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Neda Shamim
Research Scholar, Department of Food, Nutrition and Public Health, ECHS, SHUATS, Uttar Pradesh, India

Alka Gupta
Assistant Professor, Department of Food, Nutrition and Public Health, ECHS, SHUATS, Uttar Pradesh, India

Virginia Paul
Associate Professor, Department of Food, Nutrition and Public Health, ECHS, SHUATS, Uttar Pradesh, India

Eloni Vida
Research Scholar, Department of Food, Nutrition and Public Health, ECHS, SHUATS, Uttar Pradesh, India

Correspondence
Neda Shamim
Research Scholar, Department of Food, Nutrition and Public Health, ECHS, SHUATS, Uttar Pradesh, India

Nutritional genomics: A review

Neda Shamim, Alka Gupta, Virginia Paul and Eloni Vida

Abstract
Nutritional Genetics is the combination of two areas- nutrigenomics and nutrigenetics. Nutrigenomics is the study of how foods influence gene expression and how individual genetic variation affects the way an individual responds to nutrients in food. It will determine the association between diet and chronic diseases which will help to understand the etiologic aspects of chronic diseases such as cancer, type-2 diabetes, obesity and cardiovascular disease (CVS). In contrast nutrigenetics is the tool that helps to reveal the association between the genetic makeups of a particular person with the diet. It also gives an idea why and how a person reacts or responds differently by a particular food ingredient. Thus, present review highlights the interaction of genetic background with the diet and how these two areas interfere in the development of life threatening disease in both developing and developed countries.

Keywords: Nutrigenomics, Nutrigenetics, SNP, Diet related disease, Nutrient gene interaction.

1. Introduction
Nutritional genetics is not a single term that can be solely defined or explained, in fact it is combination of two broad areas-Nutrigenomics and Nutrigenetics [1].

1.1 Nutrigenomics
Nutrigenomics is the application of genomics in the field of nutrition research, enabling associations between specific nutrients and genetic factors, e.g. the way in which food or food ingredients influence gene expression. Nutrigenomics facilitate the greater understanding of how nutrition affects metabolic pathways and how this process goes away in diet-related diseases [2]. It is an attempt to study the genome-wide influences of nutrition and aims to identify the genes that influence the risk of diet-related diseases on a genome-wide scale, and to understand the mechanisms that underlie these genetic predispositions [3]. Nutrigenomics will also identify the genes involved in physiological responses to diet and the genes in which small changes, called polymorphisms, may have significant nutritional consequences and the influence of environmental factors on gene expression (figure 1) [4].

1.2 Nutrigenetics
Nutrigenetics is the tool that helps to reveal the association between the genetic makeup of a particular person with the diet. It also gives an idea why and how a person reacts or responds differently by a particular food ingredient [5]. These individual differences may be at the level of single nucleotide polymorphisms rather than at the gene level. It is envisaged that nutrigenetics may lead to individualized dietary advice [6].
These two approaches secure a vital role to understand the effects of diet on individual persons **(1)** and eventually nutrigenomics will lead to evidence-based dietary intervention strategies for restoring health and fitness and for preventing diet-related disease [7].

2. Gene Diet Disease Interaction
2.1 Nutrigenetic diseases
Human diseases like monogenic diseases are known to be associated with genes and slight modifications in the dietary intake can prevent some monogenetic diseases [8] e.g., high protein food as well as food containing amino acid phenylalanine should be avoided in case of phenylketonuria (PKU). Patients having galactosemia (lack of a liver enzyme to digest galactose) should avoid diets which contain lactose or galactose, while in case of lactose intolerance (shortage of the enzyme lactase) patients should avoid milk and milk products [9].

2.2 Nutrigenomics diseases

Nutrients are considered to be the most significant environmental stimuli [10] and Genomes are exposed to various types of environmental stimuli, including nutrition. Therefore, the genetic expressions are highly dependent and regulated by, nutrients and phytochemicals present in food [11]. Unbalanced diets can change nutrient-gene interactions; thereby can increase the possibility of occurrence of chronic diseases. Either direct or indirect dietary chemical can alter genomic expressions [12].

Nutrient imbalances may lead to aging, alcoholism/ substance abuse, behavioral disorders, cancer, cardiovascular disease (CVD), chronic fatigue, deafness, diabetes, immune disorders, macular degeneration, multiple sclerosis, neurological disorders, osteoporosis, Parkinson's disease and stroke [9]. Diseases that are known to involve in the interactions between multiple genetic and environmental factors include many cancers, diabetes, heart disease, obesity and some psychiatric disorders [9].

Therefore, both areas (nutrigenomics & nutrigenetics) aim to disclose genome-diet interactions; although their approaches and immediate goals are distinct. Nutrigenomics will reveal the most favorable diet to choose from nutritional alternatives available, whereas nutrigenetics will provide information that will be useful for identifying the optimal diet for particular subject i.e. personalized nutrition [13].

The following principles of nutritional genomics serve as a conceptual basis for understanding the focus and promise of this emerging field [4]:

1. Unbalanced diets are risk factors for developing disease.
2. Gene expression and / or genome structure can be altered or changed by dietary chemicals.
3. The extent to which diet influences the balance between healthy and disease condition may depend on an individual's genetic makeup.
4. Some diet-regulated genes are likely to play a role in the onset, incidence, progression, and/or severity of chronic diseases.

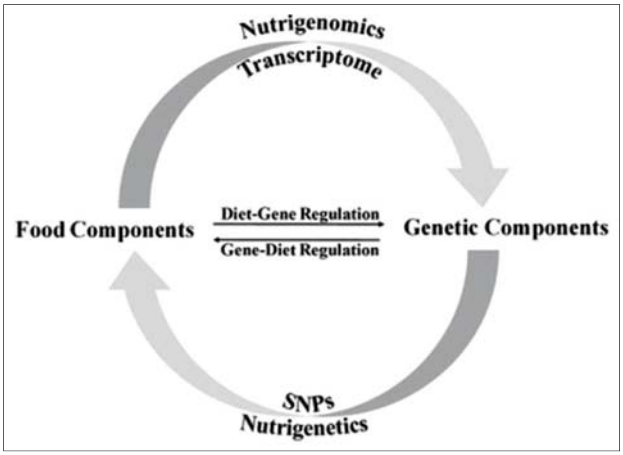

Fig 1: Distinctions between nutrigenomics and nutrigenetics. The investigation of how food components modulate changes in gene expression profile or transcriptome is defined as nutrigenomics. On the other hand, nutrigenetics is defined as the study of how genetic variations such as single nucleotide polymorphism (SNP) among individuals affect their response to specific food components. Food pyramid image obtained from: http://openi.nlm.nih.gov/detailedresult.php?img=2682937_CG-9-239_F1&req=4

3. Nutrigenomics - Direct Link to Single Nucleotide Polymorphisms (SNPS)

Single nucleotide polymorphisms (SNPs) are the most common genetic variations known as polymorphism. Although at the gene sequence level, humans are having 99.9 % identical genomic sequences [12]. Remaining 0.1 % variations in genomic sequence responsible for differences like phenotypic differences (height, weight etc.) and individual's susceptibility to disease conditions and health status. Genetic polymorphisms influence absorption as well as metabolism of dietary components. Epigenetic variations can bring changes in DNA methylation pattern and thus affects overall genetic expression. Many dietary components affect post translational events and many account for at least part of the variation [14]. As already discussed with the example of phenylketonuria there is direct link of nutrition to genetic level one more example is available that direct link nutrition with SNPs [5]. Regarding SNP's, one another best-described example is the relationship between folate and the gene MTHFR – 5, 10-methylenetetrahydrofolate reductase. MTHFR is required for supplying 5-MTHF, which is essential for remethylation of homocysteine for production of methionine. Methionine is crucial for many metabolic pathways including neurotransmitters production and genetic expression regulation. Folate is essential to the efficient functioning of MTHFR. When thymine replaces cytosine at base pair 677 in MTHFR gene, it leads to two forms of the protein: the wild type (C), which functions normally, and the thermal-labile version (T), which has a significantly reduced activity [15]. Individuals with two copies of the wildtype gene (CC) or one copy of each (CT) will have normal folate metabolism. Those persons with unstable version of both copies (TT) and low folate intake will have higher plasma homocysteine levels, which increases their risk of cardiovascular disease and premature cognitive disturbances. If supplemented with folic acid or increased folate intake, these individuals can restore their normal methionine levels through metabolising homocysteine [16]. SNP analysis provides a great tool for investigating the role of nutrition in disease and health status at molecular stage and its deliberation in metabolic and epidemiological studies can throw a light to define optimal diets [17]. Each genomic sequence is a recipe for a specific protein or group of proteins that regulates biological functions and some SNPs change the recipe for the gene that could result either a different quantity of the protein is produced or the structure is altered [4].

4. Nutrigenomics and Chronic Diseases

The present review will highlight the interaction of genetic background and diet with regard to development of life threatening chronic conditions as obesity and cancer that are responsible for the majority of deaths in developed countries [4]. The nature of these interactions is indeed very complex.

4.1 Obesity

At present obesity is alone major element of a group of metabolic abnormalities (metabolic syndrome) which includes impaired glucose tolerance, insulin resistance, hypertension and noninsulin-dependent diabetes mellitus [18]. Obesity and associated metabolic abnormalities drastically increase the risk of development of various chronic diseases that includes CVD and cancer [19-20]. Regulation of dietary habits may be affected by SNP's in the genes that encode taste receptors and a variety of peripheral signaling peptides such as cholecystokinin, insulin, ghrelin, leptin etc. Polymorphic

central regulators of energy intake include agouti-related protein, hypothalamic neuropeptide Y, melanocortin pathway factors and their receptors [21]. Understanding nutrigenomics is highly promising as it may modulate the chronic process of inflammation; one of the causes of obesity [22]. A study shows that some food contain anti-inflammatory bioactives, such as the caffeic acid (found in Yerba mate), tyrosol (found in olive oil), quercetin (present in fruits and greeneries), and lycopene (present in tomatoes, guavas, and watermelon). These molecules act inhibiting the expression of COX2 and iNOS genes through the reducing the translocation of the Kappa-B nuclear factor from the cytoplasm to the nucleus [22, 23]. α-tocoferol, a bioactive compound found in green tea, acts by decreasing the level of this chronic inflammatory process that occurs in obese individuals. Various other studies also indicate that this component can assist on the treatment of obesity [24, 25].

4.2 Cancer

Cancer is a multi-stage process when gene expression, and protein and metabolite function begin to operate abnormally [26]. Inborn mutations in genes can increase one's susceptibility for cancer. Gene diet interactions greatly affect the occurrence of cancer and chances of developing cancer can be markedly increased. Studies of twins show that the likelihood of identical twins developing the same cancer is less than 10%, indicating that the environment plays an important role in cancer susceptibility [9].

Certain micronutrient which is needed by the organism depends on the person's age, genetic background, and physical state [27, 28]. Previous researches shows that the deficiency of micronutrients, such as folic acid, vitamins B12, B6, C, and E, selenium, niacin, and zinc can cause changes into the DNA structure similar to what is seen after radiation exposure [24, 27, 28]. These changes may lead to rupture of the DNA double strand, oxidative lesions, or both. Furthermore, they narrowly related to the development of cancer [28]. DNA structure can also be altered and induce mutation by consumption of contaminated food resulting the toxic effects on health. In case of aflatoxin B1, this forms an adding compound that able to binds to the N-7 position of guanine residue, generating a new product. This generated product cleaves, then, the interaction between one sugar and one nitrogenous base of a nucleotide occur leading to the formation of anapurinic site. The mutation developed can cause severe damage to the liver, including necrosis, cirrhosis, and carcinoma [29, 30]. Another important micronutrient is the folic acid, during folate metabolism, the folic acid present in food sources, is absorbed by the intestine and, through several processes of catabolism and synthesis, it is changed into 5-methyltetrahydrofolate. This component is necessary for the synthesis of methionine, which in turn is used during the process of DNA mutilation. Thus, a diet deficit in folic acid can change this process and interfere on DNA replication, leading also to an increased risk of cancer development [24–27, 28]. On the other hand, there are minerals which work as protectors against cancer development [28]. Such as: (i) selenium, by stimulating the production of glutathione peroxidase enzyme that involves in the reduction of hydrogen peroxide and ultimately maintain the integrity of cell membranes; (ii) prostacyclins, reduce the oxidative damage of DNA, lipids, and lipoproteins; (iii) zinc, involves in the maintenance of genomic stability, genetic expression, and apoptosis modulation [24, 28].

5. Ethical Issues

Nutritional genetics is an emerging science that can be used as a tool for detecting the susceptibility of disease and its association with genes to diet; however more study still need to be done to validate the health promotion and prevention of disease. In this view, selection of participants for the study may be a challenge that raises methodological as well as ethical issues. Biased individuals should be excluded from the study as they might affect the usefulness of findings. Ethical, legal and social issues rise with respect to how the general population may access nutrigenetic tests for health promotion and prevention of diseases. Nutritional genetics will likely always remain a science of probabilities because it detects disease susceptibility arising from low penetrance polymorphisms. The science reveals the answers of the curiosity of human brain, so too should the ethical and regulatory appraisal of that science. Further, it is very important to find out more about latest advancements and opportunities as well as limitations of nutrigenomics and its clinical and commercial applications in order to encourage greater public awareness, prevention strategies and understanding of the potential risks and benefits of this rapidly evolving field.

6. Conclusion

Currently, with tools of nutritional genetics, diet-gene association studies are revealing evidence on which to base gene-specific dietary intervention trials are carried out to confirm results. Nutrigenomics research provides us additional knowledge of biological function. The application of information by physicians for the prevention and treatment of complex chronic diseases, however, has not yet been widely adopted. In future, application of information should be used to large population, however, feasibility is yet to be determined, but the principles of nutrigenomics are expected to soon allow us for more targeted interventions. [15] Collective efforts by the scientific community are needed to strictly follow guidelines put forth regarding experimental designs, analysis, and data storage for nutritional research. This strategy will be helpful to generate a sound database useful for clinicians and dietetic practitioners

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