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Nutrigenomics and Human Health

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Abstract
Nutritional Genomics encompasses the integration of various omics disciplines with the study of food and nutrition. Within this field, nutrigenetics and nutrigenomics are subfields that focus on understanding the interplay between genetics, nutrition, and health. In the past, research on nutrition primarily focused on the negative impact on health and deficiencies in diet. However, with the advancements in genomics, our understanding of how nutrients influence gene and protein expression, and consequently impact cellular and overall metabolic processes, has become significantly easier. Genomics refers to a set of advanced technologies that enable the generation, analysis, and application of scientific information related to the composition and functions of genomes. This field of study, known as nutritional genomics or nutrigenomics, can be seen as the intersection of molecular nutrition and genomics. The effects of bioactive dietary components extend beyond the basic nutritional value they provide. They have specific impacts on various tissues and organs, influencing gene expression patterns (transcriptome), chromatin organization (epigenome), protein expression patterns (proteome), including modifications that occur after protein synthesis, and the profiles of metabolites (metabolome). These are just a few examples of how dietary components interact with our genetic makeup to produce specific effects at a cellular and organismal level. This review enables us to generate useful information for scientists and health professionals regarding the role of Nutrigenomics in the prevention of diet and lifestyle-related diseases.

Keywords: Genomics, molecular nutrition, transcriptome, epigenome, proteome, metabolome

Introduction

Nutrigenomics, also known as nutritional genomics, is an emerging field that explores the intricate relationship between nutrition and our genetic makeup. It focuses on how individual genetic variations affect our response to different nutrients and how nutrients, in turn, influence gene expression and overall health. Through the study of nutrigenomics, researchers aim to understand how specific dietary components interact with our genes and influence the expression of certain traits and diseases. By analyzing an individual's genetic profile, scientists can identify variations that may impact their nutritional needs and susceptibility to certain health conditions [1].

Nutrigenomics combines the fields of genomics, which involves the study of genes and their functions, and nutrition science, which examines the effects of food on our bodies. By integrating these disciplines, researchers can uncover the molecular mechanisms underlying the relationship between diet and health. This field holds great potential for personalized nutrition recommendations and preventive medicine. By understanding an individual's genetic predispositions, healthcare professionals can provide targeted dietary advice, optimizing nutrition plans to promote better health outcomes and reduce the risk of developing diet-related diseases [2].

Moreover, nutrigenomics can also contribute to the development of functional foods tailored to an individual's genetic profile. These foods can be designed to deliver specific nutrients or bioactive compounds that activate or suppress certain genes, promoting optimal health and well-being. Nutrigenomics holds significant potential for disease prevention by providing insights into how specific nutrients and dietary patterns interact with our genes to influence disease risk [3]. By understanding an individual's genetic

variations and their response to different dietary factors, personalized nutrition recommendations can be developed to mitigate the risk of certain diseases. Similar to pharmacogenomics, centers around the study of bioactive components present in everyday food and their impact on the delicate equilibrium between health and disease through their interaction with an individual's genome. Gaining understanding of the diversity within our (EPI) genome and its connection to an individual's susceptibility to complex diseases provides valuable insights into the evolutionary factors that contribute to illness. This analysis aims to explore the interplay between diet, the modern environment, and our (EPI) genome, while also considering the role of redox biology. Such investigations have significant implications for comprehending the risk factors associated with diseases and devising preventive measures [4]

According to German (2005), there are five fundamental principles of nutrigenomics [5].

i) The primary principle asserts that the components found in the food we consume, encompassing both micro- and macro-nutrients, possess the ability to directly or indirectly impact the structure and gene expression of the human genome.
ii) In specific circumstances and among certain individuals, diet can serve as a significant risk factor in the development of various diseases.
iii) It is probable that certain genes, regulated by active substances present in our diet, play a critical role in the initiation, occurrence, progression, and severity of diseases.
iv) The extent to which diet influences the equilibrium between health and disease may depend on the genetic composition of each individual.
v) Nutritional intervention, known as individualized nutrition, is based on an understanding of an individual's nutritional status, requirements, and genotype. It can be employed for preventive, mitigating, or therapeutic purposes in the context of chronic diseases.

There are numerous intricate and varied connections between diet and general health, including excellent health maintenance and disease prevention and management. Measuring and analysing each person's food intake is required in order to implement personalized strategies that improve both human and environmental health [6]

Nutrition to Nutrigenomics

Research in the field of nutrition has been evident since 400 BC when Hippocrates proposed the theory that body temperature is an inherent trait. Around 1700 AD, the "Analytical Chemistry Era" commenced, during which Lavoisier made the groundbreaking discovery of how the body metabolizes food, producing water, carbon dioxide, and energy. In the 19th century, Liebig identified various macronutrients such as carbohydrates, proteins, and lipids, which release heat during metabolism. This led to the "Chemical and Analytical Era of Nutrition" in the 18th to 20th centuries, where Antonie Lavoisier made significant advancements in understanding food metabolism and its connection to energy production, breathing, and oxidation [7]. Subsequently, the "Biological Era" of the 19th century focused on studying metabolism and chemistry, contributing to the understanding of nutrition's role in the prevention and development of chronic diseases like cancer, cardiovascular conditions, neurodegenerative disorders, and bone metabolism issues. Presently, we are in the "Post-Genomic Era," characterized by the integration of biological, social, and environmental aspects, encompassing scientific discoveries about nutritional pathophysiology and metabolism [8].

Nutrition has an impact on the metabolic processes, homeostatic regulation, and the occurrence of diet-related diseases in humans. Additionally, nutrients serve as dietary signals that are detected by cellular sensors, leading to changes in gene and protein expression, ultimately influencing the characteristics of an organism. However, comprehending the field of nutrition science presents a significant challenge due to the diverse and intricate nature of the human diet consumed worldwide. The human diet consists of a complex combination of biologically active substances, encompassing both macronutrients and micronutrients [9].

Nutrient-gene interactions can be categorized into three main conceptual groups:

- Direct interactions: Nutrients, sometimes after interacting with a receptor, act as transcription factors that have the ability to bind to DNA. This interaction leads to the acute induction of gene expression.
- Epigenetic interactions: Nutrients have the capacity to modify the structure of DNA or histone proteins in chromatin. These modifications result in long-term changes to gene expression.
- Genetic variations: Common genetic variations, specifically single-nucleotide polymorphisms (SNPs), can impact the expression or functionality of genes. These variations can lead to alterations in the metabolism of nutrients and changes in dietary requirements.

Overall, these mechanisms can cause modifications in nutrient metabolism and influence the specific dietary needs of individuals.

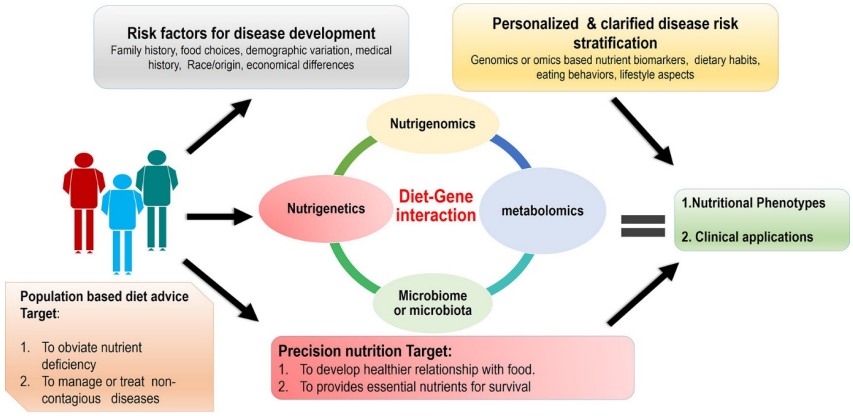

Fig 1: Diet-Gene Interaction

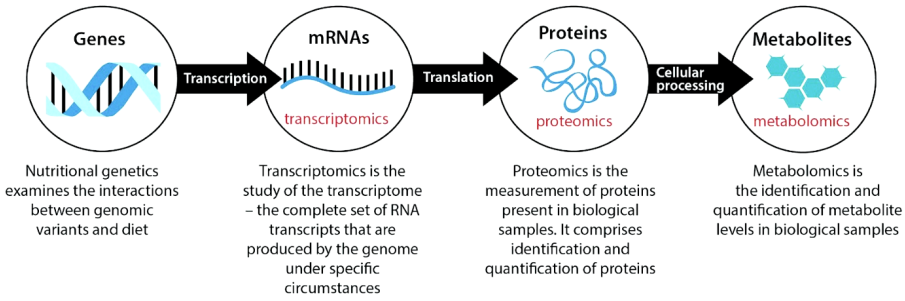

Fig 2: An overview of nutrigenomics: investigating the interactions between nutrition and the host (genome) through omics technologies.

Recent advancements in scientific knowledge have prompted a shift in nutritional research from conventional approaches such as epidemiology and physiology to more modern fields like molecular biology, genetics, and nutrigenomics. These disciplines aim to unravel the effects of nutrient deficiency and toxicity at a molecular level, examining the impact on genes, gene expression, proteins, and metabolites. From a nutrigenomics perspective, nutrients act as powerful signals or sensors that influence specific cells to undergo metabolic alterations, ultimately determining whether an individual maintains good health or develops diseases.

Following the completion of the Human Genome Project (HGP), there emerged a new phase in biological research known as the "Post-Genomic Era." During this era, significant discussions and initiatives took place, driven by advancements in bioinformatics and the field of "omics" sciences. These sciences utilize biotechnology to isolate and analyze a larger array of biomolecules within specific categories, including DNA, RNA, proteins, and metabolites [10]. Consequently, alongside genomics, other ground-breaking disciplines emerged as transformative tools, such as proteomics, metabolomics, and transcriptomics.

Today, the definition of nutrigenomics has expanded to encompass the investigation of how nutritional factors can safeguard the genome. As a result, this emerging field strives to understand the influence of dietary components on different aspects of biological data, such as the Genome, Transcriptome, Proteome, and Metabolome. By conducting a single experiment, nutrigenomics can provide multiple outcomes, underscoring the need for innovative approaches to effectively navigate this dynamic field. The application of various omics tools, including agri-genomics, nutrigenomics, Nutri-proteomics, Ayurnutrigenomics, nutri-metabolomics, and epigenomics, provides powerful means to deepen our understanding of nutrient-gene interactions and their impact on individual health [11]. These tools offer valuable insights into the complex relationship between nutrients and genes, shedding light on their effects on human well-being.

Nutritional Epigenomics

Epigenomics refers to the comprehensive examination of all epigenetic modifications occurring within a cell or tissue at a specific moment. The epigenome encompasses chemical compounds that alter or tag the genome, providing instructions on a cell's capabilities, as well as when and where they should be implemented. These marks, known as epigenetic marks, are inherited as cells divide and persist across generations. The genotype, along with environmental factors, diet, and medications, for example, influences these distinct signatures, ultimately shaping the phenotype.

Among lifestyle factors, nutrition has been extensively studied and is well recognized for its association with epigenetic modifications [12]. Within this realm, nutria-epigenetic research investigates the influence of specific foods and nutrients on the epigenetic landscape and cellular phenotypes. This field encompasses personalized and precision nutrition studies, where nutria-epigenetic approaches have been utilized to develop and provide customized dietary recommendations [13]. Diverse epigenetic mechanisms, including post-translational histone modifications and DNA methylation, interpret and respond to signals conveyed by dietary molecules [14].

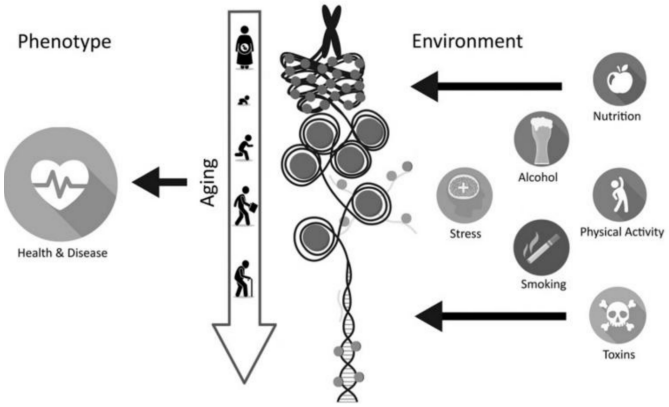

Fig 3: Environmental influences on epigenetics

Epigenetics

Studies on epigenetics concentrate on the changes that take place in DNA and proteins, particularly the interactions between DNA and histones. Without changing the underlying nucleotide sequence, these alterations can cause changes in chromatin structure. In epigenetics, information is transmitted through gene expression, which changes gradually and might be reversible over time. One noteworthy instance is the metabolism of folate, where folic acid, a vitamin critical for preserving genetic integrity, is crucial in supplying a balanced supply of deoxyribonucleotides required for DNA replication. Folic acid functions as a cofactor for the thymidylate (a protein found in RNA molecules) and nucleotide biosynthesis enzymes, acting as a universal donor of methyl groups and taking part in DNA methylation activities.

This review provides an overview of the existing understanding regarding the detrimental impacts of various factors on inflammation through epigenetic mechanisms. These factors include high-fat/high-sugar diets, deficiencies in micronutrients such as folate, manganese, and carotenoids, obesity and its associated complications, bacterial/viral infections, smoking, excessive alcohol consumption, sleep deprivation, chronic stress, air pollution, and exposure to chemicals [15].

Transcriptomics

Transcriptomics investigates the complete collection of activated RNA transcripts. These transcripts, generated in a

specific tissue and at a particular moment within an organism, reflect the dynamic nature of gene expression, which varies under different circumstances and over time. Transcription factors, once activated, migrate to the nucleus and bind to specific DNA sequences in gene promoter regions, thereby regulating or facilitating transcription processes. Physiological signals, such as those triggered by nutrients or bioactive food compounds, as well as their metabolites, hormones, pharmacological treatments, diseases, and other factors, can stimulate these transcription factors. Acting as sensors, they modulate gene transcription according to cellular requirements. In the realm of nutrition research, transcriptomics plays a vital role in unraveling the mechanisms and underlying effects of particular nutrients or diets. It also aids in the identification of genes, proteins, or metabolites that exhibit altered expression patterns in predisease states and facilitates the characterization of pathways regulated by nutrients or bioactive compounds found in foods [16].

Proteomics

Proteomics is a scientific field that investigates the complete range of proteins involved in the biological processes of a specific species. These proteins function within cells, tissues, or organs under normal conditions, but their expression levels or activities can change during different physiological or pathological situations, similar to transcriptomics. Proteins are essential molecules found in all living cells, serving diverse roles such as structural support, mechanical function, biochemical reactions, cell signaling, transport, and storage. They are also crucial components of the human diet. The number of proteins produced by an organism surpasses the number of genes it possesses, largely due to various post-transcriptional and translational modifications. Proteomics employs a range of technologies to study protein expression. These include chromatographic techniques combined with electrophoresis, sample fractionation through extraction sequences, organellar proteome analysis, and other strategies. Consequently, Proteomics serves as a fundamental tool for Nutrigenomics, bridging the gap between genome sequences and cellular behavior. It helps unravel the process of determining genetic function and how the genome is activated in response to specific diets. For instance, butyrate activity can modify the expression of numerous proteins within the ubiquitin-proteasome system. This alteration suggests that proteolysis may be the mechanism through which butyrate regulates key proteins involved in controlling the cell cycle, apoptosis, and cell differentiation [17].

Metabolomics

The metabolome encompasses a collection of small primary and secondary metabolites found in the body fluids of an organism or species. Metabolomics, a branch of functional genomics, investigates changes in these metabolites with the goal of isolating and characterizing them. Advancements in metabolomics research have the potential to enhance our understanding of how genotype relates to an individual's phenotype. From a nutritional perspective, metabolomics has various applications as it enables the identification of metabolic patterns and disorders caused by an individual's diet, as well as how these changes can impact their health and disease susceptibility. Furthermore, metabolomics examines metabolism under the influence of environmental and genetic perturbations, utilizing bioinformatics and statistical tools for analysis and interpretation. Metabolism serves as a unifying mechanism that incorporates the impacts of diet, environment, and genetics during the aging process. Aging is influenced by various factors, including genetic and epigenetic alterations, dietary choices, lifestyle habits, and exposure to the environment. The observable characteristics of an organism, known as its phenotype, are the product of interactions between its genetic makeup and the influences of diet, lifestyle, and the surrounding environment. Metabolites, which serve as a comprehensive reflection of molecular changes, effectively encapsulate the combined effects of genetics, diet, and environment on the aging process. Therefore, metabolic profiles provide a holistic view of how genetics, dietary patterns, and environmental factors contribute to the aging phenomenon [18].

Metabolites are organic molecules that reside in the cell cytosol and directly interact with proteins and other macromolecules. They are categorized as primary or secondary metabolites. Primary metabolites are directly involved in the synthesis and degradation of macromolecules, while secondary metabolites, more prevalent in plants and fungi, serve as structural components and defense mechanisms. Metabolites in living organisms can function as substrates, enzyme inhibitors or activators, molecular precursors, waste products of synthesis or degradation, and more. Metabolomics captures the combined effects of both the genetic information and environmental factors on a biological system, providing insights into the endogenous metabolites derived from genes as well as the exogenous metabolites originating from the environment. This approach facilitates the investigation of the intricate interplay between genes and the environment, uncovering potential interactions and connections [19].

In the field of nutrition, metabolomics provides insights into metabolic patterns and disturbances influenced by diet. This knowledge contributes to understanding how an individual's health or illness can be affected by the excess or deficiency of specific nutrients or compounds (including secondary metabolites) present in food. These compounds, whether nutrients or not, interact within the body, influencing metabolic pathways. For instance, perilla alcohol, a monoterpene found in strawberries, may exhibit anticancer properties under certain physiological conditions [20].

Effect of Different Nutrients on Gene Expression

Carbohydrate

Feeding high-energy diet to rats leads to early development of obesity and metabolic syndrome, apparently through an inability to cope with energy density of the diet. Obesity in the face of reduced overall weight gain was associated with decrease in mRNA levels for the oxygenic neuropeptides, NPY (neuropeptides Y), Ag RP (Agouti Related Peptide) in the ARC (Arcuate nucleus). The effects of hyperglycemia on liver angio-tensinogen (AGT) gene expression are found that hyperglycemia activated AGT gene expression in liver increased by approximately three-fold. It has been shown that lactase activity and LPH (lactase/ phlorizin hydrolase) mRNA level in the jejunum are coordinately increased by consumption of various carbohydrates, including sucrose, fructose, galactose, and glycerol. Feeding the glucose diet caused a slight increase in lactase activity, accompanied by an elevation of LPH mRNA levels. By contrast, a-methylglucoside, a non-metabolizable sugar, was unable to induce an elevation in LPH mRNA levels. It has been demonstrated that LPH mRNA levels are elevated in rats given sucrose as a dietary carbohydrate source, but not in

those given α–methylglucoside. A report also suggests that between the two constituent monosaccharides of sucrose, fructose is more active in enhancing LPH mRNA levels than glucose [21]. Therefore, it is most likely that the sucrose-induced increases in the LPH mRNA levels are attributable to fructose that is produced by the hydrolysis of sucrose. These results prompted to hypothesize that certain carbohydrates or their common metabolite might play a pivotal role in the regulation of LPH gene expression.

Dietary Fat

Dietary fat plays a crucial role as a macronutrient in supporting the growth and development of living organisms. Beyond providing energy and influencing the composition of lipid membranes, dietary fat exerts significant influence on gene expression, which in turn affects metabolism, growth, and cellular differentiation.

PUFA plays a role in regulating gene expression in both lipogenic (fat-producing) and non-lipogenic tissues. When a diet rich in linoleic acid (60%) was administered, the activity of lipogenic enzymes in the liver decreased. Several hepatic genes, including malic enzyme, acetyl co a carboxylase, L-pyruvic kinase, fatty acid synthase, and S14 protein, were affected. Although initial studies primarily focused on the liver, recent research has shed light on the impact of PUFA on adipose tissues as well. Adipocyte-specific gene regulation was observed with the n-3 fatty acid linolenate. In a cell line of adipocytes called 3T3-L1, arachidonic acid (n-6) reduced the stability of SCD1 mRNA in a dose-dependent manner (with a maximum repression of 80%), and similar effects were observed with linoleic and eicosatetraenoic acid .Dietary intake of n-6 and n-3 PUFA has also been associated with reduced triglyceride accumulation in skeletal muscle, as well as potentially in cardiomyocytes and pancreatic beta-cells. Interestingly, there have been reports indicating that short-chain fatty acids, which are generated by the gut microbiota, can influence the epigenome to modulate various pathophysiological processes in the host, such as inflammation [22].

Minerals

Like other nutrients, minerals play a role in regulating gene expression. Zinc, for example, is an essential trace element that acts as a cofactor in numerous proteins involved in intermediary metabolism, hormone secretion pathways, and immune defense mechanisms. In the case of zinc deficiency, there is an observed increase in DNA oxidation, DNA breaks, and an elevated rate of chromosome damage. A group of extracellular matrix metalloproteases (MMPs), which includes collagenases and elastases, along with their endogenous inhibitors, has been extensively studied. These enzymes play crucial roles in immunology, development, cancer, and wound healing. Zinc is essential for the catalytic activity of these enzymes, and they likely acquire zinc from intracellular sources during translation [23]. However, during zinc deficiency, the secretion of the fully functional enzyme may be reduced. Furthermore, after secretion, the enzyme's affinity for zinc may lead to an exchange of zinc with the surrounding environment, resulting in decreased enzymatic activity. Fluctuations in zinc levels, especially during deficiency, can have a direct impact on the activity of these enzymes and their associated metabolic feedback pathways. Feedback pathways may involve increased degradation of the inactive form of the enzyme or reduced stimulation of signal transduction pathways that trigger compensatory responses.

Iron serves as a crucial component of ribonucleotide reductase and mitochondrial cytochromes. When there is a deficiency of iron, the DNA repair capacity is reduced, and there is an increased susceptibility to oxidative damage, particularly in mitochondrial DNA [24]. The level of the HFE protein, which is associated with hemochromatosis and is encoded by a major histocompatibility complex (MHC) class I-like gene, is found to increase with higher cellular iron levels in human intestinal cells. This finding supports the notion that HFE protein levels in enterocytes play a role in maintaining iron homeostasis. Furthermore, studies conducted on Caco-2 cells demonstrate changes in HFE mRNA levels following iron treatment, suggesting that iron regulates HFE expression. Additionally, inhibiting protein synthesis has a significant impact on HFE mRNA levels, indicating that a short-lived protein may be involved in repressing HFE gene transcription [25].

Vitamins

Vitamins are vital micronutrients that are necessary in small quantities within the diets of humans and non-ruminant animals. While some vitamins can be internally synthesized by ruminant animals if the required precursor compounds are available. Vitamins play a significant role in regulating gene expression, thereby impacting various biological processes. For instance, Vitamin C and Vitamin E possess antioxidant properties that help prevent DNA and lipid oxidation. Insufficient levels of these vitamins are associated with increased instances of DNA strand breaks, chromosome breaks, oxidative DNA lesions, and lipid peroxide adducts on DNA [26]. Folate and vitamin B12 are essential for maintaining and methylating DNA, facilitating DNA synthesis, and effectively recycling folate. In cases of deficiency, DNA may incorporate uracil incorrectly, resulting in elevated levels of chromosome breaks, DNA hypomethylation, and other irregularities. Niacin serves as a substrate for poly (ADP-ribose) polymerase (PARP), an enzyme responsible for DNA cleavage, rejoining, and telomere length maintenance. Inadequate levels of niacin can lead to heightened levels of unrepaired nicks in DNA, chromosome breaks, rearrangements, and increased sensitivity to mutagens. Vitamin A contributes to the expression of specific genes such as PEPCK (phosphoenolpyruvate kinase) and IGF (insulin-like growth factor), while biotin is involved in gene-level synthesis of essential proteins (enzymes) like OTC (ornithine transcarbamylase). Furthermore, Vitamin C exerts influence on hepatic gene expression. More specifically, the subsequent sections describe the impact of vitamin A on PEPCK gene expression and the role of biotin in OTC gene expression [27].

Biotin

Biotin plays a direct role in crucial metabolic processes such as gluconeogenesis, fatty acid synthesis, amino acid catabolism (via pyruvate carboxylase), and acts as a component of enzymes like acetyl coenzyme carboxylase, propionyl CoA carboxylase, and β-methyl crotonyl CoA carboxylase. Additionally, it serves an important function in the expression of the ornithine transcarbamylase (OTC) gene. In humans, defects in biotin utilization lead to hyperammonemia, acidosis, and clinical symptoms including coma, seizures, skin rashes, alopecia, and pica. Ammonia removal in mammals occurs through two major mechanisms: urea synthesis catalyzed by urea cycle enzymes, and glutamine synthesis via glutamine synthetase. Biotin can

influence the activity and gene expression of enzymes involved in the urea cycle. Specifically, OTC converts ornithine to citrulline, which is a crucial step in the urea cycle. Biotin serves as a prosthetic group for OTC, and its deficiency results in reduced OTC enzyme levels, impairing normal urea cycle function and urea synthesis in the body. In biotin-deficient rats, OTC gene expression in the liver was lower compared to biotin-sufficient rats, primarily due to impaired gene transcription and decreased stabilization of OTC mRNA [28].

Conclusion

The article presents the foundational knowledge regarding the influence of various nutrients on gene expression in the body and its potential implications. To fully harness the genetic potential, it is crucial for nutrients to operate at the genetic level. This principle applies not only to beneficial nutrients but also to toxic substances found in food, necessitating an understanding of their toxicity at both the genetic and post-genetic levels to mitigate their adverse effects [29]. The integration of nutritional genomics technologies with genomic databases, encompassing sequences, inter-individual genetic variations, and disease susceptibility, can aid in identifying individuals at risk and predisposed to cancer. This, in conjunction with biomarker utilization, can contribute to the development of strategies for cancer prevention.

The article raises the question of whether nutrigenomics research can pave the way for the creation of food products that can prevent or mitigate the onset and impact of complex diseases like type 2 diabetes, cardiovascular diseases, and specific forms of cancer. Additionally, can food products be tailored to enhance the health and well-being of particular population groups based on their individual genomes? Addressing these queries necessitates a comprehensive understanding of the mechanisms that connect diet with the observable characteristics of our genes, known as the phenotype.

The concept of "Genome Health Nutrigenomics" explores how nutritional deficiencies or excesses can trigger mutations in the genome at the level of the base sequence or chromosomes [30]. The principal objective of this concept is to determine the optimal dietary intake and concentrations of tissue culture medium that minimize genomic damage. Regulations should be put in place by healthcare organizations to control these business models in order to protect patients' integrity and enhance the capability of the healthcare system to provide nutritional advice. As a result, those working in the human health sciences have the chance to continually advance the subject of nutrigenomics through their studies and the creation of cutting-edge instruments. Through these initiatives, the populace will be encouraged to adopt healthier eating habits and to improve their overall quality of life.

Future Prospects

In recent discussions on the future directions of "omics" research, there have been notable findings regarding the application of metabolomics to enhance the accuracy of dietary assessment in nutritional epidemiology. Several opportunities have been identified:

- Determining food intake by analyzing biomarker levels and employing calibration equations derived from feeding studies.
- Classifying individuals into specific dietary patterns based on urinary metabolic profiles.
- Conducting comprehensive metabolome-wide association studies to uncover associations between metabolites and dietary patterns.

However, recent studies have identified combinations of metabolites, known as metabolic signatures that are associated with specific dietary exposures and disease outcomes. According to Smith *et al*., if these metabolic signatures can be utilized to identify population groups at risk of chronic diseases, it may be possible to estimate disease risk in clinical settings with a single plasma sample, eliminating the reliance on self-reported dietary intake.

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