Review Article

Nutrigenomics: An Overview

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ABSTRACT

Nutrition and genetics both play an important role in human health as well as in disease progression. The interaction between genetic and dietary influences can result in a higher risk of disease in certain individuals and populations. Recently, diet-gene association studies have revealed evidence on which to base gene-specific dietary intervention trials to confirm results. Nutrigenomics shows a new way of working with nutrition and now, the knowledge of how food interferes with the genetic code and how the organism responds to these interferences and with the phenotype can be clarified. There are many factors that can influence the response to diet such as age, sex, physical activity, smoking and genetics. Furthermore, the development inside Nutrition Sciences, together with communication and marketing fields, provided the emergence of a personalized nutritional counseling based in Nutrigenomics. The goal of personalized nutrition is to identify individuals who benefit from a particular nutritional intervention and identify alternatives for those who do not. Since the introduction of Nutrigenomics, there is an upgrade in how physicians and other professionals evaluate and treat different diseases. In this review, we have summarized the literature on the effects of diet on whole-body metabolism (i.e., genes, proteins, and metabolites) and the influence of genotype on nutritionally related diseases.

Keywords: Chronic diseases, genetics, nutrition, nutrigenomics

INTRODUCTION

Nutrition and genetics both play an important role in human health as well as the development of chronic diseases such as cancer, osteoporosis, diabetes and cardiovascular disease. Nutrigenomics describes the scientific approach that integrates nutritional sciences and genomics and includes the application of other high-through 'omics' technologies such as transcriptomics, proteomics and metabolomics to investigate the effects of nutrition on health.[1] During the 19th century, also known as "Biological Era", studies on metabolism and chemistry were done, helping the science of nutrition on defining their role in the development and prevention of chronic diseases, such as cancer, cardiovascular, neurodegenerative and bone metabolism disorders. Nowadays, the "Post - Genomic Era" is being experienced. This era is characterized by the integration of three fields: biological, social and environmental, where scientific discoveries on nutritional pathophysiology and metabolism are included. The interaction between genetic and dietary influences can result in a higher risk of disease in certain individuals and populations. Researches indicates that diet-gene interactions play a significant role in person variability, and has clarified some of these genetic differences.[2] Variability between individuals in response to dietary intervention is a well-known phenomenon in nutrition research and practice.[3] For example, the effect of dietary changes on phenotypes such as blood cholesterol, body weight and blood pressure can differ significantly between individuals.[4] There are many factors that can influence the response to diet such as age, sex, physical activity, smoking and genetics. The goal of personalized nutrition is to identify individuals

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who benefit from a particular nutritional intervention (responders), and identify alternatives for those who do not (non-responders). Individuals should no longer be subjected to unnecessary diets they find unpleasant and ineffective when there may be an alternate dietary approach that is more effective. Personalized nutrition could be useful in both the prevention and treatment of chronic diseases by tailoring dietary advice to an individual's unique genetic profile. The daily ingestion, absorption, digestion, transport, metabolism and excretion of nutrients and food bioactives involve many proteins such as enzymes, receptors, transporters, ion channels and hormones. Variations in genes encoding proteins that affect any of these processes can alter both the amount of the protein produced as well as how well that protein functions. If a genetic variation leads to altered production or function of these proteins then nutritional status might be affected. The study of the relationship between genes and diet is called

'Nutrigenomics' (or nutritional genomics), which is an umbrella term for two complimentary approaches: how nutrients affect gene function and how genetic variation affects nutrient response. The latter is sometimes referred to as nutrigenetics and includes the study of how genetic variations affect food intake and eating behaviours.[5]

Although, the term 'nutrigenomics' is relatively new, the concept behind it has been around for some time. Perhaps the most familiar example is lactose intolerance, which is a condition resulting from an inadequate production of lactase in the small intestine due to genetic variation in the lactase gene.[6] Individuals with lactose intolerance are unable to efficiently break down the primary milk sugar (lactose) from dairy products. Consequently, the dietary recommendation is to limit lactose-containing foods or to use lactase supplements or lactose-free dairy products to prevent gastrointestinal discomfort. Also, Phenylketonuria (PKU) is an inborn error of metabolism, which represents another classic example of nutrigenomics. PKU can result from a genetic variation in phenylalanine hydroxylase (the enzyme needed to convert phenylalanine to tyrosine), which leads to a decrease in phenylalanine hydroxylase activity.[7] Individuals with PKU can develop neurological damage (severe mental retardation and seizures) from excess phenylalanine unless they follow the recommended low-phenylalanine diet. A major goal of nutrigenomics is the prevention of the onset and progression of chronic disease. The incorporation of genetics into nutritional epidemiologic studies aims to improve their consistency. Attempts are being made to develop a personalized nutrition guideline for individuals and specific sub-populations, which could decrease the risk of chronic diseases. Therefore, the aim of this review is to summarize the literature on the effects of diet on whole-body metabolism (i.e., genes, proteins, and metabolites) and the influence of genotype on nutritionally related diseases.

METHODOLOY FOR SEARCH STRATEGY

A literature search Medline and PubMed databases were searched under the following key terms: "diet," "chronic diseases," "genetics," "nutrition" and "nutrigenomics," "nutritional genomics" and "chronic periodontitis." All keywords were restricted in title or abstract without the language limitation. Only highly relevant articles from manual and electronic databases were selected for the present review. The aim of this review is to highlight the role of nutrition and genetics in human health as well as in disease progression.

LITERATURE REVIEW

Principles of Nutrigenomics

Nutrigenomics is both the examination of how nutrients affect genes (i.e. influence gene expression and function) and how genes affect diet (i.e. what an individual eats and how an individual responds to nutrients). Nutrigenomics can include the full spectrum of research strategies from basic cellular and molecular biology to, clinical trials, epidemiology and population health. The gene is the functional and physical unit of heredity passed from parent to offspring. Genes are segments of DNA that contain the information for making a specific protein. When variations in the DNA occur the result can be changes to the structure and function

of the protein. There are several different types of genetic variations, including single nucleotide polymorphisms (SNPs), which are alterations in a single nucleotide. Alleles are the variant forms of a gene at a particular location on a chromosome. The genotype is the genetic identity of an individual for a genetic site, determined from the combination of maternal and paternal alleles. Genotypes do not necessarily show as outward characteristics, and as such are different from phenotypes. A phenotype is an observable trait in an individual such as hair color, high blood sugar concentrations, or the presence of a disease. Individuals with the same genotype may have different phenotypes, in part, because of their different environments. A haplotype is a group of alleles that are inherited together and, therefore, groups of genetic polymorphisms are often inherited together. The aim of Nutigenomics is to maintain healthy a group of individuals that live in different dietary conditions. Thus, Human Genome Project came into existence.[8] The Human Genome Project (HGP) concluded new insights about the influence of nutrients into people's diet, which included

1. Will the gene expression in response to metabolic process, at cellular level, influence the health of an individual?
2. Are gene expression and metabolic response the result of the interaction between genotype and environment/nutrient?
3. Understanding how this interaction process occurs between gene and nutrient could lead to the prescription of specific diets for each individual.

Hence, in order to answer those questions, Nutrigenomics was introduced. Nutrigenomics is the area of nutrition that uses molecular tools to search, access, and understand the several responses obtained through a certain diet applied between individuals or population groups. It seeks to find how the components of a particular diet (bioactive compound) may affect the expression of genes, which may have increased its potential or which can be suppressed. This response will depend on how genes will show a changed activity or alter gene expression.

Nutrition and Gene Interaction

There is good evidence that nutrients and physical activity influence gene expression and have shaped the genome over several million years of human evolution. Genes define opportunities for health and susceptibility to disease, while environmental factors determine which susceptible individuals will develop illness. In view of changing socioeconomic conditions in developing countries, such added stress may result in exposure of underlying genetic predisposition to chronic diseases. Studies have shown the role of nutrients in gene expression; for example, researchers are currently trying to understand why omega-3 fatty acids suppress or decrease the mRNA of interleukin, which is elevated in atherosclerosis, arthritis and other autoimmune diseases, whereas the omega-6 fatty acids do not.[9,10] A recent study of the relationship between folate and cardiovascular disease revealed that a common single gene mutation that reduces the activity of an enzyme involved in folate metabolism (MTHFR) is associated with a moderate (20%) increase in serum homocysteine and higher risk of both ischemic heart disease and deep vein thrombosis.[11] Because there are genetic variations among individuals, changes in dietary patterns have a differential impact on a genetically heterogeneous population, although populations with a similar evolutionary background have more similar genotypes. Most are polygenic in nature and rapidly escalating rates suggest the importance of environmental change rather than change in genetic susceptibility.

Nutrigenomics and Disease

Since the sequencing of several eukaryotic genomes, our understanding towards human disease has progressed at an accelerated rate. Amongst 1000 genes that have so far been associated with human disease, 97% result in monogenic diseases, i.e., a single dysfunctional gene is responsible for disease.[11] Modifying the consumption of certain dietary compounds can prevent some monogenic diseases, such as galactosemia and phenylketonuria. Galactosemia arises from a rare recessive trait in galactose-1-phosphate uridyltransferase (GALT), leading to the accumulation of galactose in the blood and increasing the risk of mental retardation.[12]

Phenylketonuria is characterized by the defective phenylalanine hydroxylase (PAH) enzyme, resulting in the accumulation of phenylalanine in the blood that drastically increases the risk of neurological damage.[7] Galactose-free and phenylalanine-restricted tyrosine-supplemented diets are a means to nutritionally treat these monogenic diseases, respectively.[6] In contrast, those diseases reaching epidemic proportions in the Western world, such as cancer, obesity, diabetes and cardiovascular disease, often arise from dysfunctional biological networks, and not a single mutated gene (i.e., polygenic diseases). For example, the recent meta-analysis by Segal et al.[13] of nearly 2000 microarray studies spanning 22 different tumor types demonstrated that no single common gene mutation is responsible for the onset of these tumors, but that shared functional modules exist between various cancer types. Thus, dietary intervention to prevent the onset of such diseases is a complex and ambitious goal that requires not only knowledge of how a single nutrient may affect a biological system, but also how a complex mixture (i.e., diet) of nutrients will interact to modulate biological functions.

Nutrigenomics is widely used for studying diet-related disorders as well as heart-related disorders.[14] The higher income group who consumed excess fat and calorie-rich food had an increased prevalence of diabetes compared to the lower income group. In addition, visible fat consumption and physical inactivity showed a cumulative effect on increasing the prevalence of diabetes. Gene-diet interaction studies revealed that the adiponectin gene polymorphism contributed to insulin resistance and diabetes and this was exaggerated in those consuming diets with higher glycemic loads.[15] Gomase *et al.* explained genomics and its new aspects in cancer research. This study gave a broader idea of its history, strategies, technology, applications and current research. SNP array has significant impact on the genetic analysis of human disorders.[16] It can be used to measure both DNA polymorphism and dosage recommendations. SNP arrays are an ideal platform for identifying both somatic and germline genetic variants that lead to cancer. Nair and Pillai's (2005) reviewed on Human papillomavirus (HPV) and disease mechanisms provided a number of critical observations associated with the role of HPV in cervical and oral cancer.[17]

Nutrigenomics and Cardiovascular Diseases

CVD is the primary diet-related chronic disease of the modern time and the inflammation is emerging as underlying many chronic disorders including CVD. CVD can be characterized as a group of multifactorial conditions associated with obesity, atherosclerosis, hypertension, and thrombosis. All of these pathologic entities are known to be closely related to both genetic factors and environmental influences. Diet is considered as one of the environmental influences and a strong relationship between diet composition and CVD risk is well established.[18] Obesity is a major cardiovascular risk factor, thus polymorphic genes involved in energy balance control certainly provide ''favorable'' or ''unfavorable'' background for the development of CVD. Coronary heart disease (CHD) is one of the most prevalent diseases in Western society, and has been attributed the cause of one in every five deaths in America each year.[19]

Epidemiological studies and long-term outcome trials have established a clear link between lipids and the development of CHD, and specify two lipoproteins as primary targets in combating CHD: low density lipoproteins (LDL) and high-density lipoproteins (HDL). Lipoproteins are macromolecular complexes comprised of a lipid-rich core surrounded by a surface monolayer containing phospholipids, unesterified cholesterol, and specific proteins.[19] Various complexes exist and are defined based on their composition, which ultimately affects their density: chylomicron (high lipid to protein ratio, highest in TG as % of weight), VLDL (very low density lipoprotein, 2nd highest in TG as % of weight), IDL (intermediate density lipoprotein), LDL (highest in CE as % of weight), and HDL (high protein to lipid ratio). The first line of therapy, termed therapeutic lifestyle changes, is composed of alterations in the individual's diet and physical activity, coupled with reductions in smoking and weight if applicable. The second line of therapy involves pharmaceutical compounds, such as statins, which have been found to efficaciously inhibit the activity of

hepatic 3-hydroxy-3-methylglutaryl CoA reductase (HMG-CoA reductase) and ultimately decrease the levels of circulating cholesterol.[20]

These varying degrees of response have been accredited to genetic differences in the population, thereby emphasizing the importance of identifying those genes that play a critical role in the onset or protection from CHD, and unravel their interactions with common dietary compounds. To date, several candidate genes, and their common SNPs, have been identified and preliminary evidence supports the notion that genetic variations in such genes as cholesterol ester transfer protein (CETP), lipoprotein lipase (LPL), hepatic triglyceride lipase (HL), LDL-receptor, apolipoprotein E (APOE), apolipoprotein A1 (APOA1), ATP binding cassette transporter A1 (ABCA1), and lecithin-cholesterol acyltransferase (LCAT) will alter individual sensitivity to developing CHD.[21] Additionally, a nutrigenetic approach has begun to reveal that several of the aforementioned genes and their polymorphisms, such as APOA1 and LPL, are susceptible to dietary intervention and may modulate the onset of CHD.

Nutrigenomics and Diabetes Mellitus

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia due to defective secretion or activity of insulin.[22] A conclusive diagnosis of diabetes mellitus is made by assessing glycated hemoglobin levels; in those people with diabetes, sequential fasting plasma glucose levels will be 7 mmol/L or more. Diabetes mellitus can be classified into broad categories according to signs and symptoms.

Type 1 diabetes mellitus encompasses diabetes resulting primarily from destruction of the beta-cells in the islets of Langerhans of the pancreas. This condition often leads to absolute insulin deficiency. The cause may be idiopathic or due to a disturbance in the autoimmune process. The onset of the disease is often abrupt, and patients with this type of diabetes are more prone to ketoacidosis and wide fluctuations in plasma glucose levels. If untreated, these patients are likely to manifest the classic signs and symptoms of diabetes: polyuria (excessive urine output), polydipsia (excessive thirst) and polyphagia (excessive appetite), as well as pruritis, weakness and fatigue. These patients are more likely to suffer severe systemic complications as a result of the disease.

The causes of type 2 diabetes mellitus range from insulin resistance with relative insulin deficiency to a predominantly secretory defect accompanied by insulin resistance. The onset is generally more gradual than for type 1, and this condition is often associated with obesity. In addition, the risk of type 2 diabetes increases with age and lack of physical activity, and this form of diabetes is more prevalent among people with hypertension or dyslipidemia. People with type 2 diabetes constitute 90% of the diabetic population.

Gestational diabetes mellitus (GDM) is glucose intolerance that begins during pregnancy. The children of mothers with GDM are at greater risk of experiencing obesity and diabetes as young adults.[23] As well, there is a greater risk to the mother of developing type 2 diabetes in the future

Genetic complexity of T2DM: Identifying the genetic basis of diseases caused by single genes (monogenic diseases), such as Huntington disease or cystic fibrosis, is fairly straightforward: one analyzes how frequently a chromosomal region containing a mutated gene is found in individuals showing the disease versus the frequency in individuals who do not show symptoms. In many cases, monogenic diseases are studied in families or in populations where there is evidence of dominant inheritance (if you have the mutation, you develop the disease). Although these methods are powerful for monogenic diseases, many genetic association studies fail to identify single causative genes for chronic diseases like T2DM because multiple genes,[24] and the influence of multiple environmental factors acting on these genes, make variable contributions to the complex trait. Geneticists have developed a method called quantitative trait locus (QTL) analysis to identify regions of chromosomes that contribute to a complex trait. QTLs are found by statistical analyses of how frequently a region of a chromosome is associated with a measurable phenotype, e.g., insulin levels or glucose response for T2DM. Each of the genes within QTLs may contribute different amounts to the phenotype. For example, one QTL may

contribute 30% to the trait, while another may contribute 1% and the contribution may well be influenced by diet or other environmental variables.

Gene variants (i.e., SNPs) may therefore be associated with small to large contributions to the complex trait. The sum of the contributions from causative alleles in different QTLs produces the specific trait or disease.[25] Almost all phenotypic traits (height potential, weight potential, fasting blood glucose, susceptibility to disease, etc.) are quantitative traits. The concept of multiple genes and multiple environmental influences contributing to a complex trait can be illustrated by examining what is currently known about the chromosomal regions containing genes that contribute to T2DM.

Nutrigenetics and Obesity

Genetic differences play an important role in the development of obesity, although it is clear that these are by no means the only contributing factors. Environmental and social factors are also very important. The relative contributions of genetic and socioeconomic factors to the development of obesity, and the ways in which these interact in human societies, are largely unknown. The most recent update of the human obesity gene map emphasizes that there are currently more than 600 gene markers and chromosomal regions that have been associated or linked with human obesity. This provides immensely valuable roles of these genes, and to their contributions to key processes, most notably appetite, that influence the development of obesity. However, such syndromes are extremely rare and therefore of limited relevance to the majority of obese individuals.[26] Some recent studies include the development of technologies capable of parallel genotyping analysis for hundreds of thousands of SNPs from a single small blood or tissue sample. It is estimated that there are about 10 million SNPs in human populations. This scale currently still exceeds the capacity of the new platform technologies, but SNPs that are located close together in the DNA sequence on the same chromosome tend to be inherited together. A set of such associated SNPs is termed a 'haplotype' and it turns out that most chromosome regions have only a few common haplotypes. So, while a chromosome region may contain many SNPs, it is possible that analysing only a few 'tag' SNPs can provide most of the information on the pattern of genetic variation in that region. Defining these haplotype blocks and the most reliable tag SNPs are the goals of the International HapMap Project.[27]

Nutrigenomics and Cancer

Cancer is a process composed of multiple stages in which gene expression, and protein and metabolite function begin to operate aberrantly. In the post-genomic era, the cellular events mediating the onset of carcinogenesis, in addition to their modulation by dietary factors, has yielded important information in understanding of this disease. Inherited mutations in genes can increase one's susceptibility for cancer. The risk of developing cancer can be markedly increased if there is a gene-diet interaction. Studies of twins show that the likelihood of identical twins developing the same cancer is less than 10%, indicating that the environment plays an important role in cancer susceptibility.[28] Evidence of genome and epigenome damage biomarkers, in the absence of overt exposure of genotoxins, are themselves sensitive indicators of deficiency in micronutrients required as cofactors or as components of DNA repair enzymes, for maintenance methylation of CpG sequences and prevention of DNA oxidation and/or uracil incorporation into DNA.[29] Diet considered as a source of either carcinogens (intrinsic or cooking-generated) present in certain foods or constituents acting in a protective manner (vitamins, antioxidants, detoxifying enzyme-activating substances, etc.). It is clear that carcinogen metabolism-affecting polymorphisms may modify probability of contact between carcinogens and target cells, thus acting at the stage of cancer initiation. Dietary factors can certainly interact with hormonal regulation. Obesity strongly affects hormonal status. At the same time some food components, such as phytoestrogens are known to be processed by the same metabolic pathways as sex hormones, thus their cancer-preventive effect can be modulated by the polymorphisms.

APPLICATION OF NUTRIGENOMICS

The isolation of microsomal ω-6-desaturated gene from soybean was carried out for improving soybean seed oil profile, making it nutritionally more beneficial and stable for human consumption. Psoyatose supplementation results in an increased transcription of fibroin mRNA leading to an increased silk production of Bombyx mori L. demonstrated that soyprotein extract supplementation is of importance in regulating the fibroin gene expression at transcriptional level. Nutrigenomic analysis of intestinal response to partial soybean meal (SBM) replacement in juvenile Atlantic halibut (Hippoglossus hippoglossus L.) showed that there is no significant difference between fish fed with the fish meal (FM) and SBM diets.[30] A dialyzed aqueous extract of fenugreek seeds was investigated in vivo for hypoglycaemic potential and its effects on insulin signalling pathways in the primary targets of insulin, adipocytes and liver cells, were examined in vitro, by the use of mechanism-based innovative contemporary strategies.[30] A successful attempt of fortifying human diets with natural α - tocopherol was done by taking recourse to genetic engineering of an important oilseed crop, Brassica juncea. α - Tocopherol intakes in excess of the recommended daily allowance (RDA) are associated with decreased risk of cardiovascular diseases, improved immune function, slowing of the progression of a number of degenerative human. Curcumin and its dietary source turmeric are important for the prevention and/or treatment of diabetic retinopathy. The vascular-endothelial-growth-factor (VEGF) expression analyzed by both real time polymerase chain reaction (PCR) and immunoblotting showed that curcumin and its dietary source turmeric can inhibit VEGF expression in strepotzotocin (STZ)-induced diabetic rat retina.[31]

Nutrigenomics and Periodontal Disease

Periodontitis is a ubiquitous chronic inflammatory disease affecting the supporting structures of the teeth and if not promptly recognised and correctly managed can ultimately lead to tooth loss resulting in reduced masticatory function and subsequent alterations in dietary intake and nutritional status. The importance of successful management and treatment of periodontitis has gained added press in recent years with the recognition that periodontitis is a risk factor for a number of important systemic diseases, which include cardiovascular disease, diabetes and rheumatoid arthritis. Risk factors are important in the development and propagation of periodontal disease and act predominantly via modification of the host response to bacterial challenge, resulting in less effective clearing of pathogenic species and inflammation resolution which in turn increases host mediated tissue damage. These factors can be characterised as genetic, environmental (e.g. stress, bacterial challenge) and lifestyle/behavioural (e.g. exercise, nutrition, smoking).

Over the last few years improved understanding of ways to assess and investigate nutritional status has emerged along with the recognition of the importance of assessing nutritional intake, body composition and biochemical measures of nutrition. Measuring serum levels of various micronutrients alleviates issues surrounding self-reporting of dietary intake (poor compliance) and inadequate absorption of the dietary supplement. However, it is suggested that periodontitis is associated with reduced serum micronutrient levels, this may be due to a number of reasons including poor diet, lifestyle factors (e.g. smoking) and/or genetic factors which impact on absorption, distribution, bioavailability and synthesis of micronutrients.

Role of Nutrition in Periodontal disease

It has been acknowledged for many years that nutritional intake can impact upon the levels of inflammation seen in a number of diseases, and this is no less the case in periodontitis. Research studies using an experimental gingivitis model have shown increased levels of bleeding on probing when participants were fed with a diet high in carbohydrates when compared to those on a low sugar diet.[32] This finding has been further supported by a study investigating volunteers placed on a primitive diet which was high in fibre, anti-oxidants, and fish oils, but low in refined sugars and with no oral hygiene measures. Increases in oxidative stress is antagonised by a complex system of antioxidants which include antioxidant vitamins, however it has been

demonstrated that the most important small molecule antioxidant species is glutathione. Glutathione exists in both oxidised (GSSG) and reduced (GSH) forms. The ratio of GSH to GSSG normally favours GSH which maintains a reduced state inside the cell. GSH has a number of key characteristics that underpin its importance in antioxidant defence; it is a potent scavenger of free radicals and plays a key role in a number of other protective antioxidant systems. Other important functions of GSH include roles in cell metabolism and DNA synthesis and repair. The scientific literature identifies several studies that show that depletion of glutathione is associated with increased levels of periodontal disease. [33]

How can diet reduce oxidative stress?

Control of dietary sugar and fat intake can help reduce levels of oxidative stress and downstream inflammatory sequelae. Reductions in simple sugars, refined carbohydrates and saturated fats reduces activation of a diverse range of pathways thereby reducing oxidative stress. As previously discussed, research has shown the importance of total amounts of simple sugars, carbohydrates and fat intake entering the blood stream, but it also indicates that frequency of intake is also a key factor in generating oxidative stress, the more frequent the intake the greater the inflammation recorded in blood vessels.[34] Foods rich in antioxidants may help reduce oxidative stress, for example green leafy vegetables (broccoli, spinach etc.), berries (e.g. blueberries, blackberries, cranberries, strawberries etc.), red beans, red wine, and dark chocolate with greater than 70% cocoa are all rich in key antioxidant micronutrients. Other ways include diets that slow down gastric emptying (digestion) resulting in less pronounced spikes in blood glucose, examples of which include nuts, olive and fish oils which also have antioxidant properties, further adding to their effectiveness.[35] The physiological impact of nutrients on the host depends upon many factors including bioavailability following transit through the digestive system, absorption from the gut into the circulation, conversion to a bioactive form and transport to target cells. A dietary supplement may show excellent antioxidant/anti-inflammatory properties in the laboratory, but have no clinical efficacy if it fails to reach the target tissues in an active form. It is also becoming clear that individual differences in genetic makeup account for a diverse array of responses to dietary supplementation, something that needs to be taken into account when a new dietary intervention is proposed, i.e. “one size may not fit all”.

The Challenges and Potential of Nutrigenomics and Nutrigenetics

The potential of nutrigenomic and nutrigenetic approaches is starting to be realized. A great deal of progress has already been made and, by applying new analytical tools to the data already generated, it has proven possible not only to obtain lists of gene products and metabolites that change in response under defined conditions but also to gain insights into the overall biological processes involved. Another emerging challenge that may well carry implications for the development of obesity research is that of ‘epigenomics’. This can be defined as the study of heritable epigenetic signals, encoded in patterns of DNA methylation and histone acetylation within the chromatin, that modulate the expression of genes. Epigenetic marks have recently been shown to change in response to environmental factors over an individual’s lifetime, so even identical twins may ultimately develop differing susceptibilities to adverse environmental factors. As the massive task of mapping the human epigenome progresses, it will become possible to explore the role of epigenetic effects, both as causes and possible consequences of obesity.

Not least among the many challenges are the needs for quality control, standardization, data capture and storage of nutrigenomic and nutrigenetic data. The ‘omic’ tools produce vast quantities of data rapidly. If we are to make use of this information, rather than drown in the flood, it is essential that the data collected are of high quality and are captured in a manner that enables them to be stored and exchanged readily. Standardization of data capture for microarray studies has already been addressed and equivalent procedures are in development for proteomic and metabolomic studies. Refinements to these data capture systems are likely to include appropriate

data-quality metrics and specialty-specific metadata.

CONCLUSION

Nutrigenomics shows a new way of working with nutrition and now, the knowledge of how food interferes with the genetic code and how the organism responds to these interferences and with the phenotype can be clarified. Furthermore, the development inside Nutrition Sciences, together with communication and marketing fields, provided the emergence of a personalized nutritional counseling based in Nutrigenomics. The personalized nutritional counseling can be used not only to change diet habits and improve life style, but also mainly will permit a better diagnostic of certain diseases, retard the evolution of chronic illnesses, and assist on the treatment of others.

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REFERENCES

1. DeBusk R. Diet-related disease, nutritional genomics, and food and nutrition professionals. J Am Diet Assoc 2009;109:410-413.
2. Drayna D. Human taste genetics. Annu Rev Genomics Hum Genet 2005;6:217- 235.
3. Ordovas JM. Genotype-phenotype associations: modulation by diet and obesity. Obesity (Silver Spring) 2008;16 Suppl 3:S40-46.
4. Ordovas JM, Kaput J, Corella D. Nutrition in the genomics era: cardiovascular disease risk and the Mediterranean diet. Mol Nutr Food Res 2007;51:1293-1299.
5. Eny KM, Sohemy A El. Genetic determinants of ingestive behaviour: sensory, energy homeostasis and food reward aspects of ingestive behaviour. In Dube L, Bechara A, Dagher A, Drewnowski A, LeBel J, James P, et al. Obesity Prevention: the role of society and brain on individual behavior. 2010; pp.149-160. Elsevier Inc.
6. Swallow DM. Genetics of lactase persistence and lactose intolerance. Annu Rev Genet 2003;37:197-219.
7. DiLella AG, Kwok SC, Ledley FD, Marvit J, Woo SL. Molecular structure and polymorphic map of the human phenylalanine hydroxylase gene. Biochemistry1986;25:743-749.
8. Office of Population Genomics, National Human Genome Research Institute, and National Institutes of Health. A Catalog of Published Genome-Wide Association Studies 2009.
9. Sohemy A El, Stewart L, Khataan N, Fontaine-Bisson B, Kwong P, Ozsungur S, et al. Nutrigenomics of taste - impact on food preferences and food production. Forum Nutr 2007;60:176-182.
10. Kaput J, Rodriguez RL. Nutritional genomics: the next frontier in the postgenomic era. Physiol. Genomics 2004;16:166–177.
11. Jimenez-Sanchez G, Childs B, Valle D. Human disease genes. Nature (London) 2001;409:853–855.
12. Goppert F. Galaktosurie nach Milchzuckergabe beiangeborenem, familiaerem chronischem Leberleiden. Klin.Wschr.1917; 54: 473–477.
13. Segal E, Friedman N, Koller D, Regev A. A module map showing conditional activity of expression modules in cancer. Nat Genet 2004;36,1090–1098.
14. Singh RB, et al. Effect of an Indo- Mediterranean diet on progression of coronary artery disease in high risk patients (Indo-Mediterranean diet heart study): A randomized single-blind trial. Lancet 2002;360:1455–61.
15. Mohan V, Sandeep S, Deepa R, Shah B, Varghese C. Epidemiology of type 2 diabetes: Indian scenario. Indian J Med Res 2007;125:217–30.
16. Gomase VS, Tripathi AK, Tagore S. Genomics: new aspect of cancer research. Int J Syst Biol 2009;1:1–19.
17. Nair S and Pillai MR. Human papillomavirus and disease mechanisms: Relevance to oral and cervical cancers. Oral Dis 2005;11:350–359.
18. Lusis AJ. Atherosclerosis. Nature 2000;407:233–41.
19. Hooper L, Summerbell CD, Higgins JPT, Thompson RL, Capps NE, Smith GD, et al. Dietary fat intake and prevention of cardiovascular disease: systemic review. Br Med J 2001;322:757–63.
20. Ordovas JM. The quest for cardiovascular health in the genomic era: Nutrigenetics and plasma lipoproteins. Proc Nutr Soc 2004; 63, 145–152.
21. Knoblauch H, Bauerfeind A, Krahenbuhl C, Daury A, Rohde K, Bejanin S, et al. Common haplotypes in five genes influence genetic variance of LDL and HDL cholesterol in the general population. Hum Mol Genet 2002;11:1477–485.
22. Meltzer S, Leiter L, Daneman D, Gerstein HC, Lau D, Ludwig S, et al. Clinical practice guidelines for the management of diabetes in Canada. Canadian Diabetes Association. CMAJ 1998;159(Suppl 8):S1-29.

23. Offenbacher S, Salvi GE. Induction of prostaglandin release from macrophages by bacterial endotoxin. Clin Infect Dis 1999;28:505-13.
24. Wolford JK, Vozarova de Courten B. Genetic basis of type 2 diabetes mellitus: implications for therapy. Treat Endocrinol 2004;3:257e67.
25. Flint J, Valdar W, Shifman S, Mott R. Strategies for mapping and cloning quantitative trait genes in rodents. Nat Rev Genet 2005;6:271e86.
26. Perusse L, Rankinen T, Zuberi A, Chagnon YC, Weisnagel SJ, Argyropoulos G, et al. The human obesity gene map: The 2004 update. Obes Res 2005;13:381–490.
27. Altshuler D, Brooks LD, Chakravarti A, Collins FS, Daly MJ, Donnelly P. A haplotype map of the human genome. Nature 2005;437:1299–320.
28. Anderle P, Farmer P, Berger A, Roberts MA. Nutrigenomic approach to understanding the mechanisms by which dietary long-chain fatty acids induce gene signals and control mechanisms involved in carcinogenesis. Nutrition 2004;20:103–8.
29. Fenech. The genome health clinic and genome health nutrigenomics concepts: Diagnosis and epigenome damage on an individual basis. Mutagenesis, Oxford Journals 2005;20:255–69.
30. Kumar A, Singh RP. Obesity: prevalence, manife--stations and dietary patterns: A review. J Food Sci Technol 2009;46:287–99.
31. Lau FC, Bagchi M, Sen C, Roy S, Bagchi D. Nutrigenomics analysis of diet-gene interactions on functional supplements for weight management. Curr Genomics 2008;9:239–251.
32. Sidi A, Ashley F. Influence of frequent sugar intakes on experimental gingivitis. J Periodontol 1984;55:419-423.
33. Chapple IL, Brock G, Eftimiadi C, et al. Glutathione in gingival crevicular fluid and its relation to local antioxidant capacity in periodontal health and disease. Mol Pathol 2002;55:367-73.
34. Esposito K, Ciotola M, Carleo D, et al. Post-meal glucose peaks at home associate with carotid intima-media thickness in type 2 diabetes. J Clin Endocrin Metabol 2008;93:1345-350.
35. Chapple ILC. Potential mechanisms underpinning the nutritional modulation of periodontal. J Am Dent Assoc 2009;1402:178-84.

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