Applied Cell Biology

ISSN : 2320-1991 Online
ISSN : 2320-1983 Print
Volume 10 Issue 1
**DOI:** https://dx.doi.org/10.53043/2320-1991.acb90019

Review Article
Appl Cell Biol, 10(1), 2022 [22-25]

# Nutritional Therapy by Leveraging our Knowledge of Nutrigenomics and Nutrigenetics

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## ABSTRACT

Nutrigenomics is a tool to measure gene expressions in response to dietary intake and better understand the human body's nutrient-gene interactions. Nutrigenetics is a tool to assess how genetic variations influence nutrients' interaction result of developing various chronic diseases. Dietary factors alter genes' expression, including nutritional factors such as antioxidants, vitamins, phytochemicals, caffeine, sterols, fatty acids, and alcohol. Furthermore, genetic polymorphisms in nutrient action targets such as enzymes could alter molecular pathways that influence the physiological response to dietary intake. Identifying relevant diet-gene interactions will benefit individuals seeking personalized nutritional advice.

## Keywords

Nutrigenomics
Nutrigenetics
Gene-nutrient interaction
Personalized nutrition
Bioactive

Two people took an exact amount of the supplement, but it has a different effect on improving their health. Dietitians gave supplements to the atopic outpatients; someone has excellent result treatment, whereas someone does not work that way. Why does such a thing happen? So people need to know the two words: Nutrigenetics and Nutrigenomics. The heterogeneous response of gene variants to nutrients, dietary components, and developing nutraceuticals is called Nutrigenetics [1]. Nutrigenomics studies the interaction between nutritional elements, the genome, and regulating proteins and metabolism [1]. A healthy nutrient intake level depends on the internal levels of the bioactive substance to interact with personal genes [2].

An individual has a difference in height, weight, and physical difference, including the constitution, so it has involved different nutrients of requirement. Otherwise, people have stress as a bad thing happens, the number of nutrients needed should increase. Therefore, Nutrition science has been identifying how genes to bioactive nutrients interact through the digestion, absorption, and metabolism system and trying to discover the best way to utilize the nutrients to nutritious of the human body [3].

Molecular nutritional therapy considers being different from classic nutrition therapy. Activating the nutrients can keep 60 trillion cells as a whole to maintain the body homeostasis as health. Molecular nutrition therapy is based on individual differences with nutrients' genes expressed in the body [4]. People intake foods following a dietary guideline for Americans with supplements to assimilate their bodies daily. In that case, we call classic nutrition from a meal using the quantity as an indication. It only considered nutrient intake of quantities rather than affecting the body's quality with nutrients. The problems of classic nutrition are right here. Activating vitamins can be changed in the body condition because the human body has pharmacologic interaction in the human body if it becomes highly concentrated [5]. Therefore, how much vitamin dosage is needed depends on individual people's pharmacologic reaction, making a significant difference [6].

The human body of molecular bioactive substance is one of the enzymatic reaction. The enzymatic response in the human body to absorption and digestion of nutrients for optimal human body function. Many enzymes act in vivo. Those enzymes work alone in some cases, but many enzymes the required protein. The coenzyme is essential in a supporting

factor [7]. There are a series of chemical reactions in the body. For example, glycolysis leads to pyruvic acid and lactic acid from glucose.

The enzymatic reaction is complicated, but it is essential to explaining molecular nutrition concepts. Coenzyme (vitamins) must bind to the substrate (protein) so that enzymatic reaction happens. However, the supplement's effect makes a big difference to this coupling efficiency in an individual, leading to significant individual differences in vitamins required. That is the mean of an affinity, and it is coupling efficiency [8]. The difference between people has a different affinity; people have a one-time to around 100 times. In other words, some people take one dosage is enough, but others may need to take 100 times to effect. In addition, the required amount of the nutrient varies due to the human body of condition in the same individuals. Generally, the quantity of the necessary nutrient is significantly different when comparing little stress lived an everyday life, the athlete activating all day and working intensely. So then, the critical thing is to adjust to the nutrient needs to meet nourishment requirements.

A short while ago, we mention two words: Nutrigenomics and Nutrigenetics. Sciences completed to identify and map out a human gene in the human genetic code between 1990 and 2003 [9]. The achievement brings up all of the fields of scientific research opened up. Nutrigenetics and nutrigenomics are the two fields to benefit from that.

Nutrigenomics enables measuring the needs of nutrients, supplements, and bioactive food compounds for individuals most effectively and determining what works for each genetic type. Nevertheless, it is a quantitative analysis of their diets' gene expression [10]. Studies have shown the beneficial effects of nutrients and bioactive food compounds due to the regulation of gene expressions. For example, consuming a Mediterranean diet reduces the expression of genes inflammation and oxidative stress proteins [11]. Also, high intakes of monounsaturated fatty acids are associated with a low expression of genes involved in inflammation [12]. Moreover, taking eicosapentaenoic acid and α-lipoic acid supplements is associated with increasing fatty acid-oxidizing genes and decreased lipo-genic proinflammatory genes [13].

In contrast, high-protein diets prevent and reverse nonalcoholic fatty liver disease (NAFLD) by modulating genes involved in the liver lipid metabolism system [14]. In addition, the bioactive food of green tea, theaflavin (black tea), sulforaphane (cruciferous vegetables), resveratrol (grapes and red wine), curcumin (turmeric), genistein(soybean), and several apple polyphenols of compounds contributed to gene expression as an anti-oxidation bioactive substance in the human body [15]. Thus, epigallocatechin-3-gallate, theaflavin, curcumin, sulforaphane, and genistein have anticancer properties by decreasing tumor-promoting genes [16]. Nutrigenomics gave a tool for quantitative analysis of gene expressions of interactive efficiency between genes and bioactive foods.

Researchers have identified the effect of different dietary factors on gene expression profiles related to disease susceptibilities. For example, high-fat diets, especially rich in saturated fatty acids, have induced gene expression profiles related to inflammation. On the other hand, low-protein diets enhanced hepatic gluconeogenic gene expression with subsequent glucose intolerance [17]. Furthermore, chromium deficiency to a role in T2DM pathogenesis [18], whereas depriving selenium [19], vitamin B 12, and vitamin A could increase cardiovascular disease (CVD) susceptibility to proinflammatory and lipo-genic genes [20].

On the other hand, Nutrigenetics looks at the relationships between genes, nutrients, and how an individual's health is affected. It determines how personalized nutrition fits its nutrients and calorie intake. Nutrigenetic studies identify genetic variants associated with disease susceptibility through interaction with dietary nutrients. Nutrients and genome interaction cause disease is not new. For example, an inappropriate diet with individual genotype could be a risk factor for genetic illness; single nucleotide polymorphisms (SNPs) in the vitamin D receptor (VDR) genes, which affect vitamin D availability, it associated with osteoporosis in postmenopausal women with low calcium intakes [21].

Moreover, SNPs in genes encoding lipid proteins such as Apolipoprotein A1 (APOA1) conferred a higher risk of metabolic syndrome in people with a western dietary pattern [22]. Likewise, a genetic variant in the cytochrome P450 family one subfamily a member 2 (CYP1A2) gene was concerned about an increased risk of hypertension and CVD [23]. Furthermore, metabolic disorders are other genetic variations to diet, such as Phenylketonuria (PKU), defects associated with long-chain fatty acid oxidation with iron Absorption (hemochromatosis).

Additionally, genetic risk scores (GRS) have examined the cumulative effect of SNPs on diet interactions and disease susceptibility. Significant obesity GRS interactions with saturated fat, sugar-sweetened beverages, and fried food concern BMI and obesity [24]. For example, it showed that gene-based personalized nutrition targeting the apolipoprotein E (APOE) gene was more effective in reducing saturated fat intake than standard dietary advice [22]. Furthermore, angiotensin I converting enzyme (ACE) genotype for personalized nutrition resulted in more significant sodium

intake changes than general population-based nutritional advice [25]. Likewise, individuals who have their fatty acid desaturase 1 (FADS1) genotype was more aware of the role of omega-3 fatty acids in health than those who did not receive their personal genetic information [26]. These findings are related to a better understanding, awareness, and usefulness of genetics-based dietary recommendations than general nutritional advice.

In addition to SNPs, previous studies have found evidence of an association between Choroidal Neovascularization (CNVs) and the risk of metabolic diseases. For example, CNV in the leptin receptor (LEPR) gene was associated with metabolic traits and the risk of T2DM [27]. Another DNA biomarker is the pentanucleotide (CTTTA) Del/Ins variant in the three ′ -UTR of the LEPR gene, which was associated with the risk of T2D [28]. Analyzing the relationships between risks factors of disease genes and nutrients with a new tool of nutrigenetic is also cost-effective to link to nutrients and diseases of genes. How an individual's health is affected, and how nutrients combine into a person's chronic diseases genes to reduce their risks. That is why further studies are needed to assess possible interactions between these genetic variants and dietary intake concerning disease risk and their effects on dietary response.

As an above result, we will no longer rely on dietary recommendations that fit all American policies. Instead, we are discovering a recommended solution for personalized nutrition. We are searching for the maximum effect to increasing interaction efficiency between genes and bioactive foods in the individual human body. Designing a customized diet to optimize individual diet needs by leveraging our knowledge of Nutrigenomics and Nutrigenetics is our health care new challenge.

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**Citation:** Gao-Balch Y (2022) Nutritional Therapy by Leveraging our Knowledge of Nutrigenomics and Nutrigenetics. Appl Cell Biol, 10(1), 2022 [22-25]