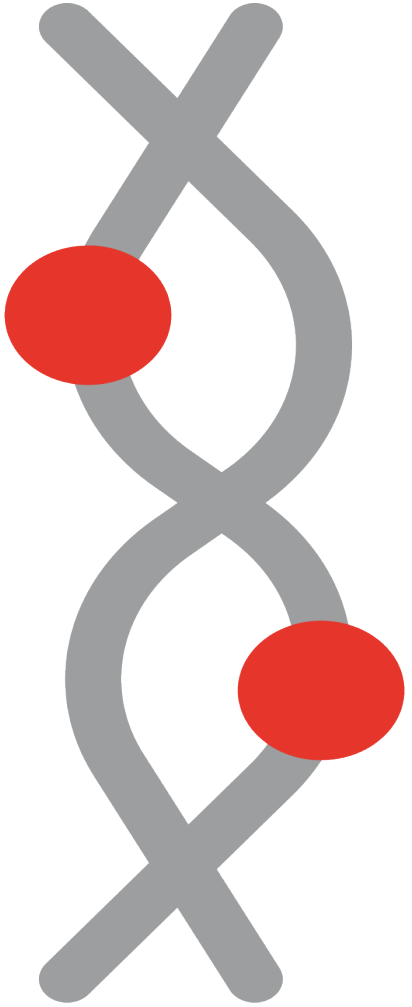
Fagron genomics



Gene Comprehensive Nutrigenomic Report

Accession Number: ##########
Specimen Collected: ##/##/####
Specimen Received: ##/##/####
Report Generated: November 30, 2022
Specimen Type: Buccal Swab
Provider: ######## ########
Patient Name: ###### ######
Patient DOB: ##/##/####
Patient Gender: Female

Do not make any decisions about your health solely based on the information contained in this report. Always consult with a licensed and experienced health practitioner when you receive this report.

GX Sciences | 844-258-5564 | www.GXSciences.com
Lab | 4150 Freidrich Lane, Ste H | Austin, TX 78744
Laboratory Director: James W. Jacobson, Ph.D

– 28 – Female

(-/-) No clinical abnormality | (+/-) Heterozygous result | (+/+) Homozygous result

rsID	Gene	Genetic Result	Therapeutics Associated With Positive Result	Highly Recommended Therapeutics	Provider Discretion: As Needed Formula Recommendations	Lifestyle Recommendations	Laboratory Recommendations
Best Diets for Weight Loss							
rs1799883	FABP2 A54T	-/-	Increased Absorption of Fatty Acids from Foods	**Low Fat / High Protein Diet Should Work Well for You** (2.5 times Expected Weight Loss with Low Fat Diet)			
rs1801282	PPARG	+/-	Polymorphism Causes Increase in Fat Storage and Decrease in Fat Mobilization Polymorphism Causes Increase Cellular Uptake of Glucose and Can Lead to Hypoglycemia				
rs9939609	FTO	+/+	Lower Calorie Intake	**Mediterranean Diet Should Work Well for this Patient** **Paleo Diet Should Work Well for this Patient**			
rs17300539	ADIPOQ	-/-	Need to Limit Saturated Fat and Ingest Low Glycemic Foods				
Fatty Acid Metabolism							
rs5082	APOA2	+/-	Increased Incidence of Obesity			Having Some High Quality Unsaturated Fats in Your Diet Should Not Affect Weight Loss	
rs662799	APOA5	-/-	Increased Risk of Hyperlipidemia				
Satiety Genes							
rs1137101	LEPR	-/-	Decreased Leptin Receptor Response Indicates Decreased Satiety				
rs696217	GHRL	-/-	Increased Hunger Reponse Increased Reward System for Alcohol and Sweets				

rs1800206	PPARA	-/-	Poor Response to Fasting		**"Fasting Mimicking Diet" May Be Beneficial for Weight Loss**	**Intermittent Fasting Should Benefit Weight Loss**	

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rsID	Gene	Genetic Result	Therapeutics Associated With Positive Result	Highly Recommended Therapeutics	Provider Discretion: As Needed Formula Recommendations	Lifestyle Recommendations	Laboratory Recommendations
Intensity of Exercise Needed for Weight Loss							
rs4994	ADRB3	-/-	**Positive Result Indicates Lower Than Expected Weight Loss Potential with Exercise**	**High Intensity Interval Training (More than 30 Mins of Exercise with Heart Rate > 70% of Maximum) Required for Significant Weight Loss**			
rs1042714	ADRB2	+/-					
rs17300539	ADIPOQ	-/-					
Insulin Resistance Risk							
rs510432	ATG5	+/-	Curcumin, Lithium Orotate, D-Chiro-Inositol, Catechins, Resveratrol, Caffeine, 12-15 Hour Fasting				
rs10210302	ATG16L1	-/-					

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rsID	Gene	Genetic Result	Therapeutics Associated With Positive Result	Highly Recommended Therapeutics	Provider Discretion: As Needed Formula Recommendations	Lifestyle Recommendations	Laboratory Recommendations
Lactose Intolerance							
rs4988235	MCM6	+/+	High Incidence of Lactose Intolerance	**You Possess a High Risk of Lactose Intolerance**		Avoid Lactose (Milk Products)	
Caffeine Metabolism							
rs762551	CYP1A2	-/-	Slow Metabolizer of Caffeine				
Chromium and Low Dose Naltrexone Efficacy							
rs1076560	DRD2	+/-	Polymorphism Indicates Better Response Rate to Chromium Picolinate and Low Dose Naltrexone	**Metabolic Stimulator™ or Chromium Picolinate 1-2 Capsules Daily**	Prescription Low Dose Naltrexone (LDN) if Patient is Craving Sugar and High Fat Foods		
Effect from Green Tea Extract / Green Coffee Bean Extract							
rs4680	COMT V158M	+/+	Improved Response to Green Tea and Green Coffee Bean Extracts	**You Should Benefit from Green Tea Extract or Green Coffee Bean Extract for Weight Loss**			
Salt Sensitivity							
rs4343	ACE	-/-	Increased Risk of Salt Retention and Hypertension	**Be Cautious with High Salt Foods**		Recommend Reducing Your Salt Intake	
rs699	AGT	+/+					
Sugar Sensitivity and Mood							
rs1800544	ADRA2A	+/+	Increased Risk of Anti-Psychotic or Anti-Depressant Induced Weight Gain			High Risk of Major Weight Gain with Anti-Psychotic and Anti-Depressant Medications	

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– 28 – Female

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rsID	Gene	Genetic Result	Therapeutics Associated With Positive Result	Highly Recommended Therapeutics	Provider Discretion: As Needed Formula Recommendations	Lifestyle Recommendations	Laboratory Recommendations
Inflammatory Environmental							
rs10156191	AOC1	+/-	Risk of Histamine Food Reaction	**GI Hist Support™ if Histamine Food Response Present**		May Have Difficulty with Histamine Containing Foods	
rs11558538	HNMT	-/-					
rs12995000	HNMT	+/-					
rs492602	FUT2	-/-	Prebiotics and Probiotics Needed				
rs2187668	HLA DQA1	-/-	High Risk of Gluten and Casein Sensitivity Broad Spectrum Enzyme				
rs2858331	HLA DQA2	+/-					

Summary for Diet / Wellness

Highly Recommended Therapeutics

- Low Fat / High Protein Diet Should Work Well for You (2.5 times Expected Weight Loss with Low Fat Diet)
- Mediterranean Diet Should Work Well for this Patient
- Paleo Diet Should Work Well for this Patient
- High Intensity Interval Training (More than 30 Mins of Exercise with Heart Rate > 70% of Maximum) Required for Significant Weight Loss
- You Possess a High Risk of Lactose Intolerance
- Metabolic Stimulator™ or Chromium Picolinate 1-2 Capsules Daily
- You Should Benefit from Green Tea Extract or Green Coffee Bean Extract for Weight Loss
- Be Cautious with High Salt Foods
- GI Hist Support™ if Histamine Food Response Present

Provider Discretion: As Needed Formula Recommendations

- "Fasting Mimicking Diet" May Be Beneficial for Weight Loss
- Prescription Low Dose Naltrexone (LDN) if Patient is Craving Sugar and High Fat Foods

Lifestyle Recommendations

- Having Some High Quality Unsaturated Fats in Your Diet Should Not Affect Weight Loss
- Intermittent Fasting Should Benefit Weight Loss
- Avoid Lactose (Milk Products)
- Recommend Reducing Your Salt Intake
- High Risk of Major Weight Gain with Anti-Psychotic and Anti-Depressant Medications
- May Have Difficulty with Histamine Containing Foods

Laboratory Recommendations

LACTOSE INTOLERANCE

VARIANTS IN THE MCM6 GENE HAS BEEN ASSOCIATED WITH LACTOSE INTOLERANCE

SYMPTOMS AFTER EATING DAIRY PRODUCTS


Diarrhea


Bloating


Occasional vomiting


Gas


Nausea


Stomach cramps

DEFINITION & CAUSES

Lactose:
the sugar found in dairy products,
is not broken down properly

Lactase:
the enzyme that breaks down lactose,
is produced in small amounts

OTHER SOURCES OF CALCIUM


Green leafy vegetables


Milk alternatives
(almond, soy)


Fish
(sardines and canned salmon)


Figs

Whey protein


Calcium
(fortified foods - breakfast
cereals, orange juice)


Seeds
(chia, poppy, sesame, celery)


Beans and lentils


Almonds


Soybean products
(edamame, tofu)

LOW FAT/HIGH PROTEIN DIET

Low Fat / High Protein Diet Definition: This diet plan limits total fat and carbohydrate intake while increasing protein intake to improve satiety and promote healthy weight loss.

FOOD SOURCES

- Egg whites
- Tofu
- Lean cuts of beef, pork (loin, etc.)
- Turkey/chicken
- Fish (cod, tilapia, shrimp, etc.)
- Vegetables (spinach, lettuce, mushrooms, broccoli)
- Fruits (oranges, apples, melons, bananas)
- Grains (quinoa)

BENEFITS:

- Improved blood lipids
- Reductions in body weight & triglycerides
- Limits risk of developing cardiovasular disease

AVOID

- High Fat Meats
- Whole Milk
- Nuts/Oils
- Limit Grains

DIETARY TIPS - *Foods to Eat*

- **Proteins:** Turkey breast (skinless), Chicken (skinless), Fish (cooked or dried): cod, halibut, haddock, flounder, albacore tuna, tilapia, pollock, Egg whites, Shrimp (fresh, frozen), Lite tofu, Lean cuts of beef or pork (look for "loin" or "round")
- **Vegetables:** Broccoli, Spinach, Kale, Arugula, Swiss chard, Lettuce, Potatoes – sweet and regular (preferably unsalted, skinned), Legumes: beans, peas and lentils, Mushrooms, Artichokes
- **Fruits:** Fresh fruit – most fruits are low fat. High protein fruits include: oranges, melons (cantaloupe, watermelon), strawberries, bananas
- **Grains:** Ancient grains – farro, bulgur, spelt, quinoa

DIETARY TIPS - *Foods to Avoid*

- **Proteins:** Dark chicken meat (drumsticks, thighs), Fatty beef, Egg yolks, Fatty fish – salmon, trout, mackerel, sardines, herring
- **Dairy:** Whole milk, Full-fat dairy products (yogurt, cheese, cream)
- **Vegetables:** Avocado
- **Nuts/Oils:** Extra virgin olive oil, Chia seeds, Nuts – almonds, walnuts, macadamia, etc, Coconut oil
- **Grains:** Bread, Cereals, Rice, Pasta

MEDITERRANEAN DIET

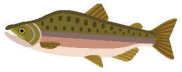
FOOD SOURCES


Olive Oil


Eggs


Nuts/Seeds


Poultry


Salmon


Fruits


Beans


Vegetables

Mediterranean Diet Definition: A diet traditional in Mediterranean countries, characterized especially by a high consumption of vegetables and olive oil and moderate consumption of protein, and thought to confer health benefits.

BENEFITS:

- Positive effects on glycemic control
- Linked to a reduced chance of degenerative diseases
- May inhibit inflammation
- Reduces chance of chronic diseases, such as coronary artery disease, rheumatoid arthritis, hypertension, and even cancer

LIMITED INTAKE

- Dairy Products
- Red Meats

DIETARY TIPS - *Foods to Eat*

- **Eat more fruits and vegetables.** Aim for 7 to 10 servings a day of fruit and vegetables.
- **Opt for whole grains.** Switch to whole-grain bread, cereal and pasta. Experiment with other whole grains, such as bulgur and farro.
- **Use healthy fats.** Try olive oil as a replacement for butter when cooking. Instead of putting butter or margarine on bread, try dipping it in flavored olive oil.
- **Eat more seafood.** Eat fish twice a week. Fresh or water-packed tuna, salmon, trout, mackerel and herring are healthy choices. Grilled fish tastes good and requires little cleanup. Avoid deep-fried fish.
- **Reduce red meat.** Substitute fish, poultry or beans for meat. If you eat meat, make sure it's lean and keep portions small.
- **Enjoy some dairy.** Eat low-fat Greek or plain yogurt and small amounts of a variety of cheeses.
- **Spice it up.** Herbs and spices boost flavor and lessen the need for salt.

DIETARY TIPS - *Foods to Avoid*

- **Added sugar.** Soda, candies, ice cream, table sugar and many others.
- **Refined grains.** White bread, pasta made with refined wheat, etc.
- **Trans fats.** Found in margarine and various processed foods.
- **Refined oils.** Soybean oil, canola oil, cottonseed oil and others.
- **Processed meat.** Processed sausages, hot dogs, etc.
- **Highly processed foods.** Anything labeled "low-fat" or "diet" or which looks like it was made in a factory.

PALEO DIET

Paleo Diet Definition: A diet based on the types of foods presumed to have been eaten by early humans, consisting chiefly of meat, fish, vegetables, and fruit, and excluding dairy, grains and processed foods.

FOOD SOURCES


Fruits


Vegetables


Nuts/Seeds


Lean meats (Steak, etc.)


Fish (Salmon, Mackerel or Tuna)


Olive Oil / Walnut Oil

BENEFITS:

- Lower triglyceride levels & diastolic pressure
- Lower BMI & HbA1c
- Improved glucose control & lipid profiles in people with Type 2 Diabetes

AVOID

- Grains, such as wheat, oats, and barley
- Legumes, such as beans, lentils, peanuts, and peas
- Dairy products
- Refined sugar
- Salt
- Potatoes
- Highly processed foods

DIETARY TIPS - *Example Daily Menu*

- **Breakfast.** Broiled salmon and cantaloupe.
- **Lunch.** Broiled lean pork loin and salad (romaine, carrot, cucumber, tomatoes, walnuts and lemon juice dressing).
- **Dinner.** Lean beef sirloin tip roast, steamed broccoli, salad (mixed greens, tomatoes, avocado, onions, almonds and lemon juice dressing), and strawberries for dessert.
- **Snacks.** An orange, carrot sticks or celery sticks.

DIETARY TIPS - *Foods to Eat*

- **Meat.** Lean cuts of beef, pork, and poultry, preferably grass-fed, organic, or free-range selections
- **Game animals.** Quail, venison, and bison
- **Eggs.** No more than six a week, and preferably free-range
- **Fish.** Including shellfish
- **Fruit.** Strawberries, cantaloupe, mango, and figs
- **Nonstarchy vegetables,** such as asparagus, onions, peppers, and pumpkin
- **Nuts and seeds,** including almonds, cashews, walnuts, and pumpkin seeds
- **Olive oil,** flaxseed oil, and walnut oil, in moderation

DIETARY TIPS - *Foods to Avoid*

- **Dairy products.** Milk, cheese, yogurt, and butter
- **Cereal grains.** Wheat, rye, rice, and barley
- **Legumes.** Beans, peanuts, and peas
- **Starchy vegetables.** Potatoes, Sweet Potatoes
- **Sweets.** All forms of candy as well as honey and sugar
- **Artificial sweeteners.**
- **Sugary soft drinks and fruit juices.**
- **Processed and cured meats.** Bacon, deli meats, and hot dogs
- **Highly processed foods.**

Gene Information Key

rsID	Gene	"-" variant	"+" variant
rs4343	ACE	A	G
rs17300539	ADIPOQ	G	A
rs1800544	ADRA2A	G	C
rs1042714	ADRB2	C	G
rs4994	ADRB3	A	G
rs699	AGT	A	G
rs10156191	AOC1	C	T
rs5082	APOA2	A	G
rs662799	APOA5	A	G
rs10210302	ATG16L1	C	T
rs510432	ATG5	C	T
rs4680	COMT V158M	G	A
rs762551	CYP1A2	A	C
rs1076560	DRD2	C	A
rs1799883	FABP2 A54T	C	T
rs9939609	FTO	T	A
rs492602	FUT2	A	G
rs696217	GHRL	G	T
rs2187668	HLA-DQA1	C	T
rs2858331	HLA-DQA2	A	G
rs11558538	HNMT	C	T
rs12995000	HNMT	C	T
rs1137101	LEPR	A	G
rs4988235	MCM6	A	G
rs1800206	PPARA	C	G
rs1801282	PPARG	C	G

Definitions

GASTROINTESTINAL	
MCM6	A mutation in a DNA control region located in the MCM6 gene is associated with expression of the lactase gene. Individuals homozygous for this polymorphism are more likely to have hypolactasia, or lactose intolerance.
General	
ADIPOQ	This gene is expressed in adipose tissue exclusively and encodes for the protein adiponectin. Adiponectin is involved with metabolic and hormonal processes. Mutations in this gene are associated with adiponectin deficiency.
ADRB2	The protein encoded by this gene belongs to the family of beta adrenergic receptors that mediate catecholamine sensitivity. This receptor is located mainly in the adipose tissue and is involved in the regulation of lipolysis and thermogenesis.
APOA2	This gene encodes apolipoprotein (apo-) A-II, which is the second most abundant protein of the high density lipoprotein particles. Defects in this gene may result in apolipoprotein A-II deficiency or hypercholesterolemia.
APOA5	The protein encoded by this gene is an apolipoprotein that plays an important role in regulating the plasma triglyceride levels, a major risk factor for coronary artery disease. It is a component of high density lipoprotein. Mutations in this gene have been associated with hypertriglyceridemia and hyperlipoproteinemia type 5.
CYP1A2	This gene encodes a member of the cytochrome P450 superfamily of enzymes typically found in the liver. These enzymes catalyze many reactions involved in drug metabolism and synthesis of cholesterol, steroids, lipids, and caffeine.
FABP2 A54T	The protein encoded by this gene is an intracellular fatty acid-binding protein that participates in the uptake, intracellular metabolism, and transport of long-chain fatty acids. The encoded protein is also involved in the modulation of cell growth and proliferation. This protein binds saturated long-chain fatty acids with high affinity, and acts as a lipid sensor to maintain energy homeostasis.
FTO	This gene codes for a nuclear protein non-haem iron and 2-oxoglutarate-dependent oxygenase superfamily. This enzyme functions to reverse alkylated DNA and RNA damage by oxidative demethylation. Studies indicate a strong association with body mass index, obesity risk, and type 2 diabetes.
GHRL	This gene encodes the ghrelin-obestatin preproprotein that is cleaved to yield two peptides, ghrelin and obestatin. Ghrelin is a powerful appetite stimulant and plays an important role in energy homeostasis. Its secretion is initiated when the stomach is empty, whereupon it binds to the growth hormone secretagogue receptor in the hypothalamus which results in the secretion of growth hormone (somatotropin). Ghrelin is thought to regulate multiple activities, including hunger, reward perception, gastric acid secretion, gastrointestinal motility, and pancreatic glucose-stimulated insulin secretion.
LEPR	This gene codes for the Leptin Receptor which is associated with the cytosolic STAT proteins. This receptor for leptin (an adipocyte-specific hormone that regulates body weight) is involved in the regulation of fat metabolism with mutations in this gene have been associated with obesity.
PPARA	The peroxisome proliferators induce the production of intracellular peroxisomes that contain enzymes for respiration and for cholesterol and lipid metabolism. The action of peroxisome proliferators is mediated via specific receptors, called PPARs, which belong to the steroid hormone receptor superfamily. PPARs affect the expression of target genes involved in cell proliferation, cell differentiation and in immune and inflammation responses. This gene encodes the subtype PPAR-alpha, which is a nuclear transcription factor.
PPARG	This gene encodes a nuclear factor called peroxisome proliferator-activated receptor (PPAR). PPARs form heterodimers with retinoid X receptors (RXRs) and these heterodimers regulate transcription of various genes. The protein encoded by this gene is PPAR-gamma and is a regulator of adipocyte differentiation.
HYPERTENSION	The polymorphisms in this category will increase the risk of developing hypertension.
ACE	Angiotensin-converting enzyme (ACE) is an important target for therapeutic drugs treating hypertension and heart failure. The best studied single nucleotide polymorphism in the ACE gene (rs4343) has been linked to a wide variety of human phenotypes: nephropathy and renal disease, cancer, and even sports performance. Interestingly, rs4343 is a member of a large family of human mutations called Alu elements.
AGT	The AGT gene codes for the angiotensinogen protein, a key regulator of blood pressure and body fluid homeostasis. Individuals carrying two copies of the rs699 C allele are at increased risk of hypertension-related disorders such as pre-eclampsia.
INFLAMMATORY	This Enzyme category has significant effects on the inflammatory state of a person's body. Polymorphisms in these specific enzymes will significantly increase the levels of inflammation in the body. By supplementing these enzyme deficiencies, the patient will effectively reduce inflammatory damage to the body.
AOC1	The SNP rs10156191 encodes a weaker form of the histamine degradation enzyme Amine Oxidase, Copper Containing 1 (AOC1). This mutation, Thr16Met, is predicted to produce an enzyme with less catalytic activity and associated higher levels of pro-inflammatory amines like histamine and putrescine.

ATG16L1 rs10210302	The ATG16L1 gene encodes a protein that is a vital component of a protein complex necessary for the cellular phenomena known as autophagy. Autophagy is the process of degrading and cleaning of inert debris of the cell. Weakness in autophagy leads to abnormal accumulation of cellular “garbage” that will eventually affect the cellular function and lead to autophagy-related disease states in including many neurological and immunological diseases, DM Type 2 and fatty liver disease.
ATG5	Autophagy-related 5 protein (ATG5) is an important intracellular mediator of the autophagy response. ATG5 is involved in a wide range of "quality control" features inside the cell: autophagy vesicle formation, innate immune system signaling, consumption of damaged mitochondria, and apoptosis. Mutations in the ATG5 gene are associated with numerous neurological, immunological and endocrine syndromes.
DRD2	Dopamine receptor D2 is an important component of the neuroinflammation process. Activation of DRD2 signaling is thought to decrease TNFalpha release from inflammatory mast cells. Polymorphisms associated with decreased DRD2 signaling activity are predicted to lead to pro-inflammatory phenotypes.
FUT2	Fucosyltransferase 2 (FUT2) is responsible for producing specific sugar groups that are secreted by the intestinal cells into the bowel to attract "good bacteria" . Polymorphisms in this gene produce "poor secreter" status. Lack of these sugars allows for gut dysbiosis and a higher risk of inflammatory bowel disease.
HLA-DQA1	Major histocompatibility complex, DQ alpha 1 (HLA-DQA1) is a human gene responsible for a cell surface receptor essential to the function of the immune system. Patients with a polymorphism in this gene are at higher risk for auto-immune based inflammatory disease including Celiac disease, Crohn's, Ulcerative Colitis, and gluten sensitivity.
HLA-DQA2	Major histocompatibility complex, DQ alpha 2 (HLA-DQA2) is a human gene responsible for a cell surface receptor essential to the function of the immune system. Patients with a polymorphism in this gene are at higher risk for auto-immune based inflammatory disease including Celiac disease, Crohn's, Ulcerative Colitis, and gluten sensitivity.
HNMT rs12995000	The HNMT gene encodes the histamine degradative enzyme, histamine N-methyltransferase. HNMT, in contrast to AOC1, requires the methyl donor S-adenosylmethionine and a complete methylation pathway for normal function. Polymorphisms in HNMT gene expression or protein-coding are predicted to prolong the pro-inflammatory effects of histamine signaling.
HNMT Thr105Ile	The HNMT gene encodes the histamine degradative enzyme, histamine N-methyltransferase. HNMT, in contrast to AOC1, requires the methyl donor S-adenosylmethionine and a complete methylation pathway for normal function. Polymorphisms in HNMT gene expression or protein coding are predicted to prolong the pro-inflammatory effects of histamine signaling.
NEUROTRANSMITTER	Neurotransmitters are chemicals that are used to produce specific effects in the nervous system. These specific neurotransmitter genomics assess a person's risk for anxiety, depression and dysphoria.
ADRA2A	ADRA2A (Adrenergic Receptor Alpha 2A) gene that determines sensitivity of the adrenergic nervous system response. Individuals with the G allele at this location predicted to be at higher risk of sugar-induced hyperactivity, and better response to ADHD treatment with typical pharmacological interventions.
COMT V158M	Catechol-O-methyltransferase (COMT) is one of several enzymes that degrade catecholamine neurotransmitters such as dopamine, epinephrine, and norepinephrine. COMT's main function is to inactivate neurotransmitters (dopamine, epinephrine, and norepinephrine) by the addition of a methyl group to the catecholamine. Normal COMT function allows people to rapidly reverse feelings of anxiety or depression. COMT (+/-) patients have sluggish ability to alter anxiety or depression episodes. COMT (+/+) patients are more prone to prolonged episodes of anxiety, depression and OCD.

Disclaimers

TESTING:

Testing Performed By: AMH

METHODOLOGY AND LIMITATIONS:

Testing for genetic variation/mutation on listed genes was performed using ProFlex PCR and Real-Time PCR with TaqMan® allele-specific probes on the QuantStudio 12K Flex. All genetic testing is performed by GX Sciences, 4150 Freidrich Lane, Ste H, Austin, TX. 78744. This test will not detect all the known alleles that result in altered or inactive tested genes. This test does not account for all individual variations in the individual tested. Test results do not rule out the possibility that this individual could be a carrier of other mutations/variations not detected by this gene mutation/variation panel. Rare mutations surrounding these alleles may also affect our detection of genetic variations. Thus, the interpretation is given as a probability. Therefore, this genetic information shall be interpreted in conjunction with other clinical findings and familial history for the administration of specific nutrients. Patients should receive appropriate genetic counseling to explain the implications of these test results. Details of assay performance and algorithms leading to clinical recommendations are available upon request. The analytical and performance characteristics of this laboratory developed test (LDT) were determined by GX Sciences' laboratory pursuant to Clinical Laboratory Improvement Amendments (CLIA) requirements.
CLIA #: 45D2144988 Laboratory Director: James Jacobson, PhD

DISCLAIMER:

This test was developed and its performance characteristics determined by GX Sciences. It has not been cleared or approved by the FDA. The laboratory is regulated under CLIA and qualified to perform high-complexity testing. This test is used for clinical purposes. It should not be regarded as investigational or for research. rsIDs for the alleles being tested were obtained from the dbSNP database (Build 142).

DISCLAIMER:

UND Result: If you have received the result Variant undetermined (UND) this indicates that we were not able to determine your carrier status based on your raw data. Please refer to the GX Sciences genetic knowledge database for more information: https://www.gxsciences.com/kb_results.asp

DISCLAIMER:

Report contents and report recommendations are created and approved by GX Sciences. Sole responsibility for the proper use of the information on the GX Sciences report rests with the user, or those professionals with whom the user may consult. Nutrigenomic Testing and Dietary Supplements are not "Designated Health Services" covered by Medicare or Medicaid and may not be reimbursed under any state or Federal health care program.

DISCLAIMER:

These products are not approved by the Food and Drug Administration and are not intended to diagnose, treat, cure or prevent disease. These recommendations are for report purposes only and an individual is not required to use such products. These are recommendations only and do not replace the advisement of your own healthcare practitioner.

GX Sciences SNP References

GASTROINTESTINAL SNP References

MCM6

• Lactose intolerance. Mayo Clinic (2020). Available at: https://www.mayoclinic.org/diseases-conditions/lactose-intolerance/symptoms-causes/syc-20374232. • Enattah, N. S. et al. Identification of a variant associated with adult-type hypolactasia. Nat. Genet. (2002). doi:10.1038/ng826 • Jennings, K.-A. Top 15 Calcium-Rich Foods (Many Are Non-Dairy). Healthline (2018). Available at: https://www.healthline.com/nutrition/15-calcium-rich-foods. • Publishing, H. H. What are the best calcium sources for people who are lactose intolerant? Harvard Health (2017). Available at: https://www.health.harvard.edu/diseases-and-conditions/what-are-the-best-calcium-sources-for-people-who-are-lactose-intolerant. • Bersaglieri, T. et al. Genetic Signatures of Strong Recent Positive Selection at the Lactase Gene. Am. J. Hum. Genet. (2004). doi:10.1086/421051

General SNP References

ADIPOQ

• Prior, S. L., Jones, D. A., Gill, G. V., Bain, S. C. & Stephens, J. W. Association of the adiponectin rs266729 C>G variant and coronary heart disease in the low risk 'Golden Years' type 1 diabetes cohort. Diabetes Res. Clin. Pract. (2011). doi:10.1016/j.diabres.2010.12.007 • Morandi, A. et al. Early detrimental metabolic outcomes of rs17300539-A Allele of ADIPOQ gene despite higher adiponectinemia. Obesity (2010). doi:10.1038/oby.2009.403 • Lu, J. fu et al. Association of ADIPOQ polymorphisms with obesity risk: A meta-analysis. Hum. Immunol. (2014). doi:10.1016/j.humimm.2014.09.004

ADRB2

• Apalasamy, Y. D., Ming, M. F., Rampal, S., Bulgiba, A. & Mohamed, Z. Gender-dependent association of a ?2-adrenergic gene variant with obesity parameters in Malaysian Malays. Asia-Pacific J. Public Heal. (2015). doi:10.1177/1010539511430250 • Jalba, M. S., Rhoads, G. G. & Demissie, K. Association of codon 16 and codon 27 ?2-adrenergic receptor gene polymorphisms with obesity: A meta-analysis. Obesity (2008). doi:10.1038/oby.2008.327 • Wang, D. W. et al. ADRB2 polymorphisms predict the risk of myocardial infarction and coronary artery disease. Genet. Mol. Biol. (2015). doi:10.1590/S1415-475738420140234

APOA2

• Lai, C. Q. et al. Epigenomics and metabolomics reveal the mechanism of the APOA2 -saturated fat intake interaction affecting obesity. Am. J. Clin. Nutr. (2018). doi:10.1093/ajcn/nqy081 • S., N., M., N. & C., S. Potential genetic association of APOA2, FTO, FADS1, LIPC, and LPL with body mass index measurements among the general population. Pharmacotherapy (2017). • Duesing, K. et al. Evaluating the association of common APOA2 variants with type 2 diabetes. BMC Med. Genet. (2009). doi:10.1186/1471-2350-10-13

APOA5

• Wang, Y. et al. The APOA5 rs662799 polymorphism is associated with dyslipidemia and the severity of coronary heart disease in Chinese women. Lipids Health Dis. (2016). doi:10.1186/s12944-016-0343-z • Hsu, M. C. et al. Central obesity in males affected by a dyslipidemia-associated genetic polymorphism on APOA1/C3/A4/A5 gene cluster. Nutr. Diabetes (2013). doi:10.1038/nutd.2013.2 • Ye, H. et al. Positive association between APOA5 rs662799 polymorphism and coronary heart disease: A case-control study and meta-analysis. PLoS One (2015). doi:10.1371/journal.pone.0135683

CYP1A2

• Guessous, I. et al. Caffeine intake and CYP1A2 variants associated with high caffeine intake protect non-smokers from hypertension. Hum. Mol. Genet. (2012). doi:10.1093/hmg/dds137 • Wang, H. et al. CYP1A2 rs762551 polymorphism contributes to cancer susceptibility: A meta-analysis from 19 case-control studies. BMC Cancer (2012). doi:10.1186/1471-2407-12-528 • Stasiukonyte, N., Liutkeviciene, R., Vilkeviciute, A., Banevicius, M. & Kriauciuniene, L. Associations between Rs4244285 and Rs762551 gene polymorphisms and age-related macular degeneration. Ophthalmic Genet. (2017). doi:10.1080/13816810.2016.1242018

FABP2 A54T

• Qiu, C. J., Ye, X. Z., Yu, X. J., Peng, X. R. & Li, T. H. Association between FABP2 Ala54Thr polymorphisms and type 2 diabetes mellitus risk: A HuGE Review and Meta-Analysis. J. Cell. Mol. Med. (2014). doi:10.1111/jcmm.12385 • Hu, X. et al. Gene Polymorphisms of ADIPOQ +45T>G, UCP2 -866G>A, and FABP2 Ala54Thr on the Risk of Colorectal Cancer: A Matched Case-Control Study. PLoS One (2013). doi:10.1371/journal.pone.0067275 • Abbas, S. et al. Association of ACE, FABP2 and GST genes polymorphism with essential hypertension risk among a North Indian population. Ann. Hum. Biol. (2015). doi:10.3109/03014460.2014.968206 • Liu, Y. et al. Association of the FABP2 Ala54Thr polymorphism with type 2 diabetes, obesity, and metabolic syndrome: A population-based case-control study and a systematic meta-analysis. Genet. Mol. Res. (2015). doi:10.4238/2015.February.6.19

FTO

• Tanofsky-Kraff, M. et al. The FTO gene rs9939609 obesity-risk allele and loss of control over eating. Am. J. Clin. Nutr. (2009). doi:10.3945/ajcn.2009.28439 • Velders, F. P. et al. FTO at rs9939609, Food Responsiveness, Emotional Control and Symptoms of ADHD in Preschool Children. PLoS One (2012). doi:10.1371/journal.pone.0049131 • Al-Attar, S. A. et al. Association between the FTO rs9939609 polymorphism and the metabolic syndrome in a non-Caucasian multi-ethnic sample. Cardiovasc. Diabetol. (2008). doi:10.1186/1475-2840-7-5

GHRL

• Imaizumi, T. et al. Effect of dietary energy and polymorphisms in BRAP and GHRL on obesity and metabolic traits. Obes. Res. Clin. Pract. (2018). doi:10.1016/j.orcp.2016.05.004 • Pabalan, N. A., Seim, I., Jarjanazi, H. & Chopin, L. K. Associations between ghrelin and ghrelin receptor polymorphisms and cancer in Caucasian populations: A meta-analysis. BMC Genet. (2014). doi:10.1186/s12863-014-0118-3 • Li, G. et al. The preliminary investigation of orexigenic hormone gene polymorphisms on posttraumatic stress disorder symptoms. Psychoneuroendocrinology (2019). doi:10.1016/j.psyneuen.2018.09.042

PPARA

• Gu, S. J. et al. Effect of obesity on the association between common variations in the PPAR gene and C-reactive protein level in Chinese Han population. Endocrine (2014). doi:10.1007/s12020-014-0218-x • Lianggeng, X., Baiwu, L., Maoshu, B., Jiming, L. & Youshan, L. Impact of Interaction Between PPAR Alpha and PPAR Gamma on Breast Cancer Risk in the Chinese Han Population. Clin. Breast Cancer (2017). doi:10.1016/j.clbc.2016.10.003 • Xie, H. J. et al. Analysis on the association between PPAR?/? polymorphisms and lipoprotein(a) in a Chinese Han population. Mol. Genet. Genomics (2014). doi:10.1007/s00438-014-0866-9

PPARG

• Phani, N. M. et al. Implications of critical PPAR?2, ADIPOQ and FTO gene polymorphisms in type 2 diabetes and obesity-mediated susceptibility to type 2 diabetes in an Indian population. Mol. Genet. Genomics (2016). doi:10.1007/s00438-015-1097-4 • Wu, L., Cui, L., Tam, W. H., Ma, R. C. W. & Wang, C. C. Genetic variants associated with gestational diabetes mellitus: A meta-analysis and subgroup analysis. Sci. Rep. (2016). doi:10.1038/srep30539 • Wang, Y. et al. Peroxisome proliferator-activated receptor gamma (PPARG) rs1801282 C>G polymorphism is associated with cancer susceptibility in asians: An updated meta-analysis. Int. J. Clin. Exp. Med. (2015).

HEALTH PRECAUTIONS SNP References

ACE

• The ACE gene and human performance: 12 years on. Puthucheary Z1, Skipworth JR, Rawal J, Loosemore M, Van Someren K, Montgomery HE. Sports Med. 2011 Jun 1;41(6):433-48. doi: 10.2165/11588720-0. • Structural details on the binding of antihypertensive drugs captopril and enalaprilat to human testicular angiotensin I-converting enzyme. (PMID: 15236580) Natesh R. … Acharya K.R.(Biochemistry 2004) • Angiotensin-converting enzyme inhibition by perindopril in the treatment of cardiovascular disease. (PMID: 19379059) Brugts J.J. … Simoons M.L.(Expert Rev Cardiovasc Ther 2009) • Meta-analysis of genetic studies in ischemic stroke: thirty-two genes involving approximately 18,000 cases and 58,000 controls. (PMID: 15534175) Casas J.P. … Sharma P.(Arch. Neurol. 2004) • Triple pharmacological blockade of the renin-angiotensin-aldosterone system in nondiabetic CKD: an open-label crossover randomized controlled trial. (PMID: 18423812) Tylicki L. … Rutkowski B.(Am. J. Kidney Dis. 2008)

HYPERTENSION SNP References

AGT

• Anton, Raymond F, Gabor Oroszi, Stephanie O'Malley, David Couper, Robert Swift, Helen M Pettinati, David Goldman, et al. 2008. "Pharmacogenomics." Edited by Yoshiaki Tsuji. Nature Genetics 16 (1). Public Library of Science: 268–78. https://doi.org/10.1016/j.ejca.2015.06.122. • Ahluwalia, Tarunveer Singh, Monica Ahuja, Taranjit Singh Rai, Harbir Singh Kohli, Anil Bhansali, Kamal Sud, and Madhu Khullar. 2009. "ACE Variants Interact with the RAS Pathway to Confer Risk and Protection against Type 2 Diabetic Nephropathy." DNA and Cell Biology 28 (3): 141–50. https://doi.org/10.1089/dna.2008.0810.

INFLAMMATORY SNP References

AOC1

• McGrath, A. P. et al. Structure and Inhibition of Human Diamine Oxidase - Biochemistry (ACS Publications). Biochemistry 48, 9810–22 (2009). • Maintz, L. & Novak, N. Histamine and histamine intolerance. Am. J. Clin. Nutr. (2007). doi:10.1093/ajcn/85.5.1185 • McGrath, A. P. et al. Structure and inhibition of human diamine oxidase. Biochemistry 48, 9810–9822 (2009). • Schwelberger, H. G. The origin of mammalian plasma amine oxidases. in Journal of Neural Transmission 114, 757–762 (2007). • Solismaa, A. et al. Histaminergic gene polymorphisms associated with sedation in clozapine-treated patients. Eur. Neuropsychopharmacol. 27, 442–449 (2017).

ATG16L1

• Mizushima, N. Autophagy: Process and function. Genes and Development (2007). doi:10.1101/gad.1599207 • Salem, M., Nielsen, O. H., Nys, K., Yazdanyar, S. & Seidelin, J. B. Impact of T300A Variant of ATG16L1 on antibacterial response, risk of culture positive infections, and clinical course of Crohn's disease. Clin. Transl. Gastroenterol. (2015). doi:10.1038/ctg.2015.47 • Begun, J. et al. Integrated Genomics of Crohn's Disease Risk Variant Identifies a Role for CLEC12A in Antibacterial Autophagy. Cell Rep. (2015). doi:10.1016/j.celrep.2015.05.045 • Cheng, J. F., Ning, Y. J., Zhang, W., Lu, Z. H. & Lin, L. T300A polymorphism of ATG16L1 and susceptibility to inflammatory bowel diseases: A meta-analysis. World J. Gastroenterol. (2010). doi:10.3748/wjg.v16.i10.1258 • Kabat, A. M. et al. The autophagy gene Atg16l1 differentially regulates Treg and TH2 cells to control intestinal inflammation. Elife (2016). doi:10.7554/eLife.12444 • Lassen, K. G. et al. Atg16L1 T300A variant decreases selective autophagy resulting in altered cytokine signaling and decreased antibacterial defense. Proc. Natl. Acad. Sci. (2014). doi:10.1073/pnas.1407001111 • Smith, G. S., Walter, G. L. & Walker, R. M. Clinical Pathology in Non-Clinical Toxicology Testing. in Haschek and Rousseaux's Handbook of Toxicologic Pathology (2013). doi:10.1016/B978-0-12-415759-0.00018-2 • Levine, B. & Kroemer, G. Autophagy in the Pathogenesis of Disease. Cell (2008). doi:10.1016/j.cell.2007.12.018 • Lindberg, S. Autophagy: Definition, Diet, Fasting, Cancer, Benefits, and More. Healthline (2014). Available at: https://www.healthline.com/health/autophagy#bottom-line. • Antunes, F. et al. Autophagy and intermittent fasting: the connection for cancer therapy? Clinics (Sao Paulo, Brazil) (2018). doi:10.6061/clinics/2018/e814s • Takagi, A., Kume, S., Maegawa, H. & Uzu, T. Emerging role of mammalian autophagy in ketogenesis to overcome starvation. Autophagy (2016). doi:10.1080/15548627.2016.1151597 • Gazouli, M. et al. NOD2/CARD15, ATG16L1 and IL23R gene polymorphisms and childhood-onset of Crohn's disease. World J. Gastroenterol. (2010). doi:10.3748/wjg.v16.i14.1753 • Kuballa, P., Huett, A., Rioux, J. D., Daly, M. J. & Xavier, R. J. Impaired autophagy of an intracellular pathogen induced by a Crohn's disease associated ATG16L1 variant. PLoS One (2008). doi:10.1371/journal.pone.0003391 • Rosentul, D. C. et al. Role of autophagy genetic variants for the risk of Candida infections. Med. Mycol. (2014). doi:10.1093/mmy/myt035 • Raju, D., Hussey, S. & Jones, N. L. Crohn disease ATG16L1 polymorphism increases susceptibility to infection with Helicobacter pylori in humans. Autophagy (2012). doi:10.4161/auto.21007 • Stappenbeck, T. S. et al. Crohn disease: A current perspective on genetics, autophagy and immunity. Autophagy (2011). doi:10.4161/auto.7.4.13074 • Csöngei, V. et al. Interaction of the major inflammatory bowel disease susceptibility alleles in Crohn's disease patients. World J. Gastroenterol. (2010). doi:10.3748/wjg.v16.i2.176 • Boada-Romero, E. et al. The T300A Crohn's disease risk polymorphism impairs function of the WD40 domain of ATG16L1. Nat. Commun. (2016). doi:10.1038/ncomms11821 • Glubb, D. M. et al. NOD2 and ATG16L1 polymorphisms affect monocyte responses in crohn's disease. World J. Gastroenterol. (2011). doi:10.3748/wjg.v17.i23.2829 • Salem, M., Ammitzboell, M., Nys, K., Seidelin, J. B. & Nielsen, O. H. ATG16L1: A multifunctional susceptibility factor in crohn disease. Autophagy (2015). doi:10.1080/15548627.2015.1017187 • Usategui-Martín, R. et al. Polymorphisms in autophagy genes are associated with paget disease of bone. PLoS One (2015). doi:10.1371/journal.pone.0128984 • Messer, J. S. et al. The Crohn's disease: Associated ATG16L1 variant and Salmonella invasion. BMJ Open (2013). doi:10.1136/bmjopen-2013-002790

ATG5

• Lindberg, S. Autophagy: Definition, Diet, Fasting, Cancer, Benefits, and More. Healthline (2014). Available at: https://www.healthline.com/health/autophagy#bottom-line. • Takagi, A., Kume, S., Maegawa, H. & Uzu, T. Emerging role of mammalian autophagy in ketogenesis to overcome starvation. Autophagy (2016). doi:10.1080/15548627.2016.1151597 • Antunes, F. et al. Autophagy and intermittent fasting: the connection for cancer therapy? Clinics (Sao Paulo, Brazil) (2018). doi:10.6061/clinics/2018/e814s • Levine, B. & Kroemer, G. Autophagy in the Pathogenesis of Disease. Cell (2008). doi:10.1016/j.cell.2007.12.018 • Smith, G. S., Walter, G. L. & Walker, R. M. Clinical Pathology in Non-Clinical Toxicology Testing. in Haschek and Rousseaux's Handbook of Toxicologic Pathology (2013). doi:10.1016/B978-0-12-415759-0.00018-2 • Mizushima, N. Autophagy: Process and function. Genes and Development (2007). doi:10.1101/gad.1599207 • Anton, R. F. et al. Pharmacogenomics. Nat. Genet. 16, 268–278 (2008). • White, K. A. M. et al. Variants in autophagy-related genes and clinical characteristics in melanoma: a population-based study. Cancer Med. 5, 3336–3345 (2016). • Yuan, J. et al. Polymorphisms in autophagy related genes and the coal workers' pneumoconiosis in a Chinese population. Gene 632, 36–42 (2017). • Martin, L. J. et al. Functional Variant in the Autophagy-Related 5 Gene Promotor is Associated with Childhood Asthma. PLoS One 7, e33454 (2012).

DRD2

• Anton, R. F. et al. Pharmacogenomics. Nat. Genet. (2008). doi:10.1016/j.ejca.2015.06.122 • Disabato, D. J., Quan, N. & Godbout, J. P. Neuroinflammation: the devil is in the details. Journal of Neurochemistry 139, 136–153 (2016). • Clarke, T. K. et al. The dopamine receptor D2 (DRD2) SNP rs1076560 is associated with opioid addiction. Ann. Hum. Genet. (2014). doi:10.1111/ahg.12046 • Sasabe, T., Furukawa, A., Matsusita, S., Higuchi, S. & Ishiura, S. Association analysis of the dopamine receptor D2 (DRD2) SNP rs1076560 in alcoholic patients. Neurosci. Lett. (2007). doi:10.1016/j.neulet.2006.10.064

FUT2

• FUT2 fucosyltransferase 2 (secretor status included) [Bos taurus (cattle)] - Gene - NCBI. Current neurology and neuroscience reports. Available at: https://www.ncbi.nlm.nih.gov/gene/281175. • Kimura, K. et al. Diversification of transcriptional modulation: Large-scale identification and characterization of putative alternative promoters of human genes. Genome Res. (2006). doi:10.1101/gr.4039406 • Strausberg, R. L. et al. Generation and initial analysis of more than 15,000 full-length human and mouse cDNA sequences. Proc. Natl. Acad. Sci. U. S. A. (2002). doi:10.1073/pnas.242603899 • Koda, Y., Soejima, M., Wang, B. & Kimura, H. Structure and expression of the gene encoding secretor-type galactoside 2-alpha-L-fucosyltransferase (FUT2). Eur. J. Biochem. (1997). doi:10.1111/j.1432-1033.1997.t01-1-00750.x • Reguigne-Arnould, I. et al. Relative positions of two clusters of human ?-L-fucosyltransferases in 19q (FUT1–FUT2) and 19p (FUT6–FUT3–FUT5) within the microsatellite genetic map of chromosome 19. Cytogenet. Genome Res. (1995). doi:10.1159/000134098 • BALL, S. P. et al. The human chromosome 19 linkage group FUT1 (H), FUT2 (SE), LE, LU, PEPD, C3, APOC2, D19S7 and D19S9. Ann. Hum. Genet. (1991). doi:10.1111/j.1469-1809.1991.tb00417.x

HLA-DQA1

• Kao, H. T. et al. Molecular analysis of the HLA class II genes in two DRw6-related haplotypes, DRw13 DQw1 and DRw14 DQw3. J. Immunol. (1989). • Todd, J. a, Fukui, Y., Kitagawa, T. & Sasazuki, T. The A3 allele of the HLA-DQA1 locus is associated with susceptibility to type 1 diabetes in Japanese. Proc. Natl. Acad. Sci. U. S. A. (1990). • Marsh, S. G. & Bodmer, J. G. HLA class II nucleotide sequences, 1992. Immunogenetics (1993). • Liu, C. P., Bach, F. H. & Wu, S. K. Molecular studies of a rare DR2/LD-5a/DQw3 HLA class II haplotype. Multiple genetic mechanisms in the generation of polymorphic HLA class II genes. J Immunol (1988). • Histocompatibility complex - Genetics Home Reference - NIH. U.S. National Library of Medicine (2020). Available at: https://ghr.nlm.nih.gov/primer/genefamily/hla. • Schmidt, H., Williamson, D. & Ashley-Koch, A. HLA-DR15 haplotype and multiple sclerosis: A HuGE review. American Journal of Epidemiology (2007). doi:10.1093/aje/kwk118 • Horn, G. T., Bugawan, T. L., Long, C. M., Manos, M. M. & Erlich, H. A. Sequence analysis of HLA class II genes from insulin-dependent diabetic individuals. Hum. Immunol. (1988). doi:10.1016/0198-8859(88)90034-1 • Schiffenbauer, J. et al. Complete sequence of the HLA DQ alpha and DQ beta cDNA from a DR5/DQw3 cell line. J. Immunol. (1987). • Jonsson, A.-K. et al. Class II genes of the human major histocompatibility complex. Comparisons of the DQ and DX ? and ? genes. J. Biol. Chem. (1987). • Blum, A. & Miller, H. The major histocompatibility complex and inflammation. Southern Medical Journal (2000). doi:10.1097/00007611-200002000-00002 • Mangalam, A. K., Taneja, V. & David, C. S. HLA Class II Molecules Influence Susceptibility versus Protection in Inflammatory Diseases by Determining the Cytokine Profile. J. Immunol. (2013). doi:10.4049/jimmunol.1201891

HLA-DQA2

• Khalil, I. et al. Trans-encoded DQ alpha beta heterodimers confer susceptibility to myasthenia gravis disease. C.R.Acad.Sci.III (1993). • Kwok, W. W. et al. Polymorphic DQ alpha and DQ beta interactions dictate HLA class II determinants of allo-recognition. Journal Of Experimental Medicine (1990). doi:10.1084/jem.171.1.85 • Hall, M. A., Lanchbury, J. S. S., Bolsover, W. J., Welsh, K. I. & Ciclitira, P. J. Celiac disease is associated with an extended HLA-DR3 haplotype which includes HLA-DPw1. Hum. Immunol. (1990). doi:10.1016/0198-8859(90)90052-Q • Sollid, L. M. Evidence for a primary association of celiac disease to a particular HLA-DQ alpha/beta heterodimer. J. Exp. Med. (1989). doi:10.1084/jem.169.1.345 • Wang, H. & He, R. HLA-DQA and DQB alleles contribute to susceptibility to insulin-dependent diabetes mellitus. Chinese Med. Sci. J. = Chung-kuo i hsueh k'o hsueh tsa chih (1993). • Bolsover, W. J., Hall, M. A., Vaughan, R. W., Welsh, K. I. & J. Ciclitira, P. A family study confirms that the HLA-DP associations with celiac disease are the result of an extended HLA-DR3 haplotype. Hum. Immunol. (1991). doi:10.1016/0198-8859(91)90012-X • Yu, L. & Sheehy, M. The cryptic HLA-DQA2 ('DX alpha') gene is expressed in human B cell lines. J Immunol. (1991). • Olerup, O. & Hillert, J. HLA class II-associated genetic susceptibility in multiple sclerosis: a critical evaluation. Tissue Antigens (1991). doi:10.1111/j.1399-0039.1991.tb02029.x • HLA-DQA2 major histocompatibility complex, class II, DQ alpha 2 [Homo sapiens (human)] - Gene - NCBI. Current neurology and neuroscience reports. Available at: https://www.ncbi.nlm.nih.gov/gene/3118. • Histocompatibility complex - Genetics Home Reference - NIH. U.S. National Library of Medicine (2020). Available at: https://ghr.nlm.nih.gov/primer/genefamily/hla. • Blum, A. & Miller, H. The major histocompatibility complex and inflammation. Southern Medical Journal (2000). doi:10.1097/00007611-200002000-00002 • Mangalam, A. K., Taneja, V. & David, C. S. HLA Class II Molecules Influence Susceptibility versus Protection in Inflammatory Diseases by Determining the Cytokine Profile. J. Immunol. (2013). doi:10.4049/jimmunol.1201891 • Khalil, I. et al. A combination of HLA-DQ beta Asp57-negative and HLA DQ alpha Arg52 confers susceptibility to insulin-dependent diabetes mellitus. J. Clin. Invest. (1990). doi:10.1172/JCI114569

HNMT

• The Food List. Histamine Intolerance Available at: https://www.histamineintolerance.org.uk/about/the-food-diary/the-food-list/. • Reference SNP (refSNP) Cluster Report: rs12995000. National Center for Biotechnology Information Available at: https://www.ncbi.nlm.nih.gov/SNP/snp_ref.cgi?rs=rs12995000. • Keeling, B. H. et al. Histamine N-methyltransferase Thr105Ile is not associated with Parkinson's disease or essential tremor. Parkinsonism Relat. Disord. (2010). doi:10.1016/j.parkreldis.2009.08.011 • Maintz, L. & Novak, N. Histamine and histamine intolerance. Am. J. Clin. Nutr. (2007). doi:10.1093/ajcn/85.5.1185 • Sighi. Normal histamine metabolism in healthy people. HIT > Histaminosis > Histamine metabolism (2020). Available at: https://www.histaminintoleranz.ch/en/histaminosis_histaminemetabolism.html. • Anton, R. F. et al. Pharmacogenomics. Nat. Genet. (2008). doi:10.1016/j.ejca.2015.06.122

NEUROTRANSMITTER SNP References

ADRA2A

• Attention-deficit/hyperactivity disorder (ADHD) in children. Mayo Clinic (2019). Available at: https://www.mayoclinic.org/diseases-conditions/adhd/diagnosis-treatment/drc-20350895#:~:text=Standard treatments for ADHD in,works best for your child. • Amanda Capritto, A. C. E.-C. P. T. What Is the Low Sugar Diet? Verywell Fit Available at: https://www.verywellfit.com/low-sugar-diet-pros-cons-and-how-it-works-4689214#:~:text=The low sugar diet involves,and vital vitamins and antioxidants. • S.N., R. et al. Association of ADRA2A and MTHFR gene polymorphisms with weight loss following antipsychotic switching to aripiprazole or ziprasidone. Human Psychopharmacology (2014). • Zhang, J. P. et al. Pharmacogenetic Associations of Antipsychotic Drug-Related Weight Gain: A Systematic Review and Meta-analysis. Schizophr. Bull. (2016). doi:10.1093/schbul/sbw058 • Kumar, S. et al. Significant role of ADRB3 rs4994 towards the development of coronary artery disease. Coron. Artery Dis. 25, 29–34 (2014). • Comings, D. E. et al. Additive effect of three noradrenergic genes (ADRA2a, ADRA2C, DBH) on attention-deficit hyperactivity disorder and learning disabilities in Tourette syndrome subjects. Clin. Genet. 55, 160–172 (1999). • Thorp, A. A. & Schlaich, M. P. Relevance of sympathetic nervous system activation in obesity and metabolic syndrome. J. Diabetes Res. (2015). doi:10.1155/2015/341583 • Davy, K. P. & Orr, J. S. Sympathetic nervous system behavior in human obesity. Neuroscience and Biobehavioral Reviews (2009). doi:10.1016/j.neubiorev.2008.05.024 • Attention-deficit/hyperactivity disorder (ADHD) in children. Mayo Clinic (2019). Available at: https://www.mayoclinic.org/diseases-conditions/adhd/diagnosis-treatment/drc-20350895#:~:text=Standard treatments for ADHD in,works best for your child.

COMT

• Lotta, T. et al. Kinetics of Human Soluble and Membrane-Bound Catechol O-Methyltransferase: A Revised Mechanism and Description of the Thermolabile Variant of the Enzyme. Biochemistry (1995). doi:10.1021/bi00013a008 • Stein, M. B., Fallin, M. D., Schork, N. J. & Gelernter, J. COMT polymorphisms and anxiety-related personality traits. Neuropsychopharmacology (2005). doi:10.1038/sj.npp.1300787 • Lee, L. O. & Prescott, C. A. Association of the catechol-O-methyltransferase val158met polymorphism and anxiety-related traits: A meta-analysis. Psychiatr. Genet. (2014). doi:10.1097/YPG.0000000000000018 • Ulmanen, I. et al. Expression and intracellular localization of catechol O-methyltransferase in transfected mammalian cells. Eur. J. Biochem. (1997). doi:10.1111/j.1432-1033.1997.0452a.x • Axelrod, J. O-methylation of epinephrine and other catechols in vitro and in vivo. Science (80-.). (1957). doi:10.1126/science.126.3270.400 • Tai, C. H. & Wu, R. M. Catechol-O-methyltransferase and Parkinson's disease. Acta Medica Okayama (2002). • Grossman, M. H., Emanuel, B. S. & Budarf, M. L. Chromosomal mapping of the human catechol-O-methyltransferase gene to 22q11.1?q11.2. Genomics (1992). doi:10.1016/0888-7543(92)90316-K • Golan, D. E., Armstrong, E. J. & Armstrong, A. W. Principles of pharmacology: the pathophysiologic basis of drug therapy. (Wolters Kluwer Health, 2017). • Bonifácio, M. J., Palma, P. N., Almeida, L. & Soares-Da-Silva, P. Catechol-O-methyltransferase and its inhibitors in Parkinson's disease. CNS Drug Reviews (2007). doi:10.1111/j.1527-3458.2007.00020.x • Wichers, M. et al. The catechol-O-methyl transferase Val158Met polymorphism and experience of reward in the flow of daily life. Neuropsychopharmacology (2008). doi:10.1038/sj.npp.1301520 • Diamond, A., Briand, L., Fossella, J. & Gehlbach, L. Genetic and Neurochemical Modulation of Prefrontal Cognitive Functions in Children. Am. J. Psychiatry (2004). doi:10.1176/appi.ajp.161.1.125 • Robinson, S., Goddard, L., Dritschel, B., Wisley, M. & Howlin, P. Executive functions in children with Autism Spectrum Disorders. Brain Cogn. (2009). doi:10.1016/j.bandc.2009.06.007 • Bruder, G. E. et al. Catechol-O-methyltransferase (COMT) genotypes and working memory: Associations with differing cognitive operations. Biol. Psychiatry (2005). doi:10.1016/j.biopsych.2005.05.010 • Chen, J. et al. Functional analysis of genetic variation in catechol-O-methyltransferase (COMT): Effects on mRNA, protein, and enzyme activity in postmortem human brain. Am. J. Hum. Genet. (2004). doi:10.1086/425589