients with high incidence of coronary heart disease

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1501299	ADIPOQ-AS1	GT	slightly high risk caused by smoking x C HD
RS1799983	NOS3	GG	moderate risk caused by smoking x CHD
RS1805081	NPC1	TT	moderate risk caused by smoking x CHD
RS36071027	intergenic	CC	moderate risk caused by smoking x CHD
RS7178051	intergenic	CT	slightly low risk caused by smoking x CH D

Risk calculation

The risk value of this test is calculated based on published academic literatures related to the risk of smoking on coronary heart disease. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of coronary heart disease among smokers and non-smokers. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk change of coronary heart disease if the user smokes, compared with the non-smoker group.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The smoking risk item for coronary heart disease uses the 5 polymorphic loci on genes such as *ADIPOQ-AS1*, *NPC1*, *NOS3* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 5 polymorphic loci detected by this test increases the risk of smoking on coronary heart disease. It does not mean the user's true smoking impact on the risk of coronary heart disease. The basis of the test is mainly from the research of Asian and European, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of smoking on coronary heart disease.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only evaluates your risk of smoking on the risk of coronary heart disease at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of coronary heart disease, or you think that you are likely to suffer from coronary heart disease, it is recommended that you consult an authoritative health professional or clinician for help.
4. Smoking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop smoking as soon as possible to reduce the risk of disease.

Test content

The test includes 5 polymorphic loci associated with the risk of smoking on coronary heart disease, including rs36071027, rs7178051, rs1805081, etc.

Population samples

The basis of the test is mainly from the research of Asian and European.

Limit of detection

Limited by the current technological means and the level of scientific cognition, the test may not cover all genes or loci that cause coronary heart disease from smoking.

Smoking x Hypertension

In general, smoking can increase the risk of hypertension. And to a certain extent, the increase of this risk is affected by genes.

My Smoking x Hypertension

Moderate elevating risk

Quit smoking knowledge

Tobacco is the largest controllable hazard affecting human health. Smoking cessation has health benefits, including a lower risk of cancer and death from cardiovascular disease. In addition to smoking cessation policies, environmental and social support, and personal determination and perseverance, appropriate smoking cessation methods should also be adopted, or a combination of multiple methods should be used. Common methods include:

- Quit smoking through willpower: Smoking cessation through willpower alone without any other supplementary means, but the success rate is very low, less than 3%.
- Drug intervention: Smoking cessation drugs can be effective in helping smokers quit their addiction. Different drugs have different efficacy, but all of them are significantly better than "Quit smoking through willpower".
- Behavioral intervention: Different levels of behavioral intervention, including speech intervention and telephone follow-up. A combination of drug interventions can greatly improve the success rate.
- E-cigarettes: E-cigarettes can be used as an alternative to tobacco, but their safety needs to be verified by further studies.
- TCM (Traditional Chinese Medicine) smoking cessation: Some studies have shown that TCM acupuncture can help smokers quit smoking by regulating endorphin levels, but this method has a high relapse rate.
- Other methods: such as Information-based smoking cessation, Aversion therapy, Hypnotherapy, etc., can be used as a second choice after drugs, behavioral intervention and other methods fail.

Knowledge

Smoking and hypertension

Smoking is an important risk factor for hypertension, and there is a significant dose effect relationship. The study found that the prevalence of hypertension in smokers was significantly higher

than that of non-smokers, and with the increase in smoking, the prevalence of hypertension also gradually increased. Compared with non-smokers, the single-factor risk of hypertension in smokers can be increased by 1.55 times.

Different genotypes interact with smoking, leading to different risks of developing hypertension. For example, the rs402007 polymorphism of ADAMTS1 gene locus is associated with the risk of hypertension in a smoking environment. Smokers with GC genotype or GG genotype at this locus are more susceptible to hypertension than smokers with CC genotype.



Time of onset of hypertension

It can occur at any age, but the prevalence of hypertension increases with age.



Prevalence of hypertension

According to data released by the Ministry of Health: The average incidence rate in China is 11.88%, and the number of patients is over 160 million. In some European and American countries, the incidence rate is as high as 20%.



Typical symptoms of hypertension

Hypertension is a clinical syndrome characterized by increased systemic arterial blood pressure. The cause of most patients is unknown. Studies have shown that hypertensive patients are more likely to have other cardiovascular and cerebrovascular diseases than those with normal blood pressure, such as atherosclerosis, and affect the functions of important organs such as the heart, brain, and kidneys, and ultimately lead to the failure of these organs. Some hypertensive patients have headaches (especially back pain and morning headaches), dizziness, tinnitus (buzzing or sizzling in the ears), visual changes or fainting episodes.



Treatment of hypertension

The cause of most hypertension patients is not clear, and most patients need long-term medication to control their blood pressure. For patients with mild and early hypertension, blood pressure can be controlled within the normal range through weight loss, diet improvement, exercise, etc., while maintaining a healthy lifestyle. If the blood pressure level drops after taking the medicine, the patient should not stop the medicine by himself, otherwise, the blood pressure rebounds or is more dangerous. Although high blood pressure cannot be completely cured, by insisting on taking medication, blood pressure can be effectively controlled and the risk of various adverse consequences can be minimized.

Other influencing factors



Controllable factors

1. Dietary factors: High-sugar and high-fat diets, eating more meat and less vegetables, excessive sodium intake, and low potassium intake will increase the risk of hypertension.
2. Alcohol intake: Alcohol can raise blood pressure and promotes the increase of certain fatty acids in the blood.
3. Unhealthy living habits: Sitting for a long time and lack of exercise may increase the risk of hypertension.
4. Psychological and emotional factors: People who have been under excessive stress, anxiety, irritability, and emotional agitation for a long time have a higher risk of illness.
5. Overweight and obesity: With the increase of BMI and waist circumference, the mean blood pressure showed a significant increase trend, and the prevalence of hypertension also increased significantly.

 Uncontrollable factors

1. Age: The prevalence of hypertension increases with age.
2. Family history: Hypertension has obvious familial clustering and is obviously related to immediate family members. People with a family history of hypertension have a higher prevalence.
3. Blood glucose and blood lipid levels: Epidemiological data show that hypertension is often accompanied by many risk factors related to metabolism, including impaired glucose tolerance, dyslipidemia, etc., which are related to hypertension.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1126742	CYP4A11	AA	moderate risk caused by smoking x hypertension
RS1801133	MTHFR	GG	moderate risk caused by smoking x hypertension
RS6495308	CHRNA3	CC	moderate risk caused by smoking x hypertension
RS7121	GNAS	TT	moderate risk caused by smoking x hypertension
RS73598374	ADA	CC	moderate risk caused by smoking x hypertension

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to the risk of smoking on hypertension. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of hypertension among smokers and non-smokers. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk change of hypertension if the user smokes, compared with the non-smoker group.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The smoking risk item for hypertension uses the 9 polymorphic loci on genes such as *MTHFR*, *CYP4A11*, *ADAMTS1* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 9 polymorphic loci detected by this test increases the risk of smoking on hypertension. It does not mean the user's true smoking impact on the risk of hypertension. The basis of the test is mainly from the research of East Asian and Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the risk of smoking on hypertension.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only evaluates your risk of smoking on the risk of hypertension at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
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3. If you have a family history of hypertension, or you think that you are likely to suffer from hypertension, it is recommended that you consult an authoritative health professional or clinician for help.
 4. Smoking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop smoking as soon as possible to reduce the risk of disease.

Test content

The test includes 9 polymorphic loci associated with the risk of smoking on hypertension, including rs1801133, rs1126742, rs402007, etc.

Population samples

The basis of the test is mainly from the research of East Asian and Caucasian.

Limit of detection

Limited by the current technological means and the level of scientific cognition, the test may not cover all genes or loci that cause hypertension from smoking.

Smoking x Lung Cancer

In general, smoking can increase the risk of lung cancer. And to a certain extent, the increase of this risk is affected by genes.

My Smoking x Lung Cancer

Slightly low elevating risk

Quit smoking knowledge

Tobacco is the largest controllable hazard affecting human health. Smoking cessation has health benefits, including a lower risk of cancer and death from cardiovascular disease. In addition to smoking cessation policies, environmental and social support, and personal determination and perseverance, appropriate smoking cessation methods should also be adopted, or a combination of multiple methods should be used. Common methods include:

- Quit smoking through willpower: Smoking cessation through willpower alone without any other supplementary means, but the success rate is very low, less than 3%.
- Drug intervention: Smoking cessation drugs can be effective in helping smokers quit their addiction. Different drugs have different efficacy, but all of them are significantly better than "Quit smoking through willpower".
- Behavioral intervention: Different levels of behavioral intervention, including speech intervention and telephone follow-up. A combination of drug interventions can greatly improve the success rate.
- E-cigarettes: E-cigarettes can be used as an alternative to tobacco, but their safety needs to be verified by further studies.
- TCM (Traditional Chinese Medicine) smoking cessation: Some studies have shown that TCM acupuncture can help smokers quit smoking by regulating endorphin levels, but this method has a high relapse rate.
- Other methods: such as Information-based smoking cessation, Aversion therapy, Hypnotherapy, etc., can be used as a second choice after drugs, behavioral intervention and other methods fail.

Knowledge



Smoking and lung cancer

Studies have shown that smoking and tobacco exposure are most closely related to lung cancer. The "Guidelines for Screening and Early Diagnosis and Treatment of Lung Cancer in China (2021)" pointed

out that current smokers are 13.1 times more likely to develop lung cancer and 11.5 times more likely to die from it than non-smokers. Former smokers are 4.06 times more likely to develop lung cancer and 4.10 times more likely to die than non-smokers. There is also a dose-response relationship between smoking and lung cancer. The more smokers smoke, the higher the risk of lung cancer.

Different genotypes interact with smoking, leading to different risks of lung cancer. For example, the polymorphism of OGG1 gene locus rs1052133 is associated with the risk of lung cancer in a smoking environment. Smokers with the CG or GG genotype at this locus are more likely to develop lung cancer than smokers with the CC genotype.

Time of onset of lung cancer

The disease can occur at any age, and people over 50 years of age who have a history of smoking may be the most susceptible to lung cancer.

Prevalence of lung cancer

Prevalence rate: 49 per 100,000 people. The cancer with the highest incidence and mortality rate among men worldwide is lung cancer. Among women, the incidence of lung cancer ranks third, and the mortality rate ranks second, second only to breast cancer. In 2012, there were 1.82 million new lung cancer patients worldwide, and 1.56 million eventually died of lung cancer, accounting for 19.4% of deaths due to cancer. The highest incidence of lung cancer is in North America, Europe and East Asia. In 2012, more than one-third of new patients appeared in China, while the incidence of lung cancer in Africa and South Asia was much lower.

Typical symptoms of lung cancer

The clinical manifestations of lung cancer are more complicated. The presence, severity, and time of symptoms and signs depend on the tumor site, pathological type, metastasis, and complications, as well as differences in patient response and tolerance. The early symptoms of lung cancer are often mild and may even be uncomfortable. The symptoms of central lung cancer appear early and severe, and the symptoms of peripheral lung cancer appear late and mild, even asymptomatic, and are often found during physical examination. The symptoms of lung cancer are roughly divided into: local symptoms, systemic symptoms, extrapulmonary symptoms, infiltration and metastasis symptoms.

1. Local symptoms: Including cough, blood or hemoptysis in sputum, chest pain, chest tightness, shortness of breath and hoarseness.
2. Systemic symptoms: Including fever, weight loss and cachexia.
3. Extrapulmonary symptoms: Due to some special active substances (including hormones, antigens, enzymes, etc.), patients may have one or more extrapulmonary symptoms, which often appear before other symptoms, and may disappear or appear with the growth and decline of tumors. Pulmonary osteoarthritis is more common in clinical practice
4. External invasion and metastasis symptoms: Including lymph node metastasis, pleural invasion and/metastasis, superior vena cava syndrome, kidney metastasis, digestive tract metastasis, bone

metastasis, central nervous system symptoms, heart invasion, metastasis and peripheral nervous system symptoms.



Treatment of lung cancer

The treatment of lung cancer includes surgical treatment, radiotherapy, chemotherapy and immunotherapy. Surgical treatment has been recognized as the first choice for the treatment of lung cancer, and the treatment plan should be selected based on the clinical stage of lung cancer. Radical resection is so far the only possible treatment for lung cancer patients to be cured and return to normal life. Before surgery, it is necessary to assess whether the patient tolerates the surgery. These tests usually include: clinical physical examination, pulmonary ventilation function, blood test and so on. For marginal patients, lung perfusion scans should be used to more accurately assess lung function, or endurance tests and coronary angiography should be used to assess heart function.

Other influencing factors



Controllable factors

1. Dietary factors: A large amount of β -carotene intake or excessive vitamin E intake may increase the risk of lung cancer.
2. Severe alcohol abuse: Excessive intake of alcohol can increase the risk of lung cancer.
3. Hormone replacement therapy: The use of hormone therapy such as estrogen and progesterone may increase the risk of lung cancer.
4. Air pollution: Car exhaust, indoor soot and other air pollutants containing nitrogen dioxide, fine particles (PM_{2.5}) and sulfate aerosols may increase the risk of lung cancer; radon is a colorless and odorless radioactive gas that may be released from foundation soil, building materials and decoration materials, and it is the second most common cause of lung cancer in the United States; asbestos can cause many lung diseases, including lung cancer.
5. Other lung carcinogens: Certain metals (aluminum products, cadmium and its compounds, etc.), some combustion products (incomplete combustion products, gas, etc.), ionizing radiation (X-rays, gamma rays, etc.), some harmful gases (methyl ether, sulfur mustard, etc.), rubber products and crystalline silica dust are all carcinogens associated with lung cancer.



Uncontrollable factors

Family history: People with a family history of lung cancer have a higher prevalence.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1042522	TP53	CC	slightly high risk caused by smoking x lung cancer
RS1052133	OGG1	CG	slightly high risk caused by smoking x lung cancer
RS1801272	CYP2A6	AA	slightly low risk caused by smoking x lung cancer
RS2279744	MDM2	GT	slightly low risk caused by smoking x lung cancer
RS2606345	CYP1A1	AC	slightly low risk caused by smoking x lung cancer

For display consideration, only some genetic details are displayed.

Risk calculation

The risk value of this test is calculated based on published academic literatures related to the risk of smoking on lung cancer. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of lung cancer among smokers and non-smokers. We use the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk change of lung cancer if the user smokes, compared with the non-smoker group.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The smoking risk item for lung cancer uses the 7 polymorphic loci on genes such as *CYP1A1*, *CYP2A6*, *TP53* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 7 polymorphic loci detected by this test increases the risk of smoking on lung cancer. It does not mean the user's true smoking impact on the risk of lung cancer. The basis of the test is mainly from the research of Chinese and Italian, other populations are for reference only.

Notification

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1. The test may not cover all genes or loci that affect the risk of smoking on lung cancer.
 2. The test results have no clinical significance.
 3. If you have clinical needs, please contact a qualified institution or doctor.
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How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only evaluates your risk of smoking on the risk of lung cancer at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of lung cancer, or you think that you are likely to suffer from lung cancer, it is recommended that you consult an authoritative health professional or clinician for help.
4. Smoking as a single factor can significantly increase the risk of cancer, cardiovascular and other diseases. Therefore, even if the genetic test results show that the risk of disease is low, you should stop smoking as soon as possible to reduce the risk of disease.

Test content

The test includes 7 polymorphic loci associated with the risk of smoking on lung cancer, including rs2606345, rs1801272, rs1042522, etc.

Population samples

The basis of the test is mainly from the research of Chinese and Italian.

Limit of detection

Limited by the current technological means and the level of scientific cognition, the test may not cover all genes or loci that cause lung cancer from smoking.

Physical Activity x T2D

Physical activity plays a protective role in type 2 diabetes (T2D). And to a certain extent, the strength of this effect is affected by genes.

My Physical Activity x T2D

Moderate protective role

Physical activity guidelines

"Exercise is a good doctor". Appropriate exercise and physical activity can effectively prevent, treat diseases and maintain health. There are individual and effect differences in exercise intervention. The key is to achieve as many health benefits as possible while avoiding exercise risks; at the same time, individual exercises should be carried out step by step and according to their ability. The World Health Organization (WHO) recommends in its 2020 physical activity guidelines:

- Children and adolescents(aged 5–17 years): Children and adolescents should do at least an average of 60 min/day of moderate-to-vigorous intensity, mostly Aerobic physical activity, across the week. Vigorous-intensity aerobic activities, as well as those that strengthen muscle and bone should be incorporated at least 3 days a week;
- Adults(aged 18–64 years): Adults should do at least 150–300 min of moderate-intensity aerobic physical activity, or at least 75–150 min of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate-intensity and vigorous-intensity activity throughout the week for substantial health benefits. Adults should also do Resistance exercise at moderate or greater intensity that involve all major muscle groups on 2 or more days a week, as these provide additional health benefits;
- Older adults(aged 65 years and older): As part of their weekly physical activity, older adults should do varied Multicomponent physical activity that emphasises functional balance and strength training at moderate or greater intensity on 3 or more days a week, to enhance functional capacity and to prevent falls;
- Special groups also need scientific and reasonable exercise.

Knowledge



Exercise and type 2 diabetes

Exercise occupies an important position in the comprehensive management of patients with type 2 diabetes (T2D). Regular exercise can help control blood sugar, reduce cardiovascular risk factors,

reduce weight, and improve happiness, and it has a significant preventive effect on people at high risk of diabetes. The study found that regular exercise for more than 8 weeks can reduce the glycosylated hemoglobin of type 2 diabetic patients by 0.66%; the mortality rate of diabetic patients who insist on regular exercise for 12 to 14 years is significantly reduced.

Different genotypes have different protective effects on type 2 diabetes under the interaction with exercise. For example, the rs17036314 polymorphism of PPARG gene can reduce the risk of type 2 diabetes under exercise conditions. Exercisers with GC and GG genotypes are less likely to develop type 2 diabetes than those with GG genotypes.



Time of onset of type 2 diabetes

It is more common in middle-aged and elderly people, 35-55 years old is the main age group of onset.



Prevalence of type 2 diabetes

Diabetes has become a global epidemic. There are currently 40 million diabetic patients in China, of which the incidence of type 2 diabetes is about 9.7%.



Typical symptoms of type 2 diabetes

There is no obvious discomfort in the early stage. As the disease progresses, symptoms of "three more and one less" appear: polydipsia, polyphagia, polyuria, weight loss.



Treatment of type 2 diabetes

Comprehensive treatment for type 2 diabetes includes diet control, exercise therapy, oral hypoglycemic drugs, insulin, blood glucose monitoring, diabetes education, etc.

Other influencing factors



Controllable factors

1. Obesity: People who are overweight ($\text{BMI} \geq 24 \text{ kg/m}^2$), obese ($\text{BMI} \geq 28 \text{ kg/m}^2$), or central obesity (male waist circumference $\geq 90 \text{ cm}$, female waist circumference $\geq 85 \text{ cm}$) are prone to type 2 diabetes.
2. Diet: Long-term consumption of high-fat and high-sugar foods will increase the risk of type 2 diabetes.
3. Mental factors: Long-term stress, anxiety, etc. can induce type 2 diabetes.
4. Hypertension and hyperlipidemia: A history of hypertension and hyperlipidemia will increase the risk of type 2 diabetes.



Uncontrollable factors

- 1. Age: The prevalence of type 2 diabetes increases with age, and it is more common in people over 55 years of age.
- 2. Family history: Type 2 diabetes has a relatively obvious tendency to cluster disease in the family. If there are diabetic patients among parents, children, or brothers and sisters, the risk of developing the disease will be greatly increased.
- 3. Race: Type 2 diabetes has a higher risk of developing the disease in ethnic groups such as South Asian, Pacific Islander, Hispanic, and Native American.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS17036314	PPARG	CG	exercise has a strong protective effect on type 2 diabetes
RS1801282	PPARG	CC	exercise has a moderate protective effect on type 2 diabetes
RS5404	SLC2A2	CC	exercise has a moderate protective effect on type 2 diabetes
RS3758947	intergenic	AG	exercise has a weak protective effect on type 2 diabetes
RS5393	SLC2A2	--	Not Detected

Risk calculation

The risk value of this test is calculated based on published academic literatures related to the protective effect of exercise on type 2 diabetes. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of type 2 diabetes among the people who exercise and those who do not exercise. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk change of type 2 diabetes if the user exercises, compared with the non-exercise group.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The protective effects of exercise on type 2 diabetes item uses the 5 polymorphic loci on genes such as *PPARG*, *SLC2A2* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 5 polymorphic loci detected by this test increases the protective effects of exercise on type 2 diabetes. It does not mean the user's true impact on the protective effects of exercise on type 2 diabetes. The basis of the test is mainly from the research of Finn, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the protective effect of exercise on type 2 diabetes.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only evaluates the impact of your exercise on the protection of type 2 diabetes at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of type 2 diabetes, or you think that you are likely to suffer from type 2 diabetes, it is recommended that you consult an authoritative health professional or clinician for help.
4. Exercise as a single factor can significantly improve the progression of cardiovascular and other diseases. Therefore, even if the genetic test results show that it has a low protective effect on the disease, scientific and reasonable exercise should be carried out to reduce the risk of disease.

Test content

The test includes 5 polymorphic loci associated with the protective effect of exercise on type 2 diabetes, including rs17036314, rs5404, rs3758947, etc.

Population samples

The basis of the test is mainly from the research of Finn.

Limit of detection

Limited by the current technological means and the level of scientific cognition, the test may not cover all genes or loci related to the protective effect of exercise on type 2 diabetes.

Physical Activity x Hypertension

Physical activity plays a protective role in hypertension. And to a certain extent, the strength of this effect is affected by genes.

My Physical Activity x Hypertension

Moderate protective role

Physical activity guidelines

"Exercise is a good doctor". Appropriate exercise and physical activity can effectively prevent, treat diseases and maintain health. There are individual and effect differences in exercise intervention. The key is to achieve as many health benefits as possible while avoiding exercise risks; at the same time, individual exercises should be carried out step by step and according to their ability. The World Health Organization (WHO) recommends in its 2020 physical activity guidelines:

- Children and adolescents(aged 5–17 years): Children and adolescents should do at least an average of 60 min/day of moderate-to-vigorous intensity, mostly Aerobic physical activity, across the week. Vigorous-intensity aerobic activities, as well as those that strengthen muscle and bone should be incorporated at least 3 days a week;
- Adults(aged 18–64 years): Adults should do at least 150–300 min of moderate-intensity aerobic physical activity, or at least 75–150 min of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate-intensity and vigorous-intensity activity throughout the week for substantial health benefits. Adults should also do Resistance exercise at moderate or greater intensity that involve all major muscle groups on 2 or more days a week, as these provide additional health benefits;
- Older adults(aged 65 years and older): As part of their weekly physical activity, older adults should do varied Multicomponent physical activity that emphasises functional balance and strength training at moderate or greater intensity on 3 or more days a week, to enhance functional capacity and to prevent falls;
- Special groups also need scientific and reasonable exercise.

Knowledge



Exercise and hypertension

Exercise can improve blood pressure levels. Aerobic exercise reduced systolic blood pressure by 3.84 mmHg and diastolic blood pressure by 2.58 mmHg on average. Studies have found that regular

exercise in patients with hypertension can reduce the risk of cardiovascular death and all-cause death. Therefore, it is recommended that non-hypertensive people (in order to reduce the risk of hypertension) or hypertensive patients (in order to reduce blood pressure), in addition to the activities of daily living, exercise regularly every week. The form of exercise can be aerobic, resistance, and stretching. Mainly aerobic exercise, anaerobic exercise as a supplement. The intensity of exercise varies from person to person. High-risk patients need to be evaluated before exercise.

Different genotypes have different protective effects on hypertension under the interaction with exercise. For example, the rs10177833 polymorphism of SLC4A5 gene can reduce the risk of hypertension under exercise conditions. Exercisers with CA and AA genotypes are less susceptible to hypertension than those with CC genotypes.



Time of onset of hypertension

It can occur at any age, but the prevalence of hypertension increases with age.



Prevalence of hypertension

According to data released by the Ministry of Health: The average incidence rate in China is 11.88%, and the number of patients is over 160 million. In some European and American countries, the incidence rate is as high as 20%.



Typical symptoms of hypertension

Hypertension is a clinical syndrome characterized by increased systemic arterial blood pressure. The cause of most patients is unknown. Studies have shown that hypertensive patients are more likely to have other cardiovascular and cerebrovascular diseases than those with normal blood pressure, such as atherosclerosis, and affect the functions of important organs such as the heart, brain, and kidneys, and ultimately lead to the failure of these organs. Some hypertensive patients have headaches (especially back pain and morning headaches), dizziness, tinnitus (buzzing or sizzling in the ears), visual changes or fainting episodes.



Treatment of hypertension

The cause of most hypertension patients is not clear, and most patients need long-term medication to control their blood pressure. For patients with mild and early hypertension, blood pressure can be controlled within the normal range through weight loss, diet improvement, exercise, etc., while maintaining a healthy lifestyle. If the blood pressure level drops after taking the medicine, the patient should not stop the medicine by himself, otherwise, the blood pressure rebounds or is more dangerous. Although high blood pressure cannot be completely cured, by insisting on taking medication, blood pressure can be effectively controlled and the risk of various adverse consequences can be minimized.

Other influencing factors

Controllable factors

1. Dietary factors: High-sugar and high-fat diets, eating more meat and less vegetables, excessive sodium intake, and low potassium intake will increase the risk of hypertension.
2. Tobacco and alcohol intake: Smokers or passive smokers have an increased risk of hypertension. Alcohol can raise blood pressure and promotes an increase of certain fatty acids in the blood.
3. Unhealthy living habits: Sitting for a long time may increase the risk of hypertension.
4. Psychological and emotional factors: People who have been under excessive stress, anxiety, irritability, and emotional agitation for a long time have a higher risk of illness.
5. Overweight and obesity: With the increase of BMI and waist circumference, the mean blood pressure showed a significant increase trend, and the prevalence of hypertension also increased significantly.

Uncontrollable factors

1. Age: The prevalence of hypertension increases with age.
2. Family history: Hypertension has obvious familial clustering and is obviously related to immediate family members. People with a family history of hypertension have a higher prevalence.
3. Blood glucose and blood lipid levels: Epidemiological data show that hypertension is often accompanied by many risk factors related to metabolism, including impaired glucose tolerance, dyslipidemia, etc., which are related to hypertension.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS10177833	SLC4A5	AC	slightly stronger protective effect produced by physical activity x hypertension
RS198389	NPPB	AG	moderate protective effect produced by physical activity x hypertension

Risk calculation

The risk value of this test is calculated based on published academic literatures related to the protective effect of exercise on hypertension. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of hypertension among the people who exercise and those who do not exercise. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all

users. By comparing with the average risk of all users tested in our products, we can give the user's risk change of hypertension if the user exercises, compared with the non-exercise group.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The protective effects of exercise on hypertension item uses the 2 polymorphic loci on genes such as *SLC4A5*, *NPPB* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 2 polymorphic loci detected by this test increases the protective effects of exercise on hypertension. It does not mean the user's true impact on the protective effects of exercise on hypertension. The basis of the test is mainly from the research of African American and Chinese, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the protective effect of exercise on hypertension.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only evaluates the impact of your exercise on the protection of hypertension at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of hypertension, or you think that you are likely to suffer from hypertension, it is recommended that you consult an authoritative health professional or clinician for help.
4. Exercise as a single factor can significantly improve the progression of cardiovascular and other diseases. Therefore, even if the genetic test results show that it has a low protective effect on the

disease, scientific and reasonable exercise should be carried out to reduce the risk of disease.

 Test content

The test includes 2 polymorphic loci associated with the protective effect of exercise on hypertension, including rs10177833 and rs198389.

 Population samples

The basis of the test is mainly from the research of African American and Chinese.

 Limit of detection

Limited by the current technological means and the level of scientific cognition, the test may not cover all genes or loci related to the protective effect of exercise on hypertension.

Physical Activity x CHD

Physical activity plays a protective role in coronary heart disease (CHD). And to a certain extent, the strength of this effect is affected by genes.

My Physical Activity x CHD

Slightly weak protective role

Physical activity guidelines

"Exercise is a good doctor". Appropriate exercise and physical activity can effectively prevent, treat diseases and maintain health. There are individual and effect differences in exercise intervention. The key is to achieve as many health benefits as possible while avoiding exercise risks; at the same time, individual exercises should be carried out step by step and according to their ability. The World Health Organization (WHO) recommends in its 2020 physical activity guidelines:

- Children and adolescents(aged 5–17 years): Children and adolescents should do at least an average of 60 min/day of moderate-to-vigorous intensity, mostly Aerobic physical activity, across the week. Vigorous-intensity aerobic activities, as well as those that strengthen muscle and bone should be incorporated at least 3 days a week;
- Adults(aged 18–64 years): Adults should do at least 150–300 min of moderate-intensity aerobic physical activity, or at least 75–150 min of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate-intensity and vigorous-intensity activity throughout the week for substantial health benefits. Adults should also do Resistance exercise at moderate or greater intensity that involve all major muscle groups on 2 or more days a week, as these provide additional health benefits;
- Older adults(aged 65 years and older): As part of their weekly physical activity, older adults should do varied Multicomponent physical activity that emphasises functional balance and strength training at moderate or greater intensity on 3 or more days a week, to enhance functional capacity and to prevent falls;
- Special groups also need scientific and reasonable exercise.

Knowledge



Exercise and coronary heart disease

Lack of exercise is the main risk factor for coronary heart disease (CHD). The study found that even after adjusting for traditional risk factors (such as obesity, alcohol consumption, etc.), lack of exercise

is still responsible for 12.2% of myocardial infarctions worldwide. A higher level of exercise intensity (referring to the intensity that most people can reach) can significantly combat the occurrence of coronary heart disease, and even less than normal exercise intensity can reduce the relative risk of coronary heart disease by 12%. Various methods and types of exercise may have a positive impact on coronary heart disease. At present, aerobic exercise is still recommended. Any single exercise method may not have a more obvious effect than joint exercise.

Different genotypes have different protective effects on coronary heart disease under the interaction with exercise. For example, the rs1042713 polymorphism of ADRB2 gene can reduce the risk of coronary heart disease under exercise conditions. Exercisers with the AA genotype are less likely to develop coronary heart disease than those with the GG and GA genotypes.

Time of onset of coronary heart disease

It mostly occurs in people over the age of 35. In developed countries, its annual deaths can account for 1/3 of the total deaths in middle-aged and elderly people.

Prevalence of coronary heart disease

According to statistics, there are about 10 cases of the disease for every 100,000 people in the average population.

Typical symptoms of coronary heart disease

The typical chest pain is caused by physical activity, emotional agitation, etc., sudden feeling of pain in the precordial area, mostly paroxysmal colic or squeezing pain, or a feeling of depression. The pain starts from the back of the breastbone or the front of the heart, and radiates upward to the left shoulder, arm, and even the little finger and ring finger. The part where chest pain disperses can also involve the neck, jaw, teeth, abdomen, etc. In addition, symptoms of gastrointestinal diseases such as upper abdominal pain, nausea, and vomiting may also occur.

Treatment of coronary heart disease

Although coronary heart disease, especially myocardial infarction, is very dangerous, the first thing to do is to solve the stenosis and infarction and restore myocardial blood supply as soon as possible, so as to quickly control the symptoms and minimize the scope and extent of myocardial necrosis. Drug treatments include sublingual nitroglycerin and other nitrate drugs, metoprolol and other β -receptor blockers, and nifedipine calcium channel blockers. Surgical procedures include heart bypass.

Other influencing factors

Controllable factors

1. Obese/overweight people have a high risk of disease.

- 2. Dietary factors: Unhealthy diets such as high fat, high cholesterol, and high calories can easily lead to coronary heart disease.
- 3. Unhealthy living habits: Excessive drinking, etc. can also induce this disease.

 Uncontrollable factors

- 1. Genetic factors: There is a family genetic tendency.
- 2. Disease factors: High blood pressure, high blood fat, high blood sugar and diabetic patients with high incidence of coronary heart disease

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1042713	ADRB2	GG	moderate protective effect produced by physical activity x CHD
RS1800588	LIPC	CC	moderate protective effect produced by physical activity x CHD

Risk calculation

The risk value of this test is calculated based on published academic literatures related to the protective effect of exercise on coronary heart disease. These literatures have found some polymorphic loci related to the risk of the disease by studying the differences in the genotypes of coronary heart disease among the people who exercise and those who do not exercise. We uses the original data in these literatures to comprehensively score the polymorphic loci involved in this test, and obtain the risk value of all users. By comparing with the average risk of all users tested in our products, we can give the user's risk change of coronary heart disease if the user exercises, compared with the non-exercise group.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of diseases induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

 Applicable situation

The protective effects of exercise on coronary heart disease item uses the 2 polymorphic loci on genes such as *ADRB2*, *LIPC* and other gene. The test results indicate that compared with all users, whether the combination of genotypes at the 2 polymorphic loci detected by this test increases the protective effects of exercise on coronary heart disease. It does not mean the user's true impact on the protective effects of exercise on coronary heart disease. The basis of the test is mainly from the research of European, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect the protective effect of exercise on coronary heart disease.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only evaluates the impact of your exercise on the protection of coronary heart disease at a genetic level. It does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.
3. If you have a family history of coronary heart disease, or you think that you are likely to suffer from coronary heart disease, it is recommended that you consult an authoritative health professional or clinician for help.
4. Exercise as a single factor can significantly improve the progression of cardiovascular and other diseases. Therefore, even if the genetic test results show that it has a low protective effect on the disease, scientific and reasonable exercise should be carried out to reduce the risk of disease.

Test content

The test includes 2 polymorphic loci associated with the protective effect of exercise on coronary heart disease, including rs1042713 and rs1800588.

Population samples

The basis of the test is mainly from the research of European.

Limit of detection

Limited by the current technological means and the level of scientific cognition, the test may not cover all genes or loci related to the protective effect of exercise on coronary heart disease.



Skin Characteristics

17 Reports

Item

Genetic risk

Anti-Tanning Reaction Ability

Weak



Antioxidant Ability

Strong



Anti-Glycation Ability

Strong



Anti-Freckle Ability

Strong



Skin Moisturizing Ability

Strong



Ability To Resist Acne

Moderate



Ability To Resist Stretch Marks

Weak



Ability To Resist Senile Plaques

Strong



Ability To Resist Sunburn

Moderate



Skin Oil Control Ability

Moderate



Ability To Resist Rosacea

Strong



Ability To Resist Cellulite

Weak



Ability To Resist Varicose Veins

Moderate



Anti-Skin Photoaging Ability

Moderate



Anti-Skin Wrinkle Ability

Moderate



Anti-Sagging Eyelid Ability

Weak



Collagen Retention Ability

Moderate



Anti-Tanning Reaction Ability

Tanning reaction refers to the process of melanin synthesis in the skin when the skin receives ultraviolet light. People with a weak anti-tanning response can tan if they are exposed to the sun for a long time.

My Anti-Tanning Reaction Ability

Weak

28.3% of We users are similar to me

Suggestions

- Your Resistance to tanning response is **Weak** and you are more prone to produce melanin under the same exposure conditions, so extra attention to sun protection for your skin is recommended
- Skin care habits such as paying attention to Sunscreen and doing After-sun whitening repair are beneficial for resistance to tanning
- Eat sun-protective foods such as kiwi, Pomegranate and other fruits rich in Vitamin C or moderate intake of Marine biomass foods
- Daily use of skin care products containing Whitening ingredients such as vitamin C, Arbutin, Nicotinamide, etc. is effective for sun protection and whitening
- Antioxidants such as Grape seed oil, Resveratrol, L-glutathione, Vitamin E, etc. can protect the skin from damages caused by absorbing UV rays
- Physical sun protections such as hats, umbrellas, masks, etc. are also key to anti-tanning.
- Appropriate use of skin care products containing After-sun repair ingredients such as Pro-xylane is often used to repair skin damage and protect the skin barrier after sun exposure

* For dietary supplements, it is recommended that you consult an authoritative health professional or clinician for assistance.

Knowledge



Reasons for Sun Protection

When the skin receives excessive UV exposure, it will damage the epidermal cells; activate the tyrosinase, accelerate pigment synthesis, destroy the moisturizing function of the skin, dry the skin, damage the elastic fibers in the dermis, cause fine lines, and cause skin inflammation and burns under intense exposure. In the case of abnormalities, it can become pigmented skin cancer.

Sunscreen Effectiveness

When choosing a sunscreen, the effectiveness of the sunscreen products in terms of UV protection can be used for assessment.

The algorithm used by brands in Japan and the United States is the sun protection factor (SPF), while in European countries, mainly France, it is expressed as "IP". There is also PA, which is an objective assessment of the effectiveness of sunscreen cosmetics in protecting against UVA.

The smaller the SPF value, the worse the sun protection; the larger the SPF value, the better the sun protection.

PA + represents a maintenance effect, and PA +++ represents a very good maintenance effect.

sunscreen food

Vitamin C fruits: guava, kiwi, strawberries, cherry tomatoes, tomatoes or citrus are rich in vitamin C.

Yellow and red fruits and vegetables: red, yellow fruits and vegetables and dark green leafy vegetables such as carrots, mangoes, tomatoes, papaya, water spinach, etc., contain a lot of carotenoids, which help antioxidant, enhance skin resistance.

Soy products: Isoflavones in soy are a kind of phytoestrogen with antioxidant ability.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1015362	intergenic	CC	weak anti-sunburn ability
RS11703668	KIAA0930	AG	moderate anti-sunburn ability
RS9561570	DCT	TT	strong anti-sunburn ability
RS746586	intergenic	CC	weak anti-sunburn ability
RS35563099	intergenic	CC	strong anti-sunburn ability

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The capacity of skin in anti-tanning item uses 17 polymorphic loci on the genes of *HERC2*, *IRF4*, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian populations, other populations are for reference only.
2. The test may not cover all genes or loci that affect the capacity of skin in anti-tanning.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your capacity of skin in anti-tanning at the genetic level. Since This test does not involve consideration of other factors, it does not represents your true status.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect the capacity of skin in anti-tanning.

Antioxidant Ability

Antioxidant ability refers to the skin's ability to resist various free radicals and other harmful oxidizing substances. Continuous contact with the outside world, including respiration, external pollution and radiation exposure, constantly accumulate free radicals in the human body, eventually leading to skin dehydration, dullness, relaxation and aging.

My Antioxidant Ability

Strong

3.9% of We users are similar to me

Suggestions

- Your antioxidant capacity is **strong**, and normal care is recommended for you to maintain a healthy skin condition
- Antioxidant refers to antioxidant free radicals, and too much Free radicals may be relevant to lots of diseases, such as cancer. Therefore, you should control the Source of free radicals.
- The foods that can help your free radical scavenging maybe the first choice for you, such as: Fruits rich in Vitamin C as kiwifruit and Pomegranate, or Marine foods in moderation.
- Antioxidant such as Grape seed oil, [^Resveratrol], [^L-glutathione]. Vitamin E is a vital part of the body's internal defense against free radicals.
- Paying attention to sun protection, reducing smoking and other good living habits are also the key to antioxidant.

* If you need dietary supplement, it is recommended that you consult an authoritative health professional or clinician for help.

Knowledge

Antioxidant theory

Anti-oxidant refers to the abbreviation of Antioxidant free radical. Free radicals are constantly generated in the human body due to continuous contact with the outside world, including respiration (oxidation reactions), external pollution, radiation exposure, and other factors. Scientific research have shown that cancer, aging or other diseases are mostly related to the production of excessive free

radicals. The mechanism of anti-oxidation can be either directly acting on free radicals, or indirectly consuming substances that are prone to generate free radicals to prevent further reactions.

Free Radical Sources

In summary, the sources of free radicals are as follows:

1. Smoking (second-hand smoke), and alcohol abuse (each cigarette produces hundreds of millions of free radicals).
2. Radiation, ultraviolet rays, electromagnetic waves, sun exposure, or radiation therapy for cancer patients, all of which produce free radicals.
3. Environmental pollution, including air pollution, drinking water pollution, industrial wastewater pollution, soil pollution, etc.
4. The abuse of chemical drugs, such as the abuse of food additives, pesticides, fruit and vegetable pollution, drugs, and therapeutic drugs.
5. Mental conditions, excessive stress, irritability, anxiety, depression, tension and other emotional problems can also produce free radicals.

Antioxidant Food

Tomato: Tomato is rich in lycopene, which has antioxidant function. In addition, how should tomatoes be eaten to be more effective in antioxidant? That is to eat them cooked. Although the vitamin C contained in cooked or processed tomatoes (tomato sauce, tomato juice, canned tomatoes) will be destroyed, the content of lycopene can be increased several times, and the antioxidant function is also stronger. **Grapes:** The anthocyanin glycosides in grape seeds have an antioxidant capacity that is 20 times that of vitamin C and 50 times that of vitamin E, which can achieve the purpose of anti-oxidation.

Salmon: The delicious salmon contains potent omega-3 polyunsaturated fatty acids, so it has a strong antioxidant effect.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1050450	GPX1	GG	weak skin antioxidant ability
RS1800566	NQO1	GG	strong skin antioxidant ability
RS35652124	NFE2L2	CC	strong skin antioxidant ability
RS4880	SOD2	AG	moderate antioxidant ability

Test details

Applicable situation

The skin antioxidant capacity item uses 4 polymorphic loci on the genes *GPX1* and *NFE2L2*: rs1050450, RS35652124, RS1800566 and RS4880.

Notification

1. The basis of the test is mainly from the research of Caucasian populations, other populations are for reference only.
2. The test may not cover all genes or loci that affect the skin antioxidant capacity.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your skin's antioxidant capacity at the genetic level. Since This test does not involve consideration of other factors, it does not represent your true status.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, and the test report may not cover all genes or loci that affect skin's antioxidant capacity.

Anti-Glycation Ability

The glycation reaction in the skin field refers to the fact that the skin's metabolism is too slow, causing excess sugar to be free in the blood for a long time, and too much sugar will adhere to the collagen, breaking or disorder the collagen. The skin will appear yellowish, wrinkled, and rough.

My Anti-Glycation Ability

Strong

56.4% of We users are similar to me

Suggestions

1. Skin care advice

a. Daily use of anti-glycation and antioxidant products: good use of anti-glycation and anti-oxidation products can effectively improve the problem of dull skin tone and reduce the glycation reaction.

2. Life advice

a. Reduce the intake of sugar in daily life: eating too much sugar will accelerate the glycation reaction of collagen in the cells, making the skin dark yellow and loose.

b. Regular work and rest: maintain a regular life, happy mood, balanced diet, adequate sleep, and proper exercise.

Knowledge

Advanced Glycation End-products (AGEs)

Advanced glycationend products (AGEs) are the combined products of excess sugar and protein. There are two sources in the body. One is the synthesis of AGEs in the body by excess sugar and protein, and the other is the presence of food in food through eating. The AGEs are ingested into the body. AGEs can combine with the tissue cells of the body and destroy them.

AGEs harm to human body

AGEs can combine with various tissue cells of the human body and destroy these tissue cells, thus causing harm to the human body. Researches have proved that: AGEs can accelerate the aging of the human body and can cause various chronic degenerative diseases, such as diabetes, Alzheimer's disease, atherosclerosis and other diseases. Therefore, reducing AGEs can play a role in anti-aging and prevent various chronic degenerative diseases.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1800624	AGER, PBX2	AA	strong anti-glycation ability
RS1800625	AGER, PBX2	AA	strong anti-glycation ability

Test details

Applicable situation

The skin anti-glycation ability item uses 2 polymorphic loci on the *AGER* gene: rs1800624 and rs1800625.

Notification

1. This test is mainly based on Caucasian studies, and other populations are for reference only.
2. The test may not cover all genes or loci that affect skin antioxidant capacity.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your skin's antioxidant capacity at the genetic level. It does not involve consideration of other factors and does not represent your true situation.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the skin's antioxidant capacity.

Anti-Freckle Ability

Freckles refer to yellowish brown punctate pigmentation spots on facial skin. Sun exposure can induce and aggravate skin lesions.

My Anti-Freckle Ability

Strong

40.5% of We users are similar to me

Suggestions

- Your anti-freckle ability is **Strong**, it is recommended to take normal care to maintain a healthy skin condition.
- Freckle is a kind of light brown spots, which is related to genetics, sun exposure, endocrine disorders and other factors. It often appears on the forehead, cheeks and cheeks.
- Pay attention to Sun protection and Face cleansing. Good skin care habits are good for the skin.
- Use some sunscreen food in moderation, such as kiwi, Pomegranate that so rich Vitamin C, or intake the right amount of Marine food.
- Grape seed oil: Resveratrol, L-Glutathione, Vitamin E anti-oxidants, etc., can protect the skin from absorbing ultraviolet rays.
- Pay attention to sun protection, regular work and rest, etc. Good living habits is also the key to anti-freckles.

*If you need to use dietary supplements or take treatment, it is recommended that you consult an authoritative health professional or clinical doctor for help.

Knowledge



Causes of freckles

The basal layer of the skin is scattered with a certain number of pigment cells. The pigment cells continuously produce the pigment, and the pigment plays a role in resisting the damage of various rays in the universe (mainly ultraviolet rays) to the human body. Freckle spots are formed by genetically mutated black cells. The size of the mutated chromatin cells is larger than that of the ordinary chromatin cells, and the dendrites are increased and enlarged. The dendrites are full of black elements, and a single black point is revealed on the skin surface.

The difference between freckles and pigment spots

Freckles and pigmented spots are both classified as pigmented moles. There is a certain difference in morphology between the two, but the difference is not very significant. Therefore, many people mix the two together. Freckles are not unfamiliar to many families. Freckles often appear on exposed parts of the body and body, and appear as brown or light brown skin spots. They usually appear after the age of 5, with a certain degree of Heredity. Freckles are only pigmented in the basal tissues of the skin. The pigment spots are larger than the freckles, which appear as small or large black patches on the skin surface. Pigment spots are mostly found when they appear, and some pigment spots can gradually darken as they grow larger. Color pigment spots are the same as freckles, they are pigmented on the surface of the skin, and there is no malignant change.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1042602	TYR	CC	weak anti-freckle ability
RS1393350	TYR	GG	strong anti-freckle ability
RS2153271	BNC2	CC	strong anti-freckle ability
RS4911414	intergenic	GG	strong anti-freckle ability
RS4911442	NCOA6	AA	strong anti-freckle ability

Test details

Applicable situation

The skin anti-freckle ability item uses 5 polymorphic loci on *BNC2*, *NCOA6* genes and between genes: rs2153271, rs4911414, rs4911442, rs1042602 and rs1393350.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or sites that affect the skin's ability to resist freckles.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your skin's antioxidant capacity at the genetic level. It does not involve consideration of other factors and does not represent your true situation.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the skin's antioxidant capacity.

Skin Moisturizing Ability

Skin moisturizing ability usually refers to the skin's ability to retain water and prevent dryness, sensitivity and other symptoms of skin moisture loss. Strong skin moisturizing ability can ensure that the skin has sufficient water channels and sufficient natural moisturizing factors to make the skin moist and transparent.

My Skin Moisturizing Ability

Strong

27.9% of We users are similar to me

Suggestions

1. Skin care recommendations

a. Use the correct skin care steps: In the skin care process, what we have to do is to moisturize first and then moisturize. Therefore, the general skin care step is to replenish moisture: toner or lotion replenishes moisture to the skin. Moisturizing: Essences, lotions, and creams consolidate and form a protective film for the skin, thereby tightly locking in moisture. The mask is generally based on moisturizing, after applying the mask, you must remember to do skin care in time and thoroughly moisturize.

b. Skin care management of different skin types: For sensitive skin, it is best to choose moisturizing products that do not contain lead and mercury; for oily skin, attention should be paid to clean, oil control, and hydration integration; for mixed skin, it is best to follow the climate conditions in different regions Adjust the maintenance plan with the skin condition of the day.

2. Life advice

a. Remember to wash your face with mild running water in the morning, so that the protective film on the skin's surface will not be damaged.

b. Be sure to add enough plain water every day, and don't wait until you are thirsty to think of drinking water.

c. Avoid spicy and greasy foods. Drink less and try not to smoke, which is harmful to the skin.

Knowledge

The three defenses of the skin moisturizing system

The first line of defense: the sebum film. The sebum membrane is the most important layer of skin's moisture retention. It can effectively lock moisture, prevent excessive evaporation of skin moisture, and prevent large amounts of external moisture and certain substances from penetrating. As a result, the moisture content of the skin remains normal.

The second line of defense: stratum corneum lipids. The lipids between the stratum corneum cells like cement. Keratinocytes+ lipids form the stratum corneum. Just like the bricks and cement on the wall, it can prevent the loss of moisture. Many people excessively degreasing and frequent physical friction. If the stratum corneum is damaged, the skin will become sensitive and easily dry and desquamated.

The third line of defense: natural moisturizing factor. There are various natural moisturizing factors in the stratum corneum, which are different from lipids. Moisturizing factors can absorb water and lock in moisture like a sponge. However, excessive exfoliation and exfoliation will cause these moisturizing factors to be lost.

Causes of Dry Skin

- a. Genetic factors.
- b. As the age increases, the skin's natural moisturizing factors are lost, sebaceous gland secretion is insufficient, mucopolysaccharide synthesis ability is reduced, cell metabolism is reduced, and the accumulation of old dead keratin is caused, which affects the skin's moisturizing ability.
- c. The keratin is hyperproliferative and tighter, making it impossible to supply water to each cell.
- d. The relative humidity of the external environment decreases, which speeds up the evaporation of epidermal moisture.
- e. Excessive sun exposure.
- f. Many improper maintenance methods can also cause dry skin. For example, using alkali-containing facial soap, over-cleaning, or using hot water with high temperature to wash your face, etc., will destroy the sebum film.
- g. Improper diet, and excessive smoking and alcohol.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137852931	ST14	GG	strong skin moisturizing ability

RS61816761	FLG	GG	strong skin moisturizing ability
RS17553719	AQP3	TT	strong skin moisturizing ability
RS11103631	intergenic	AG	moderate skin moisturizing ability

Test details

Applicable situation

The skin moisturizing ability item uses 4 polymorphic loci on *AQP3*, *ST14*, *FLG* genes and between them: rs17553719, rs137852931, rs61816761, and rs11103631.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or sites that affect the skin's ability to moisturize.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your skin's moisturizing ability at the genetic level. As it does not involve consideration of other factors, it does not represent your true situation.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to skin moisturizing ability.

Ability To Resist Acne

Acne, also known as acne vulgaris, which is closely related to factors such as excessive sebum secretion, clogged hair follicle sebaceous ducts, bacterial infection and inflammation.

My Ability To Resist Acne

Moderate

24.7% of We users are similar to me

Suggestions

- Your ability to resist acne is **Medium**, It is recommended to pay extra attention to acne protection.
- Cleansing, oil controlling and moisturizing Good skin care habits are essential to prevent acne.
- Try not to eat too salty, spicy, and greasy foods, and pay attention to eating healthily.
- Don't stay up late, pay attention to sun protection and so on Good living habits are also the key to fighting acne.
- Squeezing acne is not recommended, it is very likely to leave acne marks.
- If you have to squeeze out the acne, it is recommended to use the Correct way to squeeze out.
- The use of fruit acid peels etc. Non-drug therapy may be effective in the treatment of acne.
- In severe cases, topical A acid etc. are needed Drug therapy.

* If you need to use dietary supplements, it is recommended that you should consult an authoritative health professional or clinician for help.

Knowledge

Causes of Acne

The occurrence of acne (acne) is closely related to factors such as excessive sebum secretion, clogged hair follicle sebaceous ducts, bacterial infections and inflammatory reactions. After entering puberty, the level of androgens, especially testosterone, in the human body rises rapidly, which promotes the development of sebaceous glands and produces a large amount of sebum. At the same time, the abnormal keratinization of the hair follicle sebaceous ducts causes the ducts to be blocked, the sebum discharge is obstructed, and the keratinous plugs or micro-acne are formed. A variety of microorganisms in hair follicles, especially Propionibacterium acnes, multiply. Lipase produced by

Propionibacterium acnes decomposes sebum to generate free fatty acids. At the same time, it chemoattracts inflammatory cells and mediators, and ultimately induces and aggravates the inflammatory response.

Classification of acne (acne)

According to the nature and severity of acne lesions, acne is divided into grade 3 and grade 4.

Grade 1 (mild): Only acne.

Grade 2 (moderate): In addition to acne, there are some inflammatory papules.

Grade 3 (moderate): In addition to acne, there are more inflammatory papules or pustules.

Grade 4 (severe): In addition to acne, inflammatory papules and pustules, there are nodules, cysts or scars.

Graded Treatment of Acne

Level 1: Local treatment is generally adopted, and topical tretinoin preparations are the first choice.

Level 2: Combine topical tretinoin and benzoyl peroxide or antibiotics, if necessary, combine oral antibiotics.

Grade 3: Combination therapy is often required. Oral antibiotics combined with topical benzoyl peroxide and/or tretinoin are the first choice. Indicated female patients may also consider anti-androgen therapy.

Grade 4: Oral isotretinoin is the most effective treatment and can be used as a first-line treatment. For patients with more inflammatory papules and pustules, systemic antibiotics combined with topical benzoyl peroxide can also be applied first, and oral isotretinoin can be used for sequential treatment after the skin lesions are significantly improved.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1060573	C11orf49	AA	weak ability to resist acne
RS1159268	MIR548F3	AA	weak ability to resist acne
RS38055	intergenic	AG	moderate ability to resist acne
RS478304	intergenic	GT	moderate ability to resist acne
RS747650	C11orf49	TT	strong ability to resist acne

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The skin moisturizing ability item uses 6 polymorphic loci on *C11orf49*, *MIR548F3* genes and between genes: rs478304、rs1159268、rs38055、rs1060573、rs747650、rs7531806.

Notification

1. The basis of the test mainly comes from the research of Caucasian and Chinese Han population, other populations are for reference only.
 2. The test may not cover all genes or loci that affect the ability to resist acne.
-

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your skin's moisturizing ability at the genetic level. As it does not involve consideration of other factors, it does not represent your true situation.

Population samples

The basis of this test is mainly from the research of the people in Caucasian and Chinese Han population.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the skin's ability to resist acne.

Ability To Resist Stretch Marks

Stretch marks are pink or purplish red wavy patterns that appear on the skin of the abdomen during pregnancy. Stretch marks have both their own physical reasons and their prenatal care, but also genetic reasons.

My Ability To Resist Stretch Marks

Weak

5.5% of We users are similar to me

Knowledge



Causes of Stretch Marks

The human abdomen has many layers from outside to inside. They are skin, skin elastic fibers, subcutaneous fat layer, rectus abdominis composed of muscle fibers and tendons, preperitoneal fat layer and peritoneum. Under normal circumstances, the elastic fibers of the skin and the rectus abdominis maintain a certain degree of elasticity and can stretch freely within a certain limit. When a woman is pregnant for more than 3 months, the enlarged uterus protrudes from the pelvic cavity and develops into the abdominal cavity. The abdomen begins to swell. Affected by the enlarged uterus, skin elastic fibers and abdominal muscles begin to elongate. Especially after 6 months of pregnancy, it is more obvious. When a certain limit is exceeded, the elastic fibers of the skin are broken, and the rectus abdominis tendons are separated to varying degrees. As a result, irregular vertical cracks of pink or purple red appeared on the skin of the abdomen, that is stretch marks.



Diet health

- Eat more foods rich in collagen and collagen fibers to enhance skin elasticity, such as fish.
- Control sugar intake and eat less foods with high pigment content.
- Drink one cup of skimmed milk every morning and evening, eat fiber-rich vegetables, fruits, and foods rich in vitamins and minerals to increase the permeability of cell membranes and the metabolism of the skin.
- Vitamin E has anti-aging effects on the skin. Foods rich in vitamin E include cabbage, sunflower oil, rapeseed oil and so on. Vitamin A and B2 are also indispensable substances for smooth and moisturizing skin.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS7787362	intergenic	CC	weak ability to resist stretch marks
RS10798036	MIR548F1	GG	strong ability to resist stretch marks
RS35318931	SRPX	GG	weak ability to resist stretch marks
RS7594220	intergenic	AA	weak ability to resist stretch marks

Test details

Applicable situation

The skin resistance to stretch mark ability item uses 4 polymorphic loci on *MIR548F1*, *SRPX* genes and between genes: rs7787362、rs10798036、rs35318931、rs7594220.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect the ability to resist stretch marks.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your skin's moisturizing ability at the genetic level. As it does not involve consideration of other factors, it does not represent your true situation.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the skin's ability to resist stretch marks.

Ability To Resist Senile Plaques

Senile plaques, also known as liver spots and solar spots, are small dark areas on the skin. They vary in size and usually appear on the face, hands, shoulders and arms, the areas most exposed to the sun. senile plaques

My Ability To Resist Senile Plaques

Strong

13% of We users are similar to me

Suggestions

1. Skin care advice

Use sunscreen: Use sunscreen before being out in the sun to protect yourself from ultraviolet (UV) light. Try to use a sunscreen with a high sun protection factor. Reapply about every two hours, or more frequently if you are swimming or sweating.

2. Life Advice

- a. Sun protection: Sun protection protects you from free radicals in the sun. You should choose appropriate sun protection measures, such as using sunscreen properly, wearing long sleeves and pants, wearing a broadbrim, holding an umbrella, and avoiding going out between 10 a.m. and 2 p.m.
 - b. Regular routine: maintain a regular routine, a happy mood, a balanced diet, sufficient sleep, appropriate exercise, and quit bad habits such as smoking, drinking and staying up late.
 - c. Anti-senile plaque food: Eat fruits and vegetables rich in anti-senile plaque ingredients, such as tomatoes, ginger and kiwi fruit.
-

Knowledge



Characteristics of senile plaque

- 1. Senile plaque may affect people of all skin types, but they are more common in people with light skin.
 - 2. Increased pigmentation in areas that are mostly flat and oval; usually tan, brown, or black.
-

3. They usually appear on skin that has been exposed to the most sunlight for many years, such as the back of the hands, feet, face, shoulders, or upper back.

Causes of senile plaque

Senile plaque are caused by overactive pigment cells. Ultraviolet (UV) light accelerates the production of melanin. Senile plaque appear when melanin becomes "clumped" or is produced in high concentrations in areas of the skin that have experienced years of frequent and prolonged sun exposure. The use of commercial tanning lamps and tanning beds also contribute to the development of senile plaques.

When to see a doctor

Senile plaque are usually harmless and do not require medical attention. If dark or altered looking spots are present, a doctor may be needed to aid in the diagnosis. This is because these changes may be a symptom of melanoma, a serious type of skin cancer. It is best to have any new skin changes evaluated by a physician, especially if the spots:

1. are very dark in color
2. are rapidly expanding
3. have irregular borders
4. have an unusual combination of colors
5. are accompanied by symptoms such as itching, redness, tenderness to palpation or bleeding

Anti-senile plaque foods

Tomato: It has the effect of maintaining skin and eliminating freckles. Its rich lycopene and vitamin C are the effective ingredients to inhibit melanin formation. There is experimental evidence that regular consumption of tomatoes can effectively reduce melanin formation.

Ginger: Ginger contains a variety of active ingredients, including gingerol, which has a strong effect against senile plaque. By drinking ginger and honey water, the senile plaques on the face and back of hands may change, either disappearing, shrinking in varying degrees, or becoming lighter in color.

Kiwi fruit: Kiwi fruit is rich in vitamin C, which has a strong antioxidant capacity and can help the skin expel toxins, inhibit the production and deposition of melanin in the skin, strengthen the skin's resistance, brighten the skin tone, and restore the skin's elasticity and luster.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS35563099	intergenic	CC	weak ability to resist senile plaques

RS251468	PPARGC1B	CT	moderate ability to resist senile plaques
RS61866017	intergenic	GT	moderate ability to resist senile plaques

Test details

Applicable situation

The resistance to senile plaques item uses 3 polymorphic loci on and between the genes *PPARGC1B*: rs251468、rs35563099、rs61866017.

Notification

1. The basis of the test is mainly from the research of Japanese populations, other populations are for reference only.
2. The test may not cover all genes or loci that affect the resistance to senile plaques.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your resistance to senile plaques at the genetic level. Since This test does not involve consideration of other factors, it does not represents your true status.

Population samples

The basis of the test is mainly from the research of Japanese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect the resistance to senile plaques.

Ability To Resist Sunburn

Sunburn is mainly formed by sunlight purple rays, which are elliptical protrusions or smooth patches, and are dark brown or brownish black in color. It usually occurs on the face, the outer forearm, the back of the hand, and the front of the calf.

My Ability To Resist Sunburn

Moderate

37.4% of We users are similar to me

Suggestions

- Your ability to resist sunburn is **medium**, it is recommended to take normal care to maintain healthy skin condition
- Sunburn are mainly formed by excessive ultraviolet rays from the sun. The spots are elliptical protrusions or smooth patches, and the color is dark brown or light brown.
- Note that Sunscreen and good skin care habits such as After-sun repair are good for the skin
- Properly eat sunscreen foods, such as kiwi, Pomegranate and other fruits rich in Vitamin C or moderate intake of Marine food.
- Pay attention to sunscreen, regular work and rest, etc. Good living habits are also the key to resist sunburn
- Grape seed oil, Resveratrol, L-Glutathione, Vitamin E and other antioxidants, which can protect the skin against ultraviolet (UV) rays
- Non-drug therapy The use of laser technology etc. may be effective in the treatment of sunburn
- Drug therapy Alpha hydroxy acids (AHAs), salicylic acid, etc., often used to treat skin sunburn and abnormal melanin deposition

* If you need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

* Note that chemical drugs often have adverse reactions, such as increasing sunburn cell formation, inducing skin erythema, etc. Please follow the doctor's advice.

Knowledge



Causes of sunburn

The main reason for the formation of sunburn is excessive exposure to ultraviolet rays from the sun, that is, photoaging caused by the sun. At present, the main cause of sunburn in most sunburn patients may not be the single factor of sunlight and ultraviolet rays, but because long-term use of metal-containing cosmetics has caused certain damage to the skin, the skin's resistance has been reduced, and the skin's metabolic capacity has been disordered.

Characteristics of sunburn

Sunburn, also known as solar sunspots, are oval-shaped protrusions or smooth patches, and are dark brown or light brown in color. Generally, it is easy to appear on the face, the outside of the forearm, the back of the hand and the front of the lower leg. Before sunburn forms on the skin, it may first appear pain, itching and peeling, and then color spots will form in the later stage.

The difference between sunburn and freckles

Sunburn is formed by the sun. Generally, erythema, edema, blisters, pigmentation, and desquamation appear after normal skin exposure. First, there will be pain, itching, and peeling, and then spots will form later. The color of the sunburn is dark brown or light brown, protruding or smooth on the skin surface, with different sizes and clear boundaries. It is easy to appear on the outside of the forearm, the front of the calf, the back of the hand and the face. Freckles are a kind of hereditary spots, which generally appear around the age of five, and gradually increase with age, and are the most serious in puberty. Freckles occur frequently on the face, usually symmetrically, and are punctate, light yellow or yellowish brown in color. The spots change with the seasons. The sun will aggravate the freckles in summer and become lighter in winter. Sunburn is darker than freckles.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS12203592	IRF4	CC	strong ability to resist sunspot
RS35063026	intergenic	CC	strong ability to resist sunspot
RS62543565	intergenic	CC	strong ability to resist sunspot
RS6059655	RALY	GG	strong ability to resist sunspot
RS10733310	BNC2	GG	weak ability to resist sunspot

Test details

Applicable situation

The ability to resist sunburn item uses 5 polymorphic loci on and between genes in *IRF4* and *BNC2*, including rs12203592 and rs35063026, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect ability to resist sunburn.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your your emotional stability at the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to ability to resist sunburn.

Skin Oil Control Ability

On the one hand, the oil secreted by the skin has the functions of moisturizing and resisting environmental pollution, but if the skin's oil control ability is weak and there will be excessive secretion of oil, it may also cause acne.

My Skin Oil Control Ability

Moderate

34.2% of We users are similar to me

Suggestions

1. Skin care tips

- **Correct cleaning habits:** It is best to use mild cleaning products to clean the skin, and it is not suitable to use products with strong cleanliness. Because the skin's own oil is washed away, the skin will produce more oil in order to protect itself, causing the skin to become oilier and even cause acne. Do not wash your face frequently or always use facial cleanser. Frequent washing of your face will dry your facial skin.
- **Pay attention to skin hydration:** The skin maintains the balance of water and oil. When there is too much oil, the most essential thing is lack of water, and enough water should be added.
- **Apply an appropriate amount of sunscreen:** A thin layer of sunscreen on the face (except around the eyes) is enough. It should be noted that the application is repeated every two or three hours, and there is no need to apply too thick each time. Otherwise, the skin will not be able to breathe, and the pores will be blocked, causing too much oil to accumulate and cause acne.

2. Lifestyle tips

- **Reduce the consumption of greasy food:** Eating too greasy food is one reason why the skin is prone to oil. If too much oil accumulates in the body, it will be excreted through the pores of the skin. The pores will become thicker as a result, and it will also put pressure on the skin and make it easier to get oil.
- **Participate in physical exercise:** Exercise more, practice relaxation or meditation, and other exercises that can relieve stress. Studies have found that the human body produces higher levels of androgens under stress. This in turn stimulates the sebaceous glands, causing them to increase the secretion of oil.

Knowledge

Why is oily skin prone to acne?

The pH value of sebum (the oil from the skin) is weakly acidic and has a certain antibacterial effect. However, when the sebum secretion is excessive, it will lead to a decrease in the inhibitory effect on bacteria, and the normal flora existing on the skin surface will start to multiply and cause some skin diseases. Excessive sebum secretion can easily cause the hair follicle ducts that excrete sebum to be unable to excrete all of the sebum to the surface of the skin in time, which will block the hair follicle opening. Once the hair follicle opening is blocked, acne bacilli will multiply, and the surface sebum is easy to be contaminated with dust and dirt. In addition, there are a lot of skin flora, and under the action of multiple reasons, the skin begins to develop acne.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS174547	FADS1	CT	moderate skin oil control ability
RS1801282	PPARG	CC	strong skin oil control ability
RS743572	CYP17A1	AG	moderate skin oil control ability

Test details

Applicable situation

The skin oil control ability item uses 3 polymorphic loci on genes and between genes, including the *CYP17A1* gene, such as rs743572, rs1801282 and rs174547, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect skin oil control ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is

scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your skin oil control ability at the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to skin oil control ability.

Ability To Resist Rosacea

Rosacea, also known as acne rosacea, is a chronic inflammatory skin disease that occurs on the face. It often occurs in the middle of the face, the tip of the nose and the wing of the nose, and can also extend to the cheeks, jaw and forehead.

My Ability To Resist Rosacea

Strong

75.3% of We users are similar to me

Suggestions

1. Skin care recommendations

a. Pay attention to skin care: Many skin care products can irritate the symptoms of rosacea. Try not to use skin care products that contain ethanol, menthol, and witch hazel, and try to use mild skin care products.

b. Pay attention to sun protection: Use sunscreen before going out to prevent ultraviolet (UV) light. Try to use sunscreen with a higher SPF.

2. Life advice

a. Avoid predisposing factors: Common triggers for rosacea include overheating, cold wind blowing on your face, and eating spicy food. These may cause your rosacea to flare up. Different people may have different triggers. Find out what causes rosacea and avoid these triggers.

b. Maintain a happy mood: avoid excessive stress, participate in outdoor activities appropriately, and adjust to a comfortable rhythm of life.

Knowledge



Performance of rosacea

1. Facial redness: Rosacea usually causes continuous redness in the central part of the face. The small blood vessels on the nose and cheeks often swell and become visible.

- 2. Red and swollen skin: Many people with rosacea also form acne-like pustules on their faces and contain pus. There is a burning sensation on the skin.
- 3. Eye problems: About half of people with rosacea also experience dry, irritated and swollen eyelids, as well as red and swollen eyelids. In some people, the eye symptoms of rosacea appear before the skin symptoms.



Susceptible groups of rosacea

- 1. Between 30 and 50 years old.
- 2. Fair skin, usually seen in people with blond hair and blue eyes.
- 3. Someone in the family suffers from rosacea or severe acne.
- 4. Women are more likely to get sick than men. However, men usually have more severe symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS763035	intergenic	GG	strong ability to resist rosacea

Test details



Applicable situation

The ability to resist rosacea item uses one polymorphic locus between genes: rs763035.



Notification

- 1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
- 2. The test may not cover all genes or loci that affect ability to resist rosacea.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all

users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your your emotional stability at the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to ability to resist rosacea.

Ability To Resist Cellulite

Cellulite refers to the appearance of unevenness on the surface of the skin, showing a surface state similar to orange peel.

My Ability To Resist Cellulite

Weak

90.7% of We users are similar to me

Suggestions

1. Maintain a healthy weight: losing weight doesn't completely eliminate cellulite, but it does help prevent and reduce its appearance. This is because when you keep a healthy weight, the fat under the skin is less, thereby reducing the possibility of skin forming concave lines.
2. Adhere to physical exercise: exercise helps to reduce fat and promote blood circulation.
3. Adopt low-fat diet: eating a low-fat diet rich in lean protein and fresh vegetables can reduce body fat.

Knowledge

Cellulite is not a disease

Cellulite does not develop overnight, It usually develops slowly. Cellulite neither causes pain nor affects health, it's an aesthetic concern.

The developmental stages of cellulite

Cellulite goes through four stages of development:

Grade 0: No visible cellulite.

Grade 1: Smooth skin when standing, but orange-peel appearance when sitting.

Grade 2: Skin has orange-peel appearance when standing and sitting.

Grade 3: Skin has orange-peel appearance when standing with deep raised and depressed areas.



Factors affecting cellulite

1. Sex: Orange-peel tissue is more common in women than in men and women's fat is usually distributed in the common areas of thighs, buttocks and hips, the areas where adipose tissue is deposited.
2. Age: Most women develop some cellulite after puberty, and it becomes more common as the skin loses its elasticity.
3. Weight: Thin people can have cellulite, but it's more noticeable in those with more body fat.
4. Genetic factors: Genetic history plays a role in the development of cellulite.
5. Lack of exercise: An inactive lifestyle can also increase your chances of developing cellulite.
6. Diet: A high-fat diet increases the likelihood of cellulite.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS11549465	HIF1A	CC	weak ability to resist cellulite

Test details



Applicable situation

The ability to resist cellulite item uses one polymorphic locus of *HIF1A* gene: rs11549465.



Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect the ability to resist cellulite.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all

users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your ability to resist cellulite at the genetic level. Since This test does not involve consideration of other factors, it does not mean your true health status.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect the ability to resist cellulite.

Ability To Resist Varicose Veins

Varicose veins are abnormally tortuous and dilated blood vessels, which most often occur in the lower extremities. This is usually because the veins are under pressure for a long time, weakening and destroying the vascular valves and blood vessel walls.

My Ability To Resist Varicose Veins

Moderate

45.7% of We users are similar to me

Suggestions

1. Maintain a healthy weight: Being overweight puts unnecessary stress on your legs and circulatory system. If you are overweight, losing weight can reduce stress and thus reduce the risk of developing varicose veins.
2. Avoid standing and sitting for a long time: regular activities can promote blood reflux and reduce venous pressure through the function of calf muscle pump, which can prevent the formation of varicose veins.
3. Raise limbs during rest: promote blood reflux through gravity. Note that patients with varicose veins should not soak their feet in hot water for a long time, which will increase the flow of local arterial blood, aggravate the burden of venous blood stasis and reflux, aggravate the expansion of venous vessels, and worsen the condition.

Knowledge



How varicose veins are formed?

Our leg veins have one-way valves that keep blood flowing in the right direction, which called venous valves. Normally, these valves allow venous blood to flow from our legs up and into our heart even when we're standing, and if something goes wrong with these valves, it's like a leaky door that won't close. Blood flows back due to gravity and accumulates in the legs causing high venous pressure,

causing leg swelling and sometimes cramping and other symptoms. Over time, the veins in our legs become weaker, dilated and twisted by excessive pressure.

What kind of people are prone to varicose veins?

1. Genetics: Varicose veins are more genetics, if a family member has this condition, the chance that other members suffer from varicose veins will also be greatly increased.
2. Occupation related: it is particularly common in some occupations that need to stand for a long time, such as teachers, salespeople, waiters, etc.
3. Weight change related: obesity and pregnancy are risk factors that trigger varicose veins.
4. Age-related: The risk of varicose veins increases with age.

How do I simply predict varicose veins

Watch out for varicose veins when you have the following symptoms. From the appearance, in the early stage, the subcutaneous capillaries of the limbs will dilate, and over time, the subcutaneous blood vessels will dilate and twist into clusters, and there will be acid swelling and pain after standing for a long time; In the later stage, there will be limb swelling, concave edema, skin pruritus, dermatitis, pigmentation and ulceration in the boot area. In secondary superficial vein thrombosis, thrombophlebitis may occur with localized redness, swelling, heat, pain, or palpable swelling.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1801131	MTHFR	GT	moderate ability to resist varicose veins
RS1801133	MTHFR	GG	strong ability to resist varicose veins

Test details

Applicable situation

The ability to resist varicose veins item uses two polymorphic loci of *MTHFR* gene: rs1801131, rs1801133.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

2. The test may not cover all genes or loci that affect the ability to resist varicose veins.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your ability to resist varicose veins at the genetic level. Since This test does not involve consideration of other factors, it does not mean your true health status.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect the ability to resist varicose veins.

Anti-Skin Photoaging Ability

Photoaging is the damage caused by long-term exposure to sunlight, which promotes the premature appearance of aging changes in the skin.

My Anti-Skin Photoaging Ability

Moderate

40.1% of We users are similar to me

Suggestions

- Your anti-photoaging ability is **Medium**, it is recommended to pay extra attention to the sunscreen of the skin.
- Photoaging is a phenomenon of Skin aging caused by the destruction of elastin structure caused by long-term ultraviolet radiation.
- Pay attention to good skin care habits such as Sun protection and After-sun repair, which are beneficial to the skin.
- Appropriate consumption of sunscreen foods, such as Kiwi fruit, Pomegranate and other fruits rich in Vitamin C, or appropriate intake of Marine organisms foods.
- Pay attention to Good living habits such as sun protection, regular work and rest, etc, which are the key to anti-photoaging.
- Grape Seed Oil, Resveratrol, L-Glutathione, Vitamin E and other antioxidants can protect the skin from UV damage.
- Retinoids are often used to prevent photoaging and can promote the removal of melanin.
- Laser technology and other Non-drug therapy may be effective in the treatment of photoaging.
- Alpha hydroxy acids (AHAs), salicylic acid and other Drug therapy are commonly used to treat skin photoaging and abnormal melanin deposition.

* If you need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Knowledge



What is photoaging

Photoaging is the unnatural aging of the body caused by long-term exposure to sunlight. Ultraviolet (UV) radiation in sunlight has strong penetrating power and can reach the dermal layer of the skin,

leading to the formation of melanin, destroying the structure of elastin, and causing skin damage. Specific manifestations include pigmentation, rough skin, dryness, sagging, appearance of wrinkles, and premature skin aging.

Factors affecting skin photoaging

Photoaging is a slow development process that accumulates over time, and its influencing factors are bound to be extensive and complex. Irradiation doses of different light wavelengths, physiological factors such as age, skin color, diet and daily life, pathological factors, occupational and environmental factors can all affect the occurrence of skin photoaging.

1、Radiation spectrum and dose: The ultraviolet radiation in sunlight is the main spectrum that causes skin photoaging. UVC in ultraviolet radiation is blocked by the earth's atmosphere and cannot reach the earth's surface, so UVB and UVC radiation are the main participants in photoaging.

2、Physiological factors:

a. Age: Skin photoaging begins to accumulate since it receives sunlight. With the increase of age, skin structure will change, such as the integrity of the surface stratum corneum, the thickness of the epidermis, the color, and the content of light-absorbing substances in the skin. These factors all can affect the reflection, scattering, absorption and penetration of ultraviolet radiation, thus affecting the occurrence and development of photoaging.

b. Skin color: The color of the skin is determined by the melanin in the epidermis, which has a good absorption of ultraviolet radiation of various wavelengths. Therefore, the melanin in the epidermis is a natural barrier against ultraviolet radiation.

3、Pathological factors:

A variety of skin diseases and pathological states can enhance the sensitivity of the body to ultraviolet radiation, which can lead to or promote skin photoaging.

4、Occupational factors:

Different occupational workers receive different doses of sunlight, and the occurrence of photoaging is also very different.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS11547464	MC1R	GG	strong anti-skin photoaging ability
RS1805006	MC1R	CC	strong anti-skin photoaging ability
RS1805007	MC1R	CC	strong anti-skin photoaging ability

RS322458	intergenic	CT	moderate anti-skin photoaging ability
RS885479	MC1R	AG	moderate anti-skin photoaging ability

Test details

Applicable situation

The skin anti-photoaging ability item uses five polymorphic loci on and between *MC1R* gene: rs322458, rs11547464, etc.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect the skin's anti-photoaging ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your skin's anti-photoaging ability at the genetic level. Since This test does not involve consideration of other factors, it does not mean your true health status.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect the skin anti-photoaging ability.

Anti-Skin Wrinkle Ability

Wrinkles refer to small fine lines and wrinkles formed by the skin affected by the external environment to form free radicals, which destroy collagen and active substances in normal cell membrane tissue and oxidize cells.

My Anti-Skin Wrinkle Ability

Moderate

41.5% of We users are similar to me

Suggestions

- Your anti-wrinkle ability is **Medium**, and your skin is more prone to Wrinkles than that of your peers.
- Appropriate consumption of anti-wrinkle foods, such as Kiwi fruit, Pomegranate and other fruits rich in Vitamin C or moderate intake of Marine food.
- Grape Seed Oil, Resveratrol, L-Glutathione, Vitamin E and other antioxidants can protect the skin from UV damage.
- Daily use of skin care products containing peptides and other Anti-wrinkle ingredients is beneficial to fight wrinkles.
- Pay attention to good skin care habits such as Sun protection and moderate Facial cleansing and Pay attention to moisturizing to prevent wrinkles caused by the degradation of collagen induced by ultraviolet radiation.
- The use of Injection beauty anti-wrinkle needle, laser dot matrix, etc. Physical therapy may be effective in removing wrinkles.
- Good living habits such as reducing frowning, getting enough sleep, etc, are also the key to anti-wrinkle skin.

* If you need to use dietary supplements, it is recommended that you consult an authoritative health professional or clinician for help.

Knowledge

Causes of wrinkles

Collagen, elastic fiber and hyaluronic acid are the most abundant components in the skin. The former two are most closely related to skin thickness and elasticity, while the latter is closely related to skin moisture content. These components are synthesized by cells in the skin. With the aging of

individuals and the damage of the external environment, the cell vitality decreases, the synthesis of these components also decreases gradually, and the skin becomes loose and dry. Because ultraviolet rays can directly damage dermal cells and elastic fibers, causing skin photoaging, dark skin can block more ultraviolet radiation because of more melanin in the epidermal layer, so dermal cells and tissue fibers will suffer less damage, cell vitality will be better, and wrinkles will be less.

In addition, the hardest-hit areas of wrinkles are often around the eyes (crow's feet), forehead (forehead wrinkles) and mouth. Because these areas are rich in expression muscles, they constantly pull the skin on the surface every day. Over the years, elastic fibers will be damaged and wrinkles will also occur.



Treatments for wrinkles

1. Physical therapy: Dot matrix laser, electric wave skin pulling, thermage, photorejuvenation, etc., all exert acute physical damage on the dermis, so that the skin starts the repair mechanism, the cells are rejuvenated, the elastic fibers and collagen fibers can be rearranged to restore elasticity, and the local microcirculation will be improved. It has a good effect on early wrinkles.
2. Surgery treatment: At present, there is a new technology, which penetrates the absorbable thread with barbs into the skin and fascia system and fixes the upper end, so as to achieve the effect of minimally invasive improvement of the face and treatment of wrinkles. In addition, traditional surgery makes incisions in hidden parts (such as the hairline and behind the ears), lifts SMAS (superficial muscle aponeurosis system), and resects part of the skin, which has a significant effect on very obvious wrinkles and facial ptosis.



Anti-wrinkle foods for skin

1. Food rich in chondroitin: Dermis is the main factor affecting skin beauty. Dermis is composed of elastic fibers, and the main material of elastic fibers is chondroitin sulfate. Eating more foods rich in chondroitin sulfate can delay the occurrence of skin wrinkles and keep the skin elastic and delicate. Recommended food: pig bone soup, cow bone soup, chicken skin, chicken bone soup. All foods with more cartilage are the best source of cartilage.
2. Foods rich in vitamin C and E: Vitamin C and E are antioxidants, which can prevent subcutaneous fat oxidation, enhance the vitality of skin epidermis and dermal cells, and avoid premature skin aging. Pay attention to the intake of foods rich in vitamin C and E, which is also conducive to anti-wrinkle. Recommended food: vegetables and fruits.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2066853	AHR	AG	moderate anti-skin wrinkle ability

Test details

Applicable situation

The skin anti-wrinkle ability item uses one polymorphic locus on *AHR* gene: rs2066853.

Notification

1. The basis of the test is mainly from the research of the Chinese Han population, other populations are for reference only.
2. The test may not cover all genes or loci that affect the skin's anti-wrinkle ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your skin's anti-wrinkle ability at the genetic level. Since This test does not involve consideration of other factors, it does not mean your true health status.

Population samples

The basis of the test is mainly from the research of Chinese Han population.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect the skin anti-wrinkle ability.

Anti-Sagging Eyelid Ability

The skin of the eyelid loses its elasticity, the fat of the orbital septum droops, the wrinkles appear in the outer canthus, and the eyelids are sagging and drooping, which are more common in middle-aged and elderly people.

My Anti-Sagging Eyelid Ability

Weak

35.2% of We users are similar to me

Suggestions

1. Skin care advice

Choosing the right eye cream: Anti-age eye cream can promote the increase of collagen protein and can re-support the skin around the eyes, which may have a certain effect on improving sagging.

2. Life advice

Daily eye protection: People who often face the computer screen should be careful not to use your eyes for a long time, and do not rub your eyes severely. When you put on makeup, be gentle and don't pull the eye skin with much strength. Avoid working with your eyes under long-term sunlight.

Knowledge



Causes of sagging eyelid

1. Genetic factors: hereditary or congenital causes of sagging eyelid.
2. Causes of edema: 20-35 years old, sagging eyelid due to edema.
3. Causes of aging: The sagging eyelid that gradually appears with age after 40 years old are mainly due to skin aging. The degree and speed of aging are related to heredity.



Manifestations of sagging eyelid

1. Mild sagging eyelid: The original double eyelid is narrowed, or the shape of the double eyelid is not satisfactory, the eyelid curve is not smooth, the outer corner of the eye is slightly drooping,

the eyelashes are slightly covered by loose skin, eyelashes of people with single eyelids are covered partly.

2. Moderate and severe sagging of upper eyelid: Double eyelid is significantly narrowed or becomes epicanthic fold or multiple folds of the eyelid, increased skin wrinkles, accompanied by sunken upper eyelid, obvious sagging and drooping of the outer corner of the eye, deepening of the tail wrinkles, eyelashes may be partially or completely covered by sagging skin, eye morphology may be significantly changed, triangular eyes, sagging skin may cover pupils and vision.

Correction of sagging eyelid

1. Simple double eyelid removal: After lifting the upper eyelid skin and deep myofascial membrane, the fine lines of the eye will be stretched and disappear. After the excess skin is removed, the phenomenon of sagging and drooping of the skin and covering the pupils is improved, and the charm of the eyes is revealed.
2. Eyelid margin incision and double eyelid surgery: It is mainly aimed at elderly beauty seekers who do not want to change too obviously. Generally, the double eyelid effect is not required. They just hope to improve the current situation of blepharoptosis through surgery and make life and travel more comfortable.
3. Eyebrow lifting or eyebrow cutting operation: The operation adopts local infiltration anesthesia. The doctor selects the incision in the middle of the eyebrow, the upper edge of the eyebrow and the lower edge of the eyebrow according to the distance between eyebrows and eyes of the beauty seeker. Cut the skin, subcutaneous and orbital orbicularis oculi muscles according to the design line, and cut off the unhealthy eyebrows and loose skin.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS11876749	DLGAP1	CT	moderate ability to resist varicose veins
RS11979919	intergenic	CC	weak anti-sagging eyelid ability

Test details

Applicable situation

The anti-sagging eyelid ability item uses 2 polymorphic loci on and between *DLGAP1* gene: rs11876749, rs11979919.

Notification

1. The basis of the test is mainly from the research of Caucasian, Chinese Han population, other populations are for reference only.
2. The test may not cover all genes or loci that affect anti-sagging eyelid ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your anti-sagging eyelid ability at the genetic level. Since This test does not involve consideration of other factors, it does not mean your true health status.

Population samples

The basis of the test is mainly from the research of Caucasian, Chinese Han population.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect anti-sagging eyelid ability.

Collagen Retention Ability

Collagen is a very important protein in the human body, mainly found in connective tissues. It keeps the skin elastic, and the aging or loss of collagen can easily cause the skin to age.

My Collagen Retention Ability

Moderate

66.5% of We users are similar to me

Suggestions

- Your Collagen retention ability is **medium**, so the skin is more prone to wrinkles and sagging than people of the same age.
- Vitamin C is the main raw material for collagen production, so eating with vitamin C has a better effect on maintaining collagen in the skin.
- Appropriate supplementation of Coenzyme Q10 is beneficial to slow down skin aging through its role in repairing damaged collagen.
- Pay attention to good skin care habits such as Sun protection and After-sun repair to prevent the degradation of collagen induced by ultraviolet radiation.
- Medical beauty techniques such as Subcutaneous injection of collagen may be effective in supplementing collagen.
- Good living habits such as sun protection and regular work and rest are also the key to maintaining collagen in the skin.

* If you need to use dietary supplements, it is recommended to consult an authoritative health professional or clinician for help.

Knowledge

The effect of collagen on the skin

Collagen and elastic fibers work together to form a mesh-like support, just like the steel structure supporting skin tissue. Sufficient collagen protein can make skin cells plump, make skin moist, delicate and smooth, and can stretch fine lines and wrinkles, which can effectively prevent skin aging.

Causes of collagen loss

There are many reasons for the loss of collagen, such as the increase of age, the damage of ultraviolet, the deterioration of the environment, irregular work and rest, and so on. In addition to the irresistible factor of aging, the most important reason for the loss of collagen is UV damage.

The effect of collagen loss

The loss of collagen will first cause the skin to lose moisture, and wrinkles will appear when the skin is dry. The skin will also lose its elasticity, become sagging, and increase skin pores. In addition, after collagen is swallowed by melanin, if the activated melanin cannot be metabolized in time, it will also bring problems such as spots, uneven skin color or darkness.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1800795	intergenic	GG	strong collagen retention ability
RS3918242	intergenic	CC	weak collagen retention ability

Test details

Applicable situation

The collagen retention ability item uses 2 polymorphic loci between genes: rs1800795, rs3918242.

Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test may not cover all genes or loci that affect collagen retention ability.

Scoring model

According to the frequency of the reference sample and the control sample carrying the corresponding genotype in the literature, the weight of each locus is divided. Finally, the item is scored according to the detected genotype and the weight of the locus itself. The average score of all users and the distribution of scores are obtained through the algorithm, and the user is divided into project results accordingly.

How to use the test results

Notification

An individual's skin characteristics are determined by multiple factors such as genes and environment. This test only evaluates your collagen retention ability at the genetic level. Since This test does not involve consideration of other factors, it does not mean your true health status.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect collagen retention ability.



Genetic Characteristics

15 Reports

Item	Genetic risk
Alcohol Flushing	Less likely to have alcohol flushing
Bitter Sensitivity	May be sensitive to bitterness
Ear Wax	Dry earwax
Smoking Behaviors	Smoking is not very addictive
Fasting Plasma Glucose Levels	May have higher fasting blood glucose levels than average
Menopause Age	Normal menopause time
Autosomal Dominant Compelling Helio-Ophthalmic Outburst (ACHOO) Syndrome	High rate of sneezing when looking at light
Eating Behavior	Tend to have a normal diet
Tendency to Avoid Mistakes	May be good at avoiding mistakes
Absolute Pitch	Less likely to have the ability of absolute pitch (applies to European, Jewish, and Indian people)
ABO Blood Group	Type B
APOE Genotype	The genotype of APOE is $\epsilon 3/\epsilon 3$
Deep Sleep	Duration of deep sleep is relatively short
Genetic Height	161.7 cm
Human Milk Oligosaccharides (HMO) Secretor Status	secretor

Alcohol Flushing

Flushing of the face after alcohol consumption.

My Alcohol Flushing

Less likely to have alcohol flushing

Suggestions

People who do not have alcohol flushing are likely to drink too much in high excitement and even develop the bad habit of drinking, if not to be controlled. According to "Dietary Guidelines for Chinese Residents (2016)", the amount of alcohol consumed by an adult man per day should not exceed 25g, and that of an adult woman per day should not exceed 15g.

Knowledge

Why do you flush while drinking

Many people think that the flushing reaction is an allergy to alcohol, but in fact it is caused by the insufficient function of certain enzymes in the body. Let's first understand the metabolism of alcohol. The main component of alcohol is ethanol. When it is metabolized in the body, there are two main transporters: alcohol dehydrogenase and acetaldehyde dehydrogenase. The former breaks down alcohol into acetaldehyde, and then another enzyme converts acetaldehyde into acetic acid, which is finally metabolized into carbon dioxide and water. When acetaldehyde dehydrogenase does not work, the aldehyde will not be metabolized for a long time, and the aldehyde has the function of dilating capillaries, resulting in a common flush after drinking.

Asians are more susceptible to alcohol flushing

Also known as Asian flushing syndrome. This is because alcohol flushing is very common in East Asia. About 40%-50% of Asians have very low acetaldehyde dehydrogenase activity. As for why Europeans and Americans do not change their face when drinking, it is because their acetaldehyde dehydrogenase is almost normal.

Alcohol flushing ≠ Can drink a lot

People who flush easily after drinking are defective in metabolizing acetaldehyde. In fact, they are more likely to be harmed by aldehydes, such as dizziness, headache, panic, nausea, vomiting and other discomforts. People who really drink a lot of alcohol should have more alcohol dehydrogenase and aldehyde dehydrogenase in their bodies or with higher activity, so that they can better metabolize alcohol and avoid being drunk by a thousand cups.

Increased susceptibility to esophageal cancer in alcohol flushers

Studies have shown that people with alcohol flushing reactions drink two bottles of beer a day, and the incidence of esophageal cancer is 6-10 times that of people who do not flush.

Flush first if you want to quit drinking

Although the accumulation of acetaldehyde can make people feel uncomfortable, it is not good-for-nothing. Smart medical scientists also use this point to help people stop drinking. There is a clinical drug called disulfiram, also known as disulfiram, which can inhibit the activity of aldehyde dehydrogenase and cause aldehyde to accumulate in the body. Alcoholics who drink alcohol while taking this drug for abstinence may cause discomfort such as flushing, nausea, headache, chest tightness and elevated heart rate caused by acetaldehyde, which promotes the establishment of aversion to drinking, and gradually stops drinking to achieve the goal of abstinence.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS671	ALDH2	GG	less likely to have alcohol flushing

Test details

Applicable situation

The alcohol flushing genetic test result uses the polymorphic locus of *ALDH2* gene: rs671.

Notification

1. The basis of the test is mainly from the research of Asian, other populations are for reference only.
2. The test may not cover all genes or loci that affect the genetic features of alcohol flushing.

How to use the test results

Notification

1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
2. The test result only predicts your alcohol flushing features from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

Population samples

The basis of the test is mainly from the research of Asian, and other populations are for reference only.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the genetic features of alcohol flushing.

Bitter Sensitivity

Bitter sensitivity refers to the ability of a person to taste bitterness.

My Bitter Sensitivity

May be sensitive to bitterness

Knowledge

Can you tolerate more bitter taste

The level of bitter sensitivity affects whether we can taste the bitterness in most foods. Some people are more sensitive to bitterness and can taste the bitterness of foods such as born cabbage, kale, broccoli, and beverages such as coffee and dark beer. In addition, according to statistics statistical data, In addition, according to statistics, Asians are more sensitive to bitterness than European and African populations, and about 90% of them are more sensitive to bitterness.

'Taste Map' does not exist

Many people believe that there are specific areas on the tongue that are responsible for different taste sensations. For example, the tip of the tongue is sensitive to sweetness, and the base of the tongue is sensitive to bitterness. In fact, this kind of 「taste map」 does not exist, because all taste buds can recognize the five basic tastes of sweet, bitter, salty, sour, and fresh. Therefore, as long as the tongue area with distributed taste buds can feel different tastes.

Taste buds will also change

There are about 10,000 taste buds on the human tongue. As people age, taste buds will gradually shrink and decrease, leading to a decline in taste function. If you want to maintain the sensitivity of taste buds, you should pay attention to prevent oral diseases, quit smoking and limit alcohol, eat more vitamin-rich foods, which will help delay the aging of taste buds.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS713598	TAS2R38	CG	may have a normal bitter taste

Test details



Applicable situation

The bitter sensitivity genetic test result uses the polymorphic locus of *TAS2R38* gene: rs713598.



Notification

1. The test may not cover all genes or loci that affect the genetic features of bitter sensitivity.
-

How to use the test results



Notification

1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
2. The test result only predicts your bitter sensitivity features from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the genetic features of bitter sensitivity.

Ear Wax

Also known as cerumen, is a pale yellow viscous liquid secreted by the ceruminous glands in the external auditory canal that dries when it makes contact with air, sometimes forming a flaky wax coating. There are two primary types of earwax, wet and dry.

My Ear Wax

Dry earwax

Suggestions

1. Dry earwax is generally massive. It is not recommended to take it out forcibly, and avoid using sharp objects such as ear scoop, so as to prevent damage to the ear canal and tympanic membrane.
2. The ear canal has self-cleaning function, and most people's earwax can be discharged by themselves. If you repeatedly pick your ears, it will destroy the natural barrier of ear canal and even cause ear canal eczema. Therefore, if there is no ear discomfort, try not to pick your ears.
3. When too much earwax leads to clogging, such as ear tightness, ear pain, hearing loss and other symptoms, it is recommended to ask the otolaryngologist for help and remove the earwax with professional tools.

Knowledge

Geographical distribution of earwax types

In the beginning, human ancestors were actually wet earwax. Dramatically, before the 2000s, after our ancestors went out of Africa and entered Asia, the ABCC11 gene on chromosome 16 was mutated, causing the earwax to dry out and no longer sticky. At the same time, dry earwax is relatively adaptable, so it is retained. Therefore, as descendants of East Asia, we also enjoy these benefits. As a whole, 93%-96% of Chinese population have dry earwax, of which 99% in North China. The North Korean population is also dominated by dry earwax (99%), while the Japanese population is relatively small, with a ratio of about 84%. However, the partners in other regions are not so lucky.

Basically, they still retain the genes of their ancestors and mainly use wet earwax. The aborigines of Oceania have 60%-70% of wet earwax, about 90% of Caucasians, and 99.5% of Africans.

Types of earwax and body odor

The genes that control the type of earwax are not only mutated, it also affects other aspects. It not only determines your earwax type, but also affects your perspiration and body odor. For people with wet earwax, their sweat glands secrete a relatively large amount of fat, which is more likely to cause body odor due to the multiplication of bacteria.

Types of earwax and lactation ability

The genes that control the type of earwax are also related to the mammary glands and affect the lactation ability of the mother. According to research, the lactation ability of women with dry earwax is relatively weak, and the cases of milk secretion or inadequate secretion during lactation are significantly higher than those of women with wet earwax.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS17822931	ABCC11	TT	dry earwax

Test details

Applicable situation

The ear wax genetic test result uses the polymorphic locus of *ABCC11* gene: rs17822931.

Notification

1. The basis of the test is mainly from the research of Caucasian, Asian and African, other populations are for reference only.
2. The test may not cover all genes or loci that affect the type of ear wax.

How to use the test results

Notification

-
1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
 2. The test result only predicts your ear wax from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

Population samples

The basis of the test is mainly from the research of Caucasian, Asian and African, other populations are for reference only.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to the types of earwax.

Smoking Behaviors

Long-term smoking and addiction to nicotine contained in the smoke leads to long-term tobacco dependence.

My Smoking Behaviors

Smoking is not very addictive

Suggestions

- If you don't smoke:
 1. Do not take the initiative to smoke;
 2. Avoid exposure to tobacco environment (second-hand smoke environment, tobacco residue environment, etc.).
- If you have a smoking habit but are not addicted:
 1. Please quit smoking as soon as possible;
 2. Avoid exposure to tobacco environment (second-hand smoke environment, tobacco residue environment, etc.).
- If you have a smoking habit and have developed addictive symptoms:
 1. Please quit smoking as soon as possible and take intervention measures through medical institutions, psychological institutions and social institutions if necessary;
 2. Avoid exposure to tobacco environment (second-hand smoke environment, tobacco residue environment, etc.).

Influencing factors

Controllable factors

Tobacco smoking: It is divided into active smoking and passive smoking. After smoking, the nicotine component will cause smoking addiction.

Knowledge



Nicotine action mechanism

The mechanism of smoking addiction is nicotine addiction. Nicotine is a neurotoxin. When it enters the body, it will cause excitement in the nervous system, allowing smokers to subjectively feel that smoking can relieve fatigue and refresh their spirits. But this sense of excitement is temporary, and the nervous system is immediately suppressed after excitement. Therefore, the sensitivity and accuracy of neuromuscular response after smoking are reduced.



Quit smoking response

Sudden cessation of smoking or reduction of smoking will cause at least the following uncomfortable symptoms within 24 hours, such as: craving for smoking, irritability, depression, difficulty concentrating, restlessness, headache, drowsiness, gastrointestinal dysfunction. This is the symptom of nicotine dependence.



Harm of smoking

Smoking can cause multiple malignant tumors and other chronic diseases, leading to abnormal reproduction and development. It is also closely related to the occurrence of other diseases and health problems. Tobacco smoke contains hundreds of harmful substances, some of which damage the human body by themselves, while others chemically react with other substances inside and outside the body to derive new harmful substances to damage the human body.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1051730	CHRNA3	GG	smoking is not very addictive

Test details



Applicable situation

The smoking behaviors genetic test result uses the polymorphic locus of *CHRNA3* gene: rs1051730.



Notification

1. The basis of the test is mainly from the research of Caucasian and Asian, other populations are for reference only.
2. The test may not cover all genes or loci that affect smoking behaviors.

How to use the test results

Notification

1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
2. The test result only predicts your smoking behaviors from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian, other populations are for reference only.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all or loci that related to smoking behaviors.

Fasting Plasma Glucose Levels

Blood glucose level refers to the level of glucose in the blood. Most of the energy required for cell activity in the body comes from glucose, so blood glucose must be kept at a certain level in order to maintain the needs of various organs and tissues in the body. It is usually represented by fasting blood glucose level, that is, the blood glucose level after fasting for 8 hours.

My Fasting Plasma Glucose Levels

May have higher fasting blood glucose levels than average

Suggestions

- Your fasting blood glucose level is **high**, please be alert to Hyperglycemia and pay attention to Fasting blood glucose test results.
- If you have a family history of diabetes, obesity or habitual eating, please keep Healthy diet and exercise.
- Protective food:
 - Low glycemic index (GI) foods, such as Coarse grains, beans, milk and potatoes.
 - In the process of blood glucose control (such as weight loss), please be particularly alert to Hypoglycemia, and adjust your diet if necessary.

* If you have a disease related to abnormal blood glucose or need to use dietary supplements to control blood glucose, it is recommended that you consult an authoritative health expert or clinician for help.

Knowledge



Daily fluctuation of blood glucose levels

The fasting blood glucose of normal people is generally controlled at 2.8 ~ 6.1mmol/L to maintain the energy supply of body cells. When eating, activity or emotional tension, blood glucose will soar rapidly. At the same time, insulin (which can reduce blood glucose concentration) will also be secreted in large quantities, so as to maintain the blood glucose balance in the body. Therefore, our daily blood glucose level has inadvertently experienced peaks and valleys.



Blood glucose measurement is also exquisite

The time of blood glucose measurement is knowledgeable. It is generally divided into fasting blood glucose (fasting for 8 hours, no breakfast and exercise after getting up in the morning, measured before 8 in the morning), pre meal blood glucose (measured before eating) and 2-hour postprandial blood glucose (measured 2 hours after eating the first meal).



How to correctly measure blood glucose level

According to the guideline for prevention and treatment of type 2 diabetes in China (2013 Edition):

- Normal blood glucose level: fasting blood glucose < 6.1mmol/l and 2-hour postprandial blood glucose < 7.8mmol/l.
- High blood glucose level: fasting blood glucose is 6.1 ~ 7.0mmol/l or 2-hour postprandial blood glucose is 7.8 ~ 11.1mmol/l.
- Diabetes: fasting blood glucose is greater than 7.0mmol/L or 2 hours after meal, blood glucose is more than 11.1mmol/L.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS560887	G6PC2	CT	may have higher fasting blood glucose levels than average

Test details



Applicable situation

The fasting plasma glucose level item uses a polymorphic locus of the *G6PC2* gene: rs560887.



Notification

1. The basis of the test is mainly from the research of Caucasian and East Asian, and other populations are for reference only.
2. The genetic report may not cover all genes or loci associated with the fasting plasma glucose level.

How to use the test results



Notification

-
1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
 2. The test result only predicts your blood glucose level characteristics from the genetic level. Because it does not involve consideration of other factors, it does not represent your true situation.

Population samples

The basis of the test is mainly from the research of Caucasian, and other populations are for reference only.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to fasting plasma glucose levels.

Menopause Age

Menopause refers to the transitional period from the onset of gonadal failure to the complete loss of menstruation; menopause does not only refer to the end of menstruation. The stage of ovarian recession exists before the onset of menopause. According to a survey, this lasts from 2-4 years and is known as premenopause. After menopause, ovarian function is decreased, but does not necessarily disappear completely and will generally continue for 2-3 years, or up to 6-8 years or even longer. Therefore, menopause is a combination of premenopause, menopause, and postmenopause, and some scholars refer to it as "perimenopause".

My Menopause Age

Normal menopause time

Knowledge

What is menstruation

Menstruation is one of the signs of maturity. Every month, the body prepares for pregnancy. The lining (endometrium) of the uterus prepares each month for the implantation of an embryo. This preparation occurs under the influence of estrogen and progesterone from the ovary. If no pregnancy occurs, the uterus, or womb, sheds its lining. The menstrual blood is partly blood and partly tissue from inside the uterus. It passes out of the body through the vagina.

What is menopause

The functional activity of ovary starts from puberty and is manifested by menstruation. The menopause refers to that time in every woman's life when her periods stop and her ovaries lose their reproductive function. Usually, this occurs between the ages of 45 and 55.

What is menopausal syndrome

Menopause is a stage in life when a woman can no longer become pregnant. The average woman goes through natural menopause in her early 45-55. During this period, due to the decline of ovarian function and the fluctuation or reduction of sex hormone level, there will be a series of symptoms, such as menstrual disorder, heat and sweat, chest tightness and irritability, dizziness and panic,

insomnia and dreaminess, mental depression, namely menopausal syndrome, also known as menopausal syndrome.

What factors will lead to early menopause

1. Menarche age: menarche age is early, and menopause age may be late.
2. Menstruation, pregnancy and childbirth: the menopause age is later for those who have had more or more pregnancies in the past.
3. Living conditions: women with superior life and good maintenance have a later menopause age.
4. Bad lifestyle: smoking and drinking will lead to early menopause.
5. Race: the menopause age of whites is later than that of people of color.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS16991615	MCM8	GG	normal menopause time

Test details

Applicable situation

The menopause age item provided uses a polymorphic locus of the *MCM8* gene: rs16991615. The basis of the test is mainly from the research of Caucasian and East Asian, and other populations are for reference only.

Notification

The test may not cover all genes or loci that are related to menopause.

How to use the test results

Notification

1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
2. An individual's menopause is the common result of the interaction of genes, environment, and living habits. Since the test does not involve consideration of other factors, it does not mean your

true health status, nor does it involve assessments on your other health conditions, living habits, etc.

 Population samples

The basis of the test is mainly from the research of Caucasian, and other populations are for reference only.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to menopause age.

Autosomal Dominant Compelling Helio-Ophthalmic Outburst (ACHOO) Syndrome

Roughly 10%-35% of the population experiences nasal itching when looking at the sun or bright light, which makes it easier to sneeze.

My Autosomal Dominant Compelling Helio-Ophthalmic Outburst (ACHOO) Syndrome

High rate of sneezing when
looking at light

Suggestions

During driving, especially when returning to the light after just passing the dark tunnel, it is easy to induce light-sneezing reflex. It is recommended to slow down the vehicle and wear sunglasses before leaving the tunnel.

Knowledge

Why does bright light make some people sneeze

When we are exposed to bright light, the optic nerve will send a distress signal to the brain, hoping that the brain will order the pupils to shrink to prevent the strong light from harming the eyes. It just so happens that the optic nerve and the trigeminal nerve are a pair of close neighbors. This part of the signal is easily received by the trigeminal nerve, so the brain mistakenly believes that the nose is stimulated by the outside, and sends out the command to sneeze.

Beware of photosneezing while driving

The sneeze reflex is generally only very annoying, but it has no obvious effect on health. But during driving, especially when returning to the light just after passing a dark tunnel, it is easy to induce a light-sneezing reflex. Sneezing can cause the driver to lose control of the vehicle, cause personal injury, and damage the vehicle and the surrounding environment.



Misfortune brought by a sneeze

Fighter pilots fly at high altitudes and are susceptible to direct sunlight. If an uncontrollable sneeze occurs during combat, it can pose a considerable risk to the pilot's combat and life. In addition, for aircraft carriers or aircraft landing on coastlines, when sunlight reflects from the surrounding water, it may cause some pilots to sneeze, causing immeasurable harm. Fortunately, by adding a pair of sunglasses to the pilot or driver, similar accidents can be greatly avoided.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS10427255	intergenic	CC	high rate of sneezing when looking at light

Test details



Applicable situation

The ACHOO syndrome item uses a polymorphic locus of between the *ZEB2* and *PABPCP2* gene: rs10427255.



Notification

1. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.
2. The test report may not cover all genes or loci that affect genetic characteristics of ACHOO Syndrome.

How to use the test results



Notification

1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
2. An individual's ACHOO Syndrome is the common result of the interaction of genes, environment, and living habits. Since the test does not involve consideration of other factors, it does not mean your true health status, nor does it involve assessments on your other health conditions, living habits, etc.

Population samples

The basis of the test is mainly from the research of Caucasian, and other populations are for reference only.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to ACHOO syndrome.

Eating Behavior

A person's eating behaviour is potentially affected by his/her bitterness taste receptor genes.

My Eating Behavior

Tend to have a normal diet

Knowledge



Evaluating the basis for the potential of "foodie"

The foodie evaluation project uses the *TAS2R38* gene as the evaluation standard. The protein encoded by this gene is the bitter taste receptor and is related to the ability to taste the bitter taste of glucosinolates. Studies have found that damage to bitter receptors leads to insensitivity to the bitter taste of glucosinolates, which is significantly related to uninhibited eating behavior. Therefore, those suffering from bitter taste receptors are more likely to become "foodies".



Function of Bitter Taste Receptor

In addition to conveying taste, bitter taste receptors also have other functions:

1. **Regulating metabolic function:** Bitter taste receptors are also expressed in cell subgroups in the gastrointestinal tract of mammals, and mediate nutrient absorption and endocrine response. It causes the increase of intracellular calcium ions and the secretion of the insulin hormone glucagon-like peptide-1 (GLP-1), which is an important regulator of insulin synthesis and secretion. Through further research, it is found that the functionally impaired TAS2R bitter receptor has a negative effect on glucose homeostasis, showing an important link between food chemosensory and digestive tract diseases.
2. **Neuromodulation:** Studies have also found that two taste receptor genes *TAS2R16* and *TAS2R38* also have a significant interaction in affecting nicotine dependence.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS1726866

TAS2R38

AG

tend to have a normal diet

Test details

Applicable situation

The genetic characteristics of foodie evaluation item uses the 1 polymorphic locus rs1726866 of *TAS2R38* gene.

Notification

The test may not cover all genes or loci that can lead to the risk of genetic characteristics of foodie evaluation.

How to use the test results

Notification

1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
2. This study only predicts your foodie evaluation characteristics from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to genetic characteristics of foodie evaluation.

Tendency to Avoid Mistakes

The tendency to avoid mistakes refers to making choices that avoid negative outcomes.

My Tendency to Avoid Mistakes

May be good at avoiding mistakes

Knowledge

Mechanism

The tendency to avoid mistakes is to avoid making choices that have led to negative results again. Some past studies have shown that the neurotransmitter dopamine is related to the tendency to avoid mistakes, which means that gene changes related to dopamine signal transduction may also affect a person's learning ability. The study found that the mutant located at the rs1800497 sites of the ANKK1 gene is closely related to the decrease in the concentration of dopamine D2 receptors. Decreased dopamine D2 receptors will reduce the sensitivity of the brain to negative results, which may explain why mutation gene carriers are less good at avoiding mistakes and are more prone to addictive behaviors.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1800497	ANKK1	GG	may be good at avoiding mistakes

Test details

Applicable situation

The tendency to avoid mistakes genetic test result uses a polymorphic locus rs1800497 in the *ANKK1* gene.

 Notification

1. This test may not cover all genes or sites that affect the genetic features of the tendency to avoid mistakes.

How to use the test results

 Notification

1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
2. The test result only predicts your tendency to avoid mistakes characteristics at the genetic level, and does not represent your true situation because it does not involve consideration of other factors.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to the tendency to avoid mistakes.

Absolute Pitch

Absolute pitch, also known as perfect pitch, refers to people with the ability to sense the actual pitch of a sound.

My Absolute Pitch

Less likely to have the ability of absolute pitch (applies to European, Jewish, and Indian people)

Influencing factors



Controllable factors

1. Past music experience: past music experience has a significant impact on the establishment of absolute pitch. For example, people who emphasize the C tonic in their language and past music experience tend to show that they have an absolute pitch to the C major scale of the equal temperament.
2. Native species: the probability of absolute pitch is greater for people with more tonal variations in their native languages, such as Cantonese, Hokkien or Vietnamese, whose native speakers are more likely to have absolute pitch.



Uncontrollable factors

1. Age: the cultivation and investigation of international children's musical ability show that 3-9 years old is the sensitive period of children's absolute pitch training. During this period, a certain amount of scientific and systematic training can make ordinary children completely establish the sense of absolute pitch. But once children are beyond the age of 12, it is very difficult to develop absolute pitch.
2. Special population: studies have shown that people who are born blind due to optic nerve hypoplasia are more likely to have absolute pitch.

Knowledge



Pitch ability rating

Pitch ability can be divided into the following levels from low to high: ①He can distinguish between two sounds of different pitches; ②He can imitate exactly what he hears; ③To know the actual pitch of the sound heard and say the name of the sound; ④He can sing the actual pitch of the music directly.



Absolute pitch mechanism

There is no difference in physical function between absolute pitch owners and non-absolute pitch owners. Absolute pitch owners do not need to have sharper hearing and more precise perception, but through different brain mechanisms to achieve absolute pitch. Absolute pitch is the ability to psychologically categorize sounds according to the level of pitch received by the brain, reflecting the brain's special ability to analyze frequency information about sounds, possibly involving high levels of cortical processing. Therefore, absolute pitch is a cognitive behavior, not the primary sensation.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS3057	ASAP1	TT	less likely to have the ability of absolute pitch

Test details



Applicable situation

The absolute pitch genetic characterization item uses rs3057, a polymorphic locus on the *ASAP1* gene.



Notification

1. The basis of the test is mainly from the research of East Asian, Ashkenazi Jew, Indian, European. Other populations are for reference only.
2. The test may not cover all pathogenic genes or loci of affecting absolute pitch.

How to use the test results

Notification

1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
2. The test result only predicts your absolute pitch at the genetic level. Since the test does not involve consideration of other factors, it does not mean your true status.

Population samples

The basis of the test is mainly from the research of East Asian, Ashkenazi Jew, Indian, European. Other populations are for reference only.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all or loci that related to absolute pitch.

ABO Blood Group

ABO Blood Group is the most commonly used blood group system classifying the different types of red blood cells. Different ABO blood types refer to different types of antigens on one's red blood cells which is inheritable.

My ABO Blood Group

Type B

Knowledge

Blood group system

There are some heritable antigenic substances on the surface of red blood cells. These antigenic substances can be proteins, sugars, glycoproteins or glycolipids. Usually, these certain antigens from alleles of the same gene or coding products of several closely linked genes constitute a blood group system. At present, 30 blood type systems have been found in humans, of which the most important are the "ABO blood type system" and "Rh blood type system"- the only two indexes that are usually tested in hospitals.

ABO blood group system

In general, inheritance of the ABO blood group system is determined by a single gene, with three main alleles IA(A), IB(B) and IO(O). Alleles control the presence of the following types of antigens and antibodies in human blood that make up four different blood types: A, B, AB and O.

The distribution of B allele is mostly in central Asia, followed by Africa. While in America and Australia, there are relatively fewer B alleles. In the worldwide, the number of B allele is the lowest in the ABO group alleles, accounting for only 16% of the world population. The A allele is more abundant than the B allele, accounting for about 21% of the world population, and they are more widely distributed. Blood type O is the most common blood type in the world, accounting for 63% of the world population. They are concentrated in Central and South America, where nearly 100% of the population is blood type O, followed by Aboriginal Australia and Western Europe (especially the Celts), and in Eastern Europe and central Asia, population of blood type O are fewer.

1. People with type A blood have type A antigens on the surface of red blood cells; Their serum produces antibodies against type B antigens.

- 2. People with type B blood have type B antigens on their red blood cells; Their serum produces antibodies against type A antigens.
- 3. People with type AB blood have both type A and type B antigens on their red blood cells. Their serum does not produce antibodies against type A or B antigens.
- 4. People with type O blood have neither type A nor type B antigens on their red blood cells, and their serum produces antibodies to both antigens

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS8176719	ABO	II	may be A, B or AB blood type
RS8176746	ABO	TT	may be B blood type
RS8176747	ABO	GG	may be B blood type

Test details

 Applicable situation

The blood group genetic characteristics item uses three polymorphic loci on *ABO* gene, including rs8176719, rs8176746, and rs8176747.

 Notification

The test does not cover all genes or loci that influence the genetic characteristics of blood types.

How to use the test results

 Notification

- 1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
- 2. This test result can only predict your blood type characteristics from the genetic level. Since the test does not involve consideration of other factors, it does not mean your ture condition.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci related to genetic characteristics of blood type.

APOE Genotype

Apolipoprotein E (APOE) is involved in the conversion and metabolism of lipoprotein. *APOE* can be classified into different genotypes by specific SNP of its coding DNA. The most common *APOE* genotypes are $\epsilon 2$, $\epsilon 3$ and $\epsilon 4$, among which people carrying $\epsilon 4$ genotype tend to have higher risks of having Alzheimer's disease.

My APOE Genotype

The genotype of APOE is $\epsilon 3/\epsilon 3$

Knowledge

What is APOE genotype?

APOE gene encodes apolipoprotein E, which is a member of apolipoprotein family and plays an important role in lipid transport and lipoprotein metabolism. Single nucleotide polymorphisms (SNPs) are seen in codons 112 (rs429358) and 158 (rs7412) of *APOE* gene, which give rise to 3 common alleles in the population, showing co-dominance:

- $\epsilon 2$
- $\epsilon 3$
- $\epsilon 4$

Based on these common alleles, the population can be divided into 6 genotypes:

- Homozygotes: $\epsilon 2/\epsilon 2$, $\epsilon 3/\epsilon 3$, $\epsilon 4/\epsilon 4$
- Heterozygotes: $\epsilon 2/\epsilon 3$, $\epsilon 2/\epsilon 4$, $\epsilon 3/\epsilon 4$

Genotype	Ratio
$\epsilon 2/\epsilon 2$	0.4%
$\epsilon 2/\epsilon 3$	6.5%
$\epsilon 3/\epsilon 3$	75.9%
$\epsilon 2/\epsilon 4$	0.9%

$\epsilon 3/\epsilon 4$	14.3%
$\epsilon 4/\epsilon 4$	2.0%

Note: $\epsilon 1$ allele is rarely seen in the population.

APOE genotype and health

Blood lipid levels:

Genotype	Low-density lipoprotein (LDL)	Total cholesterol (TC)
$\epsilon 2/\epsilon 2$	low	low
$\epsilon 2/\epsilon 3$	slightly low	slightly low
$\epsilon 3/\epsilon 3$	moderate	moderate
$\epsilon 2/\epsilon 4$	slightly high	slightly high
$\epsilon 3/\epsilon 4$	high	high
$\epsilon 4/\epsilon 4$	high	high

Health risks:

Genotype	Late-onset Alzheimer's disease	Age-related macular degeneration	Cerebral infarction	Coronary heart disease
$\epsilon 2/\epsilon 2$	low	high	low	low
$\epsilon 2/\epsilon 3$	slightly low	high	low	low
$\epsilon 3/\epsilon 3$	moderate	moderate	moderate	moderate
$\epsilon 2/\epsilon 4$	slightly high	moderate	moderate	moderate
$\epsilon 3/\epsilon 4$	high	low	high	high
$\epsilon 4/\epsilon 4$	high	low	high	high

APOE and blood lipid levels

Apolipoprotein E can combine with lipids in the blood to form lipoprotein, which is responsible for transporting cholesterol and other fats in the blood and lymph. *APOE* genotype will affect the level of lipid substances and lipoprotein. Compared to $\epsilon 3$ allele carriers, the level of low density lipoprotein (LDL) in individuals with $\epsilon 4$ is more likely to increase, and the increased LDL level is closely related to cardiovascular disease. Conversely, $\epsilon 2$ allele carriers may have decreased LDL level. In addition, individuals with genotype $\epsilon 2/\epsilon 2$ have a higher risk of hyperlipoproteinemia type III, which is a rare

disease with abnormal lipid metabolism, manifested by significantly elevated triglyceride and cholesterol levels, palmar xanthoma, and early cardiovascular disease.

APOE and late-onset Alzheimer's disease

Late-onset Alzheimer's disease is a neurodegenerative disease. About 95% of Alzheimer's disease is late onset, usually after the age of 65. APOE is a component of lipoprotein granules in peripheral plasma, cerebrospinal fluid, central nervous system and brain interstitial fluid. It plays a key role in the development, maintenance and repair of the central nervous system and regulates many important signal pathways. *APOE* $\epsilon 4$ allele carriers have a higher risk of late-onset Alzheimer's disease than those with other alleles.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS429358	APOE	TT	the genotype might be $\epsilon 2/\epsilon 2$, $\epsilon 2/\epsilon 3$, or $\epsilon 3/\epsilon 3$
RS7412	APOE	CC	the genotype might be $\epsilon 3/\epsilon 3$, $\epsilon 3/\epsilon 4$, or $\epsilon 4/\epsilon 4$

Test details

Applicable situation

The APOE genotype item uses 2 polymorphic loci on *APOE* gene: rs429358 and rs7412. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all genes or loci that affect the genetic characteristics of APOE genotype.

How to use the test results

Notification

1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
2. The test results only predict your APOE genotype from the genetic level, and do not represent your real situation because other factors are not involved.

Population samples

The basis of the test is mainly from the research of Caucasian, and other populations are for reference only.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all or loci of APOE genotype.

Deep Sleep

Deep sleep, also called slow-wave sleep, refers to a slow and rhythmic sleep state where the electrical signals in the brain fall below 5 Hz, which can reflect the quality of sleep.

My Deep Sleep

Duration of deep sleep is relatively short

Suggestions

- Your deep sleep time is **relatively short**, and the effect time of sleep on eliminating fatigue, restoring energy and increasing immune ability is short.
- In addition to genetic factors, these Non-genetic factors can also reduce deep sleep time: excessive environmental stimulation, excessive caffeine intake, lack of exercise, smoking and drinking, high physical and mental stress, etc.
- Use the following methods to improve sleep:
 - Make sure to sleep in a quiet and comfortable environment, or play White noise can also improve sleep.
 - Do not drink Caffeinated drinks or Other refreshing drinks at a certain time before going to bed; If there is a lunch break, the lunch break time should also be controlled. It is suggested that it should not exceed 40 minutes.
 - Taking certain [^Sleep improving supplements] can help improve sleep, such as **melatonin**, **passion fruit** extract, glycine, etc.
 - Develop Good living habits.

* If you need additional dietary supplements, it is recommended that you seek help from an authoritative health expert or clinician.

Knowledge

Sleep stages

1. Rapid eye movement: Commonly known as 「dreaming stage」, people in this stage usually dream. Brain waves are similar to waking, but muscles are paralyzed and homeostasis is

suspended. Heart rate, ventricular pressure, cardiac output, arterial blood pressure, and respiratory rate are all irregular during this period.

2. Non-rapid eye movement: According to the different depth of sleep, physiology is subdivided into four periods, respectively is falling asleep period, light sleep period, sleeping period, deep sleep period. The first two periods are generally light sleep, the latter two periods are generally high quality of deep sleep, the latter two periods of deep sleep is referred to here non-rapid eye movement period.

Non-rapid eye movement and rapid eye movement period alternate periodically throughout an adult's night of sleep, with an interval of about 90 minutes between one rapid eye movement period and the next, and an adult experiencing 4-6 cycles per night. During the second half of sleep, there is less deep sleep in the non-rapid eye movement, but rapid eye movement duration gradually increases.

Deep sleep time

The deep sleep time you need can vary from person to person, with adults typically getting 15%~25% of a night's deep sleep. If you sleep for 8 hours, you usually get 72~120 minutes of deep sleep. In addition, the deep sleep time did not increase with the total time of sleep.

The benefits of deep sleep

During deep sleep, growth hormone is released in large quantities, which is important for the body's repair and growth. What's more, studies has shown that deep sleep plays an important role in memory consolidation.

Non-genetic factors affecting deep sleep

1. Reasonable diet, more intake of vitamin B, vitamin D rich food, calcium, magnesium and potassium ions, namely a variety of minerals, can promote deep sleep.
2. Studies shows that exercise can increase the amount of time you spend in deep sleep.
3. Alcohol, caffeine, tobacco and drugs all inhibit deep sleep.
4. Deep sleep can be adversely affected by illness or obesity.
5. Anxiety, stress, depression and other emotions can affect sleep quality and inhibit deep sleep.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS73598374	ADA	CC	common genotype, unlikely to be a deep sleeper

Test details

Applicable situation

The deep sleep item uses 1 polymorphic locus on *ADA* gene: rs73598374.

Notification

The test may not cover all genes or loci that affect the genetic characteristics of deep sleep.

How to use the test results

Notification

1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
2. The test results only predict your deep sleep from the genetic level, and do not represent your real situation because other factors are not involved.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all or loci that related to deep sleep.

Genetic Height

Height is the length of the longitudinal part of the human body, which originates from the longitudinal growth of the human body and is greatly affected by genetic factors. Some studies believe that the heritability of height can reach 80% ~ 90%.

My Genetic Height

161.7 CM

Suggestions

Height generally grows rapidly at the age of 12-13, and stops growing at the age of 19-23. The following points can help you improve your height:

1. Ensure **sleep**: children in growth period should go to bed before 11 o'clock every night. The daily sleep time: school-age children should not be less than 12 hours; And primary and secondary school students should not be less than 10 hours. Scientists have confirmed that cell proliferation, growth and development of the body are most vigorous during sleep. The secretion peak time of **growth hormone** controlling children's height is from 22 o'clock to 2:00 o'clock in the morning. If there is no sleep or repeated waking up during this period, it will affect the secretion of growth hormone.
2. Ensure balanced nutrition: Supplement enough **protein, calcium, vitamin**, etc., and appropriate amount of **calcium, iron, zinc, selenium** and other trace elements. At the same time, overnutrition should be avoided to prevent premature development due to obesity. Growing children are advised to drink a glass of **milk** before going to bed. Milk is an elevated food, which not only improves the quality of sleep but also helps the absorption of **calcium**.
3. Keep exercise: It is recommended that children in the growing period **do more exercise**, such as climbing stairs, skipping rope, pulling ups and so on.

Influencing factors

Controllable factors

1. Work and rest time: scientists have proved that cell proliferation, growth and development of the body are most vigorous during sleep. The secretion peak time of growth hormone controlling

children's height is from 22 o'clock to 2:00 o'clock in the morning. If there is no sleep or repeated waking up at this period, it will affect the secretion of growth hormone, and ultimately affect the height.

2. Nutritional factors: adequate nutrition will promote bone growth. If the intake of protein, calcium, vitamins and trace elements is insufficient, it will affect height development. Overnutrition will cause obesity, which will lead to early puberty, and thus limit height.
3. Lack of exercise: physical exercise can strengthen the blood circulation of the whole body. If the epiphyseal chondrocytes get adequate nutrition, it can accelerate the proliferation and ossification of the epiphyseal chondrocytes, make the bone grow rapidly. And thus the human body grows taller.



Uncontrollable factors

1. Genetic factors: the height is affected by heredity by about 60-80%, and the remaining 20-40% is affected by environment, mainly nutritional factors. There are many loci that influence height, but the effect of each locus is relatively low.
2. Temperature and humidity: in a hot and humid environment, the temperature of a shorter people's body rises slowly and can cool down more efficiently. Therefore, the height of people living in tropical jungle will be lower than that of people living in grassland.
3. Mental factors: Studies have found that children with severe mental trauma have growth retardation or even stagnation. This is because bad emotions will affect the function of brain and endocrine system, and thus affect the height.

Knowledge



Height and diseases

1. Height and cardiovascular disease: The shorter the height, the greater the risk of heart disease. It may be because a taller person has a stronger vascular system and better nutritional status. According to a New England magazine, people who are 6.5 cm below average have an increased risk of heart disease by about 13.5%.
2. The relationship between height and cancer: The higher the height, the higher the risk of cancer. It may be because a taller person has more cells, so may accumulate more mutations. Research shows that for every 6.5 cm increase in height, the risk of cancer increases by 4 percent.
3. The relationship between height and fracture: Taller people are more likely to have fractures. It may be because a tall person falls, and the body will be more shocked. At the same time, taller people have longer reflex arcs because nerve impulses travel longer distances.
4. The relationship between height and thrombosis: The risk of venous thromboembolism in men with a height of 180cm or more is 2.6 times that of men with a height about 170cm.

-
5. The relationship between height and cervical vertebra and spine: Taller people may have a higher risk of cervical spondylosis or scoliosis because their body is not conducive to stretching during long-term work.
-

Computational model

The height prediction model of our product uses machine learning algorithms to fit the relationship between genes and height of nearly 100,000 We users. It uses height databases in Japan, Europe and other regions as a reference. We assume that the effects of gene loci that affect height can be superimposed, calculate the eigenvalues of users at different polymorphic loci. Then we can use the model to weight the eigenvalues to calculate the deviation value of height, and then correct it with principal component analysis, age, gender and other information to obtain the final prediction result.

How to understand the prediction results

Notification

The results are predicted by the machine learning algorithm independently developed by us. As the number of feedback users continues to increase, your prediction results will become more and more accurate.

Sample source

The prediction algorithm is based on nearly 100000 user feedback datas from our products and combined with other population databases, mainly the users of Chinese, Japanese and Europeans.

Limit of prediction

The relationship between height and heredity is very complex. Science has found that a large number of genetic loci have a weak effect on height. This test is limited by the number of samples and cannot cover all loci associated with height.

Reference

- [Yengo, Loic, et al. "Meta-analysis of genome-wide association studies for height and body mass index in ~ 700000 individuals of European ancestry." Human molecular genetics 27.20 \(2018\): 3641-3649.](#)
- [Akiyama, Masato, et al. "Characterizing rare and low-frequency height-associated variants in the Japanese population." Nature communications 10.1 \(2019\): 1-11.](#)

Human Milk Oligosaccharides (HMO) Secretor Status

HMO secretor status refers to the ability to secrete specific breast milk oligosaccharides (HMO), breast-feeding women can be divided into normal secretion type and non-secretion type.

My Human Milk Oligosaccharides (HMO) Secretor Status

secretor

Suggestions

When conditions permit, adhere to breastfeeding; When infant formula is needed, the formula containing oligosaccharide is preferred.

Knowledge

What is the human milk oligosaccharides

Breast milk is an ideal substance that provides energy and nutrition for the baby 6 months before birth. Babies who lack breastfeeding may have a higher risk of immunodeficiency, reduced intelligence, and obesity and diabetes in adulthood.

The composition of breast milk is very complex. One of the important components is called human milk oligosaccharides (HMOs), which is the third highest solid component in breast milk, second only to lactose and milk fat. Human breast milk contains at least more than 200 types of oligosaccharides, of which 2'-fucose lactose (2'-FL) is the most abundant oligosaccharide in breast milk.

Human milk oligosaccharides are unique components in breast milk. Compared with cow's milk and goat's milk, the content of breast milk oligosaccharides in human breast milk is 100-1000 times higher. Therefore, milk powder made from cow's milk often lacks oligosaccharides.

The function of human milk oligosaccharides

1. Regulate infant intestinal flora: Human milk oligosaccharides are excellent substrates for probiotics such as bifidobacteria, which can reduce the nutrition of harmful flora and help

promote the formation of beneficial intestinal flora in the intestines of infants and young children. This beneficial effect can last for 2-3 years in the baby's body.

2. Promote brain development: The metabolites of human milk oligosaccharides such as sialic acid can promote brain development, neuronal transmission and synapse formation, and are potentially essential nutrients for brain development and cognitive formation.
3. Anti-adhesive antibacterial agent: The special structure of human milk oligosaccharides can prevent pathogens (viruses, bacteria, etc.) from adhering to the mucosal surface of infants, reducing the possibility of diarrhea and systemic infection.
4. Regulate intestinal epithelial cell response: Intestinal health and barrier function are the first barrier of natural immunity. Human milk oligosaccharides can regulate the response of epithelial cells to specific allergic diseases, and may also help prevent food allergies.
5. Immune regulation: More and more studies have shown that human milk oligosaccharides not only indirectly affect the infant's immune system by changing the intestinal flora, but also directly regulate the immune response by affecting the immune cell population and the secretion of cytokines to help resist microbial infections such as enterotoxigenic *Escherichia coli* and rotavirus.

Influencing factors of human milk oligosaccharides

1. Genetic factors: *FUT2* gene is related to the secretion of human milk oligosaccharides. The *FUT2* gene is a non-secreting type, and the content of 2'-fucose lactose in breast milk is significantly lower than that of a normal-secreting type.
2. Lactation stage: The content of breast milk oligosaccharides in colostrum can reach 20-25 g/L, while the content of mature milk is 10-15 g/L.
3. Geographical factors: Even if genetic differences are considered, geographic factors can affect the content of breast milk oligosaccharides.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1047781	FUT2	AT	normal content of 2'-fucosyllactose and other oligosaccharides in breast milk
RS601338	FUT2	GG	normal content of 2'-fucosyllactose and other oligosaccharides in breast milk

Test details

Applicable situation

The genetic characteristics item of HMO secretor status uses 2 polymorphic loci rs1047781 and rs601338 on the *FUT2* gene.

Notification

1. The basis of the test is mainly from the research of Caucasian and Asian, other populations are for reference only.
2. The test report may not cover all genes or loci that affect HMO secretor status.

How to use the test results

Notification

1. An individual's genetic features are the common result of the interaction of genes, environment, and time.
2. This test result only predicts your HMO secretor status from the genetic level. Since the test does not involve consideration of other factors, it does not mean your true health status.

Population samples

The basis of the test is mainly from the research of Caucasian and Asian, other populations are for reference only.

Limit of detection

Limited by the current technology and the level of scientific cognition, and the test may not cover all or loci related to HMO secretor status.



Genetic Disease

319 Reports

Normal 318
Carrier 1

Item	Genetic risk
Agenesis of The Corpus Callosum with Peripheral Neuropathy (ACCPN) Non-carrier	
Alpha-1 Antitrypsin Deficiency Non-carrier	
Phosphomannomutase 2-Congenital Disorder of Glycosylation (PMM2-CDG) Non-carrier	
Familial Hypercholesterolemia Type B Non-carrier	
Glucose-6-Phosphate Dehydrogenase Deficiency (G6PD) Non-carrier	
Hereditary Fructose Intolerance Non-carrier	
Hypertrophic Cardiomyopathy Carrier of 1 risk allele	
TTR-Related Cardiac Amyloidosis Non-carrier	
Torsion Dystonia Non-carrier	
Zellweger Syndrome Spectrum Non-carrier	
Glutaric Aciduria Type 1 (GA1) Non-carrier	

3-Methylcrotonyl CoA Carboxylase 2 Deficiency

Non-carrier



Argininosuccinate Lyase Deficiency

Non-carrier



Alport Syndrome

Non-carrier



Pendred Syndrome

Non-carrier



Stickler Syndrome

Non-carrier



Usher Syndrome

Non-carrier



X-Linked Non-Syndromic Hereditary Deafness

Non-carrier



Large Vestibular Aqueduct Syndrome

Non-carrier



Autosomal Dominant Non-Syndromic Deafness

Non-carrier



Autosomal Recessive Non-Syndromic Deafness

Non-carrier



Aminoglycoside-Induced Deafness

Non-carrier



Mitochondrial Hereditary Non-Syndromic Deafness

Non-carrier



Peroxisome Biosynthesis Disorder

Non-carrier



Branchiootorenal Spectrum Disorder

Non-carrier



Jervell-Lange-Nielsen Syndrome

Non-carrier



Mohr-Tranebjaerg Syndrome

Non-carrier



Early-Onset Alzheimer's Disease

Non-carrier



Brugada Syndrome

Non-carrier



Loeys Dietz Syndrome

Non-carrier



Wolff-Parkinson-White Syndrome

Non-carrier



Sick Sinus Syndrome

Non-carrier



Short QT Syndrome (SQTS)

Non-carrier



Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

Non-carrier



Familial Thoracic Aortic Aneurysm

Non-carrier



Dilated Cardiomyopathy

Non-carrier



Marfan Syndrome

Non-carrier



Noonan Syndrome

Non-carrier



Restrictive Cardiomyopathy

Non-carrier



Primary Pulmonary Hypertension

Non-carrier



Long QT Syndrome (LQTS)

Non-carrier



Arrhythmogenic Right Ventricular Cardiomyopathy

Non-carrier



Left Ventricular Noncompaction

Non-carrier



Familial Atrial Fibrillation

Non-carrier



Haemophilia A

Non-carrier



Haemophilia B

Non-carrier



Duchenne Muscular Dystrophy (DMD)

Non-carrier



Pulmonary Lymphangi leiomyomatosis (PLAM)

Non-carrier



Pheochromocytoma

Non-carrier



Dementia with Lewy Body (DLB)

Non-carrier



Angelman Syndrome (AS)

Non-carrier



Galactosialidosis (GSL)

Non-carrier



Galactosemia (GALAC)

Non-carrier



Megalencephalic Leukoencephalopathy with Subcortical Cysts (MLC)

Non-carrier



Osteopathia Striata with Cranial Sclerosis (OSCS)

Non-carrier



Phenylketonuria (PKU)

Non-carrier



Propionic Acidemia (PA)

Non-carrier



Pyruvate Kinase Deficiency (PKD)

Non-carrier



Pyruvate Carboxylase Deficiency (PCD)

Non-carrier



Syndactyly

Non-carrier



Vitreoretinchoroidopathy (VRCP)

Non-carrier



Osteogenesis Imperfecta (OI)

Non-carrier



Acth-independent Macronodular Adrenal Hyperplasia (AIMAH)

Non-carrier



Epidermolysis Bullosa (EB)

Non-carrier



Cholestasis

Non-carrier



Hypokalemic Periodic Paralysis (HKOPP)

Non-carrier



Hypophosphatemic Rickets (HR)

Non-carrier



Hypophosphatasia (HPP)

Non-carrier



Brachydactyly

Non-carrier



Multiple Sulfatase Deficiency (MSD)

Non-carrier



Polycystic Kidney Disease (PKD)

Non-carrier



Congenital Multicore Myopathy with External Ophthalmoplegia

Non-carrier



Multisystemic Smooth Muscle Dysfunction Syndrome (MSMDS)

Non-carrier



Frontometaphyseal Dysplasia (FMD)

Non-carrier



Otospondylomegaepiphyseal Dysplasia (OSMED)

Non-carrier



Developmental and Epileptic Encephalopathy (DEE)

Non-carrier



Maple Syrup Urine Disease (MSUD)

Non-carrier



Combined Oxidative Phosphorylation Deficiency (COXPD)

Non-carrier



Nemaline Myopathy (NM)

Non-carrier



Metaphyseal Dysplasia without Hypotrichosis (MDWH)

Non-carrier



Homocystinuria (HCU)

Non-carrier



Hyperphosphatasia with Intellectual Disability Syndrome

Non-carrier



Hyperornithinemia-Hyperammonemia-Homocitrullinuria Syndrome (HHH)

Non-carrier



Hyperinsulinemic Hypoglycemia (HH)

Non-carrier



Kabuki Syndrome (KS)

Non-carrier



Ataxia-Telangiectasia (AT)

Non-carrier



Osteopetrosis

Non-carrier



Citrullinemia

Non-carrier



Popliteal Pterygium Syndrome (PPS)

Non-carrier



Alkaptonuria

Non-carrier



Myofibrillar Myopathy (MFM)

Non-carrier



Diastrophic Dysplasia

Non-carrier



Spinal Muscular Atrophy (SMA)

Non-carrier



Spondyloperipheral Dysplasia (SPD)

Non-carrier



Spondylotarsal Synostosis Syndrome (SCT)

Non-carrier



Spondylometaphyseal Dysplasia (SMD)

Non-carrier



Spondyloepiphyseal Dysplasia (SED)

Non-carrier



Spondyloepimetaphyseal Dysplasia (SEMD)

Non-carrier



Spondylocostal Dysostosis (SCDO)

Non-carrier



Familial Partial Lipodystrophy (FPL)

Non-carrier



Hypobetalipoproteinemia (HBL)

Non-carrier



Familial Cold Autoinflammatory Syndrome (FCAS)

Non-carrier



Familial erythrocytosis

Non-carrier



Familial Amyotrophic Lateral Sclerosis (FALS)

Non-carrier



Familial Exudative Vitreoretinopathy (FEVR)

Non-carrier



Familial Dysautonomia (FD)

Non-carrier



Nail-patella Syndrome (NPS)

Non-carrier



Methylmalonic Aciduria

Non-carrier



Methylmalonic Aciduria and Homocystinuria

Non-carrier



Methylmalonic Acidemia

Non-carrier



Methylmalonic Acidemia with Homocystinuria

Non-carrier



Mevalonic Aciduria (MA)

Non-carrier



Pseudohypoparathyroidism (PHP)

Non-carrier



Pseudopseudohypoparathyroidism (PPHP)

Non-carrier



Pseudohypoaldosteronism (PHA)

Non-carrier



Progressive Pseudorheumatoid Dysplasia (PPRD)

Non-carrier



Progressive External Ophthalmoplegia (PEO)

Non-carrier



Arginase Deficiency (ARG)

Non-carrier



Focal Segmental Glomerulosclerosis (FSGS)

Non-carrier



Giant Axonal Neuropathy (GAN)

Non-carrier



Tyrosinemia

Non-carrier



Combined Pituitary Hormone Deficiency (CPHD)

Non-carrier



Leucine-Induced Hypoglycemia (LIH)

Non-carrier



Craniofrontonasal Syndrome (CFNS)

Non-carrier



Cranioectodermal Dysplasia (CED)

Non-carrier



Osteoglophonic Dysplasia

Non-carrier



Trichorhinophalangeal Syndrome (TRPS)

Non-carrier



Immunodeficiency

Non-carrier



Cystic Fibrosis (CF)

Non-carrier



Hydrocephalus Syndrome

Non-carrier



Pontocerebellar Hypoplasia (PCH)

Non-carrier



Mucopolysaccharidosis (MPS)

Non-carrier



Mucopolipidosis

Non-carrier



Ornithine Aminotransferase Deficiency (OAT)

Non-carrier



Factor V Deficiency

Non-carrier



Factor VII Deficiency

Non-carrier



Factor XI Deficiency

Non-carrier



Skin/Hair/Eye Pigmentation (SHEP)

Non-carrier



Cutis Laxa (CL)

Non-carrier



Lissencephaly

Non-carrier



GLUT1 Deficiency Syndrome

Non-carrier



Holoprosencephaly (HPE)

Non-carrier



Maturity-Onset Diabetes of the Young (MODY)

Non-carrier



Achromatopsia

Non-carrier



Holocarboxylase Synthetase Deficiency (HCSD)

Non-carrier



Hypochondroplasia

Non-carrier



Branchiooculofacial Syndrome (BOFS)

Non-carrier



Trifunctional Protein Deficiency (TFP)

Non-carrier



Incontinentia Pigmenti (IP)

Non-carrier



Neurodegeneration with Brain Iron Accumulation (NBIA)

Non-carrier



Neuronal Ceroid Lipofuscinosis (NCL)

Non-carrier



Nephropathic Cystinosis

Non-carrier



Renal-Hepatic-Pancreatic Dysplasia (RHPD)

Non-carrier



Nephronophthisis (NPHP)

Non-carrier



Renal Carnitine Transport Defect

Non-carrier



Biotinidase Deficiency (BTD)

Non-carrier



Optic Atrophy (OA)

Non-carrier



Retinitis Pigmentosa (RP)

Non-carrier



Cone-Rod Dystrophy (CRD)

Non-carrier



Hypomyelination and Congenital Cataract (HCC)

Non-carrier



Glycogen Storage Disease (GSD)

Non-carrier



Isolated Growth Hormone Deficiency (IGHD)

Non-carrier



Aspartylglucosaminuria (AGU)

Non-carrier



Sideroblastic Anemia (SA)

Non-carrier



Auditory Neuropathy (AN)

Non-carrier



Elliptocytosis

Non-carrier



Vitamin D-Dependent Rickets (VDDR)

Non-carrier



Striatonigral Degeneration (SND)

Non-carrier



Agammaglobulinemia

Non-carrier



Aniridia

Non-carrier



Choroideremia

Non-carrier



Glutaric Acidemia (GA)

Non-carrier



Mandibuloacral Dysplasia with Type A Lipodystrophy (MADA)

Non-carrier



Chediak-Higashi Syndrome (CHS)

Non-carrier



Congenital Bile Acid Synthesis Defect (CBAS)

Non-carrier



Arthrogryposis Multiplex Congenita (AMC)

Non-carrier



Coenzyme Q10 Deficiency, Primary (COQ10D)

Non-carrier



Congenital Dyserythropoietic Anemia (CDA)

Non-carrier



Dyskeratosis Congenita (DC)

Non-carrier



Congenital Stationary Night Blindness (CSNB)

Non-carrier



Congenital Disorder of Deglycosylation (CDDG)

Non-carrier



Congenital Generalized Lipodystrophy (CGL)

Non-carrier



Congenital Disorder of Glycosylation (CDG)

Non-carrier



Congenital Amegakaryocytic Thrombocytopenia (CAMT)

Non-carrier



Congenital Fibrosis of Extraocular Muscles (CFEOM)

Non-carrier



Congenital Ichthyosis

Non-carrier



Acyl-Coenzyme A Dehydrogenase Deficiency

Non-carrier



Sialidosis

Non-carrier



Mitochondrial DNA Depletion Syndrome (MDS)

Non-carrier



Mitochondrial Complex II Deficiency (MC2D)

Non-carrier



Microcephalic Osteodysplastic Primordial Dwarfism (MOPD)

Non-carrier



Microcephaly-Capillary Malformation Syndrome (MICCAP)

Non-carrier



Cardiofaciocutaneous Syndrome (CFC)

Non-carrier



Selective Tooth Agenesis (STA)

Non-carrier



Hemochromatosis

Non-carrier



Thrombocytopenia

Non-carrier



Fucosidosis

Non-carrier



Fundus Albipunctatus (FA)

Non-carrier



Oculocutaneous Albinism (OCA)

Non-carrier



Pancreatic Agenesis (PAGEN)

Non-carrier



Hereditary Sensory and Autonomic Neuropathy (HSAN)

Non-carrier



Hereditary Pancreatitis (HP)

Non-carrier



Hereditary Desmoid Disease (HDD)

Non-carrier



Hereditary Motor and Sensory Neuropathy (HMSN)

Non-carrier



Metachromatic Leukodystrophy (MLD)

Non-carrier



Isovaleric Acidemia (IVA)

Non-carrier



Thrombophilia

Non-carrier



Infantile Cerebellar-Retinal Degeneration (ICRD)

Non-carrier



Permanent Neonatal Diabetes Mellitus (PNDM)

Non-carrier



Juvenile Polyposis Syndrome (JPS)

Non-carrier



Fish-Eye Disease (FED)

Non-carrier



Primary Hyperoxaluria (PH)

Non-carrier



Distal Myopathy with Anterior Tibial Onset (DMAT)

Non-carrier



Akinesia Deformation Sequence (ADS)

Non-carrier



Xeroderma Pigmentosum (XP)

Non-carrier



Limb-Girdle Muscular Dystrophy (LGMD)

Non-carrier



Acrocallosal Syndrome (ACLS)

Non-carrier



Rhizomelic Chondrodysplasia Punctata (RCDP)

Non-carrier



Sitosterolemia

Non-carrier



Acromicric Dysplasia

Non-carrier



Thanatophoric Dysplasia

Non-carrier



Lethal Congenital Contracture Syndrome (LCCS)

Non-carrier



Intellectual Disability-Hypotonic Facies Syndrome

Non-carrier



Centronuclear Myopathy (CNM)

Non-carrier



Choroidal Dystrophy, Central Areolar (CACD)

Non-carrier



Severe Combined Immunodeficiency (SCID)

Non-carrier



Wrinkly Skin Syndrome (WSS)

Non-carrier



Spondyloenchondrodysplasia with Immune Dysregulation (SPENCDI)

Non-carrier



Lymphoproliferative Syndrome

Non-carrier



3-Methylglutaconic Aciduria (MGA)

Non-carrier



Aicardi-Goutieres Syndrome (AGS)

Non-carrier



Alstrom Syndrome (AS)

Non-carrier



Axenfeld-Rieger Syndrome (ARS)

Non-carrier



Baraitser-Winter Syndrome (BRWS)

Non-carrier



Bardet-Biedl Syndrome (BBS)

Non-carrier



Bartter Syndrome

Non-carrier



Becker Muscular Dystrophy (BMD)

Non-carrier



Bloom Syndrome (BLM)

Non-carrier



Bohring-Opitz Syndrome (BOS)

Non-carrier



Borjeson-Forssman-Lehmann Syndrome (BFLS)

Non-carrier



Bruck Syndrome (BS)

Non-carrier



Charcot-Marie-Tooth Disease (CMT)

Non-carrier



Cockayne Syndrome (CS)

Non-carrier



Coffin-Lowry Syndrome (CLS)

Non-carrier



Cohen Syndrome (COH)

Non-carrier



Cornelia de Lange Syndrome (CdLS)

Non-carrier



Costello Syndrome

Non-carrier



Crigler-Najjar Syndrome (CNS)

Non-carrier



Crouzon Syndrome

Non-carrier



D-2-hydroxyglutaric Aciduria (D2HGA)

Non-carrier



De Sanctis-Cacchione Syndrome (DSC)

Non-carrier



Dent Disease

Non-carrier



Desbuquois Dysplasia

Non-carrier



Diamond-Blackfan Anemia (DBA)

Non-carrier



Duane Syndrome (DS)

Non-carrier



Ehlers-Danlos Syndrome (EDS)

Non-carrier



Ellis-van Creveld Syndrome (EVC)

Non-carrier



Fabry Disease

Non-carrier



Fanconi Anemia (FA)

Non-carrier



FG Syndrome

Non-carrier



Floating-Harbor Syndrome (FHS)

Non-carrier



Gaucher Disease (GD)

Non-carrier



Geleophysic Dysplasia

Non-carrier



Griscelli Syndrome (GS)

Non-carrier



Hennekam Lymphangiectasia-Lymphedema Syndrome (HKLLS)

Non-carrier



Hermansky-Pudlak Syndrome (HPS)

Non-carrier



Hutchinson-Gilford Progeria Syndrom (HGPS)

[Non-carrier](#)



Johanson-Blizzard Syndrome (JBS)

[Non-carrier](#)



Joubert Syndrome (JS)

[Non-carrier](#)



KBG Syndrome

[Non-carrier](#)



Kenny-Caffey Syndrome (KCS)

[Non-carrier](#)



Kniest Dysplasia

[Non-carrier](#)



L1 Syndrome

[Non-carrier](#)



Leber Congenital Amaurosis (LCA)

[Non-carrier](#)



Leigh Syndrome

[Non-carrier](#)



Lesch-Nyhan Syndrome (LNS)

[Non-carrier](#)



Lowe Syndrome

[Non-carrier](#)



Meckel Syndrome (MKS)

[Non-carrier](#)



Meier-Gorlin Syndrome (MGS)

[Non-carrier](#)



Miyoshi Muscular Dystrophy (MMD)

[Non-carrier](#)



Mowat-Wilson Syndrome (MWS)

[Non-carrier](#)



Myhre Syndrome

[Non-carrier](#)



Nance-Horan Syndrome (NHS)

[Non-carrier](#)



Neu-Laxova Syndrome (NLS)

[Non-carrier](#)



Nicolaides-Baraitser Syndrome (NCBRS)

[Non-carrier](#)



Niemann-Pick Disease (NPD)

[Non-carrier](#)



Ogden Syndrome

[Non-carrier](#)



Pelizaeus-Merzbacher Disease (PMD)

[Non-carrier](#)



Rapadilino Syndrome

[Non-carrier](#)



Refsum Disease (RD)

[Non-carrier](#)



Renpenning Syndrome (RENS)

[Non-carrier](#)



Rett Syndrome (RTT)

[Non-carrier](#)



Revesz Syndrome

[Non-carrier](#)



Roberts Syndrome

[Non-carrier](#)



Robinow Syndrome

[Non-carrier](#)



Rothmund-Thomson Syndrome (RTS)

[Non-carrier](#)



Rubinstein-Taybi Syndrome (RSTS)

[Non-carrier](#)



Saethre-Chotzen Syndrome (SCS)

[Non-carrier](#)



Salla Disease (SD)

[Non-carrier](#)



Sandhoff Disease

[Non-carrier](#)



Seckel Syndrome

[Non-carrier](#)



Sengers Syndrome

[Non-carrier](#)



Senior-Loken Syndrome (SLSN)

[Non-carrier](#)



Shwachman-Diamond Syndrome (SDS)

[Non-carrier](#)



Smith-Lemli-Opitz Syndrome (SLOS)

[Non-carrier](#)



Smith-McCort Dysplasia (SMC)

[Non-carrier](#)



Sotos Syndrome

[Non-carrier](#)



Stargardt Disease

[Non-carrier](#)



Tay-Sachs Disease (TSD)

[Non-carrier](#)



Treacher Collins Syndrome (TCS)

[Non-carrier](#)



Troyer Syndrome

[Non-carrier](#)



Warburg Micro Syndrome (WARBM)

Non-carrier



Weill-Marchesani Syndrome (WMS)

Non-carrier



Werner Syndrome

Non-carrier



Wilson Disease

Non-carrier



Wiskott-Aldrich Syndrome (WAS)

Non-carrier



Wolfram Syndrome

Non-carrier



Yunis-Varon Syndrome (YVS)

Non-carrier



Alpha-N-Acetylgalactosaminidase Deficiency (α -NAGA)

Non-carrier



Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy is a type of common single gene hereditary cardiovascular disease, with 70% having familial HCM (FHcM). Its characteristic manifestation is the occurrence of asymmetric cardiac hypertrophy without an obvious cause of cardiac hypertrophy (such as aortic stenosis or systemic hypertension, etc.). Pathological changes mainly involve the unexpanded left ventricular septal. A relatively large sample of epidemiological studies showed that there are at least 1 million HCM patients in China. In 1995, the World Health Organization (WHO) classified it as a type of primary cardiomyopathy. Its clinical manifestations range from no obvious clinical symptoms to chest tightness, shortness of breath, dyspnea, malignant arrhythmia, heart failure and death. Hypertension, valve diseases and other diseases must be ruled out, as well as athletic hypertrophy.

Hypertrophic cardiomyopathy is not at all a rare disease. The process of clinical change in hypertrophic cardiomyopathy varies, with many patients being asymptomatic or having mild symptoms and a stable condition; some may maintain a good condition for 5-10 years. Adults who are admitted to large treatment centers have an annual mortality rate of about 3%. However, if the total number of HCM patients is counted, the annual mortality rate may be closer to 1%. Clinical progression is generally slow (except in the case of sudden death). Hypertrophic cardiomyopathy (HCM) is an important cause of sudden cardiac death in young people. The risk of sudden death in children is rather high, and can reach up to 6% each year. The clinical manifestations and course of disease of hypertrophic cardiomyopathy are diverse and complex. The majority of patients can survive long-term, but cardiac arrest, heart failure and fatal stroke, and other adverse events may occur. It is important to understand the characteristics of hypertrophic cardiomyopathy and identify patients who are at risk of hypertrophic cardiomyopathy.

My Hypertrophic Cardiomyopathy

Carrier of 1 risk allele

Knowledge

Time of onset

Onset generally in the middle and old age, the average age of onset is 38 ± 15 years old.

Prevalence

Hypertrophic cardiomyopathy affects about 1 in 500 people worldwide and is the most common inherited heart disease in the United States. According to statistics, there are about 1 million patients with hypertrophic cardiomyopathy in China.

Typical symptoms

Symptoms include exertive dyspnea, tightness and pain in the precardiac area, and frequent transient syncope. The main basis of clinical diagnosis is left ventricular hypertrophy diagnosed by two-dimensional echocardiography.

Treatment

The main principles of treatment are relief of symptoms and prevention of sudden death.

For relief: beta blockers such as propranolol, metoprolol, etc. For refractory patients, surgical treatment, implantation of a human pacemaker (ICD), and heart transplantation may also be considered.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893748	MYL3	TT	normal
RS730880336	MYBPC3	DD	normal
RS730880629	MYBPC3	TT	normal
RS730880620	intergenic	CC	normal
RS730880597	MYBPC3	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The genetic disease screening item for Hypertrophic cardiomyopathy uses the 362 polymorphic loci on the *MYBPC3* gene. The test results only indicate the user's carrier status of the disease-causing gene or locus for hypertrophic cardiomyopathy. The basis of the test is mainly from the research of Asian, other populations are for reference only.

Notification

1. The test does not cover all genes or loci that affect hypertrophic cardiomyopathy.
2. The test result is not applicable to clinical diagnosis.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by multiple factors such as genes and living environment. This test will only screen you if you carry the genes or loci that cause hypertrophic cardiomyopathy.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have hypertrophic cardiomyopathy.
4. If you have a family history of hypertrophic cardiomyopathy, or you think that you are likely to suffer from hypertrophic cardiomyopathy, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 362 polymorphic loci associated with hypertrophic cardiomyopathy including rs36212066.

Population samples

The basis of the test is mainly from the research of Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test does not cover all genes or loci that affect hypertrophic cardiomyopathy.

Agenesis of The Corpus Callosum with Peripheral Neuropathy (ACCPN)

Agenesis of the corpus callosum with peripheral neuropathy, also known as Andermann syndrome, is an autosomal recessive genetic disease in which there is agenesis, loss or deformity of the transverse nerve fiber bundle that connects the left and right hemispheres of the brain (the corpus callosum) due to sensory and motor nerve lesions.

My Agenesis of The Corpus Callosum with Peripheral Neuropathy (ACCPN)
Non-carrier

Knowledge

Time of onset

In the process of infant development, various neurological symptoms gradually appear.

Prevalence

The disease is common in northeastern Quebec and Charlevoix, with an incidence rate of about 1/2000. It is rare in other regions.

Typical symptoms

Abnormal or loss of nerve reflexes, weak muscle tone, developmental delay, mental retardation, and may be accompanied by epilepsy, head and facial abnormalities, and mental disorders.

Treatment

At present, there is no effective treatment. For concomitant epilepsy, medication can be used to control it.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121908428	SLC12A6	GG	normal

Test details

Applicable situation

The health risks of corpus callosum with peripheral nerve hypoplasia item uses a polymorphic locus in *SLC12A6*. The test results only illustrate the user's carrying situation of genes or sites that related to corpus callosum with peripheral nerve hypoplasia. The test was mainly based on Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that could lead to a risk of corpus callosum with peripheral nerve hypoplasia.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This test only screens you if you carry a disease-causing gene or locus of the corpus callosum with peripheral nerve hypoplasia.
3. It should be noted that carrying the relevant mutation does not represent your true disease state. If you do not carry the relevant mutation, it cannot be ruled out that you may have a disease of the corpus callosum with peripheral nerve hypoplasia.
4. If you have a family history of corpus callosum with peripheral nerve hypoplasia, or you think that you are likely to suffer from corpus callosum with peripheral nerve hypoplasia, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes rs121908428, a polymorphic locus related to the risk of corpus callosum with peripheral nerve hypoplasia.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci related to corpus callosum with peripheral nerve hypoplasia.

Alpha-1 Antitrypsin Deficiency

α -1 antitrypsin deficiency is an autosomal dominant genetic disease and a type of protein deficiency disease.

My Alpha-1 Antitrypsin Deficiency

Non-carrier

Knowledge



Time of onset

It can be onset at any age and is common in newborns, infants and young children.



Prevalence

It is more common in the Nordic population, and the incidence in North America is about 1/5000-7000.



Typical symptoms

Clinically, it often leads to neonatal hepatitis, cirrhosis of the liver, liver cancer and emphysema in infants and adults.



Treatment

There is currently no specific treatment, mainly symptomatic treatment. Due to the characteristics of α -1 antitrypsin deficiency (AATD), the risk of the following diseases is increased: adult chronic obstructive pulmonary disease, that is, emphysema, persistent airflow obstruction and (or) chronic bronchitis. Therefore, patients with fixed airflow obstruction are recommended to take intravenous intensive treatment (regular infusion of purified AAT to increase the serum concentration of AAT). Patients with advanced lung disease can consider lung transplantation, while for patients with advanced liver disease, liver transplantation is a radical treatment.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS17580	SERPINA1	TT	normal
RS28929474	SERPINA1	CC	normal

Test details

Applicable situation

The health risks of alpha-1 antitrypsin deficiency item uses 2 polymorphic loci in *SERPINA1*. The test results only illustrate the user's carrying situation of genes or sites that related to alpha-1 antitrypsin deficiency. The test was mainly based on Caucasian, other populations are for reference only.

Notification

1. This test may not to cover all genes or sites that could lead to a risk of alpha-1 antitrypsin deficiency.
2. The test results are not applicable to clinical practice.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This test only screens you if you carry a disease-causing gene or locus of the alpha-1 antitrypsin deficiency.
3. It should be noted that carrying the relevant mutation does not represent your true disease state. If you do not carry the relevant mutation, it cannot be ruled out that you may have a disease.
4. If you have a family history of alpha-1 antitrypsin deficiency, or you think that you are likely to suffer from alpha-1 antitrypsin deficiency, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 2 polymorphic loci related to disease, including rs17580 and rs2892947.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci related to alpha-1 antitrypsin deficiency.

Phosphomannomutase 2-Congenital Disorder of Glycosylation (PMM2-CDG)

Congenital disorders of glycosylation (CDG), formerly known as carbohydrate-deficient glycoprotein syndrome (CDGS), is a type of autosomal recessive disease caused by a defect in the process of synthesis and binding of glycans to other complexes (proteins and lipids).

My Phosphomannomutase 2-Congenital Disorder of Glycosylation (PMM2-CDG)

Non-carrier

Knowledge

Time of onset

Onset mostly in infancy.

Prevalence

To date, there are approximately 700 cases with the most common glycosylation type IA including patients from Asia, Australia, Europe, South America, and North America.

Typical symptoms

Symptoms usually appear in infancy. Symptoms include hypotonia, nipple retraction, abnormal fat distribution, strabismus, growth retardation, cerebellar dysplasia, and motor ataxia.

Treatment

Currently, there is no special treatment method, symptomatic treatment is mostly used.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS28936415	PMM2	GG	normal

Test details

Applicable situation

The congenital disorder of glycosylation type IA item provided uses the one polymorphic locus on the *PMM2* gene. The test results only indicate the user's carrier status of the disease-causing gene or locus for Congenital disorder of glycosylation type IA. It does not mean the user's true Congenital disorder of glycosylation type IA. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect Congenital disorder of glycosylation type IA.
2. The test results are not applicable to clinical practice.
3. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease is determined by multiple factors such as genes and living environment. This test will only screen you if you carry the genes or loci that cause Congenital disorder of glycosylation type IA.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Congenital disorder of glycosylation type IA.
4. If you have a family history of Congenital disorder of glycosylation type IA, or you think that you are likely to suffer from Congenital disorder of glycosylation type IA, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes rs28936415, a polymorphic locus associated with Congenital disorder of glycosylation type IA.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect congenital disorder of glycosylation type IA.

Familial Hypercholesterolemia Type B

Familial hypercholesterolemia is a rare hereditary disease with familial characteristics.

My Familial Hypercholesterolemia Type B

Non-carrier

Influencing factors



Controllable factors

1. Diet: Eating meat, animal offal, and greasy food frequently will raise your cholesterol levels.
2. Physical activity: Physical inactivity or sitting for a long time will increase the total cholesterol levels.
3. Weight: Being overweight or obese can increase the levels of Low-Density Lipoprotein Cholesterol (LDL-C).
4. Smoking: Smoking will reduce High density lipoprotein cholesterol (HDL-C) levels and increase the risk of blood clots.
5. Mental factors: Prolonged mental stress can increase the levels of blood cholesterol.



Uncontrollable factors

1. Family history: Familial hypercholesterolemia patients have genetic predisposition and family aggregation. Most people with family history suffer from this disease.
2. Age: The blood cholesterol levels in the blood increase as age, especially for men after age 40 and postmenopausal women.

Knowledge



Time of onset

The disease can occur at any age, especially for men after age 40 and postmenopausal women.



Prevalence

The incidence of Familial hypercholesterolemia is approximately 1:500.

Typical symptoms

The disease usually produce no symptoms, occasionally cause xanthomas and coronary heart disease.

Treatment

Strict diet, strengthening exercise and drug therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS12713559	APOB	GG	normal
RS144467873	APOB	GG	normal
RS112029328	intergenic	--	Not Detected

Test details

Applicable situation

The screening item for genetic diseases of Familial Hypercholesterolemia Type B uses three polymorphic loci on the *APOB* gene. The test results only indicate the user's carrier status of the disease-causing gene or locus for Familial Hypercholesterolemia Type B. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test does not cover all genes or loci that affect Familial Hypercholesterolemia Type B.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

-
1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of the disease is determined by multiple factors such as genes and living environment. This test will only screen you if you carry the genes or loci that cause Familial Hypercholesterolemia Type B.
 3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Familial Hypercholesterolemia Type B.
 4. If you have a family history of Familial Hypercholesterolemia Type B, or you think that you are likely to suffer from Familial Hypercholesterolemia Type B, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The text includes 3 polymorphic loci associated with Familial Hypercholesterolemia Type B: rs12713559, rs144467873 and rs112029328.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect Familial Hypercholesterolemia Type B.

Glucose-6-Phosphate Dehydrogenase Deficiency (G6PD)

Commonly known as fava bean disease. When patients eat fava beans, take antioxidant drugs, contact infections, etc., hemoglobin is denatured through oxidation and red blood cells are destroyed, thus causing hemolysis.

My Glucose-6-Phosphate Dehydrogenase Deficiency (G6PD)

Non-carrier

Influencing factors



Controllable factors

1. Foods: Eating broad beans or broad bean products may lead to the onset of disease.
2. Drugs: Drugs, including antimalarials, sulfonamide antibiotics, antipyretics and analgesics, etc, trigger glucose-6-phosphate dehydrogenase deficiency symptom.
3. Chemicals: Contact with camphor, menthol, pigments, etc, may trigger the disease.



Uncontrollable factors

1. Gender: It is an X-linked genetic disease. The prevalence in males is higher, and therefore, there are more male patients than female patients.
2. Region: The disease often occurs in malaria endemic areas, such as Mediterranean coast, Southeast Asia, and Africa. The incidence of broad bean disease in China is also much higher in the south than in the north (the south is more prone to malaria).

Knowledge



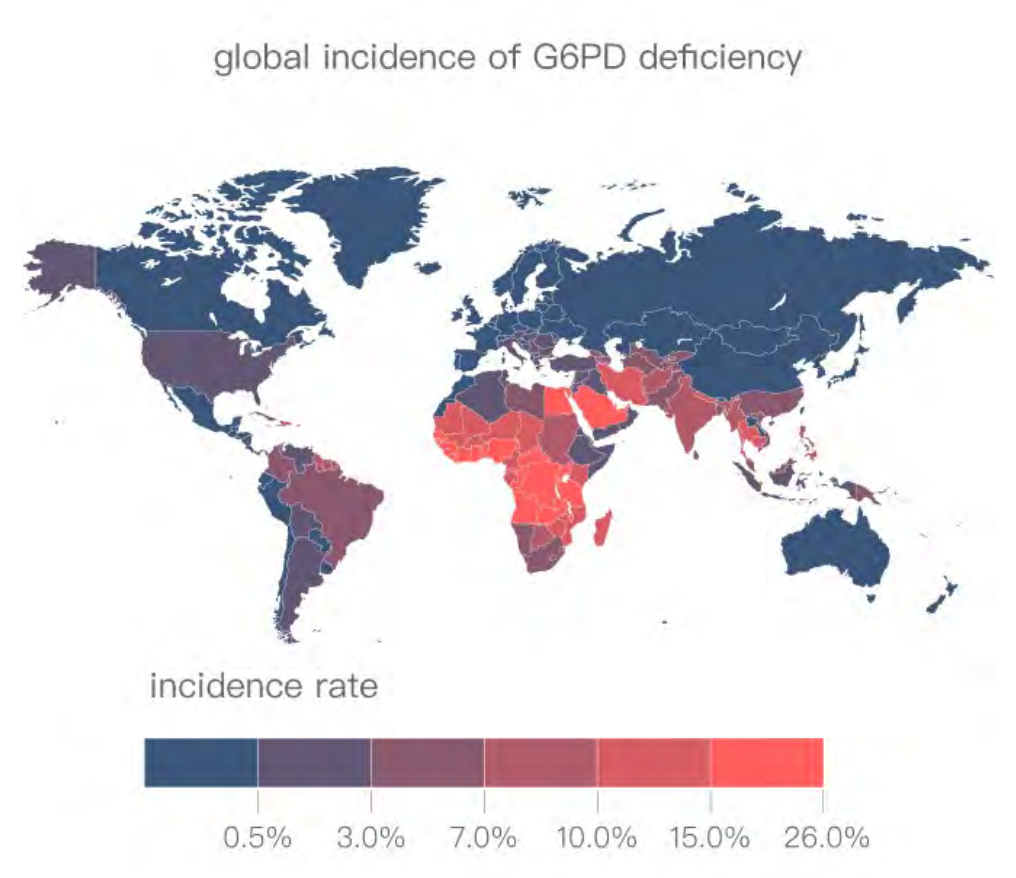
Time of onset

The onset occurs within 24 to 48 hours after eating broad beans or broad bean products (breastfeeding after the mother eats broad beans can also cause the onset of baby).



Prevalence

The disease often occurs in malaria endemic areas, such as Mediterranean coast, Southeast Asia, and Africa. In China, the incidence rate is 「higher in the south and lower in the north」 , especially in Guangdong, Guangxi, Yunnan and other areas of high incidence, incidence of 4%-15%.



 Typical symptoms

Patients have acute hemolytic reactions, such as jaundice, exhaustion, anemia, etc. Acute renal failure may occur in severe cases of hemolysis.

 Treatment

Patients with mild to moderate hemolysis are generally treated with fluid replacement. Blood transfusion is the most effective treatment, when acute hemolysis attacks, to treat acidosis and renal failure.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1050828	G6PD	CC	normal

RS72554664	G6PD	CC	normal
RS137852343	G6PD	AA	normal
RS137852344	G6PD	GG	normal
RS137852345	G6PD	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The Genetic Diseases Screening Program for Glucose-6-phosphate Dehydrogenase Deficiency item uses 49 polymorphic loci on the *G6PD* gene. The test results only indicate whether the user is carrying the disease-causing gene or locus. The basis of the test is mainly from the research of Caucasian and African populations, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect glucose-6-phosphate dehydrogenase deficiency.
2. The test is not suitable for clinical diagnosis.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens whether you are carrying the disease-causing gene or locus.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have glucose-6-phosphate dehydrogenase deficiency.
4. If you have a family history of glucose-6-phosphate dehydrogenase deficiency, or you think that you are likely to suffer from glucose-6-phosphate dehydrogenase deficiency, it is recommended

that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 49 polymorphic loci associated with glucose-6-phosphate dehydrogenase deficiency, such as rs1050828, rs1050829 and rs398123546.

 Population samples

The basis of the test is mainly from the research of Caucasian and African populations.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect glucose-6-phosphate dehydrogenase deficiency.

Hereditary Fructose Intolerance

Hereditary fructose intolerance is an autosomal recessive hereditary genetic metabolic disease which results from a deficiency of fructose diphosphate aldolase in the fructose metabolic pathway. Disease onset is related to the content of sucrose and fructose in the diet.

My Hereditary Fructose Intolerance

Non-carrier

Influencing factors

Controllable factors

Diet: Milk formula mostly contains sucrose, which when consumed by a newborn, can lead to disease.

Knowledge

Time of onset

The age of onset is related to the time of intake of fructose or sucrose, for example, a newborn can get sick when they consume complementary foods containing fructose or sucrose.

Prevalence

The incidence of Hereditary Fructose Intolerance is low, with a European incidence of approximately 1:26,100.

Typical symptoms

The patient had no clinical symptoms before consuming fructose or sucrose. When patients eat these foods, jaundice, liver enlargement, vomiting, malaise, irritability, and convulsions occur quickly. If fructose consumption continues, symptoms recede, liver and kidney failure progresses, and eventually the patient dies.

Treatment

Dietary therapy: avoid fructose and sucrose or any food containing these ingredients.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1800546	ALDOB	CC	normal
RS78340951	ALDOB	GG	normal
RS387906225	ALDOB	--	Not Detected

Test details

Applicable situation

The genetic disorders of Hereditary Fructose Intolerance item uses the three polymorphic loci on the *ALDOB* gene. The test results only indicate the carrying of the disease-causing genes or sites of the user's Hereditary Fructose Intolerance. The test is based mainly on Caucasian studies, and other populations are for informational purposes only.

Notification

1. The test report may not cover all genes or loci that affect Hereditary Fructose Intolerance.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The development of the disease is determined by multiple factors such as genes and environment. This study only screens disease-causing genes or loci of Hereditary Fructose Intolerance.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have the Hereditary Fructose Intolerance.

4. If you have a family history of Hereditary Fructose Intolerance, or you think that you are likely to suffer from Hereditary Fructose Intolerance, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 3 polymorphic loci associated with Hereditary Fructose Intolerance, including rs1800546, rs387906225, rs78340951.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that affect Hereditary Fructose Intolerance.

TTR-Related Cardiac Amyloidosis

Amyloidosis is a group of diseases resulting from deposition of insoluble fibrous amyloid substances in the extracellular space around organs and tissues due to protein folding abnormalities. Cardiac amyloidosis is a limiting cardiomyopathy caused by deposition of amyloid proteins in the myocardium.

My TTR-Related Cardiac Amyloidosis

Non-carrier

Knowledge



Time of onset

Age of onset varies between 17 and 78.



Prevalence

The disease occurs in one per 538 people in North Portugal. While in European American, the incidence is approximately 1/100,000.



Typical symptoms

Including polyneuropathy, renal insufficiency, gastrointestinal hypofunction and cardiomyopathy, etc.



Treatment

Orthotopic liver transplantation: It prevents peripheral or autonomic nerves from exacerbation. Orthotopic liver transplantation requires patients to be younger than 60 and meet the criteria, with disease duration less than 5 years, polyneuropathy limited to the lower limbs or only autonomic neuropathy, and no significant cardiac or renal insufficiency.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS76992529	TTR	GG	normal

Test details

Applicable situation

The Genetic Diseases Screening Item for TTR-related Cardiac Amyloidosis uses 1 polymorphic locus on the *TTR* gene. The test results only indicate whether the user is carrying the disease-causing gene or locus. The basis of the test is mainly from the research of American, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect TTR-related Cardiac Amyloidosis.
2. The test is not suitable for clinical diagnosis.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens whether you are carrying the disease-causing gene or locus.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have TTR-related Cardiac Amyloidosis.
4. If you have a family history of TTR-related Cardiac Amyloidosis, or you think that you are likely to suffer from TTR-related Cardiac Amyloidosis, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, rs76992529, associated with the disease.

Population samples

The basis of the test is mainly from the research of American.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect TTR-related Cardiac Amyloidosis.

Torsion Dystonia

Dystonia is a movement disorder syndrome characterized by abnormal muscle movements and postures caused by uncoordinated or excessive agonist and antagonist muscle contraction.

My Torsion Dystonia
Non-carrier

Influencing factors



Uncontrollable factors

1. Family history: Torsion dystonia has a certain genetic predisposition.
2. Nervous system diseases: Wilson's disease, Huntington's disease, gangliosidosis, etc., can lead to secondary Torsion Dystonia.

Knowledge



Time of onset

It is more common in children or adolescents between 7 and 15 years of age.



Prevalence

The incidence of this disease is about 39/100,000.



Typical symptoms

It is characterized by involuntary muscle contraction, which often leads to twisting, repetitive movements or abnormal postures.



Treatment

Medication and surgical treatment.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS80358233	TOR1A	II	normal

Test details

Applicable situation

The screening program for Torsion Dystonia uses the a polymorphic locus on the *TOR1A* gene. The test results only indicate the user's carrier status of the disease-causing gene or locus for Torsion Dystonia. The basis of the test is mainly from the research of Jewish, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that affect Torsion Dystonia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by a combination of genetic and environmental factors, and this test will only screen you if you carry the genes or loci that cause Torsion Dystonia.
3. It is important to note that carrying the associated mutation does not represent your true state of illness. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Torsion Dystonia.
4. If you have a family history of Torsion Dystonia, or you think that you are likely to suffer from Torsion Dystonia, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus related to Torsion Dystonia including rs80358233.

 Population samples

The basis of the test is mainly from the research of Jewish.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or sites of Torsion Dystonia.

Zellweger Syndrome Spectrum

Cerebrohepatorenal syndrome, also known as Zellweger syndrome, is an autosomal recessive hereditary peroxisomal disease. Clinical manifestations are characterized by multiple congenital malformations, growth retardation or agenesis, abnormal hepatorenal function, and progressive hypotonia.

My Zellweger Syndrome Spectrum

Non-carrier

Knowledge

Time of onset

The child patient tends to be in a critical condition and usually dies before the age of one year.

Prevalence

The incidence is 1:500000 and varies in race.

Typical symptoms

It is characterized by multiple congenital malformation, failure to thrive, hepatorenal dysfunction and progressive dystonia. It mainly affects the nervous system, liver and kidney, without abnormal karyotype. Both men and women can develop the disease.

Treatment

Symptomatic treatment is the general treatment. Nasogastric gavage nourishes patients with dysphagia or sucking difficulties. Anticonvulsant therapy is for those with convulsions.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS61750420	PEX1	CC	normal

Test details

Applicable situation

The Genetic Diseases Screening Program for Zellweger syndrome item uses 1 polymorphic locus on the *PEX1* gene. The test results only indicate whether the user is carrying the disease-causing gene or locus. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test does not cover all genes or loci that affect Zellweger syndrome.
2. The test is not suitable for clinical diagnosis.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens whether you are carrying the disease-causing gene or locus.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Zellweger syndrome.
4. If you have a family history of Zellweger syndrome, or you think that you are likely to suffer from Zellweger syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, rs61750420, associated with Zellweger syndrome.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test does not cover all genes or loci that affect Zellweger syndrome.

Glutaric Aciduria Type 1 (GA1)

It is a genetic disease characterized by insufficient levels of enzymes that decompose lysine, hydroxylysine and tryptophan, resulting in the body's inability to process specific proteins.

My Glutaric Aciduria Type 1 (GA1)

Non-carrier

Knowledge

Onset time

Most cases occur in infancy or early childhood.

Prevalence

The prevalence of the disease is about 1/30,000-1/40,000. This is more common in the Amish community and in the Ojibwa population of Canada, where one in 300 newborns may be affected.

Typical symptoms

Abnormally large head, and it may appear spasms, convulsions, stiffness or reduced muscle tension, cerebral hemorrhage, intraocular, intellectual disability and other symptoms.

Treatment

Regular intravenous infusion of carnitine can lead to significant clinical improvement, and the addition of choline supplements can increase carnitine intake and exposure. Dietary control may help limit the progression of nerve damage. For young children, vegetarian diet and breastfeeding can limit protein intake to avoid the risk of tryptophan being transferred to the brain. Treatment can also be accompanied by sufficient doses of ascorbic acid to stimulate collagen synthesis to prevent lysine from breaking down.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121434367	GCDH	CC	normal
RS121434369	GCDH	CC	normal
RS121434370	GCDH	GG	normal
RS121434371	GCDH	--	Not Detected

Test details

Applicable situation

The glutaric acidemia type I screening item uses four polymorphic loci on the *GCDH* gene. The test results only indicate the carrying of the user's disease-causing genes or sites of glutaric acidemia type I. The test is mainly based on the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all pathogenic genes or sites of glutaric acidemia type I.
2. The test is not suitable for clinical diagnosis.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by a combination of genetic and environmental factors, and this test only screens you for carrying the pathogenic gene or locus of glutaric acidemia type I.
3. It should be noted that carrying the relevant variant does not mean your true disease state, if you do not carry the relevant variant, it cannot be ruled out that you may have glutaric acidemia type I.
4. If you have a family history of glutaric acidemia type I, or you think that you are likely to suffer from glutaric acidemia type I, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with glutaric acidemia type I, such as rs121434371, rs121434370, rs121434369.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect glutaric acidemia type I.

3-Methylcrotonyl CoA Carboxylase 2 Deficiency

It is a rare genetic metabolic disease and the most common organic aciduria in neonatal screening.

My 3-Methylcrotonyl CoA Carboxylase 2 Deficiency

Non-carrier

Knowledge

Time of onset

It usually occurs at birth and in infants and young children.

Prevalence

The incidence of newborns worldwide is about 1/36000.

Typical Symptoms

Symptoms include: feeding difficulties, growth retardation, paroxysmal vomiting, lethargy, coma, diarrhea, "tomcat urine" smell, intractable skin damage, central nervous system abnormalities such as psychomotor developmental delay, cerebral edema, convulsions, Hyperreflexia, hypermyotonia and atonia, etc.

Treatment

Mainly take a protein-restricted diet, a high-sugar diet, and supplement L-carnitine. In acute acidosis and severe hypoglycemia, acidosis should be corrected, water and electrolyte balance should be maintained, and glucose should be infused intravenously.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS119103226	MCCC2	GG	normal

Test details

Applicable situation

The 3-methylcrotonyl CoA carboxylase 2 deficiency genetic disease screening project uses a polymorphic locus on the *MCCC2* gene. The test results only indicate the carrying status of the user's 3-methylcrotonyl-CoA carboxylase 2 deficiency disease-causing gene or locus. The basis of this test is mainly from Asian research, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that cause 3-methylcrotonyl-CoA carboxylase 2 deficiency.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This test only screens you if you carry the disease-causing gene or locus of 3-methylcrotonyl-CoA carboxylase 2 deficiency.
3. It should be noted that carrying the relevant mutation does not represent your true disease state; if you do not carry the relevant mutation, it cannot rule out the possibility that you have a certain disease.
4. If your family has a family history of 3-methylcrotonyl-CoA carboxylase 2 deficiency, or you feel that you are likely to suffer from 3-methylcrotonyl-CoA carboxylase 2 deficiency, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test included rs119103226 one polymorphic locus related to 3-methylcrotonyl-CoA carboxylase 2 deficiency.

Population samples

The basis of this test is mainly from the research of the people in Asian.

Limit of detection

Limited by the existing technological means and the level of scientific cognition, this test may not cover all the disease-causing genes or loci of 3-methylcrotonyl-CoA carboxylase 2 deficiency.

Argininosuccinate Lyase Deficiency

It is an autosomal recessive disease with hyperammonemia as the main manifestation, caused by mutations in the argininosuccinate lyase gene.

My Argininosuccinate Lyase Deficiency

Non-carrier

Knowledge

Time of onset

It usually occurs in the neonatal period.

Prevalence

About 1 in 70,000 newborn babies will develop Argininosuccinate lyase deficiency.

Typical Symptoms

Drowsiness, abnormal activities such as epilepsy, and even coma. More serious dysplasia, mental retardation, accompanied by liver, skin, and hair damage.

Treatment

Stop oral protein intake, supplement oral intravenous lipids and/or glucose, and use intravenous arginine and nitrogen scavenging therapy to quickly control hyperammonemia. If the ammonia level is abnormal, hemodialysis is required.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS28940286	ASL	CC	normal
RS367543005	ASL	CC	normal

RS367543006

ASL

CC

normal

Test details

Applicable situation

The arginyl succinic aciduria genetic disease screening project uses 3 polymorphic loci on the *ASL* gene. The test results only show the user's arginyl succinic aciduria disease-causing gene or locus carried. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

1. The test may not cover all genes or loci that cause Argininosuccinate lyase deficiency.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This test only screens you for carrying the pathogenic gene or locus of argininosuccinate lyase deficiency.
3. It should be noted that carrying the relevant mutation does not represent your true disease state; if you do not carry the relevant mutation, it cannot rule out the possibility that you have a certain disease.
4. If your family has a family history of argininosuccinate lyase deficiency, or you feel that you are likely to suffer from argininosuccinate lyase deficiency, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 3 polymorphic loci related to argininosuccinate lyase deficiency, such as rs367543006, rs367543005, rs28940286.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci of argininosuccinate lyase deficiency.

Alport Syndrome

Alport syndrome, also known as hereditary progressive nephritis or eye-ear-renal syndrome, is a rare genetic disease characterized by damage to specific basement membranes in the kidneys, ears, and eyes.

My Alport Syndrome
Non-carrier

Knowledge



Time of onset

Alport syndrome usually occurs after the age of 10, and children with X-linked dominant or autosomal recessive Alport syndrome can develop renal symptoms at an early stage.



Prevalence

According to statistics, there are 20 cases of the disease per 100,000 people in the population.



Typical symptoms

The main clinical manifestations of Alport syndrome are hematuria and progressive renal dysfunction, often accompanied by varying degrees of progressive sensorineural deafness and ocular changes (such as anterior cone lens). Alport syndrome is one of the important causes of renal failure in adolescents.



Treatment

There is currently no cure for the condition, so it is of great significance for Alport syndrome patients to apply drugs to control proteinuria and slow down the progression of Alport syndrome to renal failure. The ACE inhibitor ramipril is recommended as first-line therapy, and AT1 receptor antagonists are recommended as second-line therapy when patients experience ramipril-related adverse reactions such as dry cough. In children with hypertension and renal disease, ACE inhibitors delay proteinuria and maintain glomerular filtration rate while antihypertensive.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104886043	COL4A5	GG	normal
RS121912825	COL4A3	CC	normal
RS794727397	COL4A5	GG	normal
RS201697532	COL4A3	CC	normal
RS190598500	intergenic	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The Alport syndrome item uses 20 mutations in *COL4A5* gene and *COL4A3* gene. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. All data are interpreted based on our current understanding of disease and pathogenic genes.

Notification

1. The test may not cover all genes or loci affecting Alport syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors. This test only screens you for carrying the pathogenic gene or locus of Alport syndrome.
3. It should be noted that carrying the relevant variation does not represent your true disease status; The absence of the relevant variation does not rule out the possibility that you have a disease.
4. If there is a family history of Alport syndrome in your family, or if you think you are at risk of developing Alport syndrome, you are recommended to consult an authoritative health

professional or clinician for help.

 Test content

Twenty disease-related polymorphic loci including rs200672668 and rs190598500 were detected.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all the disease-causing genes or loci that affect Alport syndrome.

Pendred Syndrome

Pendred syndrome, also known as familial goiter and congenital deafness syndrome, is a common autosomal recessive hereditary hearing impairment syndrome.

My Pendred Syndrome

Non-carrier

Knowledge

Time of onset

The hearing loss of patients mostly occurs before learning language. Hearing impairment in some patients occurs in childhood and then worsens gradually.

Prevalence

According to statistics, there are 7.5 to 10 cases of the disease per 100,000 people in the population.

Typical symptoms

The main clinical symptom of Pendred syndrome is sensorineural deafness, which is often accompanied by inner ear malformations, with the enlargement of vestibular aqueduct being the most common deformity. Goiter is also an important clinical manifestation of Pendred syndrome. Typical goiter often occurs after deafness, usually in childhood, and is more obvious at the age of 20-30.

Treatment

Neural deafness can be treated with cochlear replacement or with built-in hearing aids to improve hearing. The goiter should not be treated with surgery. Thyroxine tablets or Euthyrox should be taken. Surgery should be considered only when the symptoms of goiter compression are obvious or the response to hormone therapy is not obvious.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS111033199	SLC26A4	GG	normal
RS121909340	FOXI1	GG	normal
RS786204426	SLC26A4	GG	normal
RS199588131	SLC26A4	CC	normal
RS541538556	intergenic	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The Pendred syndrome item uses 22 mutations in three genes including *SLC26A4* and *FOXI1* genes. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. All data are interpreted based on our current understanding of disease and pathogenic genes.

Notification

1. The test may not cover all genes or loci affecting Pendred syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors. This test only screens you for carrying the pathogenic gene or locus of Pendred syndrome.
3. It should be noted that carrying the relevant variation does not represent your true disease status; The absence of the relevant variation does not rule out the possibility that you have a disease.
4. If there is a family history of Pendred syndrome in your family, or if you think you are at risk of developing Pendred syndrome, you are recommended to consult an authoritative health

professional or clinician for help.

 Test content

Twenty-two disease-related polymorphic loci including rs786204426 and rs397516430 were detected.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all the disease-causing genes or loci that affect Pendred syndrome.

Stickler Syndrome

Stickler syndrome, also known as hereditary progressive osteoarthritis-eye disease, is an autosomal dominant genetic disease involving multiple organs such as bones, joints, mouth, eyes, and ears.

My Stickler Syndrome Non-carrier

Knowledge



Time of onset

Patients with Stickler syndrome mostly developed congenital high myopia before age 6 and degenerative joint disease of varying degrees after age 30.



Prevalence

According to statistics, there are 10 cases of the disease per 100,000 people in the population.



Typical symptoms

The clinical symptoms of Stickler syndrome include eye diseases (high myopia and retinal detachment), hearing impairment (hearing loss and frequent intraear infections), and craniofacial abnormalities (small nose, small jaw, and cleft palate). and skeletal muscle abnormalities (arthritis and joint relaxation).



Treatment

When patients with Stickler syndrome have ocular lesions, correct diagnosis and timely treatment play a very important role in restoring and maintaining patients' useful vision. For patients with retinal hiatus, laser photocoagulation or external scleral condensation can be used to seal retinal hiatus and reduce the risk of retinal detachment. If retinal detachment has occurred, additional surgery is required. Obvious cataract requires lens extraction.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121912866	COL2A1	GG	normal
RS121912882	COL2A1	GG	normal
RS121912884	COL2A1	GG	normal
RS121912885	COL2A1	GG	normal
RS121912893	COL2A1	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The Stickler syndrome item uses 13 mutations in *COL2A1* gene. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. All data are interpreted based on our current understanding of disease and pathogenic genes.

Notification

1. The test may not cover all genes or loci affecting Stickler syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors. This test only screens you for carrying the pathogenic gene or locus of Stickler syndrome.
3. It should be noted that carrying the relevant variation does not represent your true disease status; The absence of the relevant variation does not rule out the possibility that you have a disease.
4. If there is a family history of Stickler syndrome in your family, or if you think you are at risk of developing Stickler syndrome, you are recommended to consult an authoritative health

professional or clinician for help.

 Test content

Thirteen disease-related polymorphic loci including rs794727748 and rs121912882 were detected.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all the disease-causing genes or loci that affect Stickler syndrome.

Usher Syndrome

Usher syndrome, also known as retinitis pigmentosa-sensorineural deafness syndrome, is the most common type of autosomal recessive hereditary hearing loss syndrome. About half of the people with congenital deafness and blindness in the United States suffer from Usher syndrome.

My Usher Syndrome
Non-carrier

Knowledge



Time of onset

Most patients are born with hearing loss, while visual impairment caused by retinitis pigmentosa may not appear until the age of 20.



Prevalence

According to statistics, there are 3 to 5 cases of the disease per 100,000 people in the population.



Typical symptoms

Usher syndrome type I is a condition characterized by congenital severe-to-profound deafness, vestibular dysfunction, sitting and walking later than normal children, and retinopathy occurring before the age of 10. Usher syndrome type II is a condition characterized by congenital moderate-to-severe deafness, normal vestibular function. Retinopathy occurs before the age of 10 or 20. Usher syndrome type III is a condition characterized by progressive hearing loss and vestibular dysfunction. Relatively rare, about 5-15% of the population.



Treatment

There is currently no effective treatment for the disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS111033174	MYO7A	CC	normal
RS80338904	USH2A	TT	normal
RS398124619	USH2A	CC	normal
RS41298133	MYO7A	CC	normal
RS41308425	USH2A	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The Usher syndrome item uses 159 mutations in eight genes including *USH2A* and *MYO7A* genes, etc. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. All data are interpreted based on our current understanding of disease and pathogenic genes.

Notification

1. The test may not cover all genes or loci affecting Usher syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors. This test only screens you for carrying the pathogenic gene or locus of Usher syndrome.
3. It should be noted that carrying the relevant variation does not represent your true disease status; The absence of the relevant variation does not rule out the possibility that you have a disease.
4. If there is a family history of Usher syndrome in your family, or if you think you are at risk of developing Usher syndrome, you are recommended to consult an authoritative health professional or clinician for help.

Test content

159 disease-related polymorphic loci including rs373780305, rs121909762 and rs397517436 were detected.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all the disease-causing genes or loci that affect Usher syndrome.

X-Linked Non-Syndromic Hereditary Deafness

X-linked non-syndromic hereditary deafness is related to gender. As long as men carry mutations, they may get the disease. Different gene locus or the same gene locus has great phenotypic heterogeneity, and the related pathogenic gene is *POU3F4* gene.

My X-Linked Non-Syndromic Hereditary Deafness

Non-carrier

Knowledge

Time of onset

The onset age of this disease is generally early. From congenital to adolescence, hearing loss is gradually aggravated, and gradually develops into severe to very severe deafness involving full frequency.

Prevalence

It accounts for about 1% of non-syndromic deafness.

Typical symptoms

The clinical phenotypic characteristics of different gene loci of this disease display features of mixed type of deafness (e.g., DFNX2) and special type of neural deafness (e.g., DFNX5). Also, there are features of sensorineural deafness. Some patients have vestibular symptoms, but the description is not detailed at present.

Treatment

Deafness caused by *POU3F4* gene is not suitable for surgical treatment of conductive deafness, because surgery may lead to extralymphatic blowout.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS397516335

POU3F4

TT

normal

Test details

Applicable situation

The X-linked non-syndromic hereditary deafness item uses one mutation in *POU3F4* gene. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. All data are interpreted based on our current understanding of disease and pathogenic genes.

Notification

1. The test may not cover all genes or loci affecting X-linked non-syndromic hereditary deafness.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors. This test only screens you for carrying the pathogenic gene or locus of X-linked non-syndromic hereditary deafness.
3. It should be noted that carrying the relevant variation does not represent your true disease status; The absence of the relevant variation does not rule out the possibility that you have a disease.
4. If there is a family history of X-linked non-syndromic hereditary deafness in your family, or if you think you are at risk of developing X-linked non-syndromic hereditary deafness, you are recommended to consult an authoritative health professional or clinician for help.

Test content

One disease-related polymorphic locus rs397516335 was detected.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all the disease-causing genes or loci that affect X-linked non-syndromic hereditary deafness.

Large Vestibular Aqueduct Syndrome

Patients with large vestibular aqueduct syndrome have enlarged vestibular aqueducts and sensorineural hearing loss without other malformations. Large vestibular aqueduct syndrome belongs to non-syndromic deafness. Patients are sensitive to changes in intracranial pressure. Colds or traumatic brain injuries may cause changes in intracranial pressure, which can lead to sensorineural hearing loss.

My Large Vestibular Aqueduct Syndrome

Non-carrier

Knowledge

Time of onset

The onset of this condition occurs from birth to adolescence. The onset is sudden or insidious, usually preceded by a cold, mild cranial trauma, or other history of increased intracranial pressure.

Prevalence

The large vestibular aqueduct syndrome account for 1%-12% of the incidence of sensorineural deafness.

Typical symptoms

Undulating sensorineural hearing loss, mostly progressive decline.

Treatment

Patients with sudden hearing loss can adopt the treatment protocol of sudden deafness, that is, improving the inner ear circulation, taking glucocorticoid and neurotrophic drugs. After timely treatment, the hearing can be restored to the level before the decline; Hearing AIDS and even cochlear implants may be needed for patients with prolonged hearing loss.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS397516414	SLC26A4	GG	normal
RS397516416	SLC26A4	CC	normal
RS147952620	SLC26A4	CC	normal
RS786204739	SLC26A4	TT	normal
RS200712253	SLC26A4	TT	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The large vestibular aqueduct syndrome item uses six mutations in *SLC26A4* gene. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. All data are interpreted based on our current understanding of disease and pathogenic genes.

Notification

1. The test may not cover all genes or loci affecting large vestibular aqueduct syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors. This test will only screen you for carrying the pathogenic gene or locus of large vestibular aqueduct syndrome.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have large vestibular aqueduct syndrome.

4. If you have a family history of large vestibular aqueduct syndrome, or you think that you are likely to suffer from large vestibular aqueduct syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 6 disease-related polymorphic loci such as rs786204739, rs200712253, ect.

 Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect large vestibular aqueduct syndrome.

Autosomal Dominant Non-Syndromic Deafness

Autosomal dominant non-syndromic deafness is mostly post-lingual sensorineural deafness. The general course of the disease is slow, and the patients are more likely to retain their language skills. The inheritance is vertical in the family, and there are diseased individuals in each generation.

My Autosomal Dominant Non-Syndromic Deafness

Non-carrier

Knowledge

Time of onset

The age of onset of autosomal dominant non-syndromic deafness can range from child to one's fifties.

Prevalence

Autosomal dominant non-syndromic deafness accounts for 15 to 20 percent of non-syndromic deafness.

Typical symptoms

In most cases, hearing loss begins at high tones, and a few are low or middle-frequency sensorineural hearing loss. Progressive aggravation involves multiple frequencies, and most of them are not accompanied by vertigo.

Treatment

Wear hearing aids or cochlear implants.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894413	GJB2	CC	normal

RS74315318	GJB3	GG	normal
RS146670741	WFS1	GG	normal
RS786201027	TMC1	TT	normal
RS727503467	TECTA	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The autosomal dominant non-syndromic deafness item uses the 22 mutations in 7 genes such as *COL11A2* and *WFS1*. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. The interpretation of all data is based on our current understanding of the disease and the disease-causing gene(s).

Notification

1. The test may not cover all genes or loci that affect autosomal dominant non-syndromic deafness.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors, and this test only screens the genes or loci that cause autosomal dominant non-syndromic deafness.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have autosomal dominant non-syndromic deafness.
4. If your family has a history of autosomal dominant non-syndromic deafness, or you feel that you are at risk of suffering from autosomal dominant non-syndromic deafness, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 24 polymorphic loci related to autosomal dominant non-syndromic deafness, such as rs146670741 and rs397517196.

Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect autosomal dominant non-syndromic deafness.

Autosomal Recessive Non-Syndromic Deafness

Autosomal recessive non-syndromic deafness is the most common non-syndromic deafness, mostly congenital prelingual sensorineural hearing loss. The most common pathogenic genes in the Chinese population are GJB2 and SLC26A4.

My Autosomal Recessive Non-Syndromic Deafness

Non-carrier

Knowledge

 Time of onset

Autosomal recessive non-syndromic deafness is mostly congenital prelingual sensorineural hearing loss.

 Prevalence

Autosomal recessive non-syndromic deafness accounts for 80 percent of non-syndromic deafness.

 Typical symptoms

In most cases, full-tones hearing loss is manifested, and the degree of deafness is mostly severe or total deafness.

 Treatment

An artificial cochlear implant may be required.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894395	GJB2	CC	normal
RS763721044	CDH23	GG	normal

RS727505104	LOXHD1	GG	normal
RS373937326	LOXHD1	CC	normal
RS781534323	GJB2	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The autosomal recessive non-syndromic deafness item uses the 82 polymorphic loci in 22 genes such as *GJB2* and *SLC26A4*. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. The interpretation of all data is based on our current understanding of the disease and the disease-causing gene(s).

Notification

1. The test may not cover all genes or loci that affect autosomal recessive non-syndromic deafness.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors, and this test only screens the genes or loci that cause autosomal recessive non-syndromic deafness.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have autosomal recessive non-syndromic deafness.
4. If your family has a history of autosomal recessive non-syndromic deafness, or you feel that you are at risk of suffering from autosomal recessive non-syndromic deafness, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 82 polymorphic loci related to autosomal recessive non-syndromic deafness, such as rs763721044 and rs606231410.



Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect autosomal recessive non-syndromic deafness.

Aminoglycoside-Induced Deafness

People who carry the corresponding genetic mutations are very sensitive to aminoglycoside antibiotics. Even if the dose is much lower than the standard, it will still cause serious and irreversible damage to hearing, namely 'hearing loss for even one injection'. This type of deafness has the characteristics of family aggregation and maternal inheritance.

My Aminoglycoside-Induced Deafness

Non-carrier

Knowledge

Time of onset

Hearing loss usually occurs about 3 days to 3 months after using aminoglycoside drugs.

Typical symptoms

The clinical manifestations are usually bilateral, severe or extremely severe and irreversible high-frequency hearing loss.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267606617	MT-RNR1	AA	normal risk of deafness due to aminoglycoside antibiotics
RS28358569	MT-RNR1	AA	normal
RS267606619	intergenic	--	Not Detected
RS267606618	MT-RNR1	--	Not Detected

Test details

Applicable situation

The aminoglycoside antibiotics ototoxic deafness ("one shot causing deafness") item uses the 1 mutation in *RNR1* gene. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. The interpretation of all data is based on our current understanding of the disease and the disease-causing gene(s).

Notification

1. The test may not cover all genes or loci that affect aminoglycoside antibiotics ototoxic deafness ("one shot causing deafness").
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors, and this test only screens the genes or loci that cause aminoglycoside antibiotics ototoxic deafness ("one shot causing deafness").
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have aminoglycoside antibiotics ototoxic deafness ("one shot causing deafness").
4. If your family has a history of aminoglycoside antibiotics ototoxic deafness, or you feel that you are at risk of suffering from aminoglycoside antibiotics ototoxic deafness, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus related to aminoglycoside antibiotics ototoxic deafness, rs28358569.

Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect aminoglycoside antibiotics ototoxic deafness ("one shot causing deafness").

Mitochondrial Hereditary Non-Syndromic Deafness

Mitochondrial hereditary non-syndromic deafness is caused by abnormal mitochondrial function. Since mitochondria can only be passed on from the mother to the next generation, it is manifested as maternal inheritance. If the mother is sick, the child is also sick.

My Mitochondrial Hereditary Non-Syndromic Deafness

Non-carrier

Knowledge

Prevalence

Mitochondrial hereditary non-syndromic deafness accounts for 1 percent of non-syndromic deafness.

Typical symptoms

The clinical phenotypes of deafness caused by mitochondrial mutations vary greatly, and hearing loss may range from mild to severe.

Treatment

Not clear yet.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS199474667	MT-TL1	GG	normal
RS199474820	MT-TS1	TT	normal
RS199474821	MT-TS1	TT	normal

RS267606620	MT-RNR1	TT	normal
RS28358569	MT-RNR1	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The mitochondrial hereditary non-syndromic deafness item uses the 7 mutations in 3 genes such as *RNR1*, *tRNA-Ser1*. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. The interpretation of all data is based on our current understanding of the disease and the disease-causing gene(s).

Notification

1. The test may not cover all genes or loci that affect mitochondrial hereditary non-syndromic deafness.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors, and this test only screens the genes or loci that cause mitochondrial hereditary non-syndromic deafness.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have mitochondrial hereditary non-syndromic deafness.
4. If your family has a history of mitochondrial hereditary non-syndromic deafness, or you feel that you are at risk of suffering from mitochondrial hereditary non-syndromic deafness, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 7 polymorphic loci related to mitochondrial hereditary non-syndromic deafness, such as rs199474818, rs28358569.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect mitochondrial hereditary non-syndromic deafness.

Peroxisome Biosynthesis Disorder

Peroxisome biosynthesis disorder is an autosomal recessive inherited disease. Due to defects in peroxidase synthesis, it leads to a series of spectrum diseases: from the mildest infantile Refsum disease to the most serious Zellweger syndrome.

My Peroxisome Biosynthesis Disorder

Non-carrier

Knowledge

Time of onset

Deafness can occur at birth.

Prevalence

According to statistics, there are about 2.5 cases of the disease per 100,000 people in the average population.

Typical symptoms

In a series of syndromes caused by peroxisome disorders, the degree of hearing loss is different. In addition to severe damage to the auditory system, most patients with the most severe Zellweger often show mental retardation and seizures.

Treatment

At present, there is no cure method, and the current treatments for this disease are symptomatic and supportive treatment.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121434455	PEX1	AA	normal

RS61750415	PEX1	DD	normal
RS61750420	PEX1	CC	normal
RS794729652	PEX1	DD	normal
RS61750427	PEX1	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The peroxisome biosynthesis disorder item uses the 8 mutations in *PEX1* gene and *PEX6* gene. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. The interpretation of all data is based on our current understanding of the disease and the disease-causing gene(s).

Notification

1. The test may not cover all genes or loci that affect peroxisome biosynthesis disorder.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors, and this test only screens the genes or loci that cause peroxisome biosynthesis disorder.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have peroxisome biosynthesis disorder.
4. If your family has a history of peroxisome biosynthesis disorder, or you feel that you are at risk of suffering from peroxisome biosynthesis disorder, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 8 polymorphic loci related to peroxisome biosynthesis disorder, such as rs794729652, rs61750427.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect peroxisome biosynthesis disorder.

Branchiootorenal Spectrum Disorder

Branchiootorenal spectrum disorder, also known as Branchio-oto-renal syndrome, is a rare autosomal dominant syndrome type deaf disease. Clinically, it is divided into branchiootorenal (BOR) syndrome and branchiootic (BOS) syndrome according to whether the kidney abnormality is included.

My Branchiootorenal Spectrum Disorder

Non-carrier

Knowledge

Time of onset

Deafness can occur at birth.

Prevalence

According to statistics, there are about 2.5 cases of the disease per 100,000 people in the average population.

Typical symptoms

The clinical manifestations are mainly branchial cleft dysplasia (branchial cleft fistula or branchial cleft cyst), deafness, preauricular fistula, and kidney malformations. Ear abnormalities include abnormal ear development and hearing impairment, and hearing impairment occurs in 78%-93% of patients. Clinically, it is divided into branchio-oto-renal syndrome and branchiootic syndrome according to whether it is accompanied by urinary system malformations. The clinical manifestations of patients with branchiootorenal spectrum disorders are extremely variable, and there are great differences in the clinical manifestations of different patients in the same family, and even the same patient on the left and right sides.

Treatment

Otology symptoms and branchial arch anomalies can be corrected by surgery. Patients with asymptomatic kidney abnormalities or mild symptoms generally do not need to be treated; severe renal malformations lead to renal failure requiring dialysis or kidney transplantation. Patients with external auditory canal atresia should consider external auditory canal plasty. In addition, renal malformations such as vesicoureteral reflux can lead to progressive renal failure, and surgical

correction can effectively avoid the occurrence of secondary complications. At the same time, nephrotoxic drugs should be used with caution.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS397517917	intergenic	CC	normal
RS201509408	EYA1	CC	normal
RS727504494	EYA1	CC	normal

Test details

Applicable situation

The branchiootorenal spectrum disorder item uses the 3 mutations in *EYA1* gene. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. The interpretation of all data is based on our current understanding of the disease and the disease-causing gene(s).

Notification

1. The test may not cover all genes or loci that affect branchiootorenal spectrum disorder.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors, and this test only screens the genes or loci that cause branchiootorenal spectrum disorder.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have branchiootorenal spectrum disorder.

4. If your family has a history of branchiootorenal spectrum disorder, or you feel that you are at risk of suffering from branchiootorenal spectrum disorder, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes 3 polymorphic loci related to branchiootorenal spectrum disorder, such as rs201509408, rs727504494.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect branchiootorenal spectrum disorder.

Jervell-Lange-Nielsen Syndrome

Jervell and Lange-Nielsen syndrome is a rare clinical type of Long QT syndrome, which is a rare and severe cardiac and auditory abnormality syndrome.

My Jervell-Lange-Nielsen Syndrome

Non-carrier

Knowledge

Time of onset

Most patients develop cardiac symptoms before the age of 3 years.

Prevalence

Jervell and Lange-Nielsen syndrome is uncommon; it affects an estimated 1.6 to 6 per 1 million people worldwide.

Typical symptoms

The main clinical manifestations are congenital sensorineural hearing loss, as well as symptoms such as long QT interval, syncope and sudden death caused by abnormal T waves and ventricular tachycardia.

Treatment

At present, beta-adrenergic blocking agents are conventional drugs for the treatment of Jervell and Lange-Nielsen syndrome, but the effect is not good. Left heart sympathetic resection and implantable cardioverter defibrillator have been shown to reduce sudden death to a certain extent rate.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS786204778	KCNQ1	II	normal

Test details

Applicable situation

The Jervell-Lange-Nielsen syndrome item uses the 1 mutation in the *KCNQ1* gene. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. The interpretation of all data is based on our current understanding of the disease and the disease-causing gene(s).

Notification

1. The test may not cover all genes or loci that affect Jervell-Lange-Nielsen syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors, and this test only screens the genes or loci that cause Jervell-Lange-Nielsen syndrome.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Jervell-Lange-Nielsen syndrome.
4. If your family has a history of Jervell-Lange-Nielsen syndrome, or you feel that you are at risk of suffering from Jervell-Lange-Nielsen syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus related to Jervell-Lange-Nielsen syndrome, rs786204778.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect Jervell-Lange-Nielsen syndrome.

Mohr-Tranebjaerg Syndrome

Mohr-Tranebjaerg syndrome, also known as deafness dystonia syndrome, is a rare X-linked recessive genetic disease.

My Mohr-Tranebjaerg Syndrome

Non-carrier

Knowledge



Time of onset

The first symptom of DDON syndrome (also known as Mohr-Tranebjaerg syndrome) is hearing loss caused by nerve damage in the inner ear (sensorineural hearing loss), which begins in early childhood. The hearing impairment worsens over time, and most affected individuals have profound hearing loss by age 10. Progressive neuropathy that affects the brain and nerves occurs in adulthood.



Prevalence

This disease is a rare disorder, and no accurate statistics on the incidence have been released.



Typical symptoms

Symptoms are recessive neurodegenerative syndrome. The first symptom is progressive post-lingual sensorineural hearing loss in childhood, followed by a series of multiple neurological diseases.



Treatment

At present, there is no cure method, and the current treatments for this disease are symptomatic and supportive treatment. For the treatment of deafness, the effect of cochlear implantation is poor.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS80356560	TIMM8A	GG	normal

Test details

Applicable situation

The Mohr-Tranebjaerg syndrome item uses the 1 mutation in *TIMM8A* gene. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. The interpretation of all data is based on our current understanding of the disease and the disease-causing gene(s).

Notification

1. The test may not cover all genes or loci that affect Mohr-Tranebjaerg syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors, and this test only screens the genes or loci that cause Mohr-Tranebjaerg syndrome.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Mohr-Tranebjaerg syndrome.
4. If your family has a history of Mohr-Tranebjaerg syndrome, or you feel that you are at risk of suffering from Mohr-Tranebjaerg syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus related to Mohr-Tranebjaerg syndrome, rs80356560.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect Mohr-Tranebjaerg syndrome.

Early-Onset Alzheimer's Disease

The symptoms of early-onset Alzheimer's disease are similar to those of late-onset Alzheimer's disease in dementia, but the age of onset is often less than 65 years old.

My Early-Onset Alzheimer's Disease

Non-carrier

Knowledge



Time of onset

Age of onset is before 65 years (or 60 years).



Prevalence

Early-onset Alzheimer's Disease affects about 6 in 100,000 people.



Typical symptoms

1. Early stage: The symptoms of patients are mostly hidden and easy to be ignored, often with near memory loss as the main manifestation. For example, forget the things you just did. They also have time disorientation and can't even find their way home.
2. Advanced stage: Memory impairment and disorientation gradually worsened. Obvious symptoms of dementia include repeated asking the same question, poor hygiene, and decreased judgment. It is difficult to recall the names of family and friends, relatives and colleagues, and facial recognition is difficult, and self-care skills are beginning to appear problems.
3. Late stage: The patient loses the ability to take care of himself, and his ability to recognize time, place, and people is further reduced. Emotions will also gradually change, depression, negativity or restlessness.



Treatment

There are two main types of treatment for Alzheimer's disease:

- ①Control psychopathological symptoms. Such as taking anti-anxiety, anti-depressant, and anti-psychotic drugs to improve symptoms.
- ②Improve cognitive function and delay disease progression. Such as taking drugs that improve brain

neurotransmission function or drugs that improve cerebral blood circulation and brain cell metabolism.

Alzheimer's disease does not necessarily require hospitalization, but there are often some acute symptoms that require urgent treatment. Such as severe abnormal mental behavior or complicated infection, stroke, etc., hospitalization is better for the patient. However, no matter whether it is drug therapy, physical therapy, or psychotherapy, it cannot interrupt the progression of Alzheimer's disease. It can only delay the development of the disease, and there is no way to completely cure the disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS28936379	PSEN2	AA	normal
RS63750687	PSEN1	CC	normal
RS63751320	PSEN1	AA	normal
RS63751235	PSEN1	CC	normal
RS63751229	PSEN1	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The early-onset Alzheimer's disease item uses the 27 mutations in *APP* gene, *PSEN1* gene and *PSEN2* gene. The test results only report that among the pathogenic genes within the detection range, the tested person carries at least one mutation of pathogenic genotype. The interpretation of all data is based on our current understanding of the disease and the disease-causing gene(s).

Notification

1. The test may not cover all genes or loci that affect early-onset Alzheimer's Disease.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence of this disease is determined by genetic factors, and this test only screens the genes or loci that cause early-onset Alzheimer's disease.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have early-onset Alzheimer's disease.
4. If your family has a history of early-onset Alzheimer's disease, or you feel that you are at risk of suffering from early-onset Alzheimer's disease, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 27 polymorphic loci related to early-onset Alzheimer's disease, such as rs63749824, rs63749885, rs63750666.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or loci that related to early-onset Alzheimer's Disease.

Brugada Syndrome

Brugada syndrome is an autosomal dominant inherited disorder. It is characterized by an ST segment elevation in the right precordial electrocardiogram leads, disruption of the heart's normal rhythm, risk of malignant arrhythmia and family history of SCD.

My Brugada Syndrome

Non-carrier

Knowledge



Time of onset

The syndrome typically manifests during adulthood, with a mean age of sudden death of 41 +/- 15 years.



Prevalence

The prevalence of Brugada syndrome affects 1 or 5 in 10,000 people worldwide that occurs much more frequently in Japanese and Southeast Asian populations.



Typical symptoms

Brugada syndrome is characterized by ventricular fibrillation (VF) or sudden cardiac death (SCD), fainting, nocturnal difficulty breathing, palpitations, chest discomfort. These complications typically occur when an affected person is resting, asleep, fever or other state of vagus nerve tension, rarely appear during exercise.



Treatment

The implantable cardioverter - defibrillator (ICD) implantation is the only effective treatment for Brugada syndrome patients to prevent sudden cardiac death so far. However, ICD also has many shortcomings, especially for young patients who need multiple operations to replace ICD during their lifetime.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS12720441	KCNH2	GG	normal
RS199473611	SCN5A	CC	normal
RS199473588	SCN5A	AA	normal
RS199473592	SCN5A	CC	normal
RS199473597	SCN5A	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The Brugada syndrome item uses the 177 polymorphic loci in genes such as *SCN5A*, ect. The test results indicate carrying status of the gene or locus of Brugada syndrome. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all genes or loci that affect Brugada syndrome. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are the result of the interaction of multiple factors such as genes and environment, and this test only screens the genes or loci that cause Brugada syndrome.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Brugada syndrome.
4. If your family has a history of Brugada syndrome, or you feel that you are at risk of suffering from Brugada syndrome, it is recommended that you consult an authoritative health professional or

clinician for help.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect Brugada syndrome.

Loeys Dietz Syndrome

Loeys dietz syndrome is an autosomal dominant inherited connective tissue disorder characterized by vascular and skeletal manifestations. Some patients have craniofacial involvement consisting of hypertelorism or cleft palate.

My Loeys Dietz Syndrome

Non-carrier

Knowledge



Time of onset

Infancy, Neonatal.



Typical symptoms

Loeys-Dietz syndrome is characterized by vascular findings (cerebral, thoracic, and abdominal arterial aneurysms and/or dissections), skeletal manifestations (pectus excavatum or pectus carinatum, scoliosis, joint laxity, arachnodactyly, talipes equinovarus, cervical spine malformation and/or instability), craniofacial features (widely spaced eyes, strabismus, bifid uvula / cleft palate, and craniosynostosis that can involve any sutures).



Treatment

1. Use beta-adrenergic blocking agents or other drugs to reduce hemodynamic resistance.
2. Surgical intervention of aneurysms actively in early age.
3. Surgery to control cervical spine instability and prevent spinal injury is required.
4. Standardized therapy to treat foot deformities and severe flatfoot is required.
5. Control and treatment of cleft palate and premature craniofacial closure by the craniofacial medical team.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS104893809	TGFBR2	CC	normal
RS730880215	SMAD3	GG	normal
RS387906856	SMAD3	GG	normal
RS387907278	TGFB2	CC	normal
RS397516840	TGFBR2	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The Loeys dietz syndrome item uses the 48 polymorphic loci in genes such as *TGFBR2*, ect. The test results indicate carrying status of the gene or locus of Loeys dietz syndrome. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all genes or loci that affect Loeys dietz syndrome. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are the result of the interaction of multiple factors such as genes and environment, and this test only screens the genes or loci that cause Loeys dietz syndrome.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Loeys dietz syndrome.
4. If your family has a history of Loeys dietz syndrome, or you feel that you are at risk of suffering from Loeys dietz syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect Loeys dietz syndrome.

Wolff-Parkinson-White Syndrome

Wolff-Parkinson-White syndrome is an autosomal dominant inherited disorder characterized by abnormal electrical pathways in the heart that cause a disruption of the heart's normal rhythm.

My Wolff-Parkinson-White Syndrome

Non-carrier

Knowledge



Time of onset

All ages.



Prevalence

Wolff-Parkinson-White syndrome affects 1 to 3 in 1,000 people worldwide, mostly male.



Typical symptoms

The typical symptom of a patient with an arrhythmia for the first time within 20 years is paroxysmal supraventricular tachycardia suddenly occurs in the exercise. The attack may last from a few seconds to several hours. However, tachycardia can make the patient feel extremely uncomfortable and even coma. A small number of patients will experience fainting. Older people will experience paroxysmal supraventricular tachycardia, which often produces more symptoms, such as syncope, shortness of breath, and chest pain.



Treatment

The current first-line treatment for Wolff-Parkinson-White syndrome is radiofrequency ablation.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121908987	PRKAG2	CC	normal

RS3218716

MYH7

CC

normal

Test details

Applicable situation

The Wolff-Parkinson-White syndrome item uses the 2 polymorphic loci in *PRKAG2* gene and *MYH7* gene. The test results indicate carrying status of the gene or locus of Wolff-Parkinson-White syndrome. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all genes or loci that affect Wolff-Parkinson-White syndrome. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are the result of the interaction of multiple factors such as genes and environment, and this test only screens the genes or loci that cause Wolff-Parkinson-White syndrome.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Wolff-Parkinson-White syndrome.
4. If your family has a history of Wolff-Parkinson-White syndrome, or you feel that you are at risk of suffering from Wolff-Parkinson-White syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect Wolff-Parkinson-White syndrome.

Sick Sinus Syndrome

Sick sinus syndrome, also known as sinus node disease or sinus node dysfunction, is an autosomal dominant or autosomal recessive inherited disorder.

It is caused by impaired autonomy of the sinus node (the ability of cells to generate electrical impulses) or impaired the conduction of electrical impulses generated by the sinus node to the surrounding atrial muscles. Electrocardiogram typically shows sinus bradycardia, sinoatrial block, and/or sinus arrest.

My Sick Sinus Syndrome Non-carrier

Knowledge

Time of onset

Although common in the elderly, sick sinus syndrome can also occur at any age.

Prevalence

It accounts for 1/600 of heart disease patients over 65 years of age. The incidence of this condition increases with age.

Typical symptoms

Sinus arrest without proper escape rhythm may cause syncope or near syncope, depending on the duration of sinus arrest. Tachycardia usually produces palpitations, and subsequent inhibition of sinoatrial node can also lead to syncope or near syncope at the end of palpitation.

Treatment

It is recommended to implant an atrial or dual-chamber pacemaker to control the patient's symptoms. Antiarrhythmic drugs may usually aggravate the sinus node dysfunction. If drugs are needed to control tachycardia, a pacemaker is usually implanted. Sinus bradycardia or sinus arrest can lead to tachyarrhythmia.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894485	HCN4	CC	normal
RS121908411	HCN4	CC	normal
RS137854613	SCN5A	GG	normal
RS28937319	SCN5A	GG	normal
RS869025519	SCN5A	CC	normal

Test details

Applicable situation

The sick sinus syndrome genetic disease screening item uses 5 polymorphic loci on *SCN5A* and *HCN4* genes. The test results only indicate the carrying status of the disease-causing gene or loci of the sick sinus syndrome in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect sick sinus syndrome. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. The test results only screen your carrying status of the disease-causing gene or loci of the sick sinus syndrome.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have sick sinus syndrome.
4. If you have a family history of sick sinus syndrome, or you think that you are likely to suffer from sick sinus syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect sick sinus syndrome.

Short QT Syndrome (SQTS)

Short QT syndrome is a cardiac channelopathy associated with a predisposition to atrial fibrillation and sudden cardiac death. It is characterized by electrocardiography (ECG) exhibiting abbreviated QTc (Bazett's corrected QT) intervals.

My Short QT Syndrome (SQTS)

Non-carrier

Knowledge

Time of onset

The first clinical symptoms are early, and the onset can occur in childhood.

Prevalence

Short QT syndrome appears to be rare. At least 70 cases have been identified worldwide since the condition was discovered in 2000.

Typical symptoms

Mild cases have no clinical symptoms. When ventricular arrhythmia occurs, it will lead to syncope. Sudden cardiac death (SCD) occurs in severe cases. Central atrial fibrillation is common in patients with short QT syndrome.

Treatment

Currently, automatic implantable cardioverter defibrillator can prevent sudden death from short QT syndrome.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894584	KCNJ2	GG	normal

Test details

Applicable situation

The short QT syndrome genetic disease screening item uses one polymorphic locus on *KCNJ2* gene. The test results only indicate the carrying status of the disease-causing gene or loci of short QT syndrome in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect short QT syndrome. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of short QT syndrome.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have short QT syndrome.
4. If you have a family history of short QT syndrome, or you think that you are likely to suffer from short QT syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect short QT syndrome.

Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

Catecholaminergic polymorphic ventricular tachycardia is an autosomal dominant or autosomal recessive inherited disorder with a normal heart structure and sensitivity to catecholamines. It is characterized by bidirectional ventricular tachycardia (bVT), which often manifests as syncope, cardiac arrest, SCD, and it is more common in young people.

My Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

Non-carrier

Knowledge

Time of onset

The age of first onset can range from infancy to about 40 years old.

Prevalence

The prevalence of CPVT is estimated to be about 1 in 10,000 people.

Typical symptoms

Syncope caused by exercise or acute emotions is usually the first symptom observed. Sudden death may be the first manifestation of the disease in some patients (10-20%). The typical arrhythmia of catecholaminergic polymorphic ventricular tachycardia (CPVT) is bidirectional ventricular tachycardia, and supraventricular tachycardia and atrial fibrillation are rare.

Treatment

Beta blockers are the first choice of treatment for patients with this disease, and the maximum tolerated dose should be used to control arrhythmia. Recently, flecainide (a sodium channel blocker) has shown good effects in suppressing arrhythmia in CPVT patients. Implantable cardioverter-defibrillator (ICD) is recommended for CPVT patients with recurrent syncope to prevent cardiac arrest and sudden death.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121434549	CASQ2	CC	normal
RS201211033	RYR2	GG	normal
RS886037908	RYR2	CC	normal
RS886037907	RYR2	CC	normal
RS794728813	RYR2	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The catecholaminergic polymorphic ventricular tachycardia genetic disease screening item uses 31 polymorphic loci on genes such as *RYR2* and *CALM1*. The test results only indicate the carrying status of the disease-causing gene or loci of catecholaminergic polymorphic ventricular tachycardia in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect catecholaminergic polymorphic ventricular tachycardia. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of catecholaminergic polymorphic ventricular tachycardia.

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3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have catecholaminergic polymorphic ventricular tachycardia.
 4. If you have a family history of catecholaminergic polymorphic ventricular tachycardia, or you think that you are likely to suffer from catecholaminergic polymorphic ventricular tachycardia, it is recommended that you consult an authoritative health professional or clinician for help.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect catecholaminergic polymorphic ventricular tachycardia.

Familial Thoracic Aortic Aneurysm

Familial thoracic aortic aneurysm is an autosomal dominant inherited cardiovascular system disorder characterized by the familial occurrence of thoracic aortic aneurysm, dissection or dilatation.

My Familial Thoracic Aortic Aneurysm

Non-carrier

Knowledge

Time of onset

It can begin in childhood or not occur until late in life.

Prevalence

Familial thoracic aortic aneurysm is believed to account for at least 20 percent of thoracic aortic aneurysms and dissections. While aortic aneurysms are common worldwide, it is difficult to determine their exact prevalence because they usually cause no symptoms unless they rupture.

Typical symptoms

1. Dilation of the ascending thoracic aorta in the aortic sinus or ascending aortic segment (or both).
2. Thoracic aortic dissection, involving the ascending or descending aorta.

Treatment

1. Drug treatment: It is recommended to use drugs that reduce the hemodynamic pressure of the aorta, such as β -adrenergic blockers.
2. Surgical treatment: When the expansion rate of the ascending aorta is close to 0.5 cm per year or the diameter reaches 4.2-5.0 cm (depending on the mutation basis or family history), it is recommended to perform the ascending aorta repair at a selected time to prevent life-threatening aortic dissection or rupture.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS112901682	ACTA2	GG	normal
RS397515330	PRKG1	GG	normal
W16015829259	MYH11	II	normal
W03123366197	MYLK	II	normal
RS886039303	ACTA2	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The familial thoracic aortic aneurysm genetic disease screening item uses 17 polymorphic loci on genes such as *LOX* and *ACTA2*. The test results only indicate the carrying status of the disease-causing gene or loci of familial thoracic aortic aneurysm in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect familial thoracic aortic aneurysm. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of familial thoracic aortic aneurysm.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have familial thoracic aortic aneurysm.

4. If you have a family history of familial thoracic aortic aneurysm, or you think that you are likely to suffer from familial thoracic aortic aneurysm, it is recommended that you consult an authoritative health professional or clinician for help.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect familial thoracic aortic aneurysm.

Dilated Cardiomyopathy

Dilated cardiomyopathy is an autosomal dominant or autosomal recessive or X-linked inherited disorder characterized by left or right or both cardiac dilatation and reduced systolic function.

My Dilated Cardiomyopathy

Non-carrier

Knowledge



Time of onset

It can occur at any age, and the incidence of infants is higher.



Prevalence

The incidence is about 1.1-1.2/100,000.



Typical symptoms

The disease is characterized by enlargement and dilation of one or both of the ventricles along with impaired contractility, with or without congestive heart failure. Ventricular or atrial arrhythmia is more common. The disease is progressive, and death can occur at any stage of the disease.



Treatment

1. Keep normal rest, use sedatives if necessary, and eat low salt diet in case of heart failure.
2. Prevention and treatment of arrhythmia and cardiac insufficiency.
3. If there is a large amount of pleural effusion, perform a pleural aspiration.
4. Severe patients may consider artificial ventricular assist devices or heart transplantation, and may undergo cardiac resynchronization therapy (CRT).
5. Symptomatic and supportive treatment.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894655	TCAP	CC	normal
RS759710792	TTN	II	normal
RS727505352	TTN	GG	normal
RS730880132	LMNA	TT	normal
RS730880343	TTN	DD	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The dilated cardiomyopathy genetic disease screening item uses 261 polymorphic loci on or between genes such as *TTN*. The test results only indicate the carrying status of the disease-causing gene or loci of dilated cardiomyopathy in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect dilated cardiomyopathy. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of dilated cardiomyopathy.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have dilated cardiomyopathy.

4. If you have a family history of dilated cardiomyopathy, or you think that you are likely to suffer from dilated cardiomyopathy, it is recommended that you consult an authoritative health professional or clinician for help.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect dilated cardiomyopathy.

Marfan Syndrome

Marfan syndrome is an autosomal dominant inherited connective tissue disorder. It can affect many systems, often causing abnormalities in the heart, blood vessels, eyes, bones, and joints.

My Marfan Syndrome Non-carrier

Knowledge



Time of onset

The features of Marfan syndrome can become apparent anytime.



Prevalence

The incidence of Marfan syndrome is approximately 1 in 5,000, and there is no significant difference between sexes.



Typical symptoms

1. Musculoskeletal system: individuals with Marfan syndrome are usually tall and slender, have elongated fingers and toes (arachnodactyly), have an arm span that exceeds their body height and droops over their knees, and have the lower segment of the body that is greater than the upper segment.
2. Eyes: mainly contain lenticular dislocation or subluxation, high myopia, cataract, detachment of retina, iridodonesis, etc.
3. Cardiovascular system: About 80% of patients have congenital cardiovascular malformations. Including a leak in the valve that connects two of the four chambers of the heart (mitral valve prolapse) or the valve that regulates blood flow from the heart into the aorta (aortic valve regurgitation). Leaks in these valves can cause shortness of breath, fatigue, and an irregular heartbeat felt as skipped or extra beats (palpitations).



Treatment

1. Dilation of the aorta: use drugs (β -blockers), pay attention to reducing exercise, and regularly monitor the aorta.

- 2. Skeletal abnormalities: surgery can be used to stabilize the spine or repair thoracic deformities in the case of scoliosis.
- 3. Eye abnormalities: laser treatment or replacement of dislocated lens.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS111231312	FBN1	GG	normal
RS869025417	FBN1	AA	normal
RS794728280	FBN1	TT	normal
RS794728281	FBN1	CC	normal
RS794728283	FBN1	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The Marfan syndrome genetic disease screening item uses 301 polymorphic loci on genes such as *FBN1*. The test results only indicate the carrying status of the disease-causing gene or loci of Marfan syndrome in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect Marfan syndrome. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

-
1. The test is not suitable for clinical diagnosis.
 2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of Marfan syndrome.
 3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Marfan syndrome.
 4. If you have a family history of Marfan syndrome, or you think that you are likely to suffer from Marfan syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect Marfan syndrome.

Noonan Syndrome

Noonan syndrome is an autosomal dominant inherited disorder that affects many areas of the body. It is characterized by mildly unusual facial features, short stature, heart defects, skeletal malformations, and many other signs and symptoms.

My Noonan Syndrome Non-carrier

Knowledge



Time of onset

Infancy, newborns.



Prevalence

Noonan syndrome occurs in approximately 1 in 1,000 to 2,500 newborns.



Typical symptoms

distinctive facial features, short stature, congenital heart disease and variable degree of developmental delay.



Treatment

1. Cardiovascular abnormalities: The treatment method is the same as that of patients with common cardiovascular abnormalities.
2. Developmental disabilities: Formulate early intervention programs and personalized education strategies.
3. Severe bleeding: Understand the specific factors that cause the defect or consider whether it is caused by abnormal platelet aggregation, and then treat it according to the situation.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS104894360	KRAS	TT	normal
RS397517041	KRAS	CC	normal
RS397517172	SOS1	TT	normal
RS397517164	SOS1	CC	normal
RS397517159	SOS1	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The Noonan syndrome genetic disease screening item uses 113 polymorphic loci on genes such as *NRAS* and *SOS1*. The test results only indicate the carrying status of the disease-causing gene or loci of Noonan syndrome in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect Noonan syndrome. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of Noonan syndrome.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Noonan syndrome.
4. If you have a family history of Noonan syndrome, or you think that you are likely to suffer from Noonan syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect Noonan syndrome.

Restrictive Cardiomyopathy

Restrictive Cardiomyopathy is an autosomal dominant inherited disorder characterized by impaired unilateral or bilateral ventricular filling and reduced diastolic volume in the presence of normal systolic function and normal or near-normal myocardial thickness.

My Restrictive Cardiomyopathy

Non-carrier

Knowledge

Time of onset

It can appear anytime from childhood to adulthood.

Prevalence

In the United States and Europe, restrictive cardiomyopathy accounts for less than 5% of all cardiomyopathies. In China, it is mainly distributed in subtropical regions such as Guangxi Zhuang Autonomous Region, Guangdong Province and Guizhou Province, and scattered cases have also been reported in temperate regions.

Typical symptoms

It can be divided into left ventricular type, right ventricular type and mixed type, with the left ventricular type being the most common. In the early stage, the patient may be asymptomatic. With the progress of the disease, symptoms such as impaired exercise tolerance, lassitude, fatigue, exertional dyspnea, and chest pain may appear. This is mainly because the cardiac output of patients with restrictive cardiomyopathy cannot increase with the acceleration of heart rate.

Treatment

The treatment is mainly to control heart failure. Avoid fatigue and prevent infection. Diuretics should be used for those with edema and ascites.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894730	TNNI3	TT	normal
RS121917760	TNNI3	AA	normal
RS121917761	TNNI3	CC	normal
RS727503499	TNNI3	GG	normal
RS104894729	TNNI3	--	Not Detected

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The restrictive cardiomyopathy genetic disease screening item uses 6 polymorphic loci on the *TNNI3* gene. The test results only indicate the carrying status of the disease-causing gene or loci of restrictive cardiomyopathy in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect restrictive cardiomyopathy. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of restrictive cardiomyopathy.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have restrictive cardiomyopathy.

4. If you have a family history of restrictive cardiomyopathy, or you think that you are likely to suffer from restrictive cardiomyopathy, it is recommended that you consult an authoritative health professional or clinician for help.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect restrictive cardiomyopathy.

Primary Pulmonary Hypertension

Primary pulmonary hypertension is an autosomal dominant inherited disorder characterized by extensive obstruction and occlusion of the smallest peripheral pulmonary artery.

My Primary Pulmonary Hypertension

Non-carrier

Knowledge

Time of onset

It can occur at any age, and the average age of disease is 36 years.

Prevalence

There are about 300-1000 patients in the general population of the United States and Europe each year, and the incidence is about (2-3)/1 million.

Typical symptoms

The initial symptoms of patients at an early stage: Include dyspnea, syncope, chest pain, and lower extremity edema. Progressive heart failure occurs when the right ventricle cannot continue to compensate for burden.

Treatment

1. Drug treatment: The use of vasodilators (such as prostacyclin, nifedipine) is effective in reducing pulmonary hypertension.
2. Surgical treatment: Perform unilateral or bilateral lung transplantation for primary pulmonary hypertension.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121909287	ACVRL1	CC	normal
RS28936401	ACVRL1	CC	normal
RS28936688	ACVRL1	GG	normal
RS63751422	PMS2	GG	normal
RS1057517944	ACVRL1	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The primary pulmonary hypertension genetic disease screening item uses 8 polymorphic loci on genes such as *ACVRL1*. The test results only indicate the carrying status of the disease-causing gene or loci of primary pulmonary hypertension in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect primary pulmonary hypertension. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of primary pulmonary hypertension.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have primary pulmonary hypertension.
4. If you have a family history of primary pulmonary hypertension, or you think that you are likely to suffer from primary pulmonary hypertension, it is recommended that you consult an authoritative

health professional or clinician for help.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect primary pulmonary hypertension.

Long QT Syndrome (LQTS)

Long QT syndrome is an autosomal dominant or autosomal recessive inherited disorder caused by mutations in encoding cardiac ion channels. It is characterized by normal heart structure, prolonged QT interval, abnormal T-wave and polymorphic ventricular arrhythmias (torsade de pointes). These cardiac arrhythmias may result in recurrent syncope, seizure, or sudden death.

My Long QT Syndrome (LQTS)

Non-carrier

Knowledge

Time of onset

It mostly occurs during exercise or emotional excitement, and it may also occur during rest or sleep.

Prevalence

The incidence is about 1/2000.

Typical symptoms

The heart structure is normal, the QT interval is prolonged and the T wave is abnormal. When symptoms occur, they are generally caused by arrhythmias, most commonly a form of ventricular tachycardia called Torsades de pointes, prone to syncope, convulsions, and sudden death.

Treatment

In terms of treatment, medications can be used, taking β -blockers, or surgically implanting a cardioverter defibrillator (Implantable Cardioverter Defibrillator, ICD).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893715	CAV3	GG	normal

RS199473623	SCN5A	CC	normal
RS199473457	KCNQ1	CC	normal
RS199473460	KCNQ1	TT	normal
RS199473479	KCNQ1	TT	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The long QT syndrome genetic disease screening item uses 238 polymorphic loci on genes such as *KCNH2*, *KCNQ1*, and *SCN5A*. The test results only indicate the carrying status of the disease-causing gene or loci of long QT syndrome in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect long QT syndrome. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of long QT syndrome.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have long QT syndrome.
4. If you have a family history of long QT syndrome, or you think that you are likely to suffer from long QT syndrome, it is recommended that you consult an authoritative health professional or clinician for help.

Study population

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect long QT syndrome.

Arrhythmogenic Right Ventricular Cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy, also known as right ventricular cardiomyopathy or arrhythmogenic right ventricular dysplasia, is an autosomal dominant inherited disorder that caused by right ventricular cardiomyopathy. It characterized by ventricular arrhythmias, heart failure and sudden cardiac death.

My Arrhythmogenic Right Ventricular Cardiomyopathy

Non-carrier

Knowledge

Time of onset

It is more common in teenagers.

Prevalence

The incidence is estimated to be between 0.02% and 0.1%, with a high prevalence in some regions (for example, northern Italy) and a male to female prevalence ratio is about 2.7 to 1 among young people. There is still a lack of large sample epidemiological data in China.

Typical symptoms

It is characterized by symptomatic right ventricular arrhythmia, which can lead to sudden death, accompanied by obvious structural and functional abnormalities of the right ventricle.

Treatment

1. ICD implantation: For patients with arrhythmogenic right ventricular cardiomyopathy (ARVC), the prevention of sudden cardiac death (SCD) is the most important.
2. Radiofrequency ablation: Radiofrequency ablation is suitable for those patients who are not suitable for ICD implantation or who cannot control ventricular tachycardia with antiarrhythmic agent after implantation of ICD, resulting in frequent discharge of ICD.
3. Drug treatment: Antiarrhythmic agent is used to control the symptoms of ARVC patients.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121434420	PKP2	GG	normal
RS786204389	intergenic	TT	normal
RS869025392	DSP	GG	normal
RS869025388	DSG2	TT	normal
RS796756333	intergenic	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The arrhythmogenic right ventricular cardiomyopathy genetic disease screening item uses 88 polymorphic loci on genes such as *DSP* and *PKP2*. The test results only indicate the carrying status of the disease-causing gene or loci of arrhythmogenic right ventricular cardiomyopathy in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect arrhythmogenic right ventricular cardiomyopathy. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of arrhythmogenic right ventricular cardiomyopathy.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have arrhythmogenic right

ventricular cardiomyopathy.

4. If you have a family history of arrhythmogenic right ventricular cardiomyopathy, or you think that you are likely to suffer from arrhythmogenic right ventricular cardiomyopathy, it is recommended that you consult an authoritative health professional or clinician for help.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect arrhythmogenic right ventricular cardiomyopathy.

Left Ventricular Noncompaction

Left ventricular noncompaction is a rare autosomal dominant inherited congenital cardiomyopathy disorder characterized by ventricular arrhythmias, heart failure, systemic embolism.

My Left Ventricular Noncompaction

Non-carrier

Knowledge

Time of onset

It can occur at any age, and it is more common in children.

Prevalence

The incidence rate is 5/10000~24/10000.

Typical symptoms

The clinical manifestations are quite different and lack specificity, ranging from asymptomatic to arrhythmia, heart failure, systemic embolism, and even sudden cardiac death.

Treatment

At present, there is no specific method for the treatment of this disease, mainly symptomatic treatment. Malignant arrhythmias can cause sudden death in patients, which needs to be controlled by antiarrhythmic agents. Cardioverter defibrillator can be implanted for those with arrhythmias that are difficult to control.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121909376	MYBPC3	TT	normal

RS121964860	TNNT2	CC	normal
RS121964861	TNNT2	CC	normal
RS199476319	TPM1	AA	normal
RS267606909	MYH7	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The left ventricular noncompaction genetic disease screening item uses 11 polymorphic loci on genes such as *SCN5A*. The test results only indicate the carrying status of the disease-causing gene or loci of left ventricular noncompaction in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect left ventricular noncompaction. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of left ventricular noncompaction.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have left ventricular noncompaction.
4. If you have a family history of left ventricular noncompaction, or you think that you are likely to suffer from left ventricular noncompaction, it is recommended that you consult an authoritative health professional or clinician for help.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect left ventricular noncompaction.

Familial Atrial Fibrillation

Familial atrial fibrillation is an autosomal dominant inherited disorder of the arrhythmia. It can increase the risk of heart rhythm failure and stroke.

My Familial Atrial Fibrillation

Non-carrier

Knowledge

Time of onset

Atrial fibrillation increases sharply with age. The incidence of adults under 40 is close to 1%, the incidence of adults over 40 is 2.3%, and the incidence of elderly over 65 is more than 5%..

Prevalence

The overall prevalence is about 0.89%. The prevalence increases rapidly with age.

Typical symptoms

1. Palpitations: feeling of heartbeat, irregular and fast heartbeat, physical fatigue or tiredness.
2. Dizziness: dizziness or fainting.
3. Chest discomfort: pain, compression or discomfort.
4. Shortness of breath: feeling of difficulty breathing during light physical activity or rest.

Treatment

1. Restoration of sinus rhythm: This is the best result of atrial fibrillation treatment. Only the restoration of sinus rhythm (normal heart rhythm) can achieve the purpose of complete treatment of atrial fibrillation. Therefore, any patient with atrial fibrillation should try to restore sinus rhythm.
2. Control of rapid ventricular rate: For patients with atrial fibrillation who cannot restore sinus rhythm, drugs can be used to slow down the rapid ventricular rate.
3. Prevention of thrombosis and stroke: If sinus rhythm cannot be restored during atrial fibrillation, anticoagulant drugs can be used to prevent thrombosis and stroke.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS120074192	KCNQ1	AA	normal
RS121434557	GJA5	CC	normal
RS121908590	KCNA5	GG	normal
RS121908593	KCNA5	GG	normal
RS137854618	SCN5A	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The familial atrial fibrillation genetic disease screening item uses 12 polymorphic loci in genes such as *GJA5* and *SCN5A*. The test results only indicate the carrying status of the disease-causing gene or loci of familial atrial fibrillation in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect familial atrial fibrillation. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of familial atrial fibrillation.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have familial atrial

fibrillation.

4. If you have a family history of familial atrial fibrillation, or you think that you are likely to suffer from familial atrial fibrillation, it is recommended that you consult an authoritative health professional or clinician for help.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect familial atrial fibrillation.

Haemophilia A

Hemophilia A is an X-linked recessive genetic disease that causes bleeding tendency due to functional defects of coagulation factor VIII.

My Haemophilia A
Non-carrier

Knowledge

Time of onset

It usually occurs after birth and in infancy.

Prevalence

The two main forms of hemophilia are more common in men than in women. The incidence of hemophilia A in males is about 1/5000, and the incidence of female is rare.

Typical symptoms

The main manifestations are bleeding tendency, such as long-term bleeding caused by a minor injury and surgery and spontaneous bleeding. Early symptoms can be seen in continuous bleeding after a needle is tapped into the skin, such as intravenous injection. Severe bleeding is also common in hemarthrosis, muscle haematoma and Intracranial Hematomas.

Treatment

Replacement therapy: The bleeding symptoms of patients with hemophilia A can be treated by factor VIII concentrates.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137852366	F8	CC	normal

RS137852378	F8	TT	normal
RS137852382	F8	AA	normal
RS137852383	F8	AA	normal
RS137852402	F8	TT	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The haemophilia A genetic disease screening item uses 10 polymorphic loci on the *F8* gene. The test results only indicate the carrying status of the disease-causing gene or loci of haemophilia A in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of haemophilia A.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have haemophilia A.
4. If you have a family history of haemophilia A, or you think that you are likely to suffer from haemophilia A, it is recommended that you consult an authoritative health professional or clinician for help.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that affect haemophilia A.

Haemophilia B

Hemophilia B is an X-linked recessive genetic disease that causes bleeding tendency due to functional defects of coagulation factor IX.

My Haemophilia B
Non-carrier

Knowledge



Time of onset

It usually occurs within 1-2 years after birth.



Prevalence

The two main forms of hemophilia are more common in men than in women. The incidence of hemophilia B in males is about 1/30000, and the incidence of females is rare.



Typical symptoms

The typical features are prolonged bleeding or spontaneous bleeding after injury of mucosa, soft tissue and joints.



Treatment

Replacement therapy: Coagulation factor IX replacement therapy is the main treatment scheme, and factor IX concentrates can be supplemented by blood transfusion.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137852229	F9	AA	normal
RS137852274	F9	GG	normal

RS137852261	F9	CC	normal
RS137852262	F9	AA	normal
RS137852265	F9	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The haemophilia B genetic disease screening item uses 48 polymorphic loci on the *F9* gene. The test results only indicate the carrying status of the disease-causing gene or loci of haemophilia B in the user. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all the pathogenic genes or loci of that affect haemophilia B. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This study only screens your carrying status of the disease-causing gene or loci of haemophilia B.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have haemophilia B.
4. If you have a family history of haemophilia B, or you think that you are likely to suffer from haemophilia B, it is recommended that you consult an authoritative health professional or clinician for help.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, this test may not cover all disease-causing genes or loci that related to haemophilia B.

Duchenne Muscular Dystrophy (DMD)

Duchenne muscular dystrophy is inherited in an X-linked recessive pattern, muscular dystrophy mainly occurs in boys with progressive muscle weakness and muscle atrophy.

My Duchenne Muscular Dystrophy (DMD)

Non-carrier

Knowledge



Time of onset

It begins in early childhood and usually begins around the age of 3.



Prevalence

According to statistics, one out of every 3,500 newborn boys in the world suffers from this disease, which is the most common muscular dystrophy disease.



Typical symptoms

1. The first symptoms are mostly developmental delays, especially delays in learning to walk, and difficulties in walking, running, jumping and climbing stairs.
2. Frequent falls, staggering walking, often toe-walking, difficulty standing up from a sitting position. Children usually lose the ability to walk completely at the age of 7 and 12, and die at the age of 20 due to weakness of myocardium and lung muscles.
3. About one-third of children have mild, non-progressive intellectual disabilities that primarily affect speech skills.



Treatment

There is no effective treatment for the disease. physiotherapy, accessories, and surgical correction can help relieve symptoms. Patients with weak breathing muscles can be equipped with breathing aids. Drugs can use corticosteroids to slow muscle degeneration; anti-convulsive drugs can be used to control seizures and abnormal muscle movements; and immunosuppressants can delay muscle cell death and damage.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894787	DMD	GG	normal
RS863224979	DMD	GG	normal
RS863224987	DMD	TT	normal
RS863224986	DMD	GG	normal
RS863224985	DMD	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The Duchenne muscular dystrophy screening item uses 88 polymorphic loci on the *MCM6* gene. The test results only describe the carrying of the user's Duchenne muscular dystrophy-causing gene or site. The basis of the test is mainly from the research of Caucasian, and other populations are for reference only.

Notification

1. The test may not cover all disease-causing genes or loci of Duchenne muscular dystrophy.
2. The test is not suitable for clinical diagnosis. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The development of the disease are determined by multiple factors such as genes and environment. This study only screens the disease-causing genes or loci of Duchenne muscular dystrophy that you carry.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have Duchenne muscular

dystrophy.

4. If you have a family history of Duchenne muscular dystrophy, or you think that you are likely to suffer from Duchenne muscular dystrophy, it is recommended that you consult an authoritative health professional or clinician for help.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci of Duchenne muscular dystrophy.

Pulmonary Lymphangiomyomatosis (PLAM)

Pulmonary lymphangiomyomatosis is a rare diffuse lung disease. The lesions is mainly about abnormal proliferation of immature smooth muscle in the lung interstitium, bronchi, blood vessels and lymphatic vessels, and even abdominal lymphatic vessels.

My Pulmonary Lymphangiomyomatosis (PLAM)

Non-carrier

Knowledge

Time of onset

The usual incidence is in premenopausal women of childbearing age.

Prevalence

The disease is rare and mainly occurs in young women.

Typical symptoms

The initial symptoms are difficulty breathing, as well as unusual coughs, chest pain and hemorrhage. There are few signs of disease, but some women have a pronunciation and dry tone. Many patients develop spontaneous airways. they can also show signs of blockages in the lymphatic system, including celiac chest, celiac ascites and celiac urine. Symptoms worsen during pregnancy and may worsen during air travel.

Treatment

1. There is lack of effective means of treatment. Generally, treatment is used to improve the lung function of patients, closed thoracic drainage was performed for pneumothorax and chylothorax. Lung transplantation can be considered in severe cases.
2. Other supportive treatment: if the reversible test of lung function is positive, it can be given β_2 receptor Stimulants; Use pneumonia and influenza vaccine to prevent lung infection.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS28934872	TSC2	GG	normal
RS45483392	TSC2	CC	normal
RS45469298	TSC2	CC	normal
RS45517412	TSC2	CC	normal

Test details

Applicable situation

The pulmonary lymphangioleiomyomatosis item provided uses 4 polymorphic loci of the *TSC2* gene. The test results only indicate the user's pulmonary lymphangioleiomyomatosis disease-causing gene or locus carrying status. The basis of the test is mainly from the research of Caucasian, and other populations are for reference only.

Notification

1. The test may not cover all disease-causing genes or loci of pulmonary lymphangioleiomyomatosis.
2. The test results are not applicable to clinical practice. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This study only screens you for carrying pulmonary lymphangioleiomyomatosis genes or loci.
3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may suffer from pulmonary lymphangioleiomyomatosis.
4. If you have a family history of pulmonary lymphangioleiomyomatosis, or you think that you are likely to suffer from pulmonary lymphangioleiomyomatosis, it is recommended that you consult

an authoritative health professional or clinician for help.

 Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci related to pulmonary lymphangioleiomyomatosis.

Pheochromocytoma

Pheochromocytoma is a chromosomal dominant inheritance disease that originates from the adrenal medulla, sympathetic ganglia or other chromaffin tissues. This tumor continuously or intermittently releases large amounts of catecholamines, causing a persistent or paroxysmal hypertension and multiple organs functional and metabolic disorders.

My Pheochromocytoma

Non-carrier

Knowledge

Time of onset

The disease is most common between 20 and 50 years old.

Prevalence

The incidence rate is approximately 0.8 per 100,000.

Typical Symptoms

Paroxysmal headache, sweating, and palpitation, may be accompanied by tremors, pale complexion, difficulty breathing, general weakness and sudden panic.

Treatment

1. Surgical treatment: Surgical tumor resection is the preferred treatment.
2. Medication: α -blocker and β -blocker are used in combination to control hypertension.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894304	SDHD	AA	normal

RS104894307	SDHD	AA	normal
RS5030808	VHL	GG	normal
RS74315368	SDHB	CC	normal
RS74315370	SDHB	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

The screening for hereditary diseases of pheochromocytoma item uses the 9 polymorphic loci of *SDHD*, *SDHB* genes. The test results only indicate the carrying status of the user's pheochromocytoma disease-causing gene or locus. The basis of the test is mainly from the research of Caucasian, other populations are for reference only.

Notification

The test may not cover all genes or loci that can lead to the risk of pheochromocytoma. The test is not suitable for clinical diagnosis. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of the disease are determined by multiple factors such as genes and environment. This test will only screen you for the disease-causing genes or loci of pheochromocytoma.
3. It should be noted that whether or not you carry the relevant variant does not indicate your true disease state. Still, it cannot be ruled out that you may suffer from pheochromocytoma.
4. If you have a family history of pheochromocytoma, or you think that you are likely to suffer from pheochromocytoma, it is recommended that you consult with an authoritative health professional or clinician for further advice.

Study population

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that related to pheochromocytoma.

Dementia with Lewy Body (DLB)

Dementia with lewy body is a progressive cognitive deficit disease characterized by the formation of Lewy bodies in nerve cells. It is inherited in autosomal dominant.

My Dementia with Lewy Body (DLB)

Non-carrier

Knowledge

Time of onset

People over 60 years old are prone to the disease.

Prevalence

Dementia with lewy body accounts for 15% - 20% of senile dementia, second only to Alzheimer's disease. Based on a small number of demographic surveys, the prevalence of dementia with lewy body is 0.1% - 2.0% in the elderly over 65 years old and 5.0% in the population over 75 years old.

Typical symptoms

The symptoms of dementia with lewy body are very similar to Alzheimer's disease, including amnesia, disorientation, and disturbances in memory, thinking, understanding, communication and control behavior. However, dementia with lewy body can be distinguished by the following symptoms:

1. Early neurological function fluctuates obviously, and dramatic changes can occur in a few days to weeks or even in a very short time.
2. In the early stage of the disease, the impairment of attention and alertness is earlier than the memory loss, including the memory loss of recent events.
3. The impairment of copying and drawing ability is more serious than other neurological impairment symptoms.
4. Psychiatric symptoms such as hallucinations, delusions, and paranoia are common, and hallucinations appear earlier.

Treatment

The general treatment is the same as that of all dementia, including improving safety and supportive treatment. There is no special treatment for Lewy body dementia, and drugs such as rivastigmine may be effective in the treatment of Alzheimer's disease. This drug can improve mental function. In Lewy body dementia, try not to use antipsychotics to treat hallucinations and delusions. This drug tends to aggravate muscle and motor symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS78973108	GBA	CC	normal
RS80356771	GBA	--	Not Detected

Test details

Applicable situation

The health risks of dementia with lewy body item We uses the 2 polymorphic loci of *GBA* gene. The test results show whether the user has a higher prevalence of dementia with lewy body than all users of We. The test cannot represent the user's real risk of dementia with lewy body. The test is mainly based on the research of Caucasian, and other populations are only for reference.

Notification

1. The test may not cover all genes or loci that affect dementia with lewy body.
2. The test results have no clinical significance.
3. If you have clinical needs, please contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of diseases are determined by multiple factors such as genes and environment. This test will only screen you for the disease-causing genes or loci of of dementia with lewy body.

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3. It should be noted that carrying the relevant variant does not mean your true disease state; if you do not carry the relevant variant, it cannot be ruled out that you may have dementia with lewy body.
 4. If you have a family history of dementia with lewy body, or you think that you are likely to suffer from dementia with lewy body, it is recommended that you consult an authoritative health professional or clinician for help.

Study population

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all disease-causing genes or loci that affect dementia with lewy body.

Angelman Syndrome (AS)

It is a neuro-genetic disorder characterized by developmental delay, intellectual disability, speech impairments, and movement and balance disorders.

My Angelman Syndrome (AS)

Non-carrier

Knowledge

Time of onset

Symptoms of this syndrome usually appear between 6 and 12 months old and become more evident as the child grows.

Prevalence

The prevalence of it is estimated to be 1 in 12,000 to 20,000 individuals worldwide.

Typical symptoms

The typical symptoms of this syndrome include epilepsy, sleep disorders, abnormally small head, strabismus, abnormal spinal curvature, clapping or hand swinging, and attention deficit and hyperactivity disorder.

Treatment

The treatment for this syndrome are typically focused on managing symptoms and improving quality of life, and may include various therapies and medications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS587780570	intergenic	II	normal
RS587784515	UBE3A	AA	normal

RS797046087	intergenic	DD	normal
RS797046086	intergenic	DD	normal
RS797046084	intergenic	DD	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Angelman Syndrome (AS) uses 27 polymorphic loci on the *UBE3A* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Angelman Syndrome (AS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Angelman Syndrome (AS), or if you feel that you have a risk of developing Angelman Syndrome (AS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 27 polymorphic loci associated with the risk of Angelman Syndrome (AS), such as rs587780570, rs587780577 and rs587781204.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Angelman Syndrome (AS).

Galactosialidosis (GSL)

It is a rare genetic lysosomal storage disease caused by a deficiency in the enzyme protective protein cathepsin A. The disease progresses slowly and leads to organ, tissue, and nerve damage.

My Galactosialidosis (GSL)

Non-carrier

Knowledge



Time of onset

Symptoms of it may appear in childhood or adolescence, and the severity of the disease may vary.



Prevalence

It is an exceedingly rare disease with only a few cases diagnosed worldwide.



Typical symptoms

Typical symptoms of it include developmental delays, mental retardation, and a progressive decline in intellectual function. In addition, skeletal deformities, joint stiffness, hearing impairment, and vision problems can occur.



Treatment

Treatment of it is primarily symptomatic and supportive. There are no cures, but enzyme replacement therapy, bone marrow transplantation, and gene therapy are being researched.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137854547	CTSA	GG	normal
RS786200859	CTSA	AA	normal

Test details

Applicable situation

Genetic disease screening test for Galactosialidosis (GSL) uses 2 polymorphic loci on the *CTSA* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Galactosialidosis (GSL).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Galactosialidosis (GSL), or if you feel that you have a risk of developing Galactosialidosis (GSL), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Galactosialidosis (GSL), such as rs137854547 and rs786200859.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Galactosialidosis (GSL).

Galactosemia (GALAC)

It is a rare genetic disorder in which the body is unable to properly metabolize galactose, a sugar found in dairy products and many other foods. This results in a buildup of galactose that can damage various organs and cause severe complications if left untreated.

My Galactosemia (GALAC)

Non-carrier

Knowledge

Time of onset

Symptoms of it can emerge within days or weeks of birth because newborns typically consume a lactose-based milk diet. However, some people with milder forms of the disease may not experience symptoms until later in life.

Prevalence

It is a rare disease, affecting approximately 1 in 60,000 to 80,000 newborns.

Typical symptoms

Typical symptoms of it include feeding difficulties, jaundice, and lethargy. Later symptoms may include developmental delays, speech difficulties, and motor problems. Adults with galactosemia may suffer from cataracts, cognitive impairment, and infertility.

Treatment

The main treatment for this disease is not to eat foods containing lactose or galactose for life. In severe cases, infants may require emergency medical intervention, including hospitalization, to prevent brain or other organ damage.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS111033658	GALT	CC	normal
RS111033739	GALT	CC	normal
RS786204763	GALT	CC	normal
RS111033768	GALT	AA	normal
RS398123179	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Galactosemia (GALAC) uses 24 polymorphic loci on the *GALT* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Galactosemia (GALAC).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Galactosemia (GALAC), or if you feel that you have a risk of developing Galactosemia (GALAC), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 24 polymorphic loci associated with the risk of Galactosemia (GALAC), such as rs111033658, rs111033661 and rs111033667.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Galactosemia (GALAC).

Megalencephalic Leukoencephalopathy with Subcortical Cysts (MLC)

It is a progressive condition that affects brain development and function. Individuals with this condition typically have an enlarged brain (megalencephaly) that is evident at birth or within the first year of life. Affected people also have leukoencephalopathy, an abnormality of the brain's white matter.

My Megalencephalic Leukoencephalopathy with Subcortical Cysts (MLC)

Non-carrier

Knowledge

Time of onset

It typically presents in early childhood, with symptoms appearing between the age of 1 to 3.

Prevalence

It is estimated to affect 1 in 100,000 individuals.

Typical symptoms

Typical symptoms include delayed motor development, ataxia, spasticity, cognitive impairment and epilepsy.

Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support. Antiepileptic drugs may be prescribed to control seizures, while physiotherapy and occupational therapy may help individuals maintain their mobility.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS387907053

HEPACAM

CC

normal

Test details

Applicable situation

Genetic disease screening test for Megalencephalic Leukoencephalopathy with Subcortical Cysts (MLC) uses 1 polymorphic locus on the *HEPACAM* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Megalencephalic Leukoencephalopathy with Subcortical Cysts (MLC).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Megalencephalic Leukoencephalopathy with Subcortical Cysts (MLC), or if you feel that you have a risk of developing Megalencephalic Leukoencephalopathy with Subcortical Cysts (MLC), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Megalencephalic Leukoencephalopathy with Subcortical Cysts (MLC), namely rs387907053.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Megalencephalic Leukoencephalopathy with Subcortical Cysts (MLC).

Osteopathia Striata with Cranial Sclerosis (OSCS)

It is a rare genetic disorder that affects bone development in different parts of the body. It is characterized by linear striations or dense, ivory-like areas visible on X-rays of the long bones and skull.

My Osteopathia Striata with Cranial Sclerosis (OSCS)

Non-carrier

Knowledge



Time of onset

It typically appears during early childhood or adolescence.



Prevalence

The prevalence of this disease is not known, but it is considered to be very rare.



Typical symptoms

Typical symptoms include short stature, bowed limbs, and thickened bones in the skull, as well as dental problems, hearing loss, and intellectual disability.



Treatment

Regular monitoring for hearing loss and dental abnormalities is required; Physical therapy may help prevent joint contractures and improve ambulation; Orthopedic surgery may be recommended in severe cases.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS398122877	AMER1	II	normal

Test details

Applicable situation

Genetic disease screening test for Osteopathia Striata with Cranial Sclerosis (OSCS) uses 1 polymorphic locus on the *AMER1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Osteopathia Striata with Cranial Sclerosis (OSCS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Osteopathia Striata with Cranial Sclerosis (OSCS), or if you feel that you have a risk of developing Osteopathia Striata with Cranial Sclerosis (OSCS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Osteopathia Striata with Cranial Sclerosis (OSCS), namely rs398122877.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Osteopathia Striata with Cranial Sclerosis (OSCS).

Phenylketonuria (PKU)

It is a genetic disorder characterized by the inability of the body to process an amino acid called phenylalanine, leading to its accumulation in the blood and brain. This can cause severe intellectual disability, seizures, behavioral problems, and skin disorders.

My Phenylketonuria (PKU)

Non-carrier

Knowledge

Time of onset

Symptoms appear within a few months after birth, once the baby begins to consume breast milk or formula containing phenylalanine.

Prevalence

It is a rare disease and its prevalence varies among different populations. In the United States, it affects approximately 1 in 10,000 to 15,000 newborns.

Typical symptoms

Common symptoms in infants include vomiting, irritability, an unusually small head size, and developmental delays. If left untreated, the disorder can cause severe intellectual disability, seizures, and behavioral problems.

Treatment

This disease can be managed through a special diet low in phenylalanine, which is typically started shortly after birth and maintained throughout life. Additional treatment may include medication and close medical monitoring.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS199475566	intergenic	II	normal
RS199475565	intergenic	II	normal
RS62508694	PAH	CC	normal
RS62508730	PAH	CC	normal
RS62509015	PAH	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Phenylketonuria (PKU) uses 43 polymorphic loci on the *PAH* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Phenylketonuria (PKU).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Phenylketonuria (PKU), or if you feel that you have a risk of developing Phenylketonuria (PKU), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 43 polymorphic loci associated with the risk of Phenylketonuria (PKU), such as rs140175796, rs180819807 and rs199475565.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Phenylketonuria (PKU).

Propionic Acidemia (PA)

It is a rare metabolic genetic disease characterized by a lack of propionyl CoA carboxylase, an enzyme involved in the catabolism of essential amino acids valine, methionine, isoleucine, and threonine. If lacking, it can lead to the accumulation of propionyl CoA in the body, which can damage the brain, heart, kidneys, liver, etc.

My Propionic Acidemia (PA)

Non-carrier

Knowledge



Time of onset

It typically appears during infancy or early childhood.



Prevalence

It is estimated to affect 1 in 100,000 to 150,000 newborns worldwide.



Typical symptoms

Typical symptoms include poor feeding, vomiting, dehydration, lethargy, hypotonia, and delayed physical and mental development.



Treatment

Patients may require specialized formulas or medications to help manage their symptoms, while regular monitoring of blood chemistry and other indicators can help prevent complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS202247822	PCCB	TT	normal

RS572246667	PCCB	CC	normal
RS796052019	PCCA	GG	normal
RS794727620	intergenic	GG	normal
RS794726976	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Propionic Acidemia (PA) uses 19 polymorphic loci on the *PCCB*, *UQCRFS1* and *PCCA* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Propionic Acidemia (PA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Propionic Acidemia (PA), or if you feel that you have a risk of developing Propionic Acidemia (PA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 19 polymorphic loci associated with the risk of Propionic Acidemia (PA), such as rs1553778865, rs1568346416 and rs202247822.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Propionic Acidemia (PA).

Pyruvate Kinase Deficiency (PKD)

It is a rare genetic disorder characterized by a deficiency of the enzyme pyruvate kinase, which plays a key role in the breakdown of glucose for energy production in red blood cells. Without adequate levels of this enzyme, red blood cells are unable to maintain their shape and function properly, leading to hemolysis (destruction of red blood cells) and anemia.

My Pyruvate Kinase Deficiency (PKD)

Non-carrier

Knowledge



Time of onset

It typically appears in infancy or early childhood.



Prevalence

It is estimated to affect 1 in 20,000 individuals worldwide.



Typical symptoms

The most common symptoms include fatigue, pallor, jaundice, shortness of breath, abdominal pain, and splenomegaly.



Treatment

In some cases, blood transfusions and splenectomy may be necessary. In severe cases, bone marrow transplant may be considered.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS201953584	PKLR	CC	normal

Test details

Applicable situation

Genetic disease screening test for Pyruvate Kinase Deficiency (PKD) uses 1 polymorphic locus on the *PKLR* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Pyruvate Kinase Deficiency (PKD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Pyruvate Kinase Deficiency (PKD), or if you feel that you have a risk of developing Pyruvate Kinase Deficiency (PKD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Pyruvate Kinase Deficiency (PKD), namely rs201953584.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Pyruvate Kinase Deficiency (PKD).

Pyruvate Carboxylase Deficiency (PCD)

It is a rare genetic disorder that affects the metabolism of carbohydrates, causing neurological and developmental problems. The body has difficulty using carbohydrates for energy, leading to an accumulation of toxic by-products in the blood and brain.

My Pyruvate Carboxylase Deficiency (PCD)

Non-carrier

Knowledge



Time of onset

Symptoms usually begin in early infancy.



Prevalence

It has an estimated incidence of around 1 in 250,000 to 1 in 2,700,000 individuals in the general population.



Typical symptoms

Symptom may include developmental delay, feeding problems, hypotonia, seizures, and neurologic abnormalities.



Treatment

Treatment may include medications to control seizures or alleviate acidosis, as well as regular monitoring of blood and urinary biomarkers. A low-carbohydrate and high-fat diet may also be recommended.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS113994145	PC	CC	normal

RS28940591

PC

AA

normal

Test details

Applicable situation

Genetic disease screening test for Pyruvate Carboxylase Deficiency (PCD) uses 2 polymorphic loci on the *PC* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Pyruvate Carboxylase Deficiency (PCD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Pyruvate Carboxylase Deficiency (PCD), or if you feel that you have a risk of developing Pyruvate Carboxylase Deficiency (PCD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Pyruvate Carboxylase Deficiency (PCD), such as rs113994145 and rs28940591.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Pyruvate Carboxylase Deficiency (PCD).

Syndactyly

It is a congenital abnormality characterized by the fusion of two or more digits or fingers. It occurs due to a failure of apoptosis during limb development in utero.

My Syndactyly

Non-carrier

Knowledge



Time of onset

It is present at birth.



Prevalence

It is estimated to affect approximately 1 in 2,000 live births.



Typical symptoms

The most obvious symptom is the fusion of two or more digits or fingers. In some cases, the webbing may be complete and extend along the entire length of the digits.



Treatment

Surgical intervention is the most common treatment. The goal of surgery is to separate the fused digits and create new soft tissue and skin between them. The timing of surgery depends on the severity of the condition and the age of the patient.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS200750564	HOXD13	CC	normal

Test details

Applicable situation

Genetic disease screening test for Syndactyly uses 1 polymorphic locus on the *HOXD13* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Syndactyly.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Syndactyly, or if you feel that you have a risk of developing Syndactyly, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Syndactyly, namely rs200750564.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Syndactyly.

Vitreoretinchoroidopathy (VRCP)

It is a genetic disorder that affects the eyes. It is characterized by abnormalities in the vitreous, retina, and choroid, which can lead to degeneration and vision loss.

My Vitreoretinchoroidopathy (VRCP)

Non-carrier

Knowledge



Time of onset

Some individuals experiencing symptoms in childhood while others do not show any symptoms until later in life.



Prevalence

The prevalence of this disease is currently unknown.



Typical symptoms

Typical symptoms include blurry or distorted vision, floaters, and difficulty seeing in low light conditions.



Treatment

Treatment focus on managing symptoms and preventing complications. For example, surgery to correct retinal or vitreous abnormalities, as well as regular eye exams to monitor changes in vision.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS281865238	BEST1	CC	normal

Test details

Applicable situation

Genetic disease screening test for Vitreoretinopathopathy (VRCP) uses 1 polymorphic locus on the *BEST1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Vitreoretinopathopathy (VRCP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Vitreoretinopathopathy (VRCP), or if you feel that you have a risk of developing Vitreoretinopathopathy (VRCP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Vitreoretinopathopathy (VRCP), namely rs281865238.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Vitreoretinopathopathy (VRCP).

Osteogenesis Imperfecta (OI)

It is a group of genetic disorders that affect the bone and increase the propensity for fractures.

My Osteogenesis Imperfecta (OI)

Non-carrier

Knowledge



Time of onset

The symptoms can begin at birth or during childhood, and can become more severe over time.



Prevalence

It is estimated to affect 1 in every 20,000 live births worldwide.



Typical symptoms

Typical symptoms include frequent fractures, blue sclerae, short stature and skeletal deformities.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support, for example, prevention of fractures and the use of mobility aids such as braces or wheelchairs.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS118203996	P3H1	GG	normal
RS72658154	COL1A2	GG	normal
RS794727470	COL1A2	GG	normal
RS794726873	COL1A1	GG	normal

RS786201032

IFITM5

GG

normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Osteogenesis Imperfecta (OI) uses 29 polymorphic loci on genes such as *P3H1*, *ALPL* and *COL1A1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Osteogenesis Imperfecta (OI).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Osteogenesis Imperfecta (OI), or if you feel that you have a risk of developing Osteogenesis Imperfecta (OI), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 29 polymorphic loci associated with the risk of Osteogenesis Imperfecta (OI), such as rs118203996, rs121918011 and rs193922143.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Osteogenesis Imperfecta (OI).

Acth-independent Macronodular Adrenal Hyperplasia (AIMAH)

It is a rare endocrine disorder in which cortisol is unregulated by the hypothalamic adrenocorticotrophic cortex and is hypersecreted as a result of adrenal dysplasia. Thereby causing various physical symptoms.

My Acth-independent Macronodular Adrenal Hyperplasia (AIMAH)

Non-carrier

Knowledge



Time of onset

The onset time for it is variable, and it can develop at any age.



Prevalence

The prevalence of this disease is unknown, but it is considered a rare condition.



Typical symptoms

The disease causes a hypersecretory state of cortisol, androgens, or aldosterone that may cause cushing's syndrome, with typical symptoms including hypertension, hypokalemia, weight gain, muscle weakness, fatigue, and mood changes. some patients may also present with diabetes, osteoporosis, and menstrual irregularities in women.



Treatment

The treatment for this disease depends on the specific hormone excess present. surgery is the preferred treatment, with the goal of removing excess hormone production. alternatively, treatment may involve hormone-suppressing medications, such as ketoconazole, mitotane, and metyrapone. follow-up examinations are necessary to monitor recurrence and adrenal function post-surgery.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS369721476	ARMC5	CC	normal

Test details

Applicable situation

Genetic disease screening test for Acth-independent Macronodular Adrenal Hyperplasia (AIMAH) uses 1 polymorphic locus on the *ARMC5* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Acth-independent Macronodular Adrenal Hyperplasia (AIMAH).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Acth-independent Macronodular Adrenal Hyperplasia (AIMAH), or if you feel that you have a risk of developing Acth-independent Macronodular Adrenal Hyperplasia (AIMAH), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Acth-independent Macronodular Adrenal Hyperplasia (AIMAH), namely rs369721476.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Acth-independent Macronodular Adrenal Hyperplasia (AIMAH).

Epidermolysis Bullosa (EB)

It is a genetic disorder that affects the skin and mucous membranes, causing them to be incredibly fragile and prone to blistering and tearing.

My Epidermolysis Bullosa (EB)

Non-carrier

Knowledge



Time of onset

The for it varies depending on the type and severity of the disease, with some forms appearing at birth, while others may not present until later in childhood or even into adulthood.



Prevalence

While this disease is considered rare, there are still thousands of individuals affected by it worldwide.



Typical symptoms

The typical symptoms of it include blistering of the skin and mucous membranes, scarring, difficulty eating, and a higher risk of infection due to the fragile nature of the skin.



Treatment

Unfortunately, there is currently no cure for it, but treatment options may include wound care, infection prevention, pain management, and surgery to correct any physical deformities resulting from the disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121912839	COL7A1	CC	normal
RS1057516241	intergenic	II	normal

RS886058642	intergenic	CC	normal
RS886037957	KLHL24	GG	normal
RS886037956	KLHL24	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Epidermolysis Bullosa (EB) uses 23 polymorphic loci on genes such as *LAMB3*, *COL7A1* and *KRT5*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Epidermolysis Bullosa (EB).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Epidermolysis Bullosa (EB), or if you feel that you have a risk of developing Epidermolysis Bullosa (EB), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 23 polymorphic loci associated with the risk of Epidermolysis Bullosa (EB), such as rs1057516241, rs1057516486 and rs121912839.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Epidermolysis Bullosa (EB).

Cholestasis

It is a condition where bile flow from the liver to the small intestine is impaired. This can cause a buildup of bile acids and bilirubin in the liver and bloodstream, resulting in symptoms such as itching, jaundice, and fatigue.

My Cholestasis

Non-carrier

Knowledge

Time of onset

It can occur at any time but is most commonly seen during the later stages of pregnancy.

Prevalence

It occurs in approximately 1-2% of pregnancies and is more common in certain populations, such as women of South American or Scandinavian descent.

Typical symptoms

The most common symptoms of it include itching, jaundice, and fatigue. Other symptoms may include dark urine, clay-colored stools, and upper abdominal pain.

Treatment

Treatment for it typically involves managing symptoms and reducing the risk of complications. This may include medications to reduce itching, increased monitoring of fetal well-being in pregnancy, and delivery of the baby if necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121909098	ATP8B1	CC	normal

RS72549402	ABCB11	TT	normal
RS758069019	intergenic	CC	normal
RS769910565	intergenic	AA	normal
RS863225298	ABCB4	GG	normal

Test details

Applicable situation

Genetic disease screening test for Cholestasis uses 5 polymorphic loci on the *ATP8B1*, *ABCB11* and *ABCB4* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Cholestasis.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Cholestasis, or if you feel that you have a risk of developing Cholestasis, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Cholestasis, such as rs121909098, rs72549402 and rs758069019.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Cholestasis.

Hypokalemic Periodic Paralysis (HKOPP)

It is a rare genetic disorder characterized by sudden episodes of muscle weakness or paralysis that may last for a few minutes or hours. It is caused by a mutation in one of three genes encoding for skeletal muscle calcium, sodium, or potassium channels, which affects the balance of ions in the muscle cells and leads to weakness.

My Hypokalemic Periodic Paralysis (HKOPP)

Non-carrier

Knowledge



Time of onset

It usually begins in childhood or adolescence, with symptoms appearing between the ages of 7 and 21. However, some cases may not present until adulthood.



Prevalence

The prevalence of it is estimated to be between 1 in 100,000 to 1 in 25,000 individuals worldwide.



Typical symptoms

The hallmark symptom of it is sudden episodes of muscle weakness or paralysis that may affect any part of the body, most commonly the arms, legs, and trunk. These episodes may be triggered by factors such as exercise, emotional stress, cold temperature, or high carbohydrate meals.



Treatment

The treatment for it aims to prevent and manage episodes of muscle weakness or paralysis. This may involve avoiding triggers, such as exercise or certain foods, and taking medications that help to balance the levels of ions in the muscle cells. In some cases, periodic potassium supplements or acetazolamide may be prescribed.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121908552	SCN4A	CC	normal
RS267606698	CACNA1S	AA	normal
RS527236150	SCN4A	CC	normal
RS80338777	CACNA1S	CC	normal
RS80338785	intergenic	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Hypokalemic Periodic Paralysis (HKOPP) uses 9 polymorphic loci on the *SCN4A* and *CACNA1S* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hypokalemic Periodic Paralysis (HKOPP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.

4. If you have a family history of Hypokalemic Periodic Paralysis (HKOPP), or if you feel that you have a risk of developing Hypokalemic Periodic Paralysis (HKOPP), it is recommended that you consult a qualified health professional or clinician for help.

 Test content

This test includes 9 polymorphic loci associated with the risk of Hypokalemic Periodic Paralysis (HKOPP), such as rs121908552, rs1553252746 and rs1558056376.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hypokalemic Periodic Paralysis (HKOPP).

Hypophosphatemic Rickets (HR)

It is a rare genetic disorder that affects the bones. It is caused by low levels of phosphate in the blood, which leads to abnormal bone development and growth.

The disease is characterized by abnormally curved bones, short stature, and dental problems, among other symptoms.

My Hypophosphatemic Rickets (HR)

Non-carrier

Knowledge

Time of onset

It typically manifests during childhood or adolescence, but can also affect adults.

Prevalence

The prevalence of it is estimated to be around 1 in 20,000 to 1 in 100,000 individuals worldwide.

Typical symptoms

The symptoms of it include bowed legs or knock knees, short stature, dental abnormalities, bone pain, and weakness.

Treatment

Treatment for it may include oral phosphate and vitamin D supplements, as well as other medications that can help regulate phosphate levels in the blood. In some cases, surgery may also be necessary to correct bone deformities.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121908248	intergenic	GG	normal

RS193922702	FGF23	CC	normal
RS28937882	FGF23	GG	normal
RS763922486	intergenic	GG	normal
RS797044813	CLCN5	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Hypophosphatemic Rickets (HR) uses 6 polymorphic loci on genes such as *ENPP1*, *FGF23* and *CLCN5*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hypophosphatemic Rickets (HR).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hypophosphatemic Rickets (HR), or if you feel that you have a risk of developing Hypophosphatemic Rickets (HR), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 6 polymorphic loci associated with the risk of Hypophosphatemic Rickets (HR), such as rs121908248, rs193922702 and rs28937882.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hypophosphatemic Rickets (HR).

Hypophosphatasia (HPP)

It is a rare genetic disorder that affects the development of bones and teeth. It is caused by a deficiency of the enzyme alkaline phosphatase which results in the accumulation of phosphates in the body, leading to weak bones and teeth.

My Hypophosphatasia (HPP)

Non-carrier

Knowledge

Time of onset

The onset of it can occur at any age, from before birth to late adulthood.

Prevalence

It is a rare disease and its prevalence is estimated to be 1 in 100,000 to 1 in 300,000 individuals worldwide.

Typical symptoms

The symptoms of it can vary widely and may include weak bones, soft and brittle teeth, short stature, skeletal deformities, muscle weakness, nerve compression, and respiratory problems.

Treatment

Treatment for it is supportive and aims to manage symptoms. This may include medication to control pain and prevent fractures, physical therapy to improve mobility and strength, and surgery to correct skeletal deformities or nerve compression. Enzyme replacement therapy is also an emerging treatment option.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918000	ALPL	GG	normal

RS121918013	ALPL	GG	normal
RS121918014	ALPL	AA	normal

Test details

Applicable situation

Genetic disease screening test for Hypophosphatasia (HPP) uses 3 polymorphic loci on the *ALPL* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hypophosphatasia (HPP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hypophosphatasia (HPP), or if you feel that you have a risk of developing Hypophosphatasia (HPP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Hypophosphatasia (HPP), such as rs121918000, rs121918013 and rs121918014.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hypophosphatasia (HPP).

Brachydactyly

It is a genetic disorder that affects the development of fingers and toes, resulting in shorter or missing bones in the hands and feet.

My Brachydactyly
Non-carrier

Knowledge



Time of onset

Symptoms of brachydactyly are usually evident at birth or early childhood.



Prevalence

Brachydactyly is a rare condition, affecting fewer than 1 percent of the population worldwide.



Typical symptoms

The most common symptom of it is abnormally short fingers or toes. In severe cases, the affected digits may be missing entirely.



Treatment

Treatment for it is typically focused on managing symptoms and addressing functional impairments. This may involve physical therapy, adaptive equipment, or surgery to improve hand or foot function.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS28928891	HOXD13	AA	normal
RS28936397	GDF5	TT	normal
RS1014959895	intergenic	GG	normal

RS1251713297	intergenic	CC	normal
RS863225094	PPP2R1A	GG	normal

Test details

Applicable situation

Genetic disease screening test for Brachydactyly uses 5 polymorphic loci on genes such as *PRMT7*, *HOXD13* and *GDF5*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Brachydactyly.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Brachydactyly, or if you feel that you have a risk of developing Brachydactyly, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Brachydactyly, such as rs1014959895, rs1251713297 and rs28928891.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Brachydactyly.

Multiple Sulfatase Deficiency (MSD)

It is a rare genetic metabolic disease. Due to the congenital deficiency of aryl sulfatase A, B and C, sulfolipids, mucopolysaccharides, sulfate steroids, gangliosides, etc. are stored in the cerebral cortex and visceral tissues.

My Multiple Sulfatase Deficiency (MSD)

Non-carrier

Knowledge



Time of onset

It typically appears in infancy or early childhood.



Prevalence

It is an extremely rare disease, with fewer than 100 known cases reported worldwide.



Typical symptoms

Typical symptoms include developmental delay, seizures, vision and hearing problems, intellectual disability, and an enlarged liver and spleen.



Treatment

Treatment is focused on managing symptoms, with various medications and therapies used to address specific issues such as seizures, vision problems, and cognitive impairment.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137852847	SUMF1	CC	normal
RS137852849	SUMF1	GG	normal

RS770241913

intergenic

II

normal

Test details

Applicable situation

Genetic disease screening test for Multiple Sulfatase Deficiency (MSD) uses 3 polymorphic loci on the *SUMF1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Multiple Sulfatase Deficiency (MSD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Multiple Sulfatase Deficiency (MSD), or if you feel that you have a risk of developing Multiple Sulfatase Deficiency (MSD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Multiple Sulfatase Deficiency (MSD), such as rs137852847, rs137852849 and rs770241913.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Multiple Sulfatase Deficiency (MSD).

Polycystic Kidney Disease (PKD)

It is a genetic disorder that causes cysts to form on kidneys, leading to enlarged kidneys, reduced kidney function and possibly kidney failure.

My Polycystic Kidney Disease (PKD)

Non-carrier

Knowledge

Time of onset

It can be present at birth or may develop during adulthood.

Prevalence

It is estimated that the incidence of this disease is fewer than 5/10000.

Typical symptoms

Typical symptoms include pain in the back and sides, frequent urination and high blood pressure. Some people may also experience blood in their urine, urinary tract infections and kidney stones.

Treatment

Treatment options include managing blood pressure, pain relief and kidney function monitoring. In cases where kidney function is severely reduced, dialysis or kidney transplantation may be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137852947	PKHD1	GG	normal
RS1240212722	intergenic	TT	normal
RS797045101	intergenic	II	normal

RS797044745	PKHD1	DD	normal
RS797044713	PKHD1	DD	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Polycystic Kidney Disease (PKD) uses 33 polymorphic loci on the *PKHD1*, *SHOC2* and *PKD2* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Polycystic Kidney Disease (PKD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Polycystic Kidney Disease (PKD), or if you feel that you have a risk of developing Polycystic Kidney Disease (PKD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 33 polymorphic loci associated with the risk of Polycystic Kidney Disease (PKD), such as rs1240212722, rs137852947 and rs148617572.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Polycystic Kidney Disease (PKD).

Congenital Multicore Myopathy with External Ophthalmoplegia

It is a rare genetic disease, characterized by muscle weakness and wasting, as well as difficulty moving the eyes.

My Congenital Multicore Myopathy with External Ophthalmoplegia

Non-carrier

Knowledge



Time of onset

It is present from birth, although symptoms may not become apparent until later in childhood or early adulthood.



Prevalence

It is a rare disease, with an estimated prevalence of 1 in 50,000 to 100,000 individuals.



Typical symptoms

Typical symptoms of this disease include muscle weakness and wasting, difficulty moving the eyes, drooping of the eyelids, and difficulty breathing. In addition to the symptoms mentioned above, individuals with this disease may also experience scoliosis, joint contractures, and kyphosis. Over time, the disease can lead to respiratory failure and necessitate assisted breathing.



Treatment

Currently, there is no cure for this disease, and treatment is aimed at managing symptoms. Physical therapy, respiratory monitoring, and orthopedic interventions can help to preserve movement and prevent respiratory complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS118192150	RYR1	CC	normal
RS118192176	RYR1	GG	normal
RS121918596	RYR1	II	normal
RS193922781	RYR1	CC	normal
RS28933397	RYR1	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Congenital Multicore Myopathy with External Ophthalmoplegia uses 13 polymorphic loci on the *RYR1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Congenital Multicore Myopathy with External Ophthalmoplegia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Congenital Multicore Myopathy with External Ophthalmoplegia, or if you feel that you have a risk of developing Congenital Multicore Myopathy with External

Ophthalmoplegia, it is recommended that you consult a qualified health professional or clinician for help.

 Test content

This test includes 13 polymorphic loci associated with the risk of Congenital Multicore Myopathy with External Ophthalmoplegia, such as rs1057524858, rs113928116 and rs118192150.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Congenital Multicore Myopathy with External Ophthalmoplegia.

Multisystemic Smooth Muscle Dysfunction Syndrome (MSMDS)

It is a rare genetic disorder that affects the smooth muscles in various organs of the body, leading to a range of gastrointestinal, respiratory and vascular problems.

My Multisystemic Smooth Muscle Dysfunction Syndrome (MSMDS)

Non-carrier

Knowledge



Time of onset

The onset of symptoms can occur at any age, although it is most commonly diagnosed in childhood.



Prevalence

It is estimated to be very rare, with only a few dozen cases reported in medical literature.



Typical symptoms

Typical symptoms include difficulty swallowing, aspiration pneumonia, chest pain, shortness of breath, abdominal pain, constipation, diarrhea, and frequent urination.



Treatment

Treatment may include providing nutrition support, controlling hypertension, and preventing vascular complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS387906592	ACTA2	CC	normal

RS886039303

ACTA2

GG

normal

Test details

Applicable situation

Genetic disease screening test for Multisystemic Smooth Muscle Dysfunction Syndrome (MSMDS) uses 2 polymorphic loci on the *ACTA2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Multisystemic Smooth Muscle Dysfunction Syndrome (MSMDS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Multisystemic Smooth Muscle Dysfunction Syndrome (MSMDS), or if you feel that you have a risk of developing Multisystemic Smooth Muscle Dysfunction Syndrome (MSMDS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Multisystemic Smooth Muscle Dysfunction Syndrome (MSMDS), such as rs387906592 and rs886039303.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Multisystemic Smooth Muscle Dysfunction Syndrome (MSMDS).

Frontometaphyseal Dysplasia (FMD)

It is a rare genetic disorder characterized by abnormal bone development, particularly in the face, skull, and long bones of the body.

My Frontometaphyseal Dysplasia (FMD)

Non-carrier

Knowledge



Time of onset

It is a congenital condition that is present at birth and symptoms may become apparent during infancy or childhood.



Prevalence

It is extremely rare and the exact prevalence is not known. It is estimated to affect fewer than 1 in 1,000,000 people worldwide.



Typical symptoms

Typical symptoms may include pronounced forehead, wide-set eyes, a small nose with flat bridge, and a narrow jaw. The long bones of the body may have abnormal swelling or thickening.



Treatment

There is no cure for this disease. Treatment is supportive and aims to alleviate symptoms, with physiotherapy and orthopaedic care being the main focus. Surgical treatment is required in severe cases. Regular follow-up sessions with a clinical geneticist may help manage the condition.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137853312	FLNA	GG	normal

RS797045044	FLNA	CC	normal
RS863223630	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Frontometaphyseal Dysplasia (FMD) uses 3 polymorphic loci on the *FLNA* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Frontometaphyseal Dysplasia (FMD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Frontometaphyseal Dysplasia (FMD), or if you feel that you have a risk of developing Frontometaphyseal Dysplasia (FMD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Frontometaphyseal Dysplasia (FMD), such as rs137853312, rs797045044 and rs863223630.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Frontometaphyseal Dysplasia (FMD).

Otospondylomegaepiphyseal Dysplasia (OSMED)

It is a rare genetic disorder that affects bone and cartilage development, leading to short stature, hearing impairment, and joint problems.

My Otospondylomegaepiphyseal Dysplasia (OSMED)

Non-carrier

Knowledge



Time of onset

It typically appears in infancy or early childhood and progressively worsen with age.



Prevalence

It is estimated to affect fewer than 1 in 1,000,000 individuals.



Typical symptoms

Typical symptoms include short stature, hearing loss, joint stiffness, and eye problems.



Treatment

Treatment may include using hearing aids or undergoing joint surgery. Patients may also benefit from physical therapy and regular monitoring of bone health.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121912945	COL11A2	CC	normal

Test details

Applicable situation

Genetic disease screening test for Otospondylomegaepiphyseal Dysplasia (OSMED) uses 1 polymorphic locus on the *COL11A2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Otospondylomegaepiphyseal Dysplasia (OSMED).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Otospondylomegaepiphyseal Dysplasia (OSMED), or if you feel that you have a risk of developing Otospondylomegaepiphyseal Dysplasia (OSMED), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Otospondylomegaepiphyseal Dysplasia (OSMED), namely rs121912945.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Otospondylomegaepiphyseal Dysplasia (OSMED).

Developmental and Epileptic Encephalopathy (DEE)

It is a neurological disorder characterized by developmental delay, seizures, and intellectual disabilities.

My Developmental and Epileptic Encephalopathy (DEE)

Non-carrier

Knowledge



Time of onset

The varies depending on the type of this disease, and can occur from infancy to early childhood.



Prevalence

It is a rare disease, affecting about 1 in 2,500 people.



Typical symptoms

The symptoms of this disease include developmental delay, severe intellectual disability, and seizures that are often refractory to treatment.



Treatment

The treatment methods vary depending on the type and severity of the disease, and may include drugs to control epilepsy, physical therapy to improve motor function, and speech therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS118192215	KCNQ2	GG	normal
RS796052862	intergenic	DD	normal

RS796053181	SCN2A	TT	normal
RS796053180	SCN2A	GG	normal
RS796053178	SCN2A	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Developmental and Epileptic Encephalopathy (DEE) uses 119 polymorphic loci on genes such as *KCNQ2*, *PCDH19* and *SCN2A*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Developmental and Epileptic Encephalopathy (DEE).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Developmental and Epileptic Encephalopathy (DEE), or if you feel that you have a risk of developing Developmental and Epileptic Encephalopathy (DEE), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 119 polymorphic loci associated with the risk of Developmental and Epileptic Encephalopathy (DEE), such as rs1057516095, rs1057516098 and rs1060502176.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Developmental and Epileptic Encephalopathy (DEE).

Maple Syrup Urine Disease (MSUD)

It is a rare metabolic disorder named after the unique sweet scent of maple syrup in the urine of children. It is caused by deficiencies in the metabolizing enzymes of the body's branched-chain amino acids (leucine, isoleucine and valine), leading to medical problems such as brain damage, seizures, coma and death.

My Maple Syrup Urine Disease (MSUD)

Non-carrier

Knowledge

Time of onset

It typically appears within a few days after birth.

Prevalence

It is estimated to affect 1 in 185,000 newborns worldwide.

Typical symptoms

The most common symptoms include sweet odor in patient's urine or sweat, poor feeding, lethargy, vomiting and irregular sleep patterns. Severe neurological damage and developmental disabilities may occur if left untreated.

Treatment

Early diagnosis is important. This disease is managed through a low-protein diet, sometimes in combination with special supplements, medication, and other interventions such as dialysis.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121964999	DBT	AA	normal

RS398123676	DBT	CC	normal
RS796051938	intergenic	II	normal
RS768832921	intergenic	II	normal
RS727503895	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Maple Syrup Urine Disease (MSUD) uses 33 polymorphic loci on the *DBT*, *BCKDHA* and *BCKDHB* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Maple Syrup Urine Disease (MSUD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Maple Syrup Urine Disease (MSUD), or if you feel that you have a risk of developing Maple Syrup Urine Disease (MSUD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 33 polymorphic loci associated with the risk of Maple Syrup Urine Disease (MSUD), such as rs121964999, rs137852871 and rs137852874.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Maple Syrup Urine Disease (MSUD).

Combined Oxidative Phosphorylation Deficiency (COXPD)

It is a rare mitochondrial disease caused by defects in mitochondrial protein synthesis.

My Combined Oxidative Phosphorylation Deficiency (COXPD)

Non-carrier

Knowledge



Time of onset

The for this disease varies from birth to early childhood.



Prevalence

It is a rare disease, affecting an estimated 1 in 100,000 individuals worldwide.



Typical symptoms

The typical symptoms of this disease include developmental delays, hypotonia (weak muscle tone), seizures, and difficulty feeding or gaining weight.



Treatment

For this disease are limited, and management typically focuses on controlling symptoms and maximizing quality of life. Treatment may include medications to manage seizures or lactic acidosis, as well as nutritional support to manage feeding difficulties.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1370579526	intergenic	TT	normal

RS1562168768	intergenic	GG	normal
RS184469579	TRIT1	GG	normal
RS201229537	MRPS2	GG	normal
RS376766195	intergenic	AA	normal

Test details

Applicable situation

Genetic disease screening test for Combined Oxidative Phosphorylation Deficiency (COXPD) uses 5 polymorphic loci on genes such as *GATC*, *QRSL1* and *TRIT1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Combined Oxidative Phosphorylation Deficiency (COXPD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Combined Oxidative Phosphorylation Deficiency (COXPD), or if you feel that you have a risk of developing Combined Oxidative Phosphorylation Deficiency (COXPD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Combined Oxidative Phosphorylation Deficiency (COXPD), such as rs1370579526, rs1562168768 and rs184469579.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Combined Oxidative Phosphorylation Deficiency (COXPD).

Nemaline Myopathy (NM)

It is a rare genetic disorder characterized by the presence of rod-like nemaline bodies in skeletal muscle fibers, resulting in muscle weakness and poor muscle tone.

My Nemaline Myopathy (NM)

Non-carrier

Knowledge



Time of onset

This disease can affect people at different ages but is usually present at birth or during early childhood.



Prevalence

It is estimated to affect 1 in 50,000 individuals.



Typical symptoms

Symptoms can vary in severity but generally include poor muscle tone, muscle weakness and difficulty in breathing. In addition, significant motor impairment is likely to be seen.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support, for example, physical therapy, respiratory support and other supportive measures.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121964854	TPM3	GG	normal
RS398124167	intergenic	CC	normal

RS398124172	intergenic	II	normal
RS769345284	NEB	GG	normal
RS773952935	intergenic	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Nemaline Myopathy (NM) uses 9 polymorphic loci on the *TPM3* and *NEB* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Nemaline Myopathy (NM).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Nemaline Myopathy (NM), or if you feel that you have a risk of developing Nemaline Myopathy (NM), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 9 polymorphic loci associated with the risk of Nemaline Myopathy (NM), such as rs121964854, rs398124167 and rs398124172.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Nemaline Myopathy (NM).

Metaphyseal Dysplasia without Hypotrichosis (MDWH)

It is a rare autosomal recessive skeletal dysplasia. Hair is normal in most patients. Some patients develop later-onset extraskeletal manifestations including immunodeficiency and malignancy.

My Metaphyseal Dysplasia without Hypotrichosis (MDWH)

Non-carrier

Knowledge



Time of onset

This disease is present at birth and becomes more apparent as a child grows.



Prevalence

The prevalence of this disease is currently unknown.



Typical symptoms

Common symptoms are short stature, bowed legs, and an abnormal gait. Individuals may also have joint pain and stiffness, as well as an increased risk for developing osteoarthritis.



Treatment

Treatment may include braces or surgery to correct skeletal abnormalities, as well as physical therapy to improve mobility and reduce joint pain.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS786204684	RMRP	GG	normal

Test details

Applicable situation

Genetic disease screening test for Metaphyseal Dysplasia without Hypotrichosis (MDWH) uses 1 polymorphic locus on the *RMRP* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Metaphyseal Dysplasia without Hypotrichosis (MDWH).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Metaphyseal Dysplasia without Hypotrichosis (MDWH), or if you feel that you have a risk of developing Metaphyseal Dysplasia without Hypotrichosis (MDWH), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Metaphyseal Dysplasia without Hypotrichosis (MDWH), namely rs786204684.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Metaphyseal Dysplasia without Hypotrichosis (MDWH).

Homocystinuria (HCU)

It is a rare genetic disorder that affects the body's metabolism of an amino acid called methionine. People with this condition cannot properly break down methionine, which leads to a buildup of a substance called homocysteine in their blood and urine. High levels of homocysteine can cause a variety of health problems, including abnormal bone development, eye problems, and an increased risk of blood clots and heart disease.

My Homocystinuria (HCU)

Non-carrier

Knowledge



Time of onset

It is usually present from birth, although it may not be diagnosed until later in life.



Prevalence

It is a very rare disease, with an estimated of around 1 in 200,000 to 335,000 people worldwide.



Typical symptoms

The typical symptoms of it include tall stature, long limbs, and a narrow face. People with the condition may also have intellectual disability, seizures, and vision problems. may experience fatigue, weakness, and skeletal abnormalities such as scoliosis.



Treatment

Treatment for it typically involves a low-methionine diet, along with vitamin supplements to help the body break down homocysteine. Some people with the condition may need to take medication to help lower their homocysteine levels. Regular monitoring of homocysteine levels is also important to detect any potential health problems early on.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121964962	CBS	CC	normal
RS121964973	CBS	GG	normal
RS777919630	intergenic	GG	normal
RS778220779	intergenic	AA	normal
RS781444670	intergenic	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Homocystinuria (HCU) uses 13 polymorphic loci on the *CBS* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Homocystinuria (HCU).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Homocystinuria (HCU), or if you feel that you have a risk of developing Homocystinuria (HCU), it is recommended that you consult a qualified health

professional or clinician for help.

 Test content

This test includes 13 polymorphic loci associated with the risk of Homocystinuria (HCU), such as rs121964962, rs121964973 and rs1347651454.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Homocystinuria (HCU).

Hyperphosphatasia with Intellectual Disability Syndrome

Also known as Mabry syndrome, is a condition characterized by intellectual disability, distinctive facial features, increased levels of an enzyme called alkaline phosphatase in the blood (hyperphosphatasia), and other signs and symptoms.

My Hyperphosphatasia with Intellectual Disability Syndrome

Non-carrier

Knowledge



Time of onset

Suffering from hyperphosphatase within the first year of birth.



Prevalence

This disease is a rare disease, but its incidence is still unclear. More than 20 cases have been described in scientific literature.



Typical symptoms

Patients typically have moderate to severe intellectual disabilities. They usually have almost no language development and slow development of motor skills such as sitting, crawling, and walking. Many affected individuals experience hypotonia and recurrent seizures in early childhood. At the same time, the bone at the end of the finger is shortened (short phalangeal deformity), which may also lead to underdeveloped nails. Sometimes patients may experience digestive system abnormalities, including anal stenosis, anal atresia, or congenital megacolon, which is a disease that causes severe constipation or intestinal obstruction. In rare cases, affected individuals may experience hearing loss.



Treatment

There is currently no cure or specific treatment for this syndrome, but management typically involves supportive care and addressing individual symptoms. This may include therapies such as physical, occupational, or speech therapy, medications for seizures.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS139073416	PIGV	CC	normal
RS770591449	PIGO	DD	normal

Test details

Applicable situation

Genetic disease screening test for Hyperphosphatasia with Intellectual Disability Syndrome uses 2 polymorphic loci on the *PIGV* and *PIGO* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hyperphosphatasia with Intellectual Disability Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hyperphosphatasia with Intellectual Disability Syndrome, or if you feel that you have a risk of developing Hyperphosphatasia with Intellectual Disability Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Hyperphosphatasia with Intellectual Disability Syndrome, such as rs139073416 and rs770591449.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hyperphosphatasia with Intellectual Disability Syndrome.

Hyperornithinemia-Hyperammonemia-Homocitrullinuria Syndrome (HHH)

It is a rare genetic disorder that affects the metabolism of the amino acids ornithine, ammonia, and citrulline. It is caused by a defect in the transport of ornithine inside mitochondria.

My Hyperornithinemia-Hyperammonemia-Homocitrullinuria Syndrome (HH)

Non-carrier

Knowledge

Time of onset

Symptoms of it typically appear in infancy, often within the first few days or weeks of life.

Prevalence

It is an extremely rare disorder, with only a few hundred cases reported worldwide.

Typical symptoms

Common symptoms of it include elevated ammonia levels in the blood (hyperammonemia), developmental delay, seizures, spasticity, and intellectual disability.

Treatment

Treatment for it is currently limited to managing symptoms and preventing complications. This may include a protein-restricted diet, medications to control ammonia levels, and physical and occupational therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS104894424	SLC25A15	GG	normal
RS104894429	intergenic	CC	normal
RS104894430	SLC25A15	GG	normal
RS121908533	intergenic	TT	normal
RS121908535	SLC25A15	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Hyperornithinemia-Hyperammonemia-Homocitrullinuria Syndrome (HHH) uses 9 polymorphic loci on the *SLC25A15* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hyperornithinemia-Hyperammonemia-Homocitrullinuria Syndrome (HHH).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hyperornithinemia-Hyperammonemia-Homocitrullinuria Syndrome (HHH), or if you feel that you have a risk of developing Hyperornithinemia-Hyperammonemia-

Homocitrullinuria Syndrome (HHH), it is recommended that you consult a qualified health professional or clinician for help.

 Test content

This test includes 9 polymorphic loci associated with the risk of Hyperornithinemia-Hyperammonemia-Homocitrullinuria Syndrome (HHH), such as rs104894424, rs104894429 and rs104894430.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hyperornithinemia-Hyperammonemia-Homocitrullinuria Syndrome (HHH).

Hyperinsulinemic Hypoglycemia (HH)

It is a rare medical condition characterized by excessively low levels of blood sugar or glucose in the body due to high insulin levels. It occurs due to the genetic mutation that makes insulin-producing cells in the pancreas overactive, resulting in the overproduction of insulin.

My Hyperinsulinemic Hypoglycemia (HH)

Non-carrier

Knowledge

Time of onset

It can occur at any age, but most commonly presents in newborns or infants.

Prevalence

It is a rare condition, with an estimated incidence of 1 in 50,000 to 1 in 250,000 live births.

Typical symptoms

The typical symptoms of it include seizures, lethargy, pallor, sweating, irritability, and poor feeding.

Treatment

The treatment of it involves reducing the levels of insulin in the body. This is often done through medications, such as diazoxide or octreotide, or surgery to remove the affected portion of the pancreas. In severe cases, a pancreas transplant may be required.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137852671	ABCC8	CC	normal
RS139328569	intergenic	GG	normal

RS139964066	intergenic	GG	normal
RS200670692	intergenic	AA	normal
RS372307320	ABCC8	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Hyperinsulinemic Hypoglycemia (HH) uses 11 polymorphic loci on the *ABCC8* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hyperinsulinemic Hypoglycemia (HH).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hyperinsulinemic Hypoglycemia (HH), or if you feel that you have a risk of developing Hyperinsulinemic Hypoglycemia (HH), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 11 polymorphic loci associated with the risk of Hyperinsulinemic Hypoglycemia (HH), such as rs137852671, rs139328569 and rs139964066.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hyperinsulinemic Hypoglycemia (HH).

Kabuki Syndrome (KS)

It is a rare multi-systemic disorder characterized by craniofacial anomalies, intellectual disability, postnatal growth retardation, and various organ malformations.

My Kabuki Syndrome (KS)

Non-carrier

Knowledge

Time of onset

It is typically diagnosed in infancy or early childhood, but symptoms may not become apparent until later in life.

Prevalence

It is estimated to affect 1 in 32,000 to 86,000 individuals worldwide.

Typical symptoms

Common symptoms include distinct facial features, developmental delays, intellectual disability, skeletal abnormalities, short stature, abnormalities of the eyes and vision, cleft lip and/or palate, seizures, gastrointestinal problems, and hormonal imbalances. Individuals with the condition may also experience hearing loss, heart defects, and immune system dysfunction.

Treatment

Treatment is generally supportive and may include a range of therapies and interventions tailored to address an individual's specific symptoms and needs. These may include speech and developmental therapies, surgery, hearing aids, and medications to manage seizures, immune system dysfunction, and other related conditions.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS267607237	KMT2D	CC	normal
RS794727549	KMT2D	GG	normal
RS587783713	intergenic	II	normal
RS587783714	intergenic	CC	normal
RS587783715	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Kabuki Syndrome (KS) uses 49 polymorphic loci on the *KMT2D* and *KDM6A* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Kabuki Syndrome (KS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Kabuki Syndrome (KS), or if you feel that you have a risk of developing Kabuki Syndrome (KS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 49 polymorphic loci associated with the risk of Kabuki Syndrome (KS), such as rs267607237, rs398122929 and rs398123704.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Kabuki Syndrome (KS).

Ataxia-Telangiectasia (AT)

It is a rare, genetic, progressive neurodegenerative disorder that primarily affects children. It is characterized by a combination of movement problems and weakened immune system, increasing the risk of infections and cancer.

My Ataxia-Telangiectasia (AT)

Non-carrier

Knowledge



Time of onset

Onset typically occurs in early childhood, usually before the age of 5.



Prevalence

It is a rare disorder, with an estimated of 1 in 40,000 to 1 in 100,000 individuals worldwide.



Typical symptoms

The typical symptoms of this disease include abnormal eye movements, deterioration of body movements and coordination over time, resulting in unclear and irregular speech, as well as difficulty swallowing. The weakened immune system, frequent respiratory infections, and increased risk of developing various types of cancer.



Treatment

While there is currently no cure for Ataxia-telangiectasia, treatment focuses on managing symptoms and preventing complications. This may include genetic counseling, physical therapy, speech therapy, antibiotics and other medications to prevent infections and treat symptoms of cancer, and other supportive care.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121434219	ATM	CC	normal
RS786201693	ATM	CC	normal
RS774925473	intergenic	AA	normal
RS775036118	intergenic	CC	normal
RS777849257	ATM	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Ataxia-Telangiectasia (AT) uses 178 polymorphic loci on the *ATM* and *BRAF* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Ataxia-Telangiectasia (AT).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Ataxia-Telangiectasia (AT), or if you feel that you have a risk of developing Ataxia-Telangiectasia (AT), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 178 polymorphic loci associated with the risk of Ataxia-Telangiectasia (AT), such as rs1057520640, rs1060501687 and rs1060501700.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Ataxia-Telangiectasia (AT).

Osteopetrosis

It is a rare hereditary disease. It is caused by the imbalance of the mechanism of bone regeneration and destruction due to the functional defect of osteoclast, resulting in bone marrow occlusion, bone fragility, nerve compression due to narrowing of bone spaces, and abnormal hematopoietic function.

My Osteopetrosis
Non-carrier

Knowledge



Time of onset

The onset varies depending on the severity of the disease. Some cases are apparent at birth, whereas others may not present until later in life.



Prevalence

It is estimated to affect 1 in 100,000-500,000 individuals.



Typical symptoms

Typical symptoms include bone pain, fractures, short stature, dental abnormalities and vision or hearing loss. Some individuals may also experience neurologic symptoms such as blindness, deafness or paralysis.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support. Pain relief, orthopedic interventions and hearing aids may improve quality of life. In some cases, bone marrow transplantation may be considered as a potential treatment option.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121908670	LRP5	GG	normal
RS387907576	CLCN7	TT	normal
RS751881962	TCIRG1	AA	normal

Test details

Applicable situation

Genetic disease screening test for Osteopetrosis uses 3 polymorphic loci on the *LRP5*, *CLCN7* and *TCIRG1* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Osteopetrosis.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Osteopetrosis, or if you feel that you have a risk of developing Osteopetrosis, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Osteopetrosis, such as rs121908670, rs387907576 and rs751881962.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Osteopetrosis.

Citrullinemia

It is a rare genetic disorder characterized by a deficiency of the enzyme argininosuccinate synthetase (AS), which is responsible for the conversion of citrulline into argininosuccinate in the urea cycle. As a result, toxic levels of ammonia can accumulate in the blood and cause brain damage, neurological symptoms, and liver dysfunction.

My Citrullinemia
Non-carrier

Knowledge



Time of onset

It presents in infancy, but some cases may present later in childhood or adulthood.



Prevalence

The incidence of it is estimated to be 1 in 57,000 births worldwide.



Typical symptoms

The symptoms of this disease can vary widely and may include elevated levels of ammonia in the blood, elevated levels of citrulline in the urine, liver damage, and neurological symptoms such as seizures, coma, and ataxia. In severe cases, the disease can lead to death.



Treatment

Treatment for it typically involves a low-protein diet, medications to remove excess ammonia and increase arginine levels, and in some cases, liver transplantation.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121908638	ASS1	GG	normal

RS121908647	ASS1	GG	normal
RS148918985	ASS1	CC	normal
RS192838388	ASS1	GG	normal
RS371265106	ASS1	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Citrullinemia uses 7 polymorphic loci on the *ASS1* and *SLC25A13* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Citrullinemia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Citrullinemia, or if you feel that you have a risk of developing Citrullinemia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 7 polymorphic loci associated with the risk of Citrullinemia, such as rs121908638, rs121908647 and rs148918985.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Citrullinemia.

Popliteal Pterygium Syndrome (PPS)

It is a rare genetic disorder characterized by webbing of the skin on the back of the knees, which can cause limited movement, as well as various other malformations.

My Popliteal Pterygium Syndrome (PPS)

Non-carrier

Knowledge



Time of onset

It typically appears at birth.



Prevalence

It is estimated to affect 1 in 300,000 to 1,000,000 people worldwide.



Typical symptoms

The main symptom is webbing of the skin on the back of the knees, which can cause limited movement. Other symptoms may include cleft lip and/or palate, abnormal genitalia and malformations of the fingers and toes.



Treatment

Treatment mainly involves improving symptoms and may involve plastic surgery to correct the webbing of the skin on the back of the knees, or bracing for scoliosis. Specialized care may also be needed for cleft lip and palate, hearing loss or other malformations.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121434226	IRF6	GG	normal

RS769068305

IRF6

CC

normal

Test details

Applicable situation

Genetic disease screening test for Popliteal Pterygium Syndrome (PPS) uses 2 polymorphic loci on the *IRF6* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Popliteal Pterygium Syndrome (PPS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Popliteal Pterygium Syndrome (PPS), or if you feel that you have a risk of developing Popliteal Pterygium Syndrome (PPS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Popliteal Pterygium Syndrome (PPS), such as rs121434226 and rs769068305.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Popliteal Pterygium Syndrome (PPS).

Alkaptonuria

It is a rare genetic disease that affects the body's ability to break down tyrosine and phenylalanine. This leads to the accumulation of a substance called uric acid in the body, which over time may lead to joint and cartilage problems, heart problems, and blackening of the skin and urine.

My Alkaptonuria
Non-carrier

Knowledge

Time of onset

For symptoms of it typically appears in early childhood or adolescence, although some individuals may not experience symptoms until adulthood.

Prevalence

Alkaptonuria is a rare disease, affecting only about 1 in 250,000 to 1 in 1,000,000 individuals worldwide.

Typical symptoms

The typical symptoms of this disease include blackening of urine and skin, joint pain and stiffness, and heart problems. In addition, urinary melanin deposition in the eyes, ears, and nose can cause visual and hearing problems, as well as kidney problems such as stones.

Treatment

Treatment for alkaptonuria is mostly supportive and focused on managing symptoms. This can include joint replacement surgery, medication for pain and inflammation, and regular monitoring of heart and kidney function.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS120074173	HGD	TT	normal
RS120074174	HGD	CC	normal
RS28942100	HGD	GG	normal

Test details

Applicable situation

Genetic disease screening test for Alkaptonuria uses 3 polymorphic loci on the *HGD* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Alkaptonuria.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Alkaptonuria, or if you feel that you have a risk of developing Alkaptonuria, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Alkaptonuria, such as rs120074173, rs120074174 and rs28942100.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Alkaptonuria.

Myofibrillar Myopathy (MFM)

It is a rare genetic disorder that is caused by mutations in specific genes, leading to the breakdown of the structure of muscle cells.

My Myofibrillar Myopathy (MFM)

Non-carrier

Knowledge

Time of onset

Some individuals may develop symptoms during childhood, while others may not develop any symptoms until later in life.

Prevalence

This disease is considered rare, the prevalence is unknown.

Typical symptoms

Typical symptoms include muscle weakness, loss of muscle mass and difficulty with movement. Other symptoms may include difficulty in swallowing, respiratory problems and eye movement problems.

Treatment

Treatment typically involves managing symptoms and addressing complications as they arise. This may include physical therapy, respiratory support and medication to manage cardiac symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121913001	DES	AA	normal
RS267607485	DES	AA	normal

RS1562999451	intergenic	AA	normal
RS1563001456	intergenic	AA	normal
RS1564767043	intergenic	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Myofibrillar Myopathy (MFM) uses 38 polymorphic loci on the *BAG3*, *FLNC* and *DES* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Myofibrillar Myopathy (MFM).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Myofibrillar Myopathy (MFM), or if you feel that you have a risk of developing Myofibrillar Myopathy (MFM), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 38 polymorphic loci associated with the risk of Myofibrillar Myopathy (MFM), such as rs1057517945, rs1064795229 and rs1114167361.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Myofibrillar Myopathy (MFM).

Diastrophic Dysplasia

It is a disorder of cartilage and bone development. Affected individuals have short stature with very short arms and legs. Most also have early-onset joint pain (osteoarthritis) and joint deformities called contractures, which restrict movement. These joint problems often make it difficult to walk and tend to worsen with age.

My Diastrophic Dysplasia

Non-carrier

Knowledge



Time of onset

It is present at birth and symptoms can be seen soon after.



Prevalence

It is a rare disorder that affects approximately 1 in 100,000 to 200,000 newborns worldwide.



Typical symptoms

The typical symptoms of diastrophic dysplasia include short stature, joint pain and stiffness, scoliosis, clubfoot, and deformities of the hands and feet.



Treatment

Treatment for it is symptomatic and supportive. Physical therapy, orthopedic appliances, and surgical intervention may be used to manage the physical symptoms associated with this disorder. In some cases, treatment with growth hormone may be beneficial.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893916	SLC26A2	GG	normal

RS786200881

intergenic

II

normal

Test details

Applicable situation

Genetic disease screening test for Diastrophic Dysplasia uses 2 polymorphic loci on the *SLC26A2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Diastrophic Dysplasia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Diastrophic Dysplasia, or if you feel that you have a risk of developing Diastrophic Dysplasia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Diastrophic Dysplasia, such as rs104893916 and rs786200881.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Diastrophic Dysplasia.

Spinal Muscular Atrophy (SMA)

It is a genetic disorder, characterized by a reduction in the amount of survival motor neuron protein, leading to degeneration of neurons in the spinal cord and brainstem.

My Spinal Muscular Atrophy (SMA)

Non-carrier

Knowledge



Time of onset

The onset is typically in early childhood.



Prevalence

It is considered rare, with only a few reported cases in the medical literature.



Typical symptoms

Typical symptoms include muscle weakness, difficulty walking, and delayed motor milestones.



Treatment

Treatment may include physical therapy, respiratory support, and orthopedic interventions.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137852665	IGHMBP2	GG	normal
RS139497493	IGHMBP2	CC	normal
RS372000714	IGHMBP2	TT	normal
RS780136067	SIGMAR1	DD	normal

Test details



Applicable situation

Genetic disease screening test for Spinal Muscular Atrophy (SMA) uses 5 polymorphic loci on the *IGHMBP2* and *SIGMAR1* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.



Notification

1. The test may not cover all the pathogenic genes or loci of Spinal Muscular Atrophy (SMA).
 2. The test results are not applicable to clinical practice.
 3. If you have clinical needs, please contact a qualified institution or clinician.
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How to use the test results



Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Spinal Muscular Atrophy (SMA), or if you feel that you have a risk of developing Spinal Muscular Atrophy (SMA), it is recommended that you consult a qualified health professional or clinician for help.



Test content

This test includes 5 polymorphic loci associated with the risk of Spinal Muscular Atrophy (SMA), such as rs137852665, rs139497493 and rs372000714.



Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Spinal Muscular Atrophy (SMA).

Spondyloperipheral Dysplasia (SPD)

It is a rare genetic disorder that affects the development of bones and joints.

My Spondyloperipheral Dysplasia (SPD)
Non-carrier

Knowledge



Time of onset

It typically appears shortly after birth or within the first year of life.



Prevalence

Only a few cases have been reported in medical literature.



Typical symptoms

Typical symptoms include short stature, abnormal curvature of the spine, joint stiffness, and facial anomalies.



Treatment

Generally, physical therapy, braces, and surgery may be recommended to prevent further deformity and/or to correct any existing deformities.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121912874	COL2A1	GG	normal
RS121912893	COL2A1	GG	normal
RS794727261	COL2A1	GG	normal

Test details

Applicable situation

Genetic disease screening test for Spondyloperipheral Dysplasia (SPD) uses 3 polymorphic loci on the *COL2A1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Spondyloperipheral Dysplasia (SPD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Spondyloperipheral Dysplasia (SPD), or if you feel that you have a risk of developing Spondyloperipheral Dysplasia (SPD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Spondyloperipheral Dysplasia (SPD), such as rs121912874, rs121912893 and rs794727261.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Spondyloperipheral Dysplasia (SPD).

Spondylocarpotarsal Synostosis Syndrome (SCT)

It is a rare genetic disorder that affects the development of bones in the spine, wrist, and ankle. This condition is characterized by spinal fusion, carpal and tarsal coalition, and short stature.

My Spondylocarpotarsal Synostosis Syndrome (SCT)

Non-carrier

Knowledge

 Time of onset

The symptoms usually become apparent in early childhood.

 Prevalence

The prevalence of this disease is currently unknown.

 Typical symptoms

The most common symptoms include spinal fusion, carpal and tarsal coalition, short stature, and a distinctive facial appearance.

 Treatment

Treatment is generally symptomatic and may include physical therapy, surgery to address spinal or joint fusion, and pain management.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS80356517	FLNB	CC	normal

RS1555527166

intergenic

II

normal

Test details

Applicable situation

Genetic disease screening test for Spondylocarpotarsal Synostosis Syndrome (SCT) uses 2 polymorphic loci on the *MYH3* and *FLNB* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Spondylocarpotarsal Synostosis Syndrome (SCT).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Spondylocarpotarsal Synostosis Syndrome (SCT), or if you feel that you have a risk of developing Spondylocarpotarsal Synostosis Syndrome (SCT), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Spondylocarpotarsal Synostosis Syndrome (SCT), such as rs1555527166 and rs80356517.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Spondylocarpotarsal Synostosis Syndrome (SCT).

Spondylometaphyseal Dysplasia (SMD)

It is a genetic disorder that affects bone and joint development. This condition impacts the spine and causes short stature, abnormal curvature of the spine, and other skeletal abnormalities.

My Spondylometaphyseal Dysplasia (SMD)

Non-carrier

Knowledge



Time of onset

It typically appears during infancy or early childhood.



Prevalence

It is estimated to affect fewer than 1 in 1,000,000 individuals worldwide.



Typical symptoms

Typical symptoms include short stature, abnormal curvature of the spine, hip and knee deformities, and abnormalities of the ribs and pelvis.



Treatment

Treatment may include surgical interventions to correct bone and joint abnormalities, physical therapy to improve mobility and function, and supportive care to manage other symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267607144	TRPV4	CC	normal
RS387906904	TRPV4	GG	normal

Test details

Applicable situation

Genetic disease screening test for Spondylometaphyseal Dysplasia (SMD) uses 2 polymorphic loci on the *TRPV4* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Spondylometaphyseal Dysplasia (SMD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Spondylometaphyseal Dysplasia (SMD), or if you feel that you have a risk of developing Spondylometaphyseal Dysplasia (SMD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Spondylometaphyseal Dysplasia (SMD), such as rs267607144 and rs387906904.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Spondylometaphyseal Dysplasia (SMD).

Spondyloepiphyseal Dysplasia (SED)

It is a group of rare genetic disorders that affect bone and cartilage development. These conditions primarily affect the spine and joints, causing abnormalities in their growth and function.

My Spondyloepiphyseal Dysplasia (SED)

Non-carrier

Knowledge

Time of onset

It typically appears at birth or early infancy, but can also manifest during childhood or adolescence.

Prevalence

It is estimated to affect 1 in 45,000 individuals.

Typical symptoms

Typical symptoms include short stature, limited mobility, chronic pain, and spinal deformities such as scoliosis and kyphosis.

Treatment

Treatment primarily consists of managing symptoms through physical therapy, exercise, pain management, and corrective surgeries. In some cases, growth hormone therapy may be used.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS864621973	COL2A1	CC	normal
RS869312907	COL2A1	CC	normal

Test details

Applicable situation

Genetic disease screening test for Spondyloepiphyseal Dysplasia (SED) uses 2 polymorphic loci on the *COL2A1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Spondyloepiphyseal Dysplasia (SED).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Spondyloepiphyseal Dysplasia (SED), or if you feel that you have a risk of developing Spondyloepiphyseal Dysplasia (SED), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Spondyloepiphyseal Dysplasia (SED), such as rs864621973 and rs869312907.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Spondyloepiphyseal Dysplasia (SED).

Spondyloepimetaphyseal Dysplasia (SEMD)

It is a genetic disorder that affects bone growth and development. It is characterized by short stature, joint laxity, scoliosis, and distinctive facial features.

My Spondyloepimetaphyseal Dysplasia (SEMD)
Non-carrier

Knowledge

 Time of onset

It typically appears at birth, but symptoms may not be noticeable until later in childhood.

 Prevalence

The prevalence of this disease is currently unknown.

 Typical symptoms

Typical symptoms include short stature, joint hypermobility, scoliosis, platyspondyly, and distinctive facial features such as a broad forehead and flattened bridge of the nose.

 Treatment

Treatment is generally supportive and based on an individual's specific symptoms. This may include physical therapy, orthopedic management, and hearing aids.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121912880	COL2A1	CC	normal
RS797045099	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Spondyloepimetaphyseal Dysplasia (SEMD) uses 2 polymorphic loci on the *COL2A1* and *PAPSS2* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Spondyloepimetaphyseal Dysplasia (SEMD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Spondyloepimetaphyseal Dysplasia (SEMD), or if you feel that you have a risk of developing Spondyloepimetaphyseal Dysplasia (SEMD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Spondyloepimetaphyseal Dysplasia (SEMD), such as rs121912880 and rs797045099.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Spondyloepimetaphyseal Dysplasia (SEMD).

Spondylocostal Dysostosis (SCDO)

It is a genetic disorder that affects the development of the spine and ribs. It leads to abnormal curvature of the spine and deformities of the ribs that can result in reduced lung capacity and breathing difficulties.

My Spondylocostal Dysostosis (SCDO)

Non-carrier

Knowledge



Time of onset

It typically appears at birth or develops during infancy.



Prevalence

It is estimated to affect fewer than 1 in 1 million individuals.



Typical symptoms

Typical symptoms include distinct physical features, such as a short neck, flattened chest, and a protruding abdomen. Patients may also experience difficulty breathing or develop lung infections. In addition, abnormal curvature of the spine can lead to scoliosis or kyphosis.



Treatment

Treatment options aim to manage symptoms and prevent complications. This may include interventions such as physical therapy, bracing or surgery to correct spinal curvature, or treatment of lung infections or other respiratory issues. In some cases, genetic counseling and testing may be recommended for affected individuals and their families.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS786204039	intergenic	DD	normal

RS786204040	TBX6	DD	normal
RS786205519	DLL3	GG	normal

Test details

Applicable situation

Genetic disease screening test for Spondylocostal Dysostosis (SCDO) uses 3 polymorphic loci on the *TBX6* and *DLL3* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Spondylocostal Dysostosis (SCDO).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Spondylocostal Dysostosis (SCDO), or if you feel that you have a risk of developing Spondylocostal Dysostosis (SCDO), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Spondylocostal Dysostosis (SCDO), such as rs786204039, rs786204040 and rs786205519.

Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Spondylocostal Dysostosis (SCDO).

Familial Partial Lipodystrophy (FPL)

It is a rare genetic disorder that affects the body's ability to store fat tissue. It is characterized by a loss of fat tissue in the arms, legs, trunk, and buttocks, combined with an excess of fat tissue in the neck and face.

My Familial Partial Lipodystrophy (FPL)
Non-carrier

Knowledge



Time of onset

It usually develops during childhood or adolescence, but may not be diagnosed until adulthood.



Prevalence

It is a rare disease, affecting approximately 1 in 10 million individuals worldwide.



Typical symptoms

The most common symptoms of this disease include a lack of fat tissue in the arms, legs, trunk, and buttocks, combined with an excess of fat tissue in the neck and face.



Treatment

There is no cure for this disease, but treatment may include medication to manage symptoms, such as insulin-sensitizing drugs to improve insulin sensitivity and lower blood sugar levels. In some cases, liposuction or fat grafting may be used to correct the uneven distribution of fat tissue.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121909246	PPARG	CC	normal
RS72551362	PPARG	GG	normal

Test details

Applicable situation

Genetic disease screening test for Familial Partial Lipodystrophy (FPL) uses 2 polymorphic loci on the *PPARG* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Familial Partial Lipodystrophy (FPL).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Familial Partial Lipodystrophy (FPL), or if you feel that you have a risk of developing Familial Partial Lipodystrophy (FPL), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Familial Partial Lipodystrophy (FPL), such as rs121909246 and rs72551362.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Familial Partial Lipodystrophy (FPL).

Hypobetalipoproteinemia (HBL)

It is a disease that impairs the body's ability to absorb and transport fat. Its characteristic is that the cholesterol content in the blood is low. Many people with this disease may experience abnormal fat accumulation in the liver, leading to fatty liver disease. People with severe conditions may have difficulty absorbing fats and fat soluble vitamins, such as vitamin E and vitamin A.

My Hypobetalipoproteinemia (HBL)

Non-carrier

Knowledge

Time of onset

It is a congenital disorder, meaning that it is present at birth.- However, symptoms may not present until later in life.

Prevalence

The prevalence of hypobetalipoproteinemia is relatively rare, estimated to affect fewer than 1 in 1,000,000 individuals.

Typical symptoms

Individuals with it may be asymptomatic or present with mild to moderate symptoms. Common symptoms include low cholesterol and lipid levels, fatty liver disease, and neurological symptoms like ataxia and peripheral neuropathy.

Treatment

There is no cure for it, so treatment focuses on managing symptoms and preventing complications.- This can include managing lipid and cholesterol levels through diet and exercise, managing liver and neurological symptoms, and monitoring for related conditions like diabetes and high blood pressure.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918385	APOB	II	normal
RS1553383473	intergenic	GG	normal
RS878853970	APOB	GG	normal
RS797045253	intergenic	II	normal
RS766243954	APOB	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Hypobetalipoproteinemia (HBL) uses 24 polymorphic loci on the *APOB* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hypobetalipoproteinemia (HBL).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.

4. If you have a family history of Hypobetalipoproteinemia (HBL), or if you feel that you have a risk of developing Hypobetalipoproteinemia (HBL), it is recommended that you consult a qualified health professional or clinician for help.

 Test content

This test includes 24 polymorphic loci associated with the risk of Hypobetalipoproteinemia (HBL), such as rs1057518647, rs1215189537 and rs121918385.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hypobetalipoproteinemia (HBL).

Familial Cold Autoinflammatory Syndrome (FCAS)

It is a rare genetic disorder that causes episodes of fever, rash, and joint pain after exposure to cold temperatures.

My Familial Cold Autoinflammatory Syndrome (FCAS)

Non-carrier

Knowledge

Time of onset

Symptoms of it usually start in childhood and tend to be lifelong.

Prevalence

It is a rare disease, with only a few hundred cases reported worldwide.

Typical symptoms

The hallmark symptom of it is episodes of fever, rash, and joint pain that occur after exposure to cold temperatures. Other symptoms may include headache, muscle aches, and fatigue.

Treatment

Treatment for it focuses on managing symptoms. This may include avoiding cold temperatures, using nonsteroidal anti-inflammatory drugs (NSAIDs) or colchicine to control inflammation, and in some cases, using biologic drugs that target specific inflammatory proteins.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121908146	NLRP3	CC	normal

RS121908149	NLRP3	CC	normal
RS121908150	NLRP3	CC	normal
RS28937896	NLRP3	TT	normal
RS180177445	NLRP3	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Familial Cold Autoinflammatory Syndrome (FCAS) uses 6 polymorphic loci on the *NLRP3* and *NLRC4* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Familial Cold Autoinflammatory Syndrome (FCAS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Familial Cold Autoinflammatory Syndrome (FCAS), or if you feel that you have a risk of developing Familial Cold Autoinflammatory Syndrome (FCAS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 6 polymorphic loci associated with the risk of Familial Cold Autoinflammatory Syndrome (FCAS), such as rs121908146, rs121908149 and rs121908150.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Familial Cold Autoinflammatory Syndrome (FCAS).

Familial erythrocytosis

It is a rare genetic disorder characterized by an excessive production of red blood cells in the bone marrow, leading to an abnormally high number of erythrocytes in the blood.

My Familial erythrocytosis

Non-carrier

Knowledge

Time of onset

The onset of symptoms can occur at any age, with the disease usually manifesting in adulthood.

Prevalence

The exact prevalence of it is unknown, but it is considered to be a rare disease.

Typical symptoms

Patients with it may experience general symptoms such as fatigue, headaches, weakness, and dizziness. They may also experience symptoms specifically related to increased blood viscosity, such as Raynaud's phenomenon, thrombosis, and bleeding disorders.

Treatment

Treatment options include phlebotomy or medication to reduce erythrocyte production. Regular monitoring of hematologic parameters is recommended to prevent complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS33914668	HBB	TT	normal
RS33971440	HBB	CC	normal

RS34690599	HBB	GG	normal
RS34889882	HBB	II	normal
RS35004220	HBB	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Familial erythrocytosis uses 8 polymorphic loci on the *HBB* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Familial erythrocytosis.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Familial erythrocytosis, or if you feel that you have a risk of developing Familial erythrocytosis, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 8 polymorphic loci associated with the risk of Familial erythrocytosis, such as rs33914668, rs33971440 and rs34282684.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Familial erythrocytosis.

Familial Amyotrophic Lateral Sclerosis (FALS)

Also known as Lou Gehrig's disease. It is caused by mutations in one of several genes involved in the functioning of neurons that control voluntary movements.

My Familial Amyotrophic Lateral Sclerosis (FALS)

Non-carrier

Knowledge

Time of onset

The onset of symptoms in it can vary, but usually begins between the ages of 40 and 60.

Prevalence

It is rare and affects approximately 5-10% of all ALS cases worldwide.

Typical symptoms

The typical symptoms of it include muscle weakness, stiffness, and twitching, as well as difficulty speaking, breathing, and swallowing.

Treatment

Unfortunately, there is no cure for it, and treatment is focused on managing symptoms and maintaining quality of life. Some medications and therapies can help alleviate symptoms and prolong survival.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121912443	SOD1	AA	normal
RS200793464	SPG11	GG	normal

RS386134173	ALS2	II	normal
RS386134174	ALS2	II	normal
RS397514262	ERBB4	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Familial Amyotrophic Lateral Sclerosis (FALS) uses 9 polymorphic loci on genes such as *SOD1*, *NEK1* and *SPG11*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Familial Amyotrophic Lateral Sclerosis (FALS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Familial Amyotrophic Lateral Sclerosis (FALS), or if you feel that you have a risk of developing Familial Amyotrophic Lateral Sclerosis (FALS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 9 polymorphic loci associated with the risk of Familial Amyotrophic Lateral Sclerosis (FALS), such as rs121912443, rs199947197 and rs200793464.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Familial Amyotrophic Lateral Sclerosis (FALS).

Familial Exudative Vitreoretinopathy (FEVR)

It is an genetic eye disorder that affects the development of blood vessels in the retina. The abnormal growth of blood vessels can lead to retinal detachment, vision loss, and even blindness.

My Familial Exudative Vitreoretinopathy (FEVR)

Non-carrier

Knowledge

Time of onset

It symptoms can present at birth or in early childhood.

Prevalence

The prevalence of it is estimated to be around 1 in 10,000 individuals.

Typical symptoms

The typical symptoms of it include poor vision, difficulty seeing at night, sensitivity to light, and poor eye movement control.

Treatment

Treatment options for it depend on the severity of the condition and may include surgery, laser therapy, or injections of anti-VEGF drugs into the eye. Therapeutic interventions can help reduce vision loss and prevent further retinal damage.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS80358284	FZD4	TT	normal

Test details

Applicable situation

Genetic disease screening test for Familial Exudative Vitreoretinopathy (FEVR) uses 1 polymorphic locus on the *FZD4* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Familial Exudative Vitreoretinopathy (FEVR).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Familial Exudative Vitreoretinopathy (FEVR), or if you feel that you have a risk of developing Familial Exudative Vitreoretinopathy (FEVR), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Familial Exudative Vitreoretinopathy (FEVR), namely rs80358284.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Familial Exudative Vitreoretinopathy (FEVR).

Familial Dysautonomia (FD)

It is a rare genetic disorder that affects the development and function of the autonomic nervous system. This system controls involuntary actions such as regulation of blood pressure, heart rate, and digestion.

My Familial Dysautonomia (FD)

Non-carrier

Knowledge



Time of onset

Symptoms of it can appear at birth or in early infancy.



Prevalence

it is a rare disease with a of approximately 1 in 3,700 individuals of Ashkenazi Jewish descent.



Typical symptoms

Common symptoms of it include difficulty regulating body temperature, lack of tears, poor muscle tone, and increased susceptibility to pneumonia.



Treatment

Treatment options for it generally focus on managing symptoms and preventing complications. This may include medications to regulate blood pressure or improve breathing, as well as frequent monitoring for signs of infection or other complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS28939712	IKBKAP	GG	normal

Test details

Applicable situation

Genetic disease screening test for Familial Dysautonomia (FD) uses 1 polymorphic locus on the *ELP1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Familial Dysautonomia (FD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Familial Dysautonomia (FD), or if you feel that you have a risk of developing Familial Dysautonomia (FD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Familial Dysautonomia (FD), namely rs28939712.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Familial Dysautonomia (FD).

Nail-patella Syndrome (NPS)

It is a rare multisystemic disease with a classic clinical tetrad of fingernail dysplasia, hypoplasia or absence of the patella, presence of iliac horns, and elbow deformities. Ocular, renal, and neurological involvement can occur.

My Nail-patella Syndrome (NPS)

Non-carrier

Knowledge

Time of onset

It is typically diagnosed in childhood, but symptoms may not be present until later in life.

Prevalence

It is estimated to affect 1 in 50,000 individuals worldwide.

Typical symptoms

Typical symptoms include abnormally shaped, absent, or small nails, underdeveloped or absent kneecaps, and bone abnormalities.

Treatment

For bone abnormalities, bracing or surgery may be necessary; For kidney problems, medication and dialysis may be needed; For glaucoma or other eye problems, medication or surgery may be recommended.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS864621969	LMX1B	CC	normal

Test details

Applicable situation

Genetic disease screening test for Nail-patella Syndrome (NPS) uses 1 polymorphic locus on the *LMX1B* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Nail-patella Syndrome (NPS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Nail-patella Syndrome (NPS), or if you feel that you have a risk of developing Nail-patella Syndrome (NPS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Nail-patella Syndrome (NPS), namely rs864621969.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Nail-patella Syndrome (NPS).

Methylmalonic Aciduria

It is a genetic disorder that is due to the defect of methylmalonyl CoA mutase or the defect of its coenzyme cobalamin, which leads to the abnormal accumulation of toxic substances such as methylmalonic acid, resulting in multiple organ damage, especially nervous system damage, which can seriously endanger life.

My Methylmalonic Aciduria

Non-carrier

Knowledge



Time of onset

The onset of symptoms varies, while infants may develop symptoms shortly after birth, others may not be diagnosed until later in childhood or even adulthood.



Prevalence

It is estimated to affect 1 in 50,000 to 100,000 individuals worldwide.



Typical symptoms

The symptoms vary depending on the severity of the disease and the age at which it develops. It can affect various organ systems, including the kidneys, nervous system and digestive system. Patients with severe form may develop life-threatening complications, including kidney failure or sepsis. Additionally, some patients may develop neurological symptoms such as seizures, tremors or difficulty walking.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support, for example, dietary restrictions, supplementation with beneficial micronutrients and medications. In severe cases, liver or kidney transplantation may be necessary. Early diagnosis and management are important for improving health outcomes and avoiding serious complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918249	MUT	AA	normal
RS772552898	MUT	GG	normal
RS758008398	intergenic	DD	normal
RS761477436	intergenic	CC	normal
RS761525156	intergenic	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Methylmalonic Aciduria uses 38 polymorphic loci on the *MMUT*, *MMAB* and *MMAA* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Methylmalonic Aciduria.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.

4. If you have a family history of Methylmalonic Aciduria, or if you feel that you have a risk of developing Methylmalonic Aciduria, it is recommended that you consult a qualified health professional or clinician for help.

 Test content

This test includes 38 polymorphic loci associated with the risk of Methylmalonic Aciduria, such as rs121918249, rs121918254 and rs121918255.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Methylmalonic Aciduria.

Methylmalonic Aciduria and Homocystinuria

It is a rare genetic disease. Patients cannot normally decompose valine, isoleucine, threonine, methionine, cholesterol and fatty acids, which leads to the accumulation of toxic metabolites such as methylmalonic acid and methyl citrate in the body, thus causing serious health problems.

My Methylmalonic Aciduria and Homocystinuria

Non-carrier

Knowledge

 Time of onset

This disease can occur from infancy to adulthood.

 Prevalence

It is estimated to affect 1 in 50,000 to 100,000 individuals worldwide.

 Typical symptoms

Typical symptoms include metabolic acidosis, weakness, vomiting, poor appetite, failure to thrive, developmental delays, intellectual disability and vision problems.

 Treatment

Treatment for this disease involve a specific diet high in vitamin B12 and folate, as well as supplementations with medications such as betaine and carnitine. Early detection and treatment can help prevent long term complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS749272546	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Methylmalonic Aciduria and Homocystinuria uses 1 polymorphic locus on the *LMBRD1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Methylmalonic Aciduria and Homocystinuria.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Methylmalonic Aciduria and Homocystinuria, or if you feel that you have a risk of developing Methylmalonic Aciduria and Homocystinuria, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Methylmalonic Aciduria and Homocystinuria, namely rs749272546.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Methylmalonic Aciduria and Homocystinuria.

Methylmalonic Acidemia

It is a rare genetic disease. It is due to the abnormal function of methylmalonic acid coenzyme A mutase, which leads to the accumulation of methylmalonic acid and other organic acids in the body, causing damage to the nervous system, and in serious cases, it can cause ketoacidosis, hypoglycemia, hyperammonemia, hyperglycaemia.

My Methylmalonic Acidemia

Non-carrier

Knowledge



Time of onset

This disease can present at birth, infancy, childhood, or even adulthood.



Prevalence

It is estimated to affect 1 in 50,000-100,000 newborns.



Typical symptoms

Typical symptoms include abnormal levels of methylmalonic acid and other substances in the blood and urine, poor feeding, vomiting, lethargy, developmental delays, seizures and metabolic crises.



Treatment

Treatment includes a low-protein diet, supplements of specific vitamins and micronutrients. In some cases, organ transplantation is required. Regular monitoring and management of symptoms by healthcare professionals is vital.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918257	MUT	GG	normal

RS796052002

MUT

GG

normal

Test details

Applicable situation

Genetic disease screening test for Methylmalonic Acidemia uses 2 polymorphic loci on the *MMUT* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Methylmalonic Acidemia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Methylmalonic Acidemia, or if you feel that you have a risk of developing Methylmalonic Acidemia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Methylmalonic Acidemia, such as rs121918257 and rs796052002.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Methylmalonic Acidemia.

Methylmalonic Acidemia with Homocystinuria

It is a rare congenital metabolic defect in vitamin B12 (cobalamin) metabolism, characterized by megaloblastic anemia, drowsiness, developmental delay, intellectual disability, and seizures.

My Methylmalonic Acidemia with Homocystinuria

Non-carrier

Knowledge

Time of onset

This disease can occur from infancy to adulthood, with symptoms often appearing in the first few months of life.

Prevalence

It is estimated to affect 1 in 50,000 to 100,000 individuals.

Typical symptoms

Typical symptoms include metabolic acidosis, elevated levels of methionine and homocysteine in the blood and urine, poor feeding or appetite, vomiting, lethargy, developmental delays, muscle weakness and seizures.

Treatment

Treatment for this disease may include dietary management, medication to control symptoms and regular monitoring of blood levels.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS370596113	MMACHC	CC	normal

RS398124295	MMACHC	GG	normal
RS1305170860	intergenic	II	normal
RS796051995	MMACHC	CC	normal
RS796051999	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Methylmalonic Acidemia with Homocystinuria uses 5 polymorphic loci on the *MMACHC* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Methylmalonic Acidemia with Homocystinuria.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Methylmalonic Acidemia with Homocystinuria, or if you feel that you have a risk of developing Methylmalonic Acidemia with Homocystinuria, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Methylmalonic Acidemia with Homocystinuria, such as rs1305170860, rs370596113 and rs398124295.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Methylmalonic Acidemia with Homocystinuria.

Mevalonic Aciduria (MA)

It is a rare genetic disease that affects the body's ability to process the amino acid isoleucine. This leads to a build-up of mevalonic acid in the body, which can cause inflammation and damage to multiple systems including the joints, skin, and nervous system.

My Mevalonic Aciduria (MA)

Non-carrier

Knowledge



Time of onset

It typically appears in infancy or early childhood.



Prevalence

It is estimated to affect 1 in 1,000,000 individuals worldwide.



Typical symptoms

Typical symptoms include fever, joint pain and swelling, skin rashes, muscle weakness, developmental delay, and intellectual disability.



Treatment

Treatment may include managing symptoms with anti-inflammatory drugs, as well as genetic counseling and support for affected individuals and their families.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104895301	intergenic	GG	normal
RS121917790	MVK	CC	normal

RS104895298	MVK	GG	normal
RS104895311	MVK	GG	normal
RS104895360	MVK	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Mevalonic Aciduria (MA) uses 7 polymorphic loci on the *MVK* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Mevalonic Aciduria (MA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Mevalonic Aciduria (MA), or if you feel that you have a risk of developing Mevalonic Aciduria (MA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 7 polymorphic loci associated with the risk of Mevalonic Aciduria (MA), such as rs104895298, rs104895301 and rs104895311.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Mevalonic Aciduria (MA).

Pseudohypoparathyroidism (PHP)

It is a rare genetic disorder that affects the normal functioning of parathyroid hormone, calcium and phosphorus metabolism. The body's tissues and organs become unresponsive to the parathyroid hormone which leads to a decrease in calcium and an increase in phosphorus levels in the blood.

My Pseudohypoparathyroidism (PHP)

Non-carrier

Knowledge



Time of onset

It is usually diagnosed in childhood, but it can also develop in adulthood.



Prevalence

The prevalence of this disease is currently unknown.



Typical symptoms

Typical symptoms include muscle cramps and spasms, tingling and numbness in the hands and feet, susceptibility to fractures, stagnant or slow growth, intellectual disabilities and short stature.



Treatment

Treatment involves replacing the missing parathyroid hormone to maintain normal calcium and phosphate levels.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS397514456	GNAS	GG	normal

Test details

Applicable situation

Genetic disease screening test for Pseudohypoparathyroidism (PHP) uses 1 polymorphic locus on the *GNAS* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Pseudohypoparathyroidism (PHP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Pseudohypoparathyroidism (PHP), or if you feel that you have a risk of developing Pseudohypoparathyroidism (PHP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Pseudohypoparathyroidism (PHP), namely rs397514456.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Pseudohypoparathyroidism (PHP).

Pseudopseudohypoparathyroidism (PPHP)

It is a rare genetic disorder that affects bone growth and development, resulting in short stature and skeletal abnormalities.

My Pseudopseudohypoparathyroidism (PPHP)

Non-carrier

Knowledge



Time of onset

It is present from birth but may not be diagnosed until later in life when symptoms become apparent.



Prevalence

It has an estimated prevalence of fewer than one in a million.



Typical symptoms

Typical symptoms include short stature, round face, shortened fourth and fifth fingers, skeletal abnormalities.



Treatment

Treatment may include physical therapy to improve range of motion, surgery to correct skeletal abnormalities, and hormone replacement therapy to manage hormone imbalances.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1057518907	intergenic	CC	normal
RS797045046	GNAS	CC	normal

Test details

Applicable situation

Genetic disease screening test for Pseudopseudohypoparathyroidism (PPHP) uses 2 polymorphic loci on the *GNAS* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Pseudopseudohypoparathyroidism (PPHP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Pseudopseudohypoparathyroidism (PPHP), or if you feel that you have a risk of developing Pseudopseudohypoparathyroidism (PPHP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Pseudopseudohypoparathyroidism (PPHP), such as rs1057518907 and rs797045046.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Pseudopseudohypoparathyroidism (PPHP).

Pseudohypoaldosteronism (PHA)

It is a genetic disease that affects the body's ability to regulate blood potassium and sodium levels. Its characteristics include salt loss, dehydration, hypertension and low blood potassium levels.

My Pseudohypoaldosteronism (PHA)

Non-carrier

Knowledge

Time of onset

The disease may occur during infancy or early childhood.

Prevalence

The prevalence of this disease is currently unknown.

Typical symptoms

Typical symptoms include salt loss, dehydration, hypertension and low blood potassium levels.

Treatment

Treatment includes replacing lost sodium and potassium through oral supplementation or intravenous infusion; Drugs that block the action of aldosterone can also be used. In severe cases or cases that do not respond to traditional treatment, kidney transplantation may be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS111033591	WNK1	CC	normal
RS137852635	SCNN1A	GG	normal

RS137852704	SCNN1B	CC	normal
RS199469640	KLHL3	CC	normal

Test details

Applicable situation

Genetic disease screening test for Pseudohypoaldosteronism (PHA) uses 4 polymorphic loci on genes such as *WNK1*, *SCNN1A* and *SCNN1B*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Pseudohypoaldosteronism (PHA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Pseudohypoaldosteronism (PHA), or if you feel that you have a risk of developing Pseudohypoaldosteronism (PHA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Pseudohypoaldosteronism (PHA), such as rs111033591, rs137852635 and rs137852704.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Pseudohypoaldosteronism (PHA).

Progressive Pseudorheumatoid Dysplasia (PPRD)

It is a joint disease that deteriorates over time. The characteristic of this situation is the degeneration of cartilage between bones. This type of cartilage covers and protects the ends of bones, and its degradation leads to joint pain and stiffness, as well as other symptoms.

My Progressive Pseudorheumatoid Dysplasia (PPRD)

Non-carrier

Knowledge

Time of onset

Symptoms usually begin in early childhood, typically before the age of 10.

Prevalence

The prevalence of this disease is currently unknown.

Typical symptoms

Early symptoms include joint stiffness and pain, particularly in the hands, feet, and spine. Over time, affected individuals may also develop joint swelling, limited range of motion, and difficulty walking.

Treatment

Treatment may include physical therapy, pain management, and assistive devices such as braces or wheelchairs.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121908901	WISP3	CC	normal

Test details

Applicable situation

Genetic disease screening test for Progressive Pseudorheumatoid Dysplasia (PPRD) uses 1 polymorphic locus on the *CCN6* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Progressive Pseudorheumatoid Dysplasia (PPRD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Progressive Pseudorheumatoid Dysplasia (PPRD), or if you feel that you have a risk of developing Progressive Pseudorheumatoid Dysplasia (PPRD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Progressive Pseudorheumatoid Dysplasia (PPRD), namely rs121908901.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Progressive Pseudorheumatoid Dysplasia (PPRD).

Progressive External Ophthalmoplegia (PEO)

It is a rare genetic disorder that affects the muscles that control eye movement. This condition mainly causes weakness in the muscles that move the eyes and sometimes in other muscles throughout the body.

My Progressive External Ophthalmoplegia (PEO)

Non-carrier

Knowledge

Time of onset

It typically appears in adulthood between the ages of 18 and 45 years.

Prevalence

It is considered a rare condition, the prevalence is unknown.

Typical symptoms

The most common symptom is weakness in the muscles that control eye movement, leading to difficulty in moving the eyes. Other symptoms include ptosis, diplopia, dysphagia, and muscle weakness and wasting in the limbs.

Treatment

Treatment mainly involves improving symptoms. For example, medications to ease muscle weakness and spasms, surgery to correct ptosis or strabismus, and physical therapy to maintain muscle strength and flexibility.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893873	SLC25A4	GG	normal

RS104893876	SLC25A4	TT	normal
RS111033574	intergenic	GG	normal
RS111033575	intergenic	GG	normal
RS137886900	TK2	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Progressive External Ophthalmoplegia (PEO) uses 13 polymorphic loci on genes such as *SLC25A4*, *TWINK* and *TK2*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Progressive External Ophthalmoplegia (PEO).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Progressive External Ophthalmoplegia (PEO), or if you feel that you have a risk of developing Progressive External Ophthalmoplegia (PEO), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 13 polymorphic loci associated with the risk of Progressive External Ophthalmoplegia (PEO), such as rs104893873, rs104893876 and rs111033574.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Progressive External Ophthalmoplegia (PEO).

Arginase Deficiency (ARG)

It is a rare genetic metabolic disorder characterized by the inability of the body to produce enough arginase, an enzyme required for the breakdown of arginine, an amino acid. This results in the accumulation of arginine and ammonia, causing damage to the brain and other organs over time.

My Arginase Deficiency (ARG)

Non-carrier

Knowledge

Time of onset

Symptoms of it usually appear in early childhood, but it can also manifest later in life.

Prevalence

The prevalence of it is very low, with fewer than 1 in 1,000,000 individuals affected.

Typical symptoms

The typical symptoms of this disease include developmental delay, intellectual disability, seizures, spasticity, and abnormal movements. Infants with the condition may also have poor feeding and vomiting. Some cases may also have urinary symptoms like renal calculi.

Treatment

Treatment for arginase deficiency involves a low-protein diet, arginine-free medical formulas, and medications that help remove excess ammonia from the body.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893944	ARG1	CC	normal

RS104893947	ARG1	GG	normal
RS104893948	intergenic	GG	normal
RS28941474	ARG1	TT	normal
RS587776539	intergenic	GG	normal

Test details

Applicable situation

Genetic disease screening test for Arginase Deficiency (ARG) uses 5 polymorphic loci on the *ARG1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Arginase Deficiency (ARG).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Arginase Deficiency (ARG), or if you feel that you have a risk of developing Arginase Deficiency (ARG), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Arginase Deficiency (ARG), such as rs104893944, rs104893947 and rs104893948.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Arginase Deficiency (ARG).

Focal Segmental Glomerulosclerosis (FSGS)

It is a rare kidney disease that affects the filtering units (glomeruli) in the kidneys. It causes scarring and damage to the glomeruli, leading to proteinuria, edema and eventually kidney failure.

My Focal Segmental Glomerulosclerosis (FSGS)

Non-carrier

Knowledge



Time of onset

It can occur at any age, but it is more common in children and young adults. It can develop gradually over a period of months to years, or it can come on suddenly.



Prevalence

It is a rare disease, affecting about 8 in every 1 million people worldwide. It is more common in African Americans and Hispanics, and is slightly more common in men than women.



Typical symptoms

The most common symptoms of it are proteinuria, edema (swelling) in the legs, ankles and feet, and fatigue. Some patients may also experience high blood pressure, reduced urine output and weight gain.



Treatment

Treatment for it involves managing symptoms and slowing the progression of the disease. This may include medications to reduce proteinuria, control blood pressure, and prevent infections. In some cases, a kidney biopsy may be needed to determine the best course of treatment. In severe cases of FSGS, dialysis or kidney transplantation may be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121907909	WT1	GG	normal
RS267607183	INF2	GG	normal
RS387907037	INF2	TT	normal
RS530391015	INF2	GG	normal

Test details

Applicable situation

Genetic disease screening test for Focal Segmental Glomerulosclerosis (FSGS) uses 4 polymorphic loci on the *WT1* and *INF2* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Focal Segmental Glomerulosclerosis (FSGS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Focal Segmental Glomerulosclerosis (FSGS), or if you feel that you have a risk of developing Focal Segmental Glomerulosclerosis (FSGS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Focal Segmental Glomerulosclerosis (FSGS), such as rs121907909, rs267607183 and rs387907037.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Focal Segmental Glomerulosclerosis (FSGS).

Giant Axonal Neuropathy (GAN)

It is a rare genetic disorder that affects the central and peripheral nervous systems.

My Giant Axonal Neuropathy (GAN)

Non-carrier

Knowledge

Time of onset

Onset of it typically occurs in early childhood, with symptoms manifesting between the ages of 2 and 5 years.

Prevalence

The prevalence of it is relatively low, estimated to affect around 1 in every 10 million people worldwide.

Typical symptoms

Typical symptoms of it include it include difficulty walking, muscle weakness and wasting, and loss of sensation in the arms and legs. Additionally, affected individuals may experience difficulties with speech, vision, and hearing.

Treatment

Currently, there is no cure for it, and treatment is focused on managing symptoms. This may include physical therapy, use of assistive devices (such as braces or wheelchairs), and medications to control pain and muscle spasms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS747291494	intergenic	GG	normal

Test details

Applicable situation

Genetic disease screening test for Giant Axonal Neuropathy (GAN) uses 1 polymorphic locus on the *GAN* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Giant Axonal Neuropathy (GAN).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Giant Axonal Neuropathy (GAN), or if you feel that you have a risk of developing Giant Axonal Neuropathy (GAN), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Giant Axonal Neuropathy (GAN), namely rs747291494.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Giant Axonal Neuropathy (GAN).

Tyrosinemia

It is a rare genetic disorder that affects the body's ability to break down tyrosine. This can lead to a buildup of toxic substances in the liver, kidneys, and other organs, causing damage and potentially life-threatening complications.

My Tyrosinemia

Non-carrier

Knowledge



Time of onset

It can present at any age.



Prevalence

The of this disease varies depending on the type of the disease. Type I occurs most frequently in Quebec, Canada, while other forms occur throughout the world.



Typical symptoms

Typical symptoms include liver and kidney damage, growth failure, rickets, and jaundice.



Treatment

Treatment may include a restricted diet low in tyrosine and phenylalanine, medication, supplementation with cofactors, and in severe cases, liver transplantation. Continual monitoring and management throughout a patient's life are required.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121965075	FAH	GG	normal
RS137852867	intergenic	GG	normal

RS370686447	KATNAL2	GG	normal
RS80338894	KATNAL2	GG	normal
RS80338898	FAH	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Tyrosinemia uses 6 polymorphic loci on the *FAH* and *HPD* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Tyrosinemia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Tyrosinemia, or if you feel that you have a risk of developing Tyrosinemia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 6 polymorphic loci associated with the risk of Tyrosinemia, such as rs121965075, rs137852867 and rs370686447.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Tyrosinemia.

Combined Pituitary Hormone Deficiency (CPHD)

Also known as panhypopituitarism, it is a rare medical condition characterized by the inadequate production and secretion of one or more hormones by the pituitary gland located at the base of the brain.

My Combined Pituitary Hormone Deficiency (CPHD)

Non-carrier

Knowledge



Time of onset

The onset is most commonly seen during childhood or early adulthood.



Prevalence

It is estimated to affect fewer than 1 in 10,000 individuals, making it a rare disease.



Typical symptoms

The symptoms can vary depending on the specific hormones affected but commonly include fatigue, weakness, weight loss, decreased appetite, cold intolerance, decreased libido, menstrual disturbances, decreased muscle mass.



Treatment

Treatment typically involves hormone replacement therapy to address the specific hormone(s) deficient. Treatment is typically lifelong, and regular monitoring and adjustment of hormone replacement is required.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS104893763	POU1F1	TT	normal
RS104893764	POU1F1	CC	normal
RS104893765	POU1F1	CC	normal
RS104893766	POU1F1	GG	normal
RS121917840	PROP1	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Combined Pituitary Hormone Deficiency (CPHD) uses 11 polymorphic loci on the *POU1F1*, *PROP1* and *HESX1* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Combined Pituitary Hormone Deficiency (CPHD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Combined Pituitary Hormone Deficiency (CPHD), or if you feel that you have a risk of developing Combined Pituitary Hormone Deficiency (CPHD), it is recommended

that you consult a qualified health professional or clinician for help.

 Test content

This test includes 11 polymorphic loci associated with the risk of Combined Pituitary Hormone Deficiency (CPHD), such as rs104893763, rs104893764 and rs104893765.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Combined Pituitary Hormone Deficiency (CPHD).

Leucine-Induced Hypoglycemia (LIH)

It is a rare autosomal recessive metabolic disease that causes low blood sugar levels due to amino acid leucine accumulation in the body.

My Leucine-Induced Hypoglycemia (LIH)

Non-carrier

Knowledge



Time of onset

It typically appears in the first few months of life.



Prevalence

It is estimated to affect fewer than 1 in 100,000 individuals globally.



Typical symptoms

Typical symptoms include lethargy, seizures, hypoglycemia, vomiting, and delayed development.



Treatment

Treatment may include a low-protein diet to limit leucine intake, regular monitoring of blood sugar levels, and insulin therapy. In severe cases, a liver transplant may be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS193922402	ABCC8	GG	normal
RS201682634	ABCC8	GG	normal
RS773306994	intergenic	CC	normal

Test details

Applicable situation

Genetic disease screening test for Leucine-Induced Hypoglycemia (LIH) uses 3 polymorphic loci on the *ABCC8* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Leucine-Induced Hypoglycemia (LIH).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Leucine-Induced Hypoglycemia (LIH), or if you feel that you have a risk of developing Leucine-Induced Hypoglycemia (LIH), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Leucine-Induced Hypoglycemia (LIH), such as rs193922402, rs201682634 and rs773306994.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Leucine-Induced Hypoglycemia (LIH).

Craniofrontonasal Syndrome (CFNS)

It is a rare genetic disorder that mainly affects the development of the skull, face, and limbs.

My Craniofrontonasal Syndrome (CFNS)

Non-carrier

Knowledge



Time of onset

It is typically diagnosed during infancy, although mild cases may go undiagnosed until adulthood.



Prevalence

It is a rare disease, with an estimated prevalence of 1 in 100,000 to 1 in 200,000 individuals worldwide.



Typical symptoms

The most common symptoms of this disease include craniofacial anomalies (such as a high forehead, wide-set eyes, and a small or absent nose), skeletal abnormalities (such as shortened fingers and toes), and mild to moderate intellectual disability.



Treatment

Treatment for it is largely supportive and focused on managing specific symptoms. This may include surgery to correct craniofacial or skeletal abnormalities, physical therapy to improve motor function, and educational interventions to support learning and development.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894801	EFNB1	CC	normal
RS104894804	EFNB1	CC	normal

RS28936070

EFNB1

GG

normal

Test details

Applicable situation

Genetic disease screening test for Craniofrontonasal Syndrome (CFNS) uses 3 polymorphic loci on the *EFNB1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Craniofrontonasal Syndrome (CFNS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Craniofrontonasal Syndrome (CFNS), or if you feel that you have a risk of developing Craniofrontonasal Syndrome (CFNS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Craniofrontonasal Syndrome (CFNS), such as rs104894801, rs104894804 and rs28936070.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Craniofrontonasal Syndrome (CFNS).

Cranioectodermal Dysplasia (CED)

It is a rare genetic disorder that affects various organs and systems in the body. It primarily affects the development of bone, hair, and skin, as well as the nervous system, eyes, and kidneys.

My Cranioectodermal Dysplasia (CED)

Non-carrier

Knowledge

Time of onset

It is present from birth, and symptoms may be apparent in infancy or early childhood.

Prevalence

The prevalence of it is estimated to be extremely low, affecting fewer than one in a million individuals worldwide.

Typical symptoms

The typical symptoms of this disease include abnormal facial features, short stature, abnormal hair growth, delayed development of motor skills, and intellectual disability.

Treatment

Treatment for it is primarily symptomatic and supportive, as there is no cure for the disease. Medical care may involve the management of specific symptoms and complications due to the disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS786205567	intergenic	TT	normal

Test details

Applicable situation

Genetic disease screening test for Cranioectodermal Dysplasia (CED) uses 1 polymorphic locus on the *IFT122* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Cranioectodermal Dysplasia (CED).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Cranioectodermal Dysplasia (CED), or if you feel that you have a risk of developing Cranioectodermal Dysplasia (CED), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Cranioectodermal Dysplasia (CED), namely rs786205567.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Cranioectodermal Dysplasia (CED).

Osteoglophonic Dysplasia

It is a rare genetic disorder that affects bone growth and development.

My Osteoglophonic Dysplasia

Non-carrier

Knowledge

Time of onset

It typically appears in infancy or early childhood.

Prevalence

The prevalence of this disease is currently unknown.

Typical symptoms

Typical symptoms include abnormally shaped bones, specifically the skull and long bones in the limbs. Individuals with this disorder may also have hearing loss, dental abnormalities, and difficulties with movement.

Treatment

Treatment is usually aimed at managing symptoms. For example, surgery to correct abnormal bone morphology, speech therapy, and physical therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121909627	FGFR1	GG	normal

Test details

Applicable situation

Genetic disease screening test for Osteoglophonic Dysplasia uses 1 polymorphic locus on the *FGFR1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Osteoglophonic Dysplasia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Osteoglophonic Dysplasia, or if you feel that you have a risk of developing Osteoglophonic Dysplasia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Osteoglophonic Dysplasia, namely rs121909627.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Osteoglophonic Dysplasia.

Trichorhinophalangeal Syndrome (TRPS)

It is a rare genetic disorder that affects bone and hair development. It is characterized by facial anomalies, short stature, thin hair, and abnormalities in the bones of the hands and feet.

My Trichorhinophalangeal Syndrome (TRPS)

Non-carrier

Knowledge



Time of onset

The onset typically occurs during childhood.



Prevalence

It is extremely rare, affecting only a few hundred individuals worldwide.



Typical symptoms

Typical symptoms include short stature, thin hair, a small head, distinctive facial features, and abnormalities in the bones of the hands and feet.



Treatment

Treatment mainly involves improving symptoms and providing support. Patients may benefit from physical therapy to improve their range of motion, and hearing aids if hearing loss is present. Surgery may be necessary to correct bone abnormalities.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121908431	TRPS1	GG	normal

Test details

Applicable situation

Genetic disease screening test for Trichorhinophalangeal Syndrome (TRPS) uses 1 polymorphic locus on the *TRPS1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Trichorhinophalangeal Syndrome (TRPS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Trichorhinophalangeal Syndrome (TRPS), or if you feel that you have a risk of developing Trichorhinophalangeal Syndrome (TRPS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Trichorhinophalangeal Syndrome (TRPS), namely rs121908431.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Trichorhinophalangeal Syndrome (TRPS).

Immunodeficiency

It is a condition where the body's immune system is unable to function normally due to a defect in one or more of its components. This can lead to an increased vulnerability to infections and other health problems.

My Immunodeficiency
Non-carrier

Knowledge



Time of onset

It can be present from birth (congenital) or develop later in life (acquired).



Prevalence

The prevalence of it varies depending on the specific disorder. For example, severe combined immunodeficiency (SCID) is rare, affecting about 1 in every 100,000 births, while HIV/AIDS has a much higher prevalence.



Typical symptoms

Symptoms of it generally include frequent or severe infections, slow-healing wounds, and recurrent illnesses.



Treatment

Treatment of it depends on the specific disorder and may include antibiotics, antifungals, antivirals, immunosuppressive medications, stem cell transplants, and gene therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893893	IL7R	GG	normal

RS587777390	intergenic	TT	normal
RS587778405	IL7R	GG	normal
RS201559094	IL7R	CC	normal
RS74315444	IFNGR2	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Immunodeficiency uses 19 polymorphic loci on genes such as *IL7R*, *CD3E* and *STAT1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Immunodeficiency.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Immunodeficiency, or if you feel that you have a risk of developing Immunodeficiency, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 19 polymorphic loci associated with the risk of Immunodeficiency, such as rs104893893, rs121918659 and rs193922641.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Immunodeficiency.

Cystic Fibrosis (CF)

It is a genetic disorder that affects the respiratory, digestive and reproductive systems. It causes the production of thick and sticky mucus, leading to breathing difficulty, lung infections, malabsorption, and other complications.

My Cystic Fibrosis (CF)

Non-carrier

Knowledge



Time of onset

It is usually diagnosed in infancy or early childhood, but it can become apparent at any age.



Prevalence

It is a rare disease that affects approximately 1 in 2500-3000 newborns in the United States. It is most common in Caucasians of Northern European descent.



Typical symptoms

It symptoms can vary widely and may include chronic coughing, wheezing, shortness of breath, salty-tasting skin, poor growth and weight gain despite a good appetite, diarrhea, and infertility in males.



Treatment

While there is currently no cure for this disease, treatments are available to manage symptoms and improve quality of life. These may include antibiotics for infections, airway clearance techniques, nutritional supplements, and medications to improve lung function. In some cases, lung transplantation may be necessary. Gene therapy is being studied as a possible future treatment option.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS113993958	CFTR	GG	normal
RS397508416	intergenic	GG	normal
RS397508587	intergenic	II	normal
RS397508532	CFTR	GG	normal
RS397508505	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Cystic Fibrosis (CF) uses 115 polymorphic loci on the *CFTR* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Cystic Fibrosis (CF).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Cystic Fibrosis (CF), or if you feel that you have a risk of developing Cystic Fibrosis (CF), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 115 polymorphic loci associated with the risk of Cystic Fibrosis (CF), such as rs113993958, rs121908754 and rs121908760.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Cystic Fibrosis (CF).

Hydrocephalus Syndrome

It is a neurological condition that involves the accumulation of cerebrospinal fluid (CSF) in the brain's ventricles, leading to an increase in intracranial pressure (ICP).

My Hydrocephalus Syndrome

Non-carrier

Knowledge



Time of onset

It can present at any age, from infancy to adulthood.



Prevalence

Some estimates report one to two of every 1,000 babies are born with this disease.



Typical symptoms

The common symptoms of this disease include headache, nausea/vomiting, blurred vision, difficulty walking, coordination problems, and cognitive impairment. There may also be swelling of the head, thinning of the scalp, prominent scalp veins, and abnormal eye movements. Children may also exhibit bony protrusions, restlessness, and difficulty eating.



Treatment

Treatment for it involves the surgical placement of a shunt, which is a thin tube that diverts excess cerebrospinal fluid from the brain to the abdomen, bladder, or pleural cavity. In some cases, endoscopic third ventriculostomy (ETV) may be a viable alternative to shunting.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137852522	L1CAM	GG	normal

RS137852524	L1CAM	CC	normal
RS797044787	L1CAM	GG	normal

Test details

Applicable situation

Genetic disease screening test for Hydrocephalus Syndrome uses 3 polymorphic loci on the *L1CAM* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hydrocephalus Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hydrocephalus Syndrome, or if you feel that you have a risk of developing Hydrocephalus Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Hydrocephalus Syndrome, such as rs137852522, rs137852524 and rs797044787.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hydrocephalus Syndrome.

Pontocerebellar Hypoplasia (PCH)

It is a rare genetic disorder that affects the development of the brain, specifically the cerebellum and pons.

My Pontocerebellar Hypoplasia (PCH)

Non-carrier

Knowledge



Time of onset

Symptoms typically appear in infancy or early childhood.



Prevalence

It is a rare disease and its exact prevalence is not known.



Typical symptoms

Common symptoms include developmental delay, weak muscle tone, difficulty with coordination and movement, seizures and breathing problems.



Treatment

Treatment is typically supportive, focusing on managing symptoms and providing physical therapy to improve mobility and function. Anti-seizure medications may be prescribed to control seizures.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267607035	SEPSECS	CC	normal
RS587777616	CLP1	GG	normal
RS199835443	RARS2	GG	normal

RS371295780	VRK1	AA	normal
RS587780333	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Pontocerebellar Hypoplasia (PCH) uses 9 polymorphic loci on genes such as *RARS2*, *SEPSECS* and *VRK1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Pontocerebellar Hypoplasia (PCH).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Pontocerebellar Hypoplasia (PCH), or if you feel that you have a risk of developing Pontocerebellar Hypoplasia (PCH), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 9 polymorphic loci associated with the risk of Pontocerebellar Hypoplasia (PCH), such as rs199835443, rs267607035 and rs371295780.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Pontocerebellar Hypoplasia (PCH).

Mucopolysaccharidosis (MPS)

It is a rare genetic metabolic disorder that affects the production or breakdown of glycosaminoglycans (GAGs). These molecules accumulate in various tissues and organs, leading to damage and dysfunction.

My Mucopolysaccharidosis (MPS)

Non-carrier

Knowledge



Time of onset

The onset can vary depending on the type of the disease and the severity of the genetic mutation. Some forms may appear at birth or in infancy, while others may not present any symptoms until adolescence or adulthood.



Prevalence

The prevalence varies depending on the specific type of the disease. It is estimated to affect 1 in 25,000 to 100,000 births.



Typical symptoms

Common symptoms may include delayed development, joint stiffness and pain, coarse facial features, enlarged organs, problems with vision and hearing and difficulty in breathing.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support, such as enzyme replacement therapy, stem cell transplant, and medications to manage symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894641	SGSH	CC	normal

RS727504028	intergenic	GG	normal
RS398123430	GALNS	GG	normal
RS398123440	GALNS	GG	normal
RS483352895	intergenic	DD	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Mucopolysaccharidosis (MPS) uses 55 polymorphic loci on genes such as *SGSH*, *IDS* and *GALNS*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Mucopolysaccharidosis (MPS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Mucopolysaccharidosis (MPS), or if you feel that you have a risk of developing Mucopolysaccharidosis (MPS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 55 polymorphic loci associated with the risk of Mucopolysaccharidosis (MPS), such as rs104894641, rs104894856 and rs113993945.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Mucopolysaccharidosis (MPS).

Mucopolidosis

It is a group of hereditary metabolic diseases that affect the human body's ability to carry out normal turnover of various substances within cells. In this disease, abnormal amounts of carbohydrates and fatty substances accumulate in cells, Therefore, cell damage can occur, causing symptoms that range from mild learning disabilities to severe intellectual impairment and skeletal deformities.

My Mucopolidosis Non-carrier

Knowledge

Time of onset

This disease can occur at any age and varies depending on the specific type of the disease.

Prevalence

It is estimated to affect 1 in 100,000 to 600,000 individuals worldwide.

Typical symptoms

Typical symptoms include developmental delays, skeletal abnormalities, impaired vision and hearing and organ dysfunction.

Treatment

Treatment is mainly supportive and may include physical therapy, medications and surgery in some cases.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104886461	MCOLN1	AA	normal

RS398124397	GNPTAB	CC	normal
RS797044663	GNPTAB	DD	normal
RS794727302	intergenic	II	normal
RS781689303	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Mucopolidosis uses 18 polymorphic loci on the *MCOLN1*, *GNPTG* and *GNPTAB* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Mucopolidosis.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Mucopolidosis, or if you feel that you have a risk of developing Mucopolidosis, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 18 polymorphic loci associated with the risk of Mucopolipidosis, such as rs104886461, rs121908371 and rs121908373.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Mucopolipidosis.

Ornithine Aminotransferase Deficiency (OAT)

It is an genetic disorder that affects the production and breakdown of amino acids. It is caused by the lack of the ornithine aminotransferase enzyme, which leads to the buildup of ornithine and ammonia in the body.

My Ornithine Aminotransferase Deficiency (OAT)

Non-carrier

Knowledge



Time of onset

The onset varies depending on the severity of the disease. In some cases, symptoms appear shortly after birth, while in others symptoms may not appear until later in life.



Prevalence

It is estimated to affect approximately 1 in 250,000 individuals.



Typical symptoms

Symptoms can vary widely and may include vomiting, seizures, intellectual disability, developmental delay, and liver disease.



Treatment

Treatment may include a low-protein diet and supplements to help reduce ammonia levels in the body. In some cases, liver transplantation may be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121965047	OAT	CC	normal
RS121965057	OAT	GG	normal

RS200068769	OAT	GG	normal

Test details

Applicable situation

Genetic disease screening test for Ornithine Aminotransferase Deficiency (OAT) uses 3 polymorphic loci on the *OAT* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Ornithine Aminotransferase Deficiency (OAT).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Ornithine Aminotransferase Deficiency (OAT), or if you feel that you have a risk of developing Ornithine Aminotransferase Deficiency (OAT), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Ornithine Aminotransferase Deficiency (OAT), such as rs121965047, rs121965057 and rs200068769.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Ornithine Aminotransferase Deficiency (OAT).

Factor V Deficiency

It is a rare bleeding disorder that is genetic and affects blood clotting. This disease is caused by a deficiency of Factor V, a protein that plays an important role in the clotting process.

My Factor V Deficiency

Non-carrier

Knowledge



Time of onset

It can occur at any age but is usually diagnosed in infancy or childhood.



Prevalence

It is a rare disease that affects approximately 1 in 1 million individuals worldwide.



Typical symptoms

The typical symptoms of it include excessive bleeding and bruising, bleeding after minor cuts or injuries, and heavy menstrual periods in women.



Treatment

Treatment options for it include the administration of clotting factor concentrates and treatment of bleeding episodes with medications that can promote clotting. In some cases, genetic counseling may also be recommended.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS118203907	F5	TT	normal

Test details

Applicable situation

Genetic disease screening test for Factor V Deficiency uses 1 polymorphic locus on the *F5* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Factor V Deficiency.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Factor V Deficiency, or if you feel that you have a risk of developing Factor V Deficiency, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Factor V Deficiency, namely rs118203907.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Factor V Deficiency.

Factor VII Deficiency

It is a rare genetic bleeding disorder that affects the body's ability to produce the clotting protein known as factor VII. As a result, affected individuals may experience prolonged bleeding after an injury or surgery, or spontaneous bleeding in muscles and joints. The severity of the disease can vary widely, with some individuals experiencing only mild bleeding symptoms, while others may have frequent, severe bleeding episodes.

My Factor VII Deficiency
Non-carrier

Knowledge



Time of onset

It is a genetic disorder that is present from birth, but symptoms may not appear until later in life.



Prevalence

It is a rare disorder, estimated to occur in 1 in 500,000 individuals.



Typical symptoms

The most common symptoms of it are prolonged bleeding after an injury or surgery, spontaneous bleeding in muscles and joints, nosebleeds, and easy bruising. Women with it may also experience heavy and prolonged menstrual periods.



Treatment

There is currently no cure for it, but it can be managed with appropriate medical care. Treatment options may include taking medications that increase the body's production of factor VII, receiving infusions of factor VII concentrate, and taking steps to prevent bleeding before planned surgeries or procedures.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS755377592	intergenic	GG	normal

Test details

Applicable situation

Genetic disease screening test for Factor VII Deficiency uses 1 polymorphic locus on the *F7* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Factor VII Deficiency.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Factor VII Deficiency, or if you feel that you have a risk of developing Factor VII Deficiency, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Factor VII Deficiency, namely rs755377592.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Factor VII Deficiency.

Factor XI Deficiency

It is a rare genetic disorder that affects the blood clotting process. It is characterized by low levels or deficiency of the clotting protein Factor XI, which can lead to excessive bleeding after injury or surgery.

My Factor XI Deficiency

Non-carrier

Knowledge



Time of onset

The onset of it varies, but symptoms often appear in childhood or adolescence.



Prevalence

The prevalence of it is estimated to be fewer than 1 in 1 million people in the general population.



Typical symptoms

The typical symptoms of it include a prolonged clotting time, reduced clot strength, and an increased tendency for bleeding. Other symptoms may include easy bruising, nosebleeds, and bleeding in the gastrointestinal or genitourinary tracts.



Treatment

Treatment options for it may include the use of clotting factor concentrates or fresh frozen plasma to correct the clotting deficiency. Patients may also be advised to avoid medications that can interfere with blood clotting, and to take special precautions before undergoing surgery or dental procedures.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121965066	F11	CC	normal

RS139695003	intergenic	CC	normal
RS281875250	intergenic	CC	normal
RS542967227	F11	GG	normal
RS752907087	intergenic	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Factor XI Deficiency uses 9 polymorphic loci on the *F11* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Factor XI Deficiency.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Factor XI Deficiency, or if you feel that you have a risk of developing Factor XI Deficiency, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 9 polymorphic loci associated with the risk of Factor XI Deficiency, such as rs121965066, rs139695003 and rs281875250.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Factor XI Deficiency.

Skin/Hair/Eye Pigmentation (SHEP)

It is a genetic disorder that affects the production and distribution of melanin, resulting in abnormal pigmentation of the skin, hair, and eyes.

My Skin/Hair/Eye Pigmentation (SHEP)

Non-carrier

Knowledge



Time of onset

Symptoms can be present at birth or in early childhood.



Prevalence

The prevalence of this disease is currently unknown.



Typical symptoms

Typical symptoms include uneven skin pigmentation, darkening or lightening of hair color, changes in eye color, and the appearance of freckles or birthmarks.



Treatment

Treatment mainly involves improving symptoms, such as regular skin exams to monitor for skin cancer or hearing aids to address hearing loss.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894314	TYR	GG	normal
RS121908011	TYR	GG	normal
RS28940876	TYR	CC	normal

RS368124046

OCA2

CC

normal

Test details

Applicable situation

Genetic disease screening test for Skin/Hair/Eye Pigmentation (SHEP) uses 4 polymorphic loci on the *TYR* and *OCA2* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Skin/Hair/Eye Pigmentation (SHEP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Skin/Hair/Eye Pigmentation (SHEP), or if you feel that you have a risk of developing Skin/Hair/Eye Pigmentation (SHEP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Skin/Hair/Eye Pigmentation (SHEP), such as rs104894314, rs121908011 and rs28940876.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Skin/Hair/Eye Pigmentation (SHEP).

Cutis Laxa (CL)

It is a rare genetic disorder that affects the connective tissues in the body, leading to loose and saggy skin that has reduced elasticity. This condition can also affect other parts of the body, including the muscles, joints, and internal organs.

My Cutis Laxa (CL)

Non-carrier

Knowledge



Time of onset

It typically presents with symptoms at birth or within the first few months of life, although some cases might not be diagnosed until later in life.



Prevalence

The prevalence of it is unknown, but it is estimated to affect fewer than 1 in 1,000,000 people worldwide.



Typical symptoms

The most of this disease include wrinkled, sagging skin, poor wound healing, respiratory problems, and developmental delays in infants.



Treatment

Treatment for this disease are primarily supportive and aim to manage the symptoms of the disease. These can include the use of elastic garments, surgery to correct hernias or other deformities, medication to address specific complications, and physical therapy to improve muscle strength and flexibility.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS119489101	EFEMP2	CC	normal
RS121918377	PYCR1	CC	normal
RS151340631	ATP7A	CC	normal
RS151340632	intergenic	AA	normal
RS151340633	ATP7A	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Cutis Laxa (CL) uses 14 polymorphic loci on genes such as *EFEMP2*, *PYCR1* and *LTBP4*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Cutis Laxa (CL).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Cutis Laxa (CL), or if you feel that you have a risk of developing Cutis Laxa (CL), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 14 polymorphic loci associated with the risk of Cutis Laxa (CL), such as rs119489101, rs121918377 and rs1382026467.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Cutis Laxa (CL).

Lissencephaly

It is a rare genetic disorder characterized by the absence or underdevelopment of brain gyri. This results in a small head size, developmental delays and severe intellectual disability.

My Lissencephaly
Non-carrier

Knowledge



Time of onset

This disease is typically diagnosed within the first few months of life.



Prevalence

It is estimated to affect 1 in every 100,000 live births.



Typical symptoms

Typical symptoms include feeding difficulties, seizures, severe developmental delays, absent or limited speech, and muscle spasticity.



Treatment

Treatment options are limited and often focus on managing symptoms, such as applying anti-seizure medications; Using physical therapy, speech and occupational therapy to help with developmental delays.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894782	DCX	GG	normal
RS587784290	intergenic	GG	normal

RS587784485	TUBA1A	GG	normal
RS587784483	TUBA1A	GG	normal
RS587784482	intergenic	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Lissencephaly uses 81 polymorphic loci on genes such as *DCX*, *TUBA1A* and *DYNC1H1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Lissencephaly.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Lissencephaly, or if you feel that you have a risk of developing Lissencephaly, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 81 polymorphic loci associated with the risk of Lissencephaly, such as rs104894782, rs1057517843 and rs137853044.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Lissencephaly.

GLUT1 Deficiency Syndrome

It is a rare genetic disorder that affects glucose transport in the brain leading to various neurological symptoms.

My GLUT1 Deficiency Syndrome

Non-carrier

Knowledge

Time of onset

Symptoms of this disease typically manifest in early childhood, usually before the age of 2.

Prevalence

It is a rare disease, with an estimated prevalence of 1 in 90,000 individuals.

Typical symptoms

The typical symptoms of it include seizures, developmental delays, movement disorders, cognitive impairment, ataxia, and other neurological symptoms

Treatment

The mainstay of treatment for it is a ketogenic diet that provides an alternative fuel source for the brain, allowing for improved neurological function. Antiepileptic drugs may also be used in some cases, along with physical and occupational therapy to manage movement disorders.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121909739	SLC2A1	CC	normal
RS121909740	SLC2A1	CC	normal

RS267607061	SLC2A1	GG	normal
RS80359816	SLC2A1	CC	normal
RS80359818	SLC2A1	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for GLUT1 Deficiency Syndrome uses 15 polymorphic loci on the *SLC2A1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of GLUT1 Deficiency Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of GLUT1 Deficiency Syndrome, or if you feel that you have a risk of developing GLUT1 Deficiency Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 15 polymorphic loci associated with the risk of GLUT1 Deficiency Syndrome, such as rs121909739, rs121909740 and rs267607061.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of GLUT1 Deficiency Syndrome.

Holoprosencephaly (HPE)

It is a rare congenital disorder in which the brain fails to divide into two hemispheres during development, resulting in varying degrees of intellectual disability and facial abnormalities.

My Holoprosencephaly (HPE)

Non-carrier

Knowledge

Time of onset

It typically presents in early fetal development, around 3-6 weeks after conception.

Prevalence

It is a rare disorder, occurring in approximately 1 in 10,000-20,000 live births, although the true may be higher as many cases may result in miscarriage or stillbirth.

Typical symptoms

The typical symptoms of it can include varying degrees of intellectual disability, feeding difficulties, seizures, and abnormal facial features such as a single central incisor, small head size, or cleft lip and palate.

Treatment

Treatment for this disease is primarily supportive, addressing the individual medical and developmental needs of the patient. Options can include surgery to correct facial abnormalities, medications to manage seizures or other medical complications, or specialized education and therapy for intellectual disability.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121909069	TGIF1	CC	normal
RS267607047	SHH	GG	normal
RS28936675	SHH	CC	normal
RS387906995	CDON	GG	normal

Test details

Applicable situation

Genetic disease screening test for Holoprosencephaly (HPE) uses 4 polymorphic loci on the *TGIF1*, *SHH* and *CDON* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Holoprosencephaly (HPE).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Holoprosencephaly (HPE), or if you feel that you have a risk of developing Holoprosencephaly (HPE), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Holoprosencephaly (HPE), such as rs121909069, rs267607047 and rs28936675.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Holoprosencephaly (HPE).

Maturity-Onset Diabetes of the Young (MODY)

It is a rare, monogenic form of diabetes that typically affects young individuals before the age of 25 and is related to primary insulin secretion deficiency.

My Maturity-Onset Diabetes of the Young (MODY)

Non-carrier

Knowledge



Time of onset

It typically manifests in adolescence or early adulthood (age <25).



Prevalence

It is a rare form of diabetes, accounting for fewer than 5% of all cases of diabetes. However, it may be underdiagnosed due to its similarity to other forms of diabetes.



Typical symptoms

Typical symptoms include increased thirst, frequent urination, blurred vision and fatigue. However, unlike Type 1 and Type II diabetes, this disease is generally not associated with insulin resistance.



Treatment

Treatment typically involves lifestyle modifications, including diet and exercise, as well as diabetes medications such as sulfonylureas. Insulin therapy may also be necessary in certain cases. Genetic testing and counseling may also be used to help manage the disease and identify other family members who may be at risk of developing it.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894006	GCK	GG	normal

RS193922605	HNF1A	TT	normal
RS1172328722	intergenic	GG	normal
RS1131692182	intergenic	GG	normal
RS1057524905	intergenic	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Maturity-Onset Diabetes of the Young (MODY) uses 78 polymorphic loci on genes such as *GCK*, *HNF1A* and *INS*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Maturity-Onset Diabetes of the Young (MODY).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Maturity-Onset Diabetes of the Young (MODY), or if you feel that you have a risk of developing Maturity-Onset Diabetes of the Young (MODY), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 78 polymorphic loci associated with the risk of Maturity-Onset Diabetes of the Young (MODY), such as rs104894006, rs104894008 and rs104894009.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Maturity-Onset Diabetes of the Young (MODY).

Achromatopsia

It is a rare genetic disorder which impairs an individual's ability to distinguish color, resulting in complete color blindness.

My Achromatopsia
Non-carrier

Knowledge



Time of onset

It is a congenital disorder, which means it is present from birth.



Prevalence

It is a very rare disease, with estimates suggesting it affects only 1 in 30,000 to 40,000 individuals worldwide.



Typical symptoms

The individuals with it experience a range of symptoms including loss of color perception, reduced visual acuity, tonic nystagmus, and extreme light sensitivity were noted.



Treatment

While there is no known cure for it, individuals can use red-tinted glasses, contact lenses, or other vision aids to reduce sensitivity to light, improve visual acuity, and protect against further retinal damage.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893613	CNGA3	CC	normal
RS761357250	ATF6	CC	normal

RS797045173	ATF6	DD	normal
RS797045172	intergenic	GG	normal
RS797045170	ATF6	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Achromatopsia uses 21 polymorphic loci on genes such as *CNGA3*, *PDE6C* and *CNGB3*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Achromatopsia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Achromatopsia, or if you feel that you have a risk of developing Achromatopsia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 21 polymorphic loci associated with the risk of Achromatopsia, such as rs104893613, rs104893614 and rs121918539.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Achromatopsia.

Holocarboxylase Synthetase Deficiency (HCSD)

It is a genetic disease that can lead to various metabolic disorders, accumulation of toxic organic acids, metabolic disorders, and multiple organ damage.

My Holocarboxylase Synthetase Deficiency (HCSD)

Non-carrier

Knowledge



Time of onset

The onset time for it varies, but most newborns with this disorder show symptoms within days of birth.



Prevalence

It is a rare disease, with a prevalence of approximately 1 in 87,000 live births worldwide.



Typical symptoms

The typical symptoms of it include poor feeding, vomiting, lethargy, developmental delays, and metabolic acidosis.



Treatment

The treatment for it involves daily lifelong administration of biotin supplements, along with a low-protein diet and other medications to manage symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS119103227	HLCS	AA	normal
RS119103229	HLCS	GG	normal

RS119103230	HLCS	CC	normal
RS766163167	intergenic	DD	normal
RS771944310	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Holocarboxylase Synthetase Deficiency (HCSD) uses 6 polymorphic loci on the *HLCS* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Holocarboxylase Synthetase Deficiency (HCSD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Holocarboxylase Synthetase Deficiency (HCSD), or if you feel that you have a risk of developing Holocarboxylase Synthetase Deficiency (HCSD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 6 polymorphic loci associated with the risk of Holocarboxylase Synthetase Deficiency (HCSD), such as rs119103227, rs119103229 and rs119103230.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Holocarboxylase Synthetase Deficiency (HCSD).

Hypochondroplasia

It is a type of skeletal dysplasia that affects the bones and cartilage.

My Hypochondroplasia
Non-carrier

Knowledge



Time of onset

Symptoms of it may be present at birth, but often become more noticeable during childhood.



Prevalence

It affects approximately 1 in 15,000 to 40,000 people worldwide.



Typical symptoms

The typical symptoms of it include a shortened spine, bowed legs, limited elbow extension, and a prominent forehead.



Treatment

Treatment for it is generally supportive and may include physical therapy, orthopedic interventions, and surgical procedures.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121913115	FGFR3	AA	normal

Test details



Applicable situation

Genetic disease screening test for Hypochondroplasia uses 1 polymorphic locus on the *FGFR3* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

 Notification

1. The test may not cover all the pathogenic genes or loci of Hypochondroplasia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

 Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hypochondroplasia, or if you feel that you have a risk of developing Hypochondroplasia, it is recommended that you consult a qualified health professional or clinician for help.

 Test content

This test includes 1 polymorphic locus associated with the risk of Hypochondroplasia, namely rs121913115.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hypochondroplasia.

Branchiooculofacial Syndrome (BOFS)

It is a rare genetic disorder that primarily affects the development of tissues in the face, neck, and eyes.

My Branchiooculofacial Syndrome (BOFS)

Non-carrier

Knowledge



Time of onset

It is present at birth, but symptoms may not become apparent until later in childhood or adulthood.



Prevalence

It is a rare genetic disorder with an estimated prevalence of 1 in 50,000 to 100,000 individuals.



Typical symptoms

Typical symptoms of this syndrome include structural abnormalities in the face, neck, and eyes, such as clefts or fistulas in the neck or ear, a small jaw, and abnormal eyelid openings.



Treatment

Treatment for it is palliative and typically involves surgery to correct craniofacial abnormalities, such as clefts or fistulas, and to improve vision and hearing.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS793888540	TFAP2A	GG	normal
RS793888541	TFAP2A	AA	normal

Test details

Applicable situation

Genetic disease screening test for Branchiooculofacial Syndrome (BOFS) uses 2 polymorphic loci on the *TFAP2A* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Branchiooculofacial Syndrome (BOFS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Branchiooculofacial Syndrome (BOFS), or if you feel that you have a risk of developing Branchiooculofacial Syndrome (BOFS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Branchiooculofacial Syndrome (BOFS), such as rs793888540 and rs793888541.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Branchiooculofacial Syndrome (BOFS).

Trifunctional Protein Deficiency (TFP)

It is a rare autosomal recessive metabolic disorder that affects long-chain fatty acid metabolism and prevents the body from utilizing fat for energy.

My Trifunctional Protein Deficiency (TFP)

Non-carrier

Knowledge

Time of onset

Symptoms usually appear in the first few months of life, but some cases may present later in childhood or adulthood.

Prevalence

This disease is very rare, with only a few hundred cases reported worldwide.

Typical symptoms

Typical symptoms include developmental delay, seizures, hypotonia, abnormal movements, and comas.

Treatment

Treatment may include dietary modifications, medications to manage symptoms, and in severe cases, organ transplantation.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121913131	HADHB	AA	normal
RS121913133	HADHB	GG	normal

RS121913134	HADHB	GG	normal
RS137852770	HADHA	GG	normal
RS747985669	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Trifunctional Protein Deficiency (TFP) uses 10 polymorphic loci on the *HADHB* and *HADHA* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Trifunctional Protein Deficiency (TFP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Trifunctional Protein Deficiency (TFP), or if you feel that you have a risk of developing Trifunctional Protein Deficiency (TFP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 10 polymorphic loci associated with the risk of Trifunctional Protein Deficiency (TFP), such as rs121913131, rs121913133 and rs121913134.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Trifunctional Protein Deficiency (TFP).

Incontinentia Pigmenti (IP)

It is an genetic (genetic) condition that affects the skin and other systems in the body. Skin symptoms change with time and begin with a blistering rash in infancy, followed by wart-like skin growths. The growths become swirled grey or brown patches in childhood, and then swirled light patches in adulthood.

My Incontinentia Pigmenti (IP)

Non-carrier

Knowledge

Time of onset

It usually presents in infancy or early childhood.

Prevalence

The prevalence of it is estimated to be 1 in every 50,000 to 100,000 live births.

Typical symptoms

The typical symptoms of this disease can vary greatly from mild to severe cases. Skin lesions usually appear in stages with blisters and verrucous plaques turning into atrophic patches. Hair loss can be patchy, total or temporal. Dental abnormalities include missing or misshapen teeth. Nail dystrophy can range from ridges to total loss. Neurologic and ophthalmic complications can also occur.

Treatment

Treatment for this disease are focused on managing symptoms and addressing specific complications. Some options include wound care for skin lesions, hair transplantation or wigs for hair loss, dental procedures for dental anomalies, and surgery for nail dystrophy or other complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS137853323

IKBKG

CC

normal

Test details

Applicable situation

Genetic disease screening test for Incontinentia Pigmenti (IP) uses 1 polymorphic locus on the *IKBKG* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Incontinentia Pigmenti (IP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Incontinentia Pigmenti (IP), or if you feel that you have a risk of developing Incontinentia Pigmenti (IP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Incontinentia Pigmenti (IP), namely rs137853323.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Incontinentia Pigmenti (IP).

Neurodegeneration with Brain Iron Accumulation (NBIA)

It is a group of rare genetic disorders characterized by abnormal iron accumulation in the brain, leading to progressive deterioration of nervous system function.

My Neurodegeneration with Brain Iron Accumulation (NBIA)

Non-carrier

Knowledge

Time of onset

The onset varies depending on the subtype of the disease, but symptoms typically start in childhood or adolescence.

Prevalence

It is estimated to affect 1-3 in every million individuals worldwide.

Typical symptoms

Typical symptoms include progressive difficulty with movement, muscle rigidity, tremors, involuntary movements, speech difficulties and cognitive decline.

Treatment

There is currently no cure. Treatment mainly involves improving symptoms and for preserving quality of life, for example, medications, physical therapy, surgical interventions (e.g., deep brain stimulation).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121908681	PLA2G6	TT	normal
RS587784343	intergenic	II	normal
RS797046102	intergenic	CC	normal
RS797046101	WDR45	GG	normal
RS797046100	WDR45	TT	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Neurodegeneration with Brain Iron Accumulation (NBIA) uses 18 polymorphic loci on genes such as *PLA2G6*, *COASY* and *WDR45*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Neurodegeneration with Brain Iron Accumulation (NBIA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Neurodegeneration with Brain Iron Accumulation (NBIA), or if you feel that you have a risk of developing Neurodegeneration with Brain Iron Accumulation (NBIA), it

is recommended that you consult a qualified health professional or clinician for help.

 Test content

This test includes 18 polymorphic loci associated with the risk of Neurodegeneration with Brain Iron Accumulation (NBIA), such as rs121908681, rs121908686 and rs145108650.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Neurodegeneration with Brain Iron Accumulation (NBIA).

Neuronal Ceroid Lipofuscinosis (NCL)

Also known as Batten disease, it is a rare and fatal neurodegenerative disorder, which involves the abnormal accumulation of lipofuscin in the cells, leading to progressive neuronal death.

My Neuronal Ceroid Lipofuscinosis (NCL)

Non-carrier

Knowledge



Time of onset

The early-onset form usually appears in the age of 2-4, whereas the late-onset form may appear in adulthood.



Prevalence

It is estimated to affect 2-4 in every 100,000 births.



Typical symptoms

Typical symptoms include progressive vision loss, seizures, speech difficulties, cognitive impairment and movement disorders.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support, for example, anticonvulsant medications may be given to control seizures, while physical and occupational therapies may help maintain mobility and function.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894060	CLN8	CC	normal

RS796052335	CLN3	GG	normal
RS724159970	MFSD8	CC	normal
RS724159971	MFSD8	GG	normal
RS750428882	TPP1	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Neuronal Ceroid Lipofuscinosis (NCL) uses 35 polymorphic loci on genes such as *CLN8*, *TPP1* and *CLN6*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Neuronal Ceroid Lipofuscinosis (NCL).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Neuronal Ceroid Lipofuscinosis (NCL), or if you feel that you have a risk of developing Neuronal Ceroid Lipofuscinosis (NCL), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 35 polymorphic loci associated with the risk of Neuronal Ceroid Lipofuscinosis (NCL), such as rs104894060, rs119455953 and rs121908080.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Neuronal Ceroid Lipofuscinosis (NCL).

Nephropathic Cystinosis

It is a rare genetic disease that causes a buildup of cystine in cells throughout the body, leading to tissue and organ damage.

My Nephropathic Cystinosis

Non-carrier

Knowledge



Time of onset

Symptoms typically appear within the first year of life.



Prevalence

It is estimated to affect 1 in every 100,000-200,000 individuals worldwide.



Typical symptoms

Typical symptoms include poor growth, frequent urination, excessive thirst, vomiting, constipation and muscle weakness.



Treatment

Treatment primarily involves medications to reduce cystine buildup, as well as supportive care to manage symptoms and complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS113994207	CTNS	GG	normal
RS760256854	intergenic	II	normal
RS786204501	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Nephropathic Cystinosis uses 3 polymorphic loci on the *CTNS* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Nephropathic Cystinosis.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Nephropathic Cystinosis, or if you feel that you have a risk of developing Nephropathic Cystinosis, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Nephropathic Cystinosis, such as rs113994207, rs760256854 and rs786204501.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Nephropathic Cystinosis.

Renal-Hepatic-Pancreatic Dysplasia (RHPD)

It is a rare and complex genetic disorder that affects the development of the kidneys, liver, and pancreas.

My Renal-Hepatic-Pancreatic Dysplasia (RHPD)

Non-carrier

Knowledge



Time of onset

It typically appears at birth, but also adolescence or adulthood.



Prevalence

It is estimated to affect fewer than 1 in 100,000 individuals worldwide.



Typical symptoms

Typical symptoms include kidney cysts, liver fibrosis, and pancreatic cysts. Other symptoms may include an enlarged spleen, gastrointestinal bleeding, and neurologic abnormalities.



Treatment

Treatment may include kidney and liver transplantation, management of liver fibrosis, and pancreatic cyst drainage.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS119456963	NPHP3	CC	normal
RS762826555	NEK8	GG	normal
RS763300393	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Renal-Hepatic-Pancreatic Dysplasia (RHPD) uses 3 polymorphic loci on the *NPHP3* and *NEK8* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Renal-Hepatic-Pancreatic Dysplasia (RHPD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Renal-Hepatic-Pancreatic Dysplasia (RHPD), or if you feel that you have a risk of developing Renal-Hepatic-Pancreatic Dysplasia (RHPD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Renal-Hepatic-Pancreatic Dysplasia (RHPD), such as rs119456963, rs762826555 and rs763300393.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Renal-Hepatic-Pancreatic Dysplasia (RHPD).

Nephronophthisis (NPHP)

It is a rare genetic disorder characterized by the progressive destruction of renal tubules and leads to chronic kidney disease.

My Nephronophthisis (NPHP)

Non-carrier

Knowledge



Time of onset

The onset is usually in childhood, with symptoms typically appearing in the age of 5-15.



Prevalence

It is estimated to affect 1-9 in 1 million individuals worldwide.



Typical symptoms

The early symptoms are often nonspecific and can include excessive thirst, frequent urination, tiredness and loss of appetite. As the disease progresses, symptoms like polyuria, polydipsia, anemia, hypertension become more evident.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms and designed to slow down the progression of renal disease. For example, angiotensin-converting enzyme (ACE) inhibitors, dialysis or kidney transplantation in cases of end-stage renal disease (ESRD).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS119456962	NPHP3	GG	normal
RS121907899	NPHP1	CC	normal

RS137853108	TMEM67	AA	normal
RS201893408	TMEM67	TT	normal
RS267607119	TMEM67	TT	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Nephronophthisis (NPHP) uses 15 polymorphic loci on genes such as *NPHP3*, *NPHP1* and *TMEM67*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Nephronophthisis (NPHP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Nephronophthisis (NPHP), or if you feel that you have a risk of developing Nephronophthisis (NPHP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 15 polymorphic loci associated with the risk of Nephronophthisis (NPHP), such as rs119456962, rs121907899 and rs137853108.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Nephronophthisis (NPHP).

Renal Carnitine Transport Defect

It is a rare genetic disorder that affects the body's ability to absorb and utilize carnitine, an important nutrient that helps the body convert fat into energy. This condition results in a buildup of toxic byproducts in the body, which can cause muscle weakness, low blood sugar, and other potentially serious health problems.

My Renal Carnitine Transport Defect

Non-carrier

Knowledge

Time of onset

It typically appears in infancy or early childhood and its effect can be life-long.

Prevalence

It only has a handful of affected individuals worldwide.

Typical symptoms

Typical symptoms include muscle weakness, low blood sugar, fatigue, and difficulty gaining weight.

Treatment

Treatment typically involves lifelong carnitine supplementation, along with monitoring of blood sugar levels, heart function, and overall health.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS114269482	SLC22A5	CC	normal
RS121908889	SLC22A5	GG	normal

RS121908893	SLC22A5	CC	normal
RS144547521	SLC22A5	CC	normal
RS151231558	SLC22A5	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Renal Carnitine Transport Defect uses 16 polymorphic loci on the *SLC22A5* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Renal Carnitine Transport Defect.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Renal Carnitine Transport Defect, or if you feel that you have a risk of developing Renal Carnitine Transport Defect, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 16 polymorphic loci associated with the risk of Renal Carnitine Transport Defect, such as rs114269482, rs121908889 and rs121908893.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Renal Carnitine Transport Defect.

Biotinidase Deficiency (BTD)

It is an genetic metabolic disorder caused by a deficiency of the enzyme biotinidase, which is necessary for the body to process and use the vitamin biotin. Without this enzyme, the body cannot recycle biotin, leading to a buildup of toxic by-products that damage the body's organs and tissues.

My Biotinidase Deficiency (BTD)

Non-carrier

Knowledge



Time of onset

It can occur at any age, but most cases are diagnosed in infancy or early childhood.



Prevalence

It is a rare disorder that affects approximately 1 in 60,000 to 1 in 120,000 people worldwide.



Typical symptoms

The typical symptoms of this disease include seizures, developmental delay, hypotonia (weak muscle tone), vision and hearing loss, skin rashes, and hair loss.



Treatment

Treatment for this disease include lifelong biotin supplementation, which can help manage symptoms and prevent long-term complications of the disease. Early detection and treatment is crucial for optimal outcomes.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893687	BTD	CC	normal

RS397514398	BTD	II	normal
RS398123139	BTD	GG	normal
RS398123138	intergenic	II	normal
RS397514434	BTD	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Biotinidase Deficiency (BTD) uses 21 polymorphic loci on the *BTD* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Biotinidase Deficiency (BTD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Biotinidase Deficiency (BTD), or if you feel that you have a risk of developing Biotinidase Deficiency (BTD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 21 polymorphic loci associated with the risk of Biotinidase Deficiency (BTD), such as rs104893687, rs146136265 and rs200507575.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Biotinidase Deficiency (BTD).

Optic Atrophy (OA)

It is a condition that affects the optic nerve, which carries visual information from the eye to the brain. It occurs when the nerve is damaged or degrades, resulting in a reduction or loss of vision.

My Optic Atrophy (OA)
Non-carrier

Knowledge

 Time of onset

Some individuals may experience sudden vision loss, whereas others may experience a gradual decline in vision over time.

 Prevalence

It is estimated to affect 1 in 10,000 people.

 Typical symptoms

Typical symptoms include blurred vision, reduced peripheral vision and difficulty in distinguishing colors.

 Treatment

Treatment involves addressing any underlying medical conditions, such as high blood pressure or diabetes, and taking vitamins or other supplements to support optic nerve health. In some cases, surgery may be necessary in order to restore vision.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS80356523	intergenic	CC	normal

RS730882246	ISCA2	GG	normal
RS753611141	intergenic	GG	normal
RS768643552	intergenic	GG	normal
RS863223953	DNM1L	CC	normal

Test details

Applicable situation

Genetic disease screening test for Optic Atrophy (OA) uses 5 polymorphic loci on genes such as *ISCA2*, *PMPCA* and *OPA3*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Optic Atrophy (OA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Optic Atrophy (OA), or if you feel that you have a risk of developing Optic Atrophy (OA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Optic Atrophy (OA), such as rs730882246, rs753611141 and rs768643552.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Optic Atrophy (OA).

Retinitis Pigmentosa (RP)

It is a rare genetic disorder caused by mutations in one or more genes that are essential for the function of photoreceptor cells in the retina, leading to progressive vision loss and sometimes blindness.

My Retinitis Pigmentosa (RP)

Non-carrier

Knowledge



Time of onset

It typically appears during childhood or adolescence.



Prevalence

It is estimated to affect 1 in 4,000 people worldwide.



Typical symptoms

Typical symptoms include night blindness, slow loss of peripheral vision, and difficulty seeing in low light or bright light.



Treatment

There is currently no cure for this disease. Treatment may include vitamin supplements, low-vision aids, and gene therapies. Clinical trials are also underway for more advanced treatments, such as stem cell therapies and bionic retinal implants.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893791	RHO	GG	normal
RS62640589	RPGR	AA	normal

RS767745816	SPATA7	TT	normal
RS763295314	IMPG2	AA	normal
RS761238771	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Retinitis Pigmentosa (RP) uses 129 polymorphic loci on genes such as *RHO*, *RP2* and *USH2A*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Retinitis Pigmentosa (RP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Retinitis Pigmentosa (RP), or if you feel that you have a risk of developing Retinitis Pigmentosa (RP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 129 polymorphic loci associated with the risk of Retinitis Pigmentosa (RP), such as rs104893791, rs104894927 and rs111033280.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Retinitis Pigmentosa (RP).

Cone-Rod Dystrophy (CRD)

It is a rare genetic disorder that affects the rod and cone cells in the retina.

My Cone-Rod Dystrophy (CRD)

Non-carrier

Knowledge



Time of onset

Symptoms of it can appear at any age, but most often begin in childhood or adolescence.



Prevalence

The prevalence of it is estimated to be between 1 in 30,000 to 1 in 40,000 individuals.



Typical symptoms

Symptoms of this disease can include difficulty seeing in bright light, loss of color vision, blind spots, and difficulty seeing at night.



Treatment

There is currently no known cure for this disease. Treatment options may include managing symptoms and providing low vision aids.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137853041	ADAM9	CC	normal
RS397517974	USH2A	CC	normal
RS61751407	ABCA4	CC	normal
RS62635009	RPGR	TT	normal

RS62646861

ABCA4

GG

normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Cone-Rod Dystrophy (CRD) uses 13 polymorphic loci on genes such as *ADAM9*, *USH2A* and *ABCA4*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Cone-Rod Dystrophy (CRD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Cone-Rod Dystrophy (CRD), or if you feel that you have a risk of developing Cone-Rod Dystrophy (CRD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 13 polymorphic loci associated with the risk of Cone-Rod Dystrophy (CRD), such as rs137853041, rs397517974 and rs61751407.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Cone-Rod Dystrophy (CRD).

Hypomyelination and Congenital Cataract (HCC)

It is an extremely rare genetic disorder that affects the development of the nervous system and the eyes. It is characterized by the presence of cataracts at birth or soon after, as well as delayed myelination, which is the process by which nerve fibers are coated in myelin, a fatty substance that helps messages travel more efficiently through the nervous system.

My Hypomyelination and Congenital Cataract (HCC)

Non-carrier

Knowledge



Time of onset

Symptoms of it are typically present at birth or shortly thereafter.



Prevalence

It is an extremely rare disease. Its prevalence is unknown, but it has been reported in only a few families worldwide.



Typical symptoms

The most common symptoms of this disease are congenital cataract, which is an opacity of the lens of the eye that is present at birth or soon after. Individuals with this disease often have severe developmental delays, delayed motor skills, impaired vision, and a reduction in the size of their head. They may also have muscle weakness and difficulty with balance and coordination. Brain imaging may reveal delayed myelination or brain atrophy.



Treatment

There is no cure for it, but treatment focuses on managing symptoms. Surgery to remove cataracts and physical therapy to improve motor skills may be beneficial.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS72549407	FAM126A	AA	normal

Test details

Applicable situation

Genetic disease screening test for Hypomyelination and Congenital Cataract (HCC) uses 1 polymorphic locus on the *HYCC1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hypomyelination and Congenital Cataract (HCC).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hypomyelination and Congenital Cataract (HCC), or if you feel that you have a risk of developing Hypomyelination and Congenital Cataract (HCC), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Hypomyelination and Congenital Cataract (HCC), namely rs72549407.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hypomyelination and Congenital Cataract (HCC).

Glycogen Storage Disease (GSD)

It is a rare genetic disorder of glycogen metabolism, caused by defects in enzymes involved in either glycogen synthesis or breakdown. This results in an abnormal accumulation or depletion of glycogen in various tissues, leading to varying symptoms.

My Glycogen Storage Disease (GSD)

Non-carrier

Knowledge



Time of onset

The age of onset and severity of symptoms for it varies depending on the specific subtype of the disease.



Prevalence

The overall incidence of it is estimated to be 1 in 20,000 live births.



Typical symptoms

Symptoms of it can include hypoglycemia, hepatomegaly, growth retardation, myopathy, cardiomyopathy, and other metabolic abnormalities.



Treatment

Treatment for it involves managing symptoms, supporting organ function, and providing a controlled diet to balance glucose and energy intake. Enzyme replacement therapy may also be used in some cases.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894563	G6PC	CC	normal

RS370792293	AGL	AA	normal
RS398124208	PYGM	CC	normal
RS80338672	GBE1	GG	normal
RS80356488	intergenic	DD	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Glycogen Storage Disease (GSD) uses 47 polymorphic loci on genes such as *G6PC1*, *AGL* and *PYGM*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Glycogen Storage Disease (GSD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Glycogen Storage Disease (GSD), or if you feel that you have a risk of developing Glycogen Storage Disease (GSD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 47 polymorphic loci associated with the risk of Glycogen Storage Disease (GSD), such as rs104894563, rs113994127 and rs113994129.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Glycogen Storage Disease (GSD).

Isolated Growth Hormone Deficiency (IGHD)

It is a rare condition where the body does not produce enough growth hormone, leading to a decrease in overall growth and development.

My Isolated Growth Hormone Deficiency (IGHD)

Non-carrier

Knowledge

Time of onset

It usually becomes apparent in early childhood, typically between the ages of 2 and 6, when children may be shorter than their peers and have delayed development or growth.

Prevalence

It is a rare condition, occurring in only a few out of every 10,000 children born. It affects both boys and girls equally.

Typical symptoms

The most common symptom of it is a marked decrease in growth, usually leading to being significantly shorter than peers. Other symptoms may include delayed or incomplete puberty, a decrease in muscle mass, and decreased energy levels.

Treatment

The main treatment for it is recombinant human growth hormone (rhGH) injections, which can help support normal growth and development. The treatment is usually started in early childhood and continued until the child reaches adult height. Other treatments may include hormone replacement therapy to address other deficiencies, and surgery or radiation therapy for underlying pituitary gland issues.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121918117	GHRHR	GG	normal
RS121918121	GHRHR	AA	normal
RS483352872	intergenic	AA	normal

Test details

Applicable situation

Genetic disease screening test for Isolated Growth Hormone Deficiency (IGHD) uses 3 polymorphic loci on the *GHRHR* gene and intergenic region. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Isolated Growth Hormone Deficiency (IGHD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Isolated Growth Hormone Deficiency (IGHD), or if you feel that you have a risk of developing Isolated Growth Hormone Deficiency (IGHD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Isolated Growth Hormone Deficiency (IGHD), which are rs121918117, rs121918121 and rs483352872.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Isolated Growth Hormone Deficiency (IGHD).

Aspartylglucosaminuria (AGU)

It is a rare genetic disorder that affects the metabolism of glycoproteins. It is caused by a deficiency of the enzyme aspartylglucosaminidase, which leads to the buildup of certain complex sugars in the body.

My Aspartylglucosaminuria (AGU)

Non-carrier

Knowledge

Time of onset

It is typically diagnosed in early childhood.

Prevalence

It is rare, occurring in approximately 1 in 100,000 people worldwide.

Typical symptoms

Typical symptoms of this disease include delayed speech, intellectual disability, joint stiffness, skeletal abnormalities, and a coarse facial appearance.

Treatment

There is no cure for this disease but symptomatic treatment can be helpful. Physical therapy and surgery can improve the functional abilities and reduce the pain associated with the skeletal abnormalities. Enzyme replacement therapy may also be helpful in treating some of the symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121964908	AGA	GG	normal
RS121964909	AGA	AA	normal

RS386833420	AGA	II	normal
RS386833437	AGA	CC	normal

Test details

Applicable situation

Genetic disease screening test for Aspartylglucosaminuria (AGU) uses 4 polymorphic loci on the *AGA* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Aspartylglucosaminuria (AGU).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Aspartylglucosaminuria (AGU), or if you feel that you have a risk of developing Aspartylglucosaminuria (AGU), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Aspartylglucosaminuria (AGU), such as rs121964908, rs121964909 and rs386833420.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Aspartylglucosaminuria (AGU).

Sideroblastic Anemia (SA)

It is a rare type of anemia that occurs when the body fails to produce enough healthy red blood cells due to a problem with the production of heme, a component of hemoglobin. This leads to a buildup of iron in the body's cells.

My Sideroblastic Anemia (SA)

Non-carrier

Knowledge

Time of onset

It can occur at any time, but it is more commonly seen in older adults and in people with an inherited predisposition to the condition.

Prevalence

It is a rare disease that affects fewer than 200,000 people in the United States.

Typical symptoms

Typical symptoms of it may include fatigue, weakness, shortness of breath, pale skin, and frequent infections. In addition to the typical symptoms of anemia, additional clinical features of this disease may include liver and spleen enlargement, impaired growth and development in children, and muscle weakness.

Treatment

Treatment for it typically involves addressing the underlying cause of the condition, such as adjusting medications or addressing alcohol use. Additional treatments may include blood transfusions, iron chelation therapy, or bone marrow transplants in severe cases.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS137852311

ALAS2

GG

normal

Test details

Applicable situation

Genetic disease screening test for Sideroblastic Anemia (SA) uses 1 polymorphic locus on the *ALAS2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Sideroblastic Anemia (SA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Sideroblastic Anemia (SA), or if you feel that you have a risk of developing Sideroblastic Anemia (SA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Sideroblastic Anemia (SA), namely rs137852311.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Sideroblastic Anemia (SA).

Auditory Neuropathy (AN)

It is a type of hearing disorder that affects the transmission of sound signals from the inner ear to the brain.

My Auditory Neuropathy (AN)

Non-carrier

Knowledge

Time of onset

It can be present at birth or may develop later in life.

Prevalence

The prevalence of it is relatively rare, estimated to be between 1-2 per 10,000 births.

Typical symptoms

It can present with a wide range of symptoms including difficulty understanding speech, poor hearing in noisy environments, irregular response to sounds, and challenges with speech development in children.

Treatment

Treatment for this disease generally involves managing the underlying symptoms and improving communication through the use of hearing aids, cochlear implants, and assistive communication devices.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS80356586	OTOF	AA	normal
RS80356602	intergenic	CC	normal

Test details

Applicable situation

Genetic disease screening test for Auditory Neuropathy (AN) uses 2 polymorphic loci on the *OTOF* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Auditory Neuropathy (AN).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Auditory Neuropathy (AN), or if you feel that you have a risk of developing Auditory Neuropathy (AN), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Auditory Neuropathy (AN), such as rs80356586 and rs80356602.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Auditory Neuropathy (AN).

Elliptocytosis

It is a hereditary blood disorder that causes red blood cells to be abnormal in shape, usually oval or elliptical, instead of the normal disk shape. This can lead to anemia, fatigue, and jaundice.

My Elliptocytosis
Non-carrier

Knowledge

Time of onset

It is present at birth, but symptoms may not appear until later in life.

Prevalence

It is a rare disorder and its prevalence is unknown.

Typical symptoms

The most common symptoms of it are anemia, fatigue, and jaundice. Some people with elliptocytosis may also experience spleen enlargement and gallstones.

Treatment

There is no cure for it, but treatment options include blood transfusions, medications to relieve symptoms, and surgery to remove the spleen or gallbladder, if necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918634	SPTA1	AA	normal
RS121918638	SPTA1	CC	normal

RS121918641	SPTA1	CC	normal
RS121918642	intergenic	GG	normal
RS121918645	SPTB	CC	normal

Test details

Applicable situation

Genetic disease screening test for Elliptocytosis uses 5 polymorphic loci on the *SPTA1* and *SPTB* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Elliptocytosis.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Elliptocytosis, or if you feel that you have a risk of developing Elliptocytosis, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Elliptocytosis, such as rs121918634, rs121918638 and rs121918641.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Elliptocytosis.

Vitamin D-Dependent Rickets (VDDR)

It is a rare genetic disorder that affects the bones, resulting in soft, weak, and deformed bones in infants and children.

My Vitamin D-Dependent Rickets (VDDR)

Non-carrier

Knowledge



Time of onset

It typically appears in infancy or early childhood.



Prevalence

It is estimated to affect 1 in 20,000 to 100,000 births.



Typical symptoms

Typical symptoms include delayed growth and development, bowed legs, deformities of the spine and legs, muscle weakness, dental problems, and an increased risk of fractures.



Treatment

Treatment may include high doses of oral vitamin D to maintain normal levels of calcium and phosphorus in the body. Other treatment may include calcium supplements and surgery.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121909790	VDR	CC	normal
RS121909793	VDR	CC	normal
RS121909797	VDR	CC	normal

RS28934604	CYP27B1	CC	normal
RS28934606	CYP27B1	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Vitamin D-Dependent Rickets (VDDR) uses 7 polymorphic loci on the *VDR*, *CYP27B1* and *PHEX* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Vitamin D-Dependent Rickets (VDDR).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Vitamin D-Dependent Rickets (VDDR), or if you feel that you have a risk of developing Vitamin D-Dependent Rickets (VDDR), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 7 polymorphic loci associated with the risk of Vitamin D-Dependent Rickets (VDDR), such as rs121909790, rs121909793 and rs121909797.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Vitamin D-Dependent Rickets (VDDR).

Striatonigral Degeneration (SND)

It is a rare neurodegenerative disease that affects infants and young children. It is primarily characterized by the progressive degeneration of certain areas of the brain, including the basal ganglia and nigral structures.

My Striatonigral Degeneration (SND)

Non-carrier

Knowledge



Time of onset

It typically appears within the first few years of life.



Prevalence

It is considered rare, with only a small number of cases reported worldwide.



Typical symptoms

Typical symptoms include developmental delays, movement disorders, difficulty feeding, and seizures.



Treatment

Treatment is focused on managing symptoms and providing supportive care, such as physical and occupational therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS199476135	intergenic	TT	normal

Test details

Applicable situation

Genetic disease screening test for Striatonigral Degeneration (SND) uses 1 polymorphic locus on the *MT-ATP6* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Striatonigral Degeneration (SND).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Striatonigral Degeneration (SND), or if you feel that you have a risk of developing Striatonigral Degeneration (SND), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Striatonigral Degeneration (SND), namely rs199476135.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Striatonigral Degeneration (SND).

Agammaglobulinemia

It is a rare genetic disorder that affects the immune system's ability to produce antibodies, which increases the chance of infections. It commonly occurs in males and is apparent during infancy or early childhood.

My Agammaglobulinemia

Non-carrier

Knowledge

Time of onset

It is usually apparent during infancy or early childhood

Prevalence

The prevalence of it is estimated to be around 1 in 250,000 to 1 in 1,000,000 births worldwide.

Typical symptoms

Individuals with this disease have a weakened immune system, which makes them prone to frequent, severe, and recurrent infections. Infections may be bacterial, viral, or fungal and can affect many organ systems. Children may experience developmental delays, growth disturbances, and chronic diarrhea.

Treatment

Treatment for agammaglobulinemia includes regular intravenous immunoglobulin (IVIG) infusions or subcutaneous immunoglobulin replacement to replace the missing antibodies. Antibiotics may also be used to prevent and treat infections.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894770	BTK	CC	normal

RS128621208	BTK	GG	normal
RS193922132	intergenic	TT	normal
RS193922131	intergenic	CC	normal
RS193922128	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Agammaglobulinemia uses 22 polymorphic loci on the *BTK* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Agammaglobulinemia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Agammaglobulinemia, or if you feel that you have a risk of developing Agammaglobulinemia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 22 polymorphic loci associated with the risk of Agammaglobulinemia, such as rs104894770, rs128620183 and rs128620184.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Agammaglobulinemia.

Aniridia

It is a rare disease that affects the development of the eyes. It can cause partial or complete loss of the iris, which is the colored part of the eye that controls the amount of light entering the eye.

My Aniridia

Non-carrier

Knowledge



Time of onset

It is usually present from birth and is a lifelong condition.



Prevalence

The prevalence of it is estimated to be around 1 in 50,000 to 100,000 individuals worldwide.



Typical symptoms

The main symptom of it is the absence of the iris, which can cause sensitivity to light and poor vision. Other symptoms may include nystagmus, which is an involuntary movement of the eyes, and cataracts. It may also be associated with other systemic conditions such as Wilms tumor, genitourinary abnormalities, and intellectual disabilities.



Treatment

The treatment for it mainly involves managing the symptoms. This may include wearing tinted glasses or contact lenses to reduce light sensitivity, using artificial tears to moisturize the eyes, and undergoing surgery to correct cataracts or other associated eye problems.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121907900	WT1	GG	normal

RS121907906	WT1	GG	normal
RS121907916	PAX6	GG	normal
RS121907917	PAX6	GG	normal
RS121907924	PAX6	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Aniridia uses 8 polymorphic loci on the *WT1* and *PAX6* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Aniridia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Aniridia, or if you feel that you have a risk of developing Aniridia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 8 polymorphic loci associated with the risk of Aniridia, such as rs121907900, rs121907906 and rs121907916.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Aniridia.

Choroideremia

It is a rare genetic disorder that affects the retina of the eye, leading to progressive loss of vision over time.

My Choroideremia
Non-carrier

Knowledge

Time of onset

For this disease usually occurs during one's teenage years or early adulthood.

Prevalence

It is a rare disease, affecting approximately 1 in 50,000 individuals.

Typical symptoms

The typical symptoms of this disease include difficulty seeing in dimly lit environments, loss of peripheral vision, and trouble seeing at night.

Treatment

There is currently no cure for this disease, but several treatments are being developed that aim to slow the progression of the disease. These include gene therapy, stem cell therapy, and retinal implants.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS132630266	CHM	GG	normal

Test details

Applicable situation

Genetic disease screening test for Choroideremia uses 1 polymorphic locus on the *CHM* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Choroideremia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Choroideremia, or if you feel that you have a risk of developing Choroideremia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Choroideremia, namely rs132630266.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Choroideremia.

Glutaric Acidemia (GA)

It is a rare genetic disorder that affects the body's ability to process certain amino acids properly. Specifically, it impairs the breakdown of the amino acids lysine, hydroxylysine, and tryptophan, leading to a buildup of toxic substances in the body that can cause damage to the brain and other organs.

My Glutaric Acidemia (GA)

Non-carrier

Knowledge



Time of onset

It can present in infants and young children, often around 6-18 months of age. However, it can also present later in life or even in adulthood.



Prevalence

The of this disease is estimated to be around 1 in 30,000-40,000 live births.



Typical symptoms

Typical symptoms of it include it include poor feeding, vomiting, seizures, developmental delays, and muscle stiffness or weakness.



Treatment

Treatment for it include a combination of dietary management and medication to manage symptoms and prevent complications. For example, following a low-lysine and low-tryptophan diet may help to control the buildup of toxic substances in the body, while certain medications can help to manage seizures, muscle spasms, and other symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS119458971	ETFA	CC	normal
RS377656387	ETFDH	CC	normal
RS387907170	ETFDH	TT	normal
RS377686388	ETFDH	TT	normal

Test details

Applicable situation

Genetic disease screening test for Glutaric Acidemia (GA) uses 4 polymorphic loci on the *ETFA* and *ETFDH* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Glutaric Acidemia (GA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Glutaric Acidemia (GA), or if you feel that you have a risk of developing Glutaric Acidemia (GA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Glutaric Acidemia (GA), such as rs119458971, rs377656387 and rs377686388.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Glutaric Acidemia (GA).

Mandibuloacral Dysplasia with Type A Lipodystrophy (MADA)

It is a rare genetic disorder that affects bone development and fat distribution in the body.

My Mandibuloacral Dysplasia with Type A Lipodystrophy (MADA)

Non-carrier

Knowledge



Time of onset

It is typically diagnosed in childhood, but symptoms may begin to appear in utero or infancy.



Prevalence

It is a rare disease, with fewer than 100 cases reported worldwide.



Typical symptoms

Typical symptoms include short stature, craniofacial abnormalities, joint stiffness, muscle weakness, and loss of body fat particularly in the face, arms and legs. Patients may also be at increased risk for developing metabolic disorders such as diabetes and high cholesterol.



Treatment

There is currently no cure. Treatment is focused on managing symptoms and improving quality of life. This may include physical therapy to improve joint mobility and muscle strength, surgery to correct craniofacial abnormalities, and medications to treat metabolic disorders.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267607554	LMNA	CC	normal

RS386134243	LMNA	CC	normal
RS483352811	LMNA	GG	normal
RS61195471	LMNA	GG	normal
RS267607552	intergenic	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Mandibuloacral Dysplasia with Type A Lipodystrophy (MADA) uses 6 polymorphic loci on the *LMNA* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Mandibuloacral Dysplasia with Type A Lipodystrophy (MADA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Mandibuloacral Dysplasia with Type A Lipodystrophy (MADA), or if you feel that you have a risk of developing Mandibuloacral Dysplasia with Type A Lipodystrophy (MADA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 6 polymorphic loci associated with the risk of Mandibuloacral Dysplasia with Type A Lipodystrophy (MADA), such as rs267607552, rs267607554 and rs267607555.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Mandibuloacral Dysplasia with Type A Lipodystrophy (MADA).

Chediak-Higashi Syndrome (CHS)

It is a rare autosomal recessive genetic disease that affects multiple body systems. It is caused by the defect of lysosomal transport regulating protein, resulting in abnormal function of immune cells and melanocytes such as natural killer cell, neutrophils, lymphocytes, etc.

My Chediak-Higashi Syndrome (CHS)

Non-carrier

Knowledge



Time of onset

Symptoms of this syndrome usually appear in early childhood, typically around 1-4 years of age.



Prevalence

The prevalence of it is extremely low, estimated to be 1 in 1 million individuals worldwide.



Typical symptoms

The of this syndrome include recurrent infections, oculocutaneous albinism (pale skin, light hair, and light colored eyes), and easy bruising and bleeding.



Treatment

Treatment for it is mainly supportive and based on the symptoms observed. This may include antibiotics for recurrent infections and blood transfusions for bleeding disorders. Stem cell transplantation may also be an option for some patients.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS28942077	LYST	AA	normal

RS80338642	intergenic	DD	normal
RS80338643	LYST	GG	normal
RS80338644	intergenic	II	normal
RS80338652	LYST	GG	normal

Test details

Applicable situation

Genetic disease screening test for Chediak-Higashi Syndrome (CHS) uses 5 polymorphic loci on the *LYST* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Chediak-Higashi Syndrome (CHS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Chediak-Higashi Syndrome (CHS), or if you feel that you have a risk of developing Chediak-Higashi Syndrome (CHS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Chediak-Higashi Syndrome (CHS), such as rs28942077, rs80338642 and rs80338643.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Chediak-Higashi Syndrome (CHS).

Congenital Bile Acid Synthesis Defect (CBAS)

It is a rare genetic disorder that affects the production of bile acids, which are essential for the digestion and absorption of fats in the body.

My Congenital Bile Acid Synthesis Defect (CBAS)

Non-carrier

Knowledge



Time of onset

It is a congenital disorder, which means it is present at birth.



Prevalence

The prevalence of it is rare, affecting fewer than 1 in 1,000,000 individuals globally.



Typical symptoms

The typical symptoms of this disease include liver disease, failure to thrive, and developmental delay in infants. In older children and adults, symptoms may include delayed growth, osteoporosis, and severe pruritus (itching).



Treatment

Treatment for this disease aims to manage symptoms and prevent complications. This may include medications to manage pruritus, fat-soluble vitamin supplementation, and managing malabsorption through dietary changes. In severe cases, a liver transplant may be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121917814	AMACR	AA	normal
RS267606650	AKR1D1	CC	normal

RS387906288	HSD3B7	GG	normal
RS182820353	AKR1D1	GG	normal

Test details

Applicable situation

Genetic disease screening test for Congenital Bile Acid Synthesis Defect (CBAS) uses 4 polymorphic loci on the *AMACR*, *AKR1D1* and *HSD3B7* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Congenital Bile Acid Synthesis Defect (CBAS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Congenital Bile Acid Synthesis Defect (CBAS), or if you feel that you have a risk of developing Congenital Bile Acid Synthesis Defect (CBAS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Congenital Bile Acid Synthesis Defect (CBAS), such as rs121917814, rs182820353 and rs267606650.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Congenital Bile Acid Synthesis Defect (CBAS).

Arthrogryposis Multiplex Congenita (AMC)

It is a rare group of congenital diseases characterized by multiple joint contractures (stiffness) and involving systemic muscle weakness at birth. This disease should be seen as a complex symptom or descriptive term, rather than a disease, and potential causes should be sought where possible. The most common type of joint collapse is called amyloidosis, which affects both limbs and joints.

My Arthrogryposis Multiplex Congenita (AMC)

Non-carrier

Knowledge

Time of onset

The onset of symptoms can occur at birth or during pregnancy through ultrasound detection.

Prevalence

This is a rare disorder occurring in 1 out of every 3,000 live births

Typical symptoms

The typical symptoms of this disease include internal shoulder rotation; elbow extension (sometimes flexed) and forearm pronation; wrist flexion and ulnar deviation; fingers in fixed flexion and thumb-in-palm deformity; hip flexed, abducted, externally rotated, and often dislocated; knee flexion or extension; and foot deformities (clubfoot and/or vertical talus). Typically all four extremities are involved, but there are some with just upper or lower involvement. Other, more uncommon problems include scoliosis, respiratory issues, facial and jaw variations and abdominal hernias.

Treatment

There is no absolute cure for this situation. Physical and functional therapies may include stretching, casting, strengthening, activity training, and solving fine motor skills to improve range of motion and cultivate independence.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894311	TNNI2	GG	normal
RS104894312	TNNI2	CC	normal
RS121913623	MYH3	CC	normal
RS1555769818	intergenic	GG	normal
RS1555788577	intergenic	TT	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Arthrogryposis Multiplex Congenita (AMC) uses 8 polymorphic loci on genes such as *TNNI2*, *MYH3* and *RYR1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Arthrogryposis Multiplex Congenita (AMC).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.

4. If you have a family history of Arthrogryposis Multiplex Congenita (AMC), or if you feel that you have a risk of developing Arthrogryposis Multiplex Congenita (AMC), it is recommended that you consult a qualified health professional or clinician for help.

 Test content

This test includes 8 polymorphic loci associated with the risk of Arthrogryposis Multiplex Congenita (AMC), such as rs104894311, rs104894312 and rs121913623.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Arthrogryposis Multiplex Congenita (AMC).

Coenzyme Q10 Deficiency, Primary (COQ10D)

It is a rare autosomal recessive mitochondrial disorder caused by mutations in genes involved in the biosynthesis of coenzyme Q10. CoQ10 is a vital component of the electron transport chain involved in generating ATP, the energy currency of the cell. Therefore, the deficiency results in impaired energy production affecting various organs leading to a range of symptoms.

My Coenzyme Q10 Deficiency, Primary (COQ10D)

Non-carrier

Knowledge

Time of onset

Symptoms of this disease typically appear in childhood, although onset can range from infancy through adulthood.

Prevalence

The incidence of it is unknown, and there is limited data on the prevalence of the disease.

Typical symptoms

Common symptoms of this disease include muscle weakness, recurrent episodes of myoglobinuria, exercise intolerance, encephalopathy, seizures, ataxia, hearing impairment, neuropathy, hypertrophic cardiomyopathy, renal disease and liver disease. The severity of symptoms can vary widely among affected individuals regardless of age at onset.

Treatment

Treatment options for this disease include supplementation of CoQ10, vitamin C, vitamin E and antioxidants. It is important to tailor therapy to each individual's needs and monitor its effectiveness since there is little evidence supporting the use of any specific therapy for this disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918231	COQ2	CC	normal

Test details

Applicable situation

Genetic disease screening test for Coenzyme Q10 Deficiency, Primary (COQ10D) uses 1 polymorphic locus on the *COQ2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Coenzyme Q10 Deficiency, Primary (COQ10D).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Coenzyme Q10 Deficiency, Primary (COQ10D), or if you feel that you have a risk of developing Coenzyme Q10 Deficiency, Primary (COQ10D), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Coenzyme Q10 Deficiency, Primary (COQ10D), namely rs121918231.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Coenzyme Q10 Deficiency, Primary (COQ10D).

Congenital Dyserythropoietic Anemia (CDA)

It is a rare group of genetic disorders that affects the development of red blood cells. It is characterized by inadequate production and abnormal maturation of red blood cells, leading to anemia.

My Congenital Dyserythropoietic Anemia (CDA)

Non-carrier

Knowledge



Time of onset

It is usually present at birth or diagnosed during childhood.



Prevalence

The prevalence of it is unknown because it is a rare disease.



Typical symptoms

Typical symptoms of this disease include pale skin, fatigue, weakness, and shortness of breath. Other symptoms may include jaundice, enlarged spleen, and yellowing of the skin and eyes.



Treatment

Treatment for this disease include blood transfusions, iron chelation therapy, bone marrow transplantation, and erythropoietin therapy. Supportive measures such as folic acid and vitamin supplementation are also important.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS120074167	CDAN1	GG	normal
RS121918221	SEC23B	GG	normal

RS121918223	SEC23B	CC	normal
RS398124225	SEC23B	CC	normal
RS199939108	SEC23B	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Congenital Dyserythropoietic Anemia (CDA) uses 8 polymorphic loci on the *CDAN1* and *SEC23B* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Congenital Dyserythropoietic Anemia (CDA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Congenital Dyserythropoietic Anemia (CDA), or if you feel that you have a risk of developing Congenital Dyserythropoietic Anemia (CDA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 8 polymorphic loci associated with the risk of Congenital Dyserythropoietic Anemia (CDA), such as rs120074167, rs121918221 and rs121918223.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Congenital Dyserythropoietic Anemia (CDA).

Dyskeratosis Congenita (DC)

It is a rare genetic disorder that affects multiple organs and systems in the body. It is primarily characterized by bone marrow failure, changes in skin pigmentation, and abnormalities in nails.

My Dyskeratosis Congenita (DC)

Non-carrier

Knowledge



Time of onset

Symptoms of this disease may appear at any time from infancy to adulthood, although they most commonly appear around the age of 10.



Prevalence

It is an extremely rare disease, affecting approximately 1 in 1 million people worldwide.



Typical symptoms

The typical symptoms of this disease include bone marrow failure, changes in skin pigmentation, abnormal nails, and an increased risk of cancer.



Treatment

Treatment for it is supportive and aimed at managing symptoms. This may include regular blood transfusions, bone marrow transplants, and preventative cancer screenings.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121912288	DKC1	CC	normal
RS199473679	intergenic	II	normal

RS863225129	intergenic	GG	normal
RS776744306	intergenic	GG	normal
RS1555899932	intergenic	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Dyskeratosis Congenita (DC) uses 20 polymorphic loci on genes such as *DKC1*, *TINF2* and *TERT*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Dyskeratosis Congenita (DC).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Dyskeratosis Congenita (DC), or if you feel that you have a risk of developing Dyskeratosis Congenita (DC), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 20 polymorphic loci associated with the risk of Dyskeratosis Congenita (DC), such as rs121912288, rs121912289 and rs121912293.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Dyskeratosis Congenita (DC).

Congenital Stationary Night Blindness (CSNB)

It is a rare genetic disorder that affects the eyes and causes difficulty in seeing in low light conditions, such as at night or in dimly lit environments.

My Congenital Stationary Night Blindness (CSNB)

Non-carrier

Knowledge



Time of onset

It is usually present at birth, but may not be diagnosed until later in childhood or adulthood.



Prevalence

The prevalence of it is unknown, but it is estimated to affect fewer than 1 in 100,000 individuals.



Typical symptoms

Typical symptoms of this disease include difficulty seeing in low light, abnormal eye movements, and reduced visual acuity.



Treatment

There is currently no known cure for this disease, and treatment mainly focuses on managing symptoms. This may include using low vision aids, such as night vision goggles, and avoiding situations with poor lighting.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS387906862	TRPM1	GG	normal
RS180869804	TRPM1	GG	normal

RS778390089

intergenic

II

normal

Test details

Applicable situation

Genetic disease screening test for Congenital Stationary Night Blindness (CSNB) uses 3 polymorphic loci on the *TRPM1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Congenital Stationary Night Blindness (CSNB).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Congenital Stationary Night Blindness (CSNB), or if you feel that you have a risk of developing Congenital Stationary Night Blindness (CSNB), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Congenital Stationary Night Blindness (CSNB), such as rs180869804, rs387906862 and rs778390089.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Congenital Stationary Night Blindness (CSNB).

Congenital Disorder of Deglycosylation (CDDG)

It is a rare genetic disorder that affects the ability of cells to remove sugar molecules from proteins. This process, called deglycosylation, is important for proper protein function and stability in the body.

My Congenital Disorder of Deglycosylation (CDDG)

Non-carrier

Knowledge



Time of onset

It is typically diagnosed in infancy or early childhood.



Prevalence

It is a rare disorder, with only a few hundred cases reported worldwide.



Typical symptoms

Common symptoms of this disease include delayed development, intellectual disability, movement disorders, seizures, abnormal muscle tone, and difficulty with vision and hearing.



Treatment

There is currently no specific treatment for this disease, and management is focused on addressing individual symptoms and complications. This may include therapies such as physical and occupational therapy, medications to manage seizures or movement disorders, and interventions to address feeding difficulties.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS201337954	NGLY1	TT	normal

RS587776982	intergenic	II	normal
RS587777265	NGLY1	DD	normal
RS146140738	NGLY1	GG	normal

Test details

Applicable situation

Genetic disease screening test for Congenital Disorder of Deglycosylation (CDDG) uses 4 polymorphic loci on the *NGLY1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Congenital Disorder of Deglycosylation (CDDG).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Congenital Disorder of Deglycosylation (CDDG), or if you feel that you have a risk of developing Congenital Disorder of Deglycosylation (CDDG), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Congenital Disorder of Deglycosylation (CDDG), such as rs146140738, rs201337954 and rs587776982.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Congenital Disorder of Deglycosylation (CDDG).

Congenital Generalized Lipodystrophy (CGL)

It is a rare genetic disorder that affects fat distribution in the body. It is characterized by the lack of fat tissue beneath the skin and around internal organs, resulting in a muscular appearance and abnormalities in glucose and lipid metabolism.

My Congenital Generalized Lipodystrophy (CGL)

Non-carrier

Knowledge



Time of onset

Symptoms of the disease can present at birth or in early childhood.



Prevalence

The prevalence of it is estimated to be fewer than 1 in 10 million individuals globally.



Typical symptoms

The most include muscular build, loss of fat tissue, insulin resistance and related metabolic disorders, hepatomegaly, and acanthosis nigricans.



Treatment

Treatment for it is largely supportive, with a focus on managing the metabolic abnormalities associated with the disease through lifestyle modifications and medication. Patients may require insulin therapy and lipid-lowering medication to manage their symptoms and reduce their risk of complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS116807569	AGPAT2	TT	normal

RS137852975	BSCL2	CC	normal
RS797045222	AGPAT2	CC	normal

Test details

Applicable situation

Genetic disease screening test for Congenital Generalized Lipodystrophy (CGL) uses 3 polymorphic loci on the *AGPAT2* and *BSCL2* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Congenital Generalized Lipodystrophy (CGL).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Congenital Generalized Lipodystrophy (CGL), or if you feel that you have a risk of developing Congenital Generalized Lipodystrophy (CGL), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Congenital Generalized Lipodystrophy (CGL), such as rs116807569, rs137852975 and rs797045222.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Congenital Generalized Lipodystrophy (CGL).

Congenital Disorder of Glycosylation (CDG)

It is a group of rare genetic disorders caused by defects in the process of attaching sugar molecules to proteins or lipids, resulting in abnormal glycosylation. This can cause a range of symptoms affecting various systems in the body.

My Congenital Disorder of Glycosylation (CDG)

Non-carrier

Knowledge

Time of onset

It is a congenital disorder, meaning that it is present from birth, although symptoms may not always be evident immediately.

Prevalence

It is a rare disease with an estimated prevalence of 1 in 100,000 individuals worldwide.

Typical symptoms

Symptoms of this disease can vary widely and can affect multiple systems in the body. Common symptoms include developmental delay, failure to thrive, seizures, abnormal liver function, and skeletal abnormalities.

Treatment

There is currently no cure for this disease, and treatment is primarily supportive and focused on managing symptoms. This may include physical therapy, nutritional support, and management of seizures and other medical complications. In some cases, therapies such as enzyme replacement or gene therapy may be investigated.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS104894533	PMM2	TT	normal
RS121907933	ALG12	CC	normal
RS200503569	PMM2	CC	normal
RS398124401	SRD5A3	GG	normal
RS587777323	MOGS	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Congenital Disorder of Glycosylation (CDG) uses 12 polymorphic loci on genes such as *PMM2*, *ALG12* and *ALG1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Congenital Disorder of Glycosylation (CDG).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Congenital Disorder of Glycosylation (CDG), or if you feel that you have a risk of developing Congenital Disorder of Glycosylation (CDG), it is recommended that you

consult a qualified health professional or clinician for help.

 Test content

This test includes 12 polymorphic loci associated with the risk of Congenital Disorder of Glycosylation (CDG), such as rs104894533, rs121907933 and rs139716296.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Congenital Disorder of Glycosylation (CDG).

Congenital Amegakaryocytic Thrombocytopenia (CAMT)

It is a rare genetic disorder that affects the production of platelets in the bone marrow. Platelets are blood cells that help in clotting, and individuals with this disease have a reduced number of platelets, leading to an increased risk of bleeding and bruising.

My Congenital Amegakaryocytic Thrombocytopenia (CAMT)

Non-carrier

Knowledge

Time of onset

It is usually diagnosed in infants or young children, with symptoms appearing in the first few months of life.

Prevalence

It is an extremely rare disease, with an estimated incidence of 1 in 1 million live births.

Typical symptoms

The typical symptoms of this disease include excessive bleeding, easy bruising, and petechiae (red or purple spots on the skin caused by bleeding under the surface). Individuals with this disease may also experience recurrent infections, delayed growth and development, and skeletal abnormalities. They may also have an enlarged spleen and liver.

Treatment

The main treatment for it is bone marrow transplant, which can restore platelet production. Supportive treatments such as blood transfusions and antibiotics may also be used to manage symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121913611	MPL	CC	normal
RS148434485	MPL	CC	normal
RS587778515	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Congenital Amegakaryocytic Thrombocytopenia (CAMT) uses 3 polymorphic loci on the *MPL* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Congenital Amegakaryocytic Thrombocytopenia (CAMT).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Congenital Amegakaryocytic Thrombocytopenia (CAMT), or if you feel that you have a risk of developing Congenital Amegakaryocytic Thrombocytopenia (CAMT), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Congenital Amegakaryocytic Thrombocytopenia (CAMT), such as rs121913611, rs148434485 and rs587778515.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Congenital Amegakaryocytic Thrombocytopenia (CAMT).

Congenital Fibrosis of Extraocular Muscles (CFEOM)

It is a rare genetic disorder characterized by paralysis or weakness of the eye muscles, resulting in restricted eye movement, misalignment of the eyes, and poor vision.

My Congenital Fibrosis of Extraocular Muscles (CFEOM)

Non-carrier

Knowledge

Time of onset

Symptoms of it usually become apparent in infancy or early childhood.

Prevalence

The prevalence of it is unknown, but it is considered very rare.

Typical symptoms

The most common symptoms of it include limited eye movement, abnormal eye positioning, double vision, and poor vision.

Treatment

Treatment for it may include surgery to correct the position of the eyes, glasses or contact lenses to correct vision problems, and physical therapy to improve muscle strength and coordination. However, there is no cure for it and treatment varies depending on the presenting symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121912585	KIF21A	GG	normal

RS267607162	TUBB3	CC	normal
RS864321715	TUBB3	GG	normal
RS864321717	TUBB3	CC	normal

Test details

Applicable situation

Genetic disease screening test for Congenital Fibrosis of Extraocular Muscles (CFEOM) uses 4 polymorphic loci on the *KIF21A* and *TUBB3* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Congenital Fibrosis of Extraocular Muscles (CFEOM).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Congenital Fibrosis of Extraocular Muscles (CFEOM), or if you feel that you have a risk of developing Congenital Fibrosis of Extraocular Muscles (CFEOM), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Congenital Fibrosis of Extraocular Muscles (CFEOM), such as rs121912585, rs267607162 and rs864321715.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Congenital Fibrosis of Extraocular Muscles (CFEOM).

Congenital Ichthyosis

It is a rare genetic disorder that affects the skin. It results in abnormal scaling and thickening of the skin, which can cause discomfort, pain, and itching. The severity of symptoms can vary widely among individuals, ranging from mild to severe.

My Congenital Ichthyosis
Non-carrier

Knowledge



Time of onset

Symptoms of this disease present at birth or shortly thereafter.



Prevalence

It is a rare disease, affecting approximately one in 200,000 to one in 300,000 individuals worldwide.



Typical symptoms

The typical symptoms of this disease include dry, scaly skin, which is thick and hard. Cracking and bleeding increase the risk of infection. And it can lose fluids and electrolytes through the skin, which may lead to dehydration and body salt imbalance. At the same time, it may be difficult to maintain body temperature and there may be growth and developmental delays. Patients may also experience hair loss and nail deformities.



Treatment

Treatment for this disease include the use of topical creams, ointments, and emollients to hydrate and soften the skin. In severe cases, oral medications may be prescribed to reduce inflammation and improve skin texture. Genetic counseling may also be advised for affected individuals and their families.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS137853289	ABCA12	GG	normal
RS143473912	TGM1	CC	normal
RS199422216	NIPAL4	CC	normal
RS28940568	ABCA12	CC	normal
RS387906284	ABCA12	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Congenital Ichthyosis uses 9 polymorphic loci on genes such as *ABCA12*, *TGM1* and *NIPAL4*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Congenital Ichthyosis.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Congenital Ichthyosis, or if you feel that you have a risk of developing Congenital Ichthyosis, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 9 polymorphic loci associated with the risk of Congenital Ichthyosis, such as rs137853289, rs139208806 and rs143473912.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Congenital Ichthyosis.

Acyl-Coenzyme A Dehydrogenase Deficiency

It is a rare metabolic disease that impairs the body's ability to decompose fatty acids into acetyl CoA, leading to lipid oxidation disorders. This disease occurs during prolonged fasting and other conditions, and if not intervened in a timely manner, it may lead to hypoglycemia and sudden death.

My Acyl-Coenzyme A Dehydrogenase Deficiency

Non-carrier

Knowledge

Time of onset

Symptoms of this disease can manifest in infancy, childhood, or adulthood depending on the severity of the mutation.

Prevalence

It is a rare disease and the exact prevalence is unknown, but it is estimated to occur in 1 in 25,000 to 1 in 40,000 births.

Typical symptoms

Symptoms of the disease vary from mild to severe and may include burnout, loss of appetite, vomiting, hypoglycemia, muscle weakness, seizures, and coma. Sometimes it leads to sudden death. The disease may also cause heart and liver problems.

Treatment

Treatment for this disease includes dietary modifications, such as consuming a low-fat, high-carbohydrate diet. supplementation with carnitine and medium-chain triglycerides may also be beneficial. In severe cases, hospitalization and intravenous glucose may be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121434275	ACADM	TT	normal
RS758753966	intergenic	AA	normal
RS796051901	intergenic	II	normal
RS786204566	ACADM	DD	normal
RS779759347	ACADM	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Acyl-Coenzyme A Dehydrogenase Deficiency uses 19 polymorphic loci on the *ACADM* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Acyl-Coenzyme A Dehydrogenase Deficiency.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Acyl-Coenzyme A Dehydrogenase Deficiency, or if you feel that you have a risk of developing Acyl-Coenzyme A Dehydrogenase Deficiency, it is recommended that

you consult a qualified health professional or clinician for help.

 Test content

This test includes 19 polymorphic loci associated with the risk of Acyl-Coenzyme A Dehydrogenase Deficiency, such as rs121434275, rs121434277 and rs121434278.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Acyl-Coenzyme A Dehydrogenase Deficiency.

Sialidosis

It is a hereditary lysosomal storage disorder caused by a deficiency of sialidase. Lack of this enzyme will lead to abnormal accumulation of complex carbohydrates called glycosaminoglycan and fatty substances called mucolipids and damage various tissues and organs, leading to a series of symptoms.

My Sialidosis

Non-carrier

Knowledge

Time of onset

This disease can occur at any age, but symptoms typically first appear in early childhood.

Prevalence

It is very rare, with fewer than 100 cases reported worldwide.

Typical symptoms

Typical symptoms include developmental delays, hearing loss, vision problems, and problems with movement and coordination.

Treatment

Treatment mainly involves improving symptoms and providing support, for example, medications, surgery, physical therapy and occupational therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893972	NEU1	AA	normal
RS104893983	NEU1	CC	normal

RS28940583

NEU1

CC

normal

Test details

Applicable situation

Genetic disease screening test for Sialidosis uses 3 polymorphic loci on the *NEU1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Sialidosis.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Sialidosis, or if you feel that you have a risk of developing Sialidosis, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Sialidosis, such as rs104893972, rs104893983 and rs28940583.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Sialidosis.

Mitochondrial DNA Depletion Syndrome (MDS)

It is a rare genetic disorder that affects the mitochondrial DNA responsible for producing energy in the body. This condition causes a significant reduction in the amount of mitochondrial DNA leading to a shortage of energy supply, particularly in organs with high energy demand like the brain, liver and muscles.

My Mitochondrial DNA Depletion Syndrome (MDS)

Non-carrier

Knowledge



Time of onset

The onset varies depending on the specific subtype and severity of the condition. Some infants may show symptoms at birth, while some individuals may develop symptoms later in life.



Prevalence

It is estimated to affect fewer than 1 in 100,000 globally.



Typical symptoms

Typical symptoms include muscle weakness, poor muscle tone, developmental delays, failure to thrive, seizures, liver failure and anemia in some cases.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support, including nutritional support, physical therapy and medications. In some cases, liver or kidney transplantation may be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121909721	MPV17	CC	normal

RS587776944	intergenic	AA	normal
RS886041081	SLC25A4	GG	normal
RS869312995	intergenic	TT	normal
RS863224914	RRM2B	DD	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Mitochondrial DNA Depletion Syndrome (MDS) uses 30 polymorphic loci on genes such as *MPV17*, *TYMP* and *POLG*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Mitochondrial DNA Depletion Syndrome (MDS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Mitochondrial DNA Depletion Syndrome (MDS), or if you feel that you have a risk of developing Mitochondrial DNA Depletion Syndrome (MDS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 30 polymorphic loci associated with the risk of Mitochondrial DNA Depletion Syndrome (MDS), such as rs121909721, rs121909723 and rs121913037.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Mitochondrial DNA Depletion Syndrome (MDS).

Mitochondrial Complex II Deficiency (MC2D)

It is a rare genetic disorder that affects the production of energy in cells. It occurs when there is a defect in one of the protein complexes that make up the mitochondrial respiratory chain. This results in a decrease in the amount of energy produced, leading to a range of symptoms.

My Mitochondrial Complex II Deficiency (MC2D)

Non-carrier

Knowledge



Time of onset

The onset can vary, but it is usually present from birth or infancy.



Prevalence

It is estimated to affect 1 in 67,000 people.



Typical symptoms

Typical symptoms include muscle weakness, delayed development, seizures, vision and hearing loss, abnormal eye movements and exercise intolerance. Patients may also experience cardiovascular and neurological abnormalities, such as cardiomyopathy, ataxia, and neuropathy.



Treatment

Treatment is focused on managing the symptoms and providing supportive care. This may include physical therapy, speech therapy and other forms of rehabilitation. Some individuals may benefit from medications, such as anti-seizure drugs or coenzyme Q10 supplements.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS151170408	SDHA	CC	normal

RS202198133	intergenic	GG	normal
RS587782077	intergenic	II	normal
RS748089700	SDHA	CC	normal
RS766667009	intergenic	GG	normal

Test details

Applicable situation

Genetic disease screening test for Mitochondrial Complex II Deficiency (MC2D) uses 5 polymorphic loci on the *SDHA* and *SDHD* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Mitochondrial Complex II Deficiency (MC2D).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Mitochondrial Complex II Deficiency (MC2D), or if you feel that you have a risk of developing Mitochondrial Complex II Deficiency (MC2D), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Mitochondrial Complex II Deficiency (MC2D), such as rs151170408, rs202198133 and rs587782077.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Mitochondrial Complex II Deficiency (MC2D).

Microcephalic Osteodysplastic Primordial Dwarfism (MOPD)

It is a rare genetic disorder that leads to severe growth retardation and skeletal abnormalities.

My Microcephalic Osteodysplastic Primordial Dwarfism (MOPD)

Non-carrier

Knowledge

Time of onset

Symptoms of the disease are present at birth or become apparent in early infancy.

Prevalence

The prevalence of the disease is currently unknown, but it is considered extremely rare.

Typical symptoms

Typical symptoms include microcephaly, dwarfism, skeletal abnormalities such as shortening or overgrowth of bones, and developmental delays.

Treatment

There is no cure for this disease. Treatment may include surgical procedures to correct skeletal abnormalities, hearing and vision aids, and physical therapy to improve bone strength and mobility.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS119479063	PCNT	GG	normal
RS181690344	PCNT	CC	normal

RS151020551	PCNT	CC	normal
RS587779355	PCNT	GG	normal
RS587784308	PCNT	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Microcephalic Osteodysplastic Primordial Dwarfism (MOPD) uses 9 polymorphic loci on the *PCNT* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Microcephalic Osteodysplastic Primordial Dwarfism (MOPD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Microcephalic Osteodysplastic Primordial Dwarfism (MOPD), or if you feel that you have a risk of developing Microcephalic Osteodysplastic Primordial Dwarfism (MOPD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 9 polymorphic loci associated with the risk of Microcephalic Osteodysplastic Primordial Dwarfism (MOPD), such as rs119479063, rs151020551 and rs181690344.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Microcephalic Osteodysplastic Primordial Dwarfism (MOPD).

Microcephaly-Capillary Malformation Syndrome (MICCAP)

It is an genetic disorder characterized by an abnormally small head size and abnormalities of small blood vessels in the skin called capillaries (capillary malformations).

My Microcephaly-Capillary Malformation Syndrome (MICCAP)

Non-carrier

Knowledge



Time of onset

It typically appears in early childhood.



Prevalence

The prevalence of this disease is currently unknown.



Typical symptoms

Typical symptoms include an abnormally small head, malformations of the blood vessels, and developmental delays.



Treatment

Treatment may include physical therapy, speech therapy, and occupational therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS397509390	STAMPB	CC	normal
RS797046015	STAMPB	TT	normal

Test details

Applicable situation

Genetic disease screening test for Microcephaly-Capillary Malformation Syndrome (MICCAP) uses 2 polymorphic loci on the *STAMBP* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Microcephaly-Capillary Malformation Syndrome (MICCAP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Microcephaly-Capillary Malformation Syndrome (MICCAP), or if you feel that you have a risk of developing Microcephaly-Capillary Malformation Syndrome (MICCAP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Microcephaly-Capillary Malformation Syndrome (MICCAP), such as rs397509390 and rs797046015.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Microcephaly-Capillary Malformation Syndrome (MICCAP).

Cardiofaciocutaneous Syndrome (CFC)

It is a rare genetic disorder that affects multiple systems, including the heart, face, and skin. It is caused by mutations in genes that regulate a cell signaling pathway known as the MAPK pathway.

My Cardiofaciocutaneous Syndrome (CFC)

Non-carrier

Knowledge



Time of onset

Symptoms of this disease are usually present from birth or become apparent during infancy.



Prevalence

It is an extremely rare disorder, with an estimated incidence of fewer than 200 cases worldwide.



Typical symptoms

Typical symptoms of this disease include craniofacial abnormalities such as a prominent forehead, widely spaced eyes, and a small chin, heart defects, skin abnormalities, growth and developmental delays, and intellectual disability.



Treatment

Treatment for it is largely supportive and may include medications to manage symptoms such as seizures, heart defects, or gastrointestinal issues, as well as physical, occupational, and speech therapy to support development and improve quality of life.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894361	KRAS	TT	normal

RS397507483	BRAF	CC	normal
RS797044593	intergenic	AA	normal
RS794729219	BRAF	AA	normal
RS730880517	MAP2K2	TT	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Cardiofaciocutaneous Syndrome (CFC) uses 23 polymorphic loci on genes such as *KRAS*, *MAP2K1* and *MAP2K2*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Cardiofaciocutaneous Syndrome (CFC).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Cardiofaciocutaneous Syndrome (CFC), or if you feel that you have a risk of developing Cardiofaciocutaneous Syndrome (CFC), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 23 polymorphic loci associated with the risk of Cardiofaciocutaneous Syndrome (CFC), such as rs104894361, rs104894362 and rs1057519732.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Cardiofaciocutaneous Syndrome (CFC).

Selective Tooth Agenesis (STA)

It is a genetic condition that causes one or more teeth to be missing in the mouth.
It affects both baby teeth and permanent teeth and can affect any tooth in the mouth.

My Selective Tooth Agenesis (STA)

Non-carrier

Knowledge

Time of onset

It typically appears in childhood.

Prevalence

** The prevalence of Tooth Agenesis, Selective varies depending on the population, but it is estimated to affect around 1-2% of the population.

Typical symptoms

The most common symptom is a missing tooth or teeth in the mouth. Other symptoms may include abnormal spacing or gaps between teeth, a smaller than average jawline, abnormal bite patterns.

Treatment

Treatment aims to restore the function and appearance of the missing teeth using dental implants, bridges or dentures. Orthodontic treatment may also be necessary to correct bite problems.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS132630308	intergenic	TT	normal
RS132630314	EDA	GG	normal

Test details

Applicable situation

Genetic disease screening test for Selective Tooth Agenesis (STA) uses 2 polymorphic loci on the *EDA* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Selective Tooth Agenesis (STA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Selective Tooth Agenesis (STA), or if you feel that you have a risk of developing Selective Tooth Agenesis (STA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Selective Tooth Agenesis (STA), such as rs132630308 and rs132630314.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Selective Tooth Agenesis (STA).

Hemochromatosis

It is a genetic disorder that causes the body to absorb and store too much iron, leading to a build-up of iron in organs such as the liver, heart, and pancreas. Over time, this excess iron can cause serious damage to these organs and potentially lead to life-threatening conditions such as liver cancer, heart failure, and diabetes.

My Hemochromatosis

Non-carrier

Knowledge

Time of onset

The for it varies, but the symptoms typically appear between the ages of 30 and 50.

Prevalence

Type 1 hemochromatosis is one of the most common genetic disorders in the United States, affecting about 1 million people. It most often affects people of Northern European descent. The other types of hemochromatosis are considered rare and have been studied in only a small number of families worldwide.

Typical symptoms

Common symptoms of it include fatigue, joint pain, abdominal pain, and loss of sex drive.

Treatment

The treatment for it involves regular phlebotomy (blood removal) to reduce the body's iron levels, as well as maintaining a low-iron diet and avoiding alcohol. In some cases, medications may be used to help lower iron levels.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS387907377	SLC40A1	CC	normal
RS786204108	TFR2	CC	normal

Test details

Applicable situation

Genetic disease screening test for Hemochromatosis uses 2 polymorphic loci on the *SLC40A1* and *TFR2* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hemochromatosis.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hemochromatosis, or if you feel that you have a risk of developing Hemochromatosis, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Hemochromatosis, such as rs387907377 and rs786204108.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hemochromatosis.

Thrombocytopenia

It is a condition in which an individual has a low platelet count in their bloodstream. Platelets are essential for blood clotting, and a deficiency in these cells can cause excessive bleeding.

My Thrombocytopenia

Non-carrier

Knowledge



Time of onset

It can occur at any age and can vary depending on the individual.



Prevalence

It is a relatively uncommon condition, with an estimated 3 million cases per year in the United States.



Typical symptoms

Typical symptoms include fatigue, easy bruising, nosebleeds, and hemorrhaging.



Treatment

Treatment may involve medications that stimulate platelet production, platelet transfusions, and in severe cases, surgery to remove the spleen.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS132630270	intergenic	CC	normal
RS28933386	PTPN11	AA	normal
RS61751286	VWF	GG	normal

RS867593888

intergenic

TT

normal

Test details

Applicable situation

Genetic disease screening test for Thrombocytopenia uses 4 polymorphic loci on genes such as *WAS*, *PTPN11* and *VWF*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Thrombocytopenia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Thrombocytopenia, or if you feel that you have a risk of developing Thrombocytopenia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Thrombocytopenia, such as rs132630270, rs28933386 and rs61751286.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Thrombocytopenia.

Fucosidosis

It is a rare genetic disorder that affects the breakdown and use of fucose in the body. It is caused by a deficiency in the enzyme alpha-L-fucosidase, which leads to the buildup of fucose-containing compounds in various tissues and organs.

My Fucosidosis

Non-carrier

Knowledge



Time of onset

The onset time of this disease varies from person to person, with some patients experiencing symptoms during infancy and others occurring later.



Prevalence

It is an extremely rare disease, with fewer than 100 cases reported worldwide.



Typical symptoms

Typical symptoms of it include coarse facial features, thickened skin, enlarged liver and spleen, and difficulty breathing. The disease can also cause a range of neurological symptoms, including hearing loss, vision impairment, and muscle weakness.



Treatment

Treatment for this disease are largely supportive and focus on managing symptoms. Enzyme replacement therapy is also being studied as a potential treatment option.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS794727774	FUCA1	CC	normal

Test details

Applicable situation

Genetic disease screening test for Fucosidosis uses 1 polymorphic locus on the *FUCA1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Fucosidosis.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Fucosidosis, or if you feel that you have a risk of developing Fucosidosis, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Fucosidosis, namely rs794727774.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Fucosidosis.

Fundus Albipunctatus (FA)

It is a rare genetic retinal disease that affects the visual system. It is characterized by small white or yellow dots in the retina, which eventually leads to impaired night vision and decreased visual acuity.

My Fundus Albipunctatus (FA)

Non-carrier

Knowledge

Time of onset

The onset is usually in childhood or adolescence and can be noticed when the person experiences difficulty seeing in the dark.

Prevalence

It is a very rare disease with an estimated prevalence of 1 in 30,000 individuals.

Typical symptoms

The typical symptoms of it include difficulty seeing in low light conditions, impaired night vision, and a decrease in visual acuity.

Treatment

There is no cure for it, but the use of low-vision aids such as magnifying lenses can help improve visual acuity. Additionally, avoiding exposure to bright light can help reduce symptoms such as photophobia.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS62638191	RDH5	GG	normal

RS774122562

RDH5

GG

normal

Test details

Applicable situation

Genetic disease screening test for Fundus Albipunctatus (FA) uses 2 polymorphic loci on the *CD63* and *RDH5* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Fundus Albipunctatus (FA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Fundus Albipunctatus (FA), or if you feel that you have a risk of developing Fundus Albipunctatus (FA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Fundus Albipunctatus (FA), such as rs62638191 and rs774122562.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Fundus Albipunctatus (FA).

Oculocutaneous Albinism (OCA)

It is a genetic disorder that affects the production of melanin. This results in reduced or absent pigmentation in the skin, hair and eyes, and also affects the development and function of the eyes.

My Oculocutaneous Albinism (OCA)

Non-carrier

Knowledge



Time of onset

It is present from birth.



Prevalence

It is estimated to affect 1 in 20,000 individuals worldwide.



Typical symptoms

Typical symptoms include very pale skin, white or light-colored hair and light-colored or blue eyes. Additionally, individuals with this disease may experience vision problems such as reduced visual acuity, nystagmus and photophobia.



Treatment

Treatment mainly involves improving symptoms and providing support, such as wearing protective clothing and sunscreen to protect the skin from sun exposure, wearing tinted glasses to reduce glare, and vision rehabilitation therapy to improve visual acuity.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894316	TYR	GG	normal

RS62645917	TYR	CC	normal
RS797046082	TYR	AA	normal
RS797045970	SLC45A2	AA	normal
RS797045839	OCA2	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Oculocutaneous Albinism (OCA) uses 21 polymorphic loci on genes such as *TYR*, *OCA2* and *TYRP1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Oculocutaneous Albinism (OCA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Oculocutaneous Albinism (OCA), or if you feel that you have a risk of developing Oculocutaneous Albinism (OCA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 21 polymorphic loci associated with the risk of Oculocutaneous Albinism (OCA), such as rs104894316, rs142988897 and rs281865424.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Oculocutaneous Albinism (OCA).

Pancreatic Agenesis (PAGEN)

It is a rare genetic disorder that occurs due to incomplete or absent formation of the pancreas during fetal development. This results in the absence of pancreatic secretion of digestive enzymes and hormones required for controlling blood sugar levels.

My Pancreatic Agenesis (PAGEN)

Non-carrier

Knowledge



Time of onset

It is present at birth.



Prevalence

It is estimated to affect fewer than 1 in 100,000 births.



Typical symptoms

Patients may have difficulty in digesting food and controlling blood sugar levels. This can lead to malnutrition, dehydration and other complications. Symptoms may include vomiting, diarrhea, abdominal pain and low blood sugar levels.



Treatment

Treatment is focused on managing symptoms and maintaining adequate nutrition and blood sugar levels. This may include the use of pancreatic enzyme replacements, insulin injections and specialized infant formulas. Close monitoring by healthcare professionals is necessary to manage potential complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS80356661	intergenic	GG	normal
RS1555241857	intergenic	AA	normal

Test details

Applicable situation

Genetic disease screening test for Pancreatic Agenesis (PAGEN) uses 2 polymorphic loci on the *PDX1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Pancreatic Agenesis (PAGEN).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Pancreatic Agenesis (PAGEN), or if you feel that you have a risk of developing Pancreatic Agenesis (PAGEN), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Pancreatic Agenesis (PAGEN), such as rs1555241857 and rs80356661.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Pancreatic Agenesis (PAGEN).

Hereditary Sensory and Autonomic Neuropathy (HSAN)

It is a group of genetic conditions that affects the nerves responsible for detecting sensations, controlling automatic body functions, and regulating blood pressure and heart rate.

My Hereditary Sensory and Autonomic Neuropathy (HSAN)

Non-carrier

Knowledge



Time of onset

Symptoms of it can appear at any age, though the majority of cases present in childhood or adolescence.



Prevalence

The prevalence of it is rare, with estimated incidence rates ranging from 1 in 25,000 to 1 in 500,000 individuals worldwide.



Typical symptoms

Symptoms of it may vary between individuals, but commonly include loss of sensation in the limbs, inability to detect temperature changes, reduced sweating, lack of reflexes, and decreased pain perception.



Treatment

There is currently no cure for it, and treatment is primarily based on managing symptoms. This may include medications for pain management, physical and occupational therapy, and the use of mobility devices.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS797045071	SPTLC1	CC	normal

Test details

Applicable situation

Genetic disease screening test for Hereditary Sensory and Autonomic Neuropathy (HSAN) uses 1 polymorphic locus on the *SPTLC1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hereditary Sensory and Autonomic Neuropathy (HSAN).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hereditary Sensory and Autonomic Neuropathy (HSAN), or if you feel that you have a risk of developing Hereditary Sensory and Autonomic Neuropathy (HSAN), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Hereditary Sensory and Autonomic Neuropathy (HSAN), namely rs797045071.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hereditary Sensory and Autonomic Neuropathy (HSAN).

Hereditary Pancreatitis (HP)

It is a genetic disorder that causes inflammation in the pancreas, which can lead to chronic pancreatitis. This can result in permanent damage to the pancreas and lead to other health issues.

My Hereditary Pancreatitis (HP)

Non-carrier

Knowledge



Time of onset

Symptoms of it typically appear in childhood or early adulthood.



Prevalence

It is a rare disease, with an estimated incidence of 1 in every 80,000 to 100,000 individuals.



Typical symptoms

Symptoms of it may include recurrent episodes of abdominal pain, nausea and vomiting, diarrhea, fever, and jaundice.



Treatment

Treatment for it usually involves managing symptoms and preventing complications. This may include pain management, nutritional support, and monitoring for potential complications such as diabetes or pancreatic cancer.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893938	SPINK1	AA	normal
RS397508378	CFTR	GG	normal

RS78802634	CFTR	GG	normal
RS77834169	CFTR	CC	normal
RS76713772	CFTR	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Hereditary Pancreatitis (HP) uses 25 polymorphic loci on genes such as *SPINK1*, *PRSS1* and *CFTR*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hereditary Pancreatitis (HP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hereditary Pancreatitis (HP), or if you feel that you have a risk of developing Hereditary Pancreatitis (HP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 25 polymorphic loci associated with the risk of Hereditary Pancreatitis (HP), such as rs104893938, rs111033565 and rs111033567.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hereditary Pancreatitis (HP).

Hereditary Desmoid Disease (HDD)

It is a rare genetic disorder characterized by the growth of noncancerous tumors in the connective tissue cells. These tumors usually grow slowly, but can invade adjacent tissues and organs, causing obvious pain and other symptoms.

My Hereditary Desmoid Disease (HDD)

Non-carrier

Knowledge



Time of onset

Symptoms of this disease varies considerably among affected individuals. While some patients may develop the tumors during childhood, others may not experience symptoms until much later in life.



Prevalence

The prevalence of it is unknown, but it is considered a very rare condition.



Typical symptoms

Common symptoms of this disease include pain, tenderness or stiffness in the affected tissues, skin discoloration, and impaired mobility of the affected area.



Treatment

Treatment options for this disease may include surgical removal of the tumors or other affected tissues, radiation therapy, and/or chemotherapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137854573	APC	CC	normal
RS397515734	APC	CC	normal

RS145945630	APC	CC	normal
RS1554085355	intergenic	TT	normal
RS1554085382	intergenic	DD	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Hereditary Desmoid Disease (HDD) uses 11 polymorphic loci on the *APC* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hereditary Desmoid Disease (HDD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hereditary Desmoid Disease (HDD), or if you feel that you have a risk of developing Hereditary Desmoid Disease (HDD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 11 polymorphic loci associated with the risk of Hereditary Desmoid Disease (HDD), such as rs137854573, rs145945630 and rs1554085355.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hereditary Desmoid Disease (HDD).

Hereditary Motor and Sensory Neuropathy (HMSN)

It is a hereditary condition that affects the peripheral nerves. It causes a progressive damage to the muscles in the lower legs and feet, as well as the hands and arms.

My Hereditary Motor and Sensory Neuropathy (HMSN)

Non-carrier

Knowledge



Time of onset

The onset time for it can vary depending on the type of . Type 1 usually begins in childhood or early adolescence, while Type 2 tends to begin in early adulthood.



Prevalence

It affects approximately 1 in 2,500 people worldwide.



Typical symptoms

The typical symptoms of it include progressive muscle weakness and atrophy, loss of sensation in the hands and feet, and foot deformities. Other clinical features can include scoliosis, respiratory problems, and hearing loss.



Treatment

There is no cure for it, but treatment can help manage symptoms. Treatment can include physical therapy, braces or orthotics, and surgery in some cases.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS587780564

DYNC1H1

CC

normal

Test details

Applicable situation

Genetic disease screening test for Hereditary Motor and Sensory Neuropathy (HMSN) uses 1 polymorphic locus on the *DYNC1H1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hereditary Motor and Sensory Neuropathy (HMSN).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hereditary Motor and Sensory Neuropathy (HMSN), or if you feel that you have a risk of developing Hereditary Motor and Sensory Neuropathy (HMSN), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Hereditary Motor and Sensory Neuropathy (HMSN), namely rs587780564.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hereditary Motor and Sensory Neuropathy (HMSN).

Metachromatic Leukodystrophy (MLD)

Also known as sulfatide lipidosis, it is a rare genetic disease. Because cerebroside sulfate and other glycolipids containing sulfuric acid cannot be desulfated, they are deposited in the lysosomes of the whole body, mainly in the central and peripheral nervous system, eventually leading to demyelination of the nervous system.

My Metachromatic Leukodystrophy (MLD)

Non-carrier

Knowledge



Time of onset

Symptoms typically begin between the ages of 1 and 2, and the child undergoes a normal growth and development process after birth, followed by a decline in motor and intellectual abilities.



Prevalence

It is estimated to affect 1 in 40,000 individuals.



Typical symptoms

Typical symptoms include difficulty in walking, muscle stiffness and weakness, loss of muscle coordination, and problems with swallowing and speech.



Treatment

There is currently no cure. Treatment focuses on managing symptoms and slowing disease progression. Hematopoietic stem cell transplantation (HSCT) is a potential treatment option for some patients with early-stage disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS199476389	ARSA	AA	normal
RS398123414	ARSA	II	normal
RS398123415	ARSA	II	normal
RS398123418	ARSA	GG	normal
RS74315455	ARSA	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Metachromatic Leukodystrophy (MLD) uses 13 polymorphic loci on the *ARSA* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Metachromatic Leukodystrophy (MLD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Metachromatic Leukodystrophy (MLD), or if you feel that you have a risk of developing Metachromatic Leukodystrophy (MLD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 13 polymorphic loci associated with the risk of Metachromatic Leukodystrophy (MLD), such as rs199476382, rs199476389 and rs398123414.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Metachromatic Leukodystrophy (MLD).

Isovaleric Acidemia (IVA)

It is a hereditary metabolic disorder, caused by a change (mutation) in the gene encoding the enzyme isovaleryl-CoA dehydrogenase, resulting in deficient or absent activity. This enzyme is responsible for helping break down leucine, an amino acid, and its deficiency leads to a buildup of organic acids in the blood that cause symptoms

My Isovaleric Acidemia (IVA)

Non-carrier

Knowledge

Time of onset

It is a genetic disorder that is present from birth, and typically manifests in early infancy.

Prevalence

It is a rare disease, with approximately 1 in 250,000 people affected worldwide.

Typical symptoms

The typical symptoms of it include poor feeding, vomiting, lethargy, seizures, and a sweet odor to the urine or sweat. If left untreated, the disease can lead to developmental delays, intellectual disability, acidosis, and even death.

Treatment

Treatment for it typically involve strict dietary management and regular monitoring of metabolic function. This may include limiting intake of protein, supplementing with certain amino acids or enzymes, and avoiding fasting or dehydration. In severe cases, hospitalization may be necessary to manage acute metabolic decompensation.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS786204613

intergenic

II

normal

Test details

Applicable situation

Genetic disease screening test for Isovaleric Acidemia (IVA) uses 1 polymorphic locus on the *IVD* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Isovaleric Acidemia (IVA).
 2. The test results are not applicable to clinical practice.
 3. If you have clinical needs, please contact a qualified institution or clinician.
-

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Isovaleric Acidemia (IVA), or if you feel that you have a risk of developing Isovaleric Acidemia (IVA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Isovaleric Acidemia (IVA), namely rs786204613.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Isovaleric Acidemia (IVA).

Thrombophilia

It is a blood clotting disorder that increases the risk of abnormal blood clotting in the veins and arteries.

My Thrombophilia
Non-carrier

Knowledge

Time of onset

This disease can occur at any age, but it is more commonly diagnosed in middle-aged adults.

Prevalence

The incidence of inherited this disease in incident venous thrombosis is approximately 150-840 per 100,000 person years.

Typical symptoms

Typical symptoms may include swelling, redness, pain, difficulty breathing, and fainting.

Treatment

Treatment may include anticoagulant medications, compression stockings, and lifestyle modifications. In some cases, surgery may be necessary to remove blood clots or improve circulation.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918141	PROC	CC	normal
RS121918147	PROC	GG	normal

RS121918153	PROC	GG	normal
RS121918154	PROC	CC	normal
RS121918160	PROC	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Thrombophilia uses 13 polymorphic loci on genes such as *PROC*, *F9* and *PROS1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Thrombophilia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Thrombophilia, or if you feel that you have a risk of developing Thrombophilia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 13 polymorphic loci associated with the risk of Thrombophilia, such as rs121918141, rs121918147 and rs121918153.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Thrombophilia.

Infantile Cerebellar-Retinal Degeneration (ICRD)

It is a rare genetic disease that affects the cerebellum and retina, and causes progressive degeneration of the brain and vision loss.

My Infantile Cerebellar-Retinal Degeneration (ICRD)

Non-carrier

Knowledge



Time of onset

The for this disease is typically within the first two years of life.



Prevalence

The of this disease is very low, only a few hundred cases have been reported worldwide.



Typical symptoms

The of it include delayed development, abnormal eye movements, vision loss, and progressive difficulty with movement and coordination.



Treatment

Treatment for it is largely supportive, focusing on managing symptoms such as physical and occupational therapy, and vision aids.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS375761361	ACO2	CC	normal

Test details

Applicable situation

Genetic disease screening test for Infantile Cerebellar-Retinal Degeneration (ICRD) uses 1 polymorphic locus on the *ACO2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Infantile Cerebellar-Retinal Degeneration (ICRD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Infantile Cerebellar-Retinal Degeneration (ICRD), or if you feel that you have a risk of developing Infantile Cerebellar-Retinal Degeneration (ICRD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Infantile Cerebellar-Retinal Degeneration (ICRD), namely rs375761361.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Infantile Cerebellar-Retinal Degeneration (ICRD).

Permanent Neonatal Diabetes Mellitus (PNDM)

It is a type of diabetes that first appears within the first 6 months of life and persists throughout the lifespan. This form of diabetes is characterized by high blood sugar levels (hyperglycemia) resulting from a shortage of the hormone insulin.

My Permanent Neonatal Diabetes Mellitus (PNDM)

Non-carrier

Knowledge

 Time of onset

The onset is usually within the first six months of life.

 Prevalence

It is estimated to affect 1 in 300,000 to 400,000 births worldwide.

 Typical symptoms

Typical symptoms include hyperglycemia, excessive thirst, frequent urination, dehydration, weight loss and poor growth.

 Treatment

Insulin therapy is the primary intervention, which must be given multiple times a day for the remainder of the individual's life. In some cases, medication that stimulates insulin production may be effective. Lifestyle modifications, such as diet and exercise, may also help manage symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS148311934	GCK	CC	normal

RS193922283	ST6GALNAC5	GG	normal
RS193922335	GCK	TT	normal
RS193929356	KCNJ11	TT	normal
RS387906777	PDX1	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Permanent Neonatal Diabetes Mellitus (PNDM) uses 15 polymorphic loci on genes such as *GCK*, *ABCC8* and *KCNJ11*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Permanent Neonatal Diabetes Mellitus (PNDM).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Permanent Neonatal Diabetes Mellitus (PNDM), or if you feel that you have a risk of developing Permanent Neonatal Diabetes Mellitus (PNDM), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 15 polymorphic loci associated with the risk of Permanent Neonatal Diabetes Mellitus (PNDM), such as rs1085307455, rs1360415315 and rs148311934.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Permanent Neonatal Diabetes Mellitus (PNDM).

Juvenile Polyposis Syndrome (JPS)

It is a rare genetic disorder characterized by the development of multiple polyps in the gastrointestinal tract, primarily in the colon and rectum.

My Juvenile Polyposis Syndrome (JPS)

Non-carrier

Knowledge



Time of onset

It typically appears in childhood or adolescence, but the age of onset can vary.



Prevalence

The exact prevalence is unknown. Some estimated that it affects 1 in 100,000 to 160,000 individuals.



Typical symptoms

Patients may develop multiple polyps in the gastrointestinal tract, which can lead to complications such as bleeding, anemia, and obstruction. Other symptoms may include abdominal pain, diarrhea, or constipation. In some cases, the polyps can become cancerous.



Treatment

Treatment typically involves endoscopic surveillance to monitor polyps, and surgery to remove polyps that become symptomatic or cancerous. Some individuals may also benefit from medications to reduce polyp formation.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121912581	SMAD4	GG	normal
RS377767334	intergenic	DD	normal

RS377767360	SMAD4	CC	normal
RS80338963	SMAD4	CC	normal
RS1060500738	intergenic	TT	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Juvenile Polyposis Syndrome (JPS) uses 15 polymorphic loci on the *SMAD4* and *BMPRI1A* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Juvenile Polyposis Syndrome (JPS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Juvenile Polyposis Syndrome (JPS), or if you feel that you have a risk of developing Juvenile Polyposis Syndrome (JPS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 15 polymorphic loci associated with the risk of Juvenile Polyposis Syndrome (JPS), such as rs1060500738, rs1060500739 and rs1060500742.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Juvenile Polyposis Syndrome (JPS).

Fish-Eye Disease (FED)

It is a disorder that causes the clear front surface of the eyes (the corneas) to gradually become cloudy. The cloudiness, which generally first appears in adolescence or early adulthood, consists of small grayish dots of cholesterol (opacities) distributed across the corneas.

My Fish-Eye Disease (FED)

Non-carrier

Knowledge



Time of onset

Symptoms of it can occur at any age, but they usually become apparent in adulthood.



Prevalence

It is a rare condition that affects only a small number of people worldwide.



Typical symptoms

Common symptoms of it include cloudy vision, skin discoloration, and abdominal pain.



Treatment

Treatment for it may include medication to manage cholesterol levels, lifestyle changes such as a healthy diet and regular exercise, and monitoring of any complications that may arise. In some cases, surgery may be needed to remove fatty deposits from the skin or other organs.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121908053	LCAT	GG	normal

Test details

Applicable situation

Genetic disease screening test for Fish-Eye Disease (FED) uses 1 polymorphic locus on the *LCAT* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Fish-Eye Disease (FED).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Fish-Eye Disease (FED), or if you feel that you have a risk of developing Fish-Eye Disease (FED), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Fish-Eye Disease (FED), namely rs121908053.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Fish-Eye Disease (FED).

Primary Hyperoxaluria (PH)

It is a rare genetic disease that affects the liver's ability to break down oxalate. This results in high levels of oxalate in the urine, which can lead to the formation of kidney stones, damage to the kidneys, and other health problems.

My Primary Hyperoxaluria (PH)

Non-carrier

Knowledge



Time of onset

It is often diagnosed in childhood or early adulthood.



Prevalence

It is estimated to affect 1 in 100,000 individuals worldwide.



Typical symptoms

Typical symptoms include recurrent kidney stones, blood in the urine, urinary tract infections, back pain and fatigue.



Treatment

Treatment may include medications to help reduce oxalate production in the liver and dietary changes to help prevent the formation of kidney stones. In severe cases, kidney transplantation may be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS113681235	intergenic	TT	normal
RS180177313	intergenic	TT	normal

RS180177259	AGXT	GG	normal
RS180177281	intergenic	GG	normal
RS180177286	AGXT	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Primary Hyperoxaluria (PH) uses 36 polymorphic loci on the *AGXT*, *HOGA1* and *GRHPR* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Primary Hyperoxaluria (PH).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Primary Hyperoxaluria (PH), or if you feel that you have a risk of developing Primary Hyperoxaluria (PH), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 36 polymorphic loci associated with the risk of Primary Hyperoxaluria (PH), such as rs113681235, rs121908520 and rs121908521.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Primary Hyperoxaluria (PH).

Distal Myopathy with Anterior Tibial Onset (DMAT)

It is a genetic condition that has the potential to affect muscle function and cause weakness in the muscles located in the feet and lower legs. This disease is known to progress over time, and can lead to significant muscle wasting, thereby restricting mobility in those affected.

My Distal Myopathy with Anterior Tibial Onset (DMAT)

Non-carrier

Knowledge



Time of onset

The onset of it typically occurs in adulthood, with individuals usually experiencing symptoms in their 20s or 30s.



Prevalence

It is a relatively rare disease, affecting only a small percentage of the population.



Typical symptoms

The typical symptoms of it include foot drop, which is characterized by the inability to lift the front part of the foot, as well as weakness in the lower leg muscles.



Treatment

Treatment for it primarily focus on symptom management, with the use of physical therapy, braces, or medication. Surgery may also be considered in some cases, depending on the severity of the disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS398123773

DYSF

CC

normal

Test details

Applicable situation

Genetic disease screening test for Distal Myopathy with Anterior Tibial Onset (DMAT) uses 1 polymorphic locus on the *DYSF* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Distal Myopathy with Anterior Tibial Onset (DMAT).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Distal Myopathy with Anterior Tibial Onset (DMAT), or if you feel that you have a risk of developing Distal Myopathy with Anterior Tibial Onset (DMAT), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Distal Myopathy with Anterior Tibial Onset (DMAT), namely rs398123773.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Distal Myopathy with Anterior Tibial Onset (DMAT).

Akinesia Deformation Sequence (ADS)

It is a congenital disease, that affects the development of joints and skeletal muscles leading to various deformities such as joint contractures, scoliosis, and limb hypoplasia.

My Akinesia Deformation Sequence (ADS)

Non-carrier

Knowledge

Time of onset

The onset of this disease is during fetal development and symptoms are evident at birth.

Prevalence

It is a rare disease and it is estimated to be 1 in every 20,000 live births.

Typical symptoms

Typical symptoms include joint contractures, cleft palate and clubfoot, limited range of motion, muscle weakness and skeletal deformities. There can also be conditions ranging from facial dysmorphism to cardiac, renal and pulmonary complications.

Treatment

Treatment for this disease is mainly supportive and involves physical therapy, orthopedic devices, and corrective surgeries. Early intervention and multidisciplinary care can improve the patient's quality of life and functional abilities.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121909257	RAPSN	GG	normal

RS794727884

intergenic

II

normal

Test details

Applicable situation

Genetic disease screening test for Akinesia Deformation Sequence (ADS) uses 2 polymorphic loci on the *RAPSN* and *DOK7* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Akinesia Deformation Sequence (ADS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Akinesia Deformation Sequence (ADS), or if you feel that you have a risk of developing Akinesia Deformation Sequence (ADS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Akinesia Deformation Sequence (ADS), such as rs121909257 and rs794727884.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Akinesia Deformation Sequence (ADS).

Xeroderma Pigmentosum (XP)

It is a rare genetic disorder that affects the skin's ability to repair damage caused by ultraviolet (UV) radiation. This results in an increased risk of developing skin cancer and other skin-related complications.

My Xeroderma Pigmentosum (XP)

Non-carrier

Knowledge



Time of onset

It typically appears in childhood, before the age of 10.



Prevalence

It is estimated to affect 1 in 1,000,000 individuals worldwide.



Typical symptoms

Typical symptoms include severe sunburns after minimal sun exposure, freckling on sun-exposed areas, dry skin, and skin lesions.



Treatment

Treatment may include regular skin cancer screenings, avoidance of direct sunlight, and protective measures such as wearing clothing that covers the skin and using high-sun protection factors (SPF) sunscreen.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121913020	ERCC2	CC	normal
RS121913024	ERCC2	GG	normal

RS121913026	ERCC2	GG	normal
RS201940931	intergenic	TT	normal
RS754532049	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Xeroderma Pigmentosum (XP) uses 7 polymorphic loci on the *ERCC2* and *XPC* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Xeroderma Pigmentosum (XP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Xeroderma Pigmentosum (XP), or if you feel that you have a risk of developing Xeroderma Pigmentosum (XP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 7 polymorphic loci associated with the risk of Xeroderma Pigmentosum (XP), such as rs121913020, rs121913024 and rs121913026.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Xeroderma Pigmentosum (XP).

Limb-Girdle Muscular Dystrophy (LGMD)

It is a group of genetic disorders that cause progressive weakness and wasting of the muscles in the hip and shoulder areas, leading to difficulty in walking, running and climbing stairs.

My Limb-Girdle Muscular Dystrophy (LGMD)

Non-carrier

Knowledge



Time of onset

Depending on the type of mutation and the severity of the condition, symptoms may appear from early childhood to middle age.



Prevalence

It is estimated to affect 1 in 14,500 to 123,000 individuals worldwide.



Typical symptoms

Typical symptoms include progressive weakness and wasting of the muscles in the hip and shoulder areas, difficulty in walking, running, and climbing stairs, frequent falls, loss of muscle mass, muscle pain and stiffness, and cardiac and respiratory complications in some cases.



Treatment

There is currently no cure. Treatment options focus on managing symptoms and maximizing quality of life. These may include physical therapy and exercise, assistive devices and mobility aids, respiratory support and monitoring, cardiac medications and monitoring, genetic counseling and testing for affected individuals and their families.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121912938	COL6A1	GG	normal
RS121912939	COL6A1	GG	normal
RS121913572	LAMA2	CC	normal
RS121913576	LAMA2	CC	normal
RS202247791	LAMA2	DD	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Limb-Girdle Muscular Dystrophy (LGMD) uses 13 polymorphic loci on genes such as *COL6A1*, *LAMA2* and *TRAPPC11*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Limb-Girdle Muscular Dystrophy (LGMD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Limb-Girdle Muscular Dystrophy (LGMD), or if you feel that you have a risk of developing Limb-Girdle Muscular Dystrophy (LGMD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 13 polymorphic loci associated with the risk of Limb-Girdle Muscular Dystrophy (LGMD), such as rs121912938, rs121912939 and rs121913572.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Limb-Girdle Muscular Dystrophy (LGMD).

Acrocallosal Syndrome (ACLS)

It is a rare genetic disorder that affects the development of the brain, face, and limbs. It is characterized by abnormalities in the corpus callosum (a band of nerves that connects the two hemispheres of the brain), facial dysmorphism, and extra fingers and/or toes.

My Acrocallosal Syndrome (ACLS)

Non-carrier

Knowledge



Time of onset

The onset of this syndrome is typically at birth or in infancy, when symptoms begin to present.



Prevalence

It is an extremely rare disease with only a few hundred reported cases worldwide.



Typical symptoms

The typical symptoms of it include an abnormally small head, broad and short nose, prominent forehead, extra digits or toes and absent or hypoplastic corpus callosum are also associated with seizures, developmental delay, intellectual disability, vision problems, hearing loss and skeletal deformities.



Treatment

There is no known cure for this syndrome, and treatment is generally supportive and symptomatic. This may include physical therapy, speech therapy, and medications to manage symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS794727316	KIF7	GG	normal

Test details

Applicable situation

Genetic disease screening test for Acrocallosal Syndrome (ACLS) uses 1 polymorphic locus on the *KIF7* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Acrocallosal Syndrome (ACLS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Acrocallosal Syndrome (ACLS), or if you feel that you have a risk of developing Acrocallosal Syndrome (ACLS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Acrocallosal Syndrome (ACLS), namely rs794727316.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Acrocallosal Syndrome (ACLS).

Rhizomelic Chondrodysplasia Punctata (RCDP)

It is a condition that impairs the normal development of many parts of the body. The major features of this disorder include skeletal abnormalities, distinctive facial features, intellectual disability, and respiratory problems.

My Rhizomelic Chondrodysplasia Punctata (RCDP)

Non-carrier

Knowledge



Time of onset

It is present at birth.



Prevalence

It is estimated to affect 1 in 100,000 to 200,000 births.



Typical symptoms

Typical symptoms include shortening of the upper arms and thighs, joint stiffness, facial abnormalities, and intellectual disability.



Treatment

There is currently no cure and treatment is focused on managing the symptoms of the disease. This may include physical therapy, surgery, and medication for seizures and other symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121909151	PEX7	CC	normal
RS796051881	PEX5	DD	normal

Test details

Applicable situation

Genetic disease screening test for Rhizomelic Chondrodysplasia Punctata (RCDP) uses 2 polymorphic loci on the *PEX7* and *PEX5* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Rhizomelic Chondrodysplasia Punctata (RCDP).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Rhizomelic Chondrodysplasia Punctata (RCDP), or if you feel that you have a risk of developing Rhizomelic Chondrodysplasia Punctata (RCDP), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Rhizomelic Chondrodysplasia Punctata (RCDP), such as rs121909151 and rs796051881.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Rhizomelic Chondrodysplasia Punctata (RCDP).

Sitosterolemia

It is a rare genetic disease. As the intestinal absorption of plant sterols increases, the amount secreted by the liver into the bile decreases, resulting in a significant increase in blood plant sterols (such as sitosterol, campesterol and stigmasterol), which may lead to atherosclerosis.

My Sitosterolemia
Non-carrier

Knowledge



Time of onset

Symptoms may present in childhood or adulthood.



Prevalence

It is estimated to affect 1 in 1,000,000 people worldwide.



Typical symptoms

Typical symptoms include xanthomas, premature atherosclerosis, and increased risk for heart disease.



Treatment

Currently, the treatment involves a combination of a low-phytosterol and low-cholesterol diet, as well as medications such as bile acid sequestrants and ezetimibe.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS119479065	ABCG5	GG	normal
RS119479066	ABCG5	GG	normal

RS119479067	intergenic	CC	normal
RS119480070	ABCG5	CC	normal
RS137852991	ABCG8	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Sitosterolemia uses 6 polymorphic loci on the *DYNC2LI1*, *ABCG5* and *ABCG8* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Sitosterolemia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Sitosterolemia, or if you feel that you have a risk of developing Sitosterolemia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 6 polymorphic loci associated with the risk of Sitosterolemia, such as rs119479065, rs119479066 and rs119479067.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Sitosterolemia.

Acromicric Dysplasia

It is a rare genetic disorder that affects bone and cartilage development, resulting in shortened limbs and fingers, short stature, and distinctive facial features.

My Acromicric Dysplasia
Non-carrier

Knowledge



Time of onset

Symptoms of it typically appear at birth or shortly thereafter.



Prevalence

The prevalence of it is unknown due to its rarity.



Typical symptoms

Typical symptoms of it include short stature, shortening of limbs and fingers, distinctive facial features (round face, prominent eyes and small chin), joint stiffness, retarded tooth growth, pulmonary infections, hearing loss, etc.



Treatment

Treatment options for this disease are limited and are primarily focused on addressing the symptoms, which may include physical therapy, surgery for joint stiffness, and hearing aids. There is no cure for the condition.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS587776863	FBN1	DD	normal
RS1064797059	intergenic	CC	normal

RS1131692052	intergenic	AA	normal
RS1555398394	intergenic	TT	normal

Test details

Applicable situation

Genetic disease screening test for Acromicric Dysplasia uses 4 polymorphic loci on the *FBN1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Acromicric Dysplasia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Acromicric Dysplasia, or if you feel that you have a risk of developing Acromicric Dysplasia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Acromicric Dysplasia, such as rs1064797059, rs1131692052 and rs1555398394.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Acromicric Dysplasia.

Thanatophoric Dysplasia

It is a rare genetic disorder that affects bone growth and results in severe underdevelopment of the ribs, limbs, and chest.

My Thanatophoric Dysplasia

Non-carrier

Knowledge



Time of onset

It typically appears in pregnancy, and infants with this disease are typically stillborn or die shortly after birth due to respiratory failure.



Prevalence

It is estimated to affect approximately 1 in every 20,000 to 50,000 live births.



Typical symptoms

Typical symptoms include shortening of the limbs, a narrow chest, a flattened face, and an enlarged head with a prominent forehead.



Treatment

There is currently no cure. Treatment primarily involves supportive care, such as respiratory support and pain management.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121913105	FGFR3	AA	normal
RS121913482	intergenic	CC	normal

RS4647924	FGFR3	CC	normal
RS78311289	FGFR3	AA	normal

Test details

Applicable situation

Genetic disease screening test for Thanatophoric Dysplasia uses 4 polymorphic loci on the *FGFR3* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Thanatophoric Dysplasia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Thanatophoric Dysplasia, or if you feel that you have a risk of developing Thanatophoric Dysplasia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Thanatophoric Dysplasia, such as rs121913105, rs121913482 and rs4647924.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Thanatophoric Dysplasia.

Lethal Congenital Contracture Syndrome (LCCS)

It is a rare genetic disorder that is characterized by multiple joint contractures, muscle wasting and severe neurological defects.

My Lethal Congenital Contracture Syndrome (LCCS)

Non-carrier

Knowledge



Time of onset

Symptoms of the disease are usually present at birth, but they may only become apparent during the first few months of a newborn.



Prevalence

The disease is very rare and the prevalence is unknown.



Typical symptoms

Typical symptoms include multiple joint contractures, muscle wasting, hypotonia, respiratory failure and feeding difficulties.



Treatment

There is currently no cure. Treatment is mainly supportive. For example, feeding support and respiratory management.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121434407	GLE1	GG	normal
RS793888524	ADGRG6	DD	normal

RS793888525

ADGRG6

TT

normal

Test details

Applicable situation

Genetic disease screening test for Lethal Congenital Contracture Syndrome (LCCS) uses 3 polymorphic loci on the *GLE1* and *ADGRG6* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Lethal Congenital Contracture Syndrome (LCCS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Lethal Congenital Contracture Syndrome (LCCS), or if you feel that you have a risk of developing Lethal Congenital Contracture Syndrome (LCCS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Lethal Congenital Contracture Syndrome (LCCS), such as rs121434407, rs793888524 and rs793888525.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Lethal Congenital Contracture Syndrome (LCCS).

Intellectual Disability-Hypotonic Facies Syndrome

It is a rare genetic disorder that predominantly affects males. It is characterized by intellectual disability, low muscle tone, and a distinct facial appearance.

My Intellectual Disability-Hypotonic Facies Syndrome

Non-carrier

Knowledge



Time of onset

The onset of symptoms typically occurs in early infancy, with most affected individuals showing signs before six months of age.



Prevalence

The prevalence of this syndrome is unknown, but it is considered to be a rare disorder.



Typical symptoms

The main symptoms of this syndrome include intellectual disability, low muscle tone (hypotonia), and a distinct facial appearance characterized by a small head, low-set ears, and a flat nasal bridge. Some individuals may also experience congenital heart defects, structural brain abnormalities, and gastrointestinal problems.



Treatment

There is currently no cure or specific treatment for this syndrome, but management typically involves supportive care and addressing individual symptoms. This may include therapies such as physical, occupational, or speech therapy, medications for seizures, and management of hearing or vision problems.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS122445099	ATRX	GG	normal
RS398123425	ATRX	TT	normal

Test details

Applicable situation

Genetic disease screening test for Intellectual Disability-Hypotonic Facies Syndrome uses 2 polymorphic loci on the *ATRX* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Intellectual Disability-Hypotonic Facies Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Intellectual Disability-Hypotonic Facies Syndrome, or if you feel that you have a risk of developing Intellectual Disability-Hypotonic Facies Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Intellectual Disability-Hypotonic Facies Syndrome, such as rs122445099 and rs398123425.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Intellectual Disability-Hypotonic Facies Syndrome.

Centronuclear Myopathy (CNM)

It is a rare genetic disease caused by mutations in the genes responsible for the transport and organization of muscle fibers. This leads to weakness and wasting of the muscles, particularly in the arms, legs, and trunk.

My Centronuclear Myopathy (CNM)

Non-carrier

Knowledge

Time of onset

The onset can be either in childhood or adulthood, depending on the type of gene mutation.

Prevalence

It is estimated to affect 1 in 50,000 to 200,000 individuals.

Typical symptoms

The clinical features can vary greatly between individuals. Typical symptoms include muscle weakness, poor muscle tone, difficulty in walking and breathing problems.

Treatment

Treatment is supportive and focus on managing symptoms. For example, physical therapy, breathing support and surgery.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS587783343	BIN1	CC	normal

Test details

Applicable situation

Genetic disease screening test for Centronuclear Myopathy (CNM) uses 1 polymorphic locus on the *BIN1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Centronuclear Myopathy (CNM).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Centronuclear Myopathy (CNM), or if you feel that you have a risk of developing Centronuclear Myopathy (CNM), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Centronuclear Myopathy (CNM), namely rs587783343.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Centronuclear Myopathy (CNM).

Choroidal Dystrophy, Central Areolar (CACD)

It is an genetic condition that affects the central part of the retina, the tissue at the back of the eye that senses light.

My Choroidal Dystrophy, Central Areolar (CACD)

Non-carrier

Knowledge



Time of onset

The onset of symptoms for this disease usually occurs in mid-life, between the ages of 30 and 50.



Prevalence

The prevalence of this disease is currently not well established, but it is considered to be a rare condition.



Typical symptoms

Clinical features of this disease include atrophy and degeneration of the retinal pigment epithelium and choriocapillaris in the central macula, leading to loss of central vision. In some cases, there may be a subtle yellow-white or ring-like lesion in the macula, seen on fundus examination.



Treatment

Currently, there is no cure for this disease. Treatment is aimed at managing symptoms and may include interventions such as low vision aids, bright light therapy, and/or medication if there is associated choroidal neovascularization (formation of abnormal blood vessels in the eye).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918567	PRPH2	CC	normal

Test details

Applicable situation

Genetic disease screening test for Choroidal Dystrophy, Central Areolar (CACD) uses 1 polymorphic locus on the *PRPH2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Choroidal Dystrophy, Central Areolar (CACD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Choroidal Dystrophy, Central Areolar (CACD), or if you feel that you have a risk of developing Choroidal Dystrophy, Central Areolar (CACD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Choroidal Dystrophy, Central Areolar (CACD), namely rs121918567.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Choroidal Dystrophy, Central Areolar (CACD).

Severe Combined Immunodeficiency (SCID)

It is a rare genetic disorder that affects the immune system, leading to body's inability to produce functional T cells and B cells.

My Severe Combined Immunodeficiency (SCID)

Non-carrier

Knowledge

Time of onset

It is typically diagnosed within the first few months of life.

Prevalence

It is estimated to affect 1 in 50,000 to 100,000 births.

Typical symptoms

Typical symptoms include frequent infections, chronic diarrhea, failure to thrive, and a rash.

Treatment

Treatment typically involves bone marrow transplants or gene therapy. Successful treatment can lead to the development of a functioning immune system. However, without treatment, this disease is typically fatal within the first year of life due to severe infections.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS111033617	IL2RG	CC	normal
RS111033620	IL2RG	CC	normal
RS111033622	IL2RG	AA	normal

RS137852507	intergenic	TT	normal
RS137852508	IL2RG	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Severe Combined Immunodeficiency (SCID) uses 12 polymorphic loci on the *IL2RG* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Severe Combined Immunodeficiency (SCID).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Severe Combined Immunodeficiency (SCID), or if you feel that you have a risk of developing Severe Combined Immunodeficiency (SCID), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 12 polymorphic loci associated with the risk of Severe Combined Immunodeficiency (SCID), such as rs111033617, rs111033620 and rs111033622.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Severe Combined Immunodeficiency (SCID).

Wrinkly Skin Syndrome (WSS)

It is a rare genetic disorder that affects the skin and connective tissues. It is characterized by deep wrinkles on the dorsum of the hands and feet, thickened palms and soles, and joint contractures.

My Wrinkly Skin Syndrome (WSS)

Non-carrier

Knowledge



Time of onset

It typically appears at birth or in early infancy.



Prevalence

It is an extremely rare disease, with only a few cases reported worldwide.



Typical symptoms

Typical symptoms include deep wrinkles on the hands and feet, thickened palms and soles, and joint contractures.



Treatment

Treatment mainly involves improving symptoms, for example, physical therapy may be helpful in managing joint contractures. Surgical intervention may be necessary in severe cases.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS374480381	intergenic	GG	normal

Test details

Applicable situation

Genetic disease screening test for Wrinkly Skin Syndrome (WSS) uses 1 polymorphic locus on the *ATP6V0A2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Wrinkly Skin Syndrome (WSS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Wrinkly Skin Syndrome (WSS), or if you feel that you have a risk of developing Wrinkly Skin Syndrome (WSS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Wrinkly Skin Syndrome (WSS), namely rs374480381.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Wrinkly Skin Syndrome (WSS).

Spondyloenchondrodysplasia with Immune Dysregulation (SPENCDI)

It is a rare genetic disorder that affects the skeletal system and the immune system. It is characterized by abnormal growth of bones and cartilage, which can lead to short stature and skeletal abnormalities, as well as immune dysfunction, which can result in recurrent infections and autoimmune disorders.

My Spondyloenchondrodysplasia with Immune Dysregulation (SPENCDI)

Non-carrier

Knowledge



Time of onset

Symptoms can present at birth, or they can appear later in childhood.



Prevalence

It has only a small number of cases reported worldwide.



Typical symptoms

Typical symptoms include short stature, skeletal abnormalities such as scoliosis and joint deformities, and immune dysfunction such as recurrent infections and autoimmune disorders.



Treatment

Treatment is focused on supportive care, with a combination of physical therapy to manage skeletal abnormalities and immune-boosting medications to prevent infections.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS781050795	intergenic	CC	normal

Test details

Applicable situation

Genetic disease screening test for Spondyloenchondrodysplasia with Immune Dysregulation (SPENCDI) uses 1 polymorphic locus on the *ACP5* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Spondyloenchondrodysplasia with Immune Dysregulation (SPENCDI).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Spondyloenchondrodysplasia with Immune Dysregulation (SPENCDI), or if you feel that you have a risk of developing Spondyloenchondrodysplasia with Immune Dysregulation (SPENCDI), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Spondyloenchondrodysplasia with Immune Dysregulation (SPENCDI), namely rs781050795.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Spondyloenchondrodysplasia with Immune Dysregulation (SPENCDI).

Lymphoproliferative Syndrome

It is a rare genetic disorder that affects the immune system. It is caused by mutations in genes related to the immune system which leads to abnormal immune response and can result in severe infections, cancers, and autoimmune diseases.

My Lymphoproliferative Syndrome

Non-carrier

Knowledge

Time of onset

It typically appears in childhood.

Prevalence

It is estimated to affect 1 in 1 million males.

Typical symptoms

Typical symptoms include frequent and severe infections, particularly with the Epstein-Barr virus (EBV), which can cause lymphoma or other types of cancer. Other symptoms may include fever, enlarged liver or spleen, low blood count, and easy bruising or bleeding.

Treatment

Treatment may include antiviral medications, chemotherapy, immunosuppressive therapy, and stem cell transplantation. Early diagnosis and treatment are crucial to improve outcomes.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS111033623	intergenic	CC	normal

Test details

Applicable situation

Genetic disease screening test for Lymphoproliferative Syndrome uses 1 polymorphic locus on the *SH2D1A* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Lymphoproliferative Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Lymphoproliferative Syndrome, or if you feel that you have a risk of developing Lymphoproliferative Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Lymphoproliferative Syndrome, namely rs111033623.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Lymphoproliferative Syndrome.

3-Methylglutaconic Aciduria (MGA)

It is a rare genetic disease that accumulates a large amount of substance called 3-methylpentenoic acid in body fluids due to a lack of 3-methylglutaryl CoA hydratase. It can cause an increase in acid levels in the blood (metabolic acidosis) and excrete a large amount of acid in the urine (aciduria).

My 3-Methylglutaconic Aciduria (MGA)

Non-carrier

Knowledge

Time of onset

Symptoms of this disease can begin anytime between infancy and adulthood.

Prevalence

The exact prevalence is unknown, but it is a rare disease affecting an estimated 1 in 100,000 individuals.

Typical symptoms

Clinical features of it vary greatly but may include developmental delay, muscle weakness and / or involuntary movements, vision and hearing problems, cardiomyopathy, adrenal insufficiency, or mitochondrial problems, among others.

Treatment

Currently, there is no cure for this disease, but specific symptoms may be treated or managed with medications, surgery, or other therapies.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894942	intergenic	CC	normal

RS387907236	SERAC1	GG	normal
RS397515738	intergenic	CC	normal
RS397515739	intergenic	TT	normal
RS397515740	intergenic	TT	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for 3-Methylglutaconic Aciduria (MGA) uses 14 polymorphic loci on the *TAFAZZIN*, *CLPB* and *SERAC1* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of 3-Methylglutaconic Aciduria (MGA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of 3-Methylglutaconic Aciduria (MGA), or if you feel that you have a risk of developing 3-Methylglutaconic Aciduria (MGA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 14 polymorphic loci associated with the risk of 3-Methylglutaconic Aciduria (MGA), such as rs104894942, rs144078282 and rs185461628.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of 3-Methylglutaconic Aciduria (MGA).

Aicardi-Goutieres Syndrome (AGS)

It is a rare genetic disorder that affects the brain, skin, and immune system. It is caused by mutations in several genes that regulate the body's response to viral infections.

My Aicardi-Goutieres Syndrome (AGS)

Non-carrier

Knowledge



Time of onset

This syndrome typically manifests in early infancy, usually within the first few months of life.



Prevalence

It is a rare disease, with an estimated of 1 in 100,000 to 1 in 250,000 individuals.



Typical symptoms

Typical symptoms of it include irritability, feeding difficulties, seizures, abnormal muscle tone, developmental delays, skin rashes. and enlarged ventricles in the brain, calcifications in the brain, loss of white matter in the brain, and a decrease in immune cells.



Treatment

There is no cure for it. Treatment is supportive, and may include anticonvulsant medications to control seizures, physical therapy to manage muscle problems, and other therapies targeted toward specific symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS587777446	IFIH1	CC	normal

RS76642637	TREX1	II	normal
RS78218009	TREX1	CC	normal
RS376048533	IFIH1	CC	normal
RS786205483	RNASEH2B	AA	normal

Test details

Applicable situation

Genetic disease screening test for Aicardi-Goutieres Syndrome (AGS) uses 5 polymorphic loci on the *IFIH1*, *TREX1* and *RNASEH2B* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Aicardi-Goutieres Syndrome (AGS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Aicardi-Goutieres Syndrome (AGS), or if you feel that you have a risk of developing Aicardi-Goutieres Syndrome (AGS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Aicardi-Goutieres Syndrome (AGS), such as rs376048533, rs587777446 and rs76642637.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Aicardi-Goutieres Syndrome (AGS).

Alstrom Syndrome (AS)

It is a rare genetic disorder that affects multiple organ systems including the eyes, ears, liver, heart, and kidneys.

My Alstrom Syndrome (AS)

Non-carrier

Knowledge

Time of onset

It usually appears in infancy or early childhood, with symptoms becoming more apparent as the child gets older.

Prevalence

It is extremely rare, with only a few hundred cases reported worldwide.

Typical symptoms

Typical symptoms of it include retinal atrophy, deafness, metabolic abnormalities such as obesity, insulin resistance, and heart and kidney failure

Treatment

There is no cure for it, so treatment focuses on managing the individual symptoms. This may include hearing aids or cochlear implants for hearing loss, dietary changes and exercise for obesity, and medications to manage diabetes and heart failure.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS398122992	intergenic	II	normal
RS398122994	intergenic	II	normal

RS398122995	ALMS1	CC	normal
RS772136379	ALMS1	TT	normal
RS797045228	ALMS1	DD	normal

Test details

Applicable situation

Genetic disease screening test for Alstrom Syndrome (AS) uses 5 polymorphic loci on the *ALMS1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Alstrom Syndrome (AS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Alstrom Syndrome (AS), or if you feel that you have a risk of developing Alstrom Syndrome (AS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Alstrom Syndrome (AS), such as rs398122992, rs398122994 and rs398122995.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Alstrom Syndrome (AS).

Axenfeld-Rieger Syndrome (ARS)

It is a rare genetic disorder that affects the development of certain tissues and structures in the eyes, teeth and other parts of the body. It is characterized by abnormalities in the iris, which give the eyes a distinctive appearance, as well as anomalies in the teeth and facial bones.

My Axenfeld-Rieger Syndrome (ARS)

Non-carrier

Knowledge



Time of onset

this syndrome usually occurs in childhood, and there are also some cases where it may not be diagnosed until adulthood.



Prevalence

The of this syndrome is estimated to be between 1 in 100,000 and 1 in 200,000 individuals.



Typical symptoms

Some of the of this syndrome include unusual facial features, including a flattened midface and small jaw, as well as widely spaced eyes and a flattened bridge of the nose. Individuals with the condition may also have dental abnormalities and vision problems.



Treatment

Treatment for this syndrome is usually supportive, with the goal of managing symptoms and preventing complications. This may include vision correction with glasses or contact lenses, surgery to correct ocular abnormalities, and orthodontic treatment for dental problems.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS886039568

FOXC1

CC

normal

Test details

Applicable situation

Genetic disease screening test for Axenfeld-Rieger Syndrome (ARS) uses 1 polymorphic locus on the *FOXC1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Axenfeld-Rieger Syndrome (ARS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Axenfeld-Rieger Syndrome (ARS), or if you feel that you have a risk of developing Axenfeld-Rieger Syndrome (ARS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Axenfeld-Rieger Syndrome (ARS), namely rs886039568.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Axenfeld-Rieger Syndrome (ARS).

Baraitser-Winter Syndrome (BRWS)

It is a rare genetic disorder characterized by unusual facial features, intellectual disability, and musculoskeletal anomalies.

My Baraitser-Winter Syndrome (BRWS)

Non-carrier

Knowledge



Time of onset

Symptoms of this disease are usually present at birth or become apparent in early childhood.



Prevalence

It is considered a rare disease, with an estimated of 1 in 1 million individuals.



Typical symptoms

The typical symptoms of this syndrome include a small head size, wide forehead, deep-set eyes, small ears, a short nose with upturned nostrils, a small mouth and chin, and abnormalities of the hands and feet.



Treatment

Treatment options for this syndrome are focused on managing symptoms and may include physical therapy, speech therapy, and surgery to correct musculoskeletal anomalies.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS281875334	ACTB	CC	normal
RS587779770	ACTB	CC	normal

RS587780275

ACTG1

AA

normal

Test details

Applicable situation

Genetic disease screening test for Baraitser-Winter Syndrome (BRWS) uses 3 polymorphic loci on the *ACTB* and *ACTG1* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Baraitser-Winter Syndrome (BRWS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Baraitser-Winter Syndrome (BRWS), or if you feel that you have a risk of developing Baraitser-Winter Syndrome (BRWS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Baraitser-Winter Syndrome (BRWS), such as rs281875334, rs587779770 and rs587780275.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Baraitser-Winter Syndrome (BRWS).

Bardet-Biedl Syndrome (BBS)

It is a rare genetic disorder that affects multiple parts of the body such as the eyes, kidneys, and brain.

My Bardet-Biedl Syndrome (BBS)

Non-carrier

Knowledge



Time of onset

The for this syndrome varies, but it typically develops during childhood or adolescence.



Prevalence

The of this syndrome is estimated to be between 1 in 140,000 to 160,000 individuals.



Typical symptoms

The of this syndrome include obesity, vision loss or blindness, intellectual disability, limb abnormalities, and kidney dysfunction.



Treatment

There is currently no cure for this syndrome. However, treatment can involve managing the individual symptoms through special education, vision aids, hormone replacement therapy, and dialysis.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS111033571	TRIM32	CC	normal
RS727503818	intergenic	II	normal

RS863224534	BBS9	GG	normal
RS863224530	intergenic	II	normal
RS863224522	BBS10	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Bardet-Biedl Syndrome (BBS) uses 21 polymorphic loci on genes such as *ASTN2*, *BBS4* and *BBS7*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Bardet-Biedl Syndrome (BBS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Bardet-Biedl Syndrome (BBS), or if you feel that you have a risk of developing Bardet-Biedl Syndrome (BBS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 21 polymorphic loci associated with the risk of Bardet-Biedl Syndrome (BBS), such as rs111033571, rs113994190 and rs119466002.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Bardet-Biedl Syndrome (BBS).

Bartter Syndrome

It is a general term for a group of rare genetic disorders in which there are specific defects in kidney function. These defects impair the kidney's ability to reabsorb salt and cause imbalances in various electrolyte and fluid concentrations in the body. The electrolytes affected are primarily mineral salts such as potassium, calcium, magnesium, sodium, and chloride.

My Bartter Syndrome

Non-carrier

Knowledge



Time of onset

It usually presents in infancy or early childhood.



Prevalence

The of this syndrome is unknown, but it is estimated to occur in 1 in 1,000,000 individuals.



Typical symptoms

The of this syndrome include polyuria, polydipsia, and dehydration. There may be hypokalemic metabolic alkalosis, increased levels of renin and aldosterone, increased urinary calcium excretion, and hypercalciuria.



Treatment

Treatment for this syndrome include electrolyte replacement therapy and administration of nonsteroidal anti-inflammatory drugs (NSAIDs).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS140012781	SLC12A3	CC	normal

Test details

Applicable situation

Genetic disease screening test for Bartter Syndrome uses 1 polymorphic locus on the *SLC12A3* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Bartter Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Bartter Syndrome, or if you feel that you have a risk of developing Bartter Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Bartter Syndrome, namely rs140012781.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Bartter Syndrome.

Becker Muscular Dystrophy (BMD)

It is a genetic disorder that causes progressive weakness and degeneration of the muscles. It is a type of muscular dystrophy that primarily affects males.

My Becker Muscular Dystrophy (BMD)

Non-carrier

Knowledge



Time of onset

Symptoms of this disease usually appear in adolescence, but may also become apparent in early childhood or adulthood.



Prevalence

It is a relatively rare disease, affecting approximately 1 in 18,500 males.



Typical symptoms

The typical symptoms of this disease include progressive skeletal muscle weakness, cardiomyopathy, and respiratory dysfunction. Skeletal muscle weakness most commonly affects the hips, pelvic area, thighs, and shoulders. Gluteal and thigh muscles may appear enlarged. Cardiomyopathy can lead to arrhythmias and heart failure. Respiratory dysfunction can occur due to weakened chest muscles or sleep apnea



Treatment

Treatment for it is primarily supportive, and includes physical therapy to maintain muscle function, medication to alleviate symptoms, and regular monitoring of cardiac and respiratory function. Some patients may be candidates for gene therapy or experimental treatments.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS104894797	DMD	GG	normal
RS398123981	DMD	GG	normal
RS863225015	intergenic	II	normal
RS794727666	DMD	CC	normal
RS794727661	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Becker Muscular Dystrophy (BMD) uses 22 polymorphic loci on the *DMD* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Becker Muscular Dystrophy (BMD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Becker Muscular Dystrophy (BMD), or if you feel that you have a risk of developing Becker Muscular Dystrophy (BMD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 22 polymorphic loci associated with the risk of Becker Muscular Dystrophy (BMD), such as rs104894797, rs1064325 and rs128625229.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Becker Muscular Dystrophy (BMD).

Bloom Syndrome (BLM)

It is a rare genetic disorder that is characterized by short stature, sun-sensitive skin rashes, an increased risk of cancer, and a weakened immune system.

My Bloom Syndrome (BLM)
Non-carrier

Knowledge



Time of onset

The for it is usually during childhood, with symptoms appearing between the ages of 2 and 15 years old.



Prevalence

The prevalence of it is estimated to be around 1 in 50,000 to 1 in 100,000 individuals worldwide.



Typical symptoms

The of this syndrome include rowth retardation, a butterfly-shaped rash on the face, telangiectasia (enlarged blood vessels) on the skin, respiratory infections, and an increased risk of developing various types of cancer, including leukemia, lymphoma, and solid tumors..



Treatment

Currently, there is no cure for this syndrome. However, doctors can treat the symptoms of the disease with various medications, including antibiotics to prevent infections, and surgery to remove tumors.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS367543012	BLM	DD	normal
RS367543024	BLM	II	normal

RS367543035	BLM	II	normal
RS367543036	BLM	GG	normal
RS1356090839	intergenic	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Bloom Syndrome (BLM) uses 8 polymorphic loci on the *BLM* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Bloom Syndrome (BLM).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Bloom Syndrome (BLM), or if you feel that you have a risk of developing Bloom Syndrome (BLM), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 8 polymorphic loci associated with the risk of Bloom Syndrome (BLM), such as rs1356090839, rs367543012 and rs367543024.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Bloom Syndrome (BLM).

Bohring-Opitz Syndrome (BOS)

It is a rare genetic disorder that typically affects newborns and infants. It is characterized by a range of physical and developmental abnormalities, including severe intellectual disability, feeding difficulties, respiratory problems, and distinctive facial features.

My Bohring-Opitz Syndrome (BOS)

Non-carrier

Knowledge

Time of onset

This syndrome typically appear at birth or within the first few weeks of life.

Prevalence

It is an extremely rare disorder, with fewer than 100 reported cases worldwide.

Typical symptoms

The typical symptoms of this syndrome include severe intellectual disability, feeding difficulties, respiratory problems, and distinctive facial features such as a small head, wide-set eyes, and a small jaw. They may also experience seizures, heart defects, skeletal abnormalities, and hearing loss. They may have difficulty with muscle tone and coordination, leading to delayed motor development and difficulty with walking. Behavioral issues, such as self-injury, may also be present.

Treatment

Treatment for it is primarily focused on managing symptoms. This may include medication for seizures, feeding tubes for nutritional support, and surgery for heart defects or other medical issues. Early intervention services such as physical therapy, occupational therapy, and speech therapy may also be beneficial for individuals with the disorder.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS373145711	ASXL1	CC	normal
RS397515401	ASXL1	CC	normal

Test details

Applicable situation

Genetic disease screening test for Bohring-Opitz Syndrome (BOS) uses 2 polymorphic loci on the *ASXL1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Bohring-Opitz Syndrome (BOS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Bohring-Opitz Syndrome (BOS), or if you feel that you have a risk of developing Bohring-Opitz Syndrome (BOS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Bohring-Opitz Syndrome (BOS), such as rs373145711 and rs397515401.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Bohring-Opitz Syndrome (BOS).

Borjeson-Forssman-Lehmann Syndrome (BFLS)

It is a rare X-linked genetic disorder that affects multiple organs and systems. This disorder primarily affects the central nervous system, endocrine system, and skeletal muscles, resulting in intellectual disability, obesity, hypogonadism, and various physical abnormalities.

My Borjeson-Forssman-Lehmann Syndrome (BFLS)

Non-carrier

Knowledge

Time of onset

Symptoms usually present at birth or early infancy, leading to early diagnosis.

Prevalence

It is an extremely rare disorder, with an estimated prevalence of fewer than 1 in 1 million individuals worldwide.

Typical symptoms

Common symptoms of this syndrome include cognitive and developmental delays, intellectual disability, hypotonia, obesity, short stature, seizures, hypogonadism, and abnormal physical features, such as facial dysmorphism, long fingers, and prominent ears.

Treatment

There is currently no cure for this syndrome. Treatment consists of management of symptoms and supportive care, including physical, occupational, and speech therapy. Anti-epileptic drugs may be used to control seizures. Hormone replacement therapy may be necessary to treat hypogonadism.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS132630297	PHF6	CC	normal
RS132630298	PHF6	GG	normal
RS132630299	PHF6	GG	normal
RS132630301	PHF6	AA	normal
RS864309532	PHF6	GG	normal

Test details

Applicable situation

Genetic disease screening test for Borjeson-Forssman-Lehmann Syndrome (BFLS) uses 5 polymorphic loci on the *PHF6* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Borjeson-Forssman-Lehmann Syndrome (BFLS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Borjeson-Forssman-Lehmann Syndrome (BFLS), or if you feel that you have a risk of developing Borjeson-Forssman-Lehmann Syndrome (BFLS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Borjeson-Forssman-Lehmann Syndrome (BFLS), such as rs132630297, rs132630298 and rs132630299.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Borjeson-Forssman-Lehmann Syndrome (BFLS).

Bruck Syndrome (BS)

It is a rare genetic disorder that is characterized by brittle bones (osteogenesis imperfecta) and joint contractures at birth (congenital joint contractures). This condition affects bones and connective tissues, leading to significant disability and reduced quality of life.

My Bruck Syndrome (BS)

Non-carrier

Knowledge



Time of onset

Symptoms of this syndrome are typically present at birth or soon after birth.



Prevalence

The prevalence of this syndrome in the general population is unknown. It is a rare disorder with fewer than 100 cases reported in the medical literature.



Typical symptoms

The typical symptoms of Bruck Syndrome include brittle bones, joint contractures at birth, and weak muscles. Other symptoms may include short stature, scoliosis, and mild to moderate intellectual disability.



Treatment

Treatment for this syndrome is typically focused on managing symptoms and preventing complications. This may include physical therapy, orthopedic appliances (such as braces), medications for pain management, and surgical interventions for severe joint problems.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS387906960	FKBP10	GG	normal
RS794727669	COL1A2	GG	normal
RS869320752	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Bruck Syndrome (BS) uses 3 polymorphic loci on the *FKBP10* and *COL1A2* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Bruck Syndrome (BS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Bruck Syndrome (BS), or if you feel that you have a risk of developing Bruck Syndrome (BS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Bruck Syndrome (BS), such as rs387906960, rs794727669 and rs869320752.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Bruck Syndrome (BS).

Charcot-Marie-Tooth Disease (CMT)

It is a hereditary neuropathy that affects the peripheral nerves responsible for motor and sensory functions. It is named after the three physicians who first identified it.

My Charcot-Marie-Tooth Disease (CMT)

Non-carrier

Knowledge



Time of onset

The onset of this disease can occur at any age, but it is most commonly diagnosed in adolescence or early adulthood.



Prevalence

It affects approximately 1 in 3,300 people globally, making it one of the most common inherited neurological disorders.



Typical symptoms

The typical symptoms of this disease include progressive muscle weakness, loss of muscle mass, and deformities in the feet, ankles, and hands.



Treatment

There is currently no cure for it, but treatment focuses on managing symptoms and maintaining mobility through physical therapy, braces, and surgeries.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894075	intergenic	CC	normal

RS59914820	LMNA	CC	normal
RS587777875	MFN2	CC	normal
RS587777876	intergenic	CC	normal
RS587781262	PRPS1	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Charcot-Marie-Tooth Disease (CMT) uses 153 polymorphic loci on genes such as *GDAP1*, *HSPB8* and *PMP22*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Charcot-Marie-Tooth Disease (CMT).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Charcot-Marie-Tooth Disease (CMT), or if you feel that you have a risk of developing Charcot-Marie-Tooth Disease (CMT), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 153 polymorphic loci associated with the risk of Charcot-Marie-Tooth Disease (CMT), such as rs104894075, rs104894076 and rs104894078.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Charcot-Marie-Tooth Disease (CMT).

Cockayne Syndrome (CS)

It is a rare genetic disorder that affects various aspects of a person's development and growth. It is characterized by a prematurely aging appearance, abnormally small head size, and intellectual disability.

My Cockayne Syndrome (CS)

Non-carrier

Knowledge



Time of onset

The of Cockayne Syndrome varies, but it is often diagnosed in early childhood.



Prevalence

It is a rare disease with an estimated incidence of 2-5 cases per million.



Typical symptoms

The typical symptoms of this disease include growth and developmental delays, premature aging, photosensitivity, hearing loss, vision problems, and neurological impairments.



Treatment

Currently, there is no cure for Cockayne Syndrome. Treatment approaches are aimed at alleviating symptoms and may include physical therapy, medications, and surgical interventions as necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS202080674	ERCC6	GG	normal
RS786205166	intergenic	DD	normal

RS786205167	ERCC6	DD	normal
RS786205169	intergenic	II	normal
RS786205174	ERCC6	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Cockayne Syndrome (CS) uses 7 polymorphic loci on the *ERCC6* and *ERCC8* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Cockayne Syndrome (CS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Cockayne Syndrome (CS), or if you feel that you have a risk of developing Cockayne Syndrome (CS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 7 polymorphic loci associated with the risk of Cockayne Syndrome (CS), such as rs202080674, rs786205166 and rs786205167.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Cockayne Syndrome (CS).

Coffin-Lowry Syndrome (CLS)

It is a rare genetic disorder that affects the physical and intellectual development of an individual.

My Coffin-Lowry Syndrome (CLS)

Non-carrier

Knowledge



Time of onset

It is typically diagnosed in childhood as symptoms begin to appear during early childhood.



Prevalence

It is a rare disorder and the exact prevalence is unknown. It affects males and females of all ethnic groups equally.



Typical symptoms

This syndrome often have distinct facial features such as a prominent forehead, widely spaced eyes, a depressed nasal bridge, and a short, wide mouth. They may also have skeletal abnormalities such as large hands and feet, abnormal curvature of the spine, and short stature. Other clinical features include delayed speech and language development, difficulty with fine motor skills, and behavioral problems.



Treatment

Treatment for it is mainly supportive and focuses on managing symptoms such as intellectual disability and developmental delay. Physical, occupational, and speech therapy may also be helpful in improving fine motor skills and communication abilities.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS122454130	RPS6KA3	AA	normal
RS398124177	intergenic	CC	normal

Test details

Applicable situation

Genetic disease screening test for Coffin-Lowry Syndrome (CLS) uses 2 polymorphic loci on the *RPS6KA3* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Coffin-Lowry Syndrome (CLS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Coffin-Lowry Syndrome (CLS), or if you feel that you have a risk of developing Coffin-Lowry Syndrome (CLS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Coffin-Lowry Syndrome (CLS), such as rs122454130 and rs398124177.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Coffin-Lowry Syndrome (CLS).

Cohen Syndrome (COH)

It is a rare genetic disease characterized by intellectual disability, facial dysmorphism, microcephaly, and joint hypermobility.

My Cohen Syndrome (COH)

Non-carrier

Knowledge



Time of onset

Symptoms of this syndrome are typically present at birth but may not be recognized until early childhood.



Prevalence

It is a rare disease and its prevalence is unknown, although it is believed to affect fewer than 1 in 100,000 people worldwide.



Typical symptoms

Common symptoms of this syndrome include Intellectual disability, facial dysmorphism (high, broad forehead, long eyelashes, thick eyebrows, large nasal tip, thick lips), microcephaly, and joint hypermobility. It can also present with obesity, hypotonia (low muscle tone), hearing loss, and visual problems. Other features may include dental abnormalities, scoliosis, and gastrointestinal issues.



Treatment

There is no cure for this syndrome, so management is focused on addressing individual symptoms. This may include early intervention services, speech and language therapy, physical therapy, and medications to manage seizures or behavioral problems.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS140353201	VPS13B	CC	normal
RS140936527	VPS13B	CC	normal
RS180177327	intergenic	II	normal
RS180177329	intergenic	DD	normal
RS180177356	VPS13B	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Cohen Syndrome (COH) uses 16 polymorphic loci on the *VPS13B* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Cohen Syndrome (COH).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Cohen Syndrome (COH), or if you feel that you have a risk of developing Cohen Syndrome (COH), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 16 polymorphic loci associated with the risk of Cohen Syndrome (COH), such as rs140353201, rs140936527 and rs180177327.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Cohen Syndrome (COH).

Cornelia de Lange Syndrome (CdLS)

It is a genetic disorder that affects many parts of the body. It is characterized by distinctive facial features, growth delays, intellectual disability, and other physical abnormalities.

My Cornelia de Lange Syndrome (CdLS)

Non-carrier

Knowledge

Time of onset

It is typically diagnosed at birth or during infancy.

Prevalence

The prevalence of it is estimated to be 1 in 10,000 to 30,000 births.

Typical symptoms

The of this syndrome include slow growth before and after birth, distinctive facial features, such as eyebrows that meet in the middle, small head circumference, limb abnormalities, hearing loss, heart defects, and gastrointestinal problems.

Treatment

Treatment for it is symptomatic and supportive. This may involve physical therapy, occupational therapy, speech therapy, and other medical interventions to address specific symptoms and complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918267	NIPBL	CC	normal

RS587784036	NIPBL	GG	normal
RS587784026	NIPBL	CC	normal
RS587784027	intergenic	GG	normal
RS587784029	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Cornelia de Lange Syndrome (CdLS) uses 180 polymorphic loci on genes such as *NIPBL*, *HDAC8* and *SMC3*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Cornelia de Lange Syndrome (CdLS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Cornelia de Lange Syndrome (CdLS), or if you feel that you have a risk of developing Cornelia de Lange Syndrome (CdLS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 180 polymorphic loci associated with the risk of Cornelia de Lange Syndrome (CdLS), such as rs121918267, rs121918269 and rs398124465.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Cornelia de Lange Syndrome (CdLS).

Costello Syndrome

It is a rare genetic disorder that affects many parts of the body. It is characterized by distinctive facial features, developmental delay, intellectual disability, and increased tumor risk.

My Costello Syndrome
Non-carrier

Knowledge



Time of onset

It is present from birth, but symptoms may not become apparent until infancy.



Prevalence

It is extremely rare, with an estimated prevalence of 1 in 300,000 to 1 in 1,000,000 births.



Typical symptoms

Common symptoms of this syndrome include distinctive facial features, short stature, developmental delay, intellectual disability, feeding difficulties, and heart abnormalities.



Treatment

Treatment for this syndrome focuses on managing symptoms and preventing complications. This may include early intervention and therapy for developmental delays, regular monitoring for heart and tumor abnormalities, and genetic counseling for affected individuals and their families.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894227	HRAS	TT	normal
RS727503093	HRAS	CC	normal

RS730880460

HRAS

CC

normal

Test details

Applicable situation

Genetic disease screening test for Costello Syndrome uses 3 polymorphic loci on the *HRAS* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Costello Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Costello Syndrome, or if you feel that you have a risk of developing Costello Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Costello Syndrome, such as rs104894227, rs727503093 and rs730880460.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Costello Syndrome.

Crigler-Najjar Syndrome (CNS)

It is a rare genetic disorder that affects the body's ability to break down bilirubin, resulting in a buildup of the substance in the blood.

My Crigler-Najjar Syndrome (CNS)

Non-carrier

Knowledge



Time of onset

This syndrome appears within the first or second weeks after birth or later in childhood, and persists for life.



Prevalence

It is a rare disease, affecting 1 in 1,000,000 individuals globally.



Typical symptoms

The typical symptoms of this disease include jaundice, yellowing of the skin and whites of the eyes, fatigue, and abdominal pain.



Treatment

The treatment method includes phototherapy, which involves exposing the skin to blue light to help break down bilirubin. In severe cases, liver transplantation may be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS72551348	UGT1A1	AA	normal
RS797046091	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Crigler-Najjar Syndrome (CNS) uses 2 polymorphic loci on the *UGT1A* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Crigler-Najjar Syndrome (CNS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Crigler-Najjar Syndrome (CNS), or if you feel that you have a risk of developing Crigler-Najjar Syndrome (CNS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Crigler-Najjar Syndrome (CNS), such as rs72551348 and rs797046091.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Crigler-Najjar Syndrome (CNS).

Crouzon Syndrome

It is a genetic disorder characterized by the premature fusion of certain skull bones (craniosynostosis). This early fusion prevents the skull from growing normally and affects the shape of the head and face.

My Crouzon Syndrome

Non-carrier

Knowledge



Time of onset

This syndrome is present from birth and can be diagnosed early on.



Prevalence

The of this syndrome is estimated to be 1 in 60,000 to 100,000 live births.



Typical symptoms

The typical symptoms of this disease include bulging eyes, an underdeveloped upper jaw, and a beaked nose.



Treatment

Treatment options include corrective surgery to address physical abnormalities, such as cranial reconstruction and orthognathic surgery, along with supportive therapies to address symptoms such as vision issues and sleep apnea.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918488	FGFR2	AA	normal
RS121918494	FGFR2	GG	normal

RS121918501	FGFR2	AA	normal
RS776587763	FGFR2	CC	normal

Test details

Applicable situation

Genetic disease screening test for Crouzon Syndrome uses 4 polymorphic loci on the *FGFR2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Crouzon Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Crouzon Syndrome, or if you feel that you have a risk of developing Crouzon Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Crouzon Syndrome, such as rs121918488, rs121918494 and rs121918501.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Crouzon Syndrome.

D-2-hydroxyglutaric Aciduria (D2HGA)

It is a rare genetic disorder that affects the metabolism of the amino acid lysine. Patients with this disease accumulate high levels of D-2-hydroxyglutaric acid in their body fluids and tissues, resulting in damage to the brain, muscles, and other tissues.

My D-2-hydroxyglutaric Aciduria (D2HGA)

Non-carrier

Knowledge



Time of onset

The signs and symptoms of it typically appear in infancy or early childhood.



Prevalence

It is a rare disease, with an estimated incidence of 1 in 250,000 to 1 in 1,000,000 individuals worldwide.



Typical symptoms

The symptoms of this disease can vary widely depending on the severity of the disease, but may include developmental delays, intellectual disability, seizures, muscle weakness or stiffness, difficulty coordinating movements, and an abnormally large head size.



Treatment

There is currently no cure for this disease, and treatment is mainly supportive. Physical and occupational therapy can help improve motor function and mobility, while anticonvulsant medication may be used to control seizures. Some patients may also benefit from a low-lysine diet.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS797045506	intergenic	II	normal
RS797045507	intergenic	TT	normal

Test details

Applicable situation

Genetic disease screening test for D-2-hydroxyglutaric Aciduria (D2HGA) uses 2 polymorphic loci on the *D2HGDH* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of D-2-hydroxyglutaric Aciduria (D2HGA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of D-2-hydroxyglutaric Aciduria (D2HGA), or if you feel that you have a risk of developing D-2-hydroxyglutaric Aciduria (D2HGA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of D-2-hydroxyglutaric Aciduria (D2HGA), such as rs797045506 and rs797045507.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of D-2-hydroxyglutaric Aciduria (D2HGA).

De Sanctis-Cacchione Syndrome (DSC)

It is a rare genetic disorder that affects the nervous system and causes sensitivity to ultraviolet light.

My De Sanctis-Cacchione Syndrome (DSC)

Non-carrier

Knowledge



Time of onset

The onset of it is typically in infancy or early childhood, with symptoms appearing soon after exposure to sunlight.



Prevalence

It is an extremely rare disease, with only a few dozen cases reported worldwide.



Typical symptoms

The of it include redness, blistering, and peeling of the skin after minimal sun exposure, as well as neurological symptoms such as developmental delays, seizures, and intellectual disability.



Treatment

Treatment for it is primarily supportive and focused on managing symptoms. This may include avoiding sunlight and using protective clothing and sunscreen, as well as addressing any neurological complications with medications or therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121917904	ERCC6	GG	normal
RS373227647	intergenic	TT	normal

RS751838040	ERCC6	GG	normal
RS765825423	intergenic	II	normal
RS774791374	intergenic	DD	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for De Sanctis-Cacchione Syndrome (DSC) uses 7 polymorphic loci on the *ERCC6* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of De Sanctis-Cacchione Syndrome (DSC).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of De Sanctis-Cacchione Syndrome (DSC), or if you feel that you have a risk of developing De Sanctis-Cacchione Syndrome (DSC), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 7 polymorphic loci associated with the risk of De Sanctis-Cacchione Syndrome (DSC), such as rs121917904, rs373227647 and rs751838040.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of De Sanctis-Cacchione Syndrome (DSC).

Dent Disease

It is a rare genetic disorder that affects the renal tubules of the kidney, causing excessive calcium and other electrolyte loss in urine.

My Dent Disease
Non-carrier

Knowledge



Time of onset

This disease can present symptoms as early as infancy, but most commonly symptoms appear in early childhood around the age of 5-6.



Prevalence

It is a rare disorder, with an estimated prevalence of 1 in 30,000 to 40,000 individuals.



Typical symptoms

Common symptoms of Dent Disease include excessive thirst and urination, dehydration, kidney stones, proteinuria, hematuria, and progressive renal failure.



Treatment

There is currently no cure for this disease. Treatment options may include management of symptoms with diuretics, pain management for kidney stones, and electrolyte supplementation to correct imbalances. In severe cases, a kidney transplant may be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS151340620	intergenic	GG	normal
RS151340621	CLCN5	CC	normal

RS151340630	CLCN5	GG	normal
RS273585644	CLCN5	AA	normal
RS797044810	CLCN5	CC	normal

Test details

Applicable situation

Genetic disease screening test for Dent Disease uses 5 polymorphic loci on the *CLCN5* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Dent Disease.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Dent Disease, or if you feel that you have a risk of developing Dent Disease, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Dent Disease, such as rs151340620, rs151340621 and rs151340630.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Dent Disease.

Desbuquois Dysplasia

It is a rare genetic condition that affects the growth and development of bones. This condition may affect various bones in the body, but particularly long bones and the spine. It may also cause joint problems and abnormalities in other organs.

My Desbuquois Dysplasia

Non-carrier

Knowledge



Time of onset

Symptoms of this disease may become apparent before or at birth, but the condition may also not be recognized until later in childhood.



Prevalence

It is a very rare condition, with only around 50 cases reported in medical literature.



Typical symptoms

Common symptoms of this disease include short stature, joint pain, joint and spine abnormalities, and distinctive facial features such as a small nose and wide-set eyes.



Treatment

The main goal of treatment for it is to manage symptoms and prevent complications. This may involve the use of pain medication, joint surgery, hearing aids, and other supportive measures.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267606701	CANT1	GG	normal

RS587776897	intergenic	II	normal
RS538543007	CANT1	CC	normal

Test details

Applicable situation

Genetic disease screening test for Desbuquois Dysplasia uses 3 polymorphic loci on the *CANT1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Desbuquois Dysplasia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Desbuquois Dysplasia, or if you feel that you have a risk of developing Desbuquois Dysplasia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Desbuquois Dysplasia, such as rs267606701, rs538543007 and rs587776897.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Desbuquois Dysplasia.

Diamond-Blackfan Anemia (DBA)

It is a rare blood disorder in which the bone marrow fails to produce enough red blood cells, leading to anemia. The condition usually presents during the first year of life, although it may go undiagnosed until later in life.

My Diamond-Blackfan Anemia (DBA)

Non-carrier

Knowledge

Time of onset

It typically presents within the first year of life, although it may go undiagnosed until later in life.

Prevalence

It is a rare disease, affecting approximately 5 to 7 individuals per million live births.

Typical symptoms

The most common symptom of it is anemia, which may cause fatigue, weakness, and pallor. In severe cases, anemia may lead to heart failure or other complications.

Treatment

Treatment for this disease typically involves regular blood transfusions to alleviate anemia, as well as medications to help the bone marrow produce red blood cells. In some cases, bone marrow transplantation may be necessary. Patients with DBA require ongoing medical management and monitoring throughout their lives.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS116840808	RPL35A	GG	normal

RS116840809	RPL35A	CC	normal
RS61762293	RPS19	CC	normal
RS1555524354	intergenic	II	normal
RS1555524370	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Diamond-Blackfan Anemia (DBA) uses 8 polymorphic loci on genes such as *RPL35A*, *TP53* and *RPS19*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Diamond-Blackfan Anemia (DBA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Diamond-Blackfan Anemia (DBA), or if you feel that you have a risk of developing Diamond-Blackfan Anemia (DBA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 8 polymorphic loci associated with the risk of Diamond-Blackfan Anemia (DBA), such as rs116840808, rs116840809 and rs1555524354.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Diamond-Blackfan Anemia (DBA).

Duane Syndrome (DS)

It is a rare congenital disorder that affects eye movement, causing limited ability to move the eyes side-to-side. This results from abnormalities in the sixth and/or third cranial nerves controlling the lateral and medial rectus muscles, respectively.

My Duane Syndrome (DS)

Non-carrier

Knowledge

 Time of onset

It is usually diagnosed in early childhood after the age of three years old. The symptom onset can range from infancy to late childhood.

 Prevalence

Due to the rarity of this disease, exact rates are unknown.

 Typical symptoms

The main symptom of it is limited eye movement, especially in the sideways direction. Affected individuals may also tilt their head toward one shoulder or experience double vision. In some cases, the eyelid may also droop (ptosis).

 Treatment

There is no cure for this disease, but the goal of management is to improve eye alignment and reduce head tilt or double vision. Treatment options may include vision therapy, prism glasses, eye patches, or surgery to reposition the eye muscles.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS879255275	intergenic	II	normal
RS879255277	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Duane Syndrome (DS) uses 2 polymorphic loci on the *MAFB* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Duane Syndrome (DS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Duane Syndrome (DS), or if you feel that you have a risk of developing Duane Syndrome (DS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Duane Syndrome (DS), such as rs879255275 and rs879255277.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Duane Syndrome (DS).

Ehlers-Danlos Syndrome (EDS)

It is a rare genetic connective tissue disorder that affects the skin, joints, and blood vessels. It is caused by mutations in genes responsible for producing collagen, which is an essential component of connective tissue that provides strength and elasticity to various bodily structures.

My Ehlers-Danlos Syndrome (EDS)

Non-carrier

Knowledge



Time of onset

The for it can vary depending on the specific type of the disease and the severity of symptoms. Some people may be born with noticeable symptoms, while others may not experience symptoms until later in life.



Prevalence

The prevalence of it is estimated to be around 1 in 5,000 individuals worldwide, although some experts believe it may be underdiagnosed.



Typical symptoms

The typical symptoms of this disease include loose skin that is easily stretched, hypermobility of the joints, chronic pain, easy bruising, and fragile blood vessels that can lead to excessive bleeding or organ rupture.



Treatment

Treatment options for it are focused on managing and preventing symptoms. This may include physical therapy to strengthen the joints and improve mobility, medications to manage pain or prevent bleeding, and regular monitoring of the cardiovascular system to prevent complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS112371422	COL3A1	CC	normal
RS587779597	COL3A1	GG	normal
RS587779608	COL3A1	DD	normal
RS587779607	COL3A1	CC	normal
RS587779605	COL3A1	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Ehlers-Danlos Syndrome (EDS) uses 334 polymorphic loci on genes such as *COL3A1*, *CHST14* and *COL5A2*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Ehlers-Danlos Syndrome (EDS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.

4. If you have a family history of Ehlers-Danlos Syndrome (EDS), or if you feel that you have a risk of developing Ehlers-Danlos Syndrome (EDS), it is recommended that you consult a qualified health professional or clinician for help.

 Test content

This test includes 334 polymorphic loci associated with the risk of Ehlers-Danlos Syndrome (EDS), such as rs1057518075, rs1057518372 and rs1057521106.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Ehlers-Danlos Syndrome (EDS).

Ellis-van Creveld Syndrome (EVC)

It is an genetic disorder of bone growth that results in very short stature (dwarfism). People with this condition have particularly short forearms and lower legs and a narrow chest with short ribs.

My Ellis-van Creveld Syndrome (EVC)

Non-carrier

Knowledge



Time of onset

The onset of it occurs during pregnancy and symptoms may be visible at birth or shortly after.



Prevalence

The of this disease is estimated to be around 1 in 60,000 to 1 in 200,000 live births.



Typical symptoms

The typical symptoms of this syndrome include short stature, abnormal fingers and toes, dental problems, and heart defects.



Treatment

Treatment methods can include surgical and orthopedic interventions to improve mobility and address any heart defects. Some people may need special education and/or social support.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS750396637	intergenic	II	normal
RS751356206	EVC2	GG	normal

RS794726665

EVC

GG

normal

Test details

Applicable situation

Genetic disease screening test for Ellis-van Creveld Syndrome (EVC) uses 3 polymorphic loci on the *EVC2* and *EVC* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Ellis-van Creveld Syndrome (EVC).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Ellis-van Creveld Syndrome (EVC), or if you feel that you have a risk of developing Ellis-van Creveld Syndrome (EVC), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Ellis-van Creveld Syndrome (EVC), such as rs750396637, rs751356206 and rs794726665.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Ellis-van Creveld Syndrome (EVC).

Fabry Disease

It is a rare genetic disorder that occurs due to a deficiency in the enzyme alpha-galactosidase A, which leads to the accumulation of a fatty substance called globotriaosylceramide in various organs of the body.

My Fabry Disease

Non-carrier

Knowledge



Time of onset

It has a variable age of onset, but most patients begin experiencing symptoms in childhood or adolescence.



Prevalence

It is estimated to affect 1 in 40,000 to 60,000 people worldwide.



Typical symptoms

The typical symptoms of it neuropathic pain, skin rashes, gastrointestinal distress, hearing loss, and corneal opacity.



Treatment

Treatment for it includes enzyme replacement therapy, which helps to replace the missing enzyme in the body, and supportive care for associated symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894828	intergenic	CC	normal
RS869312138	intergenic	AA	normal

RS869312151	intergenic	GG	normal
RS869312149	intergenic	TT	normal
RS869312148	intergenic	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Fabry Disease uses 95 polymorphic loci on the *GLA* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Fabry Disease.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Fabry Disease, or if you feel that you have a risk of developing Fabry Disease, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 95 polymorphic loci associated with the risk of Fabry Disease, such as rs104894828, rs104894830 and rs104894831.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Fabry Disease.

Fanconi Anemia (FA)

It is a rare genetic disorder that affects the body's ability to produce healthy blood cells. It is primarily an autosomal recessive disorder which means that an individual must inherit two copies of the defective gene, one from each parent, to develop the disease.

My Fanconi Anemia (FA)

Non-carrier

Knowledge



Time of onset

Symptoms usually begin to appear in infancy or childhood.



Prevalence

It occurs in about 1 in 360,000 births in the general population.



Typical symptoms

The typical symptoms of it include fatigue, weakness, shortness of breath, and an increased susceptibility to infections.



Treatment

Treatment of it usually involves supportive care such as blood transfusions, bone marrow transplants, and medications to manage infections and other complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104886456	FANCC	TT	normal
RS730880277	intergenic	II	normal

RS587782065	intergenic	II	normal
RS587782081	intergenic	II	normal
RS587782170	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Fanconi Anemia (FA) uses 207 polymorphic loci on genes such as *FANCC*, *BRIP1* and *BRCA1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Fanconi Anemia (FA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Fanconi Anemia (FA), or if you feel that you have a risk of developing Fanconi Anemia (FA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 207 polymorphic loci associated with the risk of Fanconi Anemia (FA), such as rs104886456, rs1057516291 and rs1057519365.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Fanconi Anemia (FA).

FG Syndrome

It is a rare genetic disorder that affects the development and function of multiple body systems, including the central nervous system.

My FG Syndrome
Non-carrier

Knowledge

Time of onset

It is usually diagnosed during infancy or early childhood but can present later in life.

Prevalence

The prevalence of it is unknown, but it is considered a rare disorder.

Typical symptoms

The typical symptoms of it include developmental delays, intellectual disability, and distinctive facial features such as a wide mouth and low-set ears.

Treatment

Treatment for it is supportive and focuses on managing the symptoms of the disorder. This may include physical therapy, speech therapy, and educational interventions.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS387907360	intergenic	GG	normal
RS398122844	CASK	TT	normal

Test details

Applicable situation

Genetic disease screening test for FG Syndrome uses 2 polymorphic loci on the *MED12* and *CASK* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of FG Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of FG Syndrome, or if you feel that you have a risk of developing FG Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of FG Syndrome, such as rs387907360 and rs398122844.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of FG Syndrome.

Floating-Harbor Syndrome (FHS)

It is a rare genetic disorder that affects various parts of the body, such as bones, muscles, and facial features.

My Floating-Harbor Syndrome (FHS)

Non-carrier

Knowledge



Time of onset

The symptoms of it mostly appear during childhood or in early adulthood and may gradually worsen over the years.



Prevalence

It is a rare disease with only about 200 cases reported worldwide.



Typical symptoms

Some common symptoms of it include growth retardation that starts before and after birth, speech and language development delays, intellectual disabilities, and other behavioral problems, like sleep disturbance and anxiety. It also affects the skeletal system, leading to shorter stature, abnormal fingers, and toes, and other bone and muscle abnormalities.



Treatment

There is no cure for it, but treatments can help manage and alleviate the symptoms. Speech and language therapy can help with delayed speech development, and physical therapy can improve flexibility, motor function, and coordination. Surgery can also be done to correct the skeletal abnormalities.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS199469464	SRCAP	CC	normal
RS199469465	SRCAP	CC	normal
RS587784444	SRCAP	CC	normal

Test details

Applicable situation

Genetic disease screening test for Floating-Harbor Syndrome (FHS) uses 3 polymorphic loci on the *SRCAP* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Floating-Harbor Syndrome (FHS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Floating-Harbor Syndrome (FHS), or if you feel that you have a risk of developing Floating-Harbor Syndrome (FHS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Floating-Harbor Syndrome (FHS), such as rs199469464, rs199469465 and rs587784444.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Floating-Harbor Syndrome (FHS).

Gaucher Disease (GD)

It is a genetic disorder related to the insufficient activity of an enzyme called glucocerebrosidase. This leads to an accumulation of certain fatty substances in the organs and tissues, causing various symptoms.

My Gaucher Disease (GD)

Non-carrier

Knowledge



Time of onset

The of it varies depending on the type, but it can occur at any age from infancy to adulthood.



Prevalence

It is rare, affecting about 1 in every 50,000 to 100,000 people worldwide.



Typical symptoms

Typical symptoms of it include enlarged liver and spleen, bone pain, anemia, easy bruising, and fatigue.this disease can also cause bone fractures, growth delays, lung disease, and neurological problems. The severity and type of symptoms vary depending on the specific type of this disease.



Treatment

Treatment for this disease include enzyme replacement therapy, medication for pain and other symptoms, and in severe cases, bone marrow transplant.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104886460	intergenic	CC	normal
RS121908309	GBA	GG	normal

RS398123528	GBA	CC	normal
RS78973108	GBA	CC	normal
RS79653797	GBA	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Gaucher Disease (GD) uses 8 polymorphic loci on the *GBA1* and *MSH6* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Gaucher Disease (GD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Gaucher Disease (GD), or if you feel that you have a risk of developing Gaucher Disease (GD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 8 polymorphic loci associated with the risk of Gaucher Disease (GD), such as rs104886460, rs121908309 and rs1553333346.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Gaucher Disease (GD).

Geleophysic Dysplasia

It is a rare genetic disorder that affects various parts of the body, including the heart, lungs, and skeleton. This condition is characterized by short stature, small hands and feet, joint stiffness, thickened skin, and distinctive facial features.

My Geleophysic Dysplasia

Non-carrier

Knowledge



Time of onset

The of it is usually at birth or in early childhood.



Prevalence

It is a very rare disease, and the exact is not known.



Typical symptoms

Typical symptoms of it include short stature, distinctive facial features, limited joint mobility, thickened skin, and breathing difficulties. Some individuals with this condition may also experience heart and liver problems.



Treatment

Treatment options for it are limited, and management typically involves addressing specific symptoms and complications as they arise. Surgery may be necessary to correct skeletal abnormalities or address breathing difficulties. Some individuals may require ongoing medical care to manage heart or liver problems.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS387906622	FBN1	TT	normal

RS387906623	FBN1	CC	normal
RS387906624	FBN1	CC	normal
RS1131691804	intergenic	GG	normal

Test details

Applicable situation

Genetic disease screening test for Geleophysic Dysplasia uses 4 polymorphic loci on the *FBN1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Geleophysic Dysplasia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Geleophysic Dysplasia, or if you feel that you have a risk of developing Geleophysic Dysplasia, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Geleophysic Dysplasia, such as rs1131691804, rs387906622 and rs387906623.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Geleophysic Dysplasia.

Griscelli Syndrome (GS)

It is a rare autosomal recessive genetic disorder that affects skin pigmentation and immune system function. It is characterized by abnormally light skin, silvery hair, neurological disorders, and recurrent infections.

My Griscelli Syndrome (GS)

Non-carrier

Knowledge

Time of onset

It can present in infancy or early childhood.

Prevalence

The prevalence of it is not well established, but it is considered very rare.

Typical symptoms

The most common symptoms of it include hypopigmented skin and hair, neurological abnormalities such as seizures and ataxia, and recurrent infections.

Treatment

Treatment for it is mostly supportive, providing management for infections and neurological symptoms. Bone marrow transplantation may be necessary for those with severe immunodeficiency. Additionally, hair transplantation may help address hypopigmentation in affected individuals.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS786205551	MLPH	GG	normal

Test details

Applicable situation

Genetic disease screening test for Griscelli Syndrome (GS) uses 1 polymorphic locus on the *MLPH* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Griscelli Syndrome (GS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Griscelli Syndrome (GS), or if you feel that you have a risk of developing Griscelli Syndrome (GS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Griscelli Syndrome (GS), namely rs786205551.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Griscelli Syndrome (GS).

Hennekam Lymphangiectasia-Lymphedema Syndrome (HKLLS)

It is a rare genetic disorder characterized by lymphatic vessel abnormalities that can result in lymphedema, swelling in different parts of the body, and severe immune deficiency.

My Hennekam Lymphangiectasia-Lymphedema Syndrome (HKLLS)

Non-carrier

Knowledge



Time of onset

The symptoms of it can appear at birth or develop later in childhood.



Prevalence

It is an extremely rare disease, with only a few hundred reported cases around the world.



Typical symptoms

The typical symptoms of it include swelling in different parts of the body such as limbs, face, fingers, and toes, recurrent infections due to the immune deficiency, and abnormalities in the lymphatic vessels.



Treatment

There is no cure for it, but treatment is focused on managing the symptoms. This may include the use of compression garments, drainage procedures, and antibiotics to treat infections. In some cases, surgery may be necessary. Early diagnosis and treatment can greatly improve the quality of life for individuals with this disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121908250	CCBE1	AA	normal

Test details

Applicable situation

Genetic disease screening test for Hennekam Lymphangiectasia-Lymphedema Syndrome (HKLLS) uses 1 polymorphic locus on the *CCBE1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hennekam Lymphangiectasia-Lymphedema Syndrome (HKLLS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hennekam Lymphangiectasia-Lymphedema Syndrome (HKLLS), or if you feel that you have a risk of developing Hennekam Lymphangiectasia-Lymphedema Syndrome (HKLLS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Hennekam Lymphangiectasia-Lymphedema Syndrome (HKLLS), namely rs121908250.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hennekam Lymphangiectasia-Lymphedema Syndrome (HKLLS).

Hermansky-Pudlak Syndrome (HPS)

It is a rare genetic disorder that affects multiple organs, including the lungs, skin, and eyes. It is caused by mutations in certain genes that affect the production and function of the protein complex responsible for transporting cellular components, such as pigment granules and platelets.

My Hermansky-Pudlak Syndrome (HPS)

Non-carrier

Knowledge



Time of onset

The onset of it can vary, but it usually manifests in early childhood or adolescence.



Prevalence

It is rare, with a prevalence of 1 to 9 in 1,000,000 individuals worldwide, but is more common in certain populations. As an example, the prevalence is 1 in 1800 individuals in the northwestern region of Puerto Rico



Typical symptoms

Typical symptoms of it include bleeding disorders, vision impairment or blindness, and lung disease.



Treatment

There is currently no cure for it, and treatment is focused on managing symptoms. This may include blood transfusions, corticosteroids, oxygen therapy, and lung transplantation in severe cases.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS201227603	HPS3	GG	normal

RS281865084

intergenic

II

normal

Test details

Applicable situation

Genetic disease screening test for Hermansky-Pudlak Syndrome (HPS) uses 2 polymorphic loci on the *HPS3* and *HPS1* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hermansky-Pudlak Syndrome (HPS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hermansky-Pudlak Syndrome (HPS), or if you feel that you have a risk of developing Hermansky-Pudlak Syndrome (HPS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Hermansky-Pudlak Syndrome (HPS), such as rs201227603 and rs281865084.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hermansky-Pudlak Syndrome (HPS).

Hutchinson-Gilford Progeria Syndrom (HGPS)

It is a rare genetic disorder that causes premature aging in children. Individuals with this disease have a shorter lifespan and higher risk of developing age-associated diseases.

My Hutchinson-Gilford Progeria Syndrom (HGPS)

Non-carrier

Knowledge



Time of onset

Onset of it typically occurs in early childhood, usually before the age of 2.



Prevalence

The prevalence of it is estimated to be approximately 1 in 4 million births worldwide.



Typical symptoms

The typical symptoms of it include a characteristic facial appearance (prominent eyes, small lower jaw), hair loss, thinning and tightening of the skin, and delayed growth and development.



Treatment

There is currently no cure for it, but treatment may include medication to manage symptoms and physical therapy to maintain mobility and flexibility.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS60652225	LMNA	TT	normal

Test details

Applicable situation

Genetic disease screening test for Hutchinson-Gilford Progeria Syndrom (HGPS) uses 1 polymorphic locus on the *LMNA* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Hutchinson-Gilford Progeria Syndrom (HGPS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Hutchinson-Gilford Progeria Syndrom (HGPS), or if you feel that you have a risk of developing Hutchinson-Gilford Progeria Syndrom (HGPS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Hutchinson-Gilford Progeria Syndrom (HGPS), namely rs60652225.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Hutchinson-Gilford Progeria Syndrom (HGPS).

Johanson-Blizzard Syndrome (JBS)

It is a rare genetic disorder that affects many parts of the body.

My Johanson-Blizzard Syndrome (JBS)

Non-carrier

Knowledge

Time of onset

It typically appears in infancy or early childhood.

Prevalence

It is an extremely rare disease and its true prevalence is not well-established.

Typical symptoms

Typical symptoms include hearing loss, intellectual disability, abnormal facial features and defects affecting the anus and pancreas.

Treatment

There is currently no cure. Current treatment is based on individual symptoms, for example, surgical interventions, hearing aids and medications to treat infections, nutritional deficiencies or pancreatic insufficiency.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS797045112	UBR1	AA	normal

Test details

Applicable situation

Genetic disease screening test for Johanson-Blizzard Syndrome (JBS) uses 1 polymorphic locus on the *UBR1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Johanson-Blizzard Syndrome (JBS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Johanson-Blizzard Syndrome (JBS), or if you feel that you have a risk of developing Johanson-Blizzard Syndrome (JBS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Johanson-Blizzard Syndrome (JBS), namely rs797045112.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Johanson-Blizzard Syndrome (JBS).

Joubert Syndrome (JS)

It is a rare genetic disorder that affects the cerebellum and brainstem, leading to developmental abnormalities and impairments in muscle coordination and control.

My Joubert Syndrome (JS)
Non-carrier

Knowledge



Time of onset

It typically appears during early childhood.



Prevalence

It is estimated to affect around 1 in 80,000 to 100,000 births worldwide.



Typical symptoms

Typical symptoms include hypotonia (low muscle tone), ataxia (lack of muscle coordination), abnormal eye movements and breathing difficulties.



Treatment

There is currently no cure. Treatment is focused on addressing individual symptoms and may include the use of mobility aids, respiratory support and physical therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918128	INPP5E	CC	normal
RS796052129	KIAA0586	AA	normal

RS754279998	intergenic	II	normal
RS757350052	intergenic	GG	normal
RS757863670	B9D2	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Joubert Syndrome (JS) uses 44 polymorphic loci on genes such as *TMEM67*, *INPP5E* and *CPLANE1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Joubert Syndrome (JS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Joubert Syndrome (JS), or if you feel that you have a risk of developing Joubert Syndrome (JS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 44 polymorphic loci associated with the risk of Joubert Syndrome (JS), such as rs115195998, rs121918128 and rs137853107.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Joubert Syndrome (JS).

KBG Syndrome

It is a rare genetic disorder that is named with the first letters of the surnames of the three families that were first diagnosed. It is typically characterized by developmental delay, short stature, craniofacial abnormalities, and macrodontia (especially of the upper central incisors).

My KBG Syndrome
Non-carrier

Knowledge



Time of onset

Syndromes are present at birth or become apparent in early childhood.



Prevalence

The prevalence is not clear, but it is estimated to occur in 1 per 32,000 to 55,000 births.



Typical symptoms

Short stature, intellectual disability, developmental delays, dental abnormalities, macrodontia (especially of the upper central incisors), distinctive facial features, and skeletal abnormalities are the most prevalent symptoms.



Treatment

There is currently no cure. Medical treatment focuses on managing the symptoms that may arise from the disease, such as dental problems or developmental delays.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS797044890	intergenic	CC	normal

RS886039734	intergenic	II	normal
RS886041791	ANKRD11	GG	normal

Test details

Applicable situation

Genetic disease screening test for KBG Syndrome uses 3 polymorphic loci on the *ANKRD11* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of KBG Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of KBG Syndrome, or if you feel that you have a risk of developing KBG Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of KBG Syndrome, such as rs797044890, rs886039734 and rs886041791.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of KBG Syndrome.

Kenny-Caffey Syndrome (KCS)

It is a rare genetic disorder that affects bone development, causing short stature and thick bones in the arms and legs.

My Kenny-Caffey Syndrome (KCS)
Non-carrier

Knowledge



Time of onset

It typically appears in shortly after birth or in early childhood.



Prevalence

It is extremely rare, with only a few hundred cases reported worldwide.



Typical symptoms

Typical symptoms include short stature, thick bones in arms and legs, delayed bone age, low calcium levels in blood, intellectual disability, and seizures.



Treatment

Treatment is supportive and may include calcium and vitamin D supplements and management of seizures. Surgery may be necessary if bone deformities cause problems with mobility or other complications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS587777011	FAM111A	GG	normal

Test details

Applicable situation

Genetic disease screening test for Kenny-Caffey Syndrome (KCS) uses 1 polymorphic locus on the *FAM111A* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Kenny-Caffey Syndrome (KCS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Kenny-Caffey Syndrome (KCS), or if you feel that you have a risk of developing Kenny-Caffey Syndrome (KCS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Kenny-Caffey Syndrome (KCS), namely rs587777011.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Kenny-Caffey Syndrome (KCS).

Kniest Dysplasia

It is a rare genetic disorder that affects bone growth and development.

My Kniest Dysplasia
Non-carrier

Knowledge



Time of onset

The onset of symptoms may occur at birth or during early childhood.



Prevalence

The prevalence is estimated to be fewer than 1 in 1,000,000 individuals worldwide.



Typical symptoms

Typical symptoms include short stature, joint stiffness, a curved spine and vision and hearing problems.



Treatment

Treatment options are limited and are focused on managing symptoms. These may include physical therapy, surgery, and assistive devices such as hearing aids and glasses.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121912877	COL2A1	CC	normal

Test details



Applicable situation

Genetic disease screening test for Kniest Dysplasia uses 1 polymorphic locus on the *COL2A1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

 Notification

1. The test may not cover all the pathogenic genes or loci of Kniest Dysplasia.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

 Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Kniest Dysplasia, or if you feel that you have a risk of developing Kniest Dysplasia, it is recommended that you consult a qualified health professional or clinician for help.

 Test content

This test includes 1 polymorphic locus associated with the risk of Kniest Dysplasia, namely rs121912877.

 Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Kniest Dysplasia.

L1 Syndrome

It is a rare genetic disorder that affects the nervous system, particularly the development of nerve fibers that transmit signals within the brain and spinal cord. This syndrome results in a range of neurological issues.

My L1 Syndrome
Non-carrier

Knowledge



Time of onset

It typically begins in infancy or early childhood.



Prevalence

The estimated prevalence is 1 in 30,000 to 70,000 individuals.



Typical symptoms

Typical symptoms include muscle stiffness, difficulty with walking and coordination, intellectual disability, seizures and an abnormally small head size.



Treatment

There is currently no cure. Treatment is focused on managing the symptoms, for example, physical therapy, anti-spasticity medication, shunt surgery for hydrocephalus, and other supportive measures.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137852520	L1CAM	CC	normal
RS797045673	L1CAM	GG	normal

Test details

Applicable situation

Genetic disease screening test for L1 Syndrome uses 2 polymorphic loci on the *L1CAM* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of L1 Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of L1 Syndrome, or if you feel that you have a risk of developing L1 Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of L1 Syndrome, such as rs137852520 and rs797045673.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of L1 Syndrome.

Leber Congenital Amaurosis (LCA)

It is a rare genetic disorder that affects the retina of the eyes. Patients may experience severe vision loss or blindness.

My Leber Congenital Amaurosis (LCA)

Non-carrier

Knowledge



Time of onset

It is typically diagnosed in the first few months of life, although onset time can vary.



Prevalence

It is estimated to affect 1 in 40,000 to 80,000 newborns.



Typical symptoms

Patients typically experience severe vision loss or blindness from birth or during the first few months of life. Other symptoms may include sensitivity to light, nystagmus and poor or absent pupillary responses.



Treatment

There is currently no cure. Treatment options may include gene therapy, retinal implants, and supportive measures such as vision aids and occupational therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS386834260	RD3	CC	normal
RS773372519	CNGB3	AA	normal

RS863224863	CRX	GG	normal
RS797044761	RDH12	DD	normal
RS786205623	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Leber Congenital Amaurosis (LCA) uses 17 polymorphic loci on genes such as *GPHN*, *RPGRIP1* and *AIPL1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Leber Congenital Amaurosis (LCA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Leber Congenital Amaurosis (LCA), or if you feel that you have a risk of developing Leber Congenital Amaurosis (LCA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 17 polymorphic loci associated with the risk of Leber Congenital Amaurosis (LCA), such as rs116733939, rs192003551 and rs200125117.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Leber Congenital Amaurosis (LCA).

Leigh Syndrome

It is a rare genetic disorder that affects the deep gray nuclei of the brain. It can be caused by mutations in mitochondrial DNA or by deficiencies of pyruvate dehydrogenase. Genetic mutations in mitochondrial DNA interfere with the energy sources that run cells in an area of the brain that plays a role in motor movements.

My Leigh Syndrome Non-carrier

Knowledge

Time of onset

Symptoms typically appear in infancy or early childhood.

Prevalence

It is estimated to affect at least 1 in 40,000 newborns. However, due to difficulties in diagnosis, the true prevalence may be higher.

Typical symptoms

Typical symptoms include poor feeding, vomiting, irritability, hypotonia, developmental delay, seizures, optic atrophy and breathing problems.

Treatment

There is currently no cure. Treatment may involve supportive care such as nutrition management, respiratory support and anti-epileptic medications. Some patients may benefit from therapies that aim to improve the function of the mitochondria, including coenzyme Q10, riboflavin and other vitamins or supplements.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS118192100	intergenic	GG	normal
RS267606891	MT-ND3	GG	normal
RS782190413	SURF1	GG	normal
RS768050261	intergenic	CC	normal
RS587780529	intergenic	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Leigh Syndrome uses 22 polymorphic loci on genes such as *MT-TK*, *MT-ATP6* and *MT-TL1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Leigh Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Leigh Syndrome, or if you feel that you have a risk of developing Leigh Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 22 polymorphic loci associated with the risk of Leigh Syndrome, such as rs118192100, rs1556423632 and rs199474657.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Leigh Syndrome.

Lesch-Nyhan Syndrome (LNS)

It is a rare genetic disorder that affects how the body produces and processes purines. It is caused by a deficiency of the enzyme hypoxanthine-guanine phosphoribosyltransferase (HPRT) and is characterized by neurological dysfunction and increased levels of uric acid in the blood and urine.

My Lesch-Nyhan Syndrome (LNS)

Non-carrier

Knowledge



Time of onset

It typically appears within the first year of life, but can appear as late as age 3.



Prevalence

It is estimated to affect 1 in 235,000 to 380,000 live births.



Typical symptoms

The most notable symptom is self-injurious behavior, which may include biting, scratching, or head-banging. There may also be high levels of uric acid in blood and urine, which can lead to gout, kidney stones and other complications. Other symptoms may include developmental delays, weak muscle tone (hypotonia) and involuntary muscle movements (dystonia).



Treatment

There is currently no cure. Treatment may include medications to manage neurological symptoms, as well as behavioral and occupational therapy to help individuals with self-injurious behavior and cognitive impairments. Dietary modifications to manage uric acid levels may also be necessary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS137852480	HPRT1	TT	normal
RS137852486	HPRT1	TT	normal
RS137852487	HPRT1	GG	normal
RS137852489	HPRT1	CC	normal
RS137852490	HPRT1	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Lesch-Nyhan Syndrome (LNS) uses 9 polymorphic loci on the *HPRT1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Lesch-Nyhan Syndrome (LNS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Lesch-Nyhan Syndrome (LNS), or if you feel that you have a risk of developing Lesch-Nyhan Syndrome (LNS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 9 polymorphic loci associated with the risk of Lesch-Nyhan Syndrome (LNS), such as rs137852480, rs137852486 and rs137852487.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Lesch-Nyhan Syndrome (LNS).

Lowe Syndrome

It is a rare genetic disorder that affects the eyes, brain, and kidneys.

My Lowe Syndrome
Non-carrier

Knowledge



Time of onset

It typically appears in early infancy or childhood.



Prevalence

It has an estimated prevalence of 1 in 500,000 to 1 in a million individuals worldwide.



Typical symptoms

Typical symptoms include cataracts, glaucoma, intellectual disability, delayed development, muscle weakness, and kidney problems.



Treatment

Treatment may include medications, surgery, physical therapy, and other supportive therapies for managing symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS794727333	OCRL	CC	normal
RS794727182	intergenic	GG	normal

Test details

Applicable situation

Genetic disease screening test for Lowe Syndrome uses 2 polymorphic loci on the *OCRL* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Lowe Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Lowe Syndrome, or if you feel that you have a risk of developing Lowe Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Lowe Syndrome, such as rs794727182 and rs794727333.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Lowe Syndrome.

Meckel Syndrome (MKS)

It is a rare genetic disorder that affects multiple organ systems of the body. It is characterized by brain abnormalities, polydactyly, cystic kidney disease and hepatic ductal anomalies.

My Meckel Syndrome (MKS)

Non-carrier

Knowledge

Time of onset

The onset usually occurs during fetal development.

Prevalence

It is estimated to affect 1 in 13,000 to 140,000 births.

Typical symptoms

Typical symptoms include a small head, cleft palate or lip, bilateral polycystic kidneys, occipital encephalocele, polydactyly and hepatic fibrosis. Other clinical features may include pulmonary hypoplasia, cardiac abnormalities and genital anomalies.

Treatment

Treatment is usually palliative and supportive as there are currently no known cures. For example, management of kidney function, surgery for polydactyly and other supportive cares.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918204	RPGRIP1L	GG	normal
RS755459875	TMEM216	TT	normal

RS797045706	MKS1	GG	normal
RS794727070	intergenic	TT	normal
RS781252161	CC2D2A	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Meckel Syndrome (MKS) uses 18 polymorphic loci on genes such as *RPGRIP1L*, *TCTN2* and *CEP290*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Meckel Syndrome (MKS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Meckel Syndrome (MKS), or if you feel that you have a risk of developing Meckel Syndrome (MKS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 18 polymorphic loci associated with the risk of Meckel Syndrome (MKS), such as rs121918204, rs145665129 and rs1555293215.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Meckel Syndrome (MKS).

Meier-Gorlin Syndrome (MGS)

It is a rare genetic disorder that affects the development of several body parts, including the head, ears and bones of the limbs. It is caused by mutations in genes involved in DNA replication and cell division.

My Meier-Gorlin Syndrome (MGS)

Non-carrier

Knowledge



Time of onset

It is present at birth and usually diagnosed in infancy or early childhood.



Prevalence

It is estimated to affect 1 in 1 million individuals worldwide.



Typical symptoms

Typical symptoms include microcephaly, facial anomalies, cleft lip or palate, cardiac anomalies and hearing loss. Intellectual disability may also be present in some cases.



Treatment

Treatment mainly involves improving symptoms and may include medications to manage associated conditions, surgery to correct physical abnormalities, and physical therapy to improve mobility and strength.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS143141689	intergenic	CC	normal
RS200652608	CDT1	GG	normal

RS387906828	intergenic	CC	normal
RS387906847	ORC4	TT	normal
RS387906917	CDT1	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Meier-Gorlin Syndrome (MGS) uses 10 polymorphic loci on genes such as *ORC1*, *ORC6* and *CDT1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Meier-Gorlin Syndrome (MGS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Meier-Gorlin Syndrome (MGS), or if you feel that you have a risk of developing Meier-Gorlin Syndrome (MGS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 10 polymorphic loci associated with the risk of Meier-Gorlin Syndrome (MGS), such as rs143141689, rs200089121 and rs200652608.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Meier-Gorlin Syndrome (MGS).

Miyoshi Muscular Dystrophy (MMD)

It is a rare genetic disorder that affects the skeletal muscles in the lower limbs, causing progressive weakness and wasting of the muscles.

My Miyoshi Muscular Dystrophy (MMD)

Non-carrier

Knowledge



Time of onset

Symptoms typically begin to appear in young adulthood, usually in the age of 20-30.



Prevalence

It is estimated to affect 1 in 1,000 to 10,000 individuals.



Typical symptoms

Typical symptoms include muscle weakness and wasting in the lower limbs, difficulty in walking and climbing stairs and frequent falls.



Treatment

There is currently no cure. Treatment may include physical therapy, mobility aids and medications.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121908956	DYSF	CC	normal
RS398123800	intergenic	GG	normal
RS766016391	DYSF	GG	normal
RS758180890	DYSF	CC	normal

RS746315830

DYSF

CC

normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Miyoshi Muscular Dystrophy (MMD) uses 22 polymorphic loci on the *DYSF* and *ANO5* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Miyoshi Muscular Dystrophy (MMD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Miyoshi Muscular Dystrophy (MMD), or if you feel that you have a risk of developing Miyoshi Muscular Dystrophy (MMD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 22 polymorphic loci associated with the risk of Miyoshi Muscular Dystrophy (MMD), such as rs121908956, rs121908958 and rs121908959.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Miyoshi Muscular Dystrophy (MMD).

Mowat-Wilson Syndrome (MWS)

It is a rare genetic disorder characterized by intellectual disability, distinctive facial features, and a variety of other unique features.

My Mowat-Wilson Syndrome (MWS)

Non-carrier

Knowledge



Time of onset

This disease is usually apparent at birth, although its distinctive features may not be recognized until later.



Prevalence

It is estimated to affect fewer than 1 in 50,000 individuals.



Typical symptoms

Typical symptoms include intellectual disability, delayed development of speech and motor skills, seizures, constipation, and distinctive facial features such as a prominent chin and wide-set eyes.



Treatment

Treatment may include medication for seizures and constipation and developmental therapies such as speech and occupational therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137852981	ZEB2	GG	normal
RS730881211	intergenic	II	normal

RS797046122	ZEB2	GG	normal
RS797046121	ZEB2	GG	normal
RS797046118	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Mowat-Wilson Syndrome (MWS) uses 26 polymorphic loci on the *ZEB2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Mowat-Wilson Syndrome (MWS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Mowat-Wilson Syndrome (MWS), or if you feel that you have a risk of developing Mowat-Wilson Syndrome (MWS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 26 polymorphic loci associated with the risk of Mowat-Wilson Syndrome (MWS), such as rs137852981, rs397515448 and rs398124274.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Mowat-Wilson Syndrome (MWS).

Myhre Syndrome

It is a rare genetic disease that affects multiple organ systems in the human body, particularly characterized by physical abnormalities and developmental delays.

My Myhre Syndrome
Non-carrier

Knowledge



Time of onset

It is typically diagnosed in childhood.



Prevalence

It is extremely rare with only a few hundred cases reported in the medical literature.



Typical symptoms

Typical symptoms include short stature, distinctive facial features, hearing loss, joint stiffness, heart abnormalities, and developmental delays.



Treatment

Treatment may include surgeries to correct physical abnormalities, hearing aids for hearing loss, and physical therapy to improve joint mobility. In addition, some individuals may benefit from speech and language therapy, behavioral therapy or other developmental interventions.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS281875320	SMAD4	AA	normal
RS281875321	SMAD4	TT	normal

RS281875322	SMAD4	AA	normal
RS397518413	SMAD4	CC	normal
RS1555686624	intergenic	TT	normal

Test details

Applicable situation

Genetic disease screening test for Myhre Syndrome uses 5 polymorphic loci on the *SMAD4* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Myhre Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Myhre Syndrome, or if you feel that you have a risk of developing Myhre Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Myhre Syndrome, such as rs1555686624, rs281875320 and rs281875321.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Myhre Syndrome.

Nance-Horan Syndrome (NHS)

It is a rare genetic disorder that affects several parts of the body. It is characterized by dental abnormalities, eye abnormalities, and facial dysmorphism.

My Nance-Horan Syndrome (NHS)

Non-carrier

Knowledge



Time of onset

It typically appears at birth or in early childhood.



Prevalence

It is estimated to affect fewer than 1 in 200,000 individuals worldwide.



Typical symptoms

Typical symptoms include dental abnormalities, eye abnormalities, and facial dysmorphism. Other clinical features may include intellectual disability, developmental delays, hearing loss, and skeletal abnormalities.



Treatment

Treatment typically involves management of individual symptoms. For example, dental abnormalities may require orthodontic treatment, while cataracts may require surgery.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS132630322	NHS	CC	normal

Test details

Applicable situation

Genetic disease screening test for Nance-Horan Syndrome (NHS) uses 1 polymorphic locus on the *NHS* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Nance-Horan Syndrome (NHS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Nance-Horan Syndrome (NHS), or if you feel that you have a risk of developing Nance-Horan Syndrome (NHS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Nance-Horan Syndrome (NHS), namely rs132630322.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Nance-Horan Syndrome (NHS).

Neu-Laxova Syndrome (NLS)

It is a rare, lethal genetic disorder that affects fetal development. It is characterized by severe growth deficiency, abnormal skin and facial features, resulting in intellectual disability or stillbirth.

My Neu-Laxova Syndrome (NLS)

Non-carrier

Knowledge

Time of onset

It is present at birth, although the diagnosis can be difficult due to the complexity of symptoms.

Prevalence

It is an extremely rare disease that has been reported in fewer than 100 cases worldwide.

Typical symptoms

Typical symptoms include abnormal birth weight and body length, thick and scaly skin, microcephaly, micrognathia, a beaked nose and abnormally shaped ears. Other symptoms are cleft lip and palate, abnormal brain development, hydrocephalus, abnormal spinal curvature and heart defects.

Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support, such as, providing nutrition and respiratory support. Genetic counseling is also recommended for families affected by the disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267606948	PHGDH	GG	normal

Test details

Applicable situation

Genetic disease screening test for Neu-Laxova Syndrome (NLS) uses 1 polymorphic locus on the *PHGDH* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Neu-Laxova Syndrome (NLS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Neu-Laxova Syndrome (NLS), or if you feel that you have a risk of developing Neu-Laxova Syndrome (NLS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Neu-Laxova Syndrome (NLS), namely rs267606948.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Neu-Laxova Syndrome (NLS).

Nicolaides-Baraitser Syndrome (NCBRS)

It is a rare genetic disorder that primarily affects the development of the nervous system and causes intellectual disability along with various physical abnormalities.

My Nicolaides-Baraitser Syndrome (NCBRS)

Non-carrier

Knowledge

 Time of onset

It typically appears in infancy or early childhood.

 Prevalence

It has only about 50 cases reported in the medical literature to date.

 Typical symptoms

Typical symptoms include intellectual disability, seizures, distinctive facial features and abnormalities of the digits.

 Treatment

Treatment may include special education, physical therapy, speech therapy, and antiepileptic medication for seizures.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS281875189	SMARCA2	CC	normal
RS797045974	SMARCA2	CC	normal

RS797045976

SMARCA2

CC

normal

Test details

Applicable situation

Genetic disease screening test for Nicolaides-Baraitser Syndrome (NCBRS) uses 3 polymorphic loci on the *SMARCA2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Nicolaides-Baraitser Syndrome (NCBRS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Nicolaides-Baraitser Syndrome (NCBRS), or if you feel that you have a risk of developing Nicolaides-Baraitser Syndrome (NCBRS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Nicolaides-Baraitser Syndrome (NCBRS), such as rs281875189, rs797045974 and rs797045976.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Nicolaides-Baraitser Syndrome (NCBRS).

Niemann-Pick Disease (NPD)

It is a group of serious hereditary metabolic disorder, in which sphingomyelin accumulates in the lysosome in the cell, which will lead to hepatosplenomegaly, abdominal distension and pain. Symptoms such as decreased platelet levels occur.

My Niemann-Pick Disease (NPD)

Non-carrier

Knowledge

Time of onset

The onset and severity varies depending on the type. Type A typically presents in infancy, Type B in childhood or adolescence, and Type C may present in childhood, adolescence, or adulthood.

Prevalence

It is estimated to affect 1 in 150,000 to 250,000 live births for Type A and Type B, and 1 in 120,000 to 150,000 for Type C.

Typical symptoms

Typical symptoms include hepatosplenomegaly, neurological impairment, developmental delays, feeding difficulties and respiratory problems.

Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support. Enzyme replacement therapy has been approved for use in Type B, and clinical trials for other treatments, including gene therapy and pharmacological chaperone therapy, are ongoing.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS120074118	SMPD1	II	normal
RS80358264	NPC2	AA	normal
RS886042268	intergenic	TT	normal
RS794727897	NPC1	CC	normal
RS794727252	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Niemann-Pick Disease (NPD) uses 34 polymorphic loci on genes such as *SMPD1*, *APBB1* and *NPC1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Niemann-Pick Disease (NPD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Niemann-Pick Disease (NPD), or if you feel that you have a risk of developing Niemann-Pick Disease (NPD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 34 polymorphic loci associated with the risk of Niemann-Pick Disease (NPD), such as rs1057516403, rs120074118 and rs120074119.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Niemann-Pick Disease (NPD).

Ogden Syndrome

It is a rare genetic disorder that affects the development of the brain and other organs. The condition is characterized by severe intellectual disability, short stature, and hypertelorism.

My Ogden Syndrome
Non-carrier

Knowledge



Time of onset

It typically appears at birth or become apparent in early infancy.



Prevalence

It is extremely rare, with only a handful of cases reported worldwide.



Typical symptoms

Typical symptoms include severe intellectual disability, developmental delays, short stature, and hypertelorism. Additional symptoms may include neurological problems, hearing loss, and seizures.



Treatment

Treatment may include medications to control seizures, physical therapy to improve mobility, and early intervention programs on development.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS587780563	NAA10	GG	normal
RS797044868	NAA10	GG	normal

RS878853263

NAA10

AA

normal

Test details

Applicable situation

Genetic disease screening test for Ogden Syndrome uses 3 polymorphic loci on the *NAA10* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Ogden Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Ogden Syndrome, or if you feel that you have a risk of developing Ogden Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Ogden Syndrome, such as rs587780563, rs797044868 and rs878853263.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Ogden Syndrome.

Pelizaeus-Merzbacher Disease (PMD)

It is a rare, progressive, and degenerative central nervous system disorder that deteriorates coordination, motor abilities, and cognitive function.

My Pelizaeus-Merzbacher Disease (PMD)

Non-carrier

Knowledge



Time of onset

Symptoms are typically apparent in infancy or early childhood, but can sometimes present in adolescence or even adulthood.



Prevalence

It is estimated to affect 1 in every 200,000 births worldwide.



Typical symptoms

The major symptom is the presence of nystagmus. Other early symptoms may include delayed motor development, difficulty with walking and spasticity. Cognitive impairment is also common, often manifesting as delayed speech and language development.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support, for example, physical therapy, occupational therapy and speech therapy to address developmental and motor delays. Medications may be used to manage spasticity and other symptoms. Gene therapy is a promising avenue of research for future development.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS11543022	PLP1	CC	normal

RS132630278	PLP1	CC	normal
RS132630279	PLP1	TT	normal
RS132630281	PLP1	GG	normal
RS132630282	PLP1	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Pelizaeus-Merzbacher Disease (PMD) uses 8 polymorphic loci on the *PLP1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Pelizaeus-Merzbacher Disease (PMD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Pelizaeus-Merzbacher Disease (PMD), or if you feel that you have a risk of developing Pelizaeus-Merzbacher Disease (PMD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 8 polymorphic loci associated with the risk of Pelizaeus-Merzbacher Disease (PMD), such as rs11543022, rs132630278 and rs132630279.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Pelizaeus-Merzbacher Disease (PMD).

Rapadilino Syndrome

It is a rare genetic disorder that affects growth and development. It is characterized by short stature, hip dislocation, absent or malformed thumbs, and a distinctive facial appearance.

My Rapadilino Syndrome

Non-carrier

Knowledge



Time of onset

It may be evident at birth, but typically appear within the first year of life.



Prevalence

It is an extremely rare disorder with only a few dozen cases reported worldwide. It appears to be more common in the Finnish population.



Typical symptoms

Typical symptoms include short stature, absent or malformed thumbs, hip dislocation, cleft palate, dental abnormalities, and a unique facial appearance.



Treatment

Treatment may include physical therapy, orthopedic interventions such as braces or surgery, dental care, and speech therapy. Regular monitoring for potential complications such as scoliosis or heart defects is also recommended.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS386833844	RECQL4	GG	normal

Test details

Applicable situation

Genetic disease screening test for Rapadilino Syndrome uses 1 polymorphic locus on the *RECQL4* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Rapadilino Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Rapadilino Syndrome, or if you feel that you have a risk of developing Rapadilino Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Rapadilino Syndrome, namely rs386833844.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Rapadilino Syndrome.

Refsum Disease (RD)

Also known as phytanic acid storage disease, it is a rare genetic disease, which is caused by the accumulation of phytanic acid in the body due to the metabolic disorder in the peroxisome.

My Refsum Disease (RD)

Non-carrier

Knowledge



Time of onset

Symptoms usually begin in late childhood or adolescence.



Prevalence

It is estimated to affect 1 in 1 million people worldwide.



Typical symptoms

The most common symptoms include vision loss, nerve damage and bone abnormalities. Other symptoms may include hearing loss, skin changes and heart problems.



Treatment

There is currently no cure, but treatment can help manage symptoms. The main treatment is dietary restriction of phytanic acid, which is found in certain types of food such as dairy products and red meat. Other treatments may include medications to control symptoms or surgery to correct severe skeletal abnormalities.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894173	PHYH	CC	normal

RS104894174

PHYH

CC

normal

Test details

Applicable situation

Genetic disease screening test for Refsum Disease (RD) uses 2 polymorphic loci on the *PHYH* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Refsum Disease (RD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Refsum Disease (RD), or if you feel that you have a risk of developing Refsum Disease (RD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Refsum Disease (RD), such as rs104894173 and rs104894174.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Refsum Disease (RD).

Renpenning Syndrome (RENS)

It is a rare genetic disorder that primarily affects males. It is characterized by intellectual disability, physical abnormalities, and problems with language and speech development.

My Renpenning Syndrome (RENS)

Non-carrier

Knowledge



Time of onset

It typically appears in infancy or early childhood.



Prevalence

It is estimated to affect fewer than 1 in 1 million people worldwide.



Typical symptoms

The most common symptoms include intellectual disability, delayed speech and language development, and physical abnormalities.



Treatment

Treatment may include special education programs for those with intellectual disabilities, and speech therapy for speech and language delays.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS606231193	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Renpenning Syndrome (RENS) uses 1 polymorphic locus on the *PQBP1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Renpenning Syndrome (RENS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Renpenning Syndrome (RENS), or if you feel that you have a risk of developing Renpenning Syndrome (RENS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Renpenning Syndrome (RENS), namely rs606231193.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Renpenning Syndrome (RENS).

Rett Syndrome (RTT)

It is a rare genetic disease that affects the development and function of the central nervous system due to genetic mutations in transcription regulatory factors.

My Rett Syndrome (RTT)

Non-carrier

Knowledge



Time of onset

The onset usually occurs between 6 and 18 months of age.



Prevalence

It is estimated to affect 1 in 10,000 to 15,000 live births. Females are more prevalent.



Typical symptoms

Typical symptoms include loss of speech and purposeful hand movements, repetitive hand movements, breathing difficulties during sleep, and cognitive and motor disabilities.



Treatment

Treatment for is mostly supportive and involves speech and physical therapy. Medications for breathing and muscle stiffness may also be used. Gene therapy and stem cell therapy are promising areas of research for potential treatments.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121913678	FOXG1	GG	normal
RS61750250	CDKL5	II	normal

RS61748399	MECP2	TT	normal
RS61748400	MECP2	II	normal
RS61748402	MECP2	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Rett Syndrome (RTT) uses 71 polymorphic loci on the *FOXP1*, *CDKL5* and *MECP2* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Rett Syndrome (RTT).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Rett Syndrome (RTT), or if you feel that you have a risk of developing Rett Syndrome (RTT), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 71 polymorphic loci associated with the risk of Rett Syndrome (RTT), such as rs121913678, rs122460159 and rs267606827.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Rett Syndrome (RTT).

Revesz Syndrome

It is a rare severe phenotypic variant of dyskeratosis congenita with an onset in early childhood. excessive skin pigmentation/hypopigmentation, nail malnutrition, oral leukoplakia, bone marrow failure, cancer, developmental delay, etc. may occur.

My Revesz Syndrome

Non-carrier

Knowledge



Time of onset

It typically appears in early childhood, although symptoms may appear later in life.



Prevalence

It is an extremely rare disorder, with only a few cases reported in the medical literature.



Typical symptoms

Typical symptoms include intellectual disability, developmental delay, seizures, abnormal eye movements, skeletal abnormalities, and liver dysfunction.



Treatment

Supportive care such as physical, occupational, and speech therapy can be helpful, as can medications to control seizures and other medical problems.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121918544	TINF2	CC	normal

Test details

Applicable situation

Genetic disease screening test for Revesz Syndrome uses 1 polymorphic locus on the *TINF2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Revesz Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Revesz Syndrome, or if you feel that you have a risk of developing Revesz Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Revesz Syndrome, namely rs121918544.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Revesz Syndrome.

Roberts Syndrome

It is a rare genetic disorder that affects the growth and development of numerous parts of the body. It is characterized by limb abnormalities, facial anomalies, and distinctive craniofacial characteristics.

My Roberts Syndrome
Non-carrier

Knowledge



Time of onset

It is typically diagnosed at birth, although mild cases may go unnoticed until later in life.



Prevalence

It is estimated to affect 1 in 100,000 to 1 in 1,000,000 live births.



Typical symptoms

Typical symptoms include limb reduction defects, cleft lip/palate, microcephaly, intellectual disability, and abnormal facial features.



Treatment

Usually, skeletal deformities or cleft lip/palate are corrected through surgery; The use of hearing aids or other assistive devices can help with hearing loss issues.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS80359852	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Roberts Syndrome uses 1 polymorphic locus on the *ESCO2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Roberts Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Roberts Syndrome, or if you feel that you have a risk of developing Roberts Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Roberts Syndrome, namely rs80359852.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Roberts Syndrome.

Robinow Syndrome

It is a very rare genetic disorder. It can cause bone abnormalities, unusual facial features, genital abnormalities and other problems. The condition is usually genetic in one of two ways, but the genetic mutation can happen randomly. The autosomal recessive form is usually more severe than the autosomal dominant form.

My Robinow Syndrome

Non-carrier

Knowledge



Time of onset

It can begin before birth or in early childhood.



Prevalence

It is a very rare disease, with only a few hundred cases reported worldwide.



Typical symptoms

The most common symptoms include short stature, distinctive facial features, limb and genital abnormalities, and spinal issues.



Treatment

Treatment mainly involves improving symptoms and providing support. In severe cases, surgery may be required.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS797044834	intergenic	II	normal

RS797044836	intergenic	II	normal
RS797044837	intergenic	II	normal
RS869025216	intergenic	AA	normal
RS869025217	intergenic	GG	normal

Test details

Applicable situation

Genetic disease screening test for Robinow Syndrome uses 5 polymorphic loci on the *DVL1* and *DVL3* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Robinow Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Robinow Syndrome, or if you feel that you have a risk of developing Robinow Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Robinow Syndrome, such as rs797044834, rs797044836 and rs797044837.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Robinow Syndrome.

Rothmund-Thomson Syndrome (RTS)

It is a rare, genetic disorder that affects many parts of the body, especially the skin, eyes, bones, hair, and teeth. The main sign or symptom is a red blistering rash on the face that begins in early infancy.

My Rothmund-Thomson Syndrome (RTS)

Non-carrier

Knowledge



Time of onset

It can present at any age.



Prevalence

It is estimated to affect fewer than 1 in 1 million individuals.



Typical symptoms

Typical symptoms include a rash on the face and limbs, small stature, skeletal abnormalities, sensitivity to sunlight, cataracts, and dental abnormalities.



Treatment

The treatment of this disease aims to manage symptoms, including regular skin examinations to monitor for skin cancer; Wear protective clothing and sunscreen to reduce sunlight exposure; Surgical treatment for skeletal abnormalities or cataracts; Regular oral examination and treatment of dental abnormalities.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137853229	RECQL4	GG	normal

RS386833851	RECQL4	GG	normal
RS398124117	intergenic	CC	normal

Test details

Applicable situation

Genetic disease screening test for Rothmund-Thomson Syndrome (RTS) uses 3 polymorphic loci on the *RECQL4* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Rothmund-Thomson Syndrome (RTS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Rothmund-Thomson Syndrome (RTS), or if you feel that you have a risk of developing Rothmund-Thomson Syndrome (RTS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Rothmund-Thomson Syndrome (RTS), such as rs137853229, rs386833851 and rs398124117.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Rothmund-Thomson Syndrome (RTS).

Rubinstein-Taybi Syndrome (RSTS)

It is a rare genetic disorder that affects multiple organ systems and is characterized by physical abnormalities, developmental delays, and intellectual disability.

My Rubinstein-Taybi Syndrome (RSTS)

Non-carrier

Knowledge



Time of onset

It typically appears in infancy or early childhood.



Prevalence

It is estimated to affect around 1 in 125,000 to 720,000 live births.



Typical symptoms

Typical symptoms include distinctive facial features, broad thumbs and toes, developmental delays, intellectual disability, and health issues.



Treatment

Treatment may include physical therapy, speech therapy, medications and surgery. Early intervention is important for maximizing function and quality of life.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS200782888	intergenic	CC	normal
RS587783516	CREBBP	GG	normal

RS587783494	CREBBP	TT	normal
RS587783496	CREBBP	TT	normal
RS587783497	CREBBP	TT	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Rubinstein-Taybi Syndrome (RSTS) uses 55 polymorphic loci on the *EP300* and *CREBBP* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Rubinstein-Taybi Syndrome (RSTS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Rubinstein-Taybi Syndrome (RSTS), or if you feel that you have a risk of developing Rubinstein-Taybi Syndrome (RSTS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 55 polymorphic loci associated with the risk of Rubinstein-Taybi Syndrome (RSTS), such as rs1057517732, rs1057521737 and rs200782888.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Rubinstein-Taybi Syndrome (RSTS).

Saethre-Chotzen Syndrome (SCS)

Also known as acrocephalosyndactyly type III, it is a rare genetic disorder that affects the proper development of the skull and face, as well as the hands and feet.

My Saethre-Chotzen Syndrome (SCS)

Non-carrier

Knowledge



Time of onset

Symptoms are typically present at birth.



Prevalence

It is estimated to affect approximately 1 in 25,000 to 50,000 people in the general population.



Typical symptoms

The most common symptoms include a misshapen skull, facial asymmetry, low-set ears, and short stature.



Treatment

Treatment is primarily focused on symptom management and may include surgery to correct abnormalities in the skull, face, hands or feet, as well as physical therapy to address motor and developmental delays.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121913478	FGFR2	TT	normal
RS121918487	FGFR2	CC	normal

RS121918491	FGFR2	CC	normal
RS77543610	FGFR2	GG	normal

Test details

Applicable situation

Genetic disease screening test for Saethre-Chotzen Syndrome (SCS) uses 4 polymorphic loci on the *FGFR2* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Saethre-Chotzen Syndrome (SCS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Saethre-Chotzen Syndrome (SCS), or if you feel that you have a risk of developing Saethre-Chotzen Syndrome (SCS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Saethre-Chotzen Syndrome (SCS), such as rs121913478, rs121918487 and rs121918491.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Saethre-Chotzen Syndrome (SCS).

Salla Disease (SD)

It is a rare genetic disorder that affects the metabolism of sialic acid that is essential for brain development and function. This disease is a type of leukodystrophies, which are characterized by progressive degeneration of the white matter in the brain.

My Salla Disease (SD)

Non-carrier

Knowledge

Time of onset

The onset is typically in infancy or early childhood.

Prevalence

It is a rare genetic disorder that is more common in individuals of Finnish origin. Its prevalence is 1 in 10,000 individuals in Finland.

Typical symptoms

Typical symptoms include delayed motor development, hypotonia, ataxia, seizures, and visual impairment.

Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support, for example, medications to control seizures, physical therapy to improve muscle tone and mobility, and supportive care to manage the complications of the disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS386833990	SLC17A5	CC	normal

RS201284672	SLC17A5	AA	normal
RS727504156	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Salla Disease (SD) uses 3 polymorphic loci on the *SLC17A5* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Salla Disease (SD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Salla Disease (SD), or if you feel that you have a risk of developing Salla Disease (SD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 3 polymorphic loci associated with the risk of Salla Disease (SD), such as rs201284672, rs386833990 and rs727504156.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Salla Disease (SD).

Sandhoff Disease

It is a rare genetic disease that affects the nervous system. It is a lysine storage disease that causes damage to the function of lysosomal enzymes in the brain and other organs, as well as the accumulation of GM2 gangliosides (a type of fat).

My Sandhoff Disease

Non-carrier

Knowledge



Time of onset

It typically appears in the first few months of life.



Prevalence

It is estimated to affect 1 in 260,000 births.



Typical symptoms

Typical symptoms include developmental delay, progressive loss of motor skills, seizures, and an enlarged liver and spleen.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support. For example, physical therapy, antiepileptic medications, and palliative measures.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121907983	HEXB	GG	normal
RS398123443	intergenic	II	normal

RS398123446	intergenic	AA	normal
RS1580377105	intergenic	II	normal
RS761197472	intergenic	GG	normal

Test details

Applicable situation

Genetic disease screening test for Sandhoff Disease uses 5 polymorphic loci on the *HEXB* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Sandhoff Disease.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Sandhoff Disease, or if you feel that you have a risk of developing Sandhoff Disease, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Sandhoff Disease, such as rs121907983, rs1580377105 and rs398123443.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Sandhoff Disease.

Seckel Syndrome

It is a rare genetic disorder characterized by severe growth retardation, microcephaly, and intellectual disability.

My Seckel Syndrome
Non-carrier

Knowledge



Time of onset

It is typically diagnosed at birth or in early childhood.



Prevalence

It is estimated to affect 1 in 10,000 to 15,000 individuals worldwide.



Typical symptoms

The most common symptoms include abnormally small head, short stature, intellectual disability, and facial abnormalities.



Treatment

Treatment mainly involves improving symptoms. This can include physical therapy, surgery for skeletal abnormalities, and medication for seizures or other associated medical conditions.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS747680111	intergenic	II	normal
RS777893196	intergenic	II	normal
RS797045447	CENPJ	TT	normal

RS797045448	intergenic	II	normal
RS797045450	CENPJ	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Seckel Syndrome uses 7 polymorphic loci on the *NIN* and *CENPJ* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Seckel Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Seckel Syndrome, or if you feel that you have a risk of developing Seckel Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 7 polymorphic loci associated with the risk of Seckel Syndrome, such as rs747680111, rs777893196 and rs797045447.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Seckel Syndrome.

Sengers Syndrome

It is a rare autosomal recessive disorder that affects multiple organs and systems in the body.

My Sengers Syndrome
Non-carrier

Knowledge



Time of onset

It typically appears in infancy or early childhood.



Prevalence

It has only a few cases reported worldwide.



Typical symptoms

Common symptoms include developmental delay, muscle weakness, lactic acidosis, congenital cataracts, and cardiac abnormalities.



Treatment

Treatment may include early intervention therapies, such as physical therapy and occupational therapy, as well as medications to control seizures and balance electrolyte levels.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS746709222	AGK	CC	normal
RS766413410	AGK	CC	normal

Test details

Applicable situation

Genetic disease screening test for Sengers Syndrome uses 2 polymorphic loci on the *AGK* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Sengers Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Sengers Syndrome, or if you feel that you have a risk of developing Sengers Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Sengers Syndrome, such as rs746709222 and rs766413410.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Sengers Syndrome.

Senior-Loken Syndrome (SLSN)

It is a rare genetic disorder that affects vision and kidney function. It is characterized by the gradual degeneration of the retina and the development of kidney disease, leading to eventual blindness and end-stage renal disease.

My Senior-Loken Syndrome (SLSN)

Non-carrier

Knowledge



Time of onset

It typically appears in childhood or early adolescence, usually before the age of 10.



Prevalence

It is estimated to affect 1 in 1,000,000 individuals worldwide.



Typical symptoms

The most common is decreased visual acuity, which can progress to complete blindness. In addition, affected individuals may develop kidney disease, such as renal dysfunction or proteinuria.



Treatment

There is currently no cure. The focus of treatment is on managing the symptoms and complications of the disease. For example, medications to control blood pressure and proteinuria, or dialysis and kidney transplantation for end-stage renal disease.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137852832	CEP290	CC	normal
RS386834158	CEP290	II	normal

RS387906980	WDR19	TT	normal
RS387907009	IQCB1	CC	normal
RS397515335	intergenic	II	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Senior-Loken Syndrome (SLSN) uses 15 polymorphic loci on genes such as *CEP290*, *WDR19* and *IQCB1*. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Senior-Loken Syndrome (SLSN).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Senior-Loken Syndrome (SLSN), or if you feel that you have a risk of developing Senior-Loken Syndrome (SLSN), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 15 polymorphic loci associated with the risk of Senior-Loken Syndrome (SLSN), such as rs137852832, rs376493409 and rs386834158.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Senior-Loken Syndrome (SLSN).

Shwachman-Diamond Syndrome (SDS)

It is a rare genetic disorder that affects the bone marrow, pancreas, and skeletal system. This disease results in a decreased number of functional white blood cells, leading to an increased susceptibility to infections, a reduced ability to absorb nutrients in the gut, and skeletal abnormalities.

My Shwachman-Diamond Syndrome (SDS)

Non-carrier

Knowledge



Time of onset

It typically appears in early childhood, before the age of 2.



Prevalence

It is estimated to affect 1 in 50,000 to 100,000 newborns.



Typical symptoms

Typical symptoms include recurrent infections, diarrhea, malnutrition, short stature, and skeletal abnormalities.



Treatment

Treatment is supportive and includes antibiotics to treat infections, pancreatic enzymes to aid digestion, growth hormone therapy to stimulate growth, and bone marrow transplantation in severe cases.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS113993990	SBDS	II	normal

Test details

Applicable situation

Genetic disease screening test for Shwachman-Diamond Syndrome (SDS) uses 1 polymorphic locus on the *SBDS* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Shwachman-Diamond Syndrome (SDS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Shwachman-Diamond Syndrome (SDS), or if you feel that you have a risk of developing Shwachman-Diamond Syndrome (SDS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Shwachman-Diamond Syndrome (SDS), namely rs113993990.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Shwachman-Diamond Syndrome (SDS).

Smith-Lemli-Opitz Syndrome (SLOS)

It is a rare genetic disease that affects the way the body produces and processes cholesterol. People with this disease may experience developmental disorders in many parts of their body.

My Smith-Lemli-Opitz Syndrome (SLOS)

Non-carrier

Knowledge



Time of onset

The onset varies from infancy to childhood.



Prevalence

It is estimated to affect 1 in 20,000 to 1 in 60,000 live births.



Typical symptoms

Typical symptoms include intellectual disability, developmental delays, poor muscle tone, facial abnormalities, and growth impairments.



Treatment

Treatment may include dietary modifications, cholesterol supplementation, and vitamin supplementation.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104894212	DHCR7	CC	normal
RS104894213	DHCR7	CC	normal

RS121909768	DHCR7	CC	normal
RS80338862	DHCR7	CC	normal
RS143312232	DHCR7	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Smith-Lemli-Opitz Syndrome (SLOS) uses 10 polymorphic loci on the *DHCR7* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Smith-Lemli-Opitz Syndrome (SLOS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Smith-Lemli-Opitz Syndrome (SLOS), or if you feel that you have a risk of developing Smith-Lemli-Opitz Syndrome (SLOS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 10 polymorphic loci associated with the risk of Smith-Lemli-Opitz Syndrome (SLOS), such as rs104894212, rs104894213 and rs121909768.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Smith-Lemli-Opitz Syndrome (SLOS).

Smith-McCort Dysplasia (SMC)

It is a rare genetic disorder that affects bone growth and development. It is characterized by short stature, abnormal curvature of the spine, and joint deformities.

My Smith-McCort Dysplasia (SMC)

Non-carrier

Knowledge



Time of onset

It typically appears at birth or soon after.



Prevalence

It is a rare disease, with only a few hundred cases reported worldwide.



Typical symptoms

Typical symptoms include short stature, joint deformities, and abnormal curvature of the spine.



Treatment

Treatment is focused on managing symptoms, including physical therapy for joint deformities, surgery for spinal curvature, and hearing aids for hearing loss.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS775414124	intergenic	TT	normal

Test details

Applicable situation

Genetic disease screening test for Smith-McCort Dysplasia (SMC) uses 1 polymorphic locus on the *DYM* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Smith-McCort Dysplasia (SMC).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Smith-McCort Dysplasia (SMC), or if you feel that you have a risk of developing Smith-McCort Dysplasia (SMC), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Smith-McCort Dysplasia (SMC), namely rs775414124.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Smith-McCort Dysplasia (SMC).

Sotos Syndrome

It is a rare genetic disorder characterized by overgrowth during childhood, with symptoms including a large head, distinctive facial features, delayed development, and intellectual disability.

My Sotos Syndrome
Non-carrier

Knowledge



Time of onset

It typically appears in childhood, usually between the age of 2 to 5.



Prevalence

It is estimated to affect 1 in 14,000 to 50,000 individuals worldwide.



Typical symptoms

Typical symptoms include overgrowth during childhood, characteristic facial features, developmental delay, and intellectual disability.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms and providing support, for example, early intervention services, such as speech therapy, occupational therapy, and special education, as well as medication to manage behavioral or psychiatric symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121908070	NSD1	CC	normal
RS587784181	NSD1	CC	normal

RS587784198	intergenic	II	normal
RS587784197	intergenic	II	normal
RS587784196	NSD1	AA	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Sotos Syndrome uses 125 polymorphic loci on the *NSD1* and *SCN4A* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Sotos Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Sotos Syndrome, or if you feel that you have a risk of developing Sotos Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 125 polymorphic loci associated with the risk of Sotos Syndrome, such as rs1057520339, rs121908070 and rs398124378.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Sotos Syndrome.

Stargardt Disease

It is a hereditary eye disease that can lead to retinal degeneration and visual loss.
This disease is the most common form of hereditary macular degeneration.

My Stargardt Disease
Non-carrier

Knowledge



Time of onset

It typically appears in teenage years or early adulthood.



Prevalence

It is estimated to affect approximately 1 in 8,000 to 10,000 individuals.



Typical symptoms

Typical symptoms include blurry or distorted vision, difficulty seeing in low light, and decreased color vision.



Treatment

There is currently no cure. Treatment may include using low-vision aids such as magnifying glasses or electronic devices to help improve vision. Some individuals may also benefit from cutting-edge gene therapy or stem cell therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137853006	PROM1	GG	normal
RS150774447	ABCA4	CC	normal

RS527236098	PRPH2	CC	normal
RS61750200	ABCA4	GG	normal
RS61751383	ABCA4	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Stargardt Disease uses 16 polymorphic loci on the *PROM1*, *ABCA4* and *PRPH2* genes. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Stargardt Disease.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Stargardt Disease, or if you feel that you have a risk of developing Stargardt Disease, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 16 polymorphic loci associated with the risk of Stargardt Disease, such as rs137853006, rs150774447 and rs201471607.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Stargardt Disease.

Tay-Sachs Disease (TSD)

It is a rare, genetic neurodegenerative disorder that affects the metabolism of gangliosides. This disease is caused by a deficiency of an enzyme called hexosaminidase A, which results in the accumulation of gangliosides in brain and nerve cells. Over time, the gangliosides cause progressive damage to these cells, leading to the characteristic symptoms of the disease.

My Tay-Sachs Disease (TSD)

Non-carrier

Knowledge

Time of onset

This disease typically becomes apparent in an infant's first few months of life.

Prevalence

It is estimated to affect 1 in 320,000 live births worldwide.

Typical symptoms

The initial symptoms may include developmental delays, progressive weakness, and exaggerated startle response to loud noises. Over time, other symptoms may develop, such as loss of vision and hearing, seizures, and difficulty swallowing, breathing, and moving.

Treatment

Presently there is no specific treatment for this disease. Anticonvulsant medicine may initially control seizures. Other supportive treatment includes proper nutrition and hydration and techniques to keep the airway open. Children may eventually need a feeding tube.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS121907952	HEXA	CC	normal
RS121907957	HEXA	CC	normal
RS121907958	HEXA	CC	normal
RS121907980	intergenic	CC	normal
RS387906311	intergenic	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Tay-Sachs Disease (TSD) uses 12 polymorphic loci on the *HEXA* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Tay-Sachs Disease (TSD).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Tay-Sachs Disease (TSD), or if you feel that you have a risk of developing Tay-Sachs Disease (TSD), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 12 polymorphic loci associated with the risk of Tay-Sachs Disease (TSD), such as rs121907952, rs121907957 and rs121907958.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Tay-Sachs Disease (TSD).

Treacher Collins Syndrome (TCS)

It is a genetic disorder that affects growth and development of the head. It prevents the skull, cheek and jawbones from developing properly, causing facial anomalies and hearing loss.

My Treacher Collins Syndrome (TCS)

Non-carrier

Knowledge



Time of onset

It is present at birth and can be diagnosed during pregnancy or after birth.



Prevalence

It is estimated to affect approximately 1 in 50,000 live births worldwide.



Typical symptoms

Typical symptoms include underdeveloped cheekbones, jaw, and chin, downward-slanting eyes, and malformed or absent ears.



Treatment

Treatment may include surgery to correct facial abnormalities, hearing aids or cochlear implants to improve hearing, and therapy to assist with speech and language.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS141156009	POLR1C	CC	normal

Test details

Applicable situation

Genetic disease screening test for Treacher Collins Syndrome (TCS) uses 1 polymorphic locus on the *POLR1C* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Treacher Collins Syndrome (TCS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Treacher Collins Syndrome (TCS), or if you feel that you have a risk of developing Treacher Collins Syndrome (TCS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Treacher Collins Syndrome (TCS), namely rs141156009.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Treacher Collins Syndrome (TCS).

Troyer Syndrome

It is a neurological disorder and one of the many types of hereditary spastic paraplegia. Signs and symptoms typically begin in early childhood and may include progressive muscle weakness and stiffness (spasticity) in the legs, muscle wasting in the hands and feet, paraplegia, leg contractures.

My Troyer Syndrome

Non-carrier

Knowledge



Time of onset

Symptoms usually appear in early infancy or childhood.



Prevalence

This disease has only been reported in a few families worldwide.



Typical symptoms

The most common symptoms include developmental delays, intellectual disability, speech difficulties, muscle weakness and stiffness, and abnormal gait.



Treatment

Physical therapy to improve muscle strength and flexibility, speech therapy to improve communication, and medication to control seizures.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS730882198	intergenic	DD	normal

Test details

Applicable situation

Genetic disease screening test for Troyer Syndrome uses 1 polymorphic locus on the *SPART* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Troyer Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Troyer Syndrome, or if you feel that you have a risk of developing Troyer Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Troyer Syndrome, namely rs730882198.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Troyer Syndrome.

Warburg Micro Syndrome (WARBM)

It is a rare genetic disorder that affects brain development and causes visual impairment, intellectual disability, and movement problems.

My Warburg Micro Syndrome (WARBM)

Non-carrier

Knowledge



Time of onset

It starts to appear soon after birth.



Prevalence

It is an extremely rare disease with only a few hundred cases reported worldwide.



Typical symptoms

Typical symptoms include developmental delays, intellectual disability, inability to properly control eye movements, small head size, and seizures.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms, such as physical therapy, occupational therapy, and special education programs that help improve quality of life.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS766629205	RAB3GAP1	CC	normal
RS797045905	RAB3GAP1	TT	normal

Test details

Applicable situation

Genetic disease screening test for Warburg Micro Syndrome (WARBM) uses 2 polymorphic loci on the *RAB3GAP1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Warburg Micro Syndrome (WARBM).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Warburg Micro Syndrome (WARBM), or if you feel that you have a risk of developing Warburg Micro Syndrome (WARBM), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 2 polymorphic loci associated with the risk of Warburg Micro Syndrome (WARBM), such as rs766629205 and rs797045905.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Warburg Micro Syndrome (WARBM).

Weill-Marchesani Syndrome (WMS)

It is a rare genetic disorder that primarily affects the normal growth and development of the connective tissues in the body. It is characterized by short stature, distinctive facial and skeletal abnormalities, and eye disorders that can lead to visual impairment or blindness.

My Weill-Marchesani Syndrome (WMS)

Non-carrier

Knowledge



Time of onset

It typically appears in early childhood.



Prevalence

The prevalence of this disease is currently unknown.



Typical symptoms

Typical symptoms include short stature, a broad or flat nose, a small jaw, a small head circumference, and distinctive facial features.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms. Eye disorders may be treated with corrective lenses, surgery, or medication to lower intraocular pressure. Joint abnormalities may be managed with physical therapy or surgery. In some cases, growth hormone therapy may be used to help promote growth and development.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS111401431	FBN1	GG	normal

RS113871094	FBN1	GG	normal
RS137854467	FBN1	GG	normal
RS137854480	FBN1	GG	normal
RS193922185	FBN1	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Weill-Marchesani Syndrome (WMS) uses 16 polymorphic loci on the *FBN1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Weill-Marchesani Syndrome (WMS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Weill-Marchesani Syndrome (WMS), or if you feel that you have a risk of developing Weill-Marchesani Syndrome (WMS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 16 polymorphic loci associated with the risk of Weill-Marchesani Syndrome (WMS), such as rs111401431, rs112645512 and rs113871094.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Weill-Marchesani Syndrome (WMS).

Werner Syndrome

It is a rare genetic disorder that causes premature aging. Individuals with this disease experience accelerated aging starting in their middle age, leading to various health complications.

My Werner Syndrome
Non-carrier

Knowledge



Time of onset

Symptoms usually begin to appear in the age of 30-40.



Prevalence

It is estimated to affect 1 in 200,000 to 1,000,000 individuals.



Typical symptoms

Typical symptoms include thinning and graying of hair, skin changes, short stature, cataracts, Type II diabetes, and an increased risk of cancer.



Treatment

Treatment may include medications to control diabetes and high blood pressure, surgical intervention for cataracts, and cancer screening and treatment.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS121908446	WRN	CC	normal
RS281865160	WRN	II	normal

RS1339616347	intergenic	GG	normal
RS775802030	WRN	AA	normal
RS878854131	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Werner Syndrome uses 5 polymorphic loci on the *WRN* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Werner Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Werner Syndrome, or if you feel that you have a risk of developing Werner Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 5 polymorphic loci associated with the risk of Werner Syndrome, such as rs121908446, rs1339616347 and rs281865160.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Werner Syndrome.

Wilson Disease

It is a rare genetic disorder that causes excessive accumulation of copper in the body, predominantly in the liver and brain. This can lead to various complications such as liver disease, neurological disorders, and psychiatric symptoms.

My Wilson Disease
Non-carrier

Knowledge



Time of onset

It typically appears in childhood or adolescence.



Prevalence

It is estimated to affect 1 in 30,000 individuals worldwide, but may be higher in certain populations.



Typical symptoms

Typical symptoms include fatigue, jaundice, motor difficulties, tremors, difficulty with speech and swallowing, depression, and anxiety.



Treatment

Treatment may include copper chelation therapies, such as penicillamine and trientine, which remove the excess copper from the body, and zinc acetate, which inhibits copper absorption from the intestinal tract. In some cases, liver transplantation may be necessary for those with severe liver damage.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS137853279	ATP7B	CC	normal

RS1555296472	intergenic	AA	normal
RS753236073	ATP7B	GG	normal
RS748924063	ATP7B	DD	normal
RS376910645	intergenic	CC	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Wilson Disease uses 94 polymorphic loci on the *ATP7B* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Wilson Disease.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Wilson Disease, or if you feel that you have a risk of developing Wilson Disease, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 94 polymorphic loci associated with the risk of Wilson Disease, such as rs1038582488, rs1057516227 and rs1057516228.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Wilson Disease.

Wiskott-Aldrich Syndrome (WAS)

It is a rare genetic immunodeficiency disorder that affects the immune system, blood clotting, and platelet function.

My Wiskott-Aldrich Syndrome (WAS)

Non-carrier

Knowledge

Time of onset

The onset usually occurs during the first year of life, but can sometimes appear later in childhood or even adulthood.

Prevalence

It is estimated to affect 1 in 250,000 to 1,000,000 male births.

Typical symptoms

Typical symptoms include recurrent infections, eczema or rash, nose bleeds, easy bruising, and bloody diarrhea.

Treatment

Treatment includes antibiotics, stem cell transplantation, and administration of immunoglobulin replacement therapy.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS132630268	WAS	GG	normal
RS132630273	intergenic	CC	normal

RS193922414	WAS	CC	normal
RS387906717	intergenic	TT	normal

Test details

Applicable situation

Genetic disease screening test for Wiskott-Aldrich Syndrome (WAS) uses 4 polymorphic loci on the *WAS* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Wiskott-Aldrich Syndrome (WAS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Wiskott-Aldrich Syndrome (WAS), or if you feel that you have a risk of developing Wiskott-Aldrich Syndrome (WAS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 4 polymorphic loci associated with the risk of Wiskott-Aldrich Syndrome (WAS), such as rs132630268, rs132630273 and rs193922414.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Wiskott-Aldrich Syndrome (WAS).

Wolfram Syndrome

It is a rare genetic disorder characterized by juvenile-onset diabetes mellitus, diabetes insipidus, optic nerve atrophy, hearing loss, and neurodegeneration.

My Wolfram Syndrome
Non-carrier

Knowledge



Time of onset

The onset of symptoms typically occurs in childhood or adolescence.



Prevalence

It is estimated to affect 1 in 500,000 to 770,000 individuals.



Typical symptoms

The most common symptoms include high blood sugar, excessive thirst and urination, progressive loss of vision, and sensorineural hearing loss.



Treatment

There is currently no cure. Treatment mainly involves improving symptoms. This may include diabetes management, vision and hearing aids, and supportive care for other symptoms.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS104893879	WFS1	GG	normal
RS104893881	WFS1	CC	normal
RS28937891	WFS1	GG	normal

RS387906930	WFS1	CC	normal
RS71524377	WFS1	GG	normal

For display consideration, only some genetic details are displayed.

Test details

Applicable situation

Genetic disease screening test for Wolfram Syndrome uses 10 polymorphic loci on the *WFS1* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Wolfram Syndrome.
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Wolfram Syndrome, or if you feel that you have a risk of developing Wolfram Syndrome, it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 10 polymorphic loci associated with the risk of Wolfram Syndrome, such as rs104893879, rs104893881 and rs28937891.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Wolfram Syndrome.

Yunis-Varon Syndrome (YVS)

It is a rare genetic disorder that affects the development of bones in the skull, causing malformation of the face and head. It is also characterized by abnormalities in the development of the fingers and toes, as well as intellectual disability.

My Yunis-Varon Syndrome (YVS)

Non-carrier

Knowledge



Time of onset

It typically appears at birth, with symptoms becoming apparent in infancy.



Prevalence

It is an extremely rare disease, with only a few dozen cases reported worldwide.



Typical symptoms

Typical symptoms include craniofacial abnormalities, finger and toe malformations, and intellectual disability.



Treatment

Treatment may include surgery to correct craniofacial and limb malformations. Early intervention and support for intellectual disability is also important.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS786200937	intergenic	II	normal

Test details

Applicable situation

Genetic disease screening test for Yunis-Varon Syndrome (YVS) uses 1 polymorphic locus on the *FIG4* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Yunis-Varon Syndrome (YVS).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Yunis-Varon Syndrome (YVS), or if you feel that you have a risk of developing Yunis-Varon Syndrome (YVS), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Yunis-Varon Syndrome (YVS), namely rs786200937.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Yunis-Varon Syndrome (YVS).

Alpha-N-Acetylgalactosaminidase Deficiency (α -NAGA)

It is a rare hereditary disease that affects the decomposition of glycosphingolipid (a kind of carbohydrate) and glycosuria proteins in the body.

My Alpha-N-Acetylgalactosaminidase Deficiency (α -NAGA)

Non-carrier

Knowledge

Time of onset

This disease can vary widely depending on the severity of the mutations in the NAGA gene. Some people are born with symptoms, while others may not show any symptoms until later in childhood or adulthood.

Prevalence

It is a very rare disorder, with only a handful of cases reported worldwide.

Typical symptoms

Some of the typical symptoms of this disease include developmental delays, intellectual disability, seizures, muscle weakness, skeletal abnormalities, liver and spleen enlargement, hearing loss, and vision problems. Some people may also have a cherry red spot in their retinas.

Treatment

There is currently no cure for this disease, so treatment is focused on managing the symptoms. This may include physical therapy, medication for seizures, and hearing and vision aids.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

Test details

Applicable situation

Genetic disease screening test for Alpha-N-Acetylgalactosaminidase Deficiency (α -NAGA) uses 1 polymorphic locus on the *NAGA* gene. The test results only indicate whether the user is carrying specific pathogenic gene or locus.

Notification

1. The test may not cover all the pathogenic genes or loci of Alpha-N-Acetylgalactosaminidase Deficiency (α -NAGA).
2. The test results are not applicable to clinical practice.
3. If you have clinical needs, please contact a qualified institution or clinician.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease are determined by a combination of genetic and environmental factors, and this test only indicates whether the user is carrying specific pathogenic gene or locus.
3. It is important to note that carrying the associated mutation does not represent the true state of disease. Even if there is no carrying of the relevant variant, the risk of having the disease cannot be ruled out.
4. If you have a family history of Alpha-N-Acetylgalactosaminidase Deficiency (α -NAGA), or if you feel that you have a risk of developing Alpha-N-Acetylgalactosaminidase Deficiency (α -NAGA), it is recommended that you consult a qualified health professional or clinician for help.

Test content

This test includes 1 polymorphic locus associated with the risk of Alpha-N-Acetylgalactosaminidase Deficiency (α -NAGA), namely rs779423223.

Population samples

The basis of the test is mainly from the research of Caucasian.

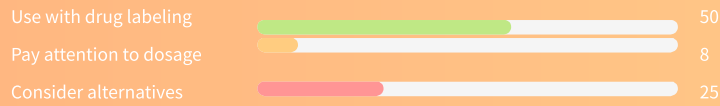
Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all pathogenic genes or loci of Alpha-N-Acetylgalactosaminidase Deficiency (α -NAGA).



Drug Guideline

83 Reports



Cardiovascular

Digoxin	Consider Dose Reduction
Simvastatin	Risk of Adverse Effect
Atorvastatin	Risk of Adverse Effect
Lovastatin	Use With Drug Labeling
Pravastatin	Risk of Adverse Effect
Rosuvastatin	Risk of Adverse Effect
Furosemide	Risk of Insufficient Efficacy
Spironolactone	Risk of Insufficient Efficacy



Hypertension

Hydrochlorothiazide	Risk of Insufficient Efficacy



Diabetes

Metformin	Risk of Insufficient Efficacy
Glibenclamide	Use With Drug Labeling
Glipizide	Use With Drug Labeling
Gliquidone	Use With Drug Labeling

Glimepiride	Use With Drug Labeling
Gliclazide	Use With Drug Labeling
Rosiglitazone	Use With Drug Labeling

Anti-infection

Amikacin	Use With Drug Labeling
Kanamycin	Use With Drug Labeling
Streptomycin	Use With Drug Labeling
Gentamicin	Use With Drug Labeling
Tobramycin	Use With Drug Labeling
Neomycin	Use With Drug Labeling
Atazanavir	Use With Drug Labeling
Daclatasvir	Use With Drug Labeling
Ribavirin	Use With Drug Labeling
Lopinavir	Use With Drug Labeling
Tenofovir	Risk of Insufficient Efficacy
Simeprevir	Use With Drug Labeling
Pyrazinamide	Use With Drug Labeling
Rifampin	Use With Drug Labeling
Ethambutol	Risk of Adverse Effect

Musculoskeletal

Adalimumab	Use With Drug Labeling
Etanercept	Use With Drug Labeling

Diclofenac	Use With Drug Labeling

Respiratory System

Budesonide	Risk of Insufficient Efficacy

Salbutamol	Use With Drug Labeling

Salmeterol	Use With Drug Labeling

Nervous System

Lamotrigine	Risk of Insufficient Efficacy

Fluoxetine	Risk of Insufficient Efficacy

Amisulpride	Use With Drug Labeling

Olanzapine	Use With Drug Labeling

Quetiapine	Use With Drug Labeling

Clozapine	Use With Drug Labeling

Paliperidone	Use With Drug Labeling

Ziprasidone	Use With Drug Labeling

Buprenorphine	Use With Drug Labeling

Fentanyl	Use With Drug Labeling

Morphine	Consider Dose Increase

Meperidine	Use With Drug Labeling

Pentazocine	Use With Drug Labeling

Oxycodone	Consider Dose Increase

Remifentanyl	Consider Dose Increase

Sufentanil	Consider Dose Increase

Methadone	Risk of Insufficient Efficacy

 Anti-tumor

Gemcitabine	Use With Drug Labeling

Methotrexate	Risk of Adverse Effect

Carboplatin	Use With Drug Labeling

Peginterferon Alfa-2A	Use With Drug Labeling

Peginterferon Alfa-2B	Use With Drug Labeling

Interferon Alfa-2B, Recombinant	Consider Dose Reduction

Trastuzumab	Use With Drug Labeling

Cetuximab	Risk of Insufficient Efficacy

Infliximab	Use With Drug Labeling

Sunitinib	Use With Drug Labeling

Anastrozole	Risk of Insufficient Efficacy

Exemestane	Risk of Insufficient Efficacy

Cyclophosphamide	Risk of Insufficient Efficacy

Epirubicin	Risk of Insufficient Efficacy

Doxorubicin	Use With Drug Labeling

Daunorubicin	Use With Drug Labeling

Docetaxel	Risk of Insufficient Efficacy

Etoposide	Use With Drug Labeling

Vincristine	Use With Drug Labeling

Paclitaxel	Risk of Insufficient Efficacy

Asparaginase

Use With Drug Labeling

 Immune System

Cyclosporine

Consider Dose Increase

Sirolimus

Consider Dose Increase

Triamcinolone

Risk of Insufficient Efficacy

 Sundry

Latanoprost

Risk of Insufficient Efficacy

Prednisolone

Use With Drug Labeling

Naloxone

Risk of Insufficient Efficacy

Leucovorin

Use With Drug Labeling

Ethanol

Risk of Adverse Effect

Hydrochlorothiazide

Hydrochlorothiazide (HCTZ or HCT) is a diuretic medication often used to treat high blood pressure and swelling due to fluid build up. Other uses include treating diabetes insipidus and renal tubular acidosis and to decrease the risk of kidney stones in those with a high calcium level in the urine. In addition, it is often used as the first-line drug for the treatment of hypertension.

My Hydrochlorothiazide

Risk of Insufficient Efficacy

EFFICACY : Patients with rs16960228-GG genotype, compared to AA or AG genotype, may have a decreased reduction of diastolic blood pressure; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Suitable for edema diseases: it can excrete excessive sodium and water in the body, reduce the volume of extracellular fluid, and eliminate edema. Common ones include congestive heart failure, liver cirrhosis, ascites, nephrotic syndrome, acute and chronic nephritis edema, early stage

of chronic renal failure, sodium and water retention caused by adrenal cortex hormone and estrogen therapy.

2. Suitable for hypertension: it can be used alone or in combination with other antihypertensive drugs, mainly used to treat essential hypertension.
3. It is suitable for central or renal diabetes insipidus.
4. Suitable for nephrolithiasis: it is mainly used to prevent calculus formed by calcium salt.



Adverse reactions

Most of the adverse reactions are related to the dose and the course of treatment.

1. Side effects caused by water and electrolyte disturbances are more common. Hypokalemia is more likely to occur and is related to the potassium excretion effect of thiazide diuretics. Long-term potassium deficiency can damage the renal tubules. Severe potassium loss can cause vacuolar changes in the renal tubular epithelium, and cause severe tachyarrhythmias and other ectopic heart rates. Low-chlorine alkalosis or low-chlorine, low-potassium alkalosis, thiazides, especially hydrochlorothiazide, often significantly increase the excretion of chloride. In addition, hyponatremia is not uncommon, leading to central nervous system symptoms and aggravating kidney damage. Dehydration causes a decrease in blood volume and renal blood flow, which also causes a decrease in glomerular filter rate. The common clinical reactions of the above-mentioned water and electrolyte disorders include dry mouth, polydipsia, muscle cramps, nausea, vomiting, extreme fatigue and weakness, etc.
2. Hyperglycemia. This drug can reduce glucose tolerance and increase blood sugar, which may be related to the inhibition of insulin release.
3. Hyperuricemia. Interference with renal tubule excretion of uric acid, a few can induce gout attacks. Since there is usually no joint pain, hyperuricemia is easily overlooked.
4. Allergic reactions, such as skin rash, urticaria, etc., are relatively rare.
5. Leukopenia or deficiency, thrombocytopenic purpura, etc., are also rare.
6. Others, such as cholecystitis, pancreatitis, hyposexual function, light sensitivity, color vision disorders, etc., but are relatively rare.



Contraindications

1. Disabled for patients with epilepsy.
2. People with severe renal dysfunction are prohibited.
3. People who are allergic to this product should not use it.
4. It is forbidden for pregnant women and lactating women.



Pregnant and lactating women

This medicine can pass the placental barrier. No preventive effect on hypertension syndrome. Therefore, pregnant women should be cautious when using it. Women who are breastfeeding should not take it.

Children

It should be used with caution in infants with jaundice, because this class of drugs can increase blood bilirubin.

Elderly

The elderly are more prone to hypotension, electrolyte imbalance and renal function damage when using this class of drugs.

Metabolism

It is metabolized by combining with glucuronic acid and oxidized in the liver. The main metabolite is glucuronic acid-binding product (about 6%), and the metabolite is excreted by the biliary tract and kidneys.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4149601	NEDD4L	GG	strong response of diuretics
RS7297610	intergenic	CC	slightly strong response of hydrochlorothiazide
RS16960228	PRKCA	GG	slightly low diastolic pressure reduction by hydrochlorothiazide

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The hydrochlorothiazide drug response test item uses 3 polymorphic loci of *PRKCA*, *NEDD4L*, and *LYZ* genes. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the

item may be affected.

4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Budesonide

Budesonide is a glucocorticoid with highly effective local anti-inflammatory effects. It can enhance the stability of endothelial cells, smooth muscle cells and lysosomal membranes, inhibit immune response and reduce antibody synthesis, thereby reducing the release and activity of histamine and other allergic active mediators, and can reduce the enzymes that are stimulated when antigen and antibody are combined. It can reduce the enzymes that are stimulated when antigen and antibody are combined, inhibit the synthesis and release of bronchoconstrictive substances, and reduce the contraction response of smooth muscles.

My Budesonide

Risk of Insufficient Efficacy

EFFICACY : Patients with rs1876828-CC genotype, compared to TT genotype, may have a decreased drug response; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

For patients with non-glucocorticoid-dependent or dependent bronchial asthma and asthmatic chronic bronchitis.

Adverse reactions

1. Mild throat irritation, coughing, hoarseness.
2. Candida infection in the oropharynx.
3. Rapid or delayed allergic reactions, including skin rash, contact dermatitis, urticaria, angioedema and bronchospasm.
4. Mental symptoms, such as nervousness, restlessness, depression, and behavioral disorders.

Contraindications

People who are allergic to this product should not use it.

Pregnant and lactating women

It has not been found that the use of inhaled budesonide during pregnancy will have adverse effects on embryos and newborns. It is not known whether budesonide can enter breast milk, but it should be used with caution. Compared with oral glucocorticoids, the systemic effect of inhaled glucocorticoids is lower when the equivalent dose of anti-asthma is reached.

Children

Children under 2 years old should be used with caution or not.

Elderly

There is no difference in safety indicators between elderly patients and younger patients.

Metabolism

Budesonide is mainly metabolized by the liver in the first pass (about 90%), and the glucocorticoid activity of the metabolites is low. The main metabolites 6b-hydroxybudesonide and 16a-hydroxyprednisolone have less than 1% of the activity of budesonide. Metabolites of budesonide are excreted through the kidneys in its original or combined form. Prototype budesonide could not be detected in the urine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1876828	CRHR1	CC	slightly weak response of budesonide

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The budesonide drug response test item uses 1 polymorphic locus of *CRHR1* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

 Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Salbutamol

Salbutamol is a medication that opens up the medium and large airways in the lungs. It is used to treat asthma, including asthma attacks, exercise-induced bronchoconstriction, and chronic obstructive pulmonary disease (COPD). It may also be used to treat high blood potassium levels.

My Salbutamol

Use With Drug Labeling

EFFICACY : Patients who are treated with salbutamol have an increased of therapeutic response; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. For the treatment of bronchial asthma or wheezing bronchitis and other respiratory diseases accompanied by bronchospasm.

Adverse reactions

1. The more common adverse reactions are: tremor, nausea, palpitations, headache, and insomnia.

2. Less common adverse reactions include dizziness, dazzle, and dry mouth and pharynx.

 Contraindications

1. People who are allergic to this product or other adrenergic receptor agonists are prohibited.

 Pregnant and lactating women

β2-agonists relax uterine smooth muscle. Pregnant and lactating women should use it with caution, and can be used under the guidance of an obstetrician when they must be used.

 Children

Follow the doctor's advice.

 Elderly

It should be used with caution, starting with a small dose.

 Metabolism

Most of the salbutamol is metabolized in the intestinal wall and liver, and less than 20% of the original drug enters the blood circulation. After oral or inhalation of salbutamol, 80% is excreted in urine within 3 days.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1042713	ADRB2	GG	normal drug response
RS6988229	COL22A1	CT	normal drug response
RS7793837	CRHR2	AA	normal drug response

How to use the test results

 Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The salbutamol drug response test item uses 3 polymorphic loci of *ADRB2*, *COL22A1*, and *CRHR2* genes. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.

-
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
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 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Salmeterol

Salmeterol is a long-acting β_2 adrenergic receptor agonist (LABA) used in the maintenance and prevention of asthma symptoms and maintenance of chronic obstructive pulmonary disease (COPD) symptoms. Symptoms of bronchospasm include shortness of breath, wheezing, coughing and chest tightness. It is also used to prevent breathing difficulties during exercise.

My Salmeterol

Use With Drug Labeling

EFFICACY : Pediatric patients with rs1042713-GG genotype and asthma, compared to AG or GG genotype, may have an increased drug response.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is used for regular treatment of reversible obstructive airway diseases, including asthma in adults and children.

Adverse reactions

1. The pharmacological side effects of β 2-agonists such as tremor, subjective palpitations and headaches have been reported, but they are all temporary and alleviated with conventional treatment.
2. Some patients may have arrhythmia (including atrial fibrillation, supraventricular tachycardia, and extrasystole), usually sensitive patients.
3. There have been reports of joint pain and allergic reactions including skin rash, edema and angioedema.
4. There have been reports of oropharyngeal irritation.
5. Rare reports of muscle cramps.

 Contraindications

1. People who have a history of allergies to this product should not be used.

 Pregnant women and lactating women

During pregnancy and lactating, only consider medication when the expected benefits to the mother outweigh any possible harm to the fetus or child. There is not enough experience in humans using salmeterol and fluticasone propionate during pregnancy and lactation.

 Children

Developmental delays in children and adolescents may occur. It is recommended that children who have been treated with inhaled corticosteroids for a long time have their height checked regularly.

 Elderly

The elderly or patients with renal impairment do not need to adjust the dose.

 Metabolism

This product is extensively metabolized by hydroxylation in the body, most of which are eliminated within 72 hours. 23% and 57% were excreted in urine and feces 168h after medication.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1042713	ADRB2	GG	normal drug response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The salmeterol drug response test item uses 1 polymorphic locus of *ADRB2* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

 Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Adalimumab

Adalimumab is a humanized monoclonal antibody against human tumor necrosis factor (TNF). TNF plays an important role in pathological inflammation and joint destruction. Adalimumab can specifically bind to TNF- α , thereby eliminating its biological function.

My Adalimumab

Use With Drug Labeling

EFFICACY : Patients with rs1800629-GG genotype, compared to AA genotype, may have an increased drug response.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Applicable to rheumatoid arthritis (RA) : Relieve signs and symptoms in adults with moderate to severe active RA, inducing major clinical response, inhibiting that progression of structural damage, and improving body function.
2. Applicable to juvenile idiopathic arthritis (JIA) : Relief of signs and symptoms of moderately to severely active polyarticular JIA in children 4 years and older.

3. Applicable to psoriatic arthritis (PsA): Reduce signs and symptoms, inhibit the progression of structural damage, and improve body function in adult patients with active PsA.
4. Applicable to ankylosing spondylitis (AS): Alleviate signs and symptoms in adult patients with active AS.
5. Applicable to Crohn's disease (CD): Reduce signs and symptoms and induce and maintain clinical remission in adult patients with moderately to severely active Crohn's disease that does not respond adequately to conventional treatment.
6. Applicable to plaque psoriasis (Ps) : Treatment of adults with moderate to severe chronic plaque psoriasis, systemic therapy or phototherapy, and other medically inappropriate alternatives for systemic therapy.



Adverse reactions

1. Severe infection: Increased risk of severe infection leading to hospitalization or death, including tuberculosis, bacterial septicaemia, invasive fungal infections, and infections due to other opportunistic pathogens.
2. Malignant diseases: Lymphoma and other malignant diseases have been reported in children and adolescents, some of which are fatal. Post-marketing treatment for adolescents and young adults has occurred cases of liver and spleen T-cell lymphoma (a rare type of T-cell lymphoma), skin cancer (except melanoma, including basal cell carcinoma and squamous cell carcinoma), benign tumors, etc.
3. Other common adverse reactions: Respiratory tract infection is the most common, and systemic and local infection is also very common; Leukocyte abnormalities, anemia, elevated blood lipid, mood changes, anxiety, insomnia, headache, migraine, nerve root compression, visual impairment, eye swelling, dizziness, tachycardia, asthma, dyspnea, gastrointestinal abnormalities, elevated liver enzymes, rash, psoriasis deterioration, urticaria, dermatitis, skeletal muscle pain, muscle spasm, renal function injury, chest pain, edema, fever, healing disorders and other laboratory indexes were abnormal.



Contraindications

1. People who are allergic to this product.
2. Active associations or other serious infectious disorders, such as sepsis and opportunistic infections.
3. Patients with moderate to severe heart failure (NYHA Division III/IV).



Pregnant and lactating women

Pregnant women can use it until the end of the second trimester of pregnancy (i.e. 30 weekends). They have insufficient experience in medication during lactation.



Children

Adalimumab is used to treat moderate to severe multiarticular adolescent idiopathic arthritis in children aged 2 years and older, and moderate to severe Crohn's disease in children aged 6 years and older.



Elderly

Dose adjustment is not necessary, but it should be used with caution. Special attention should be paid to the risk of infection in elderly treatment.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1800629	TNF, LTA	GG	normal drug response

How to use the test results



Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.



Test content

Different genetic test versions and data sources may have different coverage of the above variants.



Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.



Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only.

and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The adalimumab drug response test item uses a polymorphic locus of the *TNF* gene. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Etanercept

Etanercept is an anti-rheumatic drug that improves the condition and is used to treat rheumatoid arthritis and ankylosing spondylitis.

My Etanercept

Use With Drug Labeling

EFFICACY : Patients with rs1800629-GG genotype, compared to AA genotype, may have an increased drug response.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Adult patients with moderate to severe active rheumatoid arthritis can be treated with etanercept combined with methotrexate when anti-rheumatic drugs including methotrexate (if not contraindicated) are ineffective.
2. Adult patients with severe active ankylosing spondylitis can be treated with etanercept when conventional treatment fails.

Adverse reactions

1. Infection: The most common is lower respiratory tract infection, other infections include rhinitis, pharyngitis, pyelonephritis, bronchitis, septic arthritis, abdominal abscess, cellulitis, osteomyelitis, wound infection, pneumonia, foot abscess, leg ulcer, diarrhea, Sinusitis, sepsis. And it may increase the mortality rate of patients with original infections.
2. Cardiovascular system: heart failure, myocardial infarction, myocardial ischemia, hypertension, hypotension, deep vein thrombosis, thrombophlebitis.
3. Digestive system: cholecystitis, pancreatitis, gastrointestinal bleeding, abdominal pain, indigestion, vomiting, loss of appetite, dry mouth, oral ulcers.
4. Musculoskeletal system: bursitis, polymyositis, arthralgia, lupus-like syndrome.
5. Nervous system: headache, dizziness, cerebral ischemia, depression, multiple sclerosis, angioneurotic edema, hemiplegia, stroke, epilepsy, solitary demyelinating disease, transverse myelitis, optic neuritis.
6. Respiratory system: cough, dyspnea, pulmonary embolism, interstitial pneumonia.
7. Urogenital system: membranous glomerulonephritis.
8. Blood system: Lymph node enlargement, anemia, aplastic anemia, leukopenia, granulocytopenia, pancytopenia, thrombocytopenia.
9. Skin lesions: vasculitis, subcutaneous nodules, rubella, rash, hair loss.



Contraindications

1. Those who are allergic to the active ingredients in this product or any other ingredients.
2. Patients with sepsis or patients at risk of sepsis.
3. Patients with severe active infections, including chronic or local infections.



Pregnant and lactating women

Etanercept is not recommended for pregnant women, and women of childbearing age are advised not to become pregnant during etanercept treatment. Lactating women need to consider whether to stop breastfeeding or stop using etanercept.



Children

Not applicable.



Elderly

There is no need to adjust the dose according to the age of the patient.



Metabolism

The absorption is slower, reaching the peak plasma concentration in 69 ± 34 hours on average, and it is cleared by the protein degradation pathway in the body.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1800629	TNF, LTA	GG	normal drug response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug

response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The etanercept drug response test item uses 1 polymorphic locus of *TNF* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Diclofenac

Diclofenac is a non-steroidal anti-inflammatory drug. It has anti-inflammatory, analgesic and antipyretic effects.

My Diclofenac

Use With Drug Labeling

EFFICACY : Patients with rs1057910-AA genotype, compared to AC or CC genotype, may have a decreased, but not absent, risk of gastrointestinal bleeding; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It can be used to relieve mild to moderate pain in muscles, soft tissues and joints, such as relief of muscle and soft tissue sprains, strains, contusions, strains, pain caused by lower back injuries and joint pain, etc.
2. It can also be used for symptomatic treatment of osteoarthritis.

Adverse reactions

1. Occasionally local adverse reactions: allergic or non-allergic dermatitis such as papules, skin redness, edema, itching, small blisters, large blisters or scales, etc.
2. Systemic adverse reactions caused by topical use of this product are rare. If it is used for a large area of skin for a long time, it may appear: general rash, allergic reactions (such as asthma attacks, angioneurotic edema, photosensitized reaction, etc.) . If this happens, you should consult a physician.

Contraindications

1. People who are allergic to other non-steroidal anti-inflammatory drugs are prohibited.

Pregnant and lactating women

Pregnant women, lactating women, please consult a physician or pharmacist before use.

Children

Consult a physician for the dosage for children under 12 years old. Children must be used under adult supervision.

Elderly

It should be used with caution in people with a history of liver and kidney damage or ulcers, especially the elderly. Routine follow-up inspections of liver and kidney function should be performed during medication.

Metabolism

Diclofenac oral solution, rectal suppository, or intramuscular injection of diclofenac can be quickly absorbed. When using enteric-coated tablets, especially when taken with food, the absorption tends to be slow. Although the oral absorption of diclofenac is rapid and complete, the peak plasma concentration can be reached within 2 hours, but the first pass metabolism is obvious, and only 50% of the drug enters the systemic circulation. Diclofenac is also absorbed through the skin. At therapeutic concentrations, the protein binding rate is higher than 99%. It can penetrate into the synovial fluid and can be retained until the blood concentration drops. It can be distributed into the milk, but the amount is very low, so that it will not bring harm to the breast-feeding baby. The final plasma $t_{1/2}$ is about 1 to 2 hours. The main metabolite is hydroxydiclofenac, and the metabolites glucuronic acid and sulfuric acid are combined with urine and bile to be excreted.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS1057910

CYP2C9

AA

likely to have lower risk of gastrointestinal bleeding

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The diclofenac drug response test item uses 1 polymorphic locus of *CYP2C9* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Atazanavir

Atazanavir is a kind of azapeptide HIV-1 protease inhibitor that can selectively inhibit the specific processing of viral Gag and Gag-Pol polyproteins in HIV-1 infected cells, thereby blocking the formation of mature viruses.

My Atazanavir

Use With Drug Labeling

ADVERSE EFFECT : Possibility of jaundice is very low.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is used in combination with other antiretroviral drugs to treat HIV-1 infection.

Adverse reactions

1. Common adverse reactions are nausea, vomiting, diarrhea, stomach pain, rash, fever, cough, insomnia, depression, numbness of hands and feet, etc.
2. Yellowing of the skin and eyes, dizziness, diabetes and increased blood sugar may occur. It may increase the bleeding tendency for patients with blood diseases, and the electrocardiogram may

show a prolonged P-R interval.

3. In severe cases, metabolic acidosis can occur, and it generally occurs in women or obese people.
4. Lipid metabolism disorders rarely occur, and the incidence of jaundice is dose-related.

Contraindications

1. People who are allergic to this product should not be used.
2. Disabled for patients with severe liver damage.

Pregnant and lactating women

Only when the benefits to the fetus outweigh the disadvantages, atazanavir can be considered for pregnant women. Because there is the possibility of HIV transmission to nursing infants and serious adverse drug reactions, mothers who are receiving atazanavir treatment should be instructed not to breastfeed.

Children

The optimal dosage regimen of atazanavir in the treatment of pediatric patients has not been established. Due to the risk of kernicterus, atazanavir cannot be used in infants under 3 months.

Elderly

Because elderly patients with decreased liver, kidney, and heart function, as well as other diseases or the application of other drugs, are more likely to be cautious when applying atazanavir.

Metabolism

Atazanavir is mainly metabolized by liver microsomal cytochrome P450CYP3A isoenzymes and eliminated by bile.

My Haplotype Details

UGT1A1

[*1/*1](#)

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS887829	UGT1A10	CC	extremely low risk of jaundice

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The atazanavir drug response test item uses the rs887829 polymorphic locus and the haplotype information of the *UGT1A1* gene. The test results indicate the genotype results of the user's detected

variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

 Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Daclatasvir

Dalatavir can inhibit viral RNA replication and viral particle assembly. It is an NS5A inhibitor (a basic component of the HCV replication complex) and can be used to treat hepatitis C.

My Daclatasvir

Use With Drug Labeling

EFFICACY : Patients with rs12979860-CC genotype, compared to CT or TT genotype, may have an increased drug response; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It can be combined with other drugs to treat chronic hepatitis C virus infection in adults.

Adverse reactions

When dalatavir is used in combination with other drugs, common adverse reactions include headache, fatigue, nausea, itching, anemia, flu-like illness, insomnia, neutropenia, weakness, rash, loss of appetite, dry skin, hair loss, and fever.

Contraindications

1. For contraindications when this product is used in combination with other drugs, please refer to the instructions of the corresponding drugs.
2. This product is forbidden to patients who have been allergic to this product or any ingredient in the product in the past.
3. This product is forbidden to be used in combination with CYP3A4 potent inducers, because it may lead to a decrease in the exposure dose and curative effect of dalatavir hydrochloride tablets.

Pregnant and lactating women

Women during pregnancy or women of childbearing age who have not used contraception should not take dalatavir. Effective contraception should continue to be used within 5 weeks after completion of dalatavir treatment. Breastfeeding is not recommended for mothers who are using dalatavir hydrochloride tablets.

Children

The safety and effectiveness of dalatavir hydrochloride tablets have not been established in pediatric patients.

Elderly

No overall differences in safety or effectiveness were observed between elderly and young patients.

Metabolism

In vitro studies have shown that dalatavir is a CYP3A substrate, and CYP3A4 is the main CYP subtype involved in metabolism. It is mainly excreted in urine in the form of prototype drugs.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS12979860	IFNL4	CC	better drug response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical

medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The dalatavir drug response test item uses 1 polymorphic locus of *IFNL4* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

-
1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
 2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
 3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Ribavirin

Ribavirin is an anti-retroviral drug.

My Ribavirin

Use With Drug Labeling

EFFICACY : Patients have normal treatment response; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is suitable for viral pneumonia and bronchitis caused by respiratory syncytial virus, and skin herpes virus infection.

Adverse reactions

Common adverse reactions include anemia, fatigue, etc., which disappear after stopping the drug. Less common adverse reactions include fatigue, headache, insomnia, loss of appetite, nausea, vomiting, mild diarrhea, constipation, etc., and can cause a decrease in red blood cells, white blood cells and hemoglobin.



Contraindications

Those who are allergic to this product.



Pregnant and lactating women

It is contraindicated in pregnant women and women who may become pregnant. A small amount of the drug is excreted by breast milk and is toxic to both mother and child. Therefore, lactating women need to suspend breastfeeding during the medication period, and the breast milk should also be discarded.



Children

Very few patients can see nausea and diarrhea when used by children.



Elderly

It is not recommended for the elderly.



Metabolism

Metabolized in the liver.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS12979860	IFNL4	CC	normal drug response

How to use the test results



Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.



Test content

Different genetic test versions and data sources may have different coverage of the above variants.



Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The ribavirin drug response test item uses 1 polymorphic locus of *IFNL4* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living

environment and lifestyle factors.

5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Lopinavir

Lopinavir is an antiretroviral of the protease inhibitor class. It is used against HIV infections as a fixed-dose combination with another protease inhibitor, ritonavir (lopinavir/ritonavir).

My Lopinavir

Use With Drug Labeling

EFFICACY : Patients with rs4149056-CT genotype and HIV may have a decreased plasma levels of lopinavir as compared to CC genotype, or an increased plasma levels of lopinavir as compared to TT genotype; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for use in combination with other antiretroviral drugs to treat HIV infection.

Adverse reactions

- The most frequent adverse reaction related to the treatment of this product is mild to moderate diarrhea. The following is a list of adverse reactions according to body system organs, and their

frequency is graded according to the following methods: extremely common; common; uncommon.

- Infections and infectious diseases (uncommon: otitis media, bronchitis, sinusitis, furunculosis, bacterial infection, viral infection, pharyngitis, flu, gastroenteritis, sialadenitis)
- Tumors (including benign, malignant and unspecified) (uncommon: skin benign tumors, cysts)
- Blood and lymphatic system abnormalities (uncommon: anemia, leukopenia, lymphadenopathy)
- Immune system abnormalities (uncommon: allergic reactions)
- Endocrine abnormalities (uncommon: male hyposexual function, Cushing's syndrome, hypothyroidism)
- Nutritional and metabolic disorders (uncommon: vitamin deficiency, dehydration, increased appetite, lactic acidemia, obesity, loss of appetite, diabetes, weight gain)
- Mental disorders (uncommon: abnormal dreams, excitement, anxiety, confusion, depression, emotional instability, decreased libido, nervousness, abnormal thinking, apathy)
- Nervous system abnormalities (common, uncommon: headache, insomnia, dizziness, forgetfulness, ataxia, encephalopathy, facial paralysis, hypertonia, neuropathy, paresthesia, peripheral neuritis, lethargy, tremor, loss of taste, abnormal taste, migraine, Dyskinesia, cerebral infarction, spasm)
- Eye abnormalities (uncommon: visual abnormalities, eye discomfort)
- Ear and cochlea abnormalities (uncommon: tinnitus, dizziness)
- Heart abnormalities (uncommon: palpitations, atrial fibrillation)
- Vascular system abnormalities (uncommon: hypertension, thrombophlebitis, vasculitis, varicose veins, deep thrombophlebitis, vascular abnormalities, orthostatic hypotension)
- Breathing, thoracic and mediastinal abnormalities (uncommon: dyspnea, rhinitis, asthma, pulmonary edema)
- Gastrointestinal disorders (very common, common, uncommon: diarrhea, nausea, vomiting, abdominal pain, abnormal stool, indigestion, flatulence, abdominal enlargement, constipation, dry mouth, dysphagia, enterocolitis, belching, enteritis, Esophagitis, fecal incontinence, gastritis, hemorrhagic colitis, oral ulcers, pancreatitis, stomatitis, ulcerative stomatitis, periodontitis)
- Hepatobiliary abnormalities (uncommon: cholecystitis, hepatitis, cholangitis, jaundice)
- Skin and appendages (common, uncommon: skin rash, lipodystrophy, alopecia, dry skin, eczema, exfoliative dermatitis maculopapular rash, abnormal nails, itching, seborrheic dermatitis, skin discoloration, skin ulcers, facial edema, acne, sweating)
- Skeletal muscle and connective tissue abnormalities (uncommon: arthralgia, arthropathy, myalgia, back pain and osteonecrosis)
- Kidney and urinary system abnormalities (uncommon: kidney stones, urinary abnormalities, nephritis)
- Reproductive system and breast abnormalities (uncommon: abnormal ejaculation, breast enlargement, and feminization of male breasts)
- Systemic abnormalities and medication site conditions (common and uncommon: fatigue, chest pain, chest pain under the breastbone, chills, fever, malaise, pain, peripheral edema, drug interactions, edema, obesity)



Contraindications

1. This product is contraindicated in patients who are known to be allergic to lopinavir and ritonavir.

2. This product should not be used concurrently with drugs that mainly rely on CYP3A clearance and whose blood concentration increases cause serious and/or fatal adverse events. These drugs are listed in the table below:

- Drugs that are forbidden to be used in combination with this product:
 - Antihistamines: astemizole, terfenadine
 - Ergot derivatives: dihydroergotamine, ergonovine, ergotamine, methylergonovine
 - Gastrointestinal motility drug: cisapride
 - Neuroleptic drugs: pimozide
 - Benzodiazepines: midazolam, triazolam

 Pregnant and lactating women

The safety data of pregnant women taking this product has not been established because of the lack of adequate and well-controlled studies in pregnant women. This product can be used during pregnancy only when the potential benefits outweigh the potential risks to the fetus. It is recommended that women who are infected with HIV do not breastfeed their babies to avoid the spread of HIV after birth. Because breastfeeding may spread HIV and may cause serious adverse reactions, mothers who are taking this product should not breastfeed their babies. Whether lopinavir can be secreted through breast milk is unclear.

 Children

The safety and pharmacokinetic characteristics of this product for pediatric patients under 6 months have not been determined. In a clinical trial of HIV-infected children between 6 months and 12 years of age, the nature of the adverse events observed was similar to that observed in adult patients. Research on the evaluation of this product in pediatric patients is ongoing.

 Elderly

The lopinavir/ritonavir clinical trial did not include a sufficient number of subjects 65 years and older, so it is not yet certain whether their response will be different from younger subjects. In short, this product should be used with caution in elderly patients, because clinical monitoring found that the proportion of elderly patients with decreased liver, kidney, and heart function, the proportion of other diseases, and the proportion of receiving other drug treatments are all higher.

 Metabolism

Mainly metabolized and eliminated by the liver.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS4149056

SLCO1B1

CT

likely higher serum concentration of lopinavir

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The lopinavir drug response test item uses a polymorphic locus on the *SLCO1B1* gene. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variant.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Tenofovir

Tenofovir is a new type of nucleotide reverse transcriptase inhibitor. It can effectively fight against a variety of viruses and is used to treat viral infectious diseases.

My Tenofovir

Risk of Insufficient Efficacy

EFFICACY : Patients with rs1751034-TT genotype, compared to CT or CC genotype, may have increased tenofovir renal clearance and an decreased drug response.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for the combination of other antiretroviral drugs to treat HIV-1 infection.
2. It is suitable for the treatment of chronic hepatitis B.

Adverse reactions

1. Lactic acidosis/poisoned hepatomegaly with steatosis.

- 2. Hepatitis B worsened after treatment was discontinued.
- 3. New or worsening kidney damage.
- 4. Decreased bone mineral density.
- 5. Immune Reconstitution Syndrome.

 Contraindications

- 1. It is contraindicated in patients who have previously been allergic to tenofovir.

 Pregnant women and lactating women

Reproductive toxicity is classified as B, pregnant women should use it with caution. It is not clear whether this drug remains in human milk, and it is recommended that all women with HIV should avoid lactating.

 Children

The safety and efficacy in patients younger than 18 years of age have not been established.

 Elderly

Elderly patients should be cautious in choosing the dose, remembering that their liver, kidney, and heart function decline, and the chances of complicated diseases or being treated with other drugs increase.

 Metabolism

Tenofovir is almost not absorbed through the gastrointestinal tract, so it undergoes esterification and salt formation to become tenofovir disoproxil fumaric acid. Tenofovir dipivoxil is water-soluble and can be rapidly absorbed and degraded into the active substance tenofovir, which is then converted into the active metabolite tenofovir bisphosphate. Tenofovir reached the blood drug peak within 1 ~ 2 hours after administration. The bioavailability of this product can be increased by about 40% when taken with food. The intracellular half-life of tenofovir bisphosphate is approximately 10 hours, making it suitable for administration once a day. Because this product and tenofovir are not metabolized by CYP450 enzyme system. Therefore, the possibility of interaction with other drugs caused by this enzyme is very small. This product is mainly excreted through glomerular filtration and active tubule transport system, and about 70% to 80% is excreted in urine in its original form.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1751034	ABCC4	TT	lower effective concentration of the drug

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The tenofovir drug response test item uses a polymorphic locus of *ABCC4* gene. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

 Notification

1. Limited by the current scientific research, the genetic report may not cover all genes or loci related to the test item.
2. Limited by the technology of genetic testing, the genetic report may not cover all known genes or loci related to the test item.
3. Limited by the technology of genetic testing, the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and Asian, other populations are for reference only.

Simeprevir

Simeprevir is a HCV non-structural protein NS3/4A protease inhibitor, suitable for the treatment of adult hepatitis C virus (HCV) infection in combination with other antiviral drugs.

My Simeprevir

Use With Drug Labeling

EFFICACY : Patients with rs12979860-CC genotype, compared to CT or TT genotype, may have an increased drug response; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for the treatment of adult hepatitis C virus (HCV) infection in combination with other antiviral drugs.

Adverse reactions

1. Severe symptomatic bradycardia when combined sofosbuvir and amiodarone.

- 2. Decompensation of liver function and liver failure.
- 3. Skin rash and photosensitivity, manifested as an aggravated sunburn reaction.

 Contraindications

Since this product can only be used in combination with other antiviral drugs (including PegIFN α and RBV) to treat chronic HCV infection, the contraindications of other drugs are also applicable to the combined treatment regimen.

 Pregnant and lactating women

There are no sufficient human data to determine whether this product poses a risk to pregnancy outcomes. It is not clear whether this product and its metabolites can be secreted into human milk, affect human lactation, or affect breastfeeding infants.

 Children

The safety and efficacy of this product in pediatric patients have not yet been determined.

 Elderly

The clinical studies of this product did not include a sufficient number of patients older than 65 years old, so it is impossible to determine whether there is a difference between the response of elderly patients and young patients to the drug. Elderly patients do not need to adjust the dose of this product.

 Metabolism

Simeprevir is metabolized in the liver. In vitro experiments on human liver microsomes show that simeprevir is mainly oxidized and metabolized through the liver CYP3A system. It cannot be ruled out that CYP2C8 and CYP2C19 are also involved in metabolism. The simultaneous use of this product with CYP3A intermediate or strong inhibitors can significantly increase the plasma exposure of simeprevir, and the simultaneous use of CYP3A with intermediate or strong inducers can significantly reduce the plasma exposure of simeprevir.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS12979860	IFNL4	CC	normal drug response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The simeprevir drug response test item uses a polymorphic locus in the *IFNL4* gene. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variant.

 Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Amikacin

Amikacin is an aminoglycoside antibiotic, which works by blocking the function of the bacteria's 30S ribosomal subunit, making it unable to produce proteins. It has good effects on most Enterobacteriaceae, Pseudomonas aeruginosa and some other Pseudomonas, Acinetobacter, Alcaligenes etc., but is ineffective against anaerobic bacteria.

My Amikacin

Use With Drug Labeling

ADVERSE EFFECT : No high-risk mutation is detected in mitochondria, hence normal risk of aminoglycoside antibiotic-induced deafness; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Suitable for sensitive gram-negative bacilli (Pseudomonas aeruginosa and some other pseudomonas, Escherichia coli, Proteus, Klebsiella, Enterobacter, Serratia, Acinetobacter Genus) and Staphylococcus (methicillin-sensitive strains) caused serious infections, such as bacteremia or sepsis, bacterial endocarditis, lower respiratory tract infections, bone and joint infections,

biliary tract infections, abdominal infections, complicated urine road infections, skin and soft tissue infections, etc.

Adverse reaction

1. Patients may experience hearing loss, tinnitus, or fullness of the ears; a small number of patients may also experience symptoms such as dizziness and unstable walking. Hearing loss generally does not worsen after the drug is stopped, but some individuals may continue to develop deafness after the drug is stopped.
2. This product has certain nephrotoxicity. Patients may have hematuria, decreased urination frequency or urine output, blood urea nitrogen, increased blood creatinine, etc. Most of them are reversible and can be relieved immediately after stopping the drug, but there are also individual reports of renal failure.
3. Weakness, lethargy, dyspnea and other neuromuscular blockade are rare.
4. Other adverse reactions include headache, numbness, acupuncture infection, tremor, convulsions, joint pain, drug fever, eosinophilia, abnormal liver function, blurred vision, etc.

Contraindications

1. It is forbidden for patients allergic to amikacin or other aminoglycosides.

Pregnant and lactating women

The drug belongs to the D category of pregnant women, that is, it is harmful to humans, but the benefits may outweigh the disadvantages after the drug. This product can pass through the placenta to reach the fetal tissues, which may cause hearing damage to the fetus. Pregnant women must fully weigh the pros and cons before using this product. Lactating women should suspend lactation when taking medicine.

Children

Aminoglycosides should be used with caution in pediatrics, especially in premature infants and newborns whose kidney tissue is not yet fully developed, which prolongs the half-life of this type of drug, and the drug easily accumulates in the body to produce toxic reactions.

Elderly

The renal function of elderly patients has a certain degree of physiological decline. Even if the measured value of renal function is within the normal range, a smaller amount of treatment should be used. Elderly patients are more likely to have various toxic reactions after applying this product, and blood drug concentration should be monitored as much as possible during the course of treatment.

Metabolism

Amikacin is not metabolized in vivo. The elimination half-life of adult blood is 2 to 2.5 hours. It can enter the fetal tissue through the placenta. It is mainly discharged through glomerular filtration, and more than 90% of it is discharged within 24 hours after administration. Hemodialysis and peritoneal dialysis can remove a considerable amount of drugs from the blood.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267606617	MT-RNR1	AA	normal risk of deafness due to aminoglycoside antibiotics
RS267606618	MT-RNR1	--	Not Detected
RS267606619	intergenic	--	Not Detected

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations,

excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The amikacin drug reaction item uses 3 polymorphic loci of the *MT-RNR1* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Kanamycin

Kanamycin is a protein biosynthesis inhibitor.

My Kanamycin

Use With Drug Labeling

ADVERSE EFFECT : No high-risk mutation is detected in mitochondria, hence normal risk of aminoglycoside antibiotic-induced deafness; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Suitable for sensitive gram-negative bacilli (*Pseudomonas aeruginosa* and some other *pseudomonas*, *Escherichia coli*, *Proteus*, *Klebsiella*, *Enterobacter*, *Serratia*, *Acinetobacter* Genus) and *Staphylococcus* (methicillin-sensitive strains) caused serious infections, such as bacteremia or sepsis, bacterial endocarditis, lower respiratory tract infections, bone and joint infections, biliary tract infections, abdominal infections, complicated urine tract infections, skin and soft tissue infections, etc.

-
2. Because this product is stable to most aminoglycoside passivating enzymes, it is especially suitable for the treatment of severe infection caused by gram-negative bacilli against kanamycin, gentamicin or tobramycin-resistant strains.

Adverse reaction

1. Patients may experience hearing loss, tinnitus, or fullness of the ears; a small number of patients may also experience symptoms such as dizziness and unstable walking. Hearing loss generally does not worsen after the drug is stopped, but some individuals may continue to develop deafness after the drug is stopped.
2. This product has certain nephrotoxicity. Patients may have hematuria, decreased urination frequency or urine output, blood urea nitrogen, increased blood creatinine, etc. Most of them are reversible and can be relieved immediately after stopping the drug, but there are also individual reports of renal failure.
3. Weakness, lethargy, dyspnea and other neuromuscular blockade are rare.
4. Other adverse reactions include headache, numbness, acupuncture infection, tremor, convulsions, joint pain, drug fever, eosinophilia, abnormal liver function, blurred vision, etc.

Contraindications

1. It is forbidden for patients allergic to amikacin or other aminoglycosides.

Pregnant and lactating women

The drug belongs to the D category of pregnant women, that is, it is harmful to humans, but the benefits may outweigh the disadvantages after the drug. This product can pass through the placenta to reach the fetal tissues, which may cause hearing damage to the fetus. Pregnant women must fully weigh the pros and cons before using this product. Lactating women should suspend lactation when taking medicine.

Children

Aminoglycosides should be used with caution in pediatrics, especially in premature infants and newborns whose kidney tissue is not yet fully developed, which prolongs the half-life of this type of drug, and the drug easily accumulates in the body to produce toxic reactions.

Elderly

The renal function of elderly patients has a certain degree of physiological decline. Even if the measured value of renal function is within the normal range, a smaller amount of treatment should be used. Elderly patients are more likely to have various toxic reactions after applying this product, and blood drug concentration should be monitored as much as possible during the course of treatment.

Metabolism

This product is not metabolized in the body, and is mainly excreted in urine after glomerular filtration. 80% to 90% of it is excreted in urine within 24 hours after administration.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267606617	MT-RNR1	AA	normal risk of deafness due to aminoglycoside antibiotics
RS267606618	MT-RNR1	--	Not Detected
RS267606619	intergenic	--	Not Detected

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be

sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The kanamycin drug reaction item uses 3 polymorphic loci of the *MT-RNR1* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Streptomycin

Streptomycin sulfate is an aminoglycoside antibiotic.

My Streptomycin

Use With Drug Labeling

ADVERSE EFFECT : No high-risk mutation is detected in mitochondria, hence normal risk of aminoglycoside antibiotic-induced deafness; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. This product is mainly used in combination with other anti-tuberculosis drugs for initial cases of tuberculosis caused by *Mycobacterium tuberculosis*, or other sensitive mycobacterial infections.
2. This product can be used solely for the treatment of tularemia, or combined with other antibacterial drugs for the treatment of plague, inguinal granuloma, brucellosis, rodent bite fever, etc.
3. It can also be combined with penicillin or ampicillin to treat endocarditis caused by *Streptococcus viridans* or *Enterococcus*.



Adverse reactions

1. Hematuria, decreased urination frequency or decreased urine output, loss of appetite, thirst and other nephrotoxic symptoms, a few can produce blood urea nitrogen and increased creatinine value.
2. When the vestibular function is affected, there may be symptoms such as unstable walking and dizziness. When the auditory nerve is affected, hearing loss, tinnitus, and fullness of the ears may occur.
3. Some patients may have symptoms of peripheral neuritis such as numbness of the face or extremities, tingling, etc.
4. Occasionally, some neuromuscular block symptoms may occur, such as vision loss (optic neuritis), lethargy, weakness, and dyspnea.
5. Occasionally, rash, itching, redness and swelling may occur. A small number of patients may still have symptoms of ototoxicity after stopping the drug, such as hearing loss, tinnitus, and fullness of the ears. Such situations should be paid attention to.



Contraindications

It is forbidden for patients allergic to streptomycin or other aminoglycosides.



Pregnant and lactating women

The drug belongs to the D category of pregnant women, that is, it is harmful to humans, but the benefits may outweigh the disadvantages after the drug. This product can pass through the placenta to reach the fetal tissues, which may cause hearing damage to the fetus. Pregnant women must fully weigh the pros and cons before using this product. Lactating women should suspend lactation when taking medicine.



Children

Aminoglycosides should be used with caution in pediatrics, especially in premature infants and newborns whose kidney tissue is not yet fully developed, which prolongs the half-life of this type of drug, and the drug easily accumulates in the body to produce toxic reactions.



Elderly

Elderly patients are prone to various toxic reactions after the application of aminoglycosides, and blood drug concentration should be monitored as much as possible during the course of treatment. The renal function of elderly patients has a certain degree of physiological decline, even if the measured value of renal function is within the normal range, a smaller amount of treatment should be used.



Metabolism

Streptomycin is not metabolized in the body, and is mainly excreted through glomerular filtration. 80% to 98% is excreted in urine within 24 hours after administration, about 1% is excreted from bile, and a small amount is excreted from milk, saliva and sweat.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267606617	MT-RNR1	AA	lower risk of hearing loss
RS267606618	MT-RNR1	--	Not Detected
RS267606619	intergenic	--	Not Detected

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only

and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The streptomycin drug reaction item uses 3 polymorphic loci of the *MT-RNR1* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Gentamicin

Gentamicin is an antibiotic that has a therapeutic effect on both gram-positive bacteria and gram-negative bacteria. Indications include bone infection, endocarditis, pelvic inflammation, meningitis, pneumonia, urinary tract infection, and sepsis, etc.

My Gentamicin

Use With Drug Labeling

ADVERSE EFFECT : No high-risk mutation is detected in mitochondria, hence normal risk of aminoglycoside antibiotic-induced deafness; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It can be used for intestinal infectious diseases such as dysentery and enteritis caused by sensitive bacteria. It can also be used to clean the intestinal cavity before surgery.

Adverse reactions

- 1. Nausea, loss of appetite, vomiting, abdominal distension and other gastrointestinal symptoms, skin rashes, and elevated serum aminotransferases are rare.
- 2. It appears occasionally, like hearing loss, tinnitus or fullness of the ears (ototoxicity), hematuria, significant reduction in the frequency of urination or decreased urine output, loss of appetite, extreme thirst (nephrotoxicity), unsteady walking, dizziness (ototoxicity, effects vestibule), dyspnea, lethargy, extreme weakness (nerve-muscle block).

 Contraindications

- 1. It is forbidden for those who are allergic to this product or other aminoglycoside antibiotics.

 Pregnant and lactating women

Although this product has little absorption after oral administration, pregnant women should still use it with caution because the absorbed part can enter the fetal circulation through the placenta. Lactating women should suspend lactation during medication.

 Children

Due to potential ototoxicity and nephrotoxicity, children should use this product with caution.

 Elderly

Due to potential ototoxicity and nephrotoxicity, elderly patients should use this product with caution.

 Metabolism

The product is not metabolized in vivo, and is excreted through glomerular filtration. The urine concentration can exceed 100µg/ml, and it is excreted 50 to 93% within 24 hours.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267606617	MT-RNR1	AA	lower risk of hearing loss
RS267606618	MT-RNR1	--	Not Detected
RS267606619	intergenic	--	Not Detected

How to use the test results

 Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The gentamicin drug reaction item uses 3 polymorphic loci of the *MT-RNR1* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

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1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
 2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
 3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Tobramycin

Tobramycin is an aminoglycoside antibiotic that is commonly used for infections caused by sensitive bacteria.

My Tobramycin

Use With Drug Labeling

ADVERSE EFFECT : No high-risk mutation is detected in mitochondria, hence normal risk of aminoglycoside antibiotic-induced deafness; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for local infection of external eyes and appendages caused by sensitive bacteria.

Adverse reactions

Occasionally, local irritation symptoms, such as eyelid burning or swelling, conjunctival erythema, etc.; rare allergic reactions.

Contraindications

1. It is forbidden for those who are allergic to this product and other aminoglycoside antibiotics.

Pregnant and lactating women

A small amount of this product is absorbed into the systemic blood circulation after eye drops. Because a small amount of the absorbed part can pass through the placenta and can also be secreted into milk, pregnant women should use it with caution, and lactating women should stop lactating during the use of this product.

Children

Because this product has potential nephrotoxicity and ototoxicity, it should be used with caution in children.

Elderly

Because this product has potential nephrotoxicity and ototoxicity, it should be used with caution in elderly patients.

Metabolism

A small amount of this product is absorbed into the systemic blood circulation after eye drops.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267606617	MT-RNR1	AA	lower risk of hearing loss
RS267606618	MT-RNR1	--	Not Detected
RS267606619	intergenic	--	Not Detected

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The tobramycin drug response item uses 3 polymorphic loci of the *MT-RNR1* gene. The test results indicate the genotype results of the user's detected variation above, and interpret how these genotypes will affect the use of the drug according to existing scientific research. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.

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3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Neomycin

Neomycin is an aminoglycoside antibiotic with nephrotoxicity and inner ototoxicity, used for allergic dermatitis, skin pruritus, etc.

My Neomycin

Use With Drug Labeling

ADVERSE EFFECT : No high-risk mutation is detected in mitochondria, hence normal risk of aminoglycoside antibiotic-induced deafness; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is used for allergic dermatitis, subacute chronic eczema, scrotal eczema, pruritus and pruritus vulvae, etc.

Adverse reactions

1. Long-term and large-scale application can cause skin atrophy, telangiectasia, acne-like dermatitis, perioral dermatitis, folliculitis, and increase the susceptibility to infection.

2. It can cause allergic contact dermatitis, occasionally.



Contraindications

1. People who are allergic to this product should not use it.
2. It is forbidden for skin ulcers and eyes.
3. It is forbidden for acute and subacute inflammation and exudative erosive skin diseases.



Pregnant and lactating women

Pregnant and lactating women should weigh the pros and cons before using it with caution. Pregnant women can not use it for a long time, large area or large amount.



Children

Use with caution in children.



Elderly

Elderly patients should use this product with caution.



Metabolism

Metabolism is minimal, 12% to 25% of it is excreted by the kidney within 24 hours, and 50% to 90% of it is excreted in the bile.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS267606617	MT-RNR1	AA	normal risk of deafness due to aminoglycoside antibiotics
RS267606618	MT-RNR1	--	Not Detected
RS267606619	intergenic	--	Not Detected

How to use the test results



Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or

professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The neomycin drug response item uses 3 polymorphic loci of the *MT-RNR1* gene. The test results indicate the genotype results of the user's detected variation above, and interpret how these genotypes will affect the use of the drug according to existing scientific research. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.

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2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
 3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Pyrazinamide

Pyrazinamide has a good antibacterial effect on human tubercle bacilli, especially for tuberculosis bacilli that grow slowly in an acidic environment, and is currently the best bactericidal agent.

My Pyrazinamide

Use With Drug Labeling

ADVERSE EFFECT : Relatively lower risk of hepatotoxicity; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is only effective against mycobacteria, and is indicated for the treatment of tuberculosis when combined with other antituberculous agents (such as streptomycin, isoniazid, rifampicin and ethambutol) .

Adverse reaction

1. High incidence: joint pain (often mild and self limiting due to hyperuricemia).

2. Less incidence: anorexia, fever, fatigue or weakness, yellowing of eyes or skin (hepatotoxicity), chills.

 Contraindications

This product is highly toxic and should not be used by children.

 Pregnant and lactating women

Pregnant women with tuberculosis can be treated with isoniazid, rifampicin, and ethambutol for 9 months. Those who are resistant to any of the above drugs and may be sensitive to this product may consider using this product.

 Children

This product is highly toxic and should not be used by children. When it must be used, it must be decided after weighing the advantages and disadvantages.

 Elderly

Still uncertain.

 Metabolism

Pyrazinamide is mainly metabolized in the liver, hydrolyzed into pyrazine acid, which is a metabolite with antibacterial activity, and then hydroxylated into an inactive metabolite, which is excreted through glomerular filtration.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1041983	NAT2	CC	normal risk of hepatotoxicity
RS1799930	NAT2	GG	normal risk of hepatotoxicity

How to use the test results

 Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or

professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The pyrazinamide drug response item uses 2 polymorphic loci of the *NAT2* gene. The test results indicate the genotype results of the user's detected variation above, and interpret how these genotypes will affect the use of the drug according to existing scientific research. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.

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2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
 3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Rifampin

Rifampicin is a broad-spectrum antibiotic belonging to the rifamycin family.

My Rifampin

Use With Drug Labeling

ADVERSE EFFECT : Drug metabolism relatively increased, have decreased risk of hepatotoxicity; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. This product is combined with other anti tuberculosis drugs for the initial treatment and retreatment of all kinds of tuberculosis, including the treatment of tuberculous meningitis.
2. This product is used in combination with other drugs for the treatment of leprosy and non tuberculosis mycobacterium infection.
3. This product can be used in combination with vancomycin (intravenous) for severe infection caused by methicillin resistant Staphylococcus. The combined regimen of rifampicin and erythromycin is used for severe infection of Legionella.

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4. For asymptomatic carriers of *Neisseria meningitidis* to eliminate *Neisseria meningitidis* in nasopharynx. But it is not suitable for the treatment of *Neisseria meningitidis* infection.

Adverse reaction

1. Gastrointestinal reactions are the most common. After oral administration of this product, gastrointestinal reactions such as anorexia, nausea, vomiting, epigastric discomfort and diarrhea can occur. The incidence rate is 1.7% to 4.0%, but they are all tolerable.
2. Hepatotoxicity is the main adverse reaction of this product, with an incidence of about 1%. In the first few weeks of the course of treatment, a few patients may have elevated serum transaminases, hepatomegaly and jaundice, mostly asymptomatic transient elevated serum transaminases, which can recover by itself during the course of treatment. The elderly, alcoholics, malnutrition, original liver disease or other factors causing abnormal liver function are more likely to occur.
3. Allergy: "Influenza-like syndrome" may occasionally occur after high-dose intermittent therapy, which is manifested as afraid of cold, chills, fever, discomfort, dyspnea, dizziness, drowsiness and muscle pain. The frequency of occurrence is significantly related to the dose and interval time. Acute hemolysis or renal failure can occasionally occur. At present, it is considered that its mechanism is allergic reaction.
4. Others: After taking this product, the patient's urine, saliva, sputum and tears can be orange-red. Leukopenia, shortened prothrombin time, headache, vertigo, visual impairment, etc. are occasionally seen.

Contraindications

1. It is forbidden for those who are allergic to this product or rifamycin antibiotics.
2. It is forbidden for patients with severe liver insufficiency, biliary obstruction and pregnant women within 3 months.

Pregnant and lactating women

Rifampicin can pass through the placental barrier and has been shown to be teratogenic in animal experiments. Although there is no report of teratogenesis in humans, there is not enough data to show that it can be safely used in pregnancy. Rifampicin can be excreted by breast milk. Lactating women should fully weigh the pros and cons before deciding whether to use it.

Children

The safety of this product in children under 5 years old has not been established.

Elderly

The liver function of elderly patients has decreased, and the dosage should be reduced as appropriate.

Metabolism

This product can be rapidly deacetylated by the action of self-induced microsomal oxidase in the liver to become a metabolite with antibacterial activity, desacetyl rifampicin. After hydrolysis, it forms inactive metabolites and is excreted in urine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1041983	NAT2	CC	normal risk of hepatotoxicity
RS1799930	NAT2	GG	normal risk of hepatotoxicity

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be

sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The rifampin drug response item uses 2 polymorphic loci of the *NAT2* gene. The test results indicate the genotype results of the user's detected variation above, and interpret how these genotypes will affect the use of the drug according to existing scientific research. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Ethambutol

Ethambutol is a bacteriostatic and anti-tuberculosis drug, which is mainly used to treat tuberculosis caused by tuberculosis bacillus in combination with other anti-tuberculosis drugs.

My Ethambutol

Risk of Adverse Effect

ADVERSE EFFECT : Patients with rs1799930-AG genotype may have a decreased metabolism of drug and an increased risk of hepatotoxicity as compared to GG genotype, or an increased metabolism of drug and a decreased risk of hepatotoxicity as compared to AA genotype; Be alert to adverse effect.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1.It is suitable for the treatment of tuberculosis caused by Mycobacterium tuberculosis in combination with other anti-tuberculosis drugs.

-
2. It can also be used for the treatment of tuberculous meningitis and atypical mycobacterial infection.

Adverse reaction

1. Common optic nerve damage, such as retrobulbar optic neuritis and optic nerve central fiber damage. It may be related to the decrease of these metal elements after this product chelating with copper, zinc and other metal elements. The incidence of retrobulbar optic neuritis is about 0.8%, which is related to dose and course of treatment. It is easy to occur when the daily dose is greater than 25mg/kg. The incidence of daily dose of 15mg/kg is 1%, 25mg / kg is 6%, and 35mg/kg increases to 15%. The manifestations are blurred vision, eye pain, red-green blindness or decreased vision and reduced visual field. Early detection and timely withdrawal of the above reactions can disappear within weeks or months, and permanent loss of visual function rarely occurs.
2. Chills, joint swelling and pain (especially big toe, condyle and knee joint) and fever and tension of skin on the surface of diseased joints (acute gout and hyperuricemia) is rarely.
3. Gastrointestinal discomfort, nausea, vomiting, diarrhea, liver function damage, peripheral neuritis (often manifested as numbness, acupuncture, burning pain or weakness of hands and feet) and allergic reactions (often manifested as rash, pruritus, headache, fever and joint pain) are occasionally seen.

Contraindications

1. It is forbidden for those who are allergic to this product.
2. Patients with known optic neuritis, alcohol poisoning, and people younger than 13 years old should use it with caution.

Pregnant and lactating women

Ethambutol can penetrate the placenta and can be distributed to breast milk. Pregnant women and lactating women must fully weigh the pros and cons of medication.

Children

There is a lack of clinical data for children under 13 years old. Since it is difficult to monitor visual changes in young children, this product is not recommended for children under the age of 13.

Elderly

The elderly are often accompanied by physiological decline in renal function, so the dosage should be adjusted according to renal function.

Metabolism

After oral administration, it is absorbed by the gastrointestinal tract from 75% to 80%. It is widely distributed in body tissues and body fluids (except cerebrospinal fluid). The drug concentration in red

blood cells is equal to or twice that in plasma and lasts for 24 hours; the drug concentration in kidney, lung, saliva and urine is higher, but the concentration in pleural fluid and ascites is lower. This product cannot penetrate into normal meninges, but there may be a trace in cerebrospinal fluid of patients with tuberculous meningitis. It is mainly metabolized by liver, and about 15% of the dosage is metabolized into inactive metabolites. It is excreted by glomerular filtration and renal tubular secretion; about 80% were excreted within 24 hours after administration, at least 50% were excreted in its original form, and about 15% were inactive metabolites. About 20% is excreted in the feces in its original form. The drug concentration in breast milk is approximately equivalent to the drug concentration in maternal blood. A considerable amount of ethambutol can be removed from the body through hemodialysis and peritoneal dialysis.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1041983	NAT2	CC	decreased risk of hepatotoxicity
RS1799930	NAT2	GG	decreased risk of hepatotoxicity

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland

China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The ethambutol drug response item uses 2 polymorphic loci of the *NAT2* gene. The test results indicate the genotype results of the user's detected variation above, and interpret how these genotypes will affect the use of the drug according to existing scientific research. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain

professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Trastuzumab

Trastuzumab is a monoclonal antibody that acts on human epidermal growth factor receptor 2 (HER-2). It is mainly used to treat some HER-2 positive breast cancers.

My Trastuzumab

Use With Drug Labeling

EFFICACY : Patients who are treated with trastuzumab have an increased of drug response; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Metastatic breast cancer: This product is suitable for metastatic breast cancer with overexpression of HER2: as a single drug to treat metastatic breast cancer that has received one or more chemotherapy regimens; combined with paclitaxel or docetaxel, used for patients with metastatic breast cancer who have not received chemotherapy.
2. Adjuvant treatment of breast cancer: Single drug of this product suitable for the adjuvant treatment of HER2 overexpression breast cancer after surgery, anthracycline-containing adjuvant

chemotherapy and radiotherapy (if applicable).

3. Metastatic gastric cancer: This product combined with capecitabine or 5-fluorouracil and cisplatin is suitable for patients with metastatic gastric adenocarcinoma or gastroesophageal junction adenocarcinoma with overexpression of HER2 who have not previously received treatment for metastatic disease.
4. Trastuzumab can only be used in patients with metastatic gastric cancer with overexpression of HER2. HER2 overexpression is defined as the IHC3+ or IHC2+/FISH+ results obtained using a validated detection method.

Adverse reactions

1. The most common adverse reactions are: fever, nausea, vomiting, infusion reactions, diarrhea, infection, worsening cough, headache, fatigue, dyspnea, skin rash, neutropenia, anemia and myalgia.
2. Adverse reactions that require interruption or discontinuation of trastuzumab treatment include: congestive heart failure, significant decline in left ventricular function, severe infusion reactions, and pulmonary toxicity.
3. Other adverse reactions include: cardiomyopathy, infusion reactions, exacerbation of neutropenia caused by chemotherapy, and pulmonary toxicity.

Contraindications

1. People who are allergic to trastuzumab are prohibited.

Pregnant and lactating women

Not yet clear.

- Pregnant women During pregnancy, pregnant women should avoid using trastuzumab, and only use trastuzumab when the potential benefit to the mother is far greater than the potential risk to the fetus.
- Lactating women do not know whether trastuzumab can be secreted into human milk. The decision to stop breastfeeding or trastuzumab treatment should be based on the half-life of trastuzumab and its importance to the mother.

Children

The safety and efficacy of this product for patients younger than 18 years of age have not been established.

Elderly

It is not yet possible to judge whether the toxicity of salt touzumab in elderly patients is different from that in younger patients.

Metabolism

Studies on drug clearance for metastatic breast cancer have shown that the pharmacokinetics of 10, 50, 100, 250 and 500 mg trastuzumab intravenously administered once a week are dose-dependent. As the dose level increases, the average half-life increases and the clearance rate decreases. The average half-life of the 10 and 500 mg dose levels is 1.7 and 12 days, respectively. The volume of distribution of trastuzumab is approximately equal to the volume of serum (44mL/Kg).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1801274	FCGR2A	AA	better drug response
RS396991	FCGR3A	--	Not Detected

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only

and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The trastuzumab drug reaction item uses 2 polymorphic loci of *FCGR2A* and* *FCGR3A** gene. The test results indicate the user's genotype result of the mutation detected above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Cetuximab

Cetuximab is a chimeric monoclonal antibody that specifically binds to EGFR on the surface of tumor cells, inhibits its downstream signal transduction and mediates anti-tumor immune responses.

My Cetuximab

Risk of Insufficient Efficacy

EFFICACY : Patients with rs4444903-AA genotype, compared to GG genotype, may have a decreased drug response; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Used alone or in combination with irinotecan for the treatment of metastatic rectal cancer that overexpresses epidermal growth factor (EGF) receptors and is resistant to irinotecan-based chemotherapy regimens.

Adverse reactions

This product is well tolerated, and most of the adverse reactions are tolerable. The most common are acne-like rash, fatigue, diarrhea, nausea, vomiting, abdominal pain, fever and constipation. Other adverse reactions include decreased white blood cell count and difficulty breathing. Skin toxicity (acne-like rash, dry skin, laceration, infection, etc.) can disappear naturally. A small number of patients may have severe allergic reactions, infusion reactions, sepsis, interstitial pulmonary disease, renal failure, pulmonary embolism and dehydration. Among the patients who received the single-drug therapy of this product and the combination therapy of this product and irinotecan, 5% and 10% of patients withdrew due to adverse reactions, respectively.

Contraindications

1. Patients with severe hypersensitivity (Grade 3 or 4) to cetuximab should not use this product.

Pregnant and lactating women

Because this product can pass through the placental barrier, it may damage the fetus or affect maternal fertility, so pregnant women and women of childbearing age who have not taken contraceptive measures should use it with caution. Because this product can be secreted through breast milk, women who are breastfeeding should use it with caution.

Children

Before the safety of this product for pediatric patients has not been confirmed, children should not be used.

Elderly

Elderly patients do not need to adjust the dose. Patients over 75 years of age have limited medication experience.

Metabolism

This product can specifically bind to EGF receptors expressed on the surface of normal cells and a variety of cancer cells, and competitively block the binding of EGF and other ligands, such as transforming growth factor alpha (TGF- α). This product is an IgG1 monoclonal antibody against the EGF receptor. After the two specifically bind, it inhibits the intracellular signal transduction pathway by inhibiting the tyrosine kinase (TK) that binds to the EGF receptor, thereby inhibiting The proliferation of cancer cells induces apoptosis of cancer cells and reduces the production of matrix metalloproteinases and vascular endothelial growth factors. The pharmacokinetics of this product in single-dose treatment or combined with chemotherapy and radiotherapy showed non-linear characteristics.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4444903	EGF	AA	poor response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The cetuximab drug reaction item uses 1 polymorphic locus of *EGF* gene. The test results indicate the user's genotype result of the mutation detected above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
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6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Infliximab

Infliximab is a disease control anti-rheumatic drug used for diseases such as rheumatoid arthritis and Crohn's disease.

My Infliximab

Use With Drug Labeling

EFFICACY : Patients with rs1800629-GG genotype, compared to AA genotype, may have an increased drug response.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Applicable to rheumatoid arthritis: This product is a disease control anti-rheumatic drug. For patients with moderate to severe active rheumatoid arthritis, the combination of this product and methotrexate can be used to reduce symptoms and signs, improve physical function, and prevent patient disability.
2. Applicable to Crohn's disease: For patients with moderate to severe active Crohn's disease who have received poor traditional treatment effects, this product can be used to reduce symptoms

and signs, achieve and maintain clinical efficacy, promote mucosal healing, and improve quality of life, make patients reduce the amount of corticosteroids or stop using corticosteroids.

3. **Fistula Crohn's Disease** For patients with fistula Crohn's disease, this product can be used to: reduce the number of intestinal-cutaneous fistulas and rectal-vaginal fistulas, promote and maintain fistula healing; reduce symptoms and signs; improve the quality of life. **Ankylosing Spondylitis** For patients with active ankylosing spondylitis, this product can be used to reduce symptoms and signs, including increasing the range of activity, improving physical function, and improving the quality of life.



Adverse reactions

1. **Severe infection:** Patients treated with this product are at increased risk of serious infection, which can lead to hospitalization or death. Most patients with infections are taking methotrexate or glucocorticoids. If the patient develops severe infection or sepsis, this product should be discontinued. The reported infections include:
 - Active tuberculosis, including recurrence of latent tuberculosis. Patients with tuberculosis are often accompanied by diffuse or extrapulmonary disease. It is necessary to check whether the patient has latent tuberculosis before and during treatment with this product. If the test result is positive, the latent tuberculosis should be treated before this product is administered.
 - Invasive fungal infections, including: histoplasmosis, coccidioidomycosis, candidiasis, aspergillosis, blastomycosis and pneumocysticercosis. Patients with histoplasmosis or other invasive fungal infections are mostly diffuse rather than focal diseases. In some patients with active infection, antigen and antibody test results for histoplasmosis may be negative. If patients with severe systemic diseases are at risk of invasive fungal infections, empirical antifungal treatment should be considered.
 - Infections caused by bacteria, viruses and other conditional pathogens, including Legionella and Listeria. Before treating patients with chronic or recurrent infections, the risks and benefits of this product should be carefully considered.

During and after treatment with this product, patients should be closely monitored for signs and symptoms of infection, including patients who tested negative for latent tuberculosis infection before starting treatment.

2. **Malignant tumors:** There are reports that when children and adolescents are treated with tumor necrosis factor (TNF) inhibitors including this product, lymphomas and other malignant tumors occur, some of which are fatal. After this product was marketed, there have been reports of cases of hepatosplenic T-cell lymphoma (HSTCL) in patients receiving TNF inhibitor treatment including this product, which is a rare type of T-cell lymphoma. The course of these cases is aggressive and can lead to death. The vast majority of cases occur in patients with Crohn's disease or ulcerative colitis, and most of them are teenagers or young men. Almost all of the above patients have been treated with azathioprine or 6-mercaptopurine during or shortly before receiving TNF inhibitors.
3. The most serious adverse drug reactions related to the use of TNF inhibitors including this product also include hepatitis B virus (HBV) reactivation, congestive heart failure (CHF), serum sickness (delayed hypersensitivity) , Blood system reaction, systemic lupus erythematosus/lupus-

like syndrome, demyelinating disease, hepatobiliary events, lymphoma, intestinal or perianal abscess (Crohn's disease) and severe infusion reactions.

4. Common adverse reactions after marketing include: viral infection (such as influenza, herpes virus infection), bacterial infection (such as sepsis, cellulitis, abscess), allergic respiratory symptoms, headache, conjunctivitis, tachycardia, palpitations, Low blood pressure, high blood pressure, ecchymosis, hot flashes, flushing, upper respiratory tract infections, sinusitis, lower respiratory tract infections (such as bronchitis, pneumonia), dyspnea, nose bleeding, abdominal pain, nausea, gastrointestinal bleeding, diarrhea, digestion Poor, gastroesophageal reflux, constipation, abnormal liver function, elevated transaminases, new or worsening psoriasis, including pustular psoriasis (mainly palms and soles), urticaria, rash, itchy skin, hyperhidrosis, Dry skin, fungal dermatitis, eczema, hair loss, joint pain, myalgia, back pain, urinary tract infection, chest pain, fatigue, fever, chills, edema, etc.

Contraindications

1. Patients who are known to be allergic to mouse-derived protein or other ingredients of this product should not be used.
2. When the dose of this product is higher than 5mg/kg, it is contraindicated in patients with moderate to severe heart failure.

Pregnant women and lactating women

Since this product has no cross-reactivity with TNF α from species other than humans and chimpanzees, no studies on the reproduction of this product in animals have been carried out. Fertility and general reproductive toxicity tests were carried out on mice with similar antibodies that selectively inhibit the functional activity of mouse TNF α , and no maternal toxicity, embryo toxicity, and teratogenicity were found. The use of similar anti-TNF antibodies at a dose of 10-15 mg/kg for pharmacodynamic animal models can produce the greatest pharmacological activity. In animal reproductive toxicity studies, when the dose reached 40 mg/kg, there was no adverse effect. It is not known whether this product will cause harm to the fetus or affect fertility. For pregnant women, this product can be given only when really needed. During lactation, it is not known whether this product is excreted through breast milk and whether it is absorbed systemically after ingestion. Since many drugs and immunoglobulins can be excreted through breast milk, and because of the potential adverse reactions of this product to infants and young children, the importance of this product to the mother should be considered when deciding whether to stop breastfeeding or stop taking the drug.

Children

The use of this product in juvenile rheumatoid arthritis patients under 4 years old and children with Crohn's disease under 6 years old have not been studied. For juvenile rheumatoid arthritis and ankylosing spondylitis patients, the safety and effectiveness of this product have not been determined.

Seniors

No specific studies have been conducted on the use of this product in elderly patients. The correlation between the volume of distribution and clearance rate of this product and age has not been observed, but because the incidence of infection is usually higher in the elderly, it should be used with caution when treating elderly patients.

Metabolism

For a single intravenous infusion of this product from 3 mg/kg to 20 mg/kg, the maximum serum drug concentration has a linear relationship with the dose. The volume of distribution at steady state has nothing to do with the dose, indicating that the product is mainly distributed in the vascular cavity. The median value of the pharmacokinetic results when the therapeutic dose of rheumatoid arthritis is 3mg/kg to 10mg/kg and the therapeutic dose of Crohn's disease is 5mg/kg shows that the half-life of this product is 7.7-9.5 days. In each treatment, repeat the infusion in the 2nd and 6th weeks after the first dose of this product, and the expected drug-time curve can be obtained. Repeated administration continued, and no systemic accumulation occurred. No significant differences in clearance rate and volume of distribution were found in age or weight groups.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1800629	TNF, LTA	GG	normal drug response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The infliximab drug reaction item uses 1 polymorphic locus of *TNF* gene. The test results indicate the user's genotype result of the mutation detected above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.

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5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Sunitinib

Sunitinib is a multi-targeted oral drug for the treatment of tumors. It is used for the treatment of gastrointestinal stromal tumors and metastatic renal cell carcinoma that do not respond to or cannot tolerate standard therapies.

My Sunitinib

Use With Drug Labeling

ADVERSE EFFECT : Patients with rs1933437-AG genotype, compared to AA genotype, may have increased likelihood of myelosuppression when treated with sunitinib.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is used for inoperable advanced renal cell carcinoma (RCC).
2. It is used for gastrointestinal stromal tumors (GIST) that have failed or cannot be tolerated by imatinib mesylate.
3. It is used for unresectable, metastatic well-differentiated advanced pancreatic neuroendocrine tumor (pNET) adult patients.

Adverse reactions

The most common adverse reactions ($\geq 20\%$) in patients with gastrointestinal stromal tumor (GIST), advanced renal cell carcinoma (RCC) or pancreatic neuroendocrine tumor (pNET) are fatigue, fever, diarrhea, nausea, mucositis/stomatitis, vomiting, indigestion, abdominal pain, constipation, high blood pressure, peripheral edema, rash, hand-foot syndrome, skin fading, dry skin, hair color change, taste changes, headache, back pain, joint pain, extremity pain, cough, dyspnea, anorexia, and bleeding.

Contraindications

1. Those who are severely allergic to sunitinib should not be used.

Pregnant and lactating women

Based on animal reproduction research and its mechanism of action, pregnant women receiving sunitinib treatment may harm the fetus. There are no available data on the use of this product by pregnant women to report drug-related risks. There is no information on the presence of sunitinib and its metabolites in human milk. It is recommended that lactating women not breastfeed during treatment and for at least 4 weeks after the last dose.

Children

The safety and effectiveness of this product for pediatric patients are not yet clear. It is not recommended that this product be used in this population.

Elderly

Among the 825 patients with gastrointestinal stromal tumor (GIST) or metastatic renal cell carcinoma (RCC) who received this product, 277 (34%) were 65 years of age or older. In the pancreatic neuroendocrine tumor study, 22 patients (27%) who received sunitinib were 65 years of age and older. No difference in safety or effectiveness between young patients and elderly patients was found.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1933437	FLT3	AG	less likely to develop myelosuppression

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The sunitinib drug response test item uses a polymorphic locus of the *FLT3* gene. The test results indicate the genotype results of the user's detected the above variants and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

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1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
 2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
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 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Peginterferon Alfa-2A

It is a form of recombinant interferon usually used as part of combination therapy for the treatment of chronic hepatitis B and chronic hepatitis. Chronic hepatitis may lead to severe liver damage and liver cancer.

My Peginterferon Alfa-2A

Use With Drug Labeling

EFFICACY : Patients have normal treatment response; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for the treatment of chronic hepatitis B in adults. The patient cannot be in the decompensated stage of liver disease. Chronic hepatitis B must be diagnosed with serum markers (elevated transaminase, HBsAg, HBV DNA). It is usually also necessary to obtain histological evidence.
2. It is suitable for adult patients with chronic hepatitis C who have not received treatment before. The patient must have no manifestations of liver decompensation, and chronic hepatitis C must

be confirmed by serum markers (anti-HCV antibodies and HCV RNA). The diagnosis is usually confirmed by histology. This product is best used in combination with ribavirin in the treatment of this disease. When ribavirin is intolerant or contraindicated, this product can be used for single-drug treatment. There has not been a study of monotherapy with this product in patients with normal transaminase.

Adverse reactions

1. Chronic hepatitis B: In clinical trials of this product for the treatment of chronic hepatitis B for 48 weeks and follow-up for 24 weeks, the safety is similar to that of chronic hepatitis C, but the reported frequency of adverse events, especially depression, was significantly lower in chronic hepatitis B.
2. Chronic hepatitis C: When this product is used in combination with ribavirin, the safety indicators for HCV infected patients with normal ALT levels are consistent with those for patients with elevated ALT levels. Similarly, the combination therapy for 24 weeks is better tolerated than the combination therapy for 48 weeks.

Contraindications

1. Those who are allergic to this product.
2. Autoimmune chronic hepatitis.
3. Severe liver dysfunction or decompensated liver cirrhosis.
4. A history of severe heart disease, including unstable or uncontrolled heart disease within 6 months.
5. There is a serious mental illness or a history of serious mental illness, mainly depression.

Pregnant and lactating women

Animal experiments show that this product has reproductive toxicity, and the potential danger to humans is unknown. It is forbidden to use this product during pregnancy.

Children

The medications for patients under 18 years of age have not been fully studied. In addition, because the injection solution of this product contains benzyl alcohol, it cannot be used for newborns and infants.

Elderly

According to pharmacokinetics, pharmacodynamics, clinical tolerability and safety data, elderly patients do not need to adjust the dose.

Metabolism

The metabolic mechanism of this product has not been fully elucidated. Rat experiments show that this product is mainly metabolized in the liver, and the metabolites are mainly excreted through the kidneys.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS12979860	IFNL4	CC	normal drug response

How to use the test results

 Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

 Test content

Different genetic test versions and data sources may have different coverage of the above variants.

 Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

 Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The peginterferon Alfa-2A drug response test item uses a polymorphic locus of the *IFNL4* gene. The test results indicate the genotype results of the user's detected the above variants and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
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6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Peginterferon Alfa-2B

It is a form of recombinant interferon usually used as part of combination therapy for the treatment of chronic hepatitis B and chronic hepatitis. Chronic hepatitis may lead to severe liver damage and liver cancer.

My Peginterferon Alfa-2B

Use With Drug Labeling

EFFICACY : Patients have normal treatment response; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for the treatment of chronic hepatitis C. The patient must be ≥ 18 years old and suffer from compensatory liver disease. It is now believed that the ideal treatment for chronic hepatitis C is combined with ribavirin. When combined with ribavirin, please also refer to the product information of ribavirin.
2. It can also be used to treat HBeAg-positive chronic hepatitis B. The patient must be ≥ 18 years old and suffer from compensatory liver disease.

Adverse reactions

Most adverse reactions are mild or moderate, and treatment is not affected. According to reports, most patients experience headaches and muscle pains. Mental symptoms are not common. Life-threatening psychiatric symptoms rarely occur. These reactions include suicide, attempted suicide, suicidal ideation, irritability, aggressive behavior, and hallucinations. Similar to ordinary interferon, no unexpected adverse events occurred.

Contraindications

1. Those who are allergic to peginterferon alpha-2b.
2. Pregnant women cannot start the combination therapy of this product and ribavirin before obtaining a negative pregnancy reaction result.
3. Male patients with a pregnant spouse should not be treated with the combination of this product and ribavirin.
4. Those who have autoimmune hepatitis or have a history of their own diseases.
5. Patients with decompensated liver function.
6. Patients with severe renal insufficiency when used in combination.

Pregnant and lactating women

During the application of this product, pregnancy and breastfeeding are prohibited.

Children

It is not recommended for children or adolescents under the age of 18 to use this product.

Elderly

There is no obvious age-related pharmacokinetics of this product. The data of the elderly treated with a single dose of this product indicate that the dose of this product does not need to be changed according to age.

Metabolism

The average elimination half-life of peginterferon α -2b is about 40 ± 13.3 hours, and the apparent clearance rate is 22.0 mL/hr/kg. Although the mechanism of interferon in the human body has not been fully elucidated, the renal clearance may account for a small part (about 30%) of the apparent clearance of peginterferon α -2b.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS12979860

IFNL4

CC

normal drug response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The peginterferon Alfa-2B drug response test item uses a polymorphic locus of the *IFNL4* gene. The test results indicate the genotype results of the user's detected the above variants and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
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6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Interferon Alfa-2B, Recombinant

A drug used alone or with other drugs to treat viral diseases, such as cervical erosion, genital wart, shingles; And some tumors, such as melanoma, AIDS-related Kaposi sarcoma, hairy cell leukemia.

My Interferon Alfa-2B, Recombinant

Consider Dose Reduction

DOSE : Patients with rs1127354-CC genotype, compared to AC or AA genotype, may require a dose reduction of ribavirin when treated with recombinant interferon alfa-2b and ribavirin, or recombinant interferon alfa-2b, ribavirin and telaprevir.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is used for condyloma acuminata caused by human papilloma virus.
2. It can also be used to treat herpes zoster, herpes labialis and genital herpes.

Adverse reactions

Symptoms such as fever, headache, chills, fatigue, myalgia, and joint pain are common when using this product, which often appear in the first week of medication, and the adverse reactions mostly disappear after 48 hours of injection. In case of serious adverse reactions, the treatment plan must be modified or the medication must be stopped. Once an allergic reaction occurs, the medication should be stopped immediately. A small number of patients may also have blood abnormalities such as leukopenia and thrombocytopenia, which can return to normal after stopping the drug. Occasionally, there are adverse reactions such as anorexia, nausea, diarrhea, vomiting, hair loss, high (or low) blood pressure, and nervous system disorders.

Contraindications

1. A history of allergy to interferon alfa-2b, recombinant or any component of the preparation.
2. Suffer from severe heart disease.
3. Severe liver, kidney or bone marrow dysfunction.
4. People with epilepsy and impairment of central nervous system function.
5. People who cannot tolerate this product with other serious diseases should not be used.

Pregnant and lactating women

Pregnant women have limited medication experience, and the safe use of this product during pregnancy has not been established. Therefore, the injection of pregnant women should be given only when the condition is very necessary and after careful consideration by the clinician.

Children

Children's experience in medication is still limited. For such cases, the pros and cons should be carefully weighed before medication is prescribed by the doctor.

Elderly

For elderly patients with heart disease and advanced cancer patients, ECG should be checked before and during the treatment of this medicine, and the dosage should be adjusted according to the doctor's advice or stop using this product.

Metabolism

This product is injected intramuscularly or subcutaneously, the peak blood concentration time is 3.5-8 hours, and the elimination half-life is 4-12 hours. Kidney catabolism is the main way to eliminate interferon, and the elimination of bile secretion and liver metabolism is an important way. The absorption of intramuscular injection or subcutaneous injection exceeds 80%.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1127354	ITPA	CC	consider a dose reduction of ribavirin in combination therapy

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The interferon Alfa-2B, recombinant drug response test item uses a polymorphic locus of the *ITPA* gene. The test results indicate the genotype results of the user's detected the above variants and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

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2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
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6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Gemcitabine

Gemcitabine is a pyrimidine anti-tumor drug with the same mechanism of action as cytarabine.

My Gemcitabine

Use With Drug Labeling

EFFICACY : Patients with rs11598702-CT genotype, compared to TT genotype, may have increased metabolism of gemcitabine; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is suitable for the treatment of middle and advanced non-small cell lung cancer.

Adverse reactions

1. Blood system: Due to the myelosuppressive effect of gemcitabine, anemia, leukopenia, and thrombocytopenia may occur after gemcitabine administration. Bone marrow suppression is often mild to moderate, mostly neutropenia. Thrombocytopenia is also relatively common.

2. Digestive system: About two-thirds of patients have liver aminotransferase abnormalities, but most of them have mild, non-progressive damage, and do not need to stop the drug.
3. Kidney: Nearly half of the patients may have mild proteinuria and hematuria after medication, but they are rarely accompanied by clinical symptoms and changes in serum creatinine and urea nitrogen. However, some cases have reported unexplained renal failure.
4. Allergies: About 25% of patients may have rash, 10% of patients may have itching, usually mild rash, non-dose limiting toxicity, effective local treatment, rarely reported peeling, blisters and ulcers. During the infusion of gemcitabine, bronchospasm occurs in less than 1% of patients. The spasm is generally mild and short-lived, but may require parenteral administration. Patients who are known to be highly sensitive to this drug should not be used. It has been reported that about 10% of patients have difficulty breathing within a few hours after taking the medicine. This type of breathing difficulties often lasts for a short period of time, the symptoms are mild, almost rarely need to adjust the dose, most do not require special treatment, its pathogenesis is unclear, and the relationship with gemcitabine is also unclear.
5. Others: Approximately 20% of patients have flu-like manifestations. Most of the symptoms are mild, short-lived, and non-dose limiting. Only 1.5% of patients have severe symptoms, such as fever, headache, back pain, chills, myalgia, fatigue and anorexia are the most common symptoms. Cough, rhinitis, malaise, sweating and insomnia also occur. Some only manifested as fever and fatigue. The pathogenesis of such symptoms is still unclear, and reports have confirmed that salicylic acid drugs can alleviate symptoms. The incidence of edema/peripheral edema is about 30%, and some patients may have facial edema. The incidence of pulmonary edema is about 1%. Edema/peripheral edema often changes from mild to moderate and hardly affects the dosage of the medication. Some patients have local pain, which often reverses spontaneously after stopping the medication (gemcitabine). The mechanism that causes this toxicity is not yet clear, and there is no evidence that it is related to impaired heart, liver, and kidney function. The following adverse reactions are also commonly reported. 13% of patients have hair loss (often mild), 10% of patients are lethargic, 8% of patients have diarrhea, 7% of patients have oral toxicity (mainly ulcers and erythema), and 6% of patients have constipation. There have been case reports of hypotension, and some studies have reported myocardial infarction, congestive heart failure and arrhythmia, but there is no clear indication that the cardiotoxicity is caused by gemcitabine.

Contraindications

Patients who are allergic to this product are contraindicated.

Pregnant and lactating women

The safety of the drug for pregnant women is unknown. Animal experiments have shown that the drug has reproductive toxicity, such as reproductive defects or other toxic effects on embryo and fetal development, pregnancy and before and after delivery.

Children

The experiment has not been carried out and there is no reliable reference.

Elderly

It is also well tolerated by elderly patients over 65 years of age. Although age has an effect on the clearance and half-life of gemcitabine, there is no evidence that elderly patients need to adjust the dose.

Metabolism

Gemcitabine is rapidly metabolized by cytidine deaminase in the liver, kidney, blood and other tissues.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS11598702	NT5C2	CT	may have higher metabolic rate

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse

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Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The gemcitabine drug response test item uses a polymorphic locus of the *NT5C2* gene. The test results indicate the genotype results of the user's detected the above variants and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

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2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Methotrexate

Methotrexate is an anti-tumor drug of the antifolate type.

My Methotrexate

Risk of Adverse Effect

ADVERSE EFFECT : Patients with rs1045642-AG genotype may have an increased drug toxicity as compared to GG genotype, or a decreased drug toxicity as compared to AA genotype; Be alert to adverse effect. Patients with rs1801133-GG genotype, compared to AG or AA genotype, may have decreased risk of folic acid deficiency or toxicity, and may have an increased drug response; Consider drug dose Increase. Patients with rs1801394-AG genotype, compared to AA genotype, may have increased risk of toxicity; Be alert to adverse effect.

EFFICACY : Patients with rs4673993-CT genotype, compared to CC genotype, may have a decreased drug response; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge



Indications

1. It is used for various types of acute leukemia, especially acute lymphoblastic leukemia; malignant lymphoma, non-Hodgkin's lymphoma and granuloma fungoides and multiple myelopathy.
2. It is used for malignant hydatidiform mole, chorioepithelial carcinoma, breast cancer, ovarian cancer, cervical cancer and testicular cancer.
3. It is used for head and neck cancer, bronchial lung cancer and various soft tissue sarcomas.
4. High-dose is used for osteosarcoma. Intrathecal injection can be used to prevent and treat nerve invasion of meningeal leukemia and malignant lymphoma. This product also has a certain effect on psoriasis.



Adverse reactions

1. Gastrointestinal reactions, including stomatitis, lip ulcers, pharyngitis, nausea, vomiting, abdominal pain, diarrhea, gastrointestinal bleeding. Loss of appetite is common, and occasionally pseudomembranous or hemorrhagic enteritis.
2. Liver function damage, including jaundice, alanine aminotransferase, alkaline phosphatase, γ -glutamyl transpeptidase, etc., long-term oral administration can lead to liver cell necrosis, fatty liver, fibrosis and even cirrhosis of the liver.
3. In high-dose application, hyperuricemia nephropathy is caused by the deposition of this product and its metabolites in the renal tubules. At this time, hematuria, proteinuria, oliguria, azotemia or even uremia may occur.
4. Long-term medication can cause cough, shortness of breath, pneumonia or pulmonary fibrosis.
5. Bone marrow suppression: Mainly leukopenia and thrombocytopenia, long-term oral administration of small doses can cause significant bone marrow suppression, anemia and platelet reduction accompanied by skin or internal organ bleeding.
6. Hair loss, skin redness, itching or rash.
7. Infection can occur when leukocytes are low.



Contraindications

Patients who are known to be highly allergic to this product are contraindicated.



Pregnant and lactating women

During the application of this product, pregnancy and breastfeeding are prohibited.



Children

Children's daily 1.25mg ~ 5mg, depending on the bone marrow condition.



Elderly

The experiment has not been carried out and there is no reliable reference.

Metabolism

The plasma protein binding rate is about 50%. The amount of this product through the blood-brain barrier is very small, but a considerable amount can reach the systemic circulation after intrathecal injection. Part of it is converted into glutamate by liver cell metabolism.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1045642	ABCB1	AG	normal level of toxicity of methotrexate
RS11045879	SLCO1B1	CC	slightly low clearance of methotrexate, lower gastrointestinal toxicity
RS1801133	MTHFR	GG	slightly low risk of folic acid deficiency and poisoning, increased efficacy of methotrexate and higher doses should be used
RS1801394	MTRR	AG	slightly high level of toxicity of methotrexate
RS4673993	ATIC	CT	slightly weak response of methotrexate

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The methotrexate drug response test item uses 5 polymorphic loci of *ABCB1*, *SLCO1B1*, *MTHFR*, *MTRR* and *AT1C* genes. The test results indicate the genotype results of the user's detected the above variants and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.

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5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Carboplatin

Carboplatin is a broad-spectrum anti-tumor drug.

My Carboplatin

Use With Drug Labeling

EFFICACY : Patients have stronger treatment response and longer survival.

ADVERSE EFFECT : Administer standard dose of the drug if drug has a low toxicity profile.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is mainly used for ovarian cancer, small cell lung cancer, non-small cell lung cancer, head and neck squamous cell carcinoma, esophageal cancer, seminoma, bladder cancer, mesothelioma, etc.

Adverse reactions

1. Common reactions: Bone marrow suppression is a dose-limiting toxicity. Leukocytes and platelets reach their lowest point 21 days after medication, and usually recover about 30 days after medication; the lowest point of granulocytes occurs 21-28 days after medication, and usually

recovers around 35 days; leukopenia and thrombocytopenia are related to the dose and have an accumulation effect.

2. Rare reactions: (1) Allergic reactions (skin rash or itching, occasionally wheezing and coughing), occur within a few minutes after the medication. (2) Peripheral nerve toxicity: Numbness or tingling of fingers or toes. (3) Ototoxicity: High-frequency hearing loss occurs first, and tinnitus occurs occasionally. (4) Blurred vision, mucositis or stomatitis. (5) Nausea and vomiting, constipation or diarrhea, loss of appetite, hair loss and dizziness, occasionally allergic reactions and abnormal liver function.

Contraindications

1. Patients with obvious bone marrow suppression and liver and kidney dysfunction.
2. Those who are allergic to cisplatin or other platinum-containing compounds.

Pregnant and lactating women

The safety of carboplatin used in pregnancy is not yet understood. Carboplatin has been proved to have embryotoxicity and potential mutagenic effects in many experiments. Whether carboplatin is excreted in human milk is unclear.

Children

There is not enough information to describe the standard method of medication for children.

Elderly

Use with caution in elderly patients.

Metabolism

Carboplatin binds less to plasma proteins in the body, presents a two-compartment open model, and is mainly excreted through the kidneys.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS11615	ERCC1	GG	possibly reduced risk of toxicity, improved survival and better response
RS1801133	MTHFR	GG	lower risk of drug toxicity

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The carboplatin drug response test item uses 2 polymorphic loci of *ERCC1* and *MTHFR* genes. The test results indicate the genotype results of the user's detected the above variants and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

 Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Anastrozole

Anastrozole is a potent and selective non-steroidal aromatase inhibitor. It can inhibit the conversion of androstenedione produced in the adrenal glands of postmenopausal patients into estrone, thereby significantly reducing the plasma estrogen level and inhibiting the growth of breast tumors.

My Anastrozole

Risk of Insufficient Efficacy

EFFICACY : Patients with rs4646-AC genotype, compared to AA genotype, may have decreased drug response in pre-menopausal women and may have increased drug response in post-menopausal women; Monitoring of efficacy is required.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for the treatment of advanced breast cancer in postmenopausal women. For patients with negative estrogen receptor, this product can be considered if they have a positive clinical reaction to tamoxifen.

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2. It is suitable for adjuvant treatment of estrogen receptor positive early breast cancer in postmenopausal women.

Adverse reactions

1. Side effects mainly include skin flushing, vaginal dryness, excessive hair oil secretion, gastrointestinal dysfunction (anorexia, nausea, vomiting and diarrhea), fatigue, depression, headache or rash. Side effects are usually mild or moderate.
2. Uterine bleeding was occasionally reported, mainly in the first few weeks after patients switched from existing hormone therapy to this product.
3. There are reports of liver function changes in patients with advanced breast cancer (such as elevated peptidase and alkaline phosphatase), but many of these patients already have liver metastases or bone metastases. Studies on the causes of these changes have not yet been carried out. Clinical observations indicate that this product can slightly raise serum total cholesterol levels.

Contraindications

1. Premenopausal women.
2. Pregnant or lactating women.
3. Patients with severe renal injury (creatinine clearance < 20ml/min).
4. Patients with moderate to severe liver damage.
5. Patients known to be highly sensitive to anastrozole.

Pregnant and lactating women

Anastrozole is contraindicated in pregnant and lactating women.

Children

The safety and efficacy of anastrozole tablets have not been tested in children, so the drug should not be used in children.

Elderly

Same as adults.

Metabolism

The metabolic processes of anastrozole include N- dealkylation, hydroxylation and glucoaldehyde acidification. Its metabolites were excreted mainly through urine. Triazole, the main metabolite in plasma, did not inhibit aromatase activity.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4646	CYP19A1	AC	use of tamoxifen is less effective before menopause and more effective after menopause

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug

response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The anastrozole drug response test item uses 1 polymorphic locus of *CYP19A1* gene. The test results indicate the genotype results of the user's mutations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Exemestane

Exemestane is an anti-tumor drug used for postmenopausal advanced breast cancer patients whose condition has progressed after tamoxifen treatment.

My Exemestane

Risk of Insufficient Efficacy

EFFICACY : Patients with rs4646-AC genotype, compared to AA genotype, may have decreased drug response in pre-menopausal women and may have increased drug response in post-menopausal women; Monitoring of efficacy is required.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Adjunctive therapy for early invasive breast cancer in postmenopausal estrogen receptor-positive women after 2-3 years of adjuvant therapy with tamoxifen, up to a total of 5 years of adjuvant endocrine therapy.

Adverse reactions

It mainly includes nausea, dry mouth, constipation, diarrhea, dizziness, insomnia, rash, fatigue, fever, edema, pain, vomiting, abdominal pain, increased appetite, weight gain, etc. Hypertension, depression, anxiety, dyspnea and cough were also reported. Others include decreased lymphocyte count, abnormal liver function indexes (such as alanine transferase, etc.). In clinical trials, only 3% of patients discontinued treatment due to adverse events, mainly within the first 10 weeks of exemestane treatment: termination due to adverse events was uncommon (0.3%).

Contraindications

1. Patients allergic to this product or excipients in this product should not be allowed.
2. Pregnant women, lactating women and children are prohibited.

Pregnant and lactating women

Exemestane is contraindicated in pregnant or potential pregnant women. If the patient uses the drug during pregnancy or has pregnancy during the use of the drug, the patient should be informed of the potential harm of the drug to the fetus.

Children

Children are not allowed.

Elderly人

No special precautions.

* Metabolism*

It is mainly metabolized by cytochrome P4503A4 (CYP3A4), but the pharmacokinetics of exemestane did not change when used in combination with a potent CYP3A4 inhibitor (ketoconazole), so it appears that the CYP isozyme inhibitor has no significant effect on the pharmacokinetics of exemestane. However, the possibility that known CYP3A4 inducers reduce the level of exemestane in plasma cannot be ruled out.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4646	CYP19A1	AC	use of tamoxifen is less effective before menopause and more effective after menopause

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The exemestane drug response test item uses 1 polymorphic locus of the *CYP19A1* gene. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

 Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Asparaginase

Asparaginase is an enzyme that catalyzes the hydrolysis of asparagine to aspartic acid. It is clinically used in combination with other drugs to treat acute lymphoblastic leukemia (ALL) and certain lymphomas and mast cell tumors.

My Asparaginase

Use With Drug Labeling

ADVERSE EFFECT : Pediatric patients with rs738409-CC genotype, compared to CG or GG genotype, may have decreased risk of hepatotoxicity; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for the treatment of acute lymphocytic leukemia (ALL) , acute myeloid leukemia, acute monocytic leukemia, chronic lymphocytic leukemia, Hodgkin's disease and non-Hodgkin's lymphoma, melanoma, etc. This product has an inhibitory effect on the proliferation of the above-mentioned various tumor cells. Among them, it has the best effect on the induction of remission in children with ALL. Sometimes it may also be effective for patients who relapse after remission with some commonly used chemotherapeutic drugs, but separate application shorter remission,

and easy to produce drug resistance, so the more application form a coalition with other chemotherapy drugs, in order to improve curative effect.

Adverse reactions

It seems to be more common in adults than in children.

1. The more common ones are allergic reactions, liver damage, pancreatitis, anorexia, blood clotting factors V, VII, VIII, IX and fibrinogen decrease, etc. The main manifestations of allergic reactions are sudden onset of dyspnea, joint swelling and pain, skin rash, cutaneous pruritus, facial edema, severe respiratory distress, shock and even death may occur. The incidence of mild anaphylaxis is higher in patients with advanced childhood leukemia who are given intramuscular administration, but the incidence of severe anaphylaxis is reported to be lower than that of intravenous administration. Anaphylaxis generally occurs in patients with repeated injections, but has occurred in patients with negative intradermal sensitivity test (referred to as skin test). In addition, some allergic constitutions, even when injected skin test dose of asparaginase, will occasionally produce allergic reactions. Liver damage usually occurs within 2 weeks of starting treatment, and a variety of liver dysfunction may occur, including elevation of serum alanine aminotransferase [ALT (SGPT)], aspartate aminotransferase [AST (SGOT)], elevation of bilirubin, and decrease of serum albumin, there had cases with fatty liver lesions confirmed by liver biopsy. Patients with severe epigastric pain accompanied by nausea and vomiting should be suspected of acute pancreatitis, in which fulminant pancreatitis is very serious and may even be fatal. Other nausea, vomiting, diarrhea and so on.
2. The rare ones are hyperglycemia, hyperuricemia, high fever, mental and neurotoxicity. Patients with hyperglycemia have symptoms of polyuria, polydipsia, and thirst, and their plasma osmotic pressure may increase and blood ketone levels are normal. Hyperglycemia can be alleviated or disappeared by stopping this product, or giving an appropriate amount of insulin and rehydration, but a few serious cases can be fatal. Hyperuricemia often occurs at the beginning of treatment. Due to the rapid destruction of a large number of tumor cells, the amount of uric acid released by nucleic acid decomposition increases, which can seriously cause urinary acid nephropathy and renal failure. Mental and neurotoxicity manifests as different degrees of lethargy, mental depression, confusion, emotional agitation, hallucinations, and occasional Parkinson's syndrome. Others include leukopenia, immunosuppression, and stomatitis.
3. Rare cases include bleeding due to hypofibrinogenemia and reduction of coagulation factors, hypolipidemia, intracranial hemorrhage or thrombosis, venous thrombosis of the lower extremities, and bone marrow suppression. The reduction of coagulation factors is related to the inhibition of protein synthesis by this product.
4. Others: High blood ammonia, hair loss, thrombocytopenia, anemia, etc. It is necessary to pay attention to the adverse reactions and analyze their nature carefully. We should pay attention to the adverse reactions and analyze their nature carefully, anywhere may cause serious consequences, the use of this product should be stopped immediately, and combined with specific performance to the corresponding treatment measures, critical to active rescue.

Contraindications

1. Those who have a history of allergy to this product or a positive skin test.
2. Those who have a history of pancreatitis or are currently suffering from pancreatitis.
3. People who are currently suffering from serious infections such as chickenpox and widespread shingles.

Pregnant and lactating women

Since it cannot be ruled out that this product has potential teratogenic, mutagenic and secondary carcinogenic effects, pregnant women within 3 months of pregnancy should avoid using it. Due to the harm of this product to the baby, nursing mothers receiving treatment during breastfeeding should stop breastfeeding.

Children

The experiment has not been carried out and there is no reliable reference.

Elderly

The experiment has not been carried out and there is no reliable reference.

Metabolism

The binding rate of plasma protein is only about 30% when absorbed through muscle or vein, which can be detected in lymph after absorption, but the concentration in the cerebrospinal fluid is very low. After the injection of this product, the concentration of asparagine in the blood dropped to an undetectable level almost immediately, indicating that the product began to work soon after entering the body. The plasma T1/2 for intramuscular injection is 39 to 49 hours, and the plasma T1/2 for intravenous injection is 8 to 30 hours. The peak time after intramuscular injection is 12-24 hours, but 23-33 days after discontinuing this product, asparagine can be detected in plasma, and the excretion of this product seems to be biphasic, and only a small amount is present in the urine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS738409	PNPLA3	CC	normal risk of hepatotoxicity

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The asparaginase drug reaction test item uses a polymorphic locus of the *PNPLA3* gene. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

 Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Cyclophosphamide

Cyclophosphamide is the first broad-spectrum anti-tumor drug and is effective for both leukemia and solid tumors.

My Cyclophosphamide

Risk of Insufficient Efficacy

EFFICACY : Patients with rs4646-AC genotype, compared to AA genotype, may have decreased drug response in pre-menopausal women and may have increased drug response in post-menopausal women; Monitoring of efficacy is required. Patients with rs4880-AG genotype may have a decreased survival rate as compared to AA genotype, or an increased survival rate as compared to GG genotype; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

This product is currently a widely used anti-cancer drug. It is effective in treating malignant lymphoma, acute or chronic lymphocytic leukemia and multiple myeloma, and has certain curative

effects on breast cancer, testicular tumor, ovarian cancer, lung cancer, head and neck squamous cell carcinoma, nasopharyngeal carcinoma, neuroblastoma, rhabdomyosarcoma and osteosarcoma.

Adverse reactions

1. Myelosuppression: Leukopenia is the most common, the lowest value appearing in 1 to 2 weeks after medication, and usually recovers after 2 to 3 weeks. Less effect on platelets. May also affect liver function.
2. Gastrointestinal reactions: including anorexia, nausea and vomiting, which can disappear after stopping the drug for 1~3 days.
3. Urinary tract reaction: Hemorrhagic cystitis, manifested as bladder irritation symptoms, oliguria, hematuria and proteinuria, is caused by its metabolite acrolein stimulation of the bladder, but cyclophosphamide routine dose application, its incidence is low.
4. Other reactions include hair loss, stomatitis, toxic hepatitis, skin pigmentation, menstrual disorders, azoospermia or reduced sperm, and pulmonary fibrosis.

Contraindications

1. Anticancer drugs must be administered under the guidance of experienced specialists. Those with myelosuppression, infection, liver and kidney function damage should not be used with caution.
2. People allergic to this product are not allowed.
3. Pregnant and lactation women are forbidden.

Pregnant and lactating women

It has mutagenic and teratogenic effects, and can cause fetal death or congenital malformations. It is forbidden for pregnant women. This product can be excreted in breast milk. Breastfeeding must be stopped when the medication is started.

Children

The test has not been carried out and there is no reliable reference.

Elderly

The test has not been carried out and there is no reliable reference.

Metabolism

Phosphoramidate mustard is transformed and released in the liver, and about 50% of its metabolites are combined with protein.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1695	GSTP1	AG	stronger drug response, decreased drug toxicity
RS4646	CYP19A1	AC	may decrease premenopausal efficacy and increase postmenopausal efficacy
RS4880	SOD2	AG	higher survival rate after treatment
RS738409	PNPLA3	CC	decreased risk of hepatotoxicity

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The cyclophosphamide drug response test item We uses 4 polymorphic loci of *GSTP1*, *CYP19A1*, *SOD2*, and *PNPLA3* genes. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Epirubicin

Epirubicin is a cell cycle non-specific drug, and its main site of action is the nucleus. The mechanism of epirubicin is related to its ability to bind to DNA. Cell culture studies have shown that epirubicin can quickly penetrate into cells, enter the nucleus and bind to DNA, thereby inhibiting nucleic acid synthesis and mitosis.

My Epirubicin

Risk of Insufficient Efficacy

EFFICACY : Patients with rs4646-AC genotype, compared to AA genotype, may have decreased drug response in pre-menopausal women and may have increased drug response in post-menopausal women; Monitoring of efficacy is required.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is used to treat malignant lymphoma, breast cancer, lung cancer, soft tissue sarcoma, esophageal cancer, gastric cancer, liver cancer, pancreatic cancer, melanoma, colorectal cancer, ovarian cancer,

multiple myeloma, leukemia, etc.

Adverse reactions

1. Similar to doxorubicin, but to a lower degree, especially cardiotoxicity and myelosuppressive toxicity.
2. Other adverse reactions include: Alopecia, which can occur in 60-90% of cases, and is generally reversible, and inhibition of beard growth in men; mucositis, which appears on the 5th to 10th day of medication, usually occurs on the tongue and sublingual mucosa; gastrointestinal dysfunction, such as nausea, vomiting, diarrhea; occasional fever, chills, urticaria, hyperpigmentation, and joint pain have been reported.

Contraindications

1. Peripheral blood has less than 3,500 leukocyte or less than 50,000 platelets.
2. Patients with severe myocardial insufficiency, recent myocardial infarction, and severe arrhythmia.
3. Patients who have taken sufficient doses of daunorubicin or doxorubicin (total dose $\geq 400 \sim 500\text{mg/m}^2$) or have allergic reactions to these two drugs.
4. Patients with fever or severe infection, cachexia, dehydration, electrolyte or acid-base imbalance, gastrointestinal obstruction.
5. Patients with cardiopulmonary, liver and kidney function decompensated.

Pregnant and lactating women

This product can penetrate the placenta and can cause damage to the fetus of pregnant women. Therefore, this product is forbidden during the first 3 months of pregnancy. Women of the right age are advised to avoid pregnancy when using this product for treatment. Lactating women should stop breastfeeding before taking this medicine.

Children

The dosage for children is about 1/3 to 1/2 of that for adults, but the pharmacokinetic properties of this product for children have not been studied, and children under 2 years of age should use this product with caution.

Elderly

A certain amount of this product is excreted by the kidneys, and elderly patients should reduce the dose when using this product.

Metabolism

The main metabolite of epirubicin is a 13-hydroxy derivative. Epirubicin is mainly metabolized in the liver, eliminated by bile, and a small amount is excreted by the kidneys.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1695	GSTP1	AG	stronger drug response, decreased drug toxicity
RS4646	CYP19A1	AC	decreases premenopausal efficacy and increases postmenopausal efficacy

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The epirubicin drug response test item uses 2 polymorphic loci of *GSTP1* and *CYP19A1* genes. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Doxorubicin

Doxorubicin can directly act on DNA by inserting the double helix of DNA to unwind it, thereby changing the properties of the DNA template. Doxorubicin inhibits DNA polymerase to inhibit both DNA and RNA synthesis. It is a cycle non-specific anti-cancer chemotherapy drug.

My Doxorubicin

Use With Drug Labeling

ADVERSE EFFECT : For pediatric malignancy treatment, possibility of anthracyclines-related cardiotoxicity is relatively low, but constant monitoring is required.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Suitable for acute leukemia (lymphocytic and myelogenous), malignant lymphoma, breast cancer, lung cancer (small cell and non-small cell lung cancer), ovarian cancer, bone and soft tissue sarcoma, Wilms tumor, neuroblastoma, bladder cancer, thyroid cancer, prostate cancer, head and neck squamous cell carcinoma, testicular cancer, gastric cancer, liver cancer, etc.

Adverse reactions

1. Myelosuppression: It is the main side effect of doxorubicin. The leucocyte drops to its lowest point about 10-14 days after administration and gradually returns to normal level within 3 weeks. Anemia and thrombocytopenia are generally not serious.
2. Cardiotoxicity: Transient electrocardiogram changes may occur, manifested as supraventricular tachycardia, ventricular premature contraction and ST-T changes, generally do not affect the treatment, a small number of patients may have delayed, progressive cardiomyopathy, manifested as acute congestive heart failure, and the cumulative dose is closely related, mostly in patients with a total amount >400mg/m². These conditions may occasionally occur suddenly with no signs of abnormality in routine electrocardiogram. The cardiac lesions caused by doxorubicin usually appear 1 to 6 months after discontinuation of doxorubicin. Cardiotoxicity may be aggravated by the combination of other drugs.
3. Gastrointestinal reactions: manifested as anorexia, nausea, vomiting, and oral mucosal erythema, ulcers, esophagitis, and gastritis.
4. Alopecia: The incidence rate is more than 90%, and the growth can be restored after 1-2 months of drug withdrawal.
5. Local reaction: For example, the extravasation of the drug at the injection site can cause tissue ulcers and necrosis. Excessive drug concentration can cause phlebitis.
6. Others: A small number of patients have fever, hemorrhagic erythema, abnormal liver function and proteinuria, pigmentation at the nail bed, onycholysis, and skin redness or pigmentation in the original radiation field. Individual patients have urticaria, allergic reactions, conjunctivitis, and tearing. In addition, doxorubicin can increase the toxicity of radiotherapy and some anticancer drugs.
7. When used in patients with leukemia and malignant lymphoma, especially in those who take doxorubicin for the first time, hyperuricemia may be caused by massive destruction of tumor cells, resulting in joint pain or renal impairment.

Contraindications

1. Patients who peripheral blood images of leukocyte less than 3500/ μ L or platelet less than 50,000 / μ L are prohibited.
2. Obvious infection or fever, cachexia, dehydration, electrolyte or acid-base imbalance should not be used.
3. People with gastrointestinal obstruction, obvious jaundice or liver damage are contraindicated.
4. It is forbidden for patients with cardiopulmonary decompensation, chickenpox or herpes zoster.
5. Patients who have used other anti-tumor drugs or radiotherapy have caused myelosuppression are contraindicated.
6. Patients with severe heart disease are prohibited.

7. It is forbidden for pregnant and lactating women.



Pregnant and lactating women

Doxorubicin can penetrate the placenta and may cause miscarriage. Therefore, it is strictly forbidden to use doxorubicin during the first 3 months of pregnancy. After pregnant women use this product, the toxic reaction to the fetus can sometimes appear several years later, and it is contraindicated by lactating women.



Children

Children under 2 years old should be used with caution.



Elderly

Use with caution in elderly patients.



Metabolism

Doxorubicin is mainly metabolized in the liver, excreted by bile, 50% is excreted in its original form, 23% is excreted as the active doxorubicin metabolite, adriamitol, and only 5%-10% is excreted in urine within 6 hours.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS17863783	UGT1A6	GG	less likely to develop anthracycline-related cardiotoxicity
RS7853758	SLC28A3	GG	less likely to develop anthracycline-related cardiotoxicity
RS2229774	RARG	GG	less likely to develop anthracycline-related cardiotoxicity

How to use the test results



Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The doxorubicin drug response test item uses 3 polymorphic loci of *UGT1A6*, *RARG* and *SLC28A3* genes. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.

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3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Daunorubicin

Daunorubicin, also known as daunomycin, is a chemotherapeutic drug belonging to the class of anthracycline antibiotics. It is used to treat acute myeloid leukemia (AML), acute lymphocytic leukemia (ALL), and chronic myeloid leukemia (CGL) and Kaposi's sarcoma and other types of cancer.

My Daunorubicin

Use With Drug Labeling

ADVERSE EFFECT : Possibility of anthracyclines-related cardiotoxicity is low; Constant monitoring is recommended.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is used for acute myeloid leukemia, acute lymphocytic leukemia and chronic acute change.

Adverse reactions

1. Myelosuppression: more serious. Anemia, neutropenia, thrombocytopenia, bleeding, do not use the medicine for too long; if oral ulcers occur (mostly appear before myelosuppression), the medicine should be stopped immediately.

2. **Cardiotoxicity:** It can cause abnormal electrocardiogram, tachycardia and arrhythmia; severe cases may have heart failure. Severe myocardial damage can occur when the total dose exceeds 25 mg/kg, and arrhythmia can also occur when the intravenous injection is too fast.
3. **Gastrointestinal reactions:** Ulcerative stomatitis, loss of appetite, nausea, vomiting, abdominal pain, etc.
4. **Liver and kidney damage:** Elevated GOT, GPT, ALP, jaundice, elevated BUN, proteinuria.
5. **Local reaction:** Leakage outside the blood vessel can cause local tissue necrosis.
6. **Others:** Alopecia, fatigue, headache, dizziness and other mental symptoms, chills, dyspnea, fever, skin rash and other allergic symptoms.

Contraindications

1. It is forbidden for patients with heart disease and those with a history of heart disease.
2. Patients with a history of severe allergies to this drug are contraindicated.
3. It is forbidden for pregnant and lactating women.

Pregnant and lactating women

Prohibited to use. There are reports of teratogenicity in animal experiments (mice). It is contraindicated for pregnant women or women who may become pregnant. Avoid medication to lactating women, and stop breastfeeding when it is necessary (the safety of medication during breastfeeding has not been determined).

Children

When children take medicine, special attention should be paid to the occurrence of adverse reactions and use with caution. In children and patients of reproductive age, if medication must be given, the effect on the gonads should be considered.

Elderly

This medicine is mainly metabolized by the liver, and physiological functions such as liver function decline in elderly patients, so use it with caution.

Metabolism

Mainly metabolized by the liver, the main metabolite in the human body is daunorubicin alcohol.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS17863783	UGT1A6	GG	less likely to develop anthracycline-related cardiotoxicity
RS7853758	SLC28A3	GG	less likely to develop anthracycline-related cardiotoxicity
RS2229774	RARG	GG	less likely to develop anthracycline-related cardiotoxicity

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The daunorubicin drug response test item uses 3 polymorphic loci of *UGT1A6*, *RARG*, and *SLC28A3* genes. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Docetaxel

Docetaxel is an anti-tumor drug of the taxol class, which plays an anti-tumor effect by interfering with the microtubule network necessary for cell mitosis and interphase cell function.

My Docetaxel

Risk of Insufficient Efficacy

EFFICACY : Patients with rs4646-AC genotype, compared to AA genotype, may have decreased drug response in pre-menopausal women and may have increased drug response in post-menopausal women; Monitoring of efficacy is required.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is used for the treatment of advanced or metastatic breast cancer that has failed prior chemotherapy. Unless it is a clinical contraindication, the initial treatment should include anthracycline anticancer drugs.

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2. It is used for the treatment of advanced or metastatic non-small cell lung cancer that has failed cisplatin-based chemotherapy.



Adverse reactions

1. Myelosuppression: Neutropenia is the most common side effect and is usually serious (less than $500/\text{mm}^3$). Reversible and does not accumulate.
2. Allergic reactions: Severe allergic reactions may occur in some cases, which are characterized by hypotension and bronchospasm, requiring interruption of treatment. The patient will return to normal after cessation of infusion and immediate treatment. Mild allergic reactions may also occur in some cases, such as blushing, erythema with or without itching, chest tightness, back pain, dyspnea, medication fever, or chills.
3. Skin reactions are often manifested as erythema, mainly seen on the hands and feet, but also as a local rash on the buttocks, face and chest, sometimes accompanied by itching. The rash may usually occur within a week after the docetaxel infusion, but it can be recovered before the next infusion. Severe symptoms, such as peeling after a rash, rarely occur. Nail lesions may occur, which are characterized by hyperpigmentation or lightening, sometimes pain and fingernail shedding.
4. Fluid retention includes edema. There are also very few reports of pleural effusion, ascites, pericardial effusion, increased capillary permeability, and weight gain. After 4 weeks of treatment or a cumulative dose of $400\text{mg}/\text{m}^2$, fluid retention occurs in the lower extremities, and may develop to systemic edema, while gaining weight of 3 kg or more. After stopping docetaxel treatment, fluid retention gradually disappeared. In order to reduce fluid retention, patients should be given prophylactic corticosteroids.
5. Gastrointestinal reactions such as nausea, vomiting or diarrhea may occur.
6. There have been reports of neurotoxicity in clinical trials.
7. Cardiovascular side effects such as hypotension, sinus tachycardia, palpitations, pulmonary edema and hypertension may occur.
8. Other side effects include: hair loss, weakness, mucositis, arthralgia and muscle pain, hypotension and injection site reactions.
9. Patients with normal liver function also have elevated aminotransferase and elevated bilirubin during the treatment period, and its relationship with docetaxel is not clear.



Contraindications

1. Patients with a history of severe allergies to docetaxel or Tween-80.
2. Patients whose leukocyte count is less than $1500/\text{mm}^3$.
3. Patients with severely impaired liver function.



Pregnant and lactating women

There is no data on the use of docetaxel in pregnant women. Docetaxel shows embryonic and fetal toxicity in rabbits and mice, and reduces its fertility ability in mice. Like other cytotoxic drugs, docetaxel may be harmful to the fetus when used by pregnant women. Therefore, docetaxel cannot be used in pregnant women. Women of childbearing age should be warned to avoid pregnancy when receiving docetaxel treatment, and immediately notify the treating doctor once they become pregnant. Docetaxel is a lipophilic substance, but it is not known whether it can be excreted from human milk. Moreover, due to its potential adverse reactions to nursing infants, breastfeeding should be stopped during docetaxel treatment.

Children

The effectiveness and safety of docetaxel in children have not been determined.

Elderly

According to the results of population pharmacokinetic data, there is no special guidance on medication for the elderly. There is no data on the use of docetaxel combined with doxorubicin and cyclophosphamide in patients over 70 years of age.

Metabolism

Docetaxel and its metabolites are mainly excreted from feces. The amount excreted through feces and urine accounted for about 75% and 6% of the given dose respectively, and only a small part was excreted in its original form. In vitro studies have shown that docetaxel is metabolized by CYP3A4 isoenzymes, and this metabolism can be inhibited by CYP3A4 inhibitors.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4646	CYP19A1	AC	decreases premenopausal efficacy and increases postmenopausal efficacy

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The docetaxel drug response test item uses a polymorphic locus of the *CYP19A1* gene. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.

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3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Etoposide

Etoposide is a cell cycle specific anti-tumor drug that acts on DNA topoisomerase II and plays an anti-tumor effect by hindering DNA repair.

My Etoposide

Use With Drug Labeling

ADVERSE EFFECT : Patients with rs716274-AA genotype, compared to AG or GG genotype, may have decreased risk of death; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is mainly used to treatment of small cell lung cancer, malignant lymphoma, malignant germ cell tumor, leukemia, and has a certain effect on neuroblastoma, rhabdomyosarcoma, ovarian cancer, non-small cell lung cancer, gastric cancer and esophageal cancer.

Adverse reactions

Common adverse reactions include alopecia, anorexia, nausea, vomiting, palpitations, dizziness, hypotension, phlebitis, and myelosuppression. There are leucopenia and thrombocytopenia.



Contraindications

1. Patients who are allergic to this product, pregnant and lactating women should not be used.
2. Patients who are myelosuppression, significantly lower leukocytes and platelets are prohibited.
3. It is forbidden for those with serious impairment of heart, liver and kidney function.



Pregnant and lactating women

It is forbidden for pregnant and lactating women.



Children

The test has not been carried out and there is no reliable reference, and it is not clear.



Elderly

The test has not been carried out and there is no reliable reference, and it is not clear.



Metabolism

After oral absorption, the peak blood concentration is reached within 0.5 to 4 hours, and the bioavailability is 50%. It is mainly distributed in bile, ascites, urine, pleural fluid and lung tissue, and rarely enters the cerebrospinal fluid. It is mainly excreted in the urine in its original form and metabolites.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS716274	intergenic	AA	may have decreased risk of death

How to use the test results



Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.



Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The etoposide drug response test item uses a polymorphic locus in the intergenic region. The test results explain the genotype results of the user's variations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the

item may be affected.

4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Vincristine

Vincristine is an alkaloid extracted from the Apocynaceae plant *Catharanthus roseus*. It is an anti-tumor drug used for the treatment of acute lymphoblastic leukemia, lymphoma and breast cancer.

My Vincristine

Use With Drug Labeling

ADVERSE EFFECT : Patients have a decreased risk of hepatotoxicity and peripheral nervous system disease; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for acute leukemia, especially childhood acute leukemia, and has a significant effect on acute lymphoblastic leukemia.
2. Suitable for malignant lymphoma.
3. Suitable for germ cell tumors.
4. Suitable for small cell lung cancer, Ewing's sarcoma, nephroblastoma, neuroblastoma.

-
5. Suitable for breast cancer, chronic lymphocytic leukemia, gastrointestinal cancer, melanoma and multiple myeloma, etc.

Adverse reactions

1. Dose-limiting toxicity is the toxicity of the nervous system, which mainly causes peripheral nerve symptoms, such as numbness of fingers and neurotoxicity, etc., which are related to the cumulative amount. It is characterized by numbness of toes, dull or disappearance of tendon reflex and peripheral neuritis. Abdominal pain, constipation, and paralytic intestinal obstruction are rare occasionally. Motor nerve, sensory nerve and cranial nerve can also be damaged and produce corresponding symptoms. Neurotoxicity often occurs in people over 40 years old. The tolerance of children is better than that of adults. The tendency of neurotoxicity in patients with malignant lymphoma is higher than that in patients with other tumors.
2. Bone marrow suppression and gastrointestinal reactions are mild.
3. It can stimulate local tissue, and the drug solution cannot leak out, otherwise it may cause local necrosis.
4. Hair loss can be seen, occasionally changes in blood pressure.

Contraindications

1. It is forbidden for pregnant women.

Pregnant and lactating women

It is forbidden for pregnant women. Lactating women should weigh the importance of the drug to the mother and the damage to the fetus, and decide whether to stop lactating or stop the administration.

Children

The myelination of peripheral nerve in children under 2 years old is not perfect, so it should be used with caution.

Elderly

Not yet clear.

Metabolism

After intravenous injection of vincristine, it quickly distributes in various tissues. The concentration in nerve cells is relatively high, and it rarely penetrates the blood-brain barrier. The concentration of cerebrospinal fluid is $1/30 \sim 1/20$ of the plasma concentration. The protein binding rate is 75%. In adults, $t_{1/2\alpha}$ is less than 5 minutes, $t_{1/2\beta}$ is 50 to 155 minutes, and the terminal elimination phase $t_{1/2\gamma}$ is up to 85 hours. It is metabolized in the liver and has the highest concentration in bile. It is mainly excreted with bile, with 70% excretion in feces and 5% to 16% in urine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS738409	PNPLA3	CC	normal risk of hepatotoxicity
RS924607	intergenic	CC	normal risk of peripheral nervous disease

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug

response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The vincristine drug reaction test item uses the *PNPLA3* gene and 2 polymorphic loci in the intergenic region. The test results indicate the genotype results of the user's mutations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Paclitaxel

Paclitaxel is a diterpene alkaloid compound isolated and purified from the bark of the gymnosperm yew. It is the most excellent natural anti-cancer drug that has been discovered, and it has been widely used clinically in the treatment of breast cancer, ovarian cancer, some head and neck cancers and lung cancer.

My Paclitaxel

Risk of Insufficient Efficacy

EFFICACY : Patients with rs4646-AC genotype, compared to AA genotype, may have decreased drug response in pre-menopausal women and may have increased drug response in post-menopausal women; Monitoring of efficacy is required.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. For the first-line and second-line treatment of ovarian cancer, breast cancer and non-small cell lung cancer NSCLC (Non-Small-Cell-Lung-Cancer).
2. For head and neck cancer, esophageal cancer, seminoma, recurring non-Hoggin's lymphoma, etc.

Adverse reactions

1. Allergic reactions: The incidence rate is 39%, of which the incidence of severe allergic reactions is 2%. Most of them are type 1 allergies, manifested as bronchospasm dyspnea, urticaria and hypotension. Almost all reactions occur within the first 10 minutes after the medication.
2. Bone marrow suppression: It is the main dose-limiting toxicity, which is manifested as neutropenia and thrombocytopenia. It is rare and usually occurs 8-10 days after medication. The incidence of severe neutropenia was 47%, and the incidence of severe thrombocytopenia was 5%. Anemia is more common.
3. Neurotoxicity: The incidence of peripheral neuropathy is 62%, the most common manifestations are mild numbness and paresthesia, and the incidence of severe neurotoxicity is 6%.
4. Cardiovascular toxicity: There may be hypotension and asymptomatic short-term bradycardia. The incidence of muscle and joint pain is 55%, it occurs in the limbs and joints. The incidence and severity are dose-dependent.
5. Gastrointestinal reactions: The incidences of nausea, vomiting, diarrhea and mucositis were 59%, 43% and 39%, respectively. They were generally mild and moderate.
6. Liver toxicity: elevated ALT, AST and AKP.
7. Hair loss: The incidence rate is 80%.
8. Local reaction: Inflammation of the intravenous infusion of the drug and the local inflammation of the drug extravasation.

Contraindications

1. Paclitaxel is forbidden to people with a history of allergic reactions to paclitaxel or other drugs formulated with CremophorEL (polyoxyethyl castor oil).
2. For patients with solid tumors whose baseline neutrophil count is less than 1500/mm³, or patients with AIDS-related Kaposi's sarcoma whose baseline neutrophil count is less than 1000/mm³, paclitaxel cannot be used.

Pregnant and lactating women

Paclitaxel has been proved to affect embryo growth in animal experiments, so it is forbidden for pregnant women. Women of childbearing age should not be pregnant during treatment.

Children

The safety and efficacy of paclitaxel in children have not been established.

Elderly

There is no experience in elderly medication. Please refer to the adult dosage or follow the doctor's advice.

Metabolism

It is mainly metabolized in the liver, enters the intestine with bile, and is excreted through feces (>90%). Renal clearance only accounts for 1% to 8% of the total clearance. The metabolism of paclitaxel in patients with liver and kidney dysfunction is still unclear.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4646	CYP19A1	AC	use of tamoxifen is less effective before menopause and more effective after menopause

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be

sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The paclitaxel drug reaction test item uses 1 polymorphic locus of the *CYP19A1* gene. The test results indicate the genotype results of the user's mutations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Cyclosporine

Cyclosporine is suitable for the prevention of rejection of allogeneic kidney, liver, heart, bone marrow and other organs or tissue transplantation, and the prevention and treatment of graft-versus-host reaction that occurs during bone marrow transplantation.

My Cyclosporine

Consider Dose Increase

DOSE : Patients with rs776746-CT genotype, compared to CC genotype, may require an increased dose drug to achieve target plasma concentration.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for preventing rejection of allogeneic kidney, liver, heart, bone marrow and other organs or tissues.
2. It is also suitable for the prevention and treatment of graft-versus-host reaction during bone marrow transplantation. This product is often combined with adrenal cortex hormones and other

immunosuppressive agents to improve the efficacy.

3. In recent years, there have been reports of trial use in the treatment of uveitis, severe aplastic anemia, refractory autoimmune thrombocytopenic purpura, psoriasis, refractory lupus nephritis, etc.

Adverse reactions

1. The more common gastrointestinal reactions such as anorexia, nausea, and vomiting, gingival hyperplasia with bleeding and pain, about 1/3 of the drug users have nephrotoxicity, increased serum creatinine, urea nitrogen, and decreased glomerular filtration rate such as renal damage, high blood pressure and so on. Gingival hyperplasia generally disappears after 6 months of stopping the drug. Chronic and progressive nephrotoxicity occurs more than about 12 months after treatment.
2. Convulsion is uncommon, which may be related to the toxicity of this product to kidney and hypomagnesemia. In addition, this product can also cause aminotransferase elevation, cholestasis, hyperbilirubinemia, hyperglycemia, hirsutism, hand tremor, hyperuricemia with thrombocytopenia, microvascular hemolytic anemia, limb paresthesia, lower limb painful spasm, etc. In addition, it has been reported that this product can promote ADP-induced platelet aggregation, increase the release of thromboxane A₂ and the production of thromboplastin, enhance the activity of factor VII, reduce the production of prostacyclin, and induce thrombosis.
3. Rare allergic reactions, pancreatitis, leukopenia, Raynaud syndrome, diabetes, hematuria and so on. Severe adverse reactions are mostly related to excessive dosage. The method to prevent the reaction is to monitor the blood concentration of the product frequently, adjust the whole blood concentration of the product, so that it can be maintained the range that can play an immunosuppressive role in clinic without serious adverse reactions. In case of adverse reactions, corresponding treatment should be given immediately, and the dosage of this product should be reduced or stopped.

Contraindications

1. It is forbidden to use this product in case of virus infection, such as chickenpox, herpes zoster, etc.
2. It is forbidden for those who are allergic to this product.

Pregnant and lactating women

This product can enter breast milk. Lactating infants may have potential risks of adverse effects such as high blood pressure, nephrotoxicity, malignant tumors, etc., so it is not suitable for lactating during the use of this product.

Children

Common doses for children: The initial dose of organ transplantation is 6-11 mg/kg per day according to body weight, and the maintenance dose is 2-6 mg/kg per day.

Elderly

Elderly patients are prone to have renal insufficiency, so this product should be used with caution.

Metabolism

This product is metabolized by the liver and excreted in the feces via the biliary tract. Only 6% is excreted via the kidneys, of which about 0.1% is still excreted in its original form.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS776746	CYP3A5	CT	higher dose of drug is required

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only

and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The cyclosporine drug reaction test item uses 1 polymorphic locus of the *CYP3A5* gene. The test results indicate the genotype results of the user's mutations detected above, and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Sirolimus

Sirolimus is a new type of macrolide immunosuppressant used to prevent rejection of transplanted organs.

My Sirolimus

Consider Dose Increase

DOSE : Patients with rs776746-CT genotype, compared to CC genotype, may have increased metabolism of sirolimus, may require increased dose of drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for patients 13 years of age or older who have undergone kidney transplantation to prevent organ rejection. It is recommended that sirolimus be used in combination with cyclosporine and corticosteroids. It is recommended to monitor the blood concentration of the therapeutic drug in all patients receiving sirolimus treatment.

Adverse reactions

The most common adverse reactions (occurring in >10% of patients) are thrombocytopenia, anemia, fever, hypertension, hypokalemia, hypophosphatemia, urinary tract infection, hypercholesterolemia, hyperglycemia, hypertriglyceridemia, abdominal pain, lymphatic cyst, peripheral edema, arthralgia, acne, diarrhea, pain, constipation, nausea, headache, increased blood creatinine level and increased blood lactate dehydrogenase level (LDH).

Contraindications

1. This product is contraindicated in patients who are allergic to sirolimus and sirolimus derivatives.
2. It has an immunosuppressive effect and is not recommended for liver or lung transplant patients.

Pregnant and lactating women

The safety has not been established, pregnant and lactating women should use it with caution.

Children

The safety and efficacy of sirolimus for children under 13 years of age have not been determined.

Elderly

In clinical trials of sirolimus tablets, a sufficient number of patients 65 years of age and older were not included to determine whether the safety and efficacy of this group of drugs are different from those of younger patients. The trough blood concentration data of sirolimus suggest that for elderly kidney transplant patients, there is no need to adjust the dose according to age. The difference in response between young and elderly patients has not been identified. Generally speaking, the dose selection for elderly patients should be cautious, considering the weakening of liver function and heart function in the elderly or other comorbidities or the high frequency of co-administration of other drugs, and treatment is usually started from the low end of the dose range.

Metabolism

Sirolimus is a substrate of CYP3A4 and P-gp. Sirolimus can be extensively metabolized by CYP3A4 isoenzymes in the intestinal wall and liver, and can be reverse transported from the small intestinal epithelial cells to the intestinal lumen by the P-gp drug outflow pump. Inhibitors of CYP3A4 and P-gp can increase the concentration of sirolimus. Inducers of CYP3A4 and P-gp can reduce the concentration of sirolimus. Sirolimus is extensively metabolized via O-demethylation and/or hydroxylation. Seven major metabolites can be detected in whole blood, including hydroxylation, demethylation and hydroxyl demethylation metabolites. Some of them can also be detected in plasma, feces and urine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS776746	CYP3A5	CT	higher dose of drug is required

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The sirolimus drug response test item uses 1 polymorphic locus of *CYP3A5* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Triamcinolone

Triamcinolone is a long-acting synthetic corticosteroid, it can be used to treat eczema, psoriasis, arthritis, allergies, ulcerative colitis, lupus erythematosus, sympathetic ophthalmritis, giant cell arteritis, uveitis, eye Inflammation, imaging during vitrectomy and prevention of asthma.

My Triamcinolone

Risk of Insufficient Efficacy

EFFICACY : Patients with rs1876828-CC genotype, compared to TT genotype, may have a decreased drug response; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for connective tissue diseases such as systemic lupus erythematosus.
2. It is suitable for immune kidney diseases such as nephrotic syndrome.
3. It is suitable for immune diseases such as idiopathic thrombocytopenic purpura.
4. Applicable to other diseases applicable to prednisone acetate.

Adverse reactions

Similar to other glucocorticoids, such as:

1. Gastrointestinal reactions: upper abdominal discomfort, abdominal distension, anorexia, nausea, a small number of patients may induce gastric and duodenal ulcers and even bleeding and perforation.
2. Endocrine and metabolic abnormalities: Long-term application can induce endocrine abnormalities (such as steroid diabetes, irregular menstruation, growth inhibition in children); electrolyte disturbances; cushing's syndrome: abnormal hair distribution, full moon face, buffalo back, skin purple streaks, muscle atrophy, hypertension, hypokalemic alkalosis; osteoporosis; muscle weakness.
3. Neuropsychiatric disorders: excitement, insomnia, mild depression, drowsiness, a few can induce headaches, increased intracranial pressure, convulsions or mental disorders.
4. Ophthalmic complications: subcorneal cataract, elevated intraocular pressure and glaucoma.

Contraindications

1. All kinds of bacterial infections and systemic fungal infections are prohibited.
2. People who are allergic to triamcinolone should not be used.

Pregnant and lactating women

Use with caution in pregnant and lactating women.

Children

This product is not suitable for children, and should only be used for short-term treatment when necessary.

Elderly

Use with caution.

Metabolism

The oral absorption rate of glucocorticoid is directly proportional to its fat solubility, while the absorption rate of injection administration is proportional to its water solubility. About 90% of glucocorticoids bind to plasma proteins without biological activity, and less than 10% have biological activity. Its distribution in the body is not selective, but due to increased blood flow and vascular permeability, the drug concentration in the inflammation site can be higher than that in other organs. Glucocorticoids are mainly inactivated in the liver, and lymphocytes also have the ability to oxidize or reduce glucocorticoids. Glucocorticoids are excreted by the kidneys, the free cortisone in the urine is less than 2%, and more than 90% is inactivated, indicating that the level of active hormones in the plasma will not increase in the event of renal insufficiency.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1876828	CRHR1	CC	slightly weak response of triamcinolone

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug

response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The triamcinolone drug response test item uses 1 polymorphic locus of *CRHR1* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Lamotrigine

Lamotrigine is an antiepileptic drug.

My Lamotrigine

Risk of Insufficient Efficacy

EFFICACY : Patients with rs2011425-GT genotype, compared to TT genotype, may have decreased plasma concentration of lamotrigine and decreased drug response; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is suitable for epilepsy.

Adverse reactions

1. Very common: skin rash.
2. Rare: Stevens-Johnson syndrome.
3. Very rare: toxic epidermal necrosis and dissolution sign.



Contraindications

It is contraindicated in patients who are known to be allergic to lamotrigine.



Pregnant and lactating women

Lamotrigine should not be used by pregnant women. When it is necessary to use it, the pros and cons should be fully weighed. The potential benefits of breastfeeding should outweigh the risk of potential adverse reactions to the baby.



Children

Still uncertain.



Elderly

The pharmacokinetics of lamotrigine in the elderly are not significantly different from those in the young, so there is no need to adjust the recommended dose.



Metabolism

Lamotrigine's clearance is mainly metabolized into glucuronic acid conjugates, and then excreted in the urine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2011425	UGT1A4	GT	the serum concentration of lamotrigine is low and the drug response is slightly decreased

How to use the test results



Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.



Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The lamotrigine drug response test item uses 1 polymorphic locus of *UGT1A4* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
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3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the

item may be affected.

4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Amisulpride

Amisulpride can selectively bind to the D2 and D3 dopaminergic receptors of the limbic system, and is a psychosedative that replaces aniline.

My Amisulpride

Use With Drug Labeling

ADVERSE EFFECT : Patients with rs489693-CC genotype, compared to AA genotype, may be at a decreased risk of experiencing adverse effect, e.g., weight gain and hypertriglyceridemia; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is used to treat acute or chronic schizophrenia with positive symptoms (such as delirium, hallucinations, cognitive impairment) and/or negative symptoms (such as slow response, apathy, and social withdrawal), as well as schizophrenia characterized by negative symptoms.

Adverse reactions

1. Frequent adverse reactions: increased prolactin level in blood, weight gain, extrapyramidal syndrome (tremor, hypertonia, salivation, akathisia, hypokinesia).
2. Rarely occurring adverse reactions: lethargy, gastrointestinal dysfunction (constipation, nausea, vomiting, dry mouth).
3. Rarely occurring adverse reactions: acute dystonia (spastic torticollis, eye rotation crisis, closed teeth and other symptoms).
4. Others: tardive dyskinesia, hypotension and bradycardia, prolonged QT interval, torsade de pointes ventricular tachycardia, convulsions, malignant syndrome.

Contraindications

1. People who are known to be allergic to a certain ingredient in drugs should not be used.
2. Disabled for patients with pheochromocytoma.
3. People with prolactin-dependent tumors, pituitary prolactinoma and breast cancer are prohibited.
4. People with severe kidney damage (creatinine clearance rate 10ml/min) are forbidden.

Pregnant and lactating women

Unless the benefits outweigh the potential risks, it is not recommended to use this product during pregnancy. As there is no information on whether the drug is secreted through breast milk, it is forbidden to take this drug during breastfeeding.

Children

It is not recommended to use amisulpride in adolescents from adolescence to 18 years old; amisulpride is contraindicated in children before puberty.

Elderly

Due to the high sensitivity of the elderly to drugs (which can produce symptoms of sedation or hypotension), the elderly should pay special attention when taking drugs.

Metabolism

Amisulpride is rarely metabolized by the liver, but mainly by the kidneys, and most of it is excreted in the urine in its original form.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description

RS489693

intergenic

CC

weight gain and reduced risk of hypertriglyceridemia

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The amisulpride drug response test item uses 1 polymorphic locus of *MC4R* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Olanzapine

Olanzapine is an antipsychotic drug that acts on multiple receptor systems and has a wide range of pharmacological activities.

My Olanzapine

Use With Drug Labeling

ADVERSE EFFECT : Patientis have lower level of prolactin and lower risk of metabolic syndrome, weight gain and hypertriglyceridemia when treated with olanzapine; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Used to treat schizophrenia.
2. For patients whose initial treatment with olanzapine is effective, consolidation therapy can effectively maintain the improvement of clinical symptoms.
3. For the treatment of moderate and severe manic episodes.
4. Olanzapine can prevent the recurrence of bipolar disorder in patients with manic episodes who are effective in olanzapine treatment.

Adverse reactions

1. Common ones are drowsiness, weight gain, eosinophilia, prolactin, elevated cholesterol, elevated blood sugar, elevated triglycerides, diabetes, increased appetite, dizziness, akathisia, Parkinson's disease, leukopenia, neutropenia, dyskinesia, orthostatic hypotension, anticholinergic effect, transient asymptomatic elevation of liver transaminase, rash, tired, fatigue, fever, arthralgia, increased alkaline phosphatase, γ -glutamyl transfer increased peptidase, high uric acid, high creatine phosphokinase and edema.
2. In clinical trials of elderly patients with dementia, the olanzapine treatment group had a higher incidence of death and adverse reactions to cerebrovascular diseases than the placebo group. In this group of patients, abnormal gait and falls are common. Pneumonia, elevated body temperature, lethargy, erythema, visual hallucinations, and urinary incontinence are common.

Contraindications

1. It is contraindicated in patients who are known to be allergic to any component of the product.
2. It is contraindicated in patients who are known to be at risk of narrow-angle glaucoma.

Pregnant and lactating women

Use this medicine only when the possible benefit outweighs the potential risk to the fetus. If patients are taking olanzapine, they should be advised not to breastfeed their babies.

Children

Olanzapine is not suitable for the treatment of children and adolescents under 18 years of age.

Elderly

A lower starting dose is usually not used, but for elderly people 65 years and older, if there are clinical indications, a lower starting dose should be considered.

Metabolism

Olanzapine is metabolized in the liver through binding and oxidation reactions. The main circulating metabolite is 10-N-glucuronide. Cytochrome P450 allogeneic CYP1A2 and CYP2D5 are involved in the formation of N-desmethyl and 2-hydroxymethyl metabolites. Olanzapine is mainly excreted in the urine in the form of metabolites.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1414334	HTR2C	GG	lower risk of metabolism syndrome

RS1800497	ANKK1	GG	slightly low level of prolactin when treated with olanzapine
RS489693	intergenic	CC	weight gain and reduced risk of hypertriglyceridemia

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The olanzapine drug response testing item uses the 3 polymorphic loci of *HTR2C*, *ANKK1*, *MC4R* genes. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

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2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
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5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Quetiapine

Quetiapine belongs to the atypical antipsychotic of the dibenzothiazepine class.

My Quetiapine

Use With Drug Labeling

ADVERSE EFFECT : Patients with rs489693-CC genotype, compared to AA genotype, may be at a decreased risk of experiencing adverse effect, e.g., weight gain and hypertriglyceridemia; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

Suitable for all types of schizophrenia. This product is not only effective for positive symptoms of schizophrenia, but also for negative symptoms. It can also alleviate the affective symptoms associated with schizophrenia such as depression, anxiety and cognitive deficit symptoms.

Adverse reactions

Common adverse reactions are dizziness, lethargy, orthostatic hypotension, palpitations, dry mouth, loss of appetite, and constipation. It can also cause weight gain, abdominal pain, asymptomatic increase in ALP and increase in blood total cholesterol and triglycerides. Extrapyramidal adverse reactions are rare. Occasionally can cause excitement and insomnia.

Contraindications

1. People who are allergic to this product should not use it.
2. It is forbidden for patients with cardiovascular diseases (heart failure, myocardial infarction, conduction abnormalities) and ischemic heart disease.
3. Prohibited for patients with cerebrovascular disease.
4. Disabled for patients with coma, leukopenia, thyroid disease and epilepsy.
5. It is forbidden for patients with liver and kidney dysfunction.
6. Conditions that may induce hypotension (dehydration, hypovolemia, antihypertensive drug therapy) are prohibited.

Pregnant and lactating women

The efficacy and safety of this product when used in human pregnancy have not yet been determined. Therefore, quetiapine can only be used in pregnant patients if the benefit outweighs the potential risk. The excretion of this product in human milk is unclear. If lactating women take quetiapine, they should be advised to stop breastfeeding during the medication.

Children

The safety and effectiveness of this product for children and adolescents have not been evaluated.

Elderly

Due to the decrease in the clearance rate of this product, the dosage should be reduced as appropriate, and the dosage should be increased slowly.

Metabolism

Quetiapine is well absorbed and completely metabolized after oral administration. Eating has no significant effect on the bioavailability of quetiapine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS489693	intergenic	CC	weight gain and reduced risk of hypertriglyceridemia

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The quetiapine drug response test item uses 1 polymorphic locus of *MC4R* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

 Notification

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3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
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5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Clozapine

Clozapine is an atypical antipsychotic drug. It is mainly used for the treatment of schizophrenia for which other psychiatric drugs are ineffective. The efficacy of this product is stronger than other typical antipsychotic drugs, so it can be used to treat patients who are resistant to other drugs.

My Clozapine

Use With Drug Labeling

ADVERSE EFFECT : Patientis have lower level of prolactin and lower risk of metabolic syndrome, weight gain and hypertriglyceridemia when treated with olanzapine; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

This product is not only effective for positive symptoms of psychosis, but also for negative symptoms. It is suitable for all subtypes of acute and chronic schizophrenia, and has a good effect on hallucinations and delusions type and youth type. It can also reduce the emotional symptoms related to schizophrenia (such as depression, guilt, anxiety). For some patients whose traditional antipsychotic treatment is ineffective or not effective, it may be effective to switch to this product.

This product is also used to treat excitement, restlessness and hallucinations in mania or other psychotic disorders. Because it causes granulocytopenia, it is generally not suitable as the drug of choice.

Adverse reactions

1. Strong sedation and anticholinergic adverse reactions are common, such as dizziness, weakness, drowsiness, hyperhidrosis, salivation, nausea, vomiting, dry mouth, constipation, orthostatic hypotension, and tachycardia.
2. Increased appetite and weight gain are common.
3. Can cause abnormal changes in the electrocardiogram. Can cause EEG changes or seizures.
4. It can also cause increased blood sugar.
5. Serious adverse reactions are agranulocytosis and secondary infection.

Contraindications

1. People with severe heart, liver, kidney disease, coma, delirium, hypotension, epilepsy, glaucoma, bone marrow suppression or leukopenia are contraindicated.
2. People who are allergic to this product should not use it.

Pregnant and lactating women

Prohibited by pregnant women. Lactating women should stop breastfeeding while using this product.

Children

Not suitable for children under 12 years old.

Elderly

Use with caution or use low doses.

Metabolism

After being metabolized by the liver, 80% appears in urine and feces in the form of metabolites. The main metabolites are N-desmethyloclozapine and N-oxide of clozapine, ect.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1414334	HTR2C	GG	likely lower risk of metabolism syndrome

RS1800497	ANKK1	GG	may have lower prolactin level
RS489693	intergenic	CC	reduced likelihood of weight gain and hypertriglyceridemia

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The clozapine drug response testing item uses the 3 polymorphic loci of *HTR2C*, *ANKK1*, *MC4R* genes. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

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2. Limited by the current technology of genetic testing, the genetic report may not cover all genes or loci that are related to the test item.
3. Limited by the current technology of genetic testing, the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Paliperidone

Paliperidone is a dopamine and 5-HT_{2A} antagonist and is an atypical antipsychotic.

My Paliperidone

Use With Drug Labeling

ADVERSE EFFECT : Patients with rs489693-CC genotype, compared to AA genotype, may be at a decreased risk of experiencing adverse effect, e.g., weight gain and hypertriglyceridemia; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for the treatment of acute schizophrenia.

Adverse reactions

The most common adverse reaction in clinical trials (in any dose group, the report rate of subjects receiving this product must reach 5% or higher, at least twice the report rate of the placebo group) is

akathisia and extrapyramidal department of obstacles. The most common adverse reaction related to the withdrawal of subjects from clinical trials (causing 2% of subjects in the product treatment group to withdraw) was central nervous system disease. There are the following adverse reactions:

- Will increase the mortality rate of elderly patients with dementia-related mental illness
- Cerebrovascular adverse reactions, including stroke, dementia-related mental illness in elderly patients
- Antipsychotic Malignant Syndrome
- QT interval extension
- Tardive dyskinesia
- High blood sugar and diabetes
- Hyperprolactinemia
- Possibility of gastrointestinal obstruction
- Postural hypotension and syncope
- Possible cognitive and motor dysfunction
- Epilepsy
- Hard to swallow
- Suicide
- Priapism
- Thrombotic Thrombocytopenic Purpura (TTP)
- Destruction of body temperature regulation function
- Antiemetic effect
- Increased sensitivity in patients with Parkinson's disease or with Lewy body dementia
- Diseases or conditions that affect metabolic or hemodynamic responses



Contraindications

1. Hypersensitivity reactions have been observed in patients treated with risperidone and paliperidone, including allergic reactions and angioedema.
2. Among them, this product (paliperidone) is a metabolite of risperidone, so it is contraindicated in patients who are known to be allergic to paliperidone, risperidone or any of the ingredients in this product.



Pregnant and lactating women

There have not been adequate and well-controlled studies on pregnant women using this product. Use this product during pregnancy only when the potential benefits outweigh the possible danger to the fetus. The metabolites of this product can be secreted by breast milk. When giving this product to breast-feeding women, the drug should be used with care.



Children

The safety and effectiveness of this product in patients aged <12 years are still unclear. When used in children and adolescents, the sedative effect of this product should be closely monitored.

Elderly

No difference in safety or effectiveness between young subjects and elderly subjects was observed. This product is mainly excreted through the kidneys. Therefore, care should be taken in the choice of dosage. The advantage may require monitoring of renal function.

Metabolism

The plasma protein binding rate of paliperidone is 74%, and the apparent volume of distribution is 487L. A small amount is metabolized by the liver, mainly excreted in urine and feces, of which 59% are prototype drugs, 32% are metabolites, and the terminal half-life is about 23 hours.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS489693	intergenic	CC	weight gain and reduced risk of hypertriglyceridemia

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse

reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The paliperidone drug response test item uses 1 polymorphic locus of *MC4R* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Ziprasidone

Ziprasidone, an atypical antipsychotic (AAP; SGAs), is used to treat schizophrenia and acute mania and agitated depression associated with bipolar disorder.

Ziprasidone can also be used for the treatment of mood depression symptoms, bipolar disorder, and post-traumatic stress syndrome (PTSD) and other symptoms of drugs that are off-label therapeutic drugs.

My Ziprasidone

Use With Drug Labeling

ADVERSE EFFECT : Patients with rs489693-CC genotype, compared to AA genotype, may be at a decreased risk of experiencing adverse effect, e.g., weight gain and hypertriglyceridemia; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for the treatment of schizophrenia.

Adverse reactions

Sleepiness, akathisia, extravertebral system symptoms, dizziness, dystonia, headache, gastrointestinal discomfort, weakness, agitation, hypertonic state. A few cases have elevated SGPT, skeletal muscle discomfort, central nervous system abnormalities, rhinitis, maculopapular rash, rubella, abnormal vision, and urinary incontinence.

Contraindications

1. QT prolongation: Ziprasidone dose-dependently prolongs the QT interval, and some drugs that prolong the QT interval have been confirmed to be related to fatal arrhythmia. Ziprasidone is contraindicated in patients with a history of prolonged QT interval, patients with recent acute myocardial infarction, and patients with uncompensated heart failure. Ziprasidone should not be used in combination with drugs that prolong the QT interval, such as dofetilide, sotalol, quinidine, other class I a and III antiarrhythmic drugs, mesoridazine, thioridazine, chlorpromazine, droperidol, pimozide, sparfloxacin, gatifloxacin, moxifloxacin, halofantrine, mefloquine, pentamidine, arsenic trioxide, levomethadol (levomethadylacetate), dolasetron mesylate, probucol and tacrolimus.
2. Patients who are allergic to this product are contraindicated.
3. Patients with a significant history of cardiovascular disease (such as QT prolongation, recent acute myocardial infarction, uncompensated heart failure, arrhythmia) should avoid ziprasidone. Ziprasidone should be discontinued in patients whose QTc interval lasts longer than 500 milliseconds.

Pregnant and lactating women

Ziprasidone can be used in pregnant women only when the benefits of the drug for pregnant women outweigh the potential risk of the drug to the fetus. It is not clear whether ziprasidone is secreted into breast milk, and women taking ziprasidone should not breastfeed.

Children

The safety and efficacy of ziprasidone in children have not been evaluated.

Elderly

The starting dose should be reduced, the dose should be adjusted slowly, and the patient should be closely monitored.

Metabolism

Ziprasidone is mainly metabolized by the liver, and only a small amount of the original drug is excreted in urine (<1%) and feces (<4%). Ziprasidone is mainly eliminated through three metabolic pathways and produces four main circulating metabolites: benzisothiazole (BITP) sulfoxide, BITP-sulfone, ziprasidone sulfoxide and S-methyl-dihydrogen Ziprasidone. The excreted drugs in urine and feces are about 20% and 66%, respectively, and the original ziprasidone in serum is about 44%.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS489693	intergenic	CC	weight gain and reduced risk of hypertriglyceridemia

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The ziprasidone drug response test item uses 1 polymorphic locus of *MC4R* gene. The test results indicate the genotype results of the user's detected variations above and based on existing scientific research, interpret how genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Fluoxetine

Fluoxetine is a widely used selective 5-HT reuptake inhibitor (SSRI) in clinic, which can selectively inhibit 5-HT transporter, block the presynaptic membrane reuptake of 5-HT, prolong and increase the role of 5-HT, which produces an antidepressant effect.

My Fluoxetine

Risk of Insufficient Efficacy

EFFICACY : Patients with rs4713916-GG genotype, compared to AA genotype, may have a decreased drug response.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is used for various depressive mental disorders, depression including mild or major depression, bipolar disorder, psychogenic depression and depressive neurosis.

Adverse reactions

Common adverse reactions are dry mouth, loss of appetite, nausea, insomnia, and fatigue. Anxiety and headache can be seen in a few cases.

Contraindications

It is forbidden for people who are known to be allergic to this drug.

Pregnant and lactating women

A large amount of data shows that fluoxetine has no teratogenic effect on humans. Fluoxetine can be used during pregnancy. Be careful when used in the third trimester of pregnancy or at the beginning of childbirth. Because it has been reported that fluoxetine has the following effects on newborns: irritability, tremor, hypotonia, constant crying, difficulty sucking, or difficulty sleeping. These symptoms suggest that it may be a serotonergic effect or withdrawal syndrome. The occurrence and duration of these symptoms may be related to the longer half-life of fluoxetine (4 to 6 days) and its active metabolite, norfluoxetine (4 to 16 days). Fluoxetine and its metabolite norfluoxetine can be secreted into breast milk. Adverse events for infants have been reported. If fluoxetine must be taken, breastfeeding should be stopped; but if you want to continue breastfeeding, fluoxetine should be at the lowest effective dose.

Children

Because the safety and efficacy are not yet clear, fluoxetine is not recommended for children and adolescents (under 18 years old).

Elderly

Pay attention to the dosage or follow the doctor's advice.

Metabolism

It is mainly metabolized in the liver into the active metabolite norfluoxetine and other metabolites, which are excreted in the urine from the kidneys.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4713916	FKBP5	GG	poor drug response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical

medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The Fluoxetine drug reaction item uses a polymorphic locus of the *FKBP5* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

-
1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
 2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
 3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Remifentanyl

Remifentanyl is a potent, short-acting synthetic opioid analgesic drug. It is given to patients during surgery to relieve pain and as an adjunct to an anaesthetic. Remifentanyl is used for sedation as well as combined with other medications for use in general anesthesia.

My Remifentanyl

Consider Dose Increase

DOSE : Patients with rs4680-GG genotype, compared to AG or AA genotype, may have a decreased drug response, may require increased dose of drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Used for general anesthesia induction and maintenance analgesia during general anesthesia.

Adverse reactions

This product has the typical adverse reactions of μ opioid receptor drugs. Typical adverse reactions include nausea, vomiting, respiratory depression, bradycardia, hypotension and muscle rigidity. The

above-mentioned adverse reactions can disappear within a few minutes after stopping the drug or reducing the infusion speed. Chills, fever, dizziness, visual disturbance, headache apnea, itching, tachycardia, hypertension, agitation, hypoxemia, epilepsy, flushing and allergies have also been found in clinical studies at home and abroad. In addition, there are some rare adverse reactions:

- Digestive system: constipation, abdominal discomfort, dry mouth, gastroesophageal reflux, dysphagia, diarrhea, heartburn, intestinal obstruction.
- Cardiovascular system: myocardial ischemia and syncope.
- Musculoskeletal system: muscle rigidity, chest pain.
- Respiratory system: cough, dyspnea, bronchospasm, laryngeal spasm, wheezing, nasal congestion, pharyngitis, pleural effusion, pulmonary edema, bronchitis and rhinorrhea.
- Mental nervous system: anxiety, involuntary movement, tremor, disorientation, hallucination, irritability, nightmares, paresthesia, forgetfulness.
- Skin: rash, urticaria.
- Urinary system: urinary retention, oliguria, urinary tract interruption.
- Blood system: anemia, lymphocytopenia, leucopenia, thrombocytopenia.

Contraindications

1. Patients who are known to be allergic to this product or other fentanyl drugs are forbidden.
2. Disabled for patients with myasthenia gravis and respiratory depression.
3. Combination with monoamine oxidase inhibitors is prohibited.
4. Prohibited for patients with bronchial asthma.

Pregnant and lactating women

This product can pass through the placental barrier, and may cause respiratory depression in newborns when used by mothers. This product can be excreted in breast milk, so it is not recommended for pregnant and lactating women. When it must be used, the doctor should weigh the pros and cons.

Children

The medication for 12-year-old children is the same as that for adults. Because there is no clinical data, it is not recommended for children under 2 years old.

Elderly

As patients age, the pharmacological effects of remifentanyl increase. The initial dose for elderly patients over 65 years old is half the adult dose, and the dose should be reduced accordingly.

Metabolism

The metabolism of remifentanyl is not affected by plasma cholinesterase and anticholinesterase drugs, liver and kidney functions, age, weight, and gender. It is mainly metabolized by non-specific

esterase hydrolysis in plasma and tissues. About 95% of remifentanyl is metabolized and excreted in the urine, and the activity of the main metabolite is only 1/4600 of that of remifentanyl.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4680	COMT	GG	decreased drug response, may require higher dose

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The Remifentanil drug reaction item uses a polymorphic locus of the *COMT* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Sufentanil

Sufentanil is a phenpiperidine derivative, which mainly acts on μ -Opioid receptor and is used as an anesthetic and analgesic.

My Sufentanil

Consider Dose Increase

DOSE : Patients with rs4680-GG genotype, compared to AG or AA genotype, may have a decreased drug response, may require increased dose of drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is used for general anesthesia, compound anesthesia and analgesia. Sufentanil is used for endotracheal intubation and general anesthesia with artificial respiration. As an analgesic drug for compound anesthesia. As an anesthetic induction and maintenance drug for general anesthesia surgery.

Adverse reactions

1. Typical opioid symptoms, such as respiratory depression, apnea, skeletal muscle rigidity (foot muscle rigidity), myoclonus, hypotension, bradycardia, nausea, vomiting and dizziness, miosis and feces retention.
2. Other less common adverse reactions include: pharyngeal spasm. Allergic reactions and cardiac arrest. Due to the use of other drugs during anesthesia, it is difficult to determine whether these reactions are related to sufentanil. Occasionally, there may be reinhibition of breathing during the postoperative recovery period.



Contraindications

1. People who are allergic to sufentanil or other opioids are prohibited.
2. During childbirth, or before the umbilical cord is cut during a caesarean section, this product should not be used intravenously, because sufentanil can cause respiratory depression in newborns.
3. It is contraindicated in neonates, pregnant women and lactating women. If a breastfeeding woman must take sufentanil, she should not be able to breastfeed her baby again 24 hours after taking the drug.
4. Concurrent use with monoamine oxidase inhibitors is prohibited. Those who have used monoamine oxidase inhibitors within 14 days before using sufentanil should not use this product.
5. Acute liver porphyria is contraindicated.
6. People who have respiratory depression due to other drugs are contraindicated.
7. It is forbidden for patients with respiratory depression disease.
8. It is forbidden for patients with hypovolemia or hypotension.
9. It is forbidden for patients with myasthenia gravis.



Pregnant and lactating women

It is contraindicated during pregnancy and breastfeeding.



Children

There are very limited data on the effectiveness and safety of sufentanil in children under two years of age. The recommended total amount of medicine used for general anesthesia based on sufentanil citrate for children 2-12 years old is 10-12µg/kg body weight. If the clinical manifestations of analgesic effect is reduced, an additional dose of 1~2µg/kg body weight can be given.



Elderly

For frail patients and elderly patients, as well as patients who have used drugs that can inhibit breathing, the dosage should be reduced.



Metabolism

Sufentanil undergoes extensive biotransformation in the liver to form N-dealkylated and O-demethylated metabolites, which are excreted by the kidneys.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4680	COMT	GG	decreased drug response, may require higher dose

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The Sufentanil drug reaction item uses a haplotype information of the *COMT* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Methadone

Methadone is an opioid drug. It is suitable for pain relief and maintenance treatment, or to help people who are rehabilitated to get rid of opiate dependence.

My Methadone

Risk of Insufficient Efficacy

DOSE : Patients with rs4680-GG genotype compared to AA or AG genotype may have a decreased drug response may require an increased dose of methadone; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. This product has a slow onset and a long duration of action and is suitable for chronic pain. But its analgesia is often not complete enough. For acute trauma pain, it is rarely used because it has a slow effect and can not relieve pain immediately.
2. Alternative de escalation method was used for detoxification treatment of various opioids, especially for heroin dependence; and also used for morphine, opioid, pethidine,

dihydroetorphine etc. dependence.



Adverse reactions

1. Adverse effects are mainly hypogonadism, with little semen after use in men, and breast hyperplasia can be present. Women who use contraceptives together can become tired and fatigue all day long. If the amount is exceeded, they can gradually enter a coma, and develop right bundle branch block, tachycardia, or (and) hypotension.
2. There are also dizziness, nausea, vomiting, sweating, drowsiness, etc., and can also cause constipation and drug dependence.



Contraindications

1. Prohibited for respiratory insufficiency.



Pregnant and lactating women

During pregnancy, the drug can penetrate the placental barrier, causing chromosomal mutations in the fetus. This has caused many stillbirths and immature newborns. Neonates born to mothers with drug addiction, often presenting with delayed withdrawal symptoms, are not detected until 6-7 days after birth and persist for 6-17 days. The urinary drug concentration of these newborns can be 10-16 times that of the blood, and they are often accompanied by hypoglycemia, which is difficult to deal with. Therefore, it is strictly contraindicated during labor and delivery in pregnancy.



Children

Infant contraindicated.



Elderly

Not yet clear.



Metabolism

This product is an opioid receptor agonist, which is characterized as being orally effective, has a long duration of action to suppress withdrawal symptoms in morphine addicts, and repeated administration remains effective. It is mainly metabolized in the liver and excreted by the kidney and bile. Repeated administration has the effect of tissue accumulation.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS3745274	CYP2B6	GG	slightly high dose requirement of metha done

RS4680

COMT

GG

methadone is less effective and should
be used at higher doses

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The Methadone drug reaction item uses 2 polymorphic loci of the *CYP2B6* and *COMT* genes. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Buprenorphine

Buprenorphine is a partial opioid receptor agonist. It is a slow onset, long lasting but strong analgesic.

My Buprenorphine

Use With Drug Labeling

DOSE : Post-operative patients may have lower need for opioid analgesics as drug response is strong; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. For all kinds of postoperative pain, cancer pain, post burn pain, limb pain caused by vasculitis, angina pectoris and other visceral pain.
2. It can also be used as maintenance treatment for abstinence.

Adverse reactions

The adverse reactions were similar to morphine. Common adverse reactions include dizziness, drowsiness, nausea, emese, etc. Withdrawal symptoms may occur on the basis of the use of other

opioids

Contraindications

1. It is forbidden for patients with allergic history, severe liver injury, brain damage, blurred consciousness and elevated intracranial pressure.
2. It should not be used by children under the age of six, pregnant women, lactating women and those with slight pain or unknown cause of pain.

Pregnant and lactating women

Animal experiments have reported dystocia, lactation difficulties and low fetal survival rate. Drugs can be secreted through milk and pass through placenta and blood cerebrospinal fluid barrier. Therefore, they should not be used by pregnant women and lactating women.

Children

Not suitable for children under 7 years old.

Elderly

Use with caution for old and weak patients.

Metabolism

Buprenorphine is mainly metabolized in the liver, then excreted through bile and excreted with feces. Fewer prototypes were excreted from urine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2952768	intergenic	TT	lower requirement of opioid analgesia
RS678849	OPRD1	TT	may have increased response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or

professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The Buprenorphine drug reaction item uses 2 polymorphic loci of the *OPRD1* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.

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2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
 3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Fentanyl

Fentanyl is a powerful opioid central analgesic.

My Fentanyl

Use With Drug Labeling

DOSE : Post-operative patients may have normal need for opioid analgesics; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for all kinds of pain and analgesia after and during operation, such as surgery, gynecology and so on.
2. It is also used to prevent or reduce delirium after surgery.
3. It can also be used together with anesthetics as an auxiliary drug of anesthesia.

Adverse reactions

1. General adverse reactions include dizziness, blurred vision, nausea, emesis, hypotension, biliary sphincter spasm, laryngospasm, diaphoresis, etc. Occasionally muscle twitches.

- 2. Serious side effects include respiratory depression, asphyxia, muscle rigidity and bradycardia. If not treated in time, respiratory arrest, circulation depression and cardiac arrest may occur.
- 3. This product is addictive, but lighter than pethidine.

 Contraindications

- 1. It is forbidden for patients with bronchial asthma, respiratory depression, especially sensitive to this product and patients with myasthenia gravis.
- 2. It is forbidden to use it with monoamine oxidase inhibitors (such as Phenelzine, Pargyline, etc.).

 Pregnant and lactating women

The safety is still difficult to confirm, so use it with caution.

 Children

This test was not performed and no reliable references were available.

 Elderly

For elderly and frail patients, the first dose should be appropriately reduced, and the increase of dose should be determined according to the effect of the first dose.

 Metabolism

Oral absorption through gastrointestinal tract, but injection is generally used in clinics. Intravenous injection takes effect in 1 minute, reaches the peak in 4 minutes and lasts for 30 ~ 60 minutes. During intramuscular injections, the analgesic effect takes about 7 ~ 8 minutes and lasts for 1 ~ 2 hours. This product is mainly metabolized in the liver, and the metabolites and about 10% of the prototype drug are discharged from the kidney.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1045642	ABCB1	AG	normal requirement of opioid analgesia
RS2952768	intergenic	TT	lower requirement of opioid analgesia

How to use the test results

 Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The Fentanyl drug reaction item uses 2 polymorphic loci of the *ABCB1* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

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1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
 2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
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 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Morphine

Morphine is a pain medication of the opiate family. It acts directly on the central nervous system (CNS) to induce analgesia and alter perception and emotional response to pain. It can be taken for both acute pain and chronic pain.

My Morphine

Consider Dose Increase

DOSE : Patients with rs4680-GG genotype, compared to AG or AA genotype, may have a decreased drug response, may have an increased opioid dose requirements.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. For acute sharp pain refractory to other analgesics, such as pain from severe trauma, war injuries, burns, advanced cancer, etc. For patients with muscle infarction and normal blood pressure, the application of this product can calm the patient and reduce the burden on the heart. Applying to cardiogenic asthma can temporarily relieve the symptoms of pulmonary edema. Anesthesia and pre-surgery administration can keep the patient quiet and sleepy. Because this product has a strong excitatory effect on smooth muscles, it cannot be used alone for visceral colic (such as

biliary colic, etc.). But it should be used in combination with effective antispasmodic drugs such as atropine. This product is not suitable for long-term use in patients with chronic severe cancer pain.

Adverse reactions

1. Drug resistance develops in 3-5 days and is addictive for more than 1 week. So this drug should be used with caution. However, for patients with advanced moderate to severe cancer pain, if treatment is appropriate, dependence and addiction are rare.
2. Nausea, emesis, respiratory depression, somnolence, vertigo, constipation, dysuria, biliary colic, etc. Occasionally, pruritus, urticaria, and skin edema are observed.
3. The main symptoms of acute poisoning by this product are coma, profound respiratory depression, extremely constricted pupils, bilaterally symmetrical, or of a needle tip like appearance, decreased blood pressure, cyanosis, oliguria, decreased body temperature, skin clamminess, and muscle weakness. Severe hypoxia led to shock, circulatory collapse, mydriasis, and death.

Contraindications

Respiratory depression has been shown to be contraindicated in patients with cyanosis, increased intracranial pressure and head injury, bronchial asthma, decompensation of pulmonary origin, hypothyroidism, cortical insufficiency, prostatic hypertrophy, dysuria and severe liver dysfunction, shock not corrected before control, and inflammatory bowel infarction.

Pregnant and lactating women

This product can reach the fetus through the placental barrier, and a small amount is excreted via milk, so it is not used in infants, pregnant women, lactating women. It is contraindicated in clinician pelvic parturients because it can prolong labor by antagonizing the excitatory effect of oxytocin on the uterus.

Children

Caution is used in infants and young children and is contraindicated in immature neonates.

Elderly

Use with caution.

Metabolism

Primarily metabolized in the liver, it is excreted renally with a clearance of 15 to 23 ml/(kg · min); a small amount is excreted transmammary.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2952768	intergenic	TT	lower dose requirement of opioid analgesia
RS4680	COMT	GG	opioids are less effective and should be used at higher doses

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The Morphine drug reaction item uses 2 polymorphic loci of the *COMT* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Meperidine

Meperidine hydrochloride, also known as Durantine, Meperidine, Markeridine, Pexetine, and Demerol. Meperidine is a synthetic anesthetic and is commonly used clinically. It is usually made into an injection form and used as an anesthetic.

My Meperidine

Use With Drug Labeling

DOSE : Patients with rs2952768-TT genotype, compared to CC genotype, may have decreased opioid dose requirements after operation; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. This product is a powerful analgesic, suitable for all kinds of severe pain, such as traumatic pain, postoperative pain, pre-anaesthesia medication, or local anesthesia and intravenous inhalation combined anesthesia auxiliary medication, etc. It should be used in combination with atropine for visceral colic. When used for analgesia during childbirth, it is necessary to monitor the respiratory inhibitory effect of this product on newborns. During administration before anesthesia and artificial hibernation, it is often combined with chlorpromazine and promethazine to form an

artificial hibernation mixture for application. It is used for cardiogenic asthma to help eliminate pulmonary edema. Patients with advanced cancer with chronic severe pain should not use this product for a long time.

 Adverse reactions

1. The tolerance and addiction of this product are between morphine and codeine, and generally should not be used continuously.
2. Symptoms such as mild dizziness, diaphoresis, dry mouth, nausea, emese, tachycardia, and orthostatic hypotension may occur at the treatment dose.

 Contraindications

1. Supraventricular tachycardia, head injury, intracranial space occupying lesions, chronic obstructive pulmonary disease, bronchial asthma, severe pulmonary insufficiency, etc. are contraindicated.
2. It is strictly forbidden to use it with monoamine oxidase inhibitors.

 Pregnant and lactating women

This product can pass through the placental barrier and secrete into milk, so the dosage should be reduced when using for analgesia during childbirth and during breastfeeding.

 Children

Basic pediatric anesthesia: After thiopental sodium is 3~5mg/kg body weight for 10~15min, add pethidine 1mg/kg plus promethazine 0.5mg/kg diluted to 10ml slowly.

 Elderly

The elderly should be used with caution.

 Metabolism

Oral absorption is fast, the volume of distribution is 2.8~4.2L/kg, the protein binding rate is 64%~82%, and it is metabolized by the liver, t1/2 is 3.2~4.1 hours, and the clearance rate is 10~17ml/(kg·min).

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2952768	intergenic	TT	higher dose requirement of opioid analgesia

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The Meperidine drug reaction item uses a polymorphic locus of the interregion of the gene. The test results indicate the genotype variation results tested above of user and based on existing scientific

research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

 Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Pentazocine

Pentazocine is the first clinically used opioid receptor agonist/antagonist analgesic. It can provide an analgesic effect similar to opioid drugs including morphine and dulantine, and its drug dependence is less than other opioid drugs.

My Pentazocine

Use With Drug Labeling

DOSE : Patients with rs2952768-TT genotype, compared to CC genotype, may have decreased opioid dose requirements after operation; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Suitable for all kinds of pain, such as cancer pain, traumatic pain, pain after surgery.
2. It can also be administered before surgery or anesthesia, as an auxiliary medication for surgical anesthesia.

Adverse reactions

The adverse reactions of this product are varied:

1. Blurred vision or diplopia can occur when the pupil constricts to the needle tip size.
2. Constipation: there are local gastrointestinal factors, there are also central factors.
3. The antidiuretic effect was evident with morphine, and when ureterospasm was present, oliguria, frequency, urgency, and dysuria were observed
4. When the body position changes and blood pressure drops, there is often a sense of dizziness, unsteady gait, and fatigue.
5. When central nervous system activity is in a state of inhibition, the clinical manifestations may include drowsiness, dreams, headaches, dizziness, etc., followed by dry mouth, loss of appetite, boring diet, nausea and emese. The latter is more common in emergencies and the first time of administration.
6. The release of histamine can cause flushing of the cheeks and excessive sweating.
7. Abdominal pain can be provoked by gastrointestinal irritation and biliary spasm.
8. It can cause emotional restlessness or difficulty in sleeping and other reactions.
9. There are rare but dangerous adverse effects: frequent slowing and irregularity of breathing, small tidal volumes, suggesting severe respiratory depression, and some opioids in turn can cause hypotonia of skeletal muscles mainly of the chest wall respiratory muscles, especially when large and easily present during rapid sedation.
10. CNS toxicity manifests, most prominently with convulsions, hallucinations, tinnitus, tremor, and inability to move himself or herself.
11. The central depression is excessive, with confusion, depression, depression, and dullness, and is often infantile and may present with paroxysmal excitation and agitation, and in large doses may cause respiratory depression, an increase in blood pressure, and an acceleration of heart rate.
12. Excessive histamine release can induce elastic urticaria, itchy skin, colorful microstomia, bronchospasm, laryngeal spasm, laryngeal edema, etc.



Contraindications

1. Toxic diarrhea in which toxicants accumulate in the intestinal lumen and have not been drained.
2. Acute respiratory depression, hypoventilation.
3. In the event of haematological disease or vascular injury presenting with coagulation abnormalities, and where puncture is necessary locally in the event of inflammation, epidural or subarachnoid administration may not be permitted, nor does such administration in its break result in symptomatic improvement.



Pregnant and lactating women

Use with caution.

Children

Due to the slow clearance rate and long half-life in children, it is especially easy to cause respiratory depression, so the dosage should be lower than the usual dosage.

Elderly

The clearance rate of elderly patients is slow, and the half-life is long. It is especially easy to cause respiratory depression. The dosage should be lower than the usual dosage.

Metabolism

The plasma concentration reached a peak 15 minutes after intramuscular injection, and the peak plasma concentration 2-3 minutes after intravenous injection, $t_{1/2}$ was about 2 hours. It is mainly metabolized in the liver and excreted by the kidneys. Approximately 60% of the total discharge in 24 hours.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2952768	intergenic	TT	higher dose requirement of opioid analgesia

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The Pentazocine drug reaction item We uses a polymorphic locus of the interregion of the gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can

consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Oxycodone

Oxycodone is an opioid medication used for treatment of moderate to severe pain.

My Oxycodone

Consider Dose Increase

DOSE : Patients with rs4680-GG genotype, compared to AA or AG genotype, may have a decreased drug response, may require decreased dose of oxycodone.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is suitable for moderate to severe acute and chronic pain caused by various reasons.

Adverse reactions

There may be adverse reactions of opioid receptor agonists. May produce tolerance and dependence.

1. Common adverse reactions: constipation (laxatives can prevent constipation), nausea, vomiting, dizziness, pruritus, headache, dry mouth, hyperhidrosis, sleepiness and fatigue. If nausea and

vomiting occur, antiemetic drugs can be used for treatment.

2. Occasionally adverse reactions: anorexia, nervousness, insomnia, fever, confusion, diarrhea, abdominal pain. Vasodilation, indigestion, paraesthesia, rash, anxiety, euphoria, depression, dyspnea, postural hypotension, chills, nightmares, abnormal thinking, hiccups.
3. Rare adverse reactions: vertigo, twitching, gastritis, disorientation, flushing, emotional lability, palpitations (in the context of withdrawal), hallucinations, bronchospasm, dysphagia, bloating, intestinal obstruction, taste perversion, agitation, forgetfulness, hypertonia, hyperesthesia, hypotonia, discomfort, involuntary muscle contraction, speech disorders, tremors, visual abnormalities, withdrawal syndrome, amenorrhea, loss of libido, impotence, hypotension, supraventricular sex tachycardia, syncope, dehydration, edema, peripheral edema, thirst, dry skin, urticaria, allergy, allergic reactions, anaphylaxis, miosis, and colic. Difficulty urinating, bile duct spasm, or ureter spasm may occur. Respiratory depression may occur with overdose.

Contraindications

1. It is forbidden for those allergic to oxycodone.
2. Prohibit oxycodone in any situation where opioids are prohibited, including patients with severe respiratory depression (without monitoring devices or lack of resuscitation equipment), acute or severe bronchial asthma, and hypercapnia.
3. Oxycodone is forbidden for those suspected or known to have paralytic intestinal obstruction.

Pregnant and lactating women

Not recommended for pregnant and lactating women.

- Pregnancy: This product should not be given to pregnant women unless the doctor judges that the potential benefits outweigh the possible dangers.
- Labor or Delivery: It is not recommended for parturients to use this product before and during labor pain and delivery, as it may have a potential impact on the neonatal respiratory function.
- Lactating women: Generally, patients should not lactate while taking this product.

Children

At present, there is still a lack of medication data for patients under the age of 18, so it is not recommended for patients under the age of 18.

Elderly

Use with caution for the elderly and debilitated patients. Because the clearance rate of oxycodone in elderly patients is slightly lower than that in younger patients, when elderly patients really need to use this product, they should be highly cautious about the dosage and frequency of administration. Patients with liver and kidney insufficiency and prostatic hypertrophy should be used with caution.

Metabolism

The drug is extensively metabolized in the liver, and the metabolites are active noroxycodone and oxymorphone. Among them, noroxycodone is the main metabolite, and the area under the curve is 60% of the drug. Hydroxymorphone is metabolized by cytochrome P450 2D6 (CYP2D6), which has analgesic effect, but the blood concentration is low. The drug is mainly excreted through the kidneys, and the overall clearance rate is 0.8L/min.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4680	COMT	GG	poor drug response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be

sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The oxycodone drug reaction item uses one polymorphic locus of the *COMT* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Metformin

Metformin is the first-line medication for the treatment of type 2 diabetes, especially for patients who are overweight. It is also used in the treatment of polycystic ovary syndrome. A few studies have shown that it can also be used to prevent complications such as cardiovascular disease and cancer in diabetic patients. This product is an oral medication, and the medication will not cause weight gain.

My Metformin

Risk of Insufficient Efficacy

EFFICACY : Patients with rs11212617-AC genotype and diabetes mellitus or polycystic ovarian syndrome may have an increased drug response as compared to AA genotype, or a decreased drug response as compared to CC genotype; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. It is the first choice to use simple diet control and physical exercise to treat type 2 diabetes which is ineffective, especially obese type 2 diabetes.
2. When combined with insulin, this product can reduce the amount of insulin and prevent hypoglycemia.
3. It can be used in combination with sulfonylurea hypoglycemic agents and has a synergistic effect.

Adverse reaction

1. Occasional nausea, vomiting, diarrhea, abdominal pain, abdominal distension, indigestion, fatigue, etc.
2. Occasional tiredness, weight loss, headache, dizziness, taste perversion, rash, chills, flu like symptoms, palpitations, flushing, etc.
3. Rare lactic acidosis, manifested as vomiting, abdominal pain, hyperventilation and disturbance of consciousness.

Contraindications

1. People who are allergic to this product.
2. It is contraindicated in patients with diabetic ketoacidosis, liver and kidney dysfunction (serum creatinine exceeding 1.5mg/dl), pulmonary insufficiency, heart failure, acute myocardial infarction, severe infection and trauma, major surgery, hypotension and hypoxia, alcoholism, vitamin B12 or folic acid deficiency, severe diabetic nephropathy, diabetic retinopathy.

Pregnant and lactating women

It is forbidden for pregnant and lactating women.

Children

Clinical safety and efficacy have not been confirmed.

Elderly

As the age of elderly patients increases, renal function decreases physiologically. Therefore, it is necessary to pay attention to the selection of dose and regular examination of renal function. Usually, elderly patients should not receive the maximum dose of this product. This product should not be used in elderly patients over 80 years old unless the creatinine clearance shows no reduction in functional loss.

Metabolism

From gastrointestinal absorption, the average time of peak blood concentration was 7 hours. When taken with food, the absorption can be increased by about 50%. Repeated use will not accumulate in plasma. It is excreted in urine as a prototype, not metabolized by the liver, nor excreted by bile.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS11212617	C11orf65	AC	compared to AA genotype may have a better, compared to CC genotype may have a poor drug response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug

response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The metformin drug reaction item uses one polymorphic locus of the *C11orf65* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Glibenclamide

Glyburide is a sulfonylurea hypoglycemic agent.

My Glibenclamide

Use With Drug Labeling

EFFICACY : Patients with rs5219-CT genotype, compared to CC genotype, may have an increased drug response when receiving treatment with sulfonylureas; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is suitable for patients with mild to moderate type II diabetes who are not satisfied with the effect of diet control. The patient's pancreatic islet β cells have a certain function of secreting insulin without serious complications.

Adverse reaction

1. Diarrhea, nausea, vomiting, headache, stomachache or discomfort may occur.
2. Rash is rare.

3. Rare and serious cases include jaundice, liver function damage, bone marrow suppression, granulocytopenia (manifested as sore throat, fever and infection), thrombocytopenia (manifested as bleeding and purpura), etc.

 Contraindications

1. Patients with type 1 diabetes.
2. Patients with type 2 diabetes mellitus are accompanied by ketoacidosis, coma, severe burns, infections, trauma, and major operations.
3. Patients with liver and kidney dysfunction.
4. People who are allergic to sulfonamides.
5. Patients with leukopenia.

 Pregnant and lactating women

Pregnant women should not take it. This kind of medicine can be discharged from milk, and should not be taken by lactating women to avoid hypoglycemia in infants.

 Children

This test was not performed and no reliable references were available.

 Elderly

Elderly patients and those with renal insufficiency have decreased metabolism and excretion of this class of drugs. This product has a relatively strong hypoglycemic effect. This product should not be used. Other sulfonylurea hypoglycemic drugs with a shorter action time can be used.

 Metabolism

It is metabolized in the liver and excreted by the liver and kidneys each about 50%.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS5219	KCNJ11	CT	better response

How to use the test results

 Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The glibenclamide drug reaction item uses ong polymorphic locus of the *KCNJ11* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

-
1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
 2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
 3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
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 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Glipizide

Glipizide is mainly used for mild and moderate non-insulin-dependent patients who have not achieved good results with diet control alone.

My Glipizide

Use With Drug Labeling

EFFICACY : Patients with rs5219-CT genotype, compared to CC genotype, may have an increased drug response when receiving treatment with sulfonylureas; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is suitable for patients with mild or moderate type 2 diabetes who are not satisfied with the effect of diet control and physical exercise for 2~3 months. The pancreatic islet cells of these diabetics need to have a certain function of secrete insulin, and have no acute complications (such as infection, trauma, ketoacidosis, hyperosmolar coma, etc.), no pregnancy, no serious chronic complications.

Adverse reaction

1. The more common are gastrointestinal symptoms (such as nausea, fullness of upper abdomen), headache, etc., which can be relieved by reducing the dose.
2. Individual patients may have skin allergy.
3. Hypoglycemia is occasionally seen, especially in the elderly, weak, hyperactive, irregular eating, drinking or liver function damage.
4. There are also occasional reports of reversible changes in hematopoietic system.

Contraindications

1. People who are allergic to sulfonamides.
2. Patients with type 1 diabetes have been clearly diagnosed.
3. Patients with type 2 diabetes mellitus are accompanied by ketoacidosis, coma, severe burns, infection, trauma and major surgery.
4. Patients with liver and kidney dysfunction.
5. Patients with leukopenia.

Pregnant and lactating women

It is Forbidden for pregnant women. This kind of drug can be discharged from milk, and should not be taken by lactating women to avoid hypoglycemia in infants.

Children

This experiment was not conducted and there were no reliable references.

Elderly

Start with a small dose and adjust the dose gradually.

Metabolic principle

The drug is metabolized into inactive substances in the body, and 97% of the drug does is excreted on the first day; all of it is excreted by the kidney within three days.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS5219	KCNJ11	CT	better response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The glipizide drug reaction item uses one polymorphic locus of the *KCNJ11* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research,

interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

 Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
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5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Gliquidone

Gliquidone is classified as a second-generation sulfonylurea. Glipizide is a highly active pancreatic β -cell agent that binds to specific receptors on the pancreatic β -cell membrane and can induce the production of an appropriate amount of insulin to reduce blood glucose concentration.

My Glipizide

Use With Drug Labeling

EFFICACY : Patients with rs5219-CT genotype, compared to CC genotype, may have an increased drug response when receiving treatment with sulfonylureas; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is suitable for type 2 diabetes.

Adverse reaction

Very few people have reported skin allergic reactions, gastrointestinal reactions, mild hypoglycemic reactions and changes in the blood system.



Contraindications

1. Type 1 diabetes.
2. Diabetic coma or pre-coma.
3. Diabetes mellitus with acidosis or ketosis.
4. People who are allergic to sulfonamides.
5. Patients with pregnancy, lactation and late-stage uremia.



Pregnant and lactating women

Not suitable for pregnant and lactating women.



Children

Not yet clear.



Elderly

Not yet clear.



Metabolism

It is quickly absorbed in the gastrointestinal tract, mainly metabolized in the liver, and most metabolites (95%) are excreted in the feces through bile.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS5219	KCNJ11	CT	better response

How to use the test results



Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.



Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The gliquidone drug reaction item uses one polymorphic locus of the *KCNJ11* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the

item may be affected.

4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Glimepiride

Glimepiride is classified a third-generation sulfonylurea long-acting anti-diabetic drug.

My Glimepiride

Use With Drug Labeling

EFFICACY : Patients with rs5219-CT genotype, compared to CC genotype, may have an increased drug response when receiving treatment with sulfonylureas; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is suitable for type 2 diabetes.

Adverse reaction

1. Hypoglycemia, this product can cause hypoglycemia, especially in the early treatment frail elderly patients, or patients with irregular eating, drinking and liver and kidney function damage. It is reported that the incidence is 2%.

2. Digestive system symptoms: Nausea and vomiting, diarrhea and abdominal pain are rare.
3. A few cases have reported elevated serum liver transaminase.
4. Skin allergic reaction, pruritus, erythema and urticaria are rare.
5. Others: Headache, fatigue and dizziness are rare.



Contraindications

1. It is forbidden for those who are known to have a history of allergy to glimepiride.
2. It is forbidden for those who are diabetic ketoacidosis with or without coma. In this case, insulin therapy should be used.
3. It is forbidden for pregnant women, women in childbirth and lactation women.



Pregnant and lactating women

It is forbidden for pregnant and lactating women.



Children

There is still a lack of research data on the safety and effectiveness of this product in children.



Elderly

There is no special guidance or follow the doctor's advice.



Metabolism

This product is metabolized into metabolites without hypoglycemic activity by P450 oxide in the liver, 60% of which is excreted by urine and 40% by feces.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS5219	KCNJ11	CT	better response

How to use the test results



Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or

professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The glimepiride drug reaction item uses one polymorphic locus of the *KCNJ11* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.

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2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
 3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Gliclazide

Gliclazide is a sulfonylurea oral antidiabetic drug.

My Gliclazide

Use With Drug Labeling

EFFICACY : Patients with rs5219-CT genotype, compared to CC genotype, may have an increased drug response when receiving treatment with sulfonylureas; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is suitable for type 2 diabetes.

Adverse reaction

Occasionally mild nausea, vomiting, epigastric pain, constipation, diarrhea, erythema, urticaria, thrombocytopenia, neutropenia, anemia, etc. Most of them disappeared after drug withdrawal.

Contraindications

1. It is forbidden for those with liver and kidney dysfunction.
2. It is forbidden for those allergic to sulfonylurea.

Pregnant and lactating women

Animal tests and clinical observations have proved that gliclazide has teratogenic effect and is secreted in milk. Therefore, it is not suitable for pregnant and lactating women.

Children

This product should not be used by children.

Elderly

In elderly patients with type 2 diabetes, the dosage should be reduced when taking this product. It is advisable to use the preparations with shorter acting time to avoid the use of long-acting preparations.

Metabolism

After oral administration, it is mainly metabolized in the liver and excreted in the urine.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS5219	KCNJ11	CT	better response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

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Test details

Applicable situation

The gliclazide drug reaction item uses one polymorphic locus of the *KCNJ11* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

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environment and lifestyle factors.

5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Rosiglitazone

Rosiglitazone is a medicine used to control diabetes. It can increase insulin sensitivity and improve blood sugar control.

My Rosiglitazone

Use With Drug Labeling

EFFICACY : Patients with rs10509681-TT genotype have normal metabolism of rosiglitazone, sufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. For patients with type 2 diabetes who are still not satisfied with the effect of diet control and exercise therapy. This product can be used alone or in combination with sulfonylureas or biguanides to treat patients with type 2 diabetes who are poorly controlled by sulfonylurea or biguanides alone.

Adverse reaction

1. This product used alone rarely causes hypoglycemia (< 2%).

- 2. Effect on liver: In a comparative test for the treatment of type 2 diabetes, the incidence of elevated alanine aminotransferase (ALT) levels was 3 times higher than normal.
- 3. Mild to moderate edema and mild anemia occur in all elderly patients (≥ 65 years old), which are more common than those under 65 years old. The incidence of edema was 7.5%: 3.5%, and anemia was 2.5%: 1.7%.

 Contraindications

- 1. It is forbidden for those who are allergic to this product.

 Pregnant and lactating women

The safety of drug for pregnant women has not been determined. Pregnant women and women who may be pregnant should weigh the pros and cons. Animal (rat) experiments show that this product can be transferred into milk, and lactation should be avoided during taking this product.

 Children

The safety of drug use in children has not been determined.

 Elderly

Elderly patients do not need to adjust the dose according to their age.

 Metabolism

Rosiglitazone can be completely metabolized, and the non-prototypical drug is excreted from the urine. Its main metabolic pathway is N-demethylation and hydroxylation, combined with sulfuric acid and glucuronic acid.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS10509681	CYP2C8	TT	normal metabolism, normal efficacy

How to use the test results

 Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or

professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The rosiglitazone drug reaction item uses one polymorphic locus of the *CYP2C8* gene. The test results indicate the genotype variation results tested above of user and based on existing scientific research, interpret how these genotypes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above genotypes.

Notification

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2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
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Furosemide

Furosemide is a loop diuretic medication widely used in the treatment of congestive heart failure and edema.

My Furosemide

Risk of Insufficient Efficacy

EFFICACY : Patients with rs4961-TT genotype and liver cirrhosis, compared to GG genotype, may have a decreased response to furosemide.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Used for edematous diseases, including congestive heart failure, cirrhosis, kidney disease (nephritis, nephropathy and various causes of acute and chronic renal failure), especially when the effect of other diuretics is not good. This class of drugs may still be effective. Combined with other drugs to treat acute pulmonary edema and acute cerebral edema.
2. It is used for hypertension. In the step therapy of hypertension, it is not the first choice for the treatment of essential hypertension, but when thiazide drugs are not effective, especially when

accompanied by renal insufficiency or hypertensive crisis when this type of drug is particularly suitable.

3. It is used to prevent acute renal failure and used for various reasons that lead to insufficient blood flow in the kidney, such as dewatering, shock, intoxication, anesthesia accident, and circulatory insufficiency. It can be applied in time while correcting blood volume insufficiency to reduce Chance of acute tubular necrosis.
4. Used for hyperkalemia and hypercalcemia.
5. Used for dilutional hyponatremia, especially when the blood sodium concentration is lower than 120mmol/L.
6. Used for syndrome of inappropriate secretion of antidiuretic hormone (SIADH).
7. used for acute drug poisoning, such as barbiturate poisoning.

Adverse reactions

1. The common ones are related to water and electrolyte disorders, especially in high-dose or long-term application, such as postural hypotension, shock, hypokalemia, hyponatremia, hypochloremia, hypochloric alkalosis, hypocalcemia, thirst, fatigue, muscle soreness, arrhythmia, etc.
2. Rare cases have allergic reactions (including rash, interstitial nephritis, and even cardiac arrest), blurred vision, xanthopsia, light sensitivity, dizziness, headache, poor appetite, nausea, vomiting, abdominal pain, diarrhea, pancreatitis, muscle rigidity, etc. bone marrow suppression leads to granulocytopenia, thrombocytopenic purpura and aplastic anemia, and liver function damage, finger (toe) paresthesia, hyperglycemia, urine glucose positive, increased diabetes, hyperuricemia. Tinnitus and hearing impairment were most commonly seen in high-dose intravenous injection (more than 4~15mg per minute), mostly transient, and a few were irreversible, especially when combined with other ototoxic drugs.

Contraindications

Not yet clear

Pregnant and lactating women

This drug can pass through the placental barrier. Pregnant women, especially in the first 3 months of pregnancy, should avoid using it as much as possible. It has no preventive effect on pregnancy-induced hypertension syndrome. Animal experiments show that this product can cause fetal hydronephrosis, miscarriage and increased fetal mortality. This medicine can be secreted by breast milk and should be used with caution in lactating women.

Children

The half-life of this drug in newborns is significantly prolonged, so the interval between newborns should be extended.

Elderly

The elderly are more likely to have hypotension, electrolyte disturbances, thrombosis and renal function damage when using this medicine.

Metabolism

Mainly distributed in the extracellular fluid, the volume of distribution is 11.4% of body weight on average, and the plasma protein binding rate is 91%-97%, almost all of which are bound to albumin.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4961	ADD1	TT	worse response in diuretics treatment

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be

sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The furosemide drug reaction test item uses the 1 polymorphic locus of the *ADD1* gene. The test results indicate the genotype result of the user's mutation detected above and based on existing scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
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6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Spironolactone

Spironolactone is a diuretic commonly used to treat effusion caused by heart failure, liver cirrhosis and stomach disease. This drug is also used to treat hypertension, hypokalemia without improvement after supplementation, and female hirsutism. In addition, it is also used as an antiandrogen in cross gender female hormone replacement therapy.

My Spironolactone

Risk of Insufficient Efficacy

EFFICACY : Patients with rs4961-TT genotype and liver cirrhosis, compared to GG genotype, may have a decreased response to spironolactone.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Applicable to edema diseases: it is used in combination with other diuretics to treat edema diseases such as congestive edema, cirrhotic ascites and renal edema. Its purpose is to correct the secondary increased aldosterone secretion associated with the above diseases and resist the potassium excretion effect of other diuretics. It is also used in the treatment of idiopathic edema.

2. Applicable to hypertension: as an auxiliary drug for the treatment of hypertension.
3. Applicable to primary aldosteronism: Spironolactone can be used for the diagnosis and treatment of this disease.
4. Suitable for the prevention of hypokalemia: combined with thiazide diuretics to enhance the diuretic effect and prevent hypokalemia.



Adverse reactions

1. Common are:

1. Hyperkalemia is the most common, especially when it is used alone, eating a high-potassium diet, combined with potassium or potassium-containing drugs such as penicillin potassium, and when there is renal impairment, oliguria, or anuria; Even if combined with thiazide diuretics, the incidence of hyperkalemia can still reach 8.6% ~ 26%, and arrhythmia is often the first manifestation. Therefore, blood potassium and ECG must be closely followed up during medication;
2. Gastrointestinal reactions, such as nausea, vomiting, stomach cramps and diarrhea; there are still reports that can cause peptic ulcers.

2. Rare are:

1. Hyponatremia, which is rare when used alone, and the incidence is increased when used in combination with other diuretics;
2. Anti-androgen-like effects or effects on other endocrine systems. Long-term use of this drug can cause gynecomastia, impotence, and hyposexual function in men. In women, it can cause breast tenderness, thick voice, increased hair, and menstrual disorder, decreased sexual function;
3. Central nervous system performance, long-term or high-dose use of this drug may cause walking uncoordinated, headache, etc.

3. Rare are:

1. Allergic reactions, skin rashes and even breathing difficulties;
2. Temporary increase in plasma creatinine and urea nitrogen is mainly related to excessive diuresis, insufficient effective blood volume, and decreased glomerular filtration rate;
3. Mild hyperchloemic acidosis;
4. Tumors, there have been reports of breast cancer in 5 patients who took the drug and hydrochlorothiazide for a long time.



Contraindications

1. Disabled for patients with hyperkalemia.



Pregnant and lactating women

The drug can pass through the placenta, but its effect on the fetus is unclear. Pregnant women should take medication under the guidance of a physician, and the medication time should be as short as possible.

Children

Not yet clear.

Elderly

The elderly are more prone to hyperkalemia and excessive diuresis.

Metabolism

After entering the body, 80% is rapidly metabolized by the liver to active canrenone, which takes about 1 day to take effect and reaches a peak in 2 to 3 days. The effect can be maintained for 2 to 3 days after the drug is stopped. Inactive metabolites are excreted from the kidneys and biliary tract, and about 10% are excreted from the kidneys in their original form.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4961	ADD1	TT	worse response in diuretics treatment

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The spironolactone drug reaction test item uses the 1 polymorphic locus of the *ADD1* gene. The test results indicate the genotype result of the user's mutation detected above and based on existing scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
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5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Simvastatin

Simvastatin is the first generation of statins. Statins are widely used in the prevention and treatment of hyperlipidemia and cardiovascular and cerebrovascular diseases.

My Simvastatin

Risk of Adverse Effect

ADVERSE EFFECT : Moderate risk of myopathy.

DOSE : Consider lower dose of the drug or alternative statins, and creatine kinase monitoring is recommended.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Suitable for hyperlipidemia.
2. Applicable to coronary heart disease.

Adverse reactions

1. Such as nausea, diarrhea, rash, indigestion, pruritus, hair loss, dizziness, muscle cramps, myalgia, pancreatitis, paresthesia, peripheral neuropathy, vomiting and anemia, rhabdomyolysis and hepatitis/jaundice rarely occur.
2. Rare reports of obvious allergic reaction syndromes that include one or more of the following characteristics, such as angioneuroedema, lupus-like syndrome, rheumatic arthritis, vasculitis, thrombocytopenia, eosinophils Increased blood sedimentation rate (ESR), arthritis, arthralgia, urticaria, photosensitivity, fever, flushing, dyspnea, and discomfort.

Contraindications

1. Those who are allergic to this product.
2. Active hepatitis or unexplained persistent elevated serum aminotransferase.

Pregnant and lactating women

There is no data on the use of simvastatin in pregnant women. Because atherosclerosis is a chronic process, discontinuation of lipid-lowering drugs during pregnancy has little effect on the long-term treatment of primary hypercholesterolemia. Cholesterol and other products of its biosynthetic pathway are essential components for fetal development, including the synthesis of steroids and cell membranes. Because HMG-CoA reductase inhibitors such as simvastatin can reduce the synthesis of cholesterol and other products of its biosynthetic pathway, simvastatin is contraindicated by pregnant women. Among women of childbearing age, simvastatin can only be used for women who are unlikely to become pregnant. If a woman becomes pregnant while taking the medication, she should stop simvastatin and be informed of the possible damage to the fetus. It is not yet known whether simvastatin and its metabolites are secreted by human milk. Because many drugs are secreted by human milk and can cause serious adverse reactions, women taking simvastatin should not breastfeed (see Contraindications).

Children

The safety and effectiveness of medicine for children have not been determined. Simvastatin is not currently recommended for children.

Elderly

In elderly patients (>65 years old), in a clinical controlled study using simvastatin, its effect on reducing total cholesterol and low-density lipoprotein (LDL) cholesterol is similar to the results of other populations, adverse reactions and abnormal laboratory tests The incidence rate did not increase significantly.

Metabolism

Members of the organic anion transporter superfamily (OATPs) represent a group of important proteins involved in membrane transport of internal and external compounds. According to the latest naming principles, the mRNA or DNA is named SLCO1B1, and the protein is named OATP1B1. This

protein transporter is located in the basal membrane of liver cells, and transports simvastatin absorbed from the intestines into the liver for metabolic clearance. If the transporter gene is mutated, the transport clearance will be blocked, and the drug concentration in the blood will increase significantly.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4149056	SLCO1B1	CT	moderate risk of myopathy, consider lower dose

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be

sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The simvastatin drug reaction test item uses the 1 polymorphic locus of the *SLCO1B1* gene. The test results indicate the genotype result of the user's mutation detected above and based on existing scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

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6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Atorvastatin

Atorvastatin is a statins lipid regulating drug, which belongs to HMG CoA reductase inhibitor. The hydrolysate reduced the synthesis of cholesterol and increased the synthesis of low-density lipoprotein receptor. As a result, the levels of cholesterol and low-density lipoprotein cholesterol in blood decreased, the level of triglyceride in serum decreased moderately and the level of high-density lipoprotein in blood increased.

My Atorvastatin

Risk of Adverse Effect

ADVERSE EFFECT : Higher risk of congenital structural or functional abnormalities in the skeletal muscle; Consider alternative drug, or monitor constantly the symptoms of the muscles if no alternative drug is available.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. For the treatment of hypercholesterolemia and mixed hyperlipidemia.

2. For the prevention and treatment of coronary heart disease and stroke.

Adverse reactions

1. The most common adverse reaction is gastrointestinal discomfort. Others include headache, rash, dizziness, blurred vision and taste disturbance.
2. Occasionally, it can cause a reversible increase in blood aminotransferase. Therefore, liver function needs to be monitored.
3. Rare adverse reactions include impotence and insomnia.
4. Rare adverse reactions include myositis, myalgia and rhabdomyolysis, which are characterized by muscle pain, fatigue and fever, accompanied by elevated serum creatine phosphokinase and myoglobinuria. Rhabdomyolysis can lead to renal failure, but it is rare. This product can increase the risk of myopathy when combined with immunosuppressants, folic acid derivatives, niacin, gemfibrozil, erythromycin, etc.
5. There have been reports of hepatitis, pancreatitis and allergic reactions such as angioedema.

Contraindications

1. Patients who are allergic to atorvastatin are contraindicated. People who are allergic to other HMG-CoA reductase inhibitors should be used with caution.
2. It is contraindicated for patients with active liver disease or unexplained elevated blood aminotransferase.

Pregnant and lactating women

Since this product can cause fetal development in animal experiments and whether it is excreted in breast milk is unclear, it is not recommended for pregnant women and lactating mothers.

Children

Use in children is limited, and long-term safety has not been established.

Elderly

Elderly patients need to adjust the dose according to liver and kidney function.

Metabolism

Atorvastatin is mainly metabolized by the liver or eliminated by bile after extrahepatic metabolism, and its absolute bioavailability is low. This product is metabolized by cytochrome P4503A4 in the liver to a variety of active metabolites, most of which are excreted in the bile in the form of metabolites.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS4149056	SLCO1B1	CT	increased risk of primary structural or functional muscle disease

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The atorvastatin drug reaction test item uses the 1 polymorphic locus of the *SLCO1B* gene. The test results indicate the genotype result of the user's mutation detected above and based on existing scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Lovastatin

Lovastatin is a kind of statin medication used to treat hyperlipidemia and reduce the risk of cardiovascular disease.

My Lovastatin

Use With Drug Labeling

EFFICACY : Patients with rs662799-AA genotype, compared to AG or GG genotype, may have a higher reduction in LDL-cholesterol; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. For the treatment of hypercholesterolemia and mixed hyperlipidemia.

Adverse reactions

1. The most common adverse reactions of this product are gastrointestinal discomfort, diarrhea, flatulence, headache, rash, dizziness, blurred vision and taste disturbance.

- 2. Occasionally, it can cause a reversible increase in blood aminotransferase. Therefore, liver function needs to be monitored.
- 3. Rare adverse reactions include impotence and insomnia.
- 4. Rare adverse reactions include myositis, myalgia and rhabdomyolysis, which are characterized by muscle pain, fatigue and fever, accompanied by elevated serum creatine phosphokinase and myoglobinuria. Rhabdomyolysis can lead to renal failure, but it is rare. This product can increase the risk of myopathy when combined with immunosuppressants, folic acid derivatives, niacin, gemfibrozil, erythromycin, etc.
- 5. There have been reports of hepatitis, pancreatitis and allergic reactions such as angioedema.

 Contraindications

- 1. Patients who are allergic to lovastatin are contraindicated. People who are allergic to other HMG-CoA reductase inhibitors should be used with caution.
- 2. It is contraindicated for patients with active liver disease or unexplained elevated blood aminotransferase.

 Pregnant and lactating women

Since this product can cause fetal development in animal experiments and whether it is excreted in breast milk is unclear, it is not recommended for pregnant women and lactating mothers.

 Children

Use in children is limited, and long-term safety has not been established.

 Elderly

Elderly patients need to adjust the dose according to liver and kidney function.

 Metabolism

Oral absorption is good, but absorption is reduced by 30% on an empty stomach. This product is extensively metabolized in the liver and hydrolyzed into a variety of metabolites.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS662799	APOA5	AA	higher levels of LDL-cholesterol reduction

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The lovastatin drug reaction test item uses the 1 polymorphic locus of the *APOA5* gene. The test results indicate the genotype result of the user's mutation detected above and based on existing

scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

 Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Pravastatin

Pravastatin is a statin medication, used for preventing cardiovascular disease in those at high risk and treating abnormal lipids.

My Pravastatin

Risk of Adverse Effect

ADVERSE EFFECT : Patients with rs20455-GG genotype, compared to AA genotype, may have an increased response to pravastatin, but have an increased risk of coronary heart disease.

DOSE : Patients with rs20455-AA genotype, compared to GG genotype, may have a decreased response to pravastatin, dose increase is recommended. Patients with rs4149056-CT genotype, compared to TT genotype, may have increased plasma concentration of pravastatin; Consider drug dose reduction.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. For the prevention of paroxysmal ventricular tachycardia, paroxysmal supraventricular tachycardia and pre-excitation syndrome with supraventricular tachycardia, atrial flutter or atrial fibrillation.
2. It can also be used for the treatment of various premature beats.

Adverse reactions

1. The early adverse reactions include headache, dizziness, followed by gastrointestinal disorders, such as nausea, vomiting, constipation and so on. There are also atrioventricular block symptoms. There were two cases of cholestatic liver injury after continuous administration for two weeks. The activities of various enzymes returned to normal 2 ~ 4 weeks after withdrawal. It is believed that this pathological change belongs to allergic reaction and individual factors.
2. During the trial process, there was no damage to the lung, liver and hematopoietic system. A small number of patients experienced such minor reactions as dry mouth, headache, dizziness, and gastrointestinal discomfort. Generally, the symptoms disappeared after stopping the drug or reducing the dose. It has been reported that individual patients have atrioventricular block, prolonged Q-T interval, slightly prolonged P-R interval, and prolonged QRS time.

Contraindications

1. Patients with sinus node dysfunction, severe atrioventricular block, double bundle branch block without pacemaker protection, severe congestive heart failure, cardiogenic shock, and severe hypotension are contraindicated.
2. People who are allergic to the drug are prohibited.

Pregnant and lactating women

The safety and effectiveness of the application in pregnant women are still uncertain, so it is only used when the drug is beneficial to the fetus. It is not known whether the drug is present in breast milk, and it is recommended that women who are breastfeeding should stop using it.

Children

The safety and effectiveness of the drug in children are unclear.

Elderly

There is no increase in age-related side effects of the drug in elderly patients. However, elderly patients may experience a drop in blood pressure after medication. In addition, elderly patients are prone to damage to liver and kidney function, so they should be used with caution. The effective drug dose for elderly patients is lower than normal.

Metabolism

Mainly metabolized by the liver, the metabolite 5-hydroxy-propafenone has pharmacological activity, 90% of the metabolites are excreted from the kidneys, and about 1% of the original drug is excreted

by the kidneys.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS17244841	HMGCR	AA	normal effect of pravastatin
RS20455	KIF6	GG	pravastatin is more effective, but may cause higher risk of coronary heart disease
RS4149015	SLCO1B1	AA	slightly poor absorption of pravastatin
RS4149056	SLCO1B1	CT	increased serum concentration of pravastatin

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only

and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The pravastatin drug reaction test item uses the 4 polymorphic loci of the *HMGCR*, *KIF6* and *SLCO1B1* genes. The test results indicate the genotype result of the user's mutation detected above and based on existing scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Rosuvastatin

Rosuvastatin is a kind of statin drug, which is combined with exercise, diet control and weight loss to treat hypercholesterolemia and other related symptoms. It is also used to prevent cardiovascular disease.

My Rosuvastatin

Risk of Adverse Effect

ADVERSE EFFECT : Patients with rs4149056-CT genotype, compared to TT genotype, may have increased plasma concentration of rosuvastatin; Be alert to adverse effect.

EFFICACY : Patients with rs2231142-GG genotype, compared to TT genotype, may have a decreased drug response; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. This product is suitable for primary hypercholesterolemia (type IIa, including heterozygous familial hypercholesterolemia) or mixed dyslipidemia (type IIb) that cannot properly control

dyslipidemia after diet control and other non-drug treatments (such as exercise therapy, weight loss).

2. This product is also suitable for patients with homozygous familial hypercholesterolemia, as an auxiliary treatment for diet control and other lipid-lowering measures (such as LDL removal therapy), or used when these methods are not applicable.



Adverse reactions

The adverse reactions seen with this product are usually mild and transient. In controlled clinical trials, less than 4% of patients withdrew from the trial due to adverse events. The frequency of adverse events is arranged in the following order: common (incidence rate $>1/100$, $<1/10$); rare ($>1/1000$, $<1/100$); rare ($>1/10000$, $<1/1000$); Very rare ($<1/10000$).

- Immune system abnormalities (rare: allergic reactions, including angioneuroedema)
- Nervous system abnormalities (common: headache and dizziness)
- Gastrointestinal abnormalities (common: constipation, nausea, abdominal pain)
- Skin and subcutaneous tissue abnormalities (rare: pruritus, rash and urticaria)
- Skeletal muscle, joint and bone abnormalities (common: myalgia; rare: myopathy and rhabdomyolysis)
- Systemic abnormalities (common: weakness)

Like other HMG-CoA reductase inhibitors, the incidence of adverse reactions of this product tends to increase with increasing dose.

- Effects on the kidneys: Proteinuria (test paper test) was observed in patients receiving rosuvastatin calcium tablets, and most of the protein originated from the renal tubules. About 1% of patients had no or minimal increase in proteinuria to ++ or more during certain periods of the 10mg and 20mg treatments. Among the 40mg treatment patients, this proportion was about 3%. In the 20 mg dose treatment, a slight increase in proteinuria from no or slight increase to + was observed. In most cases, proteinuria automatically decreases or disappears after continuing treatment.
- Effect on skeletal muscle: In patients receiving various doses of this product, there are reports of effects on skeletal muscle, such as myalgia, myopathy, and rare rhabdomyolysis, especially in patients with doses greater than 20mg middle.
- The increase in creatine kinase (CK) levels has been observed in patients taking this product in a dose-related manner; most cases are mild, asymptomatic and transient. If the CK level increases ($>5 \times \text{ULN}$), treatment should be discontinued.
- Effects on the liver: Like other HMG-CoA reductase inhibitors, dose-related transaminase elevations have been observed in a small number of patients taking this product; most cases are mild, asymptomatic and transient.



Contraindications

1. Those who are allergic to rosuvastatin.
2. Patients with active liver disease, including patients with unexplained continuous elevation of serum transaminase and any patients whose serum transaminase rises more than 3 times the upper limit of normal (ULN).
3. Patients with severe renal impairment (creatinine clearance rate $<30\text{ml/min}$).

- 4. Patients with myopathy.
- 5. Patients who use cyclosporine at the same time.
- 6. During pregnancy, breastfeeding, and women who may become pregnant without using appropriate contraceptive measures.



Pregnant and lactating women

Forbidden to take



Children

The safety and effectiveness of this product in children have not been established. The experience of pediatric use is limited to a few (age> 8 years) children with homozygous familial hypercholesterolemia. Therefore, it is not recommended to use this product in pediatrics at present.



Elderly

No need to adjust the dose.



Metabolism

The plasma concentration reached its peak 5 hours after oral administration, and the absolute bioavailability was 20%. Rosuvastatin is a weak substrate of cytochrome P450 metabolism. About 90% of the dose of rosuvastatin is excreted in the feces in its original form (including absorbed and unabsorbed active substances), and the rest is excreted in urine. Approximately 5% of urine is the original form.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS2231142	ABCG2	GG	poor drug response
RS4149056	SLCO1B1	CT	higher risk of adverse events
RS4693075	COQ2	CC	lower risk of adverse muscle reaction

How to use the test results



Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The rosuvastatin drug reaction test item uses the 3 polymorphic loci of the *ABCG2*, *SLCO1B1* and *COQ2* genes. The test results indicate the genotype result of the user's mutation detected above and based on existing scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

-
1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
 2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
 3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Digoxin

Digoxin is a medium effect cardiac glycoside drug. Its effect on the heart is positive inotropic. It can enhance myocardial contractility, improve pump function, slow down heart rate, inhibit myocardial conduction system, increase cardiac output and output, and improve pulmonary circulation and systemic circulation.

My Digoxin

Consider Dose Reduction

DOSE : Patients with rs1045642-AG genotype, compared to GG genotype, may have decreased metabolism and increased plasma concentration of digoxin, may require decreased dose of drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Used for acute and chronic cardiac insufficiency.
2. Used to control the ventricular rate and supraventricular tachycardia of patients with atrial fibrillation and atrial flutter accompanied by rapid ventricular rate.

Adverse reactions

1. Common adverse reactions include arrhythmia, loss of appetite or nausea, vomiting (stimulation of the medulla oblongata), lower abdominal pain, and abnormal weakness.
2. Uncommon reactions include blurred vision or "chromatopsia" (such as yellow vision, green vision), diarrhea, and central nervous system reactions such as depression or confusion.
3. Rare reactions include drowsiness, headache and rash, urticaria (allergic reaction).
4. Among the poisoning manifestations of digitalis, pro-arrhythmia is the most important, and the most common one is ventricular premature beats, which accounts for about 33% of the adverse reactions of proarrhythmia. Followed by atrioventricular block, paroxysmal or accelerated junctional tachycardia, paroxysmal atrial tachycardia with atrioventricular block, ventricular tachycardia, sinus arrest, ventricular fibrillation and so on. Arrhythmia in children is more common than other reactions, but ventricular arrhythmia is less common than in adults. Newborns may have a prolonged P-R interval.

Contraindications

1. Forbidden to use it in combination with calcium injections.
2. Forbidden for anyone who is poisoned by cardiac glycoside preparations.
3. Forbidden for people with ventricular tachycardia and ventricular fibrillation
4. Forbidden for people with obstructive hypertrophic cardiomyopathy (applicable if accompanied by systolic insufficiency or atrial fibrillation).
5. Forbidden for people with preexcitation syndrome with atrial fibrillation or flutter.

Pregnant and lactating women

This product can pass through the placenta, so the maternal dosage in the late pregnancy may increase, and the dosage must be reduced 6 weeks after delivery. This product can be excreted into human milk, and the pros and cons must be weighed when using it.

Children

The tolerance of neonates to this product is uncertain, and their renal clearance is reduced; premature and immature infants are sensitive to this product, and the dose should be reduced according to their immaturity. In terms of body weight or body surface area, infants over 1 month use a slightly larger amount than adults.

Elderly

The elderly with liver and kidney dysfunction, decreased apparent volume of distribution or imbalanced electrolyte have low tolerance to this product and must reduce the dose.

Metabolism

Digoxin is excreted quickly in the body and has a small accumulation. Its transformation and metabolism in the body are small, and it is mainly eliminated by the kidneys in its original form.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1045642	ABCB1	AG	lower dose of drug is required

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The digoxin drug reaction test item uses the 1 polymorphic locus of the *ABCB1* gene. The test results indicate the genotype result of the user's mutation detected above and based on existing scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Ethanol

In medical use, ethanol is often used as excipient in drugs such as injection, or as disinfectant with a volume fraction of 70% ~ 75%.

My Ethanol

Risk of Adverse Effect

ADVERSE EFFECT : Patients with rs671-GG genotype, compared to AG or AA genotype, may be at an increased risk of developing alcoholism.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Applications

1. Commonly used in cold cough syrup, Huoxiang Zhengqi water, Danhong Huayu oral liquid, hydrocortisone injection, nitroglycerin injection, bromhexine hydrochloride glucose injection, paclitaxel injection and other drugs as excipients.

Adverse reactions

Allergic reactions: local red bumps, systemic pruritus, etc.

Contraindications

1. It is forbidden for those who are allergic to ethanol.
2. Ethanol also has the effects of inhibiting the central nervous system, dilating blood vessels, and enhancing the activity of liver drug enzymes. Therefore, special attention should be paid to the concurrent use of Chinese and Western medicines with similar effects.
3. Try to avoid combined use with drugs that can produce "disulfiram-like" reactions, such as glibenclamide and gliclazide in hypoglycemic drugs, cephalosporins and metronidazole in antibacterial drugs.



Pregnant and lactating women

Pregnant women use it with caution.



Children

Use with caution in premature infants, newborns and people with liver dysfunction.



Elderly

Normal use.



Metabolism

Except for a very small amount of alcohol (ethanol) ingested in the body, it is excreted through breathing and urine, and more than 95% is catabolized and metabolized in the body, and the liver is an important organ for ethanol metabolism. The catabolism of alcohol in the human body mainly relies on two enzymes: one is alcohol dehydrogenase and the other is acetaldehyde dehydrogenase. Alcohol dehydrogenase can oxidize ethanol to acetaldehyde. The acetaldehyde dehydrogenase can oxidize acetaldehyde to acetic acid.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS671	ALDH2	GG	higher risk of alcohol addiction

How to use the test results



Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or

professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The alcohol drug reaction test item uses the 1 polymorphic locus of the *ALDH2* gene. The test results indicate the genotype result of the user and based on existing scientific research, interpret how this genotype will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.

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2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
 3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
 5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Naloxone

Naloxone can alleviate the effects of excessive intake of opiates. It can be mixed with opiates to reduce the risk of poisoning in misuse.

My Naloxone

Risk of Insufficient Efficacy

EFFICACY : Patients with rs1799971-AA genotype, compared to AG or GG genotype, may have lower cortisol response when treated with naloxone; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Naloxone is mainly used for acute poisoning of morphine analgesics to relieve respiratory depression and central depression symptoms and make comatose patients recover quickly. It can also be used for the experimental diagnosis of withdrawal syndrome in drug addicts. In addition, naloxone can also be used for alcoholism to relieve respiratory depression and wake up.

Adverse reactions

This product has mild drowsiness, occasionally nausea, vomiting, tachycardia, high blood pressure and irritability.

Contraindications

1. People who are allergic to this product are forbidden.

Pregnant and lactating women

1. The reproductive toxicity test and dosage on mice and rats were 4 to 8 times the usual human dosage. The results did not show that naloxone has embryo toxicity or teratogenic toxicity. Since sufficient and effective research has not been conducted in pregnant women, pregnant women should only consider medication when necessary.
2. Maternal dependence is often accompanied by fetal dependence, so the risk to the fetus should be considered before using this product for pregnant women with known or suspected opioid dependence. Naloxone can pass through the placenta and induce withdrawal symptoms in mothers and fetuses. Patients with mild to moderate hypertension who use naloxone during labor should be closely monitored to avoid severe hypertension.
3. It is not clear whether this product affects labor and/or delivery time. However, published studies show that the use of naloxone during labor does not have adverse effects on mothers and newborns.
4. It is not clear whether this product will be secreted through human milk. Because the drug may be secreted into human milk, lactating women should use this product with caution.

Children

The use of this product on children or newborns can reverse the effects of opioids. Children with opioid poisoning have a strong response to this product, so they need to be closely monitored for at least 24 hours until the product is completely metabolized. The product is administered to the mother shortly after delivery, and the effect on prolonging the life of the newborn can only be maintained for 2 hours. If necessary, this product can be used directly on the newborn after delivery

Elderly

There are not enough clinical trials using this product in patients 65 years and older, and no difference in response to naloxone between elderly patients and young patients has been found. Under normal circumstances, the dose selection of elderly patients needs to be cautious. Considering that the liver, kidney, or heart function is reduced and the chances of concomitant disease or other drug treatment are greater, the drug should be started from a small dose.

Metabolism

It is mainly metabolized in the liver, and the metabolites are excreted in the urine after combining with glucuronic acid.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1799971	OPRM1	AA	poor drug response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

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Test details

Applicable situation

The naloxone drug reaction test item uses the 1 polymorphic locus of the *OPRM1* gene. The test results indicate the genotype result of the user and based on existing scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
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6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Leucovorin

Leucovorin is a derivative of folic acid. It is mainly used as an antidote for folic acid antagonists (such as methotrexate, pyrimidine or trimethoprim).

My Leucovorin

Use With Drug Labeling

ADVERSE EFFECT : Patients with rs1801019-CG genotype, compared to CC genotype, may have decreased risk of toxicity.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

1. Mainly used as an antidote for folic acid antagonists (such as methotrexate, pyrimethamine or trimethoprim, etc.).
2. Used to prevent severe toxic effects caused by methotrexate overdose or high-dose treatment.
3. For megaloblastic anemia caused by folic acid deficiency. When oral folic acid is not effective, it is also used for stomatitis diarrhea, malnutrition, megaloblastic anemia caused by pregnancy or infancy, but it is not suitable for vitamin B12 deficiency anemia.

4. When combined with fluorouracil, it is used to treat advanced colon cancer, rectal cancer and gastric cancer.

 Adverse reactions

Rarely, occasional rash. Allergic reactions such as measles or asthma.

 Contraindications

1. Patients with megaloblastic anemia caused by pernicious anemia and vitamin B12 deficiency should not use this product.

 Pregnant and lactating women

Although animal studies have not shown that this product has reproductive toxicity, animal experiments cannot accurately predict human response. Since leucovorin calcium has not been properly and fully controlled clinical studies in pregnant women, this product should not be used during pregnancy unless it is absolutely necessary. It is not clear whether this product is secreted by human milk, lactating women should use this product with caution.

 Children

Children taking antiepileptic drugs should use this product with caution.

 Elderly

Studies have pointed out that when this product is used in combination with fluorouracil, the risk of serious gastrointestinal toxicity in elderly patients and/or weakened persons is increased. Elderly patients use this product with caution.

 Metabolism

Leucovorin is easily absorbed by oral administration. The time for the serum reduction of leucovorin to reach the peak value was $1.72 \pm 0.8h$ after oral administration and $0.71 \pm 0.09h$ after intramuscular injection. The half-life of leucovorin reduced by intramuscular injection is 3.5h. Regardless of the route of administration, the duration of the drug's action is 3 to 6 hours. Calcium folinate is metabolized to 5-methyltetrahydrofolate by the liver and intestinal mucosa, and the metabolism is faster and more sufficient after oral administration than intramuscular injection. 80% to 90% of metabolites are excreted by the kidneys, and 5% to 8% are excreted in feces.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS1801019	UMPS	CG	normal toxicity risk

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The leucovorin drug reaction test item uses the 1 polymorphic locus of the *UMP5* gene. The test results indicate the genotype result of the user and based on existing scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

 Notification

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2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
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5. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.
6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Latanoprost

Latanoprost is a new phenyl substituted propyl ester prostaglandin F2a, which is a selective F2a receptor agonist.

My Latanoprost

Risk of Insufficient Efficacy

EFFICACY : Patients with rs3753380-CT genotype and open angle glaucoma, compared to CC genotype, may have a decreased response to latanoprost; Be alert to insufficient efficacy.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

It is used to treat glaucoma and ocular hypertension, as well as various conditions of increased intraocular pressure.

Adverse reactions

This product is usually well tolerated, with occasional blurred vision, burning pain, tingling, conjunctival hyperemia, transient punctate corneal erosion and foreign body sensation. Some patients also have brown pigmentation of the iris (7% after 6 months and 16% after 12 months).

Pigment increase is more common in races with greenish-brown, blue-gray-brown, or yellow-brown irises, and is rarer in races with pure blue, blue-gray, or green. This is caused by the stimulation of melanin formation, and the progress can be stopped after stopping the drug, but it is obviously unable to recover.

Contraindications

1. People who are known to be allergic to the ingredients of this product.
2. Wearer of corneal contact lens (contact lens).

Pregnant and lactating women

It is forbidden for pregnant and lactating women.

Children

The safety and effectiveness of drug use in children have not been established. This product is not recommended for children.

Elderly

There was no difference with adults.

Metabolism

It is a prodrug of isopropyl esterification and has no activity. It has biological activity after hydrolysis and conversion to latanoprost acid. Metabolism mainly occurs in the liver.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS3753380	PTGFR	CT	may have decreased response

How to use the test results

Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.

Test content

Different genetic test versions and data sources may have different coverage of the above variants.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

Test results are limited by the current technology and the level of scientific cognition. Depends on product package purchased, the test report may not cover all genes or loci related in each drug response. The accuracy of existing scientific research and the inherent detection error rate associated with the type of detection you purchase may also affect the accuracy of the interpretation of the item.

Test details

Applicable situation

The latanoprost drug reaction test item uses the 1 polymorphic locus in the intergenic region. The test results indicate the genotype result of the user and based on existing scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

1. Limited by the current scientific research, and the test report may not cover all genes or loci that are related to the test item.
2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.

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3. Limited by the technology of genetic testing, and the locus may be detected incorrectly. If the detection error occurs in the relevant locus of the report, the accuracy of the interpretation of the item may be affected.
 4. In addition to being affected by the genes in the report, drug metabolism will also be affected by other genetic factors, the physiological condition of the user, other drug use conditions, living environment and lifestyle factors.
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 6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.

Prednisolone

Prednisolone is an oral glucocorticoid drug, usually used to suppress the immune system and reduce inflammation, such as asthma, chronic obstructive pulmonary disease and rheumatism. Prednisone is hydrogenated to prednisolone by hydroxysteroid 11-beta dehydrogenase 1 (HSD11B1), so prednisolone may be more suitable for patients with liver disease.

My Prednisolone

Use With Drug Labeling

ADVERSE EFFECT : Pediatric patients with rs738409-CC genotype, compared to CG or GG genotype, may have decreased risk of hepatotoxicity; Administer standard dose of the drug.

Reading instructions

Explanation

The recommendations presented in this report are based on sites/haplotypes detected with higher levels of evidence in the relevant guidelines. The specific loci can be viewed in the "Scientific Evidence" section.

Notification

Due to loci selection and detection, the report may not cover all the known loci and cannot be directly used as the basis for clinical medication.

Knowledge

Indications

For allergic and autoimmune inflammatory diseases and collagen diseases, which may include rheumatism, rheumatoid arthritis, lupus erythematosus, severe bronchial asthma, nephrotic syndrome, thrombocytopenic purpura, granulocytopenia, acute lymphatic leukemia, various adrenal insufficiency, exfoliative dermatitis, blister free neurodermatitis, eczema, etc.



Adverse reactions

Glucocorticoids have no obvious adverse reactions in the application of physiological dose replacement therapy. The adverse reactions mostly occur in the application of pharmacological doses, and are closely related to the course of treatment, dosage, type of medication, usage and route of administration. Common adverse reactions are as follows:

1. Long-term use can cause the following side effects: face and posture of iatrogenic Cushing syndrome, facial appearance and posture, weight gain, lower extremity edema, purple streaks, tendency to bleeding easily, poor wound healing, acne, menstrual disorders, avascular necrosis of the humeral or femoral head, osteoporosis and fractures (including spinal compression fractures, pathological fractures of long bones), muscle weakness, muscle atrophy, hypokalemia syndrome, gastrointestinal irritation (nausea, vomiting), pancreatitis, peptic ulcer or perforation, Children's growth is inhibited, glaucoma, cataracts, benign intracranial pressure syndrome, impaired glucose tolerance, and increased diabetes.
2. The patient may have psychiatric symptoms: euphoria, agitation, delirium, restlessness, disorientation, or inhibition. Mental symptoms are caused by people who are prone to occur and suffer from chronic wasting diseases and those who have had mental disorders in the past.
3. Concurrent infection is the main adverse reaction of adrenal cortex hormones. Mainly fungi, tuberculosis, staphylococcus, proteus, *Pseudomonas aeruginosa* and various herpes viruses.
4. Glucocorticoid withdrawal syndrome. Sometimes patients have dizziness after stopping the medication, fainting tendency, abdominal pain or back pain, low fever, loss of appetite, nausea, vomiting, muscle or joint pain, headache, fatigue, weakness, after careful examination, if the adrenal cortex dysfunction and the original disease can be ruled out, and then it can be considered as a dependency syndrome on glucocorticoids.



Contraindications

1. People who are allergic to this product and steroid hormone drugs should not be used.
2. Patients with the following diseases are generally not suitable for use. Under special circumstances, the pros and cons should be weighed, and attention should be paid to the possibility of deterioration: severe mental illness (past or present) and epilepsy, active peptic ulcer disease, recent gastrointestinal anastomosis, fractures, trauma During the repair period, corneal ulcers, adrenal hyperfunction, hypertension, diabetes, pregnant women, infections that cannot be controlled by antibacterial drugs such as chickenpox, measles, fungal infections, severe osteoporosis, etc.



Pregnant and lactating women

Try to avoid.

- Medication during pregnancy: glucocorticoids can pass through the placenta. Animal experiments have confirmed that administration during pregnancy can increase the incidence of embryonic cleft palate, placental insufficiency, spontaneous abortion and intrauterine growth retardation.

Human use of pharmacological doses of glucocorticoids can increase the incidence of placental insufficiency, neonatal weight loss or stillbirth.

- Medication during lactation: Because glucocorticoids can be excreted in milk, it can cause adverse effects on infants, such as growth inhibition and adrenal cortex function inhibition. Pregnant and lactating women should avoid using it as much as possible after weighing the pros and cons.



Children

If children use adrenal cortex hormones for a long time, they must be very cautious.



Elderly

Glucocorticoids used in elderly patients are prone to hypertension and diabetes. Elderly patients, especially women after menopause, are prone to aggravate osteoporosis with glucocorticoids.



Metabolism

This product is an adrenal cortex hormone drug. It has a variety of pharmacological effects such as anti-inflammatory, anti-allergic and immune suppression. In the body, the liver has the highest content, followed by plasma. Cerebrospinal fluid, pleural and ascites fluid also have a certain content, but less in the kidney and spleen. It is excreted in urine after metabolism.

My Genetic Result Details

Gene locus	Gene name	My genotype	Description
RS738409	PNPLA3	CC	normal risk of hepatotoxicity

How to use the test results



Notification

The test does not yet have the qualifications for clinical diagnosis. The test results and medication recommendations provided are for reference only and cannot be directly used as the basis for clinical medication. If you have relevant medication needs, you can consult authoritative clinicians or professionals based on the results of this genetic test to obtain professional medication recommendations, or follow the doctor's advice for clinically qualified inspection and test.



Test content

Different genetic test versions and data sources may have different coverage of the above variants.



Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Reference information

The relevant information of the drug comes from the template of the chemical drug leaflet published by the National Medical Products Administration, the drug insert approved for marketing in Mainland China, and other publicly available reference information. The information displayed in the genetic test report (including but not limited to drug introduction, indications, interactions, adverse reactions, contraindications, metabolism, etc.), in principle, is the description of the single active ingredient of the drug, and may not include the influence of factors such as drug formulations, excipients, and compound preparations. Therefore, the information in this report is for reference only and cannot be used as a substitute for the instructions of the drug. Before using a specific drug, be sure to read its instructions carefully. When there is a conflict between the drug insert and the information in this report, please refer to and adopt the contents of the drug insert first.

Limit of detection

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Test details

Applicable situation

The prednisolone drug reaction test item uses the 1 polymorphic locus of the *PNPLA3* gene. The test results indicate the genotype result of the user and based on existing scientific research, interpret how genes will affect the use of the drug. Different genetic test versions and data sources may have different coverage of the above variants.

Notification

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2. Limited by the technology of genetic testing, and the test report may not cover all genes or loci that are related to the test item.
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environment and lifestyle factors.

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6. The basis of the test is mainly from the research of Caucasian and East Asian, other populations are for reference only.



Infection Risk

12 Reports



Virus

Hepatitis Virus	Infection risk is slightly high
Norovirus	Infection risk is moderate
HIV	Infection risk is slightly low
HPV	Infection risk is slightly high



Bacterium

M. tuberculosis	Infection risk is moderate
M. leprae	Infection risk is moderate
H. pylori	Infection risk is slightly high



Fungus

Candida	Infection risk is slightly low
Cryptococcus	Infection risk is slightly low



Parasite

Entamoeba	Infection risk is slightly high
Plasmodium	Infection risk is slightly high



Other

Prion	Infection risk is moderate

Hepatitis Virus

The virus can cause liver diseases. As the most common infection types in Chinese, hepatitis B virus (HBV) and hepatitis C virus (HCV) can lead to liver inflammation, cirrhosis, and even liver cancer. HBV is a DNA virus with vaccine, but it is difficult to cure after infection, and it is easy to progress to chronic hepatitis B; HCV is an RNA virus with mild infection symptoms. It is easy to persist undetected and progress to chronic hepatitis C. HCV can be cured and currently has no vaccine available.

My Hepatitis Virus

Infection risk is slightly high

Among the 22 genotypes associated with the risk of HBV infection, the weighted risk score is higher than the average of the population, and the infection risk is slightly high.

My Hepatitis Virus

Severity of infection is slightly high

Among the 8 genotypes associated with the severity of HBV infection, the weighted risk score is close to the average of the population, and the accumulation of virus and the aggravation of the condition is moderate when infected with HBV. Among the 20 genotypes associated with the severity of HCV infection, the weighted risk score is higher than the average of the population, and the accumulation of virus and the aggravation of the condition is slightly high when infected with HCV.

My Hepatitis Virus

Genetic risk of secondary diseases is moderate

People with the rs1012068-GT genotype may have a higher risk of progression to liver cancer when infected with HCV.

Suggestions

1. Practice safe sex and Use condoms correctly.

2. Do not share needles, and refuse unsafe injection or blood transfusion.
3. Vaccination: Hepatitis B vaccine is the most effective way to prevent hepatitis B virus infection, especially for newborns.
4. Carry out hepatitis virus related tests.

Influencing factors



Controllable factors

1. Body fluid contact: Blood, semen, vaginal secretions, milk and other body fluids of hepatitis B and C virus-infected persons are the main source of infection. For example, having sex with an infected person.
2. Medical devices: Shared or repeatedly used needles and syringes; Unsafe injection, blood transfusion and cutting or puncture without sufficient disinfection will increase the risk of hepatitis B and C virus infection.



Uncontrollable factors

1. Mother-to-child transmission: Hepatitis B virus can be transmitted from mother to child, including intrauterine transmission, transmission during delivery and postpartum transmission.
2. Heredity: Research shows that some people are prone to hepatitis virus infection due to genetics.

Knowledge



Predisposing age

All age groups are susceptible to the virus.

- Hepatitis B virus:
 - Infection at birth: chronic infection and chronic hepatitis.
 - Adult infection: incubation period is 75 days (or up to 6 months).
 - It may progress to chronic hepatitis.
- Hepatitis C virus:
 - The incubation period is 42-63 days (or up to 6 months).
 - 75-90% of the infected people progress to chronic hepatitis.



Prevalence

- Hepatitis B virus: According to the statistics of the World Health Organization (WHO) in 2017, 257 million people worldwide are suffering from chronic hepatitis B virus infection, and about 887,000

people died of hepatitis B virus infection related diseases. The prevalence rate of hepatitis B virus infection in China is about 5-6%, and there are about 70 million people with chronic hepatitis B.

- Hepatitis C virus: According to the statistics of the World Health Organization (WHO) in 2015, 71 million people worldwide are suffering from chronic hepatitis C virus infection, and about 399,000 people died of liver cirrhosis or primary hepatocellular carcinoma caused by hepatitis C virus infection. The prevalence of hepatitis C virus infection in China is about 0.43%, and there are about 10 million people with chronic hepatitis C.

Typical symptoms

- Hepatitis B virus:
 - Acute: Fatigue, nausea, vomiting, anorexia, jaundice, etc.
 - Chronic: Usually asymptomatic, may progress to liver cirrhosis, liver cancer.
- Hepatitis C virus: Usually asymptomatic, until the progression of cirrhosis.

Infection and heredity

Many studies have found that not all people infected with hepatitis B virus will become chronic hepatitis patients, and some of them can heal themselves without treatment. This may be due to the biological differences of hepatitis B virus itself and the differences in immune genetics of infected individuals. The researchers found that the adaptive T cell immune response to hepatitis B virus was particularly strong in people who recovered from acute infection with hepatitis B virus, while the adaptive T cell immune response to hepatitis B virus was weak in people with chronic infection. This shows that people who carry adaptive T cell genes encoding immune enhancement may be better able to resist hepatitis B virus infection; At the same time, T cell immune response is also an important factor and treatment idea to control hepatitis B virus infection.

Infection and hepatocellular carcinoma

In 2021, a study by *Nature Communications* found that hepatitis B virus quietly promoted the growth and survival of cancer cells. These viruses would integrate their DNA into liver cells at the early stage of tumor development, contributing to abnormal proliferation of liver cells. This kind of damage to the DNA of normal cells may have occurred 21 years before the diagnosis of cancer. The analysis results showed that the integration of viral DNA was random and could occur on any chromosome. In all integration events, the highest frequency occurred in the *TERT* gene on chromosome 5p: 11 times directly occurred in the *TERT* gene, and 9 times occurred in the upstream of the gene.

Treatment

- Hepatitis B virus:
 - Maximize the long-term inhibition of hepatitis B virus replication; Reduce or delay the deterioration and progress of hepatitis B virus-related diseases.
 - Nucleoside (acid) analogues: Entecavir, tenofovir.
 - Interferon therapy.
- Hepatitis C virus:
 - Eliminate hepatitis C virus and get cured; Eliminate or reduce the deterioration and progress of hepatitis C virus-related diseases.

- Direct antiviral drugs: Daclatasvir, asunaprevir.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS12614	CFB	CC	may have a lower infection risk when infected with HBV
RS12979860	IFNL4	CC	may have a higher risk of accumulation of virus when infected with HBV
RS1883832	CD40	TT	may have a higher infection risk when infected with HBV
RS2856718	intergenic	CT	may have a higher infection risk when infected with HBV
RS3077	HLA-DPA1	AA	may have a higher infection risk when infected with HBV

For display consideration, only some genetic details are displayed.

My haplotype details

HLA-DRB1

People without the HLA-DRB1*12:01

[*15:01/*16:02](#)

genotype may have some risk of

accumulation of virus when infected with

HCV. People without the HLA-DRB1*01:01

genotype may have some risk of

accumulation of virus when infected with

HCV. People without the HLA-DRB1*04:01

genotype may have some risk of

accumulation of virus when infected with

HCV. People without the HLA-DRB1*13:01

genotype may have some infection risk when

infected with HBV. People without the HLA-

DRB1*03:01 genotype may have some

severity of infection when infected with HCV.

People without the HLA-DRB1*11:01

genotype may have some risk of

accumulation of virus when infected with

HCV. People without the HLA-DRB1*11:02

genotype may have some infection risk when

infected with HBV.

HLA-A	People without the HLA-A*11:01 genotype may have some risk of accumulation of virus when infected with HCV. People without the HLA-A*03:01 genotype may have some infection risk when infected with HBV. People with the HLA-A*03:01 genotype may have a lower infection risk when infected with HBV People without the HLA-A*02:01 genotype may have some risk of accumulation of virus when infected with HCV. People with the HLA-A*11:01 genotype may have a lower risk of accumulation of virus when infected with HCV	*11:01/*03:01
HLA-C	People without the HLA-C*06:02 genotype may have some infection risk when infected with HBV. People without the HLA-C*07:02 genotype may have some infection risk when infected with HBV. People with the HLA-C*07:02 genotype may have a higher infection risk when infected with HBV People without the HLA-C*01:02 genotype may have some risk of accumulation of virus when infected with HCV.	*07:02/*14:02

HLA-B	People without the HLA-B*57:01 genotype may have some risk of accumulation of virus when infected with HCV. People without the HLA-B*57:03 genotype may have some risk of accumulation of virus when infected with HCV.	*51:01/*07:02
HLA-DQB1	People without the HLA-DQB1*02:01 genotype may have some infection risk when infected with HBV. People without the HLA- DQB1*03:01 genotype may have some infection risk when infected with HBV. People without the HLA-DQB1*03:01 genotype may have some risk of accumulation of virus when infected with HCV.	*06:02/*05:02

Risk calculation

The risk value of this test is calculated based on published academic literature related to Hepatitis virus. By studying the genetic differences between Hepatitis virus-infected patients and normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of Hepatitis virus infection risk of the user, as well as the severity of infection in case one is infected and the changes of genetic risk of secondary diseases.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Hepatitis virus test report uses 16 polymorphic loci on *IFNL4*, *CD40*, *MUTYH* genes and intergenic regions; Also, genotyping results of *HLA-A*, *HLA-B*, *HLA-C*, *HLA-DRB1* and *HLA-DQB1*. The results showed whether the user had a higher infection risk, severity of infection and genetic risk of secondary diseases, of Hepatitis virus than all users in the genotype combination of the 16 polymorphic loci and *HLA-A*, *HLA-B*, *HLA-C*, *HLA-DRB1* and *HLA-DQB1* genotyping results tested, and that the test did not represent the user's actual outcome of Hepatitis virus infection. The basis of the test is mainly from the research of Caucasian, Hispanic, African and Asian, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk, severity of infection and genetic risk of secondary diseases, of Hepatitis virus.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Hepatitis virus at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing Hepatitis virus, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes genotyping results of *HLA-A*, *HLA-B*, *HLA-C*, *HLA-DRB1* and *HLA-DQB1*; And 16 polymorphic loci related to the risk of Hepatitis virus, such as rs74597329, rs1883832, rs3219487.

Population samples

The basis of the test is mainly from the research of Caucasian, Hispanic, African and Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with Hepatitis virus infection.

M. tuberculosis

This bacterium is a type of aerobic gram-positive bacteria, which is the pathogen causing tuberculosis and can invade all organs of the body, most of which is pulmonary tuberculosis.

My M. tuberculosis

Infection risk is moderate

Among the 30 genotypes associated with the risk of *M. tuberculosis* infection, the weighted risk score is close to the average of the population, and the infection risk is moderate.

Suggestions

1. Avoid smoking: Avoid active smoking or passive smoking; Prevent tobacco from damaging respiratory mucosal barrier.
2. Avoid close contact with tuberculosis patients; If it is necessary to contact patients, wear masks for protection; Avoid staying in crowded places for a long period of time.
3. Improve immunity: Healthy diet, Physical exercise, keeping a good attitude and get enough sleep are means to improve immunity.
4. Infants and young children should inoculate BCG vaccine as early as possible.

Influencing factors



Controllable factors

1. Tobacco: The use of tobacco will damage the respiratory mucosal barrier, reducing the ability to resist pathogens, and make host more vulnerable to the invasion of *M. tuberculosis*.
2. Inhalation of sputum: Tuberculosis patients will produce a large number of tiny sputum containing *M. tuberculosis* into the air, while healthy people will be inadvertently infected if they inhale sputum containing bacteria when breathing normally.
3. Immune function: When the body's immunity decreases, it will increase the risk of infection.



Uncontrollable factors

1. Age: It is generally believed that children and adolescents are susceptible groups, but the prevalence of tuberculosis in the elderly is also increasing.
2. Region: More than 95% of cases and deaths occur in developing countries; South East Asia and the Western Pacific region have the largest number of new tuberculosis cases, followed by Africa.
3. Disease: HIV/AIDS patients are 20-30 times more likely to carry active *M. tuberculosis* than normal people; Patients with other immune system impairments also have a higher risk of infection.
4. Heredity: Research shows that some people are prone to *M. tuberculosis* infection due to genetics.

Knowledge



Predisposing age

Tuberculosis can be seen at any age, mostly in children and adolescents; The prevalence of tuberculosis among the elderly in China is increasing year by year.



Prevalence

About 1% of the world's population is newly infected with the disease every year. Since 2000, the number of new cases in the world has declined year by year.



Typical symptoms

The common symptom is cough, which may be accompanied by sputum and blood. Other symptoms include chest pain, fatigue, weight loss, fever and night sweats.



Treatment

- Drug treatment: 5 first-line clinical drugs are isoniazid, rifampicin, ethambutol, pyrazinamide, and streptomycin, which are effective for more than 80% of newly infected tuberculosis patients.
- Surgical treatment: Surgical treatment is still an important alternative treatment for drug-resistant tuberculosis and massive hemoptysis.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS4240897	MFN2	GG	may have a higher infection risk when infected with <i>M. tuberculosis</i>
RS4331426	intergenic	AA	may have a lower infection risk when infected with <i>M. tuberculosis</i>

RS6114027	TGM6	CT	may have a higher infection risk when infected with <i>M. tuberculosis</i>
RS2269497	RGS12	GG	may have a higher infection risk when infected with <i>M. tuberculosis</i>
RS41553512	HLA-DRB5	CT	may have a higher infection risk when infected with <i>M. tuberculosis</i>

For display consideration, only some genetic details are displayed.

My haplotype details

HLA-B	People without the HLA-B*58:02 genotype may have some infection risk when infected with <i>M. tuberculosis</i>. People without the HLA-B*58:01 genotype may have some infection risk when infected with <i>M. tuberculosis</i>.	*51:01/*07:02
HLA-DRB1	People without the HLA-DRB1*09:01 genotype may have some infection risk when infected with <i>M. tuberculosis</i>.	*15:01/*16:02
HLA-DQB1	People without the HLA-DQB1*03:03 genotype may have some infection risk when infected with <i>M. tuberculosis</i>. People without the HLA-DQB1*02:01 genotype may have some infection risk when infected with <i>M. tuberculosis</i>.	*06:02/*05:02

Risk calculation

The risk value of this test is calculated based on published academic literature related to *M. tuberculosis*. By studying the genetic differences between *M. tuberculosis*-infected patients and

normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of *M. tuberculosis* infection risk of the user.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The *M. tuberculosis* test report uses 12 polymorphic loci on *ESRRB*, *RGS12*, *MFN2* genes and intergenic regions; Also, genotyping results of *HLA-B*, *HLA-DRB1* and *HLA-DQB1*. The results showed whether the user had a higher infection risk of *M. tuberculosis* than all users in the genotype combination of the 12 polymorphic loci and *HLA-B*, *HLA-DRB1* and *HLA-DQB1* genotyping results tested, and that the test did not represent the user's actual outcome of *M. tuberculosis* infection. The basis of the test is mainly from the research of Caucasian, African and Chinese, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk of *M. tuberculosis*.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of *M. tuberculosis* at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing *M. tuberculosis*, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes genotyping results of *HLA-B*, *HLA-DRB1* and *HLA-DQB1*; And 12 polymorphic loci related to the risk of *M. tuberculosis*, such as s12437118, rs2269497, rs4240897.



Population samples

The basis of the test is mainly from the research of Caucasian, African and Chinese.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with *M. tuberculosis* infection.

M. leprae

This bacterium is a type of aerobic gram-positive bacteria, which mainly invades the skin, mucosa and peripheral nerves, causing leprosy. Leprosy is a chronic infectious disease. Failure to treat it in time can lead to deformity of eyes, hands and feet, resulting in lifelong disability.

My M. leprae

Infection risk is moderate

Among the 48 genotypes associated with the risk of *M. leprae* infection, the weighted risk score is close to the average of the population, and the infection risk is moderate.

Suggestions

1. Avoid contacting the body fluid and rash of the person infected with *M. leprae*.
2. Improve immunity: Healthy diet, Physical exercise, keeping a good attitude and get enough sleep are means to improve immunity.

Influencing factors



Controllable factors

1. Infectious source: Untreated leprosy patients with skin or mucous membrane damage containing a large number of bacteria may infect healthy people by long-time contact; In addition, healthy people may also be infected if they inhale droplets exhaled by the patients.
2. Immune function: When the body's immunity decreases, it will increase the risk of *M. leprae* infection.



Uncontrollable factors

1. Sex: Male infection rate is high.
2. Age: People aged 5-15 and over 30.
3. Region: Brazil, India, Nepal, parts of Africa and the Western Pacific are high incidence regions.
4. Disease: Patients with basic immune deficiency, such as HIV/AIDS patients and cancer patients.

5. Heredity: Research shows that some people are prone to *M. leprae* infection due to genetics.

Knowledge



Predisposing age

Leprosy can be seen at any age, mostly in people aged 5-15 and over 30. *M. leprae* grows slowly, and the incubation period generally ranges from 6 months to 10 years.



Prevalence

Leprosy is an ancient disease that has been prevalent in the world for nearly 3,000 years. India, Egypt and China are considered as the three major leprosy foci in the world. At the end of last century, the World Health Organization (WHO) proposed a grand plan for the global eradication of leprosy. According to statistics, from 1985 to 2011, the prevalence rate of 10,000 people in the world has dropped from 21.1 to 0.37. In China, the infection rate of leprosy is extremely low, with only sporadic cases. Male infection is relatively more common.



Typical symptoms

Leprosy usually starts to show symptoms after infection for more than 1 year (average 5-7 years), and the progress of symptoms is also very slow.

- Tuberculoid: Skin lesions are mainly skin rashes, which are characterized by hypoesthesia, central pigment reduction, clear boundary and bulge.
- Lepromatous: In addition to skin and neuropathy, the infected person can also affect other organs, such as nasal cavity, testis and kidney. Skin lesions can be manifested as macules, papules, nodules or plaques, which are often symmetrical.
- Borderline: It has the characteristics of both leprosy types. If it is not treated in time, its symptoms may be reduced, which is similar to tuberculoid leprosy, or it may be aggravated, which is similar to lepromatous leprosy.



Treatment

Treatment depends on the form of leprosy and involves multidrug regimens.

- For multibacillary: dapsone, rifampicin, clofazimine.
- For paucibacillary: dapsone, rifampicin.

My genetic result details

Gene locus	Gene name	My genotype	Description

RS2275606	intergenic	GG	may have a lower infection risk when infected with <i>M. leprae</i>
RS77061563	intergenic	CC	may have a higher infection risk when infected with <i>M. leprae</i>
RS2844633	PSORS1C1	CC	may have a higher infection risk when infected with <i>M. leprae</i>
RS3095309	intergenic	TT	may have a higher infection risk when infected with <i>M. leprae</i>
RS142179458	intergenic	GG	may have a higher infection risk when infected with <i>M. leprae</i>

For display consideration, only some genetic details are displayed.

My haplotype details

HLA-DRB1	People without the HLA-DRB1*15:01 genotype may have some infection risk when infected with <i>M. leprae</i>. People with the HLA-DRB1*15:01 genotype may have a higher infection risk when infected with <i>M. leprae</i>	*15:01/*16:02
HLA-DQB1	People without the HLA-DQB1*04:01 genotype may have some infection risk when infected with <i>M. leprae</i>.	*06:02/*05:02

Risk calculation

The risk value of this test is calculated based on published academic literature related to *M. leprae*. By studying the genetic differences between *M. leprae*-infected patients and normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of *M. leprae* infection risk of the user.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-

genetic factors are not taken into account.

Test details

Applicable situation

The *M. leprae* test report uses 22 polymorphic loci on *IL12B*, *BATF3*, *LACC1* genes and intergenic regions; Also, genotyping results of *HLA-DRB1* and *HLA-DQB1*. The results showed whether the user had a higher infection risk of *M. leprae* than all users in the genotype combination of the 22 polymorphic loci and *HLA-DRB1* and *HLA-DQB1* genotyping results tested, and that the test did not represent the user's actual outcome of *M. leprae* infection. The basis of the test is mainly from the research of Chinese and Vietnamese, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk of *M. leprae*.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of *M. leprae* at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing *M. leprae*, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes genotyping results of *HLA-DRB1* and *HLA-DQB1*; And 22 polymorphic loci related to the risk of HPV, such as rs6871626, rs2221593, rs3764147.

Population samples

The basis of the test is mainly from the research of Chinese and Vietnamese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with *M. leprae* infection.

Entamoeba

Commonly known as amoeba, it is a kind of anaerobic parasite, which mainly parasitizes in the digestive tract of the host, causing amebic dysentery.

My Entamoeba

Infection risk is slightly high

People with the rs58994923-II genotype may have a higher infection risk when infected with *E. histolytica*.

Suggestions

1. Pay attention to diet, environment and personal hygiene:

- Prevent human feces from polluting food and water.
- Drinking water needs to be disinfected, for example, the residual chlorine concentration of drinking water reaches the standard; Do not drink raw water.
- Follow the Five key points for food safety when handling food.
- Proper hand washing: Wash hands with soap or hand sanitizer containing antibacterial agent; Wash hands with running water for at least 20 seconds.
- Hand washing habits: Wash hands before meals and after using the toilet; Wash hands before processing food; Wash hands after taking public transport; Take care of infants and children and wash their hands at any time; Pay attention to food and drinking water hygiene.

Influencing factors



Controllable factors

1. Food hygiene: Infection occurs through ingestion of contaminated food and drinking water.
2. Hygienic habits: Poor hygienic conditions will increase the risk of infection.



Uncontrollable factors

1. Other diseases: Some studies have shown that one infected with human immunodeficiency virus (HIV) is more likely to be infected with *Entamoeba*.
2. Region: In tropical and subtropical areas, the prevalence of *Entamoeba* infection is particularly serious.
3. Heredity: Research shows that some people are prone to *Entamoeba* infection due to genetics.

Knowledge



Predisposing age

All age groups are susceptible to the parasite. After *Entamoeba* invades the colon wall, the incubation period is 1-2 weeks.



Prevalence

According to research estimates, about 350,000 to 500,000 people worldwide are infected with *Entamoeba* and about 55,000 people die every year.



Typical symptoms

- Most patients infected with *Entamoeba* are asymptomatic, but cysts can be continuously discharged from their feces. Symptoms when colon tissue is invaded include:
 - Intermittent diarrhea and constipation
 - Gastrointestinal flatulence
 - Spasmodic abdominal pain
- Amebiasis can be caused by *Entamoeba histolytica*, which is particularly common in tropical areas. It is characterized by frequent semi-liquid diarrhea. Feces often contain blood, mucus and live trophozoites, often accompanied by abdominal tenderness.
- The *Entamoeba* infection of extraintestinal dysentery originates from colon infection and can affect any organ. However, a single abscess of the liver is the most common. The symptoms include pain or discomfort in the liver area. Sometimes the pain can radiate to the right shoulder, accompanied by intermittent fever, sweating, chills, nausea, vomiting, fatigue and weight loss.



Treatment

- Initial treatment: Metronidazole and tinidazole.
- Capsule clearance: Diiodohydroxyquinoline, paromomycin or diloxanide furoate.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS58994923	intergenic	II	may have a higher infection risk when infected with <i>E. histolytica</i>

Risk calculation

The risk value of this test is calculated based on published academic literature related to *Entamoeba*. By studying the genetic differences between *Entamoeba*-infected patients and normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of *Entamoeba* infection risk of the user.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The *Entamoeba* test report uses 1 polymorphic locus on *CREM* gene. The results showed whether the user had a higher infection risk of *Entamoeba* than all users in the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome of *Entamoeba* infection. The basis of the test is mainly from the research of Bangladeshi, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk of *Entamoeba*.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of *Entamoeba* at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing *Entamoeba*, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus related to the risk of *Entamoeba*, which is rs58994923.



Population samples

The basis of the test is mainly from the research of Bangladeshi.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with *Entamoeba* infection.

Candida

This fungus widely exists in nature and is one of the normal intestinal flora of human body. It is conditionally pathogenic with *Candida albicans* being the most common type in clinical practice, which can invade local skin, mucous membrane and various tissues and organs of the whole body, causing local or systemic infectious candidiasis.

My Candida

Infection risk is slightly low

Compared with the population average, the risk of *Candida* infection is slightly low.

Suggestions

1. Improve immunity: Healthy diet, Physical exercise, keeping a good attitude and get enough sleep are means to improve immunity.
2. Build good living habits: Avoid tight or airtight clothes and keep your body dry; Get enough sleep; Control sugar intake.

Influencing factors

Controllable factors

1. Immune function: When the body's immunity is weakened, it will increase the risk of candida infection.
2. Living habits: High stress level, staying up late, insufficient sleep, excessive intake of sweet food, wearing airtight clothes, and excessive sweating making body hot and humid, are risk factors of candida infection.

Uncontrollable factors

1. Hospitalization: For hospitalized patients, *Candida* is one of the important pathogens of nosocomial bacteremia.
2. Disease: Patients with basic immune deficiency, such as HIV/AIDS patients and cancer patients.
3. Heredity: Research shows that some people are prone to *Candida* infection due to genetics.

Knowledge

Predisposing age

All age groups are susceptible to *Candida* infection.

Prevalence

The prevalence rate is about 2.1-21/100,000, and the fatality rate is 40-60%.

Typical symptoms

- Oral infection (thrush):
 - Cream like, white, painful mucosal patches.
 - A cleft in the mouth (cheilitis).
 - Red, painful and smooth tongue.
- Esophageal infection: Plaque appears, causing swallowing pain or dysphagia.
- Skin infection: Burning rash.

Other symptoms include fever, heart murmur, splenomegaly, shock and oliguria. Retina and intraocular infection can cause blindness. If the infection is further aggravated, multiple organ failure or death may occur.

Treatment

Antifungal drugs are generally used for treatment.

- Skin, mouth or vagina infection: clotrimazole, nystatin.
- Esophageal infection: fluconazole, itraconazole; If the infection is aggravated, use anidulafungin, caspofungin, micafungin injection, oral or intravenous voriconazole, etc.
- Systemic infection: anidulafungin, caspofungin or micafungin are injected intravenously or fluconazole is injected intravenously or orally.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS4845320	intergenic	AA	may have a lower infection risk when infected with <i>Candida</i>

Risk calculation

The risk value of this test is calculated based on published academic literature related to *Candida*. By studying the genetic differences between *Candida*-infected patients and normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of *Candida* infection risk of the user.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The *Candida* test report uses 1 polymorphic locus on *LCE4A* gene. The results showed whether the user had a higher infection risk of *Candida* than all users in the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome of *Candida* infection. The basis of the test is mainly from the research of Caucasian and African American, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk of *Candida*.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of *Candida* at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing *Candida*, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus related to the risk of *Candida*, which is rs4845320.

 Population samples

The basis of the test is mainly from the research of Caucasian and African American.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with *Candida* infection.

Plasmodium

It is a kind of single celled and parasitic organism, which causes malaria in humans. *Plasmodium falciparum* and *Plasmodium vivax* are the most common types.

My Plasmodium

Infection risk is slightly high

Among the 18 genotypes associated with the risk of *P. falciparum* infection, the weighted risk score is higher than the average of the population, and the infection risk is slightly high.

Suggestions

1. Mosquito bites are an important transmission route of malaria, and Methods of preventing mosquito should be mastered.
2. Do not share needles and instruments, and refuse unsafe injection or blood transfusion to avoid infection caused by medical devices contaminated by *Plasmodium* parasites.
3. Improve immunity: Healthy diet, Physical exercise, keeping a good attitude and get enough sleep are means to improve immunity.
4. Preventive medication can be taken before entering High prevalence area of malaria.

Influencing factors



Controllable factors

1. Mosquito bites: Malaria is mainly transmitted through mosquito bites, especially female *Anopheles*.
2. Medical devices: *Plasmodium* parasites live in red blood cells, so needles and syringes that have been exposed to blood will increase the risk of infection if they are shared or repeatedly used.
3. Immune function: When the body's immunity decreases, it will increase the risk of infection.



Uncontrollable factors

1. Age: Children younger than 5 years old have a higher risk of infection.

-
2. Region: Malaria is prevalent in most tropical regions, such as Central Africa, South Asia, Southeast Asia and Latin America, among which Africa is the most prevalent.
 3. Mother-to-child transmission: The mother's blood containing *Plasmodium* parasites may enter the fetus through damaged placenta or skin of the fetus when passing through the birth canal, which may cause infection.
 4. Disease: Immune disease, malignant tumor, etc.
 5. Heredity: Research shows that some people are prone to *Plasmodium* infection due to genetics.
-

Knowledge

Infection process

When the female mosquito *Anopheles* bites and sucks the blood of a person with *Plasmodium* parasites, the parasites enter the mosquito body with the blood. Under the appropriate temperature conditions, the *Plasmodium* parasites develop and reproduce to form sporozoites. The sporozoites are stored at the mosquito's salivary gland, and will enter human body with saliva when the mosquito is sucking blood again. *Plasmodium* parasites invade the liver cells of the bitten person through blood flow to parasitize and reproduce, and then invade the red blood cells to reproduce after maturity, causing the red blood cells to break up in batches periodically and malaria occurs.

Predisposing age

All age groups are susceptible to the parasite, and children under 5 years old are at a higher risk of infection.

The incubation period for *Plasmodium* infection is usually:

- *Plasmodium falciparum*: 9-14 days.
- *Plasmodium vivax*: 12-17 days.
- *Plasmodium ovale*: 16-18 days or more.
- *Plasmodium malariae*: 18-40 days or more.

Prevalence

Malaria is the most harmful parasitic disease to human beings, spreading in 95 countries and infecting more than 3 billion people. According to the statistics of the World Health Organization (WHO), 241 million malaria cases and 627,000 deaths occurred globally in 2020.

Typical symptoms

- Common clinical manifestations of malaria include:
 - Fever and chills
 - Anemia
 - Jaundice

- Splenomegaly
- Hepatomegaly
- *Plasmodium falciparum*: It is the only *Plasmodium* parasite that may be fatal without treatment, and the disease is progressing most seriously.

Infection and heredity

The change of red blood cell membrane can protect the body against malaria. Duffy protein is a receptor on the red blood cell membrane, through which *Plasmodium vivax* can invade the red blood cell. This protein is inactive in the red blood cells of almost all West African populations and more than half of Africans, which means that their red blood cells are resistant to *Plasmodium vivax*. The appearance of red blood cells of some people living in Papua New Guinea is similar to breakfast cereal. This symptom free condition is usually called Southeast Asian ovalocytosis (SAO), which makes the rest of the structure of the red blood cell scaffold harder than normal cells, and can also help individuals effectively resist the invasion of *Plasmodium* parasites.

Treatment

- *Plasmodium falciparum*: Artemisinin-based combination therapy (ACT) is a combination of highly effective short acting artemisinins and long-acting drugs to avoid artemisinin resistance and facilitate drug administration.
- *Plasmodium vivax*, *Plasmodium ovale*, *Plasmodium malariae*: Quinoline antimalarials, such as chloroquine, amodiaquine, piperaquine, quinine, quinidine, mefloquine, primaquine, benflumetol and halogens, have anti-infective activity in the inner phase of red blood cells; Primaquine can kill *Plasmodium* and gametophyte in liver phase.
- When mixed infection occurs (uncommon): Artemisinin based combination therapy (ACT) + primaquine.
- Since folic acid synthesis is a necessary pathway for parasite DNA synthesis, antifolate drugs, such as sulfonamides, pyrimidine ethylamine, chloroguanidine and dapsone, have synergistic effects on enzymes involved in folic acid synthesis.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS10900585	ATP2B4	TT	may have a higher infection risk when infected with <i>P. falciparum</i>
RS8176719	ABO	II	may have a higher infection risk when infected with <i>P. falciparum</i>
RS2365860	intergenic	TT	may have a higher infection risk when infected with <i>P. falciparum</i>
RS10900589	intergenic	TT	may have a higher infection risk when infected with <i>P. falciparum</i>

RS2365858

intergenic

GG

may have a lower infection risk when infected with *P. falciparum*

For display consideration, only some genetic details are displayed.

My haplotype details

HLA-A

People without the HLA-A*30:01 genotype

[*11:01/*03:01](#)

may have some infection risk when infected with *P. falciparum*. People without the HLA-A*33:01 genotype may have some infection risk when infected with *P. falciparum*.

HLA-B

People without the HLA-B*35:01 genotype

[*51:01/*07:02](#)

may have some infection risk when infected with *P. falciparum*.

Risk calculation

The risk value of this test is calculated based on published academic literature related to *Plasmodium*. By studying the genetic differences between *Plasmodium*-infected patients and normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of *Plasmodium* infection risk of the user.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details



Applicable situation

The *Plasmodium* test report uses 7 polymorphic loci on *ATP2B4*, *ABO*, *OR51B5* genes and intergenic regions; Also, genotyping results of *HLA-A* and *HLA-B*. The results showed whether the user had a higher infection risk of *Plasmodium* than all users in the genotype combination of the 7 polymorphic loci and *HLA-A* and *HLA-B* genotyping results tested, and that the test did not represent the user's actual outcome of *Plasmodium* infection. The basis of the test is mainly from the research of African, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk of *Plasmodium*.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of *Plasmodium* at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing *Plasmodium*, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes genotyping results of *HLA-A* and *HLA-B*; And 7 polymorphic loci related to the risk of *Plasmodium*, such as rs10900585, rs8176719, rs372091.

Population samples

The basis of the test is mainly from the research of African.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with *Plasmodium* infection.

Norovirus

The virus is a highly pathogenic and highly infectious enterovirus, which can cause acute gastroenteritis in humans; It is the main pathogen of sporadic cases and outbreaks of acute gastroenteritis worldwide.

My Norovirus

Infection risk is moderate

People with the rs1047781-AT genotype may have a higher infection risk when infected with norovirus. People with the rs601338-GG genotype may have a higher infection risk when infected with norovirus.

Suggestions

1. Pay attention to diet, environment and personal hygiene:

- Follow the Five key points for food safety when handling food.
- Use Chlorine-containing disinfectant with effective concentration to disinfect the environment regularly; Use in combination with ultraviolet radiation is recommended.
* Note: Alcohol and phenol disinfectants have poor disinfection effect on Norovirus.
- Proper hand washing: Wash hands with soap or hand sanitizer containing antibacterial agent; Wash hands with running water for at least 20 seconds.
- Hand washing habits: Wash hands before meals and after using the toilet; Wash hands before processing food; Wash hands after taking public transport; Take care of infants and children and wash their hands at any time; Pay attention to food and drinking water hygiene.

2. Avoid going to closed public places where people gather.

Influencing factors

Controllable factors

1. Food hygiene: The virus can infect people only with a very low level (≥ 18 virus particles). The risk of infection may be increased by ingesting contaminated food, drinking water, and even contacting aerosol particles from infected people.
2. Venue: Norovirus infection mostly occurs in the form of outbreaks in collective institutions, such as schools, hospitals, military, kindergartens, nursing homes, stadiums, restaurants and tourist areas.

Uncontrollable factors

1. Special population: Infants, children, the elderly and people with basic diseases such as immunodeficiency disease are vulnerable to dehydration and systemic dysfunction after infection due to their weak resistance.
2. Region: Research shows that Norovirus is more infectious in economically underdeveloped areas.
3. Season: Norovirus has obvious seasonality, with higher incidence in autumn and winter.
4. Heredity: Research shows that some people are prone to Norovirus infection due to genetics.

Knowledge

Predisposing age

All age groups are susceptible to Norovirus, with the highest incidence rate among children under 5 years old. The incubation period from infection to gastrointestinal symptoms is 1.2 days on average, the duration of symptoms is 5-6 days, and the virus excretion lasts about 2-3 weeks.

Prevalence

According to the statistics of the World Health Organization (WHO) in 2015, 684 million diarrhoeal diseases caused by Norovirus occur every year in the world, and about 35,000 people die from Norovirus infection every year. The prevalence of Norovirus infection in China reached 6.0% in 2015, with the highest incidence rate among children under 5 years old.

Typical symptoms

The main symptom was diarrhea, followed by vomiting. Other symptoms include abdominal pain, fever, headache, chills, muscle aches, etc.

Infection and heredity

FUT2 is a fucosyltransferase that transfers fucose to the end of ABO precursor in gastrointestinal cells or salivary glands to produce ABH antigen, which is considered as the receptor of human Norovirus. If the gene encoding FUT2 enzyme mutates (such as *FUT2* G428A and C571T mutations), its homozygous carrier is called a non-secretor. Because non-secretor does not produce ABH antigen, it has a strong protective effect on Norovirus. About 20% of white people are non-secretors and can be immune to Norovirus infection.

Treatment

- Norovirus infection is a self-limited disease. At present, there is no specific drug. Most patients do not need treatment after onset and can recover after 2-3 days of rest.
- In case of infectious diarrhea, it is unnecessary to take antibiotics. Sugar saline or oral rehydration salts should be supplemented to help patients balance water and electrolytes.

- Seek medical advice in case of severe vomiting or diarrhea.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS1047781	FUT2	AT	may have a higher infection risk when infected with norovirus
RS601338	FUT2	GG	may have a higher infection risk when infected with norovirus

Risk calculation

The risk value of this test is calculated based on published academic literature related to Norovirus. By studying the genetic differences between Norovirus-infected patients and normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of Norovirus infection risk of the user.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Norovirus test report uses 2 polymorphic loci on *FUT2* gene and intergenic region. The results showed whether the user had a higher infection risk of Norovirus than all users in the genotype combination of the 2 polymorphic loci tested, and that the test did not represent the user's actual outcome of Norovirus infection. The basis of the test is mainly from the research of Caucasian and East Asians, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk of Norovirus.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Norovirus at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing Norovirus, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 2 polymorphic loci related to the risk of Norovirus, such as rs601338 and rs1047781.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asians.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with Norovirus infection.

HIV

The virus has two types, among which HIV-1 can cause acquired immunodeficiency syndrome (AIDS). HIV-1 destroys the immune system of the human body, making the infected people gradually lose their resistance to various diseases, and finally leading to death.

My HIV

Infection risk is slightly low

Compared with the population average, the risk of HIV infection is slightly low.

My HIV

Severity of infection is slightly high

Among the 10 genotypes associated with the severity of HIV-1 infection, the weighted risk score is higher than the average of the population, and the accumulation of virus and the aggravation of the damage to the immune system is slightly high when infected with HIV-1.

Suggestions

1. Practice safe sex and Use condoms correctly.
2. Do not share needles, and refuse unsafe injection or blood transfusion.
3. Circumcision of the genital may be considered for men to prevent viral hiding and transmission due to excessive prepuce.
4. The spouse, sexual partner, children born to HIV-infected person, as well as the occupational exposed persons and the people suspected of infection, should carry out HIV-related counseling or testing as early as possible.

In case of suspected exposure to HIV:

1. Clean the contaminated part with soap solution and flowing water; For mucous membrane such as the eyes, rinse with a large amount of normal saline repeatedly.

-
2. When there is an open wound, gently squeeze the blood from the wound in a proximal-to-distal fashion, and then wash the wound with soap solution and flowing water; Use 75% alcohol or 0.5% iodophor to disinfect the wound.
 3. Preventive medication: Take emtricitabine/tenofovir + latiravir (or dotiravir) as soon as possible (within 2 hours).
 4. Get medical attention as soon as possible.
-

Influencing factors



Controllable factors

1. Sexual behavior: In the process of sexual behavior, HIV can infect the human body through skin damage or mucosal cleft.
2. Medical devices: Shared or repeatedly used needles and syringes; Accepting unsafe injections, blood transfusions, and cutting or punctures that are not sufficiently disinfected can increase the risk of HIV infection.
3. Male's prepuce: An excessive prepuce may increase a man's risk of acquiring HIV infection through heterosexual sex by approximately 60%.
4. Occupational exposure: health care personnel, police or other personnel are at risk of HIV infection due to their contact with blood, tissues or other body fluids of HIV-infected persons in their occupational work.



Uncontrollable factors

1. Family history of HIV: HIV/AIDS patients in the family will increase the risk of HIV infection.
 2. Mother-to-child transmission: Including transmission via placenta, perinatal transmission and breast milk transmission.
 3. Other sexually transmitted diseases (STDs): Syphilis, herpes, chlamydia, gonorrhea, bacterial vaginitis and other infections will increase the risk of HIV infection.
 4. Heredity: Research shows that some people are prone to HIV infection due to genetics.
-

Knowledge



Predisposing age

All age groups are susceptible to the virus. After HIV enters the human body, it reaches the local lymph nodes within 24-48 hours, and virus components can be detected in the peripheral blood within 5-10 days. Due to the existence of the virus repository, the host immune system cannot

completely eliminate the virus, forming chronic infection, including asymptomatic infection and symptomatic infection. It is reported that the duration of asymptomatic infection lasts about 8 years on average. China has reported that men who have sex with men infected with HIV have progressed to the AIDS stage in an average of 4.8 years.



Prevalence

According to the statistics of the World Health Organization (WHO), HIV infection has caused more than 40 million deaths worldwide. By the end of 2021, there are an estimated 38.4 million people living with HIV, of which two thirds (25.6 million) are in the African region. According to the statistics of the National Health and Family Planning Commission of the People's Republic of China in 2017, the incidence rate of HIV infection in China was 4.145/100,000, and the mortality rate was 1.1053/100,000.



Typical symptoms

- Acute phase (within 6 months after infection)
 - HIV viremia and acute injury of the immune system are mainly manifested by fever, but also accompanied by sore throat, night sweat, nausea, vomiting, diarrhea, rash, joint pain, lymph node enlargement and nervous system symptoms.
- Asymptomatic period (about 8-10 years after infection)
 - Due to the continuous replication of HIV in the body of infected persons, the immune system is damaged, and the CD4⁺ T lymphocyte count gradually decreases, which can cause symptoms such as lymph node enlargement.
- AIDS period (about 1-2 years after the diagnosis of AIDS)
 - CD4⁺ T lymphocyte count < 200/ μ L. The main manifestations are HIV-related infection symptoms, various opportunistic infections and tumors.
 - The late clinical manifestations are complex and diverse, and the types of complications are various, eventually leading to death.



Infection and heredity

Since the famous CCR5 was first discovered in 1996, scientists have gradually realized its role as an 'accomplice' in the process of HIV infection. *CCR5* gene encodes a receptor protein, which is located on the surface of white blood cells and plays a role in the process of T cells binding to specific tissues and target organs. When the 32-base pair deletion mutation of the *CCR5* gene (*CCR5* Δ 32/ Δ 32 or *CCR5* rs333-DD genotype) will prevent cells from expressing this receptor protein, thereby blocking the way HIV-1 enters CD4⁺ T cells. The results of population survey and experimental research show that the individuals carrying the *CCR5* Δ 32 mutation have normal immune function and inflammatory reaction, and show significant resistance to HIV infection. It has been found that about 10% of European people carry the *CCR5* Δ 32 mutation, and these people will hardly be infected with HIV-1. What is more critical is that there are clinical trials that transplant these natural human stem cells with HIV-1 resistance to AIDS patients, and the patients' HIV-1 virus has been completely eliminated or even cured.



Treatment

- The clinical use of antiretroviral therapy (ART) mainly includes:
 - Nucleoside reverse transcriptase inhibitors (NRTIs): Zidovudine, didanosine, zalcitabine.
 - Non-nucleoside reverse transcriptase inhibitors (NNRTIs): Nevirapine, delavirdine, efavirenz.
 - Protease inhibitors (PIs): Ritonavir, nelfinavir.
 - Integrase inhibitors (INSTIs): Raltegravir, dolutegravir.
 - Fusion inhibitors (FIs): Enfuvirtide, maraviroc, ibalizumab.
 - CCR5 inhibitors: Mostly in preclinical and clinical trials.
- For general adults and adolescents, the recommended scheme for newly treated patients is:
 - Two NRTIs drugs are combined with the third class drugs, such as tenofovir + lamivudine + rilpivirine.
 - Compound single tablet preparations, such as bictegravir/emtricitabine/tenofovir alafenamide.
 - One NRTIs drug combined with one INSTIs drug, such as dolutegravir + lamivudine.
- In clinical practice, highly effective antiretroviral therapy (commonly known as cocktail therapy) is also used, which refers to the treatment of AIDS through the combined use of three or more antiviral drugs to reduce the drug resistance brought about by a single drug.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS11884476	PARD3B	CC	may have a higher risk of progression to AIDS when infected with HIV-1
RS333	CCR5	II	may have a higher infection risk and may have a higher risk of progression to AIDS when infected with HIV-1
RS35829419	NLRP3	CC	may have a higher infection risk when infected with <i>M. leprae</i>
RS5968255	intergenic	CT	may have a higher risk of progression to AIDS when infected with HIV-1
RS11896375	intergenic	GG	may have a higher risk of progression to AIDS when infected with HIV-1

My haplotype details

HLA-B

People without the HLA-B*27:02 genotype

*51:01/*07:02

may have some risk of accumulation of virus

when infected with HIV-1. People without the

HLA-B*57:01 genotype may have some risk of

progression to AIDS when infected with HIV-1.

People without the HLA-B*27:05 genotype

may have some risk of accumulation of virus

when infected with HIV-1.

Risk calculation

The risk value of this test is calculated based on published academic literature related to HIV. By studying the genetic differences between HIV-infected patients and normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of HIV infection risk of the user, as well as the severity of infection in case one is infected.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details



Applicable situation

The HIV test report uses 5 polymorphic loci on *NLRP3*, *CCR5*, *PARD3B* genes and intergenic regions; Also, genotyping results of *HLA-B*. The results showed whether the user had a higher infection risk and severity of infection of HIV than all users in the genotype combination of the 5 polymorphic loci and *HLA-B* genotyping results tested, and that the test did not represent the user's actual outcome of HIV infection. The basis of the test is mainly from the research of Caucasian, Hispanics, African Americans and Chinese, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk and severity of infection of HIV.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of HIV at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing HIV, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes genotyping results of *HLA-B*; And 5 polymorphic loci related to the risk of HIV, such as rs35829419, rs333 and rs11884476.

Population samples

The basis of the test is mainly from the research of Caucasian, Hispanics, African Americans and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with HIV infection.

HPV

The virus is easy to infect skin and mucous membrane, causing the proliferation of squamous epithelium in humans. Currently, more than 200 HPV types have been identified, which can be generally divided into high-risk type and low-risk type according to carcinogenicity. Among them, HPV-16 and HPV-18 have the highest risk of inducing cancer, such as cervical cancer.

My HPV

Infection risk is slightly high

Among the 10 genotypes associated with the risk of HPV infection, the weighted risk score is higher than the average of the population, and the infection risk is slightly high.

My HPV

Genetic risk of secondary diseases is moderate

Among the 18 genotypes associated with genetic risk of secondary diseases following HPV infection, the weighted risk score is close to the average of the population, and risk of progression to cervical cancer is moderate when infected with HPV.

Suggestions

1. Practice safe sex and Use condoms correctly.
2. Keep the vulva and vagina clean; Try to keep the sexual partner fixed; Avoid too much sex at an early age; Reduce the number of abortions.
3. Improve immunity: Healthy diet, Physical exercise, keeping a good attitude and get enough sleep are means to improve immunity.
4. Vaccination: HPV vaccine has a significant effect on the prevention of infection, especially before first-time sex, which can achieve the maximum protection effect.
5. Take physical examination every year.

Influencing factors



Controllable factors

1. Sexual contact: HPV infection can occur after oral or genital contact with the infected area of HPV infected persons, such as oral sex, vaginal sex, anal sex, etc.
2. Sexual hygiene: The risk of HPV infection will increase if the environment around the genitals is unclean, too many sexual partners, too much sexual life too early, and abortion events.
3. Immune function: When the body's immunity decreases, the risk of HPV infection will increase.



Uncontrollable factors

1. Sex: HPV infection rate in women is higher than that in men.
2. Pregnancy: Some studies have shown that the HPV infection rate of pregnant women has increased, which is due to the fluctuation of estrogen, progesterone and other hormones.
3. Disease: immune disease, malignant tumor, etc.
4. Heredity: Research shows that some people are prone to HPV infection due to genetics.

Knowledge



Predisposing age

- All age groups are susceptible to the virus. The peak of HPV infection usually occurs within 10 years after the first sexual behavior, and warts usually appear after 1-6 months. There is evidence that HPV can enter a longer latency (several months or even decades), and some female groups will also have cervical HPV reactivation, such as HIV-infected women and older women.
- In China, high risk HPV infection in women presents two peaks: 15-24 years old and over 40 years old.



Prevalence

Female infection is more common, 70-80% of women have been infected with HPV at least once in their life. The HPV infection rate of Chinese women is about 16.8%. In 2018, there were about 570,000 new cases and 311,000 deaths of cervical cancer worldwide (almost 100% of cervical cancer is related to persistent infection of high-risk HPV), which has become the second common tumor and the third cause of death for women aged 15-44. New cases of cervical cancer in China account for about 1/4 of the total number of new cases in the world.



Typical symptoms

- Many people have no symptoms after infection, and the infection often improves on its own.
- Some types of HPV (HPV-6, HPV-11) can cause genital warts.
- Long-term HPV-infected (especially HPV-16 and HPV-18) patients are prone to other diseases:
 - Genital HPV infection can cause cervical cancer and vaginal cancer.

- Perianal HPV infection can cause anal cancer.
- HPV infection in mouth and pharynx can cause oropharyngeal cancer.

Infection and heredity

Host genetic variability may play an important role in HPV infection and post infection development, especially in controlling genetic factors involved in immune response. Human leukocyte antigen (HLA) alleles participate in the presentation of foreign antigens by immune system cells. In particular, HLA class II *DRB1* and *DQB1* genes play a key role in modifying the efficiency of T cell-mediated immune response against viruses and other pathogens. Studies have shown that people carrying *DRB104:03* and *DQB106:02* haplotypes have a higher risk of HPV infection.

Infection and cervical cancer

Under normal circumstances, invading HPV will be cleared by the human immune system. Similar to catching a viral cold, this transient infection is normal as the virus will be cleared from the body without even showing symptoms, and will not develop into cancer. Some women with weak immune function or immune mechanism defects may develop into cervical cancer because they cannot eliminate HPV in the body, resulting in persistent HPV infection. E6 and E7 proteins of HPV are the main transformation proteins of the virus, which play an important role in the carcinogenic process by inactivating the tumor suppressor genes *p53* and *Rb*, and are considered to be important carcinogens.

Treatment

Most HPV infections subside spontaneously within 8-12 months. At present, there is no completely satisfactory treatment to eliminate HPV infection. Common treatments include:

- Physical removal: Cryotherapy, electrocautery, laser or surgical resection, etc.
- Local treatment:
 - Antimitotic drugs: Podophyllotoxin, podophyllotoxin resin, 5-fluorouracil
 - Corrosive drug: Trichloroacetic acid
 - Interferon inducer: Imiquimod

My genetic result details

Gene locus	Gene name	My genotype	Description
RS13117307	EXOC1	CT	may have a higher risk of progression to cervical cancer when infected with HPV
RS2516448	intergenic	CT	may have a higher risk of progression to cervical cancer when infected with HPV

RS3117027	HLA-DPB2	CC	may have a lower risk of progression to cervical cancer when infected with HPV
RS4282438	intergenic	TT	may have a higher risk of progression to cervical cancer when infected with HPV
RS8067378	intergenic	AA	may have a lower risk of progression to cervical cancer when infected with HPV

For display consideration, only some genetic details are displayed.

My haplotype details

HLA-DRB1	Females without the HLA-DRB1*04:03 genotype may have some infection risk when infected with HPV.	*15:01/*16:02
HLA-DQB1	Females without the HLA-DQB1*06:02 genotype may have some infection risk when infected with HPV. Females with the HLA- DQB1*06:02 genotype may have a higher infection risk when infected with HPV	*06:02/*05:02

Risk calculation

The risk value of this test is calculated based on published academic literature related to HPV. By studying the genetic differences between HPV-infected patients and normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of HPV infection risk of the user, as well as the changes of genetic risk of secondary diseases.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The HPV test report uses 12 polymorphic loci on *EXOC1*, *HLA-DPB2* genes and intergenic regions; Also, genotyping results of *HLA-DRB1* and *HLA-DQB1*. The results showed whether the user had a higher infection risk and genetic risk of secondary diseases, of HPV than all users in the genotype combination of the 12 polymorphic loci and *HLA-DRB1* and *HLA-DQB1* genotyping results tested, and that the test did not represent the user's actual outcome of HPV infection. The basis of the test is mainly from the research of Caucasian and East Asian, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk and genetic risk of secondary diseases, of HPV.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of HPV at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing HPV, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes genotyping results of *HLA-DRB1* and *HLA-DQB1*; And 12 polymorphic loci related to the risk of HPV, such as rs59661306, rs13117307, rs3117027.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with HPV infection.

Prion

It is a kind of protein particle with self replication ability and infectivity.

Pathogenic prion protein (PrP^{Sc}) is formed by the conformational change of normal prion protein (PrP^C) in the body and deposited in a large number of nerve cells, causing prion diseases, such as sporadic Creutzfeldt-Jakob disease (sCJD).

My Prion

Infection risk is moderate

Among the 8 genotypes associated with the risk of prion infection, the weighted risk score is close to the average of the population, and the infection risk is moderate.

Suggestions

1. When having medical treatment, seek only professional and registered provider; Ensure that medical devices used meet hygiene standards.
2. Avoid eating tissues or processed products of the host infected with prions.

Influencing factors



Controllable factors

1. Medical devices: Prion contaminated surgical devices may infect patients receiving treatment.
2. Food intake: Eating the tissues or processed products of the host containing prion (such as the meat products of cattle with mad cow disease and the brain tissues of patients with Kuru disease) will increase the risk of prion infection.



Uncontrollable factors

1. Age: The majority of patients are elderly, and the average age of onset is about 62 years old.
2. Family history: Prion patients in the family will increase the risk of prion infection.
3. Heredity: Research shows that some people are prone to Prion infection due to genetics.

Knowledge

Predisposing age

The average age of onset is around 62. Prion disease has a long incubation period, which can last for months, years or even decades.

Prevalence

The incidence rate of human Prion diseases is low. Sporadic Creutzfeldt-Jakob disease (sCJD) is 1-3/1,000,000 per year.

Typical symptoms

The main manifestations include myoclonus, visual impairment, akinetic mutism, progressive ataxia, tremor, postural instability and other cerebellar symptoms.

The discovery of Prion

In 1957, American scientist Gajdusek found an indigenous tribe, Fore, in the eastern highlands of Papua New Guinea. Many Fore people suffered from a strange disease called Kuru disease with a long incubation period. In the early stage, patients only felt headache and joint pain, followed by ataxia, tremor, and involuntary movement. The latter, including chorea, myoclonus, etc., developed progressive dementia and neurological abnormalities in the late course of the disease. Most patients die within 3-6 months of onset. At first, Gajdusek thought that Kuru disease was a genetic disease. Later, he found that the Fore people had the custom of eating the organs of their dead relatives to show their remembrance. After research, Gajdusek found that Kuru disease was an infectious disease transmitted through the digestive tract with unknown pathogenic factors. He was awarded the Nobel Prize in Physiology or Medicine in 1976 for discovering the new pathogenesis and transmission mechanism of Kuru disease and eliminating this fatal disease. The pathogen of Kuru disease is Prion, which was discovered later.

Treatment

- There is no effective treatment for human Prion disease. Usually, symptomatic treatment can be given to deal with the neuropsychiatric symptoms that cause pain or disability, such as the use of benzodiazepines (clonazepam) to relieve myoclonus.
- Therapies under research: Flupirtine, pentosan polysulfate, quinacrine, and gene therapy.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS4921542	MTMR7	GT	may have a higher infection risk when infected with prion

RS6107516	PRNP	GG	may have a higher infection risk when infected with prion
RS7565981	intergenic	AA	may have a higher infection risk when infected with prion
RS6951643	GRM8	AG	may have a higher infection risk when infected with prion

Risk calculation

The risk value of this test is calculated based on published academic literature related to Prion. By studying the genetic differences between Prion-infected patients and normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of Prion infection risk of the user.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The Prion test report uses 4 polymorphic loci on *PRNP*, *GRM8* and *MTMR7* genes and intergenic region. The results showed whether the user had a higher infection risk of Prion than all users in the genotype combination of the 4 polymorphic loci tested, and that the test did not represent the user's actual outcome of Prion infection. The basis of the test is mainly from the research of Caucasian, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk of Prion.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of Prion at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing Prion, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 4 polymorphic loci related to the risk of Prion, such as rs6107516, rs6951643 and rs4921542.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with Prion infection.

Cryptococcus

This fungus is widely found in pigeon droppings, soil, rotten wood and other environments, as well as on the surface of human body, oral cavity and intestinal tract. It is a conditional pathogenic fungus, among which *Cryptococcus neoformans* and *Cryptococcus gattii* are the main types, mainly invading the central nervous system and the lung.

My Cryptococcus

Infection risk is slightly low

Compared with the population average, the risk of *Cryptococcus* infection is slightly low.

Suggestions

1. Improve immunity: Healthy diet, Physical exercise, keeping a good attitude and get enough sleep are means to improve immunity.
2. Keep the living environment clean and prevent inhalation of dust with bird droppings; Avoid contact with bird droppings.

Influencing factors



Controllable factors

1. Immune function: The risk of *Cryptococcus* infection will be increased when the body's immunity declines.
2. Infectious source: In cities, bird droppings (especially pigeon droppings) often become the main infectious source; Inhalation or contact with dust with bird droppings may easily cause infection.



Uncontrollable factors

1. Disease: Patients with basic immune deficiency, such as AIDS patients and cancer patients.
2. Heredity: Research shows that some people are prone to *Cryptococcus* infection due to genetics.

Knowledge

Predisposing age

All age groups are susceptible to *Cryptococcus*.

Prevalence

In 2012, the number of *Cryptococcus neoformans* patients worldwide exceeded 1,000,000 each year, with a mortality rate of 20-70%.

Typical symptoms

- Patients with pulmonary cryptococcosis may not have obvious clinical symptoms.
 - In patients with normal immune function, the main symptoms include cough, chest pain, expectoration, weight loss, fever, etc.
 - In patients with low immune function, they are more likely to develop acute respiratory distress syndrome (ARDS) or even die.
- The early symptoms and signs of cryptococcal meningitis are often not specific, which can be manifested as fever, headache, cough, dyspnea; And nausea, vomiting, neck stiffness, visual symptoms, personality changes caused by increased intracranial pressure.
- The symptoms of cryptococcal skin infection are pustular, papular, nodular, or ulcerated lesions, which sometimes resemble acne, molluscum contagiosum, or basal cell carcinoma.

Treatment

Antifungal drugs are generally used for treatment.

- For asymptomatic patients with normal immune function: Closely follow up and observe, and use fluconazole for treatment.
- For mild to moderate patients: Fluconazole is used for treatment.
- For severe patients: Amphotericin B combined with flucytosine was used for treatment, and then fluconazole was used.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS5743604	TLR1	GG	may have a lower infection risk when infected with <i>Cryptococcus</i>

Risk calculation

The risk value of this test is calculated based on published academic literature related to *Cryptococcus*. By studying the genetic differences between *Cryptococcus*-infected patients and

normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of *Cryptococcus* infection risk of the user.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The *Cryptococcus* test report uses 1 polymorphic locus on *TLR1* gene. The results showed whether the user had a higher infection risk of *Cryptococcus* than all users in the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome of *Cryptococcus* infection. The basis of the test is mainly from the research of Chinese, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk of *Cryptococcus*.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of *Cryptococcus* at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing *Cryptococcus*, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus related to the risk of *Cryptococcus*, which is rs5743604.

Population samples

The basis of the test is mainly from the research of Chinese.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with *Cryptococcus* infection.

H. pylori

This bacterium is a type of microaerobic gram-negative bacteria, which is adapted to grow and reproduce in acidic environment. It is closely related to gastritis, peptic ulcer, gastric cancer and other diseases, and belongs to Class I carcinogen.

My H. pylori

Infection risk is slightly high

People with the rs10004195-TT genotype may have a higher infection risk when infected with *H. pylori*. People with the rs368433-TT genotype may have a higher infection risk when infected with *H. pylori*.

My H. pylori

Genetic risk of secondary diseases is moderate

People with the rs2294008-CT genotype may have a higher risk of progression to duodenal ulcer when infected with *H. pylori*. People with the rs2671655-CT genotype may have a higher risk of progression to stomach cancer when infected with *H. pylori*.

Suggestions

1. Pay attention to food hygiene:

- Follow the Five key points for food safety when handling food.
- Do not drink raw water and avoid eating raw food.

2. Pay attention to eating habits:

- When many people eat together, they should avoid sharing tableware, and it is recommended to use public chopsticks; In case of infection, separate meals are recommended.
- Avoid mouth-to-mouth feeding.
- Dental appliances need to be replaced every three months; It is recommended to use mouthwash and antibacterial toothpaste regularly to alleviate oral inflammation.

3. Family members of patients with *H. pylori* infection, gastric ulcer or duodenal ulcer is suggested to have Detection test of *H. pylori*.

Influencing factors



Controllable factors

1. Food hygiene: Contaminated food and drinking water, such as drinking raw water, eating rare steak and raw fish, will increase the risk of *H. pylori* infection.
2. Eating habits: Direct contact with saliva, vomit or feces, such as kissing, sharing tableware, mouth to mouth feeding and other behaviors will increase the risk of *H. pylori* infection.



Uncontrollable factors

1. Race: The infection rate is the highest among Black, Hispanic and Asian people.
2. Family history: *H. pylori* infection has obvious aggregation in the family.
3. Patients with chronic gastric disease: May have poor gastric mucosa defense, and have a higher risk of *H. pylori* infection.
4. Heredity: Research shows that some people have certain genetic susceptibility to *H. pylori*.

Knowledge



Predisposing age

All age groups are susceptible to the bacteria. The infection peak was at the age of 41-60.



Prevalence

About 50% of the world's population is infected with *H. pylori*. In 2015, about 4.4 billion people worldwide were infected.



Typical symptoms

Many patients with *H. pylori* infection have no obvious clinical manifestations. If signs and symptoms of *H. pylori* infection occur, they may include:

- Abdominal pain or burning pain
- Increased pain when fasting
- Nausea
- Inappetence
- Hiccup frequently
- Abdominal distension
- Unexpected weight loss
- Halitosis



Infection and heredity

H. pylori is easy to reproduce in the environment with low pH (i.e. acid). The ability of gastric parietal cells to control their acid production is important in the development of *H. pylori* related diseases. *H. pylori* infection mainly occurs in the acid prominent parts of the human stomach, such as the gastric antrum. Therefore, the host with high gastric acid secretion has a high density of *H. pylori* in the gastric antrum, and mainly develops into antral gastritis. On the other hand, under the stimulation of *H. pylori*, monocytes produce a large number of cytokines, such as IL-10, IL-12. This will enhance the proinflammatory response, leading to more gastric epithelial damage and aggravating the disease. Some host genes encoding immune regulatory factors contain polymorphic regions, and the polymorphisms of these genes will change their transcription, thus affecting the inflammatory process. Therefore, the risk of *H. pylori* related diseases varies from person to person.

Infection and gastric cancer

In 2019, a study by *Oncogene* revealed the molecular mechanism by which inflammation caused by *H. pylori* promoted the proliferation of gastric epithelial stem cells, thereby triggering gastric tumors. Tumor necrosis factor α (TNF- α) promotes inflammation in the body, which will activate NOXO1 protein that can participate in the production of reactive oxygen species (ROS). ROS is a tissue-damaging molecule, and the oxidative stress reaction triggered by it will damage and mutate the DNA of gastric cells, leading to the formation of tumors. The inflammation caused by *H. pylori* infection will also produce ROS, which will increase the oxidative stress response of the stomach and increase the risk of cancer.

Treatment

- Common medication:
 - Proton pump inhibitor (PPI): It can inhibit gastric acid secretion, enhance antibiotic sensitivity, and promote the healing of erosive ulcer, such as esomeprazole, rabeprazole, omeprazole, lansoprazole, pantoprazole, etc.
 - Bismuth agent: it is used to improve antibiotic sensitivity and eradication rate, such as bismuth potassium citrate, colloidal pectin bismuth, etc.
 - Antibiotics: Amoxicillin, clarithromycin, metronidazole, furalddehyde ketone, tetracycline, etc.
- At present, combined drug therapy is commonly used, such as triple therapy (proton pump inhibitor + clarithromycin + amoxicillin/metronidazole) and quadruple therapy (proton pump inhibitor + bismuth agent + tetracycline + metronidazole).
- Some studies have shown that probiotics can enhance the inhibitory effect on *H. pylori*, and can also alleviate the adverse gastrointestinal reactions caused by drug treatment.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS10004195	intergenic	TT	may have a higher infection risk when infected with <i>H. pylori</i>

RS2294008	PSCA	CT	may have a higher risk of progression to duodenal ulcer when infected with <i>H. pylori</i>
RS2671655	intergenic	CT	may have a higher risk of progression to stomach cancer when infected with <i>H. pylori</i>
RS368433	intergenic	--	Not Detected

Risk calculation

The risk value of this test is calculated based on published academic literature related to *H. pylori*. By studying the genetic differences between *H. pylori*-infected patients and normal people, these literatures have identified some polymorphic sites associated with the risk of infection. We use the raw data in these literatures to score the polymorphic sites involved in this test and get the risk values for all users. By comparing the risk averages of all users tested in our products, we can give the changes of *H. pylori* infection risk of the user, as well as the changes of genetic risk of secondary diseases.

Notice: The risk calculation of the test item only takes into account the relevant polymorphic loci involved in the test, and the risk of infection induced by other unknown genes or variants and non-genetic factors are not taken into account.

Test details

Applicable situation

The *H. pylori* test report uses 4 polymorphic loci on *FCGR2A* and *PSCA* genes and intergenic regions. The results showed whether the user had a higher infection risk and genetic risk of secondary diseases, of *H. pylori* than all users in the genotype combination of the 4 polymorphic loci tested, and that the test did not represent the user's actual outcome of *H. pylori* infection. The basis of the test is mainly from the research of Caucasian and East Asian, other races are for reference only.

Notification

1. The test may not cover all genes or loci that could contribute to infection risk and genetic risk of secondary diseases, of *H. pylori*.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This study only predicts your risk of *H. pylori* at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have a risk of developing *H. pylori*, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 4 polymorphic loci related to the risk of *H. pylori*, such as rs368433 and rs2294008.

Population samples

The basis of the test is mainly from the research of Caucasian and East Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with *H. pylori* infection.



Exposure Risk

25 Reports



Food Exposure Risk

Shrimps	risk is slightly low
Nuts	risk is moderate
Peaches	risk is moderate
Cow's Milk	risk is moderate
Peanuts	risk is slightly high
Hen's Eggs	risk is slightly low
Processed Meat	risk is slightly low
Wheat Products	risk is slightly low
Dietary Sodium	risk is slightly low



Occupational Environment Risk

Benzene	risk is slightly high
Pesticides	risk is slightly high
Noises	risk is slightly low
Endotoxins	risk is slightly low
Cleaning Agents	risk is moderate
Night Shift	risk is moderate
Gases and Fumes	risk is slightly low
Chlorinated Solvents	risk is moderate

Mineral Dust	risk is slightly low
Occupational Stress	risk is slightly high
Metal Dust	risk is slightly low
Non-aromatic Solvents	risk is moderate

Other Exposure Risk

Dogs	risk is slightly low
Cats	risk is slightly low
Natural Rubber Latex	risk is slightly low
Dust Mites	risk is slightly high

Dogs

It is a common pet, but some people may have allergic reactions due to contact with dog hair, dandruff, saliva, etc.

My Dogs exposure

risk is slightly low

Compared with the population average, the risk of persistent wheeze after exposure to dogs in early life is slightly low.

Suggestions

1. Regular cleaning: Maintain hygiene in living spaces to reduce the accumulation of dog hair and dander. If owning a pet dog, giving it regular baths can minimize the spread of allergenic substances.
2. Air purification: Using efficient air filters (like HEPA vacuum cleaners) can help remove allergens from the environment.
3. Adjust living habits: Keep dogs away from clothes, bedrooms, and sofas; use allergen-protective bedding; wash hands and change clothes after handling dogs.
4. Limit contact: Those at high risk or with severe reactions should consider not owning a dog or limit interactions with them.
5. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and efficacy of treatment methods vary from person to person and should always be undertaken under the advice and supervision of a medical professional, avoiding self-experimentation.

Knowledge



Dog allergy

Allergic reactions occur when the body's immune system responds to typically harmless substances (allergens) mistaking them for threats. Dog hair, dander, and saliva are the main allergens, and the proteins responsible include:

- Can f 1: The primary dog allergen protein, typically found in dog dander, saliva, and urine.
- Can f 2: A common dog allergen protein, primarily found in dog dander and saliva.
- Can f 3: A protein produced by the dog's prostate, mainly present in the urine of male dogs.
- Can f 4: A protein produced by dog skin glands, a potential cause of allergies.
- Can f 5: A protein found in dog saliva, another potential cause of allergies.
- Can f 6: Another protein produced by the dog's prostate, primarily present in the urine of male dogs.

 High-risk groups

- Individuals with a family history of allergies.
- Children are more likely to develop allergic reactions.

 Prevalence

Dog allergies are one of the most common pet allergies. It is estimated that up to 30% of allergic individuals react to cats and dogs, with cat allergies being twice as prevalent as dog allergies.

 Typical symptoms

Common symptoms include sneezing, runny or stuffy nose, itchy eyes or excessive tearing, and skin rashes. In asthma patients, exposure to dogs can also trigger asthma symptoms or worsen existing asthma.

 Treatment

- Medications: Antihistamines like chlorpheniramine and benadryl can effectively alleviate symptoms of dog allergies.
- Allergen immunotherapy: By gradually introducing small doses of allergens into the patient's body, this method aims to increase tolerance to the allergens, thereby reducing or preventing allergic symptoms.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS2305480	GSDMB	GG	may have a lower risk of persistent wheeze after exposure to dogs in early life

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of dogs, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to dogs in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to dogs relative to those without allergic reactions to dogs. It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Dogs test report uses 1 polymorphic locus on *GSDMB* gene. The results showed whether the user had a higher exposure risk of dogs than all users in the genotype of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following dogs exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of dogs.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of dogs at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of dogs, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs2305480, related to exposure risk of dogs.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of dogs.

Cats

It is a common pet, but some people may have allergic reactions due to contact with cat hair, dandruff, saliva, etc.

My Cats exposure

risk is slightly low

Compared with the population average, the risk of allergic reaction after exposure to cats in early life is slightly low.

Suggestions

1. Regular cleaning: Maintain hygiene in living spaces to reduce the accumulation of cat hair and dander. If owning a pet cat, giving it regular baths can minimize the spread of allergenic substances.
2. Air purification: Using efficient air filters (like HEPA vacuum cleaners) can help remove allergens from the environment.
3. Adjust living habits: Keep cats away from clothes, bedrooms, and sofas; use allergen-protective bedding; wash hands and change clothes after handling cats.
4. Limit contact: Those at high risk or with severe reactions should consider not owning a cat or limit interactions with them.
5. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and efficacy of treatment methods vary from person to person and should always be undertaken under the advice and supervision of a medical professional, avoiding self-experimentation.

Knowledge

Allergic reactions occur when the body's immune system responds to typically harmless substances (allergens) mistaking them for threats. Cat hair, dander, and saliva are the main allergens, and the proteins responsible include:

- Fel d 1: The primary cat allergen protein, typically sourced from the skin and saliva secretions of male cats. When cats groom themselves, these proteins can deposit in their fur via saliva.
- Fel d 4: A urinary protein typically secreted by both female and male cats.
- Fel d 2: Mainly found in cat saliva. When cats lick or groom, these proteins adhere to their skin and fur.

 High-risk groups

- Individuals with a family history of allergies.
- Children are more likely to develop allergic reactions.

 Prevalence

Cat allergies are one of the most common pet allergies. It is estimated that up to 30% of allergic individuals react to cats and dogs, with cat allergies being twice as prevalent as dog allergies.

 Typical symptoms

Common symptoms include sneezing, runny or stuffy nose, itchy eyes or excessive tearing, and skin rashes. In asthma patients, exposure to cats can also trigger asthma symptoms or worsen existing asthma.

 Treatment

- Medications: Antihistamines like chlorpheniramine and benadryl can effectively alleviate symptoms of cat allergies.
- Allergen immunotherapy: By gradually introducing small doses of allergens into the patient's body, this method aims to increase tolerance to the allergens, thereby reducing or preventing allergic symptoms.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS7216389	GSDMB	TT	may have a lower risk of allergic reaction after exposure to cats in early life

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of cats, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by

studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to cats in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to cats relative to those without allergic reactions to cats.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Cats test report uses 1 polymorphic locus on *GSDMB* gene. The results showed whether the user had a higher exposure risk of cats than all users in the genotype of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following cats exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of cats.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of cats at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of cats, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs7216389, related to exposure risk of cats.

Population samples

The basis of the test is mainly from the research of Caucasian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of cats.

Benzene

It is a colorless, volatile liquid hydrocarbon with a special flavor, widely used in the chemical industry, and exists in tobacco smoke, vehicle emissions and industrial emissions. Long-term exposure to benzene may cause health problems, such as non-Hodgkin lymphoma (NHL).

My Benzene exposure

risk is slightly high

People with the rs144848-AA genotype may have a higher risk of non-hodgkin lymphoma (NHL) after exposure to benzene.

Suggestions

1. Personal protection: Wear appropriate masks, gloves, and protective gear when handling benzene.
2. Ensure ventilation: Make sure areas where benzene is used or stored are well-ventilated.
3. Limit exposure: Reduce time spent in areas with high concentrations of benzene and avoid inhaling its fumes.
4. Risk education: Understand the risks associated with benzene exposure and the methods of protection.
5. Safe storage: Store benzene in well-sealed containers in a cool, dry place away from heat or open flames.

Knowledge

Benzene

Benzene is an aromatic hydrocarbon and, as an essential chemical feedstock, is widely used to produce various chemical products, including plastics, synthetic fibers, rubber, dyes, drugs, pesticides, etc.

Non-Hodgkin Lymphoma (NHL)

NHL is a group of malignant blood cancers originating from lymphocytes, a type of white blood cell. Its exact cause remains unclear, but certain factors, including prolonged exposure to some chemicals, may increase the risk.

 Benzene and NHL

Long-term exposure to benzene is associated with an increased risk of developing NHL, as benzene can damage DNA within cells, causing mutations that lead to cancer development.

 High-risk occupations

- **Petrochemical industry workers:** Those involved in the production or regular use of benzene may be at a higher exposure risk.
- **Refinery workers:** Workers may be exposed to benzene during the crude oil refining process.
- **Chemical plant workers:** Production involving benzene-containing products presents a higher exposure risk.
- **Gas station employees:** Gasoline contains benzene which poses an exposure risk.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS144848	BRCA2	AA	may have a higher risk of non-hodgkin lymphoma (NHL) after exposure to benzene

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of benzene. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of benzene in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with benzene exposure relative to those without benzene exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Benzene test report uses 1 polymorphic locus on *BRCA2* gene. The results showed whether the user had a higher exposure risk of benzene than all users in the genotype of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following benzene exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of benzene.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of benzene at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of benzene, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs144848, related to exposure risk of benzene.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of benzene.

Shrimps

It contains tropomyosin, arginine kinase and other proteins, which may cause allergic reactions after ingestion.

My Shrimps exposure

risk is slightly low

Compared with the population average, the risk of allergic reaction after exposure to shrimps is slightly low.

Suggestions

1. Consume cooked shrimps: Cooked shrimps can reduce their allergenicity.
2. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and effectiveness of treatment methods vary from person to person. Regarding allergen immunotherapy for food, in particular, remains a relatively new field. If necessary, it should be discussed with a doctor to weigh the pros and cons and carried out under their supervision, avoiding self-experimentation.

* Individuals diagnosed with shrimp allergies should avoid consuming shrimps and related products (such as seafood seasonings). It is essential to read processed food labels. Some allergenic proteins in shrimps are also expressed in other seafood (e.g., crabs, lobsters), so there is a risk of cross-reactivity. When dining out, consult restaurant staff or chefs in advance to ensure there are no shrimps or risks of cross-contamination in the food.

Influencing factors



Controllable factors

1. Cooking level of shrimps: The risk of developing a shrimp allergy increases with undercooked shrimps.



Uncontrollable factors

1. Genetics: Individuals with a family history of seafood allergies may have a higher risk of shrimp allergy.

Knowledge

Shrimp allergy

An allergic reaction is when the body's immune system reacts to typically harmless substances (allergens) and mistakes them as threatening to itself. The main allergenic proteins in shrimps include:

- Myosin: The most common allergenic protein in shrimps. This protein has high thermal stability.
- Arginine kinase: A widely distributed allergenic protein in shrimps. This protein has poor thermal stability.
- Phosphoglycerate isomerase: A widely distributed glycolytic enzyme in shrimps. This protein has poor thermal stability.
- Hemocyanin: A potential allergenic protein in shrimps.

Prevalence

In the U.S., the population allergic to seafood products like shrimps and crabs has reached 2.3% of the total population. In Asia, shrimps account for nearly 30% of all food allergy incidents.

Typical symptoms

- Mild symptoms: Skin reactions producing hives or eczema, stomach pain, or sneezing.
- Severe symptoms: Shortness of breath, rapid heartbeat, a drop in blood pressure, or even anaphylactic shock.

Treatment

- Mild symptoms: Antihistamines (e.g., cetirizine, loratadine) can be used for treatment.
- Severe symptoms: Those experiencing anaphylactic shock should immediately use an epinephrine auto-injector and seek emergency treatment immediately.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS1800925	TH2LCRR	CC	may have a lower risk of allergic reaction after exposure to shrimps
RS20541	IL13	GG	may have a lower risk of allergic reaction after exposure to shrimps
RS9275596	intergenic	CT	may have a lower risk of allergic reaction after exposure to shrimps

RS74995702

intergenic

AA

may have a lower risk of allergic reaction after exposure to shrimps

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of shrimps, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to shrimps in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to shrimps relative to those without allergic reactions to shrimps.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Shrimps test report uses 4 polymorphic loci on *IL13* and *TH2LCRR* genes and intergenic regions. The results showed whether the user had a higher exposure risk of shrimps than all users in the genotype combination of the 4 polymorphic loci tested, and that the test did not represent the user's actual outcome following shrimps exposure. The basis of the test is mainly from the research of Asian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of shrimps.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.

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2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of shrimps at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
 3. If you feel you have some exposure risk of shrimps, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 4 polymorphic loci related to exposure risk of shrimps, i.e., rs1800925, rs20541, rs74995702 and rs9275596.

Population samples

The basis of the test is mainly from the research of Asian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of shrimps.

Natural Rubber Latex

It is a natural substance extracted from the sap of rubber trees. Latex has elasticity and durability, so it is widely used in the manufacture of various products, such as gloves, balloons and condoms. However, latex contains allergens and may cause allergic reactions upon contact.

My Natural Rubber Latex exposure

risk is slightly low

Compared with the population average, the risk of allergic reaction after exposure to natural rubber latex is slightly low.

Suggestions

1. Identify latex products: When purchasing gloves, mattresses, toys, condoms, etc., carefully read product labels to identify ingredients such as 'latex' or 'rubber'.
2. Stay dry: Latex products release allergens more easily in humid environments. Try to avoid exposing latex products to moist conditions.
3. First-aid training: Understand how to self-administer or assist others with emergency medications in the event of a severe allergic reaction.

* Individuals diagnosed with latex allergies should avoid products made from natural rubber latex; consider choosing synthetic latex or ethylene-vinyl acetate latex (EVA latex) as alternative products; be alert to cross allergic reactions. For example, latex allergy is associated with allergies to bananas, avocados, chestnuts, and kiwifruit.

Knowledge

Latex allergy

Allergic reactions occur when the body's immune system responds to typically harmless substances (allergens) mistaking them for threats. Currently, 15 allergenic proteins have been identified in latex, including:

- Hev b 5: It is a common latex allergen protein, and it is also a major allergen that causes latex glove allergy of medical workers.
- Hev b 6: Also known as hevein, it is a common latex allergen protein.
- Hev b 13: A common latex allergen protein.

In addition, vulcanizing agents added to latex products may also cause allergic reactions.

Prevalence

Data analysis from limited studies suggests that the average latex allergy prevalence among the general population worldwide is 4.3%. The prevalence might be higher among healthcare workers and those with a history of allergies.

Typical symptoms

Symptoms of latex allergy can range from mild skin redness, rash, and itching to more severe reactions like sneezing, runny nose, and asthma symptoms. In rare cases, anaphylactic shock may occur.

Treatment

- General intervention: Antihistamines can effectively alleviate symptoms of latex allergy, e.g., benadryl, cetirizine, etc.
- Emergency intervention: In cases of severe reactions like anaphylactic shock, one should seek medical attention immediately and use epinephrine.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS1800925	TH2LCRR	CC	may have a lower risk of allergic reaction after exposure to natural rubber latex

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of natural rubber latex, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to natural rubber latex in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to natural rubber latex relative to those without allergic reactions to natural rubber latex.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Natural Rubber Latex test report uses 1 polymorphic locus on *IL13* gene. The results showed whether the user had a higher exposure risk of natural rubber latex than all users in the genotype of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following natural rubber latex exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of natural rubber latex.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of natural rubber latex at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of natural rubber latex, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs1800925, related to exposure risk of natural rubber latex.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of natural rubber latex.

Pesticides

It is a large class of chemical or biological agents widely used in agriculture, horticulture and forestry, but exposure to pesticides may be related to health problems, such as thyroid dysfunction (TD) and Parkinson's disease (PD).

My Pesticides exposure

risk is slightly high

People with the rs3129882-GG genotype may have a higher risk of Parkinson's disease (PD) after exposure to pesticides.

People with the rs4244285-AG genotype may have a higher risk of thyroid dysfunction (TD) after exposure to pesticides.

Suggestions

1. Personal protection: Workers should wear appropriate masks, gloves, and protective clothing.
2. Safe storage: Store pesticides in a cool, dry place away from children and pets.
3. Standardized operations: Strictly follow the manufacturer's instructions when using pesticides; handle empty pesticide containers as per local regulations.
4. Risk education: Train workers on the potential risks associated with each pesticide and preventive measures.

Knowledge



Pesticides

Pesticides are a broad category of chemical or biological agents, including herbicides, insecticides, fungicides, plant growth regulators, etc., used to control pests, weeds, fungi, and other harmful organisms. They enhance crop yield, quality, and safeguard plant health.



Thyroid dysfunction (TD)

The thyroid is a vital endocrine gland in the human body. Hormones secreted by the thyroid play a pivotal role in metabolism, growth, development, and body temperature regulation. TD refers to a category of diseases arising from abnormal secretion of thyroid hormones, leading to excessively high (hyperthyroidism) or low (hypothyroidism) levels of thyroid hormones in the body.



Parkinson's disease (PD)

PD is a chronic, degenerative neurological disorder primarily affecting the dopamine system in the brain, leading to a decline in dopamine levels. Dopamine deficiency can result in neurological dysfunction, impairing body movement, characterized by muscle rigidity, tremors, and slowed movements.



Pesticides and TD

There is a certain correlation between pesticides and TD, especially with specific organohalogen pesticides like benzothiazoles and chloroprop.



Pesticides and PD

Studies indicate that prolonged or repetitive exposure to pesticides might lead to chronic neurological issues, including cognitive decline, behavioral issues, and neurodegeneration.



High-risk occupations

- Agricultural workers: Directly exposed to pesticides during application.
- Exterminators: Regularly exposed to insecticidal environments.
- Gardeners and horticulturists: Might frequently use pesticides to control pests in plants.
- Workers in pesticide manufacturing plants: Face exposure risks during production.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS3129882	HLA-DRA	GG	may have a higher risk of Parkinson's disease (PD) after exposure to pesticides
RS4244285	CYP2C19	AG	may have a higher risk of thyroid dysfunction (TD) after exposure to pesticides

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of pesticides. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of pesticides in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with pesticides exposure relative to those without pesticides exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Pesticides test report uses 2 polymorphic loci on *CYP2C19* and *HLA-DRA* genes. The results showed whether the user had a higher exposure risk of pesticides than all users in the genotype combination of the 2 polymorphic loci tested, and that the test did not represent the user's actual outcome following pesticides exposure. The basis of the test is mainly from the research of Caucasian and Thai.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of pesticides.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of pesticides at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of pesticides, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 2 polymorphic loci related to exposure risk of pesticides, i.e., rs4244285 and rs3129882.

Population samples

The basis of the test is mainly from the research of Caucasian and Thai.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of pesticides.

Noises

It refers to any undesired or interfering sound, which usually has a negative impact on people's auditory perception, work, sleep and quality of life. Prolonged exposure to high noise may cause health problems, the most common of which is noise-induced hearing loss (NIHL).

My Noises exposure

risk is slightly low

Compared with the population average, the risk of noise-induced hearing loss (NIHL) after exposure to noises is slightly low.

Suggestions

1. Hearing protection: Use earplugs or earmuffs in noisy environments.
2. Limit exposure: Reduce the duration of exposure to noise.
3. Safe volume: Maintain the volume of musical devices at a safe level, ideally below 60% of the maximum volume.
4. Regular check-ups: If exposed to noisy work environments, have your hearing checked regularly.
5. Noise control at work: Implement soundproofing or noise reduction measures in noisy workplaces.

Knowledge



Noises

Sound is measured in decibels (dB). Typically, sounds exceeding 85dB are considered as noise, and prolonged exposure can be harmful. Common sources of noise include traffic, entertainment activities (like concerts), industrial production, household appliances (like washing machines, vacuum cleaners), etc.



Noise-induced hearing loss (NIHL)

NIHL is a damage to the auditory system due to long-term or excessive exposure to high-intensity noise environments. This hearing loss is typically progressive, causing harm to the ears and auditory

nerves, leading to a decline in hearing. Symptoms of NIHL might include hearing reduction (especially in high frequencies), decreased sensitivity to certain frequencies, tinnitus, etc.

Noises and NIHL

Noise propagates in the form of sound waves. When these waves enter the ear, they cause sensory cells (hair cells) within the auditory system to vibrate. If an individual is exposed to intense noises for prolonged periods or frequently, these cells might get damaged or destroyed, possibly resulting in irreversible hearing loss.

High-risk occupations

- Construction workers: May be continuously exposed to construction noises.
- Musicians: Musical sounds might be a source of noise; in enclosed concert venues or recording studios, sound reflections might amplify noise levels.
- Factory workers: Might be continuously exposed to machinery noise.
- Airport ground staff: Noise from aircraft engines can be a significant source of noise.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS9321402	EYA4	AA	may have a lower risk of noise-induced hearing loss (NIHL) after exposure to noises

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of noises. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of noises in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with noises exposure relative to those without noises exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Noises test report uses 1 polymorphic locus on *EYA4* gene. The results showed whether the user had a higher exposure risk of noises than all users in the genotype of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following noises exposure. The basis of the test is mainly from the research of Chinese.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of noises.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of noises at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of noises, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs9321402, related to exposure risk of noises.

Population samples

The basis of the test is mainly from the research of Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of noises.

Nuts

Generally refers to tree nuts, including almonds, walnuts, cashews, hazelnuts, pistachios, macadamia nuts, Brazil nuts, etc. Some individuals will have allergic reactions to the ingredients (such as protein) in nuts after eating nuts and related products.

My Nuts exposure

risk is moderate

People with the rs324015-CT genotype may have a moderate risk of allergic reaction after exposure to nuts.

Suggestions

1. Improve consumption method: Avoid roasted nuts; nuts processed through boiling, fermentation, refining, and peeling might reduce the incidence of allergies.
2. Timing of first-time consumption: Introducing nuts after 1 year of age might reduce the risk of nut allergies, but one should start with small quantities; studies also suggest that non-allergic pregnant women consuming nuts might help their offspring avoid allergies.
3. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and effectiveness of treatment methods vary from person to person. Regarding allergen immunotherapy for food, in particular, remains a relatively new field. If necessary, it should be discussed with a doctor to weigh the pros and cons and carried out under their supervision, avoiding self-experimentation.

* Those diagnosed with nut allergies should avoid consuming nuts and related products (like seasonings and pastries), and check the labels of processed foods; be aware of cross-reactivity, for instance, hazelnut allergy is related to birch pollen allergy; when dining out, consult the waiter or chef in advance to ensure there are no nuts or cross-contamination risks in the food.

Influencing factors



Controllable factors

1. Consumption method: Roasted nuts may increase allergenicity.
2. First-time consumption: The timing of introducing nuts to the diet may affect the risk of nut allergies.

Uncontrollable factors

1. Genetics: Individuals with a family history of food allergies may have an increased risk.

Knowledge

Nut allergy

An allergic reaction refers to the body's immune system responding to typically harmless substances (allergens) as if they were harmful. Most allergens in nuts belong to seed storage proteins, such as:

- Pru du 6: The main allergenic protein in almonds, a type of 11S globulin.
- Jug r 1/3: The main allergenic proteins in walnuts, a type of 2S albumin, highly resistant to heat, pH, and gastrointestinal enzymes.
- Ana o 1: The main allergenic protein in cashews, a type of legumin-like 7S globulin, has weak stability.
- Ana o 3: Another main allergenic protein in cashews, a type of 2S albumin, highly resistant to heat, pH, and gastrointestinal enzymes.
- Cor a 1: The main allergenic protein in hazelnuts, belongs to the Pathogenesis-Related Protein family 10.
- Ber e 1: The main allergenic protein in Brazil nuts, a 2S sulfur-rich storage protein, highly resistant to heat, pH, and gastrointestinal enzymes.

Prevalence

- In the US, about 0.5-1% of the total population is allergic to nuts. Asia has a lower overall consumption of nuts, so the prevalence of nut-induced allergies ranges from 0.05-4.9%.
- Similar to peanut allergies, nut allergies typically persist into adulthood.
- Nut allergy deaths account for 18-40% of food allergy fatality cases.
- Common nuts causing allergies in different countries/regions:
 - US: Walnuts, cashews
 - UK: Brazil nuts, walnuts
 - Europe: Hazelnuts
 - Japan: Walnuts
 - China: Cashews

Typical symptoms

- Mild symptoms: Skin reactions (hives, itchiness), gastrointestinal discomfort, or a runny nose.
- Severe symptoms: Difficulty breathing, swollen throat, rapid or irregular heartbeat, significant drop in blood pressure, and even anaphylactic shock.



Treatment

- Mild symptoms: Antihistamines (like cetirizine, loratadine) can be used for treatment.
- Severe symptoms: Those in anaphylactic shock need an immediate adrenaline injection and should seek emergency treatment.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS324015	STAT6	CT	may have a moderate risk of allergic reaction after exposure to nuts

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of nuts, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to nuts in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to nuts relative to those without allergic reactions to nuts.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details



Applicable situation

The Nuts test report uses 1 polymorphic locus on *STAT6* gene. The results showed whether the user had a higher exposure risk of nuts than all users in the genotype of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following nuts exposure. The basis of the test is mainly from the research of Caucasian.



Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of nuts.
2. The test results have no clinical significance.

3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of nuts at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of nuts, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs324015, related to exposure risk of nuts.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of nuts.

Dust Mites

It is a kind of tiny arthropod that cannot be seen by the naked eye. It is named dust mite because it usually thrives in the dust. Dust mites use dead dander, animal and plant fibers as food sources, so indoor dust mites are generally distributed in beds, carpets, fabrics, household dust and other places. The feces, egg shells, debris and other substances produced by dust mites may cause allergic reactions.

My Dust Mites exposure

risk is slightly high

People with the rs2395185-GG genotype may have a higher risk of allergic reaction after exposure to dust mites. People without the HLA-DRB1-*04:05 genotype may have some risk of allergic reaction after exposure to dust mites. People without the HLA-DRB1-*04:11 genotype may have some risk of allergic reaction after exposure to dust mites.

Suggestions

1. **Maintain hygiene:** Ensure sanitation in the living environment, especially in fabric-covered areas like bedding, carpets, and curtains.
2. **Regular cleaning of bedding:** It is recommended to clean sheets, duvet covers, pillowcases, etc., every 1-2 weeks using hot water and high-temperature drying. Cleaning duvets, pillows, and mattresses every 2-3 months is also recommended.
3. **Air purification:** Using efficient air filters (like HEPA vacuums) can filter dust mites and their fecal particles from the air.
4. **Control humidity:** Dust mites breed more easily in humid environments. It is suggested to keep indoor relative humidity between 30%-50%.
5. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and efficacy of treatment methods vary from person to person and should always be undertaken under the advice and supervision of a medical professional, avoiding self-experimentation.

Knowledge

Dust mite allergy

Allergic reactions occur when the body's immune system responds to typically harmless substances (allergens) mistaking them for threats. While dust mites generally do not pose a direct health threat to most people, some are allergic to them. The main allergenic proteins of dust mites include:

- Der p1: The main allergen protein from *Dermatophagoides pteronyssinus*, belonging to the cystatin family, can disrupt intercellular connections, leading to a decrease in epithelial cell barrier function, making it easier for other allergens to enter the human body.
- Der p2: The main allergen protein from *Dermatophagoides pteronyssinus*.
- Der f1: The main allergen protein from *Dermatophagos farinae*, belonging to the cystatin family, can disrupt intercellular connections, leading to a decrease in epithelial barrier function, making it easier for other allergens to enter the human body.
- Der f2: The main allergen protein from *Dermatophagoides farinae*.

High-risk individuals

- People with asthma or existing respiratory diseases.
- Those with a family history of allergies.
- Individuals living in high humidity environments or homes without proper ventilation.

Prevalence

- Dust mite allergy has a relatively high incidence rate in developed countries, which may be because people usually live in a cleaner and closed indoor environment, resulting in more frequent contact with dust mite allergens, increasing the risk of allergy.
- From the north to the south of China, with changes in temperature and humidity, the positive rate of dust mites also increases.

Typical symptoms

Common symptoms include nasal congestion, stuffiness, runny nose, sneezing; coughing (especially at night or upon waking up); itchy, red, painful, and teary eyes; possible itchy skin or eczema.

Treatment

- Medication: Antihistamines can effectively alleviate symptoms of dust mite allergy, e.g., benadryl, cetirizine, etc.
- Allergen immunotherapy: Introducing small doses of allergens gradually to the patient's body to increase their tolerance to the allergens, thereby reducing or preventing allergic symptoms.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS2395185	intergenic	GG	may have a higher risk of allergic reaction after exposure to dust mites

My haplotype details

HLA-DQB1	People without the HLA-DQB1-*06:01 genotype may have some risk of allergic reaction after exposure to dust mites.	*06:02/*05:02
HLA-DRB1	People without the HLA-DRB1-*04:05 genotype may have some risk of allergic reaction after exposure to dust mites. People without the HLA-DRB1-*08:03 genotype may have some risk of allergic reaction after exposure to dust mites. People without the HLA-DRB1-*04:11 genotype may have some risk of allergic reaction after exposure to dust mites.	*15:01/*16:02

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of dust mites, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to dust mites in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to dust mites relative to those without allergic reactions to dust mites.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Dust Mites test report uses 1 polymorphic locus within intergenic regions; Also, genotyping results of *HLA-DQB1* and *HLA-DRB1*. The results showed whether the user had a higher exposure risk of dust mites than all users in the genotype combination of the 1 polymorphic locus and *HLA-DQB1* and *HLA-DRB1* genotyping results tested, and that the test did not represent the user's actual outcome following dust mites exposure. The basis of the test is mainly from the research of Chinese and Brazilian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of dust mites.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of dust mites at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of dust mites, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes genotyping results of *HLA-DQB1* and *HLA-DRB1*; And 1 polymorphic locus, i.e., rs2395185, related to exposure risk of dust mites.

Population samples

The basis of the test is mainly from the research of Chinese and Brazilian.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of dust mites.

Peaches

It contains Pru p 1, Pru p 3 and other proteins, which may cause allergic reactions after ingestion.

My Peaches exposure

risk is moderate

People with the rs28359884-AC genotype may have a moderate risk of allergic reaction after exposure to peaches.

Suggestions

1. Improve consumption method: Heating can reduce the allergenicity of the allergenic proteins in peaches; since there are allergens in peach skin, it is recommended to peel before eating.
2. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and effectiveness of treatment methods vary from person to person. Regarding allergen immunotherapy for food, in particular, remains a relatively new field. If necessary, it should be discussed with a doctor to weigh the pros and cons and carried out under their supervision, avoiding self-experimentation.

* Those diagnosed with peach allergy should avoid intake or contact with peaches and related products, and pay attention to product labels; some allergenic proteins in peaches are also expressed in apples, apricots, pears, strawberries, and birch pollen, be wary of cross-allergic reactions.

Influencing factors

Controllable factors

1. Consumption method: Eating fresh peaches, especially without peeling, may increase the risk of allergic reactions.

Uncontrollable factors

1. Genetics: Having a family history of allergies, especially food allergies, might increase susceptibility.
2. Age: Peach allergy is more common in children.
3. Other allergies: Individuals allergic to certain pollens (like birch pollen), fruits (like apples) might also be allergic to peaches.

Knowledge

Peach allergy

Allergic reactions are when the body's immune system reacts to typically harmless substances (allergens), mistaking them as threats. The main allergens in peaches are present in both the skin and the flesh. The major allergenic proteins in peaches include:

- Pru p 1 (Pathogenesis-related protein family 10): The main allergenic protein in peaches, highly expressed in the skin. This protein is very sensitive to heat treatment and gastric protease digestion.
- Pru p 2 (Profilin protein family): Highly expressed in mature fruits, generally causes fewer allergic reactions.
- Pru p 3 (Non-specific lipid transfer protein family): An allergenic protein for over 90% of individuals allergic to peaches, highly expressed in the skin. Its tight three-dimensional structure makes it highly resistant to high temperatures and gastric protease digestion.
- Pru p 4 (Thaumatococcus-like protein family): Widely present in the skin and flesh of peaches, generally causes fewer allergic reactions.
- Pru p 7 (Gibberellin-regulated protein family): Highly expressed in both the skin and flesh of peaches, similar to Pru p 3 protein, has high conservation and thermal stability, posing a higher allergenicity risk.

Note that skin discomfort caused by touching vellus hair (peach fuzz) or peach skin is often due to skin sensitivity, where the skin easily reacts to various non-specific stimuli (e.g., temperature changes, friction, acidity/alkalinity), and usually is not an allergic reaction.

Prevalence

- Globally, 0.1-4.3% of the population suffer from fruit allergies, with peaches being a common trigger. Peaches are widely distributed in temperate, subtropical, and tropical areas, making allergies common among populations in Europe, the Americas, and Asia.
- A study from Beijing's Peking Union Medical College Hospital on children aged 4-9 showed that peaches are the most common fruit causing allergic reactions in children.

Typical symptoms

- Mild symptoms: Oral itching or stinging, hives, and stomach discomfort.
- Severe symptoms: Swelling of the face, throat, or other body parts, difficulty breathing, dizziness, or anaphylactic shock.

Treatment

- Mild symptoms: Antihistamines (like cetirizine, loratadine) can be used for treatment.
- Severe symptoms: If any signs of anaphylactic shock appear, an epinephrine auto-injector and emergency medical assistance should be sought immediately.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS28359884	intergenic	AC	may have a moderate risk of allergic reaction after exposure to peaches

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of peaches, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to peaches in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to peaches relative to those without allergic reactions to peaches.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Peaches test report uses 1 polymorphic locus within intergenic regions. The results showed whether the user had a higher exposure risk of peaches than all users in the genotype of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following peaches exposure. The basis of the test is mainly from the research of Japanese.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of peaches.
2. The test results have no clinical significance.

3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of peaches at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of peaches, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs28359884, related to exposure risk of peaches.

Population samples

The basis of the test is mainly from the research of Japanese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of peaches.

Cow's Milk

It is rich in whey protein and casein, which may cause allergic reactions after ingestion.

My Cow's Milk exposure

risk is moderate

Among the 8 genotypes associated with the risk of allergic reaction after exposure to cow's milk, the weighted risk score is close to the average of the population, and the risk of allergic reaction after exposure to cow's milk is moderate.

Suggestions

1. **Breastfeeding for newborns:** It is recommended that newborns be exclusively breastfed for the first 6 months. Mothers should avoid consuming cow's milk and its products. If Formula is needed, consider choosing a hypoallergenic formula.
2. **Dietary avoidance during breastfeeding:** Avoidance of cow's milk protein and related products in the mother's diet can reduce the risk of newborn allergies.
3. **Timing of first-time consumption:** Research suggests that introducing dairy products early in an infant's life may reduce the risk of developing allergies. However, it should start with a small amount of cow's milk.
4. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and effectiveness of treatment methods vary from person to person. Regarding allergen immunotherapy for food, in particular, remains a relatively new field. If necessary, it should be discussed with a doctor to weigh the pros and cons and carried out under their supervision, avoiding self-experimentation.

* Individuals diagnosed with a cow's milk allergy should avoid consuming cow's milk and related products (such as cheese, butter, baked goods, ice cream, whey protein powder). Check processed food labels; consider plant proteins and meats as alternative nutritional sources. Be aware of cross-reactivity, for instance, cow's milk allergy is related to sheep's milk allergy. When dining out, consult the restaurant staff or chef in advance to ensure there is no cow's milk or cross-contamination risk.

Influencing factors



Controllable factors

1. Newborn feeding method: Research indicates that adequate breastfeeding may affect the risk of cow's milk allergy.
2. Transfer through breast milk: A small amount of cow's milk protein can sensitize an infant when transferred through breast milk.
3. First-time consumption: The timing of an infant's intake of cow's milk may influence the risk of cow's milk allergy.



Uncontrollable factors

1. Genetics: Individuals with a family history of food allergies may have increased risk.
2. Age: Cow's milk allergies are more common in infants.

Knowledge



Cow's milk allergy

An allergic reaction occurs when the body's immune system reacts to normally harmless substances (allergens) as if they are threatening. The allergenic proteins in cow's milk mainly include:

- Whey protein (approximately 20% of cow's milk protein): Of which, β -lactoglobulin has high allergenicity. This protein is not present in breast milk, hence can trigger an immune response. This protein is resistant to digestive enzymes.
- Casein (approximately 80% of cow's milk protein): Of which, α -S1-casein has high allergenicity. This protein is not present in breast milk, hence can trigger an immune response.



Cow's milk allergy and lactose intolerance

- Cow's milk allergy involves the immune system and can cause allergic symptoms like hives and anaphylactic shock. Those allergic to cow's milk can digest it normally.
- Lactose intolerance generally does not involve the immune system. Due to a lack of lactase in the body, lactose in cow's milk cannot be effectively digested; a high concentration of lactose in the small intestine can cause watery diarrhea. At the same time, lactose is fermented by bacteria in the colon, producing gas and leading to bloating and abdominal cramps.



Prevalence

- Globally, about 2-7.5% of the population may develop a cow's milk allergy.
- Cow's milk allergy is common among infants. Research indicates that the incidence rate of cow's milk protein in infants under 1 year old in China is 2.69%.
- As age increases, some children may see an improvement in their cow's milk allergy symptoms. Statistics show that over 50% of children allergic to cow's milk and eggs may develop spontaneous tolerance after the age of 5-10 years, lasting a lifetime.

Typical symptoms

- Mild to moderate symptoms: rashes, itching, hives or swelling; abdominal pain, diarrhea, nausea or vomiting; nasal congestion, flu-like symptoms or sore throat.
- Severe symptoms: shortness of breath or asthma; severe abdominal pain, persistent diarrhea, severe nausea and vomiting; anaphylactic shock, including a sudden drop in blood pressure, rapid breathing, accelerated heart rate, and can be life-threatening.

Treatment

- Mild to moderate symptoms: Antihistamine treatments like cetirizine, prednisone; for diarrhea, montmorillonite and probiotics can be used.
- Severe symptoms: When any signs of anaphylactic shock appear, use an epinephrine auto-injector immediately and seek emergency medical assistance.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS17616434	intergenic	CC	may have a higher risk of allergic reaction after exposure to cow's milk
RS1800896	intergenic	TT	may have a higher risk of allergic reaction after exposure to cow's milk
RS2069772	intergenic	CT	may have a higher risk of allergic reaction after exposure to cow's milk
RS12123821	intergenic	CC	may have a lower risk of allergic reaction after exposure to cow's milk

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of cow's milk, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to cow's milk in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to cow's milk relative to those without allergic reactions to cow's milk.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Cow's Milk test report uses 4 polymorphic loci within intergenic regions. The results showed whether the user had a higher exposure risk of cow's milk than all users in the genotype combination of the 4 polymorphic loci tested, and that the test did not represent the user's actual outcome following cow's milk exposure. The basis of the test is mainly from the research of Caucasian and Chinese.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of cow's milk.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of cow's milk at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of cow's milk, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 4 polymorphic loci related to exposure risk of cow's milk, i.e., rs1800896, rs2069772, rs12123821 and rs17616434

Population samples

The basis of the test is mainly from the research of Caucasian and Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of cow's milk.

Peanuts

It contains various seed storage proteins, which may cause allergic reactions after ingestion.

My Peanuts exposure

risk is slightly high

Among the 12 genotypes associated with the risk of allergic reaction after exposure to peanuts, the weighted risk score is higher than the average of the population, and the risk of allergic reaction after exposure to peanuts is slightly high.

Suggestions

1. Improve consumption methods: Avoid eating roasted peanuts.
2. Timing of first-time consumption: Some studies have found that introducing peanuts into the diet of infants between 4-6 months might help prevent allergies, but it is advised to start with a small amount of peanuts.
3. Breastfeeding for newborns: It is recommended that newborns are exclusively breastfed for the first 6 months, providing all the necessary nutrients and allergy protection.
4. Supplement vitamin D: The general population can obtain enough vitamin D through moderate sun exposure and diet.
5. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and effectiveness of treatment methods vary from person to person. Regarding allergen immunotherapy for food, in particular, remains a relatively new field. If necessary, it should be discussed with a doctor to weigh the pros and cons and carried out under their supervision, avoiding self-experimentation.

* Individuals diagnosed with peanut allergies should avoid consuming peanuts and related products (such as condiments, pastries). Be sure to check the labels on processed foods; many who are allergic to peanuts (not a nut) also frequently have a nut allergy. When dining out, consult restaurant staff or chefs in advance to ensure no peanuts or cross-contamination risks.

Influencing factors



Controllable factors

1. Consumption method: Roasted peanuts can significantly increase the risk of allergies.
2. First-time consumption: The timing of introducing peanuts into the diet can affect the risk of peanut allergies.
3. Newborn feeding method: Studies have shown that whether there is sufficient breastfeeding may influence the risk of peanut allergies.
4. Vitamin D: Research indicates that a lack of vitamin D might increase the risk of peanut allergies.



Uncontrollable factors

1. Genetics: A family history of allergies, especially food allergies, may increase susceptibility.

Knowledge



Peanut allergies

Allergic reactions occur when the body's immune system reacts to a typically harmless substance (allergen), mistakenly identifying it as a threat. Currently, 18 allergenic proteins have been identified in peanuts, most of which are seed storage proteins, including:

- Ara h 1: The most prevalent allergenic protein in peanuts with an allergy rate of about 65%. This protein has a highly stable homotrimeric structure, making it resistant to enzymatic digestion and heat-stable.
- Ara h 2: A major allergenic protein in peanuts with an allergy rate of about 85%. This protein is structurally stable, heat-stable, and resistant to enzymatic digestion.
- Ara h 3/4: A major allergenic protein in peanuts with an allergy rate of about 53%. This protein is structurally stable, heat-stable, and resistant to enzymatic digestion.
- Ara h 6: A major allergenic protein in peanuts with an allergy rate of about 38%, typically related to mild allergic reactions. This protein is structurally stable, heat-stable, acid-resistant, and resistant to enzymatic digestion.



Prevalence

- The incidence of peanut allergies in the general population is approximately 1.5%. In the US, the rate is about 2.2%, in Australia about 3%, and in Europe about 9.8%. A survey on self-reported food allergies among Chinese college students indicated a 0.6% incidence rate for peanut allergies.
- Unlike other food allergies, peanut allergies are less likely to disappear with age. Approximately 90% of individuals with peanut allergies will have life-long allergies and even small amounts can trigger severe reactions.
- Peanut allergies account for the highest number of deaths among food allergies.



Typical symptoms

- Mild symptoms: Reddened skin, hives or itching, runny nose, or stomach cramps.

- Moderate symptoms: Tightness in the throat, hoarse voice, wheezing or coughing.
- Severe symptoms: Can lead to anaphylactic shock, manifested by a sudden drop in blood pressure, accelerated pulse, difficulty breathing, and loss of consciousness.

Treatment

- Mild to moderate symptoms: Antihistamines (such as cetirizine, loratadine) can be used for treatment.
- Severe symptoms: Seek professional medical assistance immediately; those at high risk should always carry an epinephrine auto-injector.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS7192	HLA-DRA	GT	may have a higher risk of allergic reaction after exposure to peanuts
RS9275596	intergenic	CT	may have a higher risk of allergic reaction after exposure to peanuts
RS12123821	intergenic	CC	may have a lower risk of allergic reaction after exposure to peanuts
RS9273440	intergenic	CT	may have a higher risk of allergic reaction after exposure to peanuts
RS11949166	intergenic	AT	may have a higher risk of allergic reaction after exposure to peanuts

My haplotype details

HLA-DQB1	People without the HLA-DQB1-*02 genotype	*06:02/*05:02
	may have some risk of allergic reaction after exposure to peanuts.	

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of peanuts, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic

reactions to peanuts in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to peanuts relative to those without allergic reactions to peanuts.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Peanuts test report uses 5 polymorphic loci on *HLA-DRA* and *HLA-DQB1* genes and intergenic regions; Also, genotyping results of *HLA-DQB1*. The results showed whether the user had a higher exposure risk of peanuts than all users in the genotype combination of the 5 polymorphic loci and *HLA-DQB1* genotyping results tested, and that the test did not represent the user's actual outcome following peanuts exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of peanuts.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of peanuts at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of peanuts, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes genotyping results of *HLA-DQB1*; And 5 polymorphic loci related to exposure risk of peanuts, such as rs7192, rs9273440 and rs9275596



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of peanuts.

Hen's Eggs

It contains proteins such as ovomucoid and ovalbumin, which may cause allergic reactions after ingestion.

My Hen's Eggs exposure

risk is slightly low

Compared with the population average, the risk of allergic reaction after exposure to hen's eggs is slightly low.

Suggestions

1. Consume well-cooked eggs: Heat treatment can change the allergenic properties of proteins in eggs. Allergic potential in order of likelihood: raw egg > soft-boiled egg > hard-boiled egg > braised egg.
2. Timing of first-time consumption: It is recommended to introduce eggs into an infant's diet at 6 months, starting with a small amount of egg yolk.
3. Breastfeeding for newborns: Exclusive breastfeeding for the first 6 months is recommended, providing all necessary nutrients and allergy protection.
4. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and effectiveness of treatment methods vary from person to person. Regarding allergen immunotherapy for food, in particular, remains a relatively new field. If necessary, it should be discussed with a doctor to weigh the pros and cons and carried out under their supervision, avoiding self-experimentation.

* Those diagnosed with an egg allergy should avoid consuming eggs and related products (like cakes, egg rolls, and bread). Always read labels on processed foods. Consider choosing plant or animal proteins as an alternative source of nutrition. When dining out, consult the restaurant staff or chef in advance to ensure there is no egg content or risk of cross-contamination.

Influencing factors



Controllable factors

1. Cooking degree of the egg: The risk of an allergic reaction increases with undercooked eggs.
2. First-time consumption: The timing of introducing eggs into the diet may influence the risk of developing an allergy.
3. Newborn feeding method: Studies suggest that exclusive breastfeeding might influence the risk of developing an egg allergy.



Uncontrollable factors

1. Genetics: Having a family history of allergies, especially food allergies, can increase susceptibility.
2. Age: Allergies to hen's eggs are more common in children.
3. Other diseases: Individuals with atopic dermatitis or other types of allergies might be more susceptible to developing an allergy to hen's eggs.

Knowledge



Egg allergy

An allergic reaction is when the body's immune system reacts to a usually harmless substance (allergen) as if it was harmful. Egg allergens are mainly found in egg whites, with fewer in egg yolks. The main allergenic proteins in eggs include:

- Gal d 1 (Ovomucoid): A pancreatic protease inhibitor glycoprotein in egg white and a major allergen. It is heat-stable and not easily denatured during cooking.
- Gal d 2 (Ovalbumin): A water-soluble glycoprotein that is most abundant in egg white and a major allergen.
- Gal d 3 (Ovotransferrin): Present in egg white and can trigger allergies. Heating can change its structure, reducing its allergenicity.
- Gal d 4 (Lysozyme): Present in egg white, it has antimicrobial properties and protects the egg. It rarely triggers allergic reactions.
- Gal d 5 (α -Livitin): Found in egg yolk, it mainly causes allergies through the respiratory tract. Heating may not necessarily reduce its allergenicity.
- Gal d 6 (Yolk glycoprotein 42): Present in egg yolk and can trigger allergies. Heating may not necessarily reduce its allergenicity.



Prevalence

- Egg allergy is one of the most common food allergies in children, with a global prevalence of about 1-2% in infants and toddlers. A study targeting Chinese children aged 3-12 showed that 8.4% had food allergies, with more than half allergic to eggs.
- According to various studies, about 70% of children allergic to eggs will develop a tolerance before adolescence. However, if the allergy persists into adulthood, improvement is unlikely.



Typical symptoms

- Mild symptoms: Skin redness and itching, runny nose, tearing, stomach pain, nausea, or diarrhea.
- Severe symptoms: Breathing difficulties, wheezing, swelling of the face or throat, increased heart rate, drop in blood pressure, and anaphylactic shock.

 Treatment

- Mild symptoms: Antihistamines (e.g., cetirizine, loratadine) can be used for treatment.
- Severe symptoms: Seek immediate professional medical assistance; use an epinephrine auto-injector for first aid and call emergency services.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS12123821	intergenic	CC	may have a lower risk of allergic reaction after exposure to hen's eggs
RS11949166	intergenic	AT	may have a higher risk of allergic reaction after exposure to hen's eggs
RS1243064	intergenic	AT	may have a higher risk of allergic reaction after exposure to hen's eggs

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of hen's eggs, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to hen's eggs in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to hen's eggs relative to those without allergic reactions to hen's eggs.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

 Applicable situation

The Hen's Eggs test report uses 3 polymorphic loci within intergenic regions. The results showed whether the user had a higher exposure risk of hen's eggs than all users in the genotype combination of the 3 polymorphic loci tested, and that the test did not represent the user's actual outcome following hen's eggs exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of hen's eggs.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of hen's eggs at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of hen's eggs, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes genotyping results of 3 polymorphic loci related to exposure risk of hen's eggs, i.e., rs1243064, rs12123821 and rs11949166.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of hen's eggs.

Endotoxins

It refers to a kind of lipopolysaccharide substances that may be exposed in agriculture, textile industry, sewage treatment and other industries, which may cause a series of symptoms of lung dysfunction such as wheezing.

My Endotoxins exposure

risk is slightly low

Compared with the population average, the risk of wheeze after exposure to endotoxins is slightly low.

Suggestions

1. Personal protection: Workers should wear appropriate masks, gloves, and protective clothing.
2. Personal hygiene: Regular hand washing and shower disinfection can reduce the risk of endotoxin exposure.
3. Clean working environment: Installing exhaust ventilation, using wet dust suppression methods, and maintaining a clean workspace are recommended.
4. Risk education: Train workers on the risks associated with endotoxins and protection measures.
5. Environmental monitoring: Regular air quality assessment and endotoxin level measurements help ensure a safe environment.

Knowledge

Endotoxins

Toxins play a crucial role in bacterial pathogenicity and are categorized into exotoxins and endotoxins. Specifically, endotoxins are components of the outer membrane cell wall of Gram-negative bacteria like *E. coli* and *Salmonella*. The primary active ingredient is lipopolysaccharide (LPS). When these bacteria divide or die, endotoxins are released, which can cause various pathogenic effects on the human body, including fever, endotoxemia, endotoxic shock, and even death. Even a minute amount of endotoxins entering the body can cause fever and discomfort. Hence, endotoxin testing is required in many biopharmaceutical production processes and products.

Asthma

Asthma is characterized by a high-pitched whistling sound during exhalation, usually resulting from narrowed or obstructed airways. It might also be triggered by allergic asthma and lower respiratory tract inflammation.

 Endotoxins and asthma

Airborne endotoxins, combined with particles like dust, form aerosols that, when inhaled, can cause an inflammatory reaction in the respiratory tract. The inflammation caused by endotoxin exposure can lead to bronchoconstriction, increased mucus production, and airway edema. These physiological changes in the airways are the reason for the occurrence of asthma.

 High-risk occupations

- Agricultural workers: Contaminated crops, grain dust, and animal products can all be sources of endotoxin exposure.
- Textile workers: Cotton and other organic fibers might contain endotoxins.
- Waste recycling workers: Decomposition of waste might release endotoxins.
- Wastewater treatment workers: Raw sewage might contain high levels of Gram-negative bacteria and endotoxins.

 Endotoxin tolerance

Endotoxin tolerance is a crucial mechanism in the body's ability to maintain immune balance when faced with potential threats. It helps regulate immune responses, preventing excessive inflammatory reactions and maintaining overall health.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS2569190	CD14	AA	may have a lower risk of wheeze after exposure to endotoxins
RS2569191	CD14	CC	may have a lower risk of wheeze after exposure to endotoxins
RS2915863	intergenic	CC	may have a lower risk of wheeze after exposure to endotoxins
RS10808798	intergenic	CT	may have a higher risk of wheeze after exposure to endotoxins

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of endotoxins. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of endotoxins in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with endotoxins exposure relative to those without endotoxins exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Endotoxins test report uses 4 polymorphic loci on *CD14* gene and intergenic regions. The results showed whether the user had a higher exposure risk of endotoxins than all users in the genotype combination of the 4 polymorphic loci tested, and that the test did not represent the user's actual outcome following endotoxins exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of endotoxins.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of endotoxins at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of endotoxins, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 4 polymorphic loci related to exposure risk of endotoxins, i.e., rs2915863, rs2569191, rs2569190 and rs10808798.



Population samples

The basis of the test is mainly from the research of Caucasian.



Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of endotoxins.

Cleaning Agents

It is a chemical used to remove dirt, stains and odor on the surface of objects. Although cleaning agents help maintain hygiene, their volatiles or residues may damage the health of the respiratory system and trigger diseases such as adult-onset asthma.

My Cleaning Agents exposure

risk is moderate

Among the 10 genotypes associated with the risk of adult-onset asthma after exposure to cleaning agents, the weighted risk score is close to the average of the population, and the risk of adult-onset asthma after exposure to cleaning agents is moderate.

Suggestions

1. Follow usage instructions: Strictly adhere to the safety guidelines provided by the cleaning agent product.
2. Ensure ventilation: Always maintain good air circulation in areas where cleaning agents are used.
3. Use personal protective equipment: Wear masks and gloves when dealing with strong cleaning agents (e.g., bleach).
4. Switch products: Opt for cleaning agents that do not contain harmful chemical ingredients.
5. Safe storage: Store cleaning agents in a cool, dry place, away from children and pets.

Knowledge

Cleaning agents

Cleaning agents typically contain one or more active ingredients, such as surfactants, acidic and alkaline components, oxidizers, etc. Depending on the usage scenario, they can be categorized as detergents, disinfectants, abrasives, and solvents, and are widely used in households, industries, medical settings, and the catering industry.

Adult-onset asthma

Unlike childhood asthma, adult-onset asthma usually occurs in individuals aged 20 and above. Symptoms include wheezing, shortness of breath, chest tightness, and persistent coughing.

Cleaning agents and adult-onset asthma

Certain cleaning agents may contain volatile organic compounds (VOCs). These substances can evaporate into the air and irritate the respiratory system, causing airway inflammation and asthma symptoms.

High-risk occupations

- Cleaning staff: Frequently exposed to disinfection environments in various locations and areas.
- Industrial cleaners: Often exposed to disinfection environments around different machinery and equipment.
- Medical professionals: Regularly exposed to the disinfection of medical devices and facilities.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS931127	SIPA1, PCNXL3	GG	may have a higher risk of adult-onset asthma after exposure to cleaning agents
RS2667026	intergenic	AG	may have a higher risk of adult-onset asthma after exposure to cleaning agents
RS7949980	intergenic	CT	may have a higher risk of adult-onset asthma after exposure to cleaning agents
RS1958980	PRKD1	AG	may have a higher risk of adult-onset asthma after exposure to cleaning agents
RS11847351	PRKD1	AG	may have a higher risk of adult-onset asthma after exposure to cleaning agents

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of cleaning agents. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of cleaning agents in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with cleaning agents exposure relative to those without cleaning agents exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Cleaning Agents test report uses 5 polymorphic loci on *SIPA1* and *PRKD1* genes and intergenic regions. The results showed whether the user had a higher exposure risk of cleaning agents than all users in the genotype combination of the 5 polymorphic loci tested, and that the test did not represent the user's actual outcome following cleaning agents exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of cleaning agents.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of cleaning agents at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of cleaning agents, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 5 polymorphic loci related to exposure risk of cleaning agents, such as rs931127, rs1958980 and rs11847351.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of cleaning agents.

Processed Meat

It includes sausage, bacon, ham and other cured or smoked meat. Processing technology can extend the shelf life of meat and enhance the flavor of meat, but long-term intake of processed meat may lead to an increased risk of colorectal cancer.

My Processed Meat exposure

risk is slightly low

Compared with the population average, the risk of colorectal cancer after exposure to processed meat is slightly low.

Suggestions

1. Adjust dietary habits: Reduce the intake of processed meats and choose lean meats or plant-based proteins.
2. Increase physical activity: Regular exercise can help reduce the risk of colorectal cancer.
3. Quit smoking and limit alcohol: Limiting alcohol intake and avoiding smoking can lower the risk of colorectal cancer.
4. Regular screenings: Regular colorectal screenings (e.g., colonoscopy) can help detect early cancerous changes.

Influencing factors



Controllable factors

1. Dietary habits: Long-term excessive consumption of processed meat can increase the risk of colorectal cancer.
2. Lifestyle: A sedentary lifestyle may increase the risk of colorectal cancer.
3. Smoking and drinking: Smoking or excessive alcohol consumption can increase the risk of colorectal cancer.



Uncontrollable factors

1. Genetics: Individuals with a family history of colorectal cancer may have a higher risk.

2. Age: The incidence of colorectal cancer increases with age.

Knowledge



Processed meat and colorectal cancer

The World Health Organization (WHO) has classified processed meat as a Group 1 carcinogen, meaning there is sufficient evidence of its carcinogenicity. The International Agency for Research on Cancer (IARC) points out that consuming 50 grams of processed meat daily (equivalent to about two slices of bacon or a sausage) is associated with an increased risk of colorectal cancer. Specific factors contributing to the increased risk include:

- **Cooking methods:** When processed meats are cooked at high temperatures (e.g., frying or grilling), harmful compounds such as polycyclic aromatic hydrocarbons (PAHs) and heterocyclic amines (HCAs) may form, increasing cancer risk.
- **Preservatives and additives:** Processed meats often contain nitrates and nitrites, which can convert into N-nitroso compounds (NOCs) in the body, known carcinogens.
- **High fat content:** Processed meats often have a high fat content, and high-fat diets are linked to colorectal cancer.
- **Gut microbiota:** Gut microbes ferment some residues of processed red meat that cannot be digested by intestinal enzymes, such as protein residues and bile acids stimulated by fats, leading to inflammation or carcinogenic metabolites, increasing cancer risk.



Prevalence

- Processed meat, especially pork, is favored by consumers due to its taste and convenience. It is especially popular among African-Americans and Hispanic populations.
- The global processed meat market is steadily growing. In 2023, the USA was the largest revenue country for processed meat. In 2021, Asia's demand for processed meat accounted for 41.7% of the global market.
- A Western diet rich in processed and red meats may increase the incidence of colorectal cancer.
- Colorectal cancer is the 3rd most common cancer worldwide, characterized by high incidence and mortality rates. According to IARC statistics, millions are diagnosed with colorectal cancer every year, with developed countries generally reporting higher rates.



Typical symptoms of colorectal cancer

Common symptoms include changes in bowel habits, rectal bleeding, persistent abdominal discomfort, weakness or fatigue, and unexplained weight loss.



Treatment for colorectal cancer

- **Surgical intervention:** Surgically removing cancerous polyps or affected parts is a common treatment method.
- **Other treatments:** Depending on the stage and location of the cancer, treatments may also include radiation, chemotherapy, or targeted drug therapy.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS4143094	intergenic	GG	may have a lower risk of colorectal cancer after exposure to processed meat

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of processed meat, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to processed meat in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to processed meat relative to those without allergic reactions to processed meat.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Processed Meat test report uses 1 polymorphic locus on *GATA3* gene. The results showed whether the user had a higher exposure risk of processed meat than all users in the genotype of the 1 polymorphic loci tested, and that the test did not represent the user's actual outcome following processed meat exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of processed meat.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of processed meat at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of processed meat, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs4143094, related to exposure risk of processed meat.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of processed meat.

Night Shift

It refers to working at night or late at night. Working at night will affect the normal periodic rhythm of human life activities (i.e. biological clock), which may damage health in the long run. For example, the risk of stroke may increase in night shift workers.

My Night Shift exposure

risk is moderate

People with the rs10830963-CG genotype may have a moderate risk of stroke after exposure to night shift.

Suggestions

1. Regular sleep schedule: Try to maintain a consistent sleep schedule (including fixed sleeping and waking times) to help regulate the biological clock.
2. Quality rest: Use blackout curtains, reduce noise, control temperature, etc., to create an environment conducive to deep sleep.
3. Maintain lighting: Ensure that the night shift workspace is well-lit, simulating daytime conditions to aid biological clock regulation.
4. Flexible scheduling: Implement rotating shifts to avoid prolonged night work.
5. Regular check-ups: Periodically undergo medical check-ups and be informed about the potential risks associated with night shifts.

Knowledge



Night shift

Night shift typically involves working hours outside the traditional 9-to-5 schedule. It is a common work arrangement, especially in industries that require 24/7 services, such as healthcare, transportation, hospitality, emergency services, etc.



Stroke

Stroke, sometimes called a brain attack, is a sudden interruption or rupture of blood supply to the brain. As brain tissues cannot get adequate oxygen and nutrients, this can lead to brain cell death,

resulting in severe neurological and cognitive problems. Symptoms might include sudden numbness, confusion, vision problems, and severe headaches.

 Night shift and stroke

Research suggests that night shift work might be associated with an increased risk of stroke. The disruption of the biological clock due to night shift can lead to metabolic disturbances, hypertension, and inflammation, all of which are related to stroke risk.

 High-risk occupations

- Medical professionals: Nurses, doctors, and other healthcare workers required to work at night.
- Factory production Workers: Individuals involved in 24-hour production cycles.
- Security personnel: Guards and monitoring staff required to work at night.
- Transportation workers: Truck drivers, train operators, and pilots often need to work during night hours.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS10830963	MTNR1B	CG	may have a moderate risk of stroke after exposure to night shift

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of night shift. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of night shift in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with night shift exposure relative to those without night shift exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Night Shift test report uses 1 polymorphic locus on *MTNR1B* gene. The results showed whether the user had a higher exposure risk of night shift than all users in the genotype combination of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following night shift exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of night shift.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of night shift at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of night shift, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs10830963, related to exposure risk of night shift.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of night shift.

Wheat Products

Wheat has high nutritional value and palatability, and can be processed into many foods, such as bread, beer, etc. Some individuals will have allergic reactions to ingredients (such as protein) in wheat products after ingestion, contact or inhalation of wheat products.

My Wheat Products exposure

risk is slightly low

Compared with the population average, the risk of allergic reaction after exposure to wheat products is slightly low.

Suggestions

1. Occupational protection: Wear masks and goggles when baking; ensure ventilation and regular cleaning in the baking area.
2. Timing of first-time consumption: There is controversy over whether to introduce or avoid wheat products early for infants. You can try to gradually introduce small amounts of wheat products into the diet and closely observe.
3. **Desensitization therapy:** Even in instances of allergy, prolonged exposure to allergens at low doses may lead to the gradual development of adaptability in the body. This forms the theoretical basis of Immunotherapy. The approach involves administering gradually increasing doses of the allergen to patients, guiding the immune system to generate regulatory responses, thereby reducing the frequency and severity of allergic reactions. As the treatment progresses, there is a potential for alleviation or disappearance of symptoms in some patients. It is crucial to emphasize that the choice and effectiveness of treatment methods vary from person to person. Regarding allergen immunotherapy for food, in particular, remains a relatively new field. If necessary, it should be discussed with a doctor to weigh the pros and cons and carried out under their supervision, avoiding self-experimentation.

* Individuals diagnosed with wheat allergies should avoid ingesting, contacting, or inhaling wheat products and related products (including cosmetics); pay attention to product labels; consider choosing almond flour, coconut flour, rice flour, and potato flour as alternative nutrition sources; be vigilant for cross-reactive allergies, e.g., wheat allergy might be related to peach allergy; when dining out, consult restaurant staff or chefs in advance to ensure no wheat or cross-contamination risks.

Influencing factors

Controllable factors

1. Occupation: Engaging in baking may lead to frequent inhalation of wheat dust, thus increasing the risk of allergies.
2. First-time consumption: The timing of introducing wheat products into the diet may affect the risk of wheat allergies.

Uncontrollable factors

1. Genetics: Individuals with a family history of allergies or allergic diseases (such as asthma, eczema) may have an increased risk.
2. Age: Wheat allergies are more common in children.

Knowledge

Wheat allergy

An allergic reaction is when the body's immune system responds to typically harmless substances (allergens) as if they are threatening. Those allergic to wheat have an abnormal immune response to at least one protein in wheat. 28 allergenic proteins in wheat have been published so far, including:

- Tri a 14 (Non-specific lipid transfer protein 1): A major allergen that can lead to rapid-onset wheat allergy.
- Tri a 19 (ω -5-gliadin): A seed storage protein and a main allergen.
- Tri a 36 (Low-molecular-weight glutenin): A seed storage protein leading to rapid-onset wheat allergy.
- Tri a 37 (α -amylase inhibitor): A plant defense protein present in wheat seeds, which can increase allergy risk.

Wheat allergy, celiac disease, and non-celiac wheat sensitivity

Different allergenic proteins, pathways, and organ responses can lead to various wheat-related conditions:

- Wheat allergy: Triggered upon ingestion, it is a rapid-onset allergic reaction (mainly IgE mediated) caused by allergenic proteins in wheat.
- Celiac disease: Triggered upon ingestion, it is an autoimmune disease. Gluten (a family of storage proteins that are naturally found in wheat) triggers specific immunity, causing the body's antibodies to attack its own intestinal tissue and affect the whole body.
- Non-celiac wheat sensitivity: Triggered upon ingestion, it does not involve allergy or autoimmune reactions. Its symptoms are similar to celiac disease, possibly due to other substances in wheat products causing intestinal inflammation.

Prevalence

- Globally, the prevalence of wheat allergy is around 1%. A meta-analysis in 2023 reported that the positive detection rate for wheat allergy in China is 7.30%, especially in northern areas like Shaanxi,

Henan, and Inner Mongolia where the rate exceeds 10%.

- Wheat allergy is more common in children. In Europe, the prevalence in children is between 0.5-9%.
- A retrospective study of anaphylaxis patients from 2000-2014 at Peking Union Medical College Hospital showed that wheat was the main culprit, accounting for 37% of all triggers. Wheat caused 57% of severe allergic reactions.

Typical symptoms

- Mild symptoms: Hives, difficulty breathing, sneezing, abdominal discomfort.
- Severe symptoms: Swelling, difficulty breathing, drop in blood pressure, and even anaphylactic shock.

Treatment

- Mild symptoms: Antihistamines (e.g., cetirizine, loratadine) can be used for treatment.
- Severe reactions: Administer epinephrine immediately and seek emergency care.
- Ongoing management: A strict wheat-free diet is an essential health management measure; consultation with a dietitian familiar with wheat allergy is recommended for support.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS9271588	intergenic	TT	may have a lower risk of allergic reaction after exposure to wheat products
RS59325236	intergenic	GG	may have a lower risk of allergic reaction after exposure to wheat products

My haplotype details

HLA-DQB1	People without the HLA-DQB1-*06 genotype may have some risk of allergic reaction after exposure to wheat products. People with the HLA-DQB1-*06 genotype may have a lower risk of allergic reaction after exposure to wheat products.	*06:02/*05:02

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of wheat products, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to wheat products in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to wheat products relative to those without allergic reactions to wheat products.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Wheat Products test report uses 2 polymorphic loci within intergenic regions; Also, genotyping result of *HLA-DQB1*. The results showed whether the user had a higher exposure risk of wheat products than all users in the genotype combination of the 2 polymorphic loci and *HLA-DQB1* genotyping result tested, and that the test did not represent the user's actual outcome following wheat products exposure. The basis of the test is mainly from the research of Japanese.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of wheat products.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of wheat products at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.

3. If you feel you have some exposure risk of wheat products, it is recommended that you consult an authoritative health professional or clinician for help.

 Test content

The test includes genotyping result of *HLA-DQB1*; And 2 polymorphic loci related to exposure risk of wheat products, i.e., rs59325236 and rs9271588

 Population samples

The basis of the test is mainly from the research of Japanese.

 Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of wheat products.

Gases and Fumes

It is air pollutants produced by various industrial processes, combustion and other human activities. Prolonged exposure to certain industrial fumes may lead to the development or exacerbation of respiratory diseases, including chronic obstructive pulmonary disease (COPD).

My Gases and Fumes exposure

risk is slightly low

Compared with the population average, the risk of chronic obstructive pulmonary disease (COPD) after exposure to gases and fumes is slightly low.

Suggestions

1. Personal protection: Individuals at high exposure risk should wear appropriate masks and respirators.
2. Ensure ventilation: Ensure adequate ventilation in areas where gases and fumes are generated or released.
3. Risk education: Understand potential risks associated with specific gases and fumes and protective measures.
4. Regular monitoring: Periodically monitor air quality in potentially exposed locations.
5. For those at high exposure risk, quitting smoking and avoiding secondhand smoke are effective preventive measures.

Knowledge



Gases and fumes

Gases and fumes typically consist of various emissions like exhaust gases, smoke, etc. These emissions often contain various chemicals and particulates such as sulfur dioxide (SO₂), nitrogen oxides (NO_x), volatile organic compounds (VOCs), particulate matter (PM), heavy metals like lead, mercury, cadmium, and other harmful substances.



Chronic obstructive pulmonary disease (COPD)

COPD is a chronic inflammatory lung disease leading to obstructed airflow in the lungs. Symptoms include shortness of breath, cough, mucus production, and wheezing. Long-term exposure to irritant gases or particulates is a major cause.

 Gases and fumes and COPD

Long-term exposure to harmful substances in certain gases and fumes, such as welding fumes, mining dust, and chemical industry emissions, can adversely affect the respiratory system and increase the risk of developing COPD.

 High-risk occupations

- Welders: Often exposed to welding smoke and gases.
- Miners: Potential exposure to various underground gases.
- Chemical plant workers: Frequently in contact with various gases, posing higher exposure risks.
- Firefighters: Exposure to smoke produced by combustion.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS159497	intergenic	TT	may have a lower risk of chronic obstructive pulmonary disease (COPD) after exposure to gases and fumes
RS516732	intergenic	CC	may have a lower risk of chronic obstructive pulmonary disease (COPD) after exposure to gases and fumes

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of gases and fumes. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of gases and fumes in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with gases and fumes exposure relative to those without gases and fumes exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Gases and Fumes test report uses 2 polymorphic loci within intergenic regions. The results showed whether the user had a higher exposure risk of gases and fumes than all users in the genotype combination of the 2 polymorphic loci tested, and that the test did not represent the user's actual outcome following gases and fumes exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of gases and fumes.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of gases and fumes at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of gases and fumes, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 2 polymorphic loci related to exposure risk of gases and fumes, i.e., rs159497、rs516732.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of gases and fumes.

Chlorinated Solvents

It is a kind of chemical substance mainly used for industrial purposes, used as degreasing agent, cleaner and chemical synthesis. Prolonged exposure to these solvents may cause health problems, such as non-Hodgkin lymphoma (NHL).

My Chlorinated Solvents exposure

risk is moderate

People with the rs1805794-CC genotype may have a higher risk of non-hodgkin lymphoma (NHL) after exposure to chlorinated solvents.

Suggestions

1. Personal protection: Wear appropriate masks, gloves, and protective gear when handling chlorinated solvents.
2. Ensure ventilation: Ensure areas where solvents are used or stored are well-ventilated.
3. Minimize exposure: Limit the amount of time spent in areas with high concentrations of chlorinated solvents.
4. Risk education: Understand the potential risks of chlorinated solvents and protective measures.
5. Safe storage: Store the solvents in well-sealed containers in a cool, dry place away from heat or open flames.

Knowledge



Chlorinated solvents

Chlorinated solvents are organic solvents containing chlorine atoms, including trichloroethylene (TCE), tetrachloroethylene (PCE), and dichloromethane (DCM). They are commonly used in metal cleaning, paint stripping, and the production of adhesives and pharmaceuticals.



Non-Hodgkin lymphoma (NHL)

NHL is a group of malignant blood cancers originating from lymphocytes, a type of white blood cell. Its exact cause remains unclear, but certain factors, including prolonged exposure to some chemicals, may increase the risk.



Chlorinated solvents and NHL

Long-term exposure to chlorinated solvents may increase the risk of developing NHL, as the solvents can potentially damage DNA within cells, causing mutations that lead to cancer development.



High-risk occupations

- Industrial workers: Individuals involved in the production or regular use of chlorinated solvents may be at higher exposure risk.
- Dry cleaning employees: Tetrachloroethylene is commonly used as a solvent in dry cleaning.
- Mechanical workers: Degreasing of metal surfaces might involve the use of chlorinated solvents.
- Painters: Chlorinated solvents might be used in thinners and paint strippers.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS12917	MGMT	CC	may have a lower risk of non-hodgkin lymphoma (NHL) after exposure to chlorinated solvents
RS1805794	NBN	CC	may have a higher risk of non-hodgkin lymphoma (NHL) after exposure to chlorinated solvents

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of chlorinated solvents. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of chlorinated solvents in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with chlorinated solvents exposure relative to those without chlorinated solvents exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Chlorinated Solvents test report uses 2 polymorphic loci on *MGMT* and *NBN* genes. The results showed whether the user had a higher exposure risk of chlorinated solvents than all users in the genotype combination of the 2 polymorphic loci tested, and that the test did not represent the user's actual outcome following chlorinated solvents exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of chlorinated solvents.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of chlorinated solvents at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of chlorinated solvents, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 2 polymorphic loci related to exposure risk of chlorinated solvents, i.e., rs12917 and rs1805794.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of chlorinated solvents.

Mineral Dust

It refers to the fine particles produced from soil, rocks and minerals in various human activities and natural processes. Prolonged inhalation of mineral dust (especially from occupational sources) may lead to lung diseases, including chronic obstructive pulmonary disease (COPD).

My Mineral Dust exposure

risk is slightly low

Compared with the population average, the risk of chronic obstructive pulmonary disease (COPD) after exposure to mineral dust is slightly low.

Suggestions

1. Personal protection: Individuals at high exposure risk should wear appropriate masks and respirators.
2. Ensure ventilation: Ensure adequate ventilation in areas where mineral dust is generated.
3. Wet processing: Use wet processes when cutting or grinding minerals or rocks to suppress dust.
4. Risk education: Thoroughly understand the hazards of mineral dust and the importance of protective measures.
5. Regular monitoring: Conduct periodic air quality assessments in places prone to mineral dust exposure.

Knowledge

Mineral dust

Mineral dust primarily consists of silica (silicate minerals) but may also contain other minerals such as calcium carbonate, iron oxides, alumina, magnesium carbonate, etc. Activities like mining, construction, and agriculture can produce significant amounts of mineral dust.

Chronic obstructive pulmonary disease (COPD)

COPD is a chronic inflammatory lung disease that leads to obstructed airflow in the lungs. Symptoms include shortness of breath, cough, mucus production, and wheezing. Long-term exposure to irritant

gases or particulates is a major cause.

Mineral dust and COPD

Dust particles can cause irritation and inflammation in the airways, leading to narrowing and damage. With prolonged inhalation and repeated irritation, chronic symptoms and complications associated with COPD gradually develop.

High-risk occupations

- Miners: Those involved in coal, quartz, or hard rock mining face higher exposure risks.
- Construction workers: Those involved in demolition or significant soil-moving tasks have higher exposure risks.
- Agricultural workers: Dust produced during plowing and tilling poses an exposure risk.
- Stonemasons and bricklayers: Individuals involved in cutting, shaping, and installing stone face higher exposure risks.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS13278529	intergenic	TT	may have a lower risk of chronic obstructive pulmonary disease (COPD) after exposure to mineral dust

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of mineral dust. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of mineral dust in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with mineral dust exposure relative to those without mineral dust exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Mineral Dust test report uses 1 polymorphic locus within intergenic regions. The results showed whether the user had a higher exposure risk of mineral dust than all users in the genotype of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following mineral dust exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of mineral dust.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of mineral dust at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of mineral dust, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs13278529, related to exposure risk of mineral dust.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of mineral dust.

Occupational Stress

It refers to the psychological, emotional and physical pressure experienced by staff due to work-related factors. Long-term occupational stress may cause various health problems, such as hypertension and sleep disturbances.

My Occupational Stress exposure

risk is slightly high

People with the rs1801131-GT genotype may have a higher risk of hypertension after exposure to occupational stress.
People with the rs2304672-GG genotype may have a higher risk of sleep disturbances after exposure to occupational stress.

Suggestions

1. Job content adjustment: Change job responsibilities to reduce workload or mitigate occupational stress through role ambiguity.
2. Regular breaks: Encourage regular breaks to alleviate mental and physical fatigue of workers.
3. Team support: Create a supportive work environment through team building and effective communication.
4. Psychological counseling: Offer employee assistance programs or counseling services for stress management.
5. Enrich leisure life: Encourage cultivating personal hobbies and interests outside of work to achieve a work-life balance.

Knowledge



Occupational stress

Occupational Stress arises from a series of factors, including high job demands, lack of control, poor work relationships, and a sense of job insecurity. Persistent stress can impair mental health, reduce work efficiency and job satisfaction, and even lead to physiological alterations.



Hypertension

Hypertension is a condition where the force of blood against the arterial walls is abnormally high. Persistent hypertension can lead to severe complications, increasing the risk of heart diseases, stroke, and other health problems.

Sleep disturbances

Sleep disturbances encompass various conditions that affect an individual's ability to get regular, quality sleep. Influenced by lifestyle to various medical conditions, chronic sleep disturbances can lead to health problems such as obesity, diabetes, cardiovascular diseases, and weakened immune system.

Occupational stress and hypertension

Certain jobs with high stress and high demands can lead to prolonged occupational stress, directly associated with the onset and exacerbation of hypertension. Continuous activation of the body's stress response can keep blood pressure elevated for extended periods.

Occupational stress and sleep disturbances

Occupational stress is a significant contributor to sleep disturbances. Job-related psychological and physiological stress can make it challenging to relax and get restorative sleep. This can create a vicious cycle where sleep deprivation exacerbates the stress experienced during work hours, making high-quality sleep even more elusive.

High-risk occupations

- Corporate executives: Leadership pressures and major decision-making might elevate stress levels, potentially leading to hypertension and sleep disturbances.
- Medical professionals: Regularly dealing with emergencies and patient care are more likely to result in sleep disturbances and elevated blood pressure.
- Emergency responders: Traumatic events and high-adrenaline work scenarios can impact sleep quality and cardiovascular health.
- Customer service representatives: Regularly handling complaints and high client demands can induce significant stress, potentially affecting sleep quality and cardiovascular health.

Self-stress management

Mindfulness meditation, deep breathing exercises, physical activities, etc., are all effective ways to alleviate occupational stress.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS1801131	MTHFR	GT	may have a higher risk of hypertension after exposure to occupational stress

RS1801133	MTHFR	GG	may have a lower risk of hypertension after exposure to occupational stress
RS1805193	SELE	CC	may have a lower risk of hypertension after exposure to occupational stress
RS2304672	PER2	GG	may have a higher risk of sleep disturbances after exposure to occupational stress

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of occupational stress. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of occupational stress in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with occupational stress exposure relative to those without occupational stress exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Occupational Stress test report uses 4 polymorphic loci on *PER2*, *SELE* and *MTHFR* genes. The results showed whether the user had a higher exposure risk of occupational stress than all users in the genotype combination of the 4 polymorphic loci tested, and that the test did not represent the user's actual outcome following occupational stress exposure. The basis of the test is mainly from the research of Chinese.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of occupational stress.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of occupational stress at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of occupational stress, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 4 polymorphic loci related to exposure risk of occupational stress, i.e., rs2304672, rs1805193, rs1801131 and rs1801133.

Population samples

The basis of the test is mainly from the research of Chinese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of occupational stress.

Metal Dust

They are tiny particles produced by grinding, welding or polishing of metals.
Inhalation of metal dust may cause respiratory symptoms, such as cough.

My Metal Dust exposure

risk is slightly low

Compared with the population average, the risk of cough after exposure to metal dust is slightly low.

Suggestions

1. Personal protection: Workers should wear suitable dust masks or respirators when operating.
2. Ensure ventilation: Ensure adequate ventilation in areas where metal dust is generated.
3. Wet processing: Use wet methods for cutting, grinding, and polishing to reduce dust generation.
4. Risk education: Train on the risks of inhaling metal dust and protective measures.
5. Regular cleaning: Regularly clean areas exposed to metal dust to reduce dust accumulation.

Knowledge



Metal dust

Metal dust can be fine metallic powders or tiny particles generated during processes like machining, grinding, cutting, welding, etc. The production of metal dust typically occurs in metalworking, manufacturing, construction, and other industrial activities.



Cough

A cough is a natural defense reaction of the body aimed at clearing mucus or irritants from the airways to maintain clear breathing passages. It can be triggered by various causes, including infections, irritants, diseases, etc. When coughing becomes persistent or chronic, it is crucial to consult a doctor for an accurate diagnosis and treatment recommendations.



Metal dust and cough

Inhaling metal dust can irritate the respiratory tract, leading to persistent coughing. Prolonged exposure to metal dust can cause severe respiratory diseases, with coughing being a common

warning symptom.

 High-risk occupations

- Welders: Exposed to metal fumes and dust during welding operations.
- Machinists: Metal cutting, grinding, and polishing operations pose exposure risks.
- Metal casters: Exposure occurs during the metal casting process.
- Construction workers: Potential exposure when working with metal materials.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS7588395	intergenic	AA	may have a lower risk of cough after exposure to metal dust

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of metal dust. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of metal dust in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with metal dust exposure relative to those without metal dust exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

 Applicable situation

The Metal Dust test report uses 1 polymorphic locus within intergenic regions. The results showed whether the user had a higher exposure risk of metal dust than all users in the genotype of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following metal dust exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of metal dust.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of metal dust at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of metal dust, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs7588395, related to exposure risk of metal dust.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of metal dust.

Dietary Sodium

Sodium plays a key role in the regulation of human blood pressure. Sodium intake mainly comes from salt, various food materials and processed food. Long-term high-salt diet may increase the risk of cardiovascular disease, especially hypertension.

My Dietary Sodium exposure

risk is slightly low

Compared with the population average, the risk of elevated systolic blood pressure after exposure to dietary sodium is slightly low.

Suggestions

1. Adjust dietary habits: Prioritize fresh, unprocessed foods; check sodium content when buying packaged and processed foods, and avoid High-sodium foods; use salt-restricted spoons to control total salt use; during cooking, incorporate salt-free condiments or ingredients like ginger, green onions, garlic, chili, shiitake mushrooms, lemon, crown daisy, celery to enhance the flavor of dishes.
2. Increase physical activity: Regular exercise can help counteract the negative effects of high sodium and reduce blood pressure.
3. Avoid alcohol: Limiting alcohol intake can help reduce the negative impacts of a high-sodium diet and increased blood pressure on cardiovascular health.

Influencing factors



Controllable factors

1. Dietary habits: Habits like a high-sodium diet, not paying attention to food ingredient labels can lead to excessive sodium intake.
2. Lifestyle: Sedentary behavior can have a negative effect on sodium metabolism and blood pressure.
3. Alcohol consumption: Excessive alcohol consumption can disrupt the kidney's sodium filtering mechanism, leading to increased blood pressure.



Uncontrollable factors

1. Genetics: Some individuals are sensitive to the sodium in table salt. When sodium intake is high, their risk of developing hypertension increases.
2. Age: As age increases, the kidneys may reduce their efficiency in eliminating sodium, which can lead to increased blood pressure.
3. Race: Groups such as African Americans are more likely to be affected by sodium's impact on blood pressure.

Knowledge



Role of sodium

- Sodium is vital for fluid balance, nerve impulse transmission, and muscle contraction. However, excessive sodium can lead to fluid retention and increased blood pressure.
- For adults, the World Health Organization (WHO) recommends consuming less than 2000 mg of sodium per day (equivalent to less than 5 grams of table salt, or less than a teaspoon).



Prevalence

- Due to the popularity of processed foods, many people's daily diets contain more sodium than the recommended intake. According to the WHO, about 30% of the global population exceeds the recommended daily salt intake limit.
- Survey on dietary nutrients intake of Chinese residents between 2010 and 2012 showed that the dietary sodium intake of adults was 5013 mg per day, with over 80% of Chinese residents exceeding the recommended intake level.
- Hypertension is the primary risk factor for cardiovascular disease and premature death, with up to one-third of adults worldwide suffering from hypertension. High-sodium diets have a significant impact on the global hypertension prevalence rate.



Typical symptoms of hypertension

Most people with hypertension do not show noticeable symptoms in the early stages. However, if blood pressure is not controlled and remains at a relatively high level for a long time, patients may experience symptoms such as dizziness, headaches, fatigue, and palpitations.



Intervention methods for hypertension

- Dietary intervention: A low-sodium diet, rich in fruits, vegetables, and whole grains helps control sodium intake and reduce the risk of hypertension.
- Exercise intervention: Regular exercise helps lower blood pressure and manage weight, further reducing the risk of hypertension.
- Medication: Blood pressure medications can be combined with lifestyle interventions to effectively control blood pressure.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS8022678	intergenic	GG	may have a lower risk of elevated systolic blood pressure after exposure to dietary sodium

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of dietary sodium, such as allergic reaction. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of allergic reactions to dietary sodium in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with allergic reactions to dietary sodium relative to those without allergic reactions to dietary sodium.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Dietary Sodium test report uses 1 polymorphic locus within intergenic regions. The results showed whether the user had a higher exposure risk of dietary sodium than all users in the genotype of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following dietary sodium exposure. The basis of the test is mainly from the research of Japanese.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of dietary sodium.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of dietary sodium at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of dietary sodium, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs8022678, related to exposure risk of dietary sodium.

Population samples

The basis of the test is mainly from the research of Japanese.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of dietary sodium.

Non-aromatic Solvents

It is a kind of organic solvent without aromatic ring structure, which is widely used in cleaning, degreasing and as a solvent for paints and coatings. Exposure to these solvents may cause respiratory symptoms, such as excessive sputum production.

My Non-aromatic Solvents exposure

risk is moderate

People with the rs7758025-CT genotype may have a moderate risk of phlegm after exposure to non-aromatic solvents.

Suggestions

1. Personal protection: Wear appropriate masks, gloves, and protective equipment when handling non-aromatic solvents.
2. Ensure ventilation: Make sure areas where solvents are used or stored are properly ventilated.
3. Risk education: Understand the potential risks and protection methods associated with non-aromatic solvents.
4. Safe storage: Store the solvents in well-sealed containers, in a cool and dry place, away from heat sources or open flames.

Knowledge



Non-aromatic solvents

Non-aromatic solvents are organic solvents that lack aromatic ring structures and include alcohols, esters, ethers, and ketones. These solvents have a wide range of industrial and chemical applications, including in paints, cleaners, adhesives, coatings, and as solvents in chemical reactions.



Phlegm production

Phlegm is a viscous fluid secreted by the mucous membranes of the respiratory tract. Its primary function is to protect the respiratory tract by capturing and clearing foreign objects, bacteria, viruses, and other harmful substances. While phlegm production is usually a part of a normal physiological process, noticeable changes in its color, texture, or quantity might indicate potential health issues,

such as infections, irritants, diseases, etc. Consultation with a doctor for accurate diagnosis and treatment advice is recommended when such changes occur.

 Non-aromatic solvents and phlegm production

Exposure to non-aromatic solvents can irritate the respiratory tract, leading to increased mucus production and inflammation. Prolonged exposure can intensify this reaction, possibly resulting in chronic bronchitis or other respiratory diseases.

 High-risk occupations

- Painters: May use non-aromatic solvents in paints and thinners.
- Mechanical workers: Might use non-aromatic solvents for cleaning mechanical parts.
- Printers: Could be exposed to non-aromatic solvents in printing inks.
- Chemical plant workers: Have opportunities to come in contact with non-aromatic solvents during production processes.

My genetic result details

Gene locus	Gene name	My genotype	Description
RS7758025	ARHGAP18	CT	may have a moderate risk of phlegm after exposure to non-aromatic solvents

Risk calculation

The risk value of this test is calculated based on the published literature evidence related to exposure risk of non-aromatic solvents. Genetic polymorphism(s) related to the test is identified by studying the risk genotype differences of specific effects brought by the presence/absence of non-aromatic solvents in the population. We comprehensively scored the polymorphic loci involved in this test through the original data in published literature evidence, and obtained the risk value of all users. By comparing with the average risk value of all users detected at We, we can give the risk changes of users with non-aromatic solvents exposure relative to those without non-aromatic solvents exposure.

It should be noted that the risk calculation of this test only takes into account the relevant genetic polymorphism(s) with respect to the test, and the risks of other unknown genetic factors and non-genetic factors are not included.

Test details

Applicable situation

The Non-aromatic Solvents test report uses 1 polymorphic locus on *ARHGAP18* gene. The results showed whether the user had a higher exposure risk of non-aromatic solvents than all users in the genotype of the 1 polymorphic locus tested, and that the test did not represent the user's actual outcome following non-aromatic solvents exposure. The basis of the test is mainly from the research of Caucasian.

Notification

1. The test may not cover all genes or loci that could contribute to exposure risk of non-aromatic solvents.
2. The test results have no clinical significance.
3. If you have a clinical need, contact a qualified institution or doctor.

How to use the test results

Notification

1. The test is not suitable for clinical diagnosis.
2. The occurrence and development of disease is determined by a combination of genetic and environmental factors. This report only predicts your exposure risk of non-aromatic solvents at a genetic level and does not represent your true health status or your other health conditions, lifestyle, etc.
3. If you feel you have some exposure risk of non-aromatic solvents, it is recommended that you consult an authoritative health professional or clinician for help.

Test content

The test includes 1 polymorphic locus, i.e., rs7758025, related to exposure risk of non-aromatic solvents.

Population samples

The basis of the test is mainly from the research of Caucasian.

Limit of detection

Limited by the current technology and the level of scientific cognition, the test may not cover all genes or sites associated with exposure risk of non-aromatic solvents.