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A RECENT REVIEW ON NUTRIGENETICS AND NUTRIGENOMICS: CURRENT APPROACHES AND FUTURE ENDEAVORS

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ABSTRACT
The disciplines of nutrigenetics and nutrigenomics offer an in-depth knowledge for enhanced dietary counseling and recommendations to individuals, genetic subgroups, ethnic groups, and the general population as a whole. Thorough understanding of nutrition, biochemistry, genetics, Omic technologies has led to the application of preventative techniques for health improvement, preventing the onset of diseases, and reducing their severity. Research publications on Google Scholar, PubMed, Sci Hub, and the HEC digital library were examined. 87 publications that were pertinent to the topic were included in this study. The basic idea, technical terms, and technological advances of nutrigenomics and nutrigenetics are illustrated in this review, along with how this rapidly advancing knowledge can be applied to improve health, manage, and prevent disease, as well as the potential effects of changing nutritional and dietetic practices. As preventive medicine and nutrition grow more and more intertwined, it is crucial that dieticians, medical professionals, and geneticists receive the required training in the area of nutritional genomics in order to make the best use of its ideas and practices. Education must also be included in allied health courses so that students can teach the public about using omics technologies to improve healthcare.

Key words: nutrigenetics, nutrigenomics**,** dietetics, nutrition

INTRODUCTION

Human health is significantly influenced by diet and nutrition (Visioli *et al.*, 2022). Growth is affected by diet and nutrition throughout a person's life. The fact that normal growth is hampered by inadequate consumption of both macro- and micronutrients serves as evidence of this (Inzaghi *et al.*, 2022). In addition to having an effect on physical health, nutrition and dietary habits also have an effect on mental health (Elshahat *et al.*, 2023). Diet and nutrition status are two such challenges, both of which serve as important factors in determining one's well-being and state of health (Ishikawa and Yokoyama, 2023). It is not a new idea that one's diet has an impact on their health. Furthermore, it is widely acknowledged that everyone requires a different amount of nutrients. Numerous studies have concluded that the two main determinants of one's health and illness are the environment and diet (both quality and quantity) (Lal *et al.*, 2022).

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Nutrient-gene interaction

The health and disease status of a person are impacted by the bilateral interactions between genes and diet (Ahluwalia, 2021). The genetic makeup of a person best describes its characteristics with respect to development and growth. It is preserved and passed down through the generations while maintaining an ongoing balance with other non-living elements and the environment. Diet and nutrition are the main environmental influences. At essentially three separate levels—genome, proteome, and metabolome—diet/nutrition interacts with and modifies a variety of underlying molecular pathways critical to diverse physiological activities (Bahinipati *et al.*, 2021).

The genetic profile of an individual in reaction to a particular food is shown by the association among specific nutrients and connected genes involving a certain organ. For certain groups with identical genotypes, the relationship of genes with diet creates chances for the inclusion of bioactive substances. Determining the impact of nutrients and how they are utilized in the body is made easier by the modest variation in human genetic material (Lal *et al.*, 2022). Throughout their lifespan, genomes are subjected to a range of external stressors, including nutrition, and nutrients are typically recognized as the most potent environmental stimulant. Therefore, dietary minerals and phytochemicals play a substantial role in gene expression. Unbalanced diets can cause gene-nutrient interactions that increase the risk of developing chronic degenerative illnesses by having a direct or indirect impact on the expression of the genome (Vyas, 2022). For instance, genes that impact the metabolic process of vitamin B12 may result in cardio metabolic characteristics that are crucial in the setting of obesity (Matusheski *et al.*, 2021). Studies on vitamin A, vitamin D, and fatty acids, show that these vitamins directly cause nuclear receptors to activate and drive transcription of genes. As an example, distinct genetic variations in metabolic pathway-related genes impact the consumption and use of specific micronutrients (Kiani *et al.*, 2022).

Recent research has shown that nutrients modify gene and protein expression, which in turn affects organismal and cellular metabolism. Human health and fertility can be affected by nutrition, a modifiable fundamental element that can interact with both the genome and epigenome. Particular genetic variations can affect how the body reacts to food components and nutrient needs, and the diet itself has the power to control the regulation of genes (Franzago *et al.*, 2020).

Genes have long been regarded as the physically fixed blueprints of our bodies. And while genes are important in defining some aspects of our health in advance, a more recent branch of science termed as epigenetics has taught us that genes may be altered in response to the environment. Nutrition has a huge impact on the way genes are turned on or off since what we consume plays a significant role in determining our internal environment (Christie and ND, 2023). .By altering the chromatin structure (methylation of DNA and histone modification), non-coding RNA, signaling cascades, activating transcription factors, or by binding directly to nuclear receptors, bioactive ingredients in the diet control gene expression (Mierziak *et al.*, 2021), (Tan *et al.*, 2022).

The complexities of our eating environment, our living conditions, our exercise levels, as well as social, economic, and other personality characteristics interact with our genetic and epigenetic profiles, gut microbiota and metabolome, and our circadian rhythms to define the likelihood of

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diseases. To create individualized nutrition advice for people and increase treatment effectiveness, one must have a solid understanding of the interrelationships among the aforementioned components (Kalea and Klimis-Zacas, 2021).

The activity of genes involved in the metabolism of some of the most often consumed nutrients by humans is influenced by common single nucleotide polymorphisms (SNPs), which have been shown to have large population-based changes in allele frequency. Additionally, evolutionary studies demonstrate that humans genetically adjusted to their ancestral diets and regional settings and also genetically drifted apart, resulting in the patterns of sequence variation that are present globally (Mullins *et al.*, 2020).

Understanding how nutrients affect the regulation of genes might therefore further our understanding of metabolic illnesses and perhaps lead to the development of novel therapeutic strategies based on dietary modifications and a patient's unique genetic profile (HaroMarrero and Relat, 2019).

OMICS

Methods for examining biological systems have been significantly transformed by "OMICS," which is defined as examining and evaluating enormous amounts of data that depict the function and structure of a whole biological system at a specific level (Dai and Shen, 2022); (Radzikowska *et al.*, 2022).

In addition to interacting with dietary/nutritional factors, these omics technologies additionally involve genomics (polymorphisms and other structural genetic variants), epigenomics (DNA methylation, histone modifications, long non-coding RNA, telomere length), metagenomics (gut microbiota composition, enterotypes), transcriptomics (RNA expression patterns), proteomics (protein amounts), and metabolomics (metabolite profiles) (Radzikowska *et al.*, 2022). Molecular genetics, biology, and nutrigenomics, which concentrate on determining the genomic, transcriptomic, proteomic, and metabolomic consequences of both deficiencies in nutrients and toxicity, have been recommended to take the place of traditional epidemiological and physiological in nutritional research (Mishra *et al.*, 2022).

Nutrigenetics and Nutrigenomics: two sides of a coin

An emerging field of study called nutritional genomics, also known as nutrigenetics or nutrigenomics, aims to define the interactions between dietary consumption and the corresponding diseases with the genome of an individual (Koromina *et al.*, 2020). The contribution of nutrigenetics and nutrigenomics along with OMICS will provide insight into molecular analyses as well as the needs-based nutritional needs (Singh, 2023).

At the 4th International Conference on Nutrition and Fitness, held in Athens in 2000, Robert F. Murray, Jr. coined the term "nutrigenomics," defining it as "the study of the genomic basis for individuality or individual variability in the response to specific nutrients." The "new frontier of nutrition science" was referred to the next year, and it was anticipated to change both nutrition research and its application for clients via dieticians and nutrition specialists (Ferguson and Barnett, 2022).

Nutritional genomics, which includes both "nutrigenetics" and "nutriepigenomics," is the study of how dietary intake interacts with the DNA. It focuses on identifying the molecular mechanisms underlying both transient and long-lasting interactions between nutrients and health, and it may be a fruitful area of research to advance clinical and nutritional practices (Meroni *et al.*, 2020).

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While nutrigenomics examines how foods affect patterns in the genome and transcriptome, nutrigenetics investigates the impact of diet at the gene level. A notion of customized medicine that integrates nutrition and healthcare is feasible by carefully analyzing the relationship among patients' genomic profiles and their food consumption (Irimie *et al.*, 2019).

The study of nutrigenetics describes how genes influence nutrition, a phenomenon that is typically seen in conditions like lactose intolerance and phenylketonuria. To learn how different people react to food based on their genetic make-up, nutritional genetics integrates both the fields of nutrition and genetics. These minute changes control how nutrients interact with our bodies and how our systems process the food we consume. Individualized nutrition ties together this mutually beneficial interaction between foods and genes (Uthpala *et al.*, 2020).

With the help of high-throughput genomic technologies, nutrigenomics has been able to determine how nutrients affect how genes are expressed. While nutritional genetics (nutrigenetics) concentrate on the varied responses of gene variations to dietary components and nutrients. Application of nutrigenomics and nutrigenetics, along with system biology, produces customized nutrition as a relevant, specific advice for dietary modification, which will boost the motivation and maintain of those receiving the intervention (Meiliana and Wijaya, 2020).

It is crucial for future developments of both customized nutrition and precision healthcare, as nutrigenomics/nutrigenomics plays an integral part not just in addressing diseases and illnesses but also in improving health and wellness via basic and clinical research (Marcum 2020). Nutrition may be able to affect a patient's clinical result by influencing their particular epigenetic expression alongside their distinct genetic background, according to discoveries from the fields of nutrigenetics and nutrigenomics (Dallio *et al.*, 2021).

Goals of Nutrigenetics and Nutrigenomics

The objective is to identify diseases and health parameters that depend on nutrition. By examining proteomics and metabolic pathways, it is possible to examine food composition and quality in another significant field related to these disciplines(Dable-Tupas *et al.*, 2023). The main objective of nutritional investigations is to pinpoint the people who can benefit by dietary modifications and to come up with solutions for those who won't take to them. By altering dietary treatments based on a person's genetic composition, this is beneficial for both preventing and management of long-term illnesses (Bahinipati *et al.*, 2021).

Personalized Nutrition

In order to encourage dietary behavior changes that may have demonstrable health advantages, (de Hoogh *et al.*, 2023). The term "precision nutrition" refers to the process of analyzing physiological or pathological responses to specific food elements or diets, determining the biological impacts of foods and their affect on health, identifying people with particular dietary deficiencies, providing information on inter-individual modifications, or helping to create individualized dietary recommendations to promote optimal health based on specific phenotypes(Picó *et al.*, 2019). The vast majority of the research studies that were included used a biological justification for personalization; the methods to carry out these interventions were developed and supplied customized dietary recommendations on the basis of phenotypic or/and genotypic data. Some treatments, for instance, chose genotypes known to affect the likelihood of developing chronic diseases, such as the disclosure of HLA-DRB1, which can triple the chance of developing rheumatoid arthritis. Other research, however, focused on genotypes known to affect food metabolism, such as Apolipoprotein E, which controls lipoprotein metabolism and responds to saturated fat consumption (Livingstone *et al.*, 2022).

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The individualized nutrition approach: One size does not fit all. Regardless of acknowledged causal connections among health and diet outcomes, traditional nutritional interventions designed aimed at lowering the load of persistent illnesses have resulted in a limited impact. This can be due to a combination of factors, including the assumption that individual nutritional needs as well as responses resemble the average response found in study populations (Berciano *et al.*, 2022).

Nutritional Epigenomics

The third subfield of nutritional genomics is nutritional epigenomics. This field examines how nutrition affects the epigenetic systems that control gene expression and function. DNA methylation, histone modifications (acetylation, phosphorylation, and methylation), and noncoding RNA activity are all included in the field of epigenetics (Corrêa *et al.*, 2020).

"The study of the entire array of epigenetic changes occurring in a cell or within a tissue at a specific time" is the definition of epigenomics. The term "epigenome" refers to a set of chemical substances that alter or identify the genome in a way that can be used to identify what a cell is capable of doing and when and where it is able to do it. 'Epigenetic marks' are the name given to these markings. These "epigenetic marks" are transferred from a cell to another during cell division, and as a result, they are passed down from one generation to the next. The phenotype will be determined by these signals, which are modified by genotype in the external environment, such as the environment, diet, and medications (Alam *et al.*, 2019). For many years, the term "epigenetics" has been technically defined as a shift in a gene's or trait's state of expression that does not entail a mutation but is nonetheless passed down across generations (preferably through a mitotic divisions) because of the absence of a signal or incident that caused the alteration (Dhar *et al.*, 2021).

DNA Methylation and Post-translational Histone Modification

DNA methylation, one of the most prevalent and well researched epigenetic alterations, is crucial for healthy cellular biology and development. Transcriptome changes and cellular pathways dysregulation are caused by global changes in the DNA methylation pattern (Dhar *et al.*, 2021), (SkvortsovaStirzaker and Taberlay, 2019);. The epigenetic alteration that affects gene expression that has received the most research is DNA methylation (Pinho and Maga, 2021).

The recruitment of proteins with methyl-binding domains and additional transcriptional co-repressors as a result of this reversible DNA alteration causes the silence of gene expression. The promoter regions of a broad range of human genes have an aggregation of CpG dinucleotides, or CpG islands. As a result, modifications to the DNA methylation pattern influence the transcription of several genes (Swiatowy, 2021); (Yang *et al.*, 2021). Specifically within CpG dinucleotide settings (5-methylcytosine; 5mC), cells use DNA methyltransferase (DNMT) enzymes that catalyze the binding of a methyl group to the fifth carbon location on cytosine (SkvortsovaStirzaker and Taberlay, 2019).

For example, DNA methylation in normal cells and cancer cells**:** Between healthy and cancerous cells, there are significant variations in the DNA methylation patterns that span the entire genome and include all gene regulatory components. The majority of CpG sites in the normal genome carry 5mC, and CpG island regions and distal enhancer elements are resistant to DNMT action. Cancer cells exhibit the anomalous presence of punctuated elevations in DNA methylation across enhancers and promoters, as well as a general loss of 5mC. Together, these changes in distribution result in the suppression of tumor-suppressor genes and a parallel rise in the expression of oncogenes, that are responsible for carcinogenesis (SkvortsovaStirzaker and

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Taberlay, 2019). Cardiovascular disease, risk for mortality, depression as well, and physiological processes including chronic inflammation have all been linked to DNA profiles (McDade, (2019).).

Histone proteins undergo covalent post-translational modification (PTM), which mostly comprises of methylation, phosphorylation, ubiquitination, and ADP ribosylation. By changing the chromatin structure, the PTMs that have occurred in histone proteins can influence gene expression (RamaziAllahverdi and Zahiri, 2020). The tails of histones are mainly involved in DNA and histone-histone interaction and can be covalently modified through post-translational modifications. Histone modifications are generated by enzymes which includes families of histone acetyltranferases (HATs), histone methyltransferases (HMTs) and histone ubiquitinating enzymes (Kunadis *et al.*, 2021).

Defects in the PTMs pathway have been linked to the onset and progression of numerous human diseases, including cancer, heart failure, autoimmune diseases, inflammatory diseases, neurodegenerative disorders like Parkinson's disease, Alzheimer's disease, and Huntington's disease, coronary artery disease, type 2 diabetes, asthma, multiple chronic lung disorders, and inflammatory bowel disease (Lin *et al.*, 2022), (RamaziAllahverdi and Zahiri, 2020).

Nutrigenomics and Biomarkers

The importance of the medicinal properties of food has increased as the notion of a biomarker in nutrigenomics has developed as a key method to prevent disease and slow down the aging process (Pal, 2022). An objectively measurable property known as a nutritional biomarker can be used to assess the nutritional status of various biological samples in relation to the consumption or metabolism of dietary components (Picó *et al.*, 2019). It has been suggested that biochemical measures of nutritional stress, often known as biomarkers, can provide more precise information about consumer nutrition. For instance, studying folate, iron, and vitamin B12 as well as copper and zinc might help determine possible nutritional reasons of anemia (JonesShafer and Frost, 2023).

Single nucleotide polymorphisms, or SNPs, are the main type of genetic polymorphisms that are used to identify genetic biomarkers. The lactase polymorphism 13910C>T (rs4988235), which is found on the MCM6 gene but affects the lactase gene (LCT), is a well-known example. It has a high correlation with lactase production persistence and, consequently, lactose tolerance or intolerance. Circulating lipid profiles and urea levels in the blood or urine are further biomarkers for assessing a person's nutritional condition (Picó *et al.*, 2019).

Nutrigenomics is the study of the effects of diet on the whole genome (a person's complete genetic makeup, involving epigenetic modifications) and its stability (DNA damage at the chromosomal and molecular levels), transcriptomics (the expression of RNA and micro-RNA), proteomics (the expression of proteins), and metabolomics (the changes in metabolites), all of which can be studied separately or jointly to diagnose a person's health status and/or disease trajectory (Batool *et al.*, 2022); (Rubio *et al.*, 2023). The only one of these biomarkers that is clearly indicative of fundamental illness is DNA damage, which may be reduced by encouraging the death of genetically abnormal cells or by slowing the rate at which DNA damage accumulates (Alhmoud *et al.*, 2021).

The following are a variety of complementary methods for diagnosing DNA damage: Damage to single bases includes: (i) DNA adducts, which are formed when a hydroxyl radical is added to guanine as a result of oxidative stress; (ii) abasic regions in the DNA sequence, which can be detected using an aldehyde-reactive probe; (iii) DNA strand breaks, which are frequently

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detected using the Comet assay; (iv) shortening of the telomere (as measured by terminal length of restriction fragment analysis(v) Mitochondrial DNA damage (typically assessed as deletions or base degradation within a circular mitochondrial DNA sequence), (vi) chromosome breakage or loss (typically assessed using micronucleus cytome tests or metaphase chromosome analyses), and (v) chromosome breakage or loss. On the basis of studies related to disease and nutrients, the application of damaged DNAs to serve as biomarker was recently verified at multiple levels (Mondal and Panda, 2020).

Dietary signals and nutrient sensors

"Nutritional signaling" is a catch-all term used to describe a variety of pathways of cell signaling that are influenced by food availability. The molecular architecture of the nutrients' molecules offers guidance on how to activate specific signaling pathways to arrive to the desired location. Signaling cascades are activated when nutrient levels change, changing critical cellular functions like metabolic activities, proliferation, secretion, and autophagy (Shah *et al.*, 2021); (Wen *et al.*, 2022).

An experiment using the model organism Drosophila showed that every nutrient has a variety of targeting locations with different affinities and specificities. More than any other model organism, it was discovered that drosophila possesses adipose-like tissues and a lipid transportation system, which are similar to those in humans in terms of obesity and associated disorders (Ugrankar *et al.*, 2019).

In addition to controlling the expression of specific genes, activator receptors operate as sensors for nutrients. These target genes are engaged in a wide range of metabolic processes, including the oxidation of fatty acids, the generation of ketone bodies, the synthesis of glucose from non-carbohydrate precursors, etc. Fasting causes adipose tissue to release free fatty acids (FFAs), which makes PPAR crucial. In the liver, FFAs undergo partial or complete oxidation. By attaching to the promoters of PPAR, these fatty acids regulate the expression of genes. Elevated fatty acids act as PPAR agonists by enhancing PPAR signaling, which stimulates the production of gluconeogenesis and glucose. A greater comprehension of the role of PPAR in type 2 diabetes may aid studies relating obesity and type 2 diabetes (CarielloPiccinin and Moschetta, 2021); (Kim *et al.*, 2022).

Experimental Approaches and Technologies Used in Studying Nutrigenetics and Nutrigenomics

To better understand nutrition science and implement interventions for better health, several experimental strategies, nutrigenomics and nutrigenetics research advancements, and interventional designs for studies have been developed (Otero and Bernolo, 2023).

Scientists and nutrition practitioners must not only choose methods that are acceptable for a particular experiment design or line of inquiry, but also ensure that the data interpretation is appropriate in order to effectively tackle the varied and intricate nature of nutrigenomic approaches and output data. (Chirita-Emandi and Niculescu, 2020). In order to investigate the function of particular substances and their regulatory route in the biological system, the omics study might be utilized to identify the functional genes, RNA, proteins, and metabolite products. For the compilation, evaluation, as well as interpretation of the vast amounts of data acquired through omics technologies, bioinformatics methodologies could be applied. Such high-throughput datasets need to be acquired, managed, stored, retrieved, and analyzed in bioinformatics (Prakash *et al.*, 2020)

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Bioactive food ingredients change the amounts and functions of proteins and gene expression, which has a variety of beneficial impacts on human health. Global analysis and integration can be used to gather functional information from metabolites, protein levels and post-translational modifications, gene variations and expression, epigenetic regulation, and epigenetic regulation of proteins. By combining the study of food and nutrition with the use of omics technologies, foodomics enables the examination of cellular processes, functional processes, and molecules that are involved in it, along with the determination of targets for bioactive compounds helpful for advancing the creation of nutritional intervention plans, and the identification of biomarkers linking nutrition and health (Ortea, 2022).

The numerous 'omic' technologies must be taken into account in human studies along with information gathered on dietary, lifestyle, clinical, physiological, demographic, and environmental aspects. Understanding the function of gut microbiota of the gut and the relationships that develop among the gut microbiome and host genome is of great interest. The gut microbiota interacts directly with host signaling cascades, affects the epigenetic landscape, induces differential splicing, and remodels host chromatin. The link connecting the microbiome of the gut and host expression of genes may one day be better understood thanks to cutting-edge methods such as single-cell sequencing of RNA and organoid creation. We get insight into the physiological processes necessary for promoting the vast cross-kingdom connections and, ultimately, human health by combining the microbiome and host gene expression (Nichols and Davenport, 2021). After engaging a particular receptor, which strongly binds to DNA and intensely promotes gene expression, nutrients act as transcription factors. For instance, consider the lac operon that is by default disabled in cells (such as bacterial cells) that do not contain lactose. Since the RNA polymerase is prevented from transcription by the lacI inhibitor gene's binding to the promoter region, no transcription of DNA occurs (Jabeen *et al.*, 2023).

Single Nucleotide Polymorphism

There is growing understanding of the complexity of genetic diversity throughout the human genome. The majority of sequence diversity in the human genome occurs as single nucleotide polymorphisms (SNPs). SNPs, or single nucleotide polymorphisms, can be related to the genes that are linked to a number of complicated diseases, including Alzheimer's, schizophrenia, diabetes, cancer, and blood pressure. They may also relate to the genes that are linked to other complex diseases, such as heart disease, migraine, cancer, and blood pressure. These SNPs typically occur into a gene or at a regulatory region close to a gene, and they may change how the gene functions to have a more direct impact on disease (Kaur *et al.*, 2019). Single nucleotide polymorphisms, or SNPs, are the basis for the majority of genetic biomarkers. One well-known instance involves the lactase polymorphism 13910C>T (rs4988235), that is found on the MCM6 gene but affects the lactase gene (LCT). It has a substantial correlation with the persistence of lactase synthesis and, consequently, to the ability to tolerate or intolerant to lactose (Picó *et al.*, 2019). Because SNPs are primarily found in the important gene regulatory systems involved in the transportation and metabolism of glucose or lipids, they also have a substantial impact on the onset and progression of obesity as well as associated metabolic illnesses (Chen and Chen, 2022).

With the development of new computational approaches and statistical tools that can analyze and filter such data, nutrigenetics and nutrigenomics are being studied in great detail thanks to the incorporation of Omic innovations and applications. Then, advances in next-generation sequencing (NGS), high-throughput platforms, mass spectrometry, and bioinformatic tools

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enabled the fields of epigenomics, RNA (transcriptomics), metabolites (metabolomics), proteins (proteomics), and (microbiomics), with the capability to combine different categories of 'omics' data ('multi-omics' or'system biology'). The study of complex human diseases has been revolutionized by the development of modern "omics" methodologies and techniques that offer an unprecedented genome-wide perspective of genetic diversity, gene expression, interactions with microbes, and environmentally responsive epigenetic alterations (Cassotta *et al.*, 2021); (Pinu *et al.*, 2019).

Numerous studies have unmistakably demonstrated that nutrition influences how genetic information is expressed at various stages of transmission of signals, gene regulation, as well as through changes in the structure of chromatin and protein function. Diet impacts gene expression through altering epigenetics (such as methylating DNA). Alterations in gene expression profiles might represent molecular 'signatures' that reflect exposure to particular nutrients. This information can be used by dietitians to better understand how the patient's diet interacts with their genome, which in turn facilitates further planning and intervention (Ilango *et al.*, 2020).

Precision nutrition requires dietary assessment, which is the assessment of each person's nutritional intake. The two most popular techniques are food-frequency questionnaires (FFQs) and 24-hour recall (24HR). These techniques are also employed in the research of nutrigenomics and nutrigenetics. The development of individualized nutrition and medicine as well as the voyage from the My pyramid to the My plate were all made possible by the cutting-edge methods of nutrigenetics and nutrigenomics (Mattes *et al.*, 2022).

Nutritional genomics and Chronic Diseases

More than 60% of deaths worldwide occur from non-communicable diseases (NCDs), and if current trends continue, this number is expected to rise tenfold by 2025. The science of nutrition has recently gained importance for everyone's health as well as physical fitness and appearance. In actuality, nutrition has a significant role in both prevention and treatment of nutrition-related non-communicable diseases (NR-NCDs), which include diseases such as diabetes, hypertension, obesity, cardiovascular disease, metabolic syndrome, and malignancies (Soldati *et al.*, 2015).

In the vast majority of nations around the world, cardiovascular disease (CVD) continues to be the leading cause of mortality. Numerous SNPs linked to CVD risk factors have been discovered by GWAS and the traditional gene candidate technique, enabling focused research to incorporate nutritional information. According to the findings, people who are homozygous of the C allele of an SFA-rich diet-related polymorphism found in the apolipoprotein A2 gene promoter (APOA2 rs5082) have considerably higher BMIs than T allele carriers. The same gene-diet interaction was also linked to greater LDL-C levels and a higher LDL-C/high-density lipoprotein-cholesterol (HDL-C) ratio in those with diabetes, according to a more recent study. Therefore, our findings imply that a low-SFA diet will be advantageous for people who have the CC genotype for this APOA2 SNP in order to prevent hypercholesterolemia (Desjardins and Vohl, 2023).

Cardiovascular illnesses, comprising strokes and coronary coronary artery disease (CAD), is now one of the main causes of death, in addition to the trend of increased longevity and better health. Over seven million individuals are thought to pass away from the illness each year. According to biology and epidemiological data, CAD is a complicated condition brought on by the interaction of hereditary and non-genetic factors: (1) genetic diversity; (2) physiologic variables, including hypertension, diabetes, obesity, and more; (3) smoking, drinking, dieting, exercising, and other unhealthy lifestyle choices; (4) exposure to environmental pollution; and (5) interactions between various factors. The risk of CAD is greatly influenced by genetic factors. First genetic

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risk variation for CAD was found on chromosome 9p21 in 2007 and replicated. In order to find CAD-related susceptibility loci, an increasing number of extensive investigations have been carried out in certain populations. Studies were routinely publishing the results of 35 newly found susceptibility loci linked to CAD risk utilizing the trans-ancestry meta-analysis up until recently (Koyama *et al.*, 2020). Over 200 loci associated with CAD susceptibility have been discovered in the last 20 years (Hartiala *et al.*, 2021).

These risk variations have been discovered to influence the risk of CAD through a variety of pathogeneses and mechanisms, including lipid metabolism (e.g., PCSK9, LPA, APOB, APOE, and BCMO1), alcohol metabolism (e.g., ALDH2), adipose tissue formation (e.g., ADCY3), blood pressure (e.g., SH2B3), and other pathogeneses and processes. For instance, the proprotein convertase subtilisin/kexin type 9 (PCSK9) coding protein may affect the receptors for low-density lipoprotein cholesterol (LDL-C), and PCSK9 inhibitors may be used to lower plasma LDL-C levels and affect the occurrence of CAD. A crucial oxidase linked to alcohol metabolism in cell mitochondria, aldehyde dehydrogenase 2 (coded by ALDH2) acts as a preventative against cell damage brought on by oxidative stimulation. By decreasing the development of vascular endothelial inflammation, it can also slow the progression of cardiovascular illnesses. These newly identified genetic risk variants may be utilized to develop genetic risk scores, which would enhance risk prediction beyond conventional risk variables (Ye *et al.*, 2022).

Despite the intense discussion, obesity is seen as a result of unhealthy eating habits and inactivity, especially when considered in the context of a larger "obesogenic" environment. Newer evidence, meanwhile, increasingly supports the idea that genetics can play a causal role as well. More than 50 candidate genes, including the melanocortin-4-receptor (MC4R) gene for obesity, have recently been discovered by genome-wide association analyses, providing evidence in favor of this (Asghar and Khalid, 2023). Numerous common SNPs have been linked to obesity by candidate gene and genome-wide association studies. The greatest common genetic predictor of obesity now known is the fat mass and obesity-associated gene (FTO) variations, which have been consistently linked to obesity features in a variety of populations. FTO has so far demonstrated the highest correlation between BMI and weight, with the FTO SNP increasing the probability of obesity 120–132 times in Europeans and 125 times in Asians. The FTO mutation, rs9939609, raised the risk of obesity 115 times, which is comparable to a rise in BMI of 030 kg/m2 per effect allele, according to the most recent meta-analysis of data comprising eight Indian studies (Vimaleswaran, 2020).

A recent study investigated the relationships between 15 gene variations and cardiometabolic variables and tested if dietary intake may change these relationships. 110 Minangkabau women from Padang, Indonesia, with a body mass index (BMI) of 25.13 4.2 kg/m2 and ages ranging from 25 to 60 were included in the study. On the basis of 15 SNPs related to cardiometabolic illness, a genetic risk score (GRS) was created. There was a strong correlation between GRS and BMI, with those with 6 or more risk alleles having greater BMI than those with 5 or fewer risk alleles. A substantial relationship between GRS and protein intake was also shown to affect triglyceride levels and waist circumference (WC). The WC and triglyceride concentrations of women with six or more risk alleles were substantially lower than those of women with five or less risk alleles among those who consumed less protein (13.51± 1.18% of the total daily energy intake) (Alsulami *et al.*, 2020).

Half of pregnancies globally and almost 40% of pregnancies in Brazil are impacted by excessive gestational weight gain (GWG). Preterm birth, cesarean section, large-for-gestational-age

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newborn, macrosomia, neonatal distress, and neonatal abnormalities are all more likely in pregnant women with pregestational diabetes mellitus (PGDM) who have excessive GWG. Pregnant women with pregestational diabetes mellitus who were 18 years of age or older, less than 28 weeks along, carrying a single fetus, abstaining from alcohol, tobacco, or drugs, testing negative for sexually transmitted diseases like syphilis or genital herpes, eating disorders, or psychiatric illnesses like anxiety or depression were the participants in a study. The A allele carriers for the FTO gene and the ADRB2 gene, respectively, had a risk of reaching GWG sooner than double that of the TT and GG genotypes in a sample of 70 pregnant Brazilian women with pregestational diabetes mellitus (Santos *et al.*, 2022).

Type 2 diabetes (T2D) is a diverse collection of metabolic illnesses brought on by the combination of many environmental variables and genetic susceptibility. fifty people were included in a case-control study on the population of Egypt. Diabetic patients made up half of them. From the patient database, complete demographic and clinical information was gathered. After that, peripheral whole blood was drawn from each participant in the study and placed on EDTA. Serum samples were also taken, and then the following laboratory tests were run: Blood sugar one at random. (2-Glycated Hemoglobin; HbA1C). 3- Genotyping of the TCF7L2 gene polymorphism using real-time PCR (Polymerase Chain Reaction). Interleukin 6 (IL6) ELISA assay.In diabetic patients, the current study found a statistically significant positive link between BMI and measures of glycemic control (random blood glucose and HbA1c); this means that the higher the BMI, the worse the glycemic control. According to the study's findings, elevated levels of inflammatory markers are a hallmark of obesity. They attributed this correlation to the growth of abdominal fat mass in obese men, that secretes IL-6 in an endocrine mechanism and causes an increase in hepatic CRP production as well. In a related study, it was revealed that one of the main adipokines, IL-6, increased with obesity, particularly visceral obesity in males (Elghazawy *et al.*, 2020).

A quarter of the world's population is now known to have non-alcoholic fatty liver disease (NAFLD), which has a prevalence rate that is comparable to that of obesity. Comparative proteome, photoproteomic, and lipidomic studies of the livers of mice fed a high-fat (HFD) and low-fat (LFD) diet were carried out. Six male mice were used in this investigation; they were housed in a humidity- and temperature-controlled environment with a 12:12 h light/dark cycle. Mice were given a chow diet containing 62.1% of their calories from carbohydrates (starch), 24.7% from protein, and 13.2% from fat from the time they were weaned until they were 5 weeks old.Mice were randomized at 5 weeks of age to either the low-fat diet (LFD, 10% calories from fat) or the high-fat diet (HFD, 60% calories from fat) for an additional 12 weeks. A variety of metabolic pathways that were considerably changed were discovered by an integrated proteome and lipidomic investigation of HFD-induced fatty liver tissues from animal models. The work demonstrated how fluctuations in lipid composition and amount are linked with changes in the liver's proteome and phosphoproteome, or vice versa, using this comprehensive multi-omic method (Kim *et al.*, 2022).

Nutritional genomics and Cancer Management

According to the findings, diet and exercise modifications can reduce the risk of developing cancer. About 5% of all cases of human cancer are caused by mutational events, while the remaining 95% are rare occurrences brought on by exposure to environmental and nutritional variables. According to the data, smoking accounts for roughly 25%–30% of all cancer-related fatalities, followed by food (30–35%), infections (15–20%), radiations, anxiety, physical activity,

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environmental contaminants, and other variables.Research has also shown that a number of bioactive food components, such as phytochemicals found in plants, zoochemicals found in animals and fish, such as conjugated linoleic acid as well as omega-3 fatty acids, accordingly, fungochemicals obtained from mushrooms, and bacteriochemicals produced by microbial fermentation, may alter sensitivity to malignancy. Apoptosis, differentiation of cells, inflammation, DNA alteration, and carcinogen metabolism are just a few of the many routes that nutrients play in (Parmar *et al.*, 2019).Because it is a leading cause of death worldwide, cancer has drawn the attention of many researchers. The level of expression of oncogenes is modulated by genetic and epigenetic changes, which causes the conversion of healthy, normal cells into cancerous cells. Nutritional deficiency has been found to play a significant influence in the development of the tumor (GairolaDubey and Bahuguna, 2023).

Single nucleotide polymorphisms and nutrigenetics in Cancer

When exposed to sunshine, a person's skin can produce vitamin D, a fat-soluble vitamin that is present in food. A dietary intake of vitamin D may not be necessary if there is sufficient ultraviolet radiation (UVB) in the wavelength range of 290-315 nm. A dietary dose of vitamin D is required to prevent skeletal illnesses that deteriorate bones, that include rickets and osteomalacia, since sufficient exposure to UVB isn't always achievable for a wide range of reasons.The 1,25-dihydroxy vitamin D3 (1,25(OH)2 D3), which serves as the physiologically active kind of vitamin D, is known to mediate the biological actions of this nutrient by regulating a number of target genes linked to cell proliferation and differentiation. This is known as the 1,25-dihydroxy vitamin D receptor (VDR), a nuclear hormone receptor. There are several VDR polymorphisms that are known, but only a small number of them have been demonstrated to have functional effects or affect how the body reacts to different dietary ingredients and illness risk.One specific VDR polymorphism, called Fok 1, causes the production of a VDR protein whose length is three amino acids longer compared to that of people who carry the non-variant F allele. According to reports, those with the Ff or ff genotype have a 51% and 84% higher chance of having colorectal cancer, respectively. It was discovered that people with the ff genotype compared to those with the FF genotype had an elevated risk with colorectal cancer that was more than twice as high. This polymorphism may therefore act as a prognostic marker for individuals that are going to benefit greatly from ensuring proper food intakes in the case of specific malignancies.Fok 1 F allele was also found as a defense towards prostate cancer only in males who receive enough vitamin D exposure, which further complicates the situation. It has been demonstrated through the examination of many demographic groups in regard to vitamin D status that this molecule possesses preventive qualities, particularly in relation to colon, neck and head, breast, ovarian, and prostate cancers. The emergence of prostate cancer, colon cancer, and breast cancer may be caused by vitamin D deficiency (Irimie *et al.*, 2019); (Chattopadhyay, 2020); (Dhanapal and Vimaleswaran, 2022).

MNSOD gene polymorphism and breast cancer risk

Manganese superoxide dismutase (MnSOD), a mitochondrial enzyme that is essential in the detoxification of oxygen species that are reactive, is an intriguing example that illustrates the fact that fruits and vegetables, which include a variety of dietary components, are also good sources of antioxidants. A valine to alanine alteration within the mitochondrial targeting region, which is expected to affect how the enzyme is transported into mitochondria, has been linked to an increased risk of breast cancer.It has been discovered that this link is more prominent in women who consume fewer fruits and vegetables. Risk for prostate cancer has also been examined in

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relation to MnSOD, genotype, and plasma antioxidant prediagnostic levels. Each inverse relationship between the plasma levels of selenium, lycopene, and a-tocopherol and risk of prostate cancer was modified by associations between prostate cancer risk and the antioxidant score (the sum of lycopene, a-tocopherol, and selenium status) and the MnSOD polymorphism.More so than for antioxidants evaluated separately, there was a higher association between the total antioxidant score and the MnSOD polymorphism. When genotype-specific analyses were performed on men homozygous for the variation A allele, high (vs low) antioxidant scores were substantially linked with: lower overall risk of developing prostate cancer (Sunil and Janghel, 2020); (Tuncel *et al.*, 2022).

Nutrition Impact on Epigenetics Modifications associated with Cancer

It is generally recognized that changes in the epigenome can alter how cancer develops and progresses. Tumors frequently undergo epigenetic reprogramming, and around 50% of all human malignancies contain alterations in genes coding epigenetic regulators. Even malignancies without these mutations show aberrant DNA or histone modification profiles, which coincide with alterations in chromatin modifier expression or activity. Further restrict or permissive chromatin may be produced as a result of epigenetic changes, which might affect a cell's ability to differentiate or adapt (IzzoAffronti and Wellen, 2021). The environment, especially nutrition, which is a source of metabolic substrates that affect the production of the cofactors and substrates for chromatin and RNA modifying enzymes, has a significant impact on epigenetics. Additionally, plants are a typical source of bioactives that can alter the functioning of these enzymes directly (Barrero *et al.*, 2022).

Through chemical connection with methyl product, epigenetic active substances, such as folate and cobalamin, have been shown to have an important function in the metabolism of DNA and maintenance of DNA methylation pattern. In a study, the p16 gene tumor suppressor, a significant cancer-linked gene with a constant DNA methylation silencer on its promoter, and dietary folic acid intake were positively connected. Age-related changes in p16 gene expression in old mouse colons are accompanied by a contemporaneous decline in DNMT expression. Furthermore, it was noted that low folate consumption is associated with hypomethylation, which raises the risk of pancreatic and colorectal cancer. Furthermore, it has been demonstrated by epidemiology that low levels of folate reduce DNA methylation capacity, which is connected to the development of cancer. This suggests that changed epigenetic processes may be used to control folate's anticarcinogenic characteristics (Adetunji *et al.*, 2022).

OBJECTIVES

The goal of this review was to give an overview of:

1. The fundamental ideas, terminologies, and technology underlying nutrigenetics and nutrigenomic research.
2. How to use this information to promote health and treat and prevent disease.
3. The possible impact of such knowledge on how nutrition and dietetics are practiced, as well as the consequences of such a change.

METHODOLOGY

Nutrigenetics and Nutrigenomics: Viewpoints on the Current Status and Applications in Nutritional Research and Practices were the focus of the review. In order to compile pertinent information for this review over the course of four months, research publications on Google Scholar, PubMed, Sci Hub, and the HEC digital library were examined. 87 publications that

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were pertinent to the topic out of more than 250 that had been studied were included in this study.

DISCUSSION

This review clarified the relationship between diet and nutrition and how it links to the human genome and the way it can impact a person's general health. The study of nutritional genomics helps us to better understand how nutrition, health, and the genome are related in this regard. Numerous research provided evidence in favor of the hypothesis that genetic activity and its expression as well as health are influenced by nutrition. Mother Nature has essentially genetically identically shaped all people, yet it takes only a 0.1% variance to distinguish one person from another in terms of phenotype, individual vulnerability to sickness or health, and different dietary responses. Strangely enough, some people may be at risk by following the same diet while others may benefit from it. Also, while some diets operate as potential disease-inducers, others may control genes to aid in the preservation of health. In order to keep a balance between health and disease and to provide a healthy life, tailored nutrition and diet recommendations can be produced according to information regarding individual nutritional requirements, nutritional status, and genotype. A major obstacle is that an individually tailored nutrition systemic approach is a pipe dream in a large country like ours where individuals are still struggling to meet their fundamental necessities. How to address this public health issue is currently the most important query. Although the nutrigenomics technique is the best and only option, it will not be as affordable for the general public to use. Additionally, it is a very challenging task to manage the massive population with nutritional assistance as it will demand enough qualified personnel in addition to cutting-edge lab facilities. Public health awareness campaigns can currently act as an important substitute in a variety of ways to prevent individuals from these diseases in a more general sense. It will primarily concentrate on the early detection of those who are at risk and the appropriate intervention, such as weight loss, dietary adjustments, and increased physical activity, to assist avoid or at minimum delaying the onset of dietary disorders.

CONCLUSION AND RECOMMENDATIONS

The only method that can guarantee that the knowledge produced by nutrigenetics and nutrigenomics science is appropriately applied and examined is the evidence-based approach, Given that nutritional interventions are incapable of being recommended in a manner that is "one size fits all" once nutrient-gene interactions are taken into account, this idea gave rise to the My Pyramid to My Plate concept, which enables people to receive precision nutrition based on their needs and nutritional status.

As preventive medicine and nutrition grow more and more intertwined, it is crucial that dieticians, medical professionals, and geneticists receive the required training in the area of nutritional genomics in order to make the best use of its ideas and practices. Education must also be included in allied health courses so that students can teach the public about using omics technologies to improve healthcare.

REFERENCES

Adetunji, J. B., C. O. Adetunji, S. Islam and D. Thangadurai, 2022. Nutritional epigenomics and disease prevention. In: Nutriomics. CRC Press: pp: 49-61.

Ahluwalia, M. K., 2021. Nutrigenetics and nutrigenomics—a personalized approach to nutrition. Advances in Genetics, 108: 277-340.

ISSN E: 3006-1466
ISSN P: 3006-1458
CONTEMPORARY JOURNAL OF SOCIAL SCIENCE REVIEW
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CONTEMPORARY JOURNAL OF SOCIAL SCIENCE REVIEW
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Alam, M. Z., M. A. Hoque, G. J. Ahammed, R. McGee and L. Carpenter-Boggs, 2019. Arsenic accumulation in lentil (lens culinaris) genotypes and risk associated with the consumption of grains. Scientific Reports, 9(1): 9431.

Alhmoud, J. F., J. F. Woolley, A.-E. Al Moustafa and M. I. Mallei, 2021. DNA damage/repair management in cancers. Advances in Medical Biochemistry, Genomics, Physiology, and Pathology: 309-339.

Alsulami, S., A. Aji, U. Ariyasra, S. Sari, N. Tasrif, F. Yani, J. A. Lovegrove, I. Sudji, N. Lipoeto and K. S. Vimaleswaran, 2020. Interaction between the genetic risk score and dietary protein intake on cardiometabolic traits in southeast asian. Genes & nutrition, 15: 1-10.

Asghar, W. and N. Khalid, 2023. Nutrigenetics and nutrigenomics, and precision nutrition. SAGE Publications Sage UK: London, England: pp: 169-170.

Bahinipati, J., R. Sarangi, S. Mishra and S. Mahapatra, 2021. Nutrigenetics and nutrigenomics: A brief review with future prospects. Biomedicine, 41(4): 714-719.

Barrero, M. J., P. Cejas, H. W. Long and A. Ramirez de Molina, 2022. Nutritional epigenetics in cancer. Advances in Nutrition, 13(5): 1748-1761.

Batool, A., U. Farooq, A. Shafi and Z. Khan, 2022. Nutrigenomics: Challenges and opportunities. Trends and Innovations in Food Science.

Berciano, S., J. Figueiredo, T. D. Brisbois, S. Alford, K. Koecher, S. Eckhouse, R. Ciati, M. Kussmann, J. M. Ordovas and K. Stebbins, 2022. Precision nutrition: Maintaining scientific integrity while realizing market potential. Frontiers in nutrition, 9: 979665.

Cariello, M., E. Piccinin and A. Moschetta, 2021. Transcriptional regulation of metabolic pathways via lipid-sensing nuclear receptors ppars, fxr, and lxr in nash. Cellular and Molecular Gastroenterology and Hepatology, 11(5): 1519-1539.

Cassotta, M., T. Y. Forbes-Hernandez, D. Cianciosi, M. Elexpuru Zabaleta, S. Sumalla Cano, I. Dominguez, B. Bullon, L. Regolo, J. M. Alvarez-Suarez and F. Giampieri, 2021. Nutrition and rheumatoid arthritis in the 'omics' era. Nutrients, 13(3): 763.

Chattopadhyay, I., 2020. Role of nutrigenetics and nutrigenomics in cancer chemoprevention. Pharmacotherapeutic Botanicals for Cancer Chemoprevention: 167-188.

Chen, M. and W. Chen, 2022. Single-nucleotide polymorphisms in medical nutritional weight loss: Challenges and future directions. Journal of Translational Internal Medicine, 10(1): 1-4.

Chirita-Emandi, A. and M. Niculescu, 2020. Methods for global nutrigenomics and precision nutrition. In: Principles of nutrigenetics and nutrigenomics. Elsevier: pp: 49-58.

Christie, J. and C. ND, 2023. How to use nutrigenomics testing to personalize your patients optimal diet. Nutrition.

Corrêa, T. A. F., B. J. Quintanilha, M. M. Norde, M. A. d. S. Pinhel, C. B. Nonino and M. M. Rogero, 2020. Nutritional genomics, inflammation and obesity. Archives of endocrinology and metabolism, 64: 205-222.

Dable-Tupas, G., C. J. Capirig, M. Roy and A. Pathak, 2023. Nutrigenomics research: Methods and applications. In: Role of nutrigenomics in modern-day healthcare and drug discovery. Elsevier: pp: 35-82.

Dai, X. and L. Shen, 2022. Advances and trends in omics technology development. Frontiers in Medicine, 9: 911861.

Dallio, M., M. Romeo, A. G. Gravina, M. Masarone, T. Larussa, L. Abenavoli, M. Persico, C. Loguercio and A. Federico, 2021. Nutrigenomics and nutrigenetics in metabolic-(dysfunction) associated fatty liver disease: Novel insights and future perspectives. Nutrients, 13(5): 1679.

ISSN E: 3006-1466
ISSN P: 3006-1458
CONTEMPORARY JOURNAL OF SOCIAL SCIENCE REVIEW
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CONTEMPORARY JOURNAL OF SOCIAL SCIENCE REVIEW
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de Hoogh, I. M., M. J. Reinders, E. L. Doets, F. P. Hoevenaars and J. L. Top, 2023. Design issues in personalized nutrition advice systems. Journal of Medical Internet Research, 25: e37667.
Desjardins, L.-C. and M.-C. Vohl, 2023. Precision nutrition for cardiovascular disease prevention. Lifestyle Genomics, 16(1): 73-82.
Dhanapal, A. C. T. A. and K. S. Vimaleswaran, 2022. Vitamin d supplementation and immune-related markers: An update from nutrigenetic and nutrigenomic studies. British journal of nutrition, 128(8): 1459-1469.
Dhar, G. A., S. Saha, P. Mitra and R. Nag Chaudhuri, 2021. DNA methylation and regulation of gene expression: Guardian of our health. The Nucleus, 64(3): 259-270.
Elghazawy, M., L. A. A. ElShawarby, R. Saleh and N. Fouad, 2020. Tcf7l2 polymorphism and inflammation in type ii diabetes: A nutrigenetic pilot study. Acta Scientific Pharmaceutical Sciences, 4(1): 02-08.
Elshahat, S., T. Moffat, O. Gagnon, L. Charkatli and E. D. Gomes-Szoke, 2023. The relationship between diet/nutrition and the mental health of immigrants in western societies through a holistic bio-psycho-socio-cultural lens: A scoping review. Appetite: 106463.
Ferguson, L. and M. Barnett, 2022. Nutrigenomics and nutrigenetics research in new zealand, and its relevance and application to gastrointestinal health. Nutrients, 14(9): 1743.
Franzago, M., D. Santurbano, E. Vitacolonna and L. Stuppia, 2020. Genes and diet in the prevention of chronic diseases in future generations. International Journal of Molecular Sciences, 21(7): 2633.
Gairola, K., S. K. Dubey and A. Bahuguna, 2023. Nutrigenomics in cancer chemoprevention. In: Natural products and nano-formulations in cancer chemoprevention. CRC Press: pp: 165-181.
Haro, D., P. F. Marrero and J. Relat, 2019. Nutritional regulation of gene expression: Carbohydrate-, fat-and amino acid-dependent modulation of transcriptional activity. International Journal of Molecular Sciences, 20(6): 1386.
Hartiala, J. A., Y. Han, Q. Jia, J. R. Hilser, P. Huang, J. Gukasyan, W. S. Schwartzman, Z. Cai, S. Biswas and D.-A. Tregouet, 2021. Genome-wide analysis identifies novel susceptibility loci for myocardial infarction. European heart journal, 42(9): 919-933.
Ilango, S., B. Paital, P. Jayachandran, P. R. Padma and R. Nirmaladevi, 2020. Epigenetic alterations in cancer. Frontiers in Bioscience-Landmark, 25(6): 1058-1109.
Inzaghi, E., V. Pampanini, A. Deodati and S. Cianfarani, 2022. The effects of nutrition on linear growth. Nutrients, 14(9): 1752.
Irimie, A. I., C. Braicu, S. Pasca, L. Magdo, D. Gulei, R. Cojocneanu, C. Ciocan, A. Olariu, O. Coza and I. Berindan-Neagoe, 2019. Role of key micronutrients from nutrigenetic and nutrigenomic perspectives in cancer prevention. Medicina, 55(6): 283.
Ishikawa, M. and T. Yokoyama, 2023. The relationship between individual and environmental factors related to health, nutritional status, and diet in elderly people living alone in japan. Nutrition reviews, 81(1): 91-94.
Izzo, L. T., H. C. Affronti and K. E. Wellen, 2021. The bidirectional relationship between cancer epigenetics and metabolism. Annual review of cancer biology, 5: 235-257.
Jabeen, A., G. Malik, J. I. Mir and R. Rasool, 2023. Nutrigenomics: Linking food to genome. Italian Journal of Food Science, 35(1): 26-40.
Jones, C. L., A. B. Shafer and P. C. Frost, 2023. Characterizing nutritional phenotypes using experimental nutrigenomics: Is there nutrient-specificity to different types of dietary stress? Molecular Ecology, 32(5): 1073-1086.

ISSN E: 3006-1466
ISSN P: 3006-1458
CONTEMPORARY JOURNAL OF SOCIAL SCIENCE REVIEW
CJSSR
CONTEMPORARY JOURNAL OF SOCIAL SCIENCE REVIEW
Vol.02 No.04 (2024)

Kalea, A. Z. and D. Klimis-Zacas, 2021. Nutrition, precision and nature: The focus on the individual, not the average individual. Current Opinion in Clinical Nutrition & Metabolic Care, 24(4): 287-288.

Kaur, S., A. Ali, U. Ahmad, Y. Siahbalaei, A. Pandey and B. Singh, 2019. Role of single nucleotide polymorphisms (snps) in common migraine. The Egyptian Journal of Neurology, Psychiatry and Neurosurgery, 55(1): 1-7.

Kiani, A. K., G. Bonetti, K. Donato, J. Kaftalli, K. L. Herbst, L. Stuppia, F. Fioretti, S. Nodari, M. Perrone and P. Chiurazzi, 2022. Polymorphisms, diet and nutrigenomics. Journal of Preventive Medicine and Hygiene, 63(2 Suppl 3): E125.

Kim, S. Q., R. Mohallem, J. Franco, K. K. Buhman, K.-H. Kim and U. K. Aryal, 2022. Multi-omics approach reveals dysregulation of protein phosphorylation correlated with lipid metabolism in mouse non-alcoholic fatty liver. Cells, 11(7): 1172.

Koyama, S., K. Ito, C. Terao, M. Akiyama, M. Horikoshi, Y. Momozawa, H. Matsunaga, H. Ieki, K. Ozaki and Y. Onouchi, 2020. Population-specific and trans-ancestry genome-wide analyses identify distinct and shared genetic risk loci for coronary artery disease. Nature genetics, 52(11): 1169-1177.

Kunadis, E., E. Lakiotaki, P. Korkolopoulou and C. Piperi, 2021. Targeting post-translational histone modifying enzymes in glioblastoma. Pharmacology & Therapeutics, 220: 107721.

Lal, M. K., E. Sharma, R. K. Tiwari, R. Devi, U. N. Mishra, R. Thakur, R. Gupta, A. Dey, P. Lal and A. Kumar, 2022. Nutrient-mediated perception and signalling in human metabolism: A perspective of nutrigenomics. International Journal of Molecular Sciences, 23(19): 11305.

Lin, Y., T. Qiu, G. Wei, Y. Que, W. Wang, Y. Kong, T. Xie and X. Chen, 2022. Role of histone post-translational modifications in inflammatory diseases. Frontiers in Immunology, 13: 852272.

Livingstone, K. M., O. Ramos-Lopez, L. Perusse, H. Kato, J. M. Ordovas and J. A. Martínez, 2022. Precision nutrition: A review of current approaches and future endeavors. Trends in Food Science & Technology.

Mattes, R. D., S. B. Rowe, S. D. Ohlhorst, A. W. Brown, D. J. Hoffman, D. J. Liska, E. J. Feskens, J. Dhillon, K. L. Tucker and L. H. Epstein, 2022. Valuing the diversity of research methods to advance nutrition science. Advances in Nutrition, 13(4): 1324-1393.

Matusheski, N. V., A. Caffrey, L. Christensen, S. Mezgec, S. Surendran, M. F. Hjorth, H. McNulty, K. Pentieva, H. M. Roager and B. K. Seljak, 2021. Diets, nutrients, genes and the microbiome: Recent advances in personalised nutrition. British journal of nutrition, 126(10): 1489-1497.

McDade, T. W., Ryan, C. P., Jones, M. J., Hoke, M. K., Borja, J., Miller, G. E., … &Kobor, M. S. , (2019). Genome wide analysis of DNA methylation in relation to socioeconomic status during development and early adulthood. . American journal of physical anthropology,, 169((1),): 3-11.

Meiliana, A. and A. Wijaya, 2020. Nutrigenetics, nutrigenomics and precision nutrition. The Indonesian Biomedical Journal, 12(3): 189-200.

Meroni, M., M. Longo, A. Rustichelli and P. Dongiovanni, 2020. Nutrition and genetics in nafld: The perfect binomium. International Journal of Molecular Sciences, 21(8): 2986.

Mierziak, J., K. Kostyn, A. Boba, M. Czemplik, A. Kulma and W. Wojtasik, 2021. Influence of the bioactive diet components on the gene expression regulation. Nutrients, 13(11): 3673.

Mishra, U. N., D. Jena, C. Sahu, R. Devi, R. Kumar, R. Jena, E. A. Irondi, S. Rout, R. K. Tiwari and M. K. Lal, 2022. Nutrigenomics: An inimitable interaction amid genomics, nutrition and health. Innovative Food Science & Emerging Technologies, 82: 103196.

ISSN E: 3006-1466
ISSN P: 3006-1458
CONTEMPORARY JOURNAL OF SOCIAL SCIENCE REVIEW
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CONTEMPORARY JOURNAL OF SOCIAL SCIENCE REVIEW
Vol.02 No.04 (2024)

Mondal, S. and D. Panda, 2020. Nutrigenomics: An interface of gene-diet-disease interaction. In: Mineral deficiencies-electrolyte disturbances, genes, diet and disease interface. IntechOpen.
Mullins, V. A., W. Bresette, L. Johnstone, B. Hallmark and F. H. Chilton, 2020. Genomics in personalized nutrition: Can you "eat for your genes"? Nutrients, 12(10): 3118.
Nichols, R. G. and E. R. Davenport, 2021. The relationship between the gut microbiome and host gene expression: A review. Human genetics, 140(5): 747-760.
Ortea, I., 2022. Foodomics in health: Advanced techniques for studying the bioactive role of foods. TrAC Trends in Analytical Chemistry, 150: 116589.
Otero, M. C. B. and L. F. Bernolo, 2023. General principles in nutrigenomics and nutrigenetics. In: Role of nutrigenomics in modern-day healthcare and drug discovery. Elsevier: pp: 3-18.
Pal, A., 2022. Protocols in advanced genomics and allied techniques. Springer.
Picó, C., F. Serra, A. M. Rodríguez, J. Keijer and A. Palou, 2019. Biomarkers of nutrition and health: New tools for new approaches. Nutrients, 11(5): 1092.
Pinho, R. and E. Maga, 2021. DNA methylation as a regulator of intestinal gene expression. British journal of nutrition, 126(11): 1611-1625.
Pinu, F. R., D. J. Beale, A. M. Paten, K. Kouremenos, S. Swarup, H. J. Schirra and D. Wishart, 2019. Systems biology and multi-omics integration: Viewpoints from the metabolomics research community. Metabolites, 9(4): 76.
Prakash, B., P. P. Singh, A. Kumar and V. Gupta, 2020. Prospects of omics technologies and bioinformatics approaches in food science. In: Functional and preservative properties of phytochemicals. Elsevier: pp: 317-340.
Radzikowska, U., K. Baerenfaller, J. A. Cornejo-Garcia, C. Karaaslan, E. Barletta, B. E. Sarac, D. Zhakparov, A. Villaseñor, I. Eguiluz-Gracia and C. Mayorga, 2022. Omics technologies in allergy and asthma research: An eaaci position paper. Allergy, 77(10): 2888-2908.
Ramazi, S., A. Allahverdi and J. Zahiri, 2020. Evaluation of post-translational modifications in histone proteins: A review on histone modification defects in developmental and neurological disorders. Journal of biosciences, 45: 1-29.
Rubio, K., E. Y. Hernández-Cruz, D. G. Rogel-Ayala, P. Sarvari, C. Isidoro, G. Barreto and J. Pedraza-Chaverri, 2023. Nutriepigenomics in environmental-associated oxidative stress. Antioxidants, 12(3): 771.
Santos, K. d., E. L. Rosado, A. C. P. da Fonseca, G. P. Belfort, L. B. G. da Silva, M. Ribeiro-Alves, V. M. Zembrzuski, J. A. Martínez and C. Saunders, 2022. Fto and adrb2 genetic polymorphisms are risk factors for earlier excessive gestational weight gain in pregnant women with pregestational diabetes mellitus: Results of a randomized nutrigenetic trial. Nutrients, 14(5): 1050.
Shah, K., A. Al-Haidari, J. Sun and J. U. Kazi, 2021. T cell receptor (tcr) signaling in health and disease. Signal transduction and targeted therapy, 6(1): 412.
Singh, V., 2023. Current challenges and future implications of exploiting the 'omics'data into nutrigenetics and nutrigenomics for personalized diagnosis and nutrition-based care. Nutrition: 112002.
Skvortsova, K., C. Stirzaker and P. Taberlay, 2019. The DNA methylation landscape in cancer. Essays in biochemistry, 63(6): 797-811.
Soldati, L., A. Kerkadi, P. Amuna and A. Terranegra, 2015. State of art and science advances on nutrition and nutrigenetics in nutrition-related non-communicable diseases in middle east. Journal of Translational Medicine, 13: 1-1.

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Vol.02 No.04 (2024)

Sunil, R. and D. K. Janghel, 2020. Chapter-6 nutrigenomics. ADVANCES IN: 79.
Swiatowy, W. J., Drzewiecka, H., Kliber, M., Sąsiadek, M., Karpiński, P., Pławski, A., &Jagodziński, , 2021. Physical activity and DNA methylation in humans. . International Journal of Molecular Sciences, 22((23)): 12989.
Tan, P. Y., J. B. Moore, L. Bai, G. Tang and Y. Y. Gong, 2022. In the context of the triple burden of malnutrition: A systematic review of gene-diet interactions and nutritional status. Critical reviews in food science and nutrition: 1-29.
Tuncel, G., Q. Hoti, G. Mocan and M. C. Ergoren, 2022. A review of the mediterranean diet and nutritional genomics in relation to cancer in women. Journal of Preventive Medicine and Hygiene, 63(2 Suppl 3): E81.
Ugrankar, R., J. Bowerman, H. Hariri, M. Chandra, K. Chen, M.-F. Bossanyi, S. Datta, S. Rogers, K. M. Eckert and G. Vale, 2019. Drosophila snazarus regulates a lipid droplet population at plasma membrane-droplet contacts in adipocytes. Developmental cell, 50(5): 557-572. e555.
Uthpala, T., H. Fernando, A. Thibbotuwawa and M. Jayasinghe, 2020. Importance of nutrigenomics and nutrigenetics in food science.
Vimaleswaran, K. S., 2020. A nutrigenetics approach to study the impact of genetic and lifestyle factors on cardiometabolic traits in various ethnic groups: Findings from the genuine collaboration. Proceedings of the Nutrition Society, 79(2): 194-204.
Visioli, F., F. Marangoni, A. Poli, A. Ghiselli and D. Martini, 2022. Nutrition and health or nutrients and health? International Journal of Food Sciences and Nutrition, 73(2): 141-148.
Vyas, S., 2022. Advances in nutrigenomics and applications in public health: A recent update. Current Research in Nutrition and Food Science Journal, 10(3): 1092-1104.
Wen, X., B. Zhang, B. Wu, H. Xiao, Z. Li, R. Li, X. Xu and T. Li, 2022. Signaling pathways in obesity: Mechanisms and therapeutic interventions. Signal transduction and targeted therapy, 7(1): 298.
Yang, Y., X. Fan, J. Yan, M. Chen, M. Zhu, Y. Tang, S. Liu and Z. Tang, 2021. A comprehensive epigenome atlas reveals DNA methylation regulating skeletal muscle development. Nucleic acids research, 49(3): 1313-1329.
Ye, C.-y., J.-r. Xin, Z. Li, X.-y. Yin, S.-l. Guo, J.-M. Li, T.-Y. Zhao, L. Wang and L. Yang, 2022. Aldh2, adcy3 and bcmo1 polymorphisms and lifestyle-induced traits are jointly associated with cad risk in chinese han people. Gene, 807: 145948.