# Role of Nutrigenomics in Human Health and Diseases

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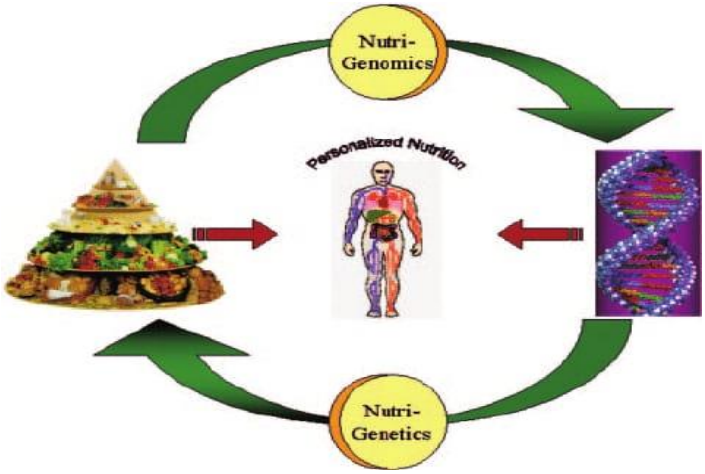
With the completion of human genome sequencing and incoming the-Omics area, the new term "Nutritional Genomics" tends to replace the former "nutrient-gene interactions". Nutritional genomics is the study of the interactions between our genetic makeup and the foods we consume and the health outcomes that may occur. Nutritional genomics is a relatively new field of study that involves two distinct fields: nutrigenomics and nutrigenetics. The influence of nutrients on genes expression is called Nutrigenomics. It is the science that examines the response of individuals to food compounds using post-genomic and related technologies which includes genomics, transcriptomics, proteomics, metabolomics etc. It will also determine the individual nutritional requirements based on the genetic makeup of the person as well as the association between diet and chronic diseases which will help to understand the etiologic aspects of chronic diseases such as obesity, cardiovascular disease (CVD), type-2 diabetes and cancer. In contrast, the heterogeneous response of gene variants to nutrients, dietary components and developing nutraceuticals is called Nutrigenetics. It also reveals why and how people respond differently to the same nutrient. In this way, considering different aspects of gene–nutrient interaction and designing appropriate diet for every specific genotype that optimize individual health, diagnosis and nutritional treatment of genome instability, we could prevent and control conversion of healthy phenotype to diseases.

**Principles of nutrigenomics**

These are 5 basic principles of nutrigenomics:

i. Substances contained in the food (micro- and macro-nutrients) can directly or indirectly affect the human genome through changes in its structure and gene expression.

ii. Some genes regulated by active substances in the diet probably play a crucial role in the onset, incidence, progression and severity of the disease.




iii. The degree to which diet influences the balance between health and disease may depend on individual's genetic makeup.

iv. Under certain circumstances and in some individuals the diet can be an important risk factor for the development of the number of diseases.

v. Nutritional intervention is based on the knowledge of individual's nutritional status and needs as well as genotype (individualized nutrition) and can be used for prevention, mitigation or healing the chronic diseases.

**Strategies related to nutrigenomics**

The use of genomic techniques in molecular nutrition research can be divided into **two different strategies** which are complementary to each other.

**Strategy I:** It uses the conventional hypothesis-driven strategy, i.e., specific genes and proteins. Nutrients have an impact on the expression of these specific genes and proteins. These are discovered utilizing genetic technologies including transcriptomics, proteomics, and metabolomics, which subsequently enables the identification of the regulatory pathways by which food affects homeostasis.

**Strategy II:** It is the system biology approach, which includes gene, protein and metabolite signatures. They are linked to particular nutrients or dietary patterns and may serve as "early warning" molecular indicators for nutrient-induced changes to homeostasis. The first strategy will provide us with

detailed molecular data on the interaction between nutrition and the genome, whereas the second strategy might be more important for human nutrition, given the difficulty of collecting tissue samples from 'healthy' individuals.

**Nutrient-gene interaction can occur in three ways:**

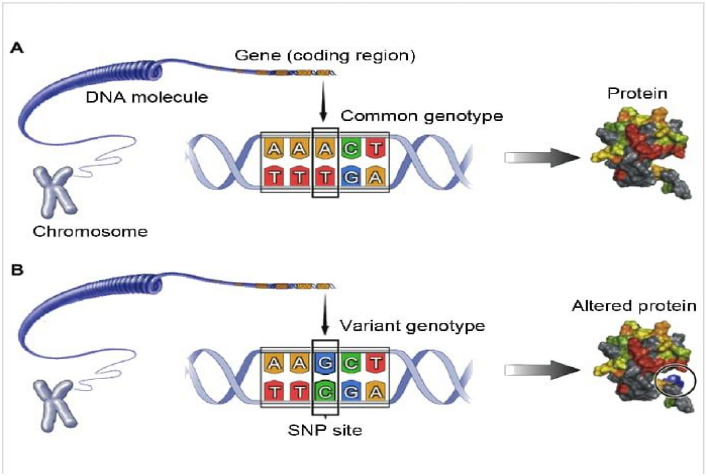
(i) **Direct interactions:** When nutrients interact with a receptor, they may behave as transcription factors that bind to DNA and abruptly activate the expression of certain genes

(ii) **Epigenetic interactions:** Nutrients can alter the structure of DNA (or of histone proteins in chromatin) so that gene expression is chronically altered. The sustained effects of epigenetic mechanisms are mediated by methylation of DNA or by methylation, biotinylation of histones or acetylation, or by both functions. The resulted epigenetic modifications can result in changes in gene expression that can last throughout a person's life and can even persist across generations.

(iii) **Genetic variations**: Common genetic variations (single-nucleotide polymorphisms (SNPs)) can alter the expression or functionality of genes. All the three mechanisms can result in altered mechanism of and altered dietary requirements for nutrients.

**Gene-nutrient interaction (i.e., Nutrigenetics)**

Nutrigenetics term was used first time by Dr R.O Brennan in 1975 in his book Nutrigenetics. Nutrigenetics is defined as the science that studies the effect of genetic variation on dietary response. Its main goal is to investigate the impact of genetic variation, particularly as a single-nucleotide polymorphism (SNP), on an individual's response to dietary intake, especially in terms of how genetic variation influences an individual's metabolic state. SNPs are the most common genetic variation, occur at about 500-2000 bp throughout the human genome, and normally found in at least 1per cent of the population. When the genetic sequences of two individuals are aligned and compared, only about 1 of every 1000 base pairs of nucleotide sequence of human DNA, which is about 0.1%, exhibits variance and many of these variations




are found in just a single base pair/letter in the DNA code, for example, a cytosine (C) in place of guanine (G). This variation involving a single base pair is called SNP.

In this sense, several SNPs have been associated with common chronic diseases through interactions with the intakes of macro and micronutrients, or with the consumption of particular foods and dietary patterns. Examples include polymorphisms in taste-related genes such as the sweet taste receptor (TAS1R2) and cluster of differentiation (CD36), which have been linked to dyslipidaemia in Mexican patients who consume high levels of carbohydrates and fats, respectively. Low intakes of folate, vitamin B6 and vitamin B12 have been related to an increased risk for breast cancer in people with common variations in the genes regulating homocysteine metabolism, such as methylenetetrahydrofolate reductase (MTHFR) and methionine synthase (MTR). Additionally, it has been noted that a number of polymorphisms in the genes that make up the vitamin D pathway can also affect vitamin D status, by altering the biological activities of the vitamin D in the body. It's interesting to note that postmenopausal women with low calcium intakes have an increased risk of osteoporosis due to SNPs in the vitamin D receptor (VDR) gene, which impact vitamin D availability. Moreover, SNPs in genes encoding lipid proteins such as apolipoprotein C3,(APOC3) and apolipoprotein A 1(APOA1) conferred a higher risk of metabolic syndrome in subjects with a Western dietary pattern. Likewise, a genetic variant in the cytochrome P450 family 1 subfamily A member (CYP1A2) gene was associated

with an increased risk of hypertension and CVD in moderate and heavy coffee drinkers.

**Role of nutrigenomics in human health and diseases**

Nutrigenomics aims to identify the effects of several nutrients, including macronutrients and micronutrients on the genome and explores the interaction between genes and nutrients or food bioactive and their effects on human health. It investigates the impact of diet and nutrition on gene expression, especially through epigenomic (e.g., histone methylation), transcriptomic (e.g., RNA transcription), proteomic (e.g., protein synthesis) and metabolomic (e.g., metabolite synthesis) high-throughput assays. Transcriptomics, proteomics, and metabolomics are technologies that apply in Nutrigenomics research. According to numerous studies, nutrients can alter the expression of genes at the level of gene regulation, signal transduction, chromatin structure and protein function.

**Nutrigenomics effect on human health and diseases**

According to estimates based on body mass index (BMI), more than 35% of the world's population (2100 million people) is either overweight or obese. Obesity is linked to a wide range of health problems, including dyslipidaemias, cardiovascular diseases (CVD), type 2 diabetes mellitus (T2DM), non-alcoholic fatty liver disease (NAFLD) and various types of cancer. These issues come with significant financial and societal implications.

High-fat diets, particularly those high in saturated fatty acids, have increased the expression of neuropeptides involved in the development of obesity and gene expression profiles related to inflammation, glucose intolerance, and liver lipid accumulation. However, low-protein diets increased the expression of the gluconeogenic genes in the liver, which led to the development of glucose intolerance. Additionally, diets low in choline and folate were linked to the deregulation of genes involved in lipid metabolism, which affected the susceptibility and severity of NAFLD. While selenium, vitamin B12, and vitamin A deficiency could raise CVD vulnerability by upregulating proinflammatory and lipogenic genes, chromium insufficiency downregulated insulin signalling genes, showing a role in T2DM pathogenesis.

Experimental studies have shown the beneficial effects of nutrients and bioactive food compounds as a result of the regulation of critical gene expressions. In this regard, it has been shown that eating a Mediterranean diet lowers the postprandial expression of genes that encode proteins linked to inflammation, endoplasmic reticulum stress, atherogenesis and oxidative stress. A low expression of genes associated to inflammation and inappropriate lipid accumulation has also been linked to large intakes of monounsaturated fatty acids from the consumption of olive oil. Diets with a high content of polyunsaturated fatty acids favourably regulate the expression of neuropeptide genes involved in energy homeostasis. Moreover, energy-restricted diets supplemented with eicosapentaenoic acid, and Ł-lipoic acid have been associated with upregulation of fatty acid-oxidizing genes, as well as downregulation of lipogenic and proinflammatory genes. In contrast, high-protein diets prevent and reverse NAFLD by modulating the expression of genes involved in liver lipid metabolism.

**Effects of bioactive food compounds on gene expression**

Effects of bioactive food chemicals on gene expression have been extensively investigated, and some of the most well-known examples are green tea, theaflavin in black tea, sulforaphane in cruciferous vegetables, resveratrol in grapes and red wine, curcumin in turmeric, genistein in soy beans, and various polyphenols in apples. As a result, substances including genistein, theaflavin, curcumin, epigallocatechin-gallate, and sulforaphane may have anticancer effects by upregulating tumor suppressor genes and downregulating tumor-promoting genes. Additionally, curcumin and resveratrol have demonstrated antiatherogenic benefits by reducing the expression of matrix metalloproteinases, which are crucial for plaque formation and progression. The modulation of genes involved in adipogenesis, lipolysis, and fatty acid oxidation by apple polyphenols is noteworthy because it appears to prevent diet-induced obesity.

## Conclusion

Nutrigenomics is a rapidly developing new body of knowledge that will change future research and practice in human nutrition. Nutrigenomics is anticipated to produce biomarkers for health, early biomarkers for disease propensity, dietary responders and non-responders and bioactive food components. In the long run, nutrigenomics will enable efficient dietary-intervention strategies to restore normal homeostasis and prevent diet-related disorders. Undoubtedly, nutrigenomics research is still in its infancy, and much more work needs to be done to properly understand the mechanism and get over any obstacles or limits.

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