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INTERACTION BETWEEN DIET AND GENETIC PREDISPOSITION

Elemuo, J.F. [1]; Udeh, Kester U. [2]
Department of Biochemistry, School of Science and Technology, Federal Polytechnic, Oko, Anambra State, Nigeria
judithfrancis90@gmail.com 08132322921, 07032355979

Abstract
This paper examined the complex relationship between diet and genetic predisposition, highlighting how these elements interact to shape health outcomes. Genetic predisposition refers to inherited traits that increase the risk of developing specific health conditions, while diet encompasses the nutritional choices and eating habits that can modify these risks. Research shows that while genetics provide a baseline for health, dietary habits can significantly influence the expression of genetic traits through mechanisms like epigenetics. For example, certain nutrients can activate or inhibit genes associated with metabolism, inflammation, and chronic diseases. The interaction diet and genetics offers insights into the differences in disease susceptibility among individuals, underscoring the significance of personalized nutrition.
Keywords: genetic predisposition, diet, interaction, nutrition, diseases.

Introduction

Nutrition is a modifiable key factor that is able to interact with both the genome and epigenome to influence human health and fertility. In particular, specific genetic variants can influence the response to dietary components and nutrient requirements, and conversely, the diet itself is able to modulate gene expression. In this context and the era of precision medicine, nutrigenetic and nutrigenomic studies offer significant opportunities to improve the prevention of metabolic disturbances, such as Type 2 diabetes, gestational diabetes, hypertension, and cardiovascular diseases, even with transgenerational effects. Understanding **gene-**diet interaction has the potential to promote diet modifications on the basis of genetic makeup. Several recent large-scale studies found reproducible evidence showing consumption of sugar sweetened beverages or dietary patterns might modulate genetic predisposition to obesity or cardiovascular disease. Genome-wide association studies (GWAS) have discovered many genetic variants associated with specific nutrition-related traits including nutrient absorption, lipid metabolism, nutrient utilization, and fat accumulation that in turn can result in gene–diet interactions and human diseases. Risk factors for several of the major chronic diseases, such as cardiovascular disease, hypertension, diabetes, obesity, and cancer, are often observed during childhood. Therefore, preventive measures adopted early in life may help reduce the prevalence of these diseases in adulthood. Diet is undoubtedly such a risk factor that could benefit from early intervention.

Nutritional genomics

The interaction between the nutrients and cellular/genetic processes is being referred to as “nutritional Genomics” (Kaptur, *et al*., 2004). This term describes the interface of biochemistry genomics, human nutrition, understanding of reactions and interactions at the molecular genomic levels. The conceptual basis for this genomic research can be summarized with the following five principles: 1) Common dietary chemicals act on the human genome, either directly or indirectly, to alter gene expression and/or structure. 2) Under certain circumstances and in some individuals, diet can be a serious risk factor for a number of diseases. 3) Some diet-regulated genes (and their normal, common variants) are likely to play a role in the onset, incidence, progression, and/or severity of chronic diseases. 4) The degree to which diet influences the balance between healthy and disease states may depend on an individual’s genetic background. 5) Dietary intervention based

on knowledge of individual nutritional requirement, nutritional status, and genotype (i.e., "individualized nutrition" GV) can be used to prevent, relieve, or cure chronic diseases.

Application of Nutritional Genomics

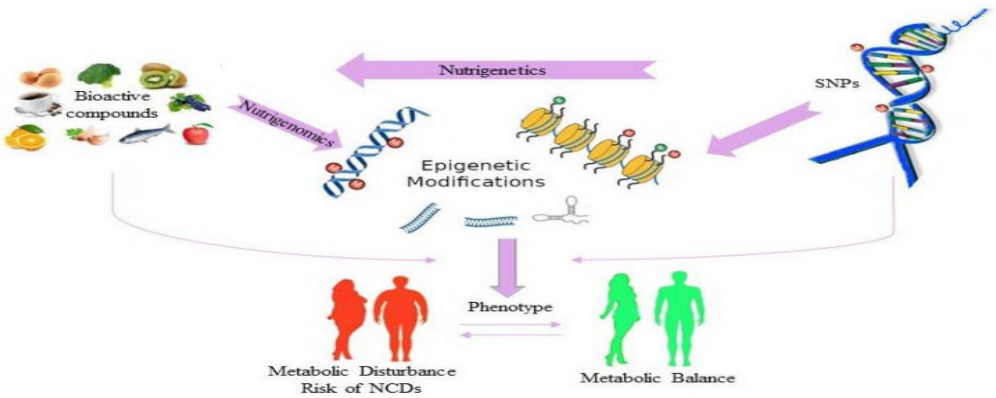
The applications of nutritional genomics are multiple. With personalized assessment some disorders (diabetes, metabolic syndrome) can be identified. Nutrigenomics can help with personalized health and nutrition intake by assessing individuals and make specific nutritional requirements. The focus is in the prevention and the correction of specific genetic disorders. Examples of genetic related disorders that improve with nutritional correction according to US national centre for Biotechnology, are obesity, coronary heart disease (CHD), hypertension and diabetes mellitus type1. Genetic disorders that can often be prevented by proper nutritional intake of parents include spina bifida, alcoholism and phenylketouria.

Nutrigenomics

Nutrigenomics aims to identify the effects of several nutrients, including macronutrients and micronutrients on the genome (Rivera, *et al*., 2005) and explores the interaction between genes and nutrients or food bioactives and their effects on human health (El- shoemy, 2008). The influence of nutrients on the transcription activity, gene expression, and heterogeneous response of gene variants is also referred to as "Nutrigenomics".Nutrigenomics also describes the use of functional genomic tools to study a biological system to understanding of how nutritional molecules affect metabolic pathways and homeostatic control. This branch of science will reveal the optimal diet form within a series of nutritional changes, whereas Nutrigenetics will yield critically important information that assist clinicians in identifying the optimal diet for a given individual, i.e. personalized nutrition (David, *et al*., 2005). Transcriptomics, proteomics, and metabolomics are also technologies that apply in Nutrigenomics research (El- shoemy, 2008).

Nutrigenetics

Nutrigenetics points to understanding how the genetic background of an individual impact to the diet (Rivera, *et al*., 2005). The study of gene-nutrient interaction is a developing area of science. This idea that adverse diet/genome interaction can cause disease is not new and the unsuitable diet for any individual genotype could be a risk factor for monogenetic and polygenetic disease (Kaptur, *et al*., 2004). Genetic polymorphisms can influence response to environmental elements, such as enzymatic activities changes that affect circulating concentrations and ultimately the effectiveness of chemicals and their metabolites Furthermore, metabolic disorders are other examples of influence of the genetic variations to diet such as PKU, defects associated with long chain fatty acid oxidation, iron absorption (haemochromatosis), which can be reasonably well managed with dietary restrictions.



Effects of diets on genes

Several genes within our genome are known to influence the metabolism of nutrients (Goni, *et al.,* 2015). The main genes, whose variants are related to body weight loss in response to hypocaloric diets and/or physical

activity programs in adults, are involved in the regulation of lipid metabolism and adipogenesis; others are related to carbohydrate metabolism, energy intake and expenditure, and the circadian system (Goni, *et al.,* 2015). Research is growing at a fast pace, acknowledging that gene–diet interactions play an enormous role in human health and are typically only partially assessed. Healthcare providers, on the other hand, receive scarce knowledge of these interactions, thus jeopardizing the application of precision nutrition (Joffe, et al., 2020). At the same time, market initiatives have been developing to fulfill the market's increasing interest in prevention through nutrition in order to optimize people's health. This Special Issue on the effects of gene–diet interactions in human health includes six original research articles showing data that will bring readers closer to the state-of-the-art developments in the field of nutritional genomics. (Rivera-Iñiqguez et al., 2022) studied how a nutrigenetic intervention impacted emotion, rewarding behaviors, and self-efficacy in an exploratory pilot study in Mexico, which is a topic that has not yet been thoroughly assessed. They included twenty-eight individuals during a 6-month intervention. The authors concluded that when dietary advice is culturally acceptable and genetically compatible, it can reduce negative emotions and unhealthy eating behavior while increasing self-efficacy. These results open a wide new field of recommendations that psychologists and therapists could use to treat people with unhealthy and emotional eating behaviors.

Diet-Gene Interaction in certain diseases

Diabetes mellitus

Diabetes mellitus (DM), a group of metabolic diseases, results from defects in insulin secretion and insulin activity or both which is characterized by hyperglycemia. (Georgoulis, *et al*., 2014) reported that due to this metabolic disease DM, various organs like blood vessels, heart and kidneys are dysfunction and/or failure, and nowadays this disease is considered a global burden. The International Diabetes Federation's recent estimates indicate that 8.3% of adults (382 million individuals) have diabetes, and the number of individuals with this disease is expected to rise beyond 592 million in less than 25 years (IDF, 2013). DM fall into two broad pathogenetic categories: type 1 and type 2 DM known as T1DM and T2DM, respectively. The epidemic global obesity noted by Prentice, 2006 he stated that virtually T2DM will be a major health issue in the world create a major drain on health budgets. Individual with obesity increases the risk of developing the disease DM by at least 10-fold as compared to normal one. In developing countries, peoples are shifted their lifestyle from traditional diets system to modernized fast food eating habit which include frequent consumption of red meat, refined carbohydrates and saturated fats is leads to obesity (Popkin, 2001). The insulin hormone, which is an important controller of glucose and fat metabolism is secreted from β-cells of pancreas. Irregular secretion insulin is observed in both the cases i.e. obesity and T2DM. Glucolipotoxicity is the results of high sugar and saturated fatty acid in diet on regular basis and it negatively controls the secretion of insulin of from the β-cells, and results hyperglycemia and hyperlipidemia.

Cancer

Cancer is a multiple stages process in which gene expression, and protein and metabolite function begin to run aberrantly. In today's genomic era, the cellular events which intercede the activation of carcinogenesis upon modulation by dietary factors, has led to flow of significant information which helped in understanding of this disease (Anderle, *et al*., 2004). Cancer susceptibility may increase due to inherited mutations in genes. Gene diet interaction may increase the risk of developing cancer. Endogenous reactions, such as oxidations or from exogenous agents, sunlight exposure (skin cancer), such as tobacco smoke (lung cancer), aflatoxin (liver cancer), and relatively high doses of ionizing radiations (many types of cancers) induces cancer. It is mandatory to have communication between nutrition, metabolism, and gene expression for upholding body homeostasis. Human genome and nutrition jointly interacts to do the same. Individual's health condition and susceptibility to disease may get affected due to this (Gregori, *et al.,* 2011). Nutrient regulates transcription factors at the

molecular level which then modifies the gene expression (up or down), consequently to adjust the metabolic responses.

Diet is a blend of protective, carcinogenic, and mutagenic agents all together and are metabolized by the enzymes of biotransformation process. Risk of developing cancer can be modified through genetic polymorphisms that change protein expression or the function of these enzymes. Foods ingested by humans are proposed to contain more than 25,000 (Komduur, *et al.,* 2011) Role of different bioactive food ingredients in cancer pathogenesis has been studied and found that, among these, more than 500 types of bioactive food ingredients is proved to be possible predisposing agent.

Conclusion

Nutritional genomics elucidate the interaction among nutrients, metabolic intermediates, and the mammalian genome. The response to bioactive food components is dependent on genetic background (Nutrigenetics effects) that can influence absorption and metabolism targets or sites of action. Likewise, the response to food components depends on DNA methylation and other epigenetic events. The ability of bioactive food components to influence gene expression patterns (nutrigenomics effects) is also a factor in determining the overall response. Finally, bioactive food components may influence protein synthesis, degradation, and posttranslational modification. Understanding the interrelationships among human genetics diversity, genome function, and dietary components will enable precise manipulation of genome function and stability throughout the life cycle for optimal human health and disease prevention.

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