Chronic diseases and Nutrigenomics

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Abstract

Nutrigenomics is an emerging approach in the field of nutritional research which studies gene-diet interactions and can provide a key role in adding knowledge for the prevention and management of a wide range of diseases. Today, the concept of nutrigenomics is not new in the area of the scientific and medical technology for the contribution and light on the mechanism on the induction and progression of a number of chronic diseases like obesity, hypertension, diabetes, cardiovascular diseases and cancer. Study related to nutrigenomics is one step forward in nutritional research involving the techniques of nutrition, molecular biology, genomics, bioinformatics, molecular medicine and epidemiology together to understand the role of food as an epigenetic factor which unravels its role in the occurrence of these diseases. Hence, under the prevailing scenario of world health, it has become an urgency to boost nutrigenomics and related research to find cure for chronic diseases caused due to nutrition and allied environmental factors. Thus, such a type of research findings ensures the effective benefit of genomic revolution for mankind in the near future.

Keywords: Nutrigenomics, Obesity, Hypertension, Diabetes, CVD, Cancer

Introduction

A globally emerging science, Nutrigenomics, studies about the effect of genetic variations inresponse to food. In today's molecular era, every nutrient is considered as "signalling molecules" through which dietary signals are transmitted and translated into the cell and it changes the gene expression in the nucleus leading to the alterations in protein and metabolite expression within the cellular system (Wellen, 2005). Here comes a big question: what actually happens within the cellular system whenever a food is taken in less or excess amounts? The science of nutrigenomics actually studies the mechanism of food- gene interlinking signalling and is based on three main mechanisms i.e. firstly, there exists a strong inherited diversity of genomes between different ethnic groups and individuals affected by the bioavailability of nutrients and their metabolism. Secondly, cultural, geographical, economical, and taste perception differences in choice and food habit/ nutrient availability may differ every person greatly with each other. Thirdly, malnutrition (excess or deficiency) can affect gene expression and genome stability.

In the present, nutrition research has shown a greater concentration on nutrient deficiencies leading to impairment of health and fitness. The advent of genomics can be considered as a suite of high throughput technologies for the purpose of generating, processing, and applying scientific knowledge related to the chemical constituents and functions of genomes. This important information can create endless opportunities for enhancing the concepts of the science behind modulation of genes by nutrients and the expression of protein which ultimately effects the metabolism of cells and organism. Nutrigenomics is the combination of molecular nutrition and genomics as it is the junction between health, diet, and genomics. The vast scope of nutrigenomics can help in the promotion of an increased or a higher approach of understanding the mechanism of effect of nutrition on metabolic pathways and homeostatic controls, understanding how the regulation alters in the early phases of diseases related to diet, and to know the extent of contribution in such type of diseases by individual sensitizing genotypes. In general, the study of nutrigenomics can contribute to planning strategies which are truly evidence- based and developing dietary intervention for the restoration of health, prevention and management of diet and lifestyle related diseases.

Gene, nutrients and chronic diseases: scope of nutrigenomics

As nutrigenomics deals with the complexities of the genome of humans, it has the efficiency to decipher variability of genome in terms of wide range of nutrient concentration and a variety of food through identification of

specific dietary signal, signal sensor or receptors. It has been evident by nutrigenomics research that each nutrient has multiple target sites with various affinities and specificities (Ruden et al, 2016). Studies have also recognised mechanisms of specialized cellular- sensing in the human body which consider nutrients and dietary metabolites as signalling elements (Müller, 2003). Naturally, the structure of molecules of each nutrient are made up in a way that it contains all the information regarding the activation of every specific signalling pathway for hitting the target site. The design is so sensitive that even a minute structural change can affect the activation of sensor pathways to a great extent. For the scientist and technologists, actually it is a great challenge to recognise and identify the pathways of molecules and the up/downstream regulation by every nutrient. Study of the science of nutrition together with genomics can help to identify molecular pathways by genome-wide characterization of nutritional target genes.

Research in this field is basically based on the principle of nutrition-gene- disease interaction of individuals and developing a scientific way to protect mankind with a wide range of diseases including chronic diseases as most of the diseases are mediated by exposure of particular food components. The detection of gene alteration and cellular modification done by food is done by biomarkers which may be lipid profile, blood pressure, insulin sensitivity or a metabolic syndrome. These biomarkers by nature are generally single proteins or metabolites or specific body functions or responses. The information generated through biomarkers proves to be a detector for proteomics and metabolic changes in the body which depends on the particular person’s genotype and may be a potential causative agent of a variety of chronic diseases. In the light of this wisdom the researchers can get a great help for understanding the action of nutrients and its linkage with diet which act as an important role in fitness and diseases. Planning healthful food, advices related to lifestyle and dietary interventions can be made possible by the intense research in the field of nutrigenomics. Broadly, it indeed requires a collaborative effort in the protection of mankind from chronic diseases and thereby maintaining a better balance in genetics and public health industries, food science andculinary. The future of nutrition science can be spread in the extend of prescribing personalized diet charts by the dieticians based on genomic construction and present trends leading to lifestyle diseases. Present chapter focuses the approach of nutrigenomics based on the interaction of gene- diet in the relevance to present and existing researches for understanding its present and future prospect.

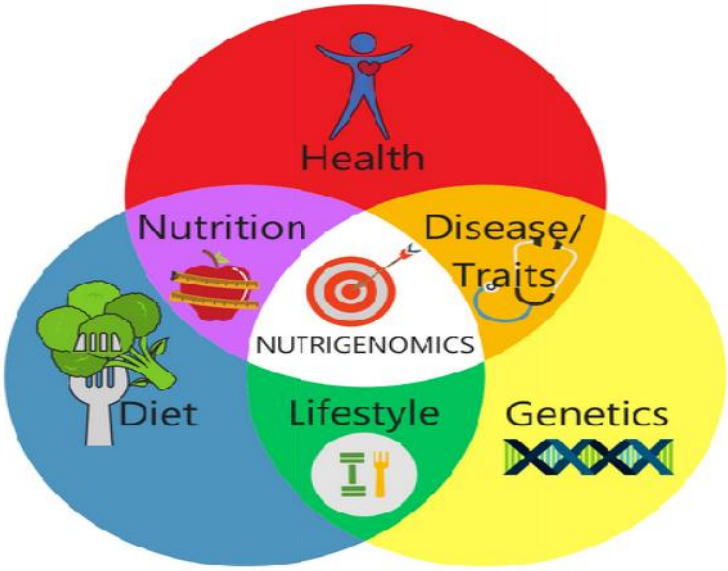

Figure- Relationship of nutrigenomics with diseases, health, genes and nutrition.

Source- adopted from https://goqii.com/blog/nutrigenomics-you/

Obesity and Nutrigenomics

Obesity is a chronic low – grade nutrition related disorder prevalent and significant among all age groups globally. Obesity is caused by various factors, including environment, behaviour, and genetics. Since 2006, more than 50 genes have been found associated with obesity. A range of co-morbidities and complexities associated with obesity includes hyperinsulinemia, hypertension, type 2 diabetes, cardiovascular diseases and a number of cancers (Ukkola and Bouchard, 2001, Maury et al, 2010). Though there has been the contribution of multifactorial etiology in the progression and development of obesity, lifestyle changes and physical activity are considered as the most important elements to treat it (Bary, 2008). As abnormal lifestyle practices lead to the progression of obesity, nutrigenomics and nutrigenetics has a vital role and contribution for its study. It has been believed that obesity is essentially present in an environment which contains positive factors or an obesogenic environment giving rise to it, but the studies of nutrigenomics denies it. Many studies states that the occurrence of obesity is based on genetic variability and environment factors that promotes obesity as dietary habits, alcohol, tobacco, substance abuse, sleep, age, gender, ethnicity, physical activity, use of medications and

depression (Nakamura et al, 2015, Nettleton et al, 2015, Reddon et al, 2016). Nutrigenomics gives a conclusive statement that the primary cause of obesity is the environmental factors in relation to gene-nutrient-disease interaction. If a person follows a healthy dietary practice with proper and specific timings and indulge in physical activity then he/ she can avoid obesity and its related comorbidities to a great extent even after the presence of an obesogenic environment. Nutrigenomics studies the interactions of genome, which are so complex in nature and explains its regulation differences among the obese phenotype that vary both within and across populations (Joffe YT et al, 2010, Joffe YT et al. 2011, Stryjecki, 2011).

However, the deposition of energy in the body is the result of increased energy intake and decreased energy output. The concept explains about resting metabolic rate, absorption and metabolism of dietary nutrients, heat production or thermogenesis, and physical activity stating energy which is represented in the form of calories states that excess of energy creates a positive balance results to promote deposition of triacylglycerol within adipose tissue. Likely, in vice-versa condition, a state of negative energy balance results to promote lipolysis of triacylglycerol and mobilization of fatty acids from adipose tissue (Hill et al, 2012).

Association of epigenetics and obesity explains the science behind the fact known to us that the embryo in mother's womb can depict a dramatic variation in the phenotype without changing the genomic constitution of the offspring while providing a moderate environmental constraint during specific periods of time in the development of the embryo. Prenatal and early postnatal periods also have a critical role in initiation of obesity. Studies proved that the nutritional environment of the fetus can enhance the susceptibility to develop obesity in later life due to the role of epigenetics (Goldberg et al. 2007). Thus, epigenetics are potent enough to induce heritable changes in gene expression without altering the gene sequences. Basically epigenetics is the integral regulating and determining factor of expression of a specific gene. The study on detailed methylation action pattern of epigenetics explains methylation at the 5´position of cytosine in DNA within a CpG (cytosine and guanine nucleotides linked by phosphate) dinucleotide is very common in mammalian genomes and leave a stable epigenetic mark which is transmitted through DNA replication and cell division (Bird, 2002).

In addition to these, different studies and experiments by nutrigenomic scientists show versatile results related to obesity. Some facts show that lower birth weight infants who undergo early catch-up in growth have higher chances

of becoming obese in later life as compared with infants of high body weight at birth. The reason behind is accumulation of greater fat mass than the lean body mass in infants (Ong et al, 2000). Similarly, it has also been concluded that the infants with lower birth weight who were formula milk fed and having a catch-up growth show higher chances of cardio- vascular disease in later life (Singhal, 2006). Greater incidence of obesity in adults has also been reported in formula fed as compared to formula fed during infancy, though there are exceptions too (Owen, 2005).

These examples illustrate that it is not enough to be told or even to know what impact a gene has on human body function and what disease associations have been reported in the scientific literature. Nutrigenomics must exist in the context of nutrition science to provide a deeper understanding of the biochemical pathways that have been impacted. A detailed and advanced understanding of nutrigenomics can prove to be a vital element in developing effective weight management interventions.

Hypertension and Nutrigenomics

It is a well-known fact that genetics and heredity plays a crucial role in the development of hypertension in an individual with an average increased risk of four fold. But despite this fact the gene identification in hypertension has been a slower process. A large number of genes are involved in blood pressure control pathways so these have small effects on the phenotypes being measured. Because of the importance of blood pressure control, there are surplus compensatory pathways for blood pressure normalization. Therefore, a gene that may compromise one pathway may not be found to be associated with hypertension because other pathways can adequately compensate and normalize the phenotypes being studied (Hunt, 2010). In Asian populations risk influencing polymorphisms in certain genes have been identified which serve crucial in testing for predisposition to hypertension. A person with such variations in the DNA makeup is more likely to develop hypertension in earlier life.

There are a variety of factors contributing to the progression and development of hypertension, including genetic and environmental factors.

Oxidative stress, inflammation and autoimmune dysfunction initiate and propagate hypertension and cardiovascular disease. There is a role for the selected use of single and component nutraceutical supplements, vitamins, antioxidants and minerals in the treatment of hypertension based on scientifically controlled studies which complement optimal nutrition, coupled with other lifestyle modifications (Housten, 2014).

The pathogenesis of hypertension involves the complex mechanism of various pathophysiological processes which in turn reflects the variation at individual level as it depends on both the environmental exposures and inherited genetic makeup. Acute compensating mechanisms can hide the initiating gene effects during or after an intervention, so that early phenotype assessments during the intervention may be more likely to detect the genetic initiators. On the other hand the compensatory mechanisms are variably effective in minimizing the blood pressure rise making the genetic initiating mechanisms more difficult to understand (Hunt, 2010).

Diabetes and Nutrigenomics

Diabetes mellitus (DM) is a syndrome of multiple aetiology. The main characteristic is hyperglycemia (high concentrations of glucose in the bloodstream) with dysfunctions in the metabolism of protein and lipids. The hyperglycaemia is generally associated with insufficient insulin production, secretion, absorption or set of more than abnormality (Kuzuya et al, 2002). As the disease progresses, it can affect various organs as blood vessels, heart and kidneys adversely and lead to greater complications. Type 2 diabetes (T2DM) or non-insulin dependent diabetes mellitus represents 90% of cases, occurring when the body is not able to produce or absorb sufficient insulin for proper maintenance of blood glucose. Diabetes shows a pathogenesis associated with genetic and environmental aspects. Obesity increases the risk for developing diabetes for about 10 times. People with sedentary, modernised lifestyles with greater intake of refined carbohydrates, red meats and saturated fats are likely to develop obesity and chances of diabetes increases with it. The main reason behind is, both obesity and diabetes has a common characteristic of irregular secretion creating insulin resistance. Insulin hormone is secreted from β-cells of pancreas and is an important controller of glucose as well as fat metabolism in the human body (Popkin, 2001). Insulin resistance and pancreatic β-cells dysfunction are associated with oxidative stress. An oxidative stress is the occurrence of an imbalance between the production of free radicals and the defending antioxidants that results in the damage of vital biomolecules and DNA, protein and lipids (Wu and Cederbaum, 2003).

Nutrigenomics studies the role of genes in the regulation and expression of antioxidant protein that acts as a protective agent against oxidative stress. It has been proved that specific genes are responsible for preventing the production of glucose, cholesterol and triglycerides by promoting the oxidation of fatty acids. In addition, genes are also responsible for the de acetylation processes, thereby modulating several other genes in the body and hence can

control glucose production, lipid metabolism and insulin production to a significant level (Price et al, 2012, Rato et al, 2014). Similarly, there are many genes affecting the pancreatic cells and inflammatory responses on the body. The nutrigenomics has a greater role in management and prevention of diabetes by the inclusion of certain functional foods. These are foods with naturally occurring bioactive components which directly interactwith the genome and can perform an additional health benefit to the body together with basic nutritional function (Henry, 2010).

Studies on bioactive components as flavonoids which includes flavones, flavonols, anthrocyanins shows protective role on maintenance of glucose in the body. Specific molecules such as luteolin, quercetin and others have a greater impact on the intracellular signalling pathways. (Kerimi, 2016) These pathways are directly related to insulin secretion, glucose uptake, enhancing mitochondrial pathways and suppression of inflammatory cytokines. Phenolic compounds are also responsible for carbohydrate metabolism and digestion (Xiao et al, 2015). The health care professionals including doctors and dieticians are working in accordance with these follow- up studies to promote higher consumption of bioactive compounds particularly apples, pears, and blueberries which are inversely associated with T2DM (Wedick et al, 2012). Phenolic compounds present in coffee also play a protective role in T2DM. It has been seen that Resveratrol, a natural phenol present in the skin of grapes, blueberries, raspberries, peanuts and red wine, are helpful in reducing the risk of complications of diabetes in various organs including liver (Bagul, 2015). In addition, nutritional biochemistry and gene studies also relate the risk of prevalence and predisposition of diseases as T2DM due to the deficiency of certain micronutrients. Afzal et al., concluded the fact that lower vitamin D levels indicates a risk factor for incident of T2DM in humans. Timely supplementation and action can reduce the chances of developing the disease.

With the gene variation studies and related information one way to prevent T2DM is to know the gene and identify the food and role of specific nutrient that can directly interact with it and bring about a positive outcome in health. Many of the bioactive foods as flavones, anthrocyanins, quercetin, resveratrol and any more have established a positive effects on insulin production, inflammatory processes and macronutrient metabolism to prevent T2DM yet, there is a need of further study on the role of these genes in the prevention and treatment of the disease. Most of the studies have already established the evidence to prove the change in the expression of the genes or not but still there is a need of more information about the mode of action of these bioactive components and micronutrients to understand the mechanism to

such an extent that a treatment of diabetes can be established. This knowledge can give a clearer idea on the aetiology or control of the disease as there are not many studies on the compounds that can modulate their expression.

Cardiovascular disease and Nutrigenomics

Disease affecting the heart and blood vessels including heart arteries, capillaries and veins is known as cardiovascular diseases (CVD). It includes atherosclerosis, coronary and ischemic heart diseases. Individuals with CVD have plaques throughout the inner walls of arteries of the heart and are extremely prone for heart attacks. The causative and progressive factors of CVD have a multifactorial spectrum including age, gender and genomic constitution. A stressful and busy life leading to other risk factors such as hypertension, hyperlipidaemia, obesity, diabetes, substance abuse and thrombosis are also very potent in the induction of CVD.

Interestingly, the effects of dietary factors as high fat content shows different effects in individuals as some respond with high cholesterol levels while others have normal levels of cholesterol. This is due to genetic variation or polymorphisms, based on genetic makeup or genotype. In the genes, the polymorphism involved in the development of atherogenesis, especially in the metabolism of lipoprotein, determines the exact mechanism and action on how an individual's blood vessels respond to a particular dietary factor such as fibre. The response on various parameters can be determined by nutrigenomics as cholesterol, low- density lipoprotein- cholesterol (LDLC), high density lipoprotein-cholesterol (HDL-C), triglycerides, and lipoprotein particles (Engler, 2009). The nutrigenomics consider diet as an environmental factor and has a direct connection with the development of the disease. Based on the complex aetiology of CVD which has both genetic and environmental components, nutrigenomics narrates some dietary recommendations based on the genetic constitutions of an individual for the prevention and management of CVD (Juma et al, 2014). Various studies has well proved the fact that diet compositions can have a direct link with the development of CVD in every age and ethnicity (Hooper, 2001, Corella, 2009).

American Heart Association provides a detailed and comprehensive review and recommendations describing the importance of nutrigenomics in CVD. Genetic linkage studies and gene linkage studies show evidence on the connection of genetic basis of myocardial infarction and atherosclerotic CVD (Arnett, 2007). It has been proved in gene association studies that that gene even has an influence on a specific trait such as high LDL cholesterol or low HDL cholesterol. The likely genes associated with specific traits related to

CVD are selected and tested for single- nucleotide polymorphism (SNP) for both, carrier of the trait and non- carrier (control). The genome study on CVD focuses on the analysis of linkage of families to examine genetic and phenotypic information as high blood cholesterol levels (Franchini, 2008). By identifying the traits that are inherited along with mutations, this information can greatly help in finding the disease- causing mutations of genes. Specific polymorphism in genes encoding lipid transport proteins, their receptors, and lipid-processing enzymes and inflammation related proteins are also reported to be associated with the characteristic changes in blood lipid concentrations (Weinberg, 2002). Moreover, the role of atherosclerosis in the pathogenesis of CVD, describes that a complex combination of lipid transport and metabolism disorder with chronic inflammation can be regarded as a major key factor. It has been observed in nutrigenomics studies that the levels of total cholesterol, LDL cholesterol, and triglycerides elevated permanently in the blood plasma is causative agent in the progression of coronary artery plagues and increased levels of high density lipoprotein (HDL) depicts a protective role for CVD (Loktionov, 2003).

For identifying susceptible individuals for CVD it is quite important to screen family history as CVD and its risk factors are multifactorial with many genes interacting with each other. As behavioural and environmental factors play an important role in inducing CVD, the role of diet come in major picture for prevention and management of the disease. However, a deep and intense debate/ discussion on the best suitable dietary plan consisting of combination of macronutrients specially the amount of total fats along with different fatty acids as polyunsaturated fatty acids content is important (Jacobsen, 2009). Based on the facts, counselling related to nutrition related changes promoting optimum weight and normalising blood lipid profiles is the best approach to prevent CVD.

Thus, it is clear and evident from several studies that risk of CVD can be determined by the interaction between genetic variations and dietary intake the scope of nutrigenomics can be promising in minimising the risk of CVD in individuals. As the genetic variation in CVD is well established, the future research and scope of nutrigenomics should majorly focus on the characterization of disease associated genes and its variants across individuals, communities and populations. This knowledge will foster the use of nutrigenomics in personalized nutrition therapy to promote heath health and prevent CVD in the long term.

Cancer and Nutrigenomics

The incidence of cancer has been projected to increase in future and there is a huge need for effective preventive strategy to face this challenge globally. Cancer is a disease characterised by a multistage process in which gene expression, protein and metabolite function of the body disrupts. Today, significant understanding of the disease is available about the cellular event which initiates the activation of carcinogenesis upon modulation by dietary and other environmental factors (Go et al, 2003). Inherited mutation of genes and gene diet interaction increases the risk of cancer. Endogenous reactions as oxidative stress and excess exposure to exogenous agents as ultraviolet rays from sunlight, high intake of alcohol and tobacco and high doses of other ionizing radiations causes cancer (Setlow, 2001).

For maintaining homeostasis of the body it is essential to have a proper communication between nutrition, metabolism and gene expression. The synergy between these components determines an individual's health and his/her susceptibility to develop chronic diseases. It is a notable fact that the transcription factors at the molecular level are regulated by nutrients which are modified by the genetic expression to adjust metabolic responses for the development and progression of serious conditions such as cancer (Debusk et al, 2010). Diet of any individual on the basis of quality can be a source of protective, carcinogenic and mutagenic agents which are metabolized by the enzymes of the process of biotransformation. Genetic polymorphism that can change the protein expression or the function of these enzymes can modify the susceptibility for cancer. Food consumed by humans contains thousands of bioactive compounds that have a role in the pathogenesis of cancer. Among these around 500 bioactive food ingredients have already been studied and proved to be possible predisposing agents (Komduur, 2011).Certain vitamins, antioxidants and detoxifying enzyme activating substances found in fruits and vegetables acts in a protective manner for carcinogens by mechanisms such as blocking metabolic activation through increasing detoxification (Keum, 2004). Detoxification enzymes as flavonoids, phenols, isothiocyanates, allylsulfur compounds, indoles, and selenium can be modulated on consuming plant foods. The initiation stage of cancer starts with the polymorphism by the contact of carcinogen and target cell due to the metabolism of carcinogens in the body. Tumours in breasts, prostate, ovaries and endometrial lining, which are hormone dependent, influences polymorphisms of gene encoding factors involved in hormonal regulation are most strongly manifested. Polymorphisms in sex hormone receptor genes comprising those encoding oestrogen receptors, progesterone receptor, and androgen receptor have been shown to be associated

with cancer risk modulation (Lotionov, 2003). Dietary factors can influence the hormonal regulation of the body as food components like phytoestrogens can be processed by the pathways similar to sex hormones (Adlercreutz, 2002).

Genetic studies have shown various examples on the effect of diets on the risk of cancer. High consumption of red meat increases the risk of colon cancer, high intake of dietary irritants as salts and certain preservatives is carcinogen for gastric cancer (Turnpenny, 2007), increased risk for cancer is associated with low consumption of vitamin B12, vitamin B6 and methionine in the diet (Slattery, 1999), dietary fibres and fish oil have a potent protective role in prevention of colon cancer (Calder, 1998).

Thus, nutrigenomics studies on the prevention of cancer reported the evidence that all the major signalling pathways deregulated in various types of cancers are exclusively affected by nutrients. In recent years, the major emphasis is laid on decoding the underlying molecular mechanisms of the activities of these agents, although the results are a bit complicated as the effect of different nutrients is exclusively cell type- specific and dose- dependent. Dietary components are considered to be the major determinants of risk of cancer in humans due to the fact that genetic polymorphism alteration happens in response to absorption and metabolism influenced by dietary components. DNA methylation patterns changes by the epigenetic events and thereby influencing an overall change in gene expression occurs in response to food components. The importance of nutritional chemistry is and nutrient gene interaction powerful effects are obvious as per the above state studies on the prevention of cancer. For the future strategies of cancer care, studies of dietary components using cellular model systems in nutrigenomics can provide a better understanding of inter- relations among different elements and pathogenesis. As the field of molecular nutrition expands and the functions of the human genome are better understood, a greater understanding of how foods and their components influence cancer will ensue.

Conclusion

As a rapidly growing field of science, nutrigenomics has opened a wide variety of fields in the studies of nutrition and gene relationship. Nutrigenomics is expected to bring a revolution in the healthcare system by formulating tailor-made diets and personalized nutrition for optimum health and disease prevention. Based on genetic variation among individuals it can influence the responsiveness to dietary factors or nutrients, specific dietary recommendations and accordingly nutritional interventions could be made. With the knowledge of nutrigenomics the healthcare professionals can render their services more

effectively and this will foster delivery of competent care and facilitate incorporation of these areas into clinical practice, education and research. Due to new advances in genome technology, new disciplines in nutrition research are emerging to facilitate an understanding of the relationship and the interactions between diet and health. Nutrigenomics research can facilitate the approach to evidence-based healthy food or nutrients and therapeutic dietary interventions. An understanding of genetic variants and diet will promote the use of nutrigenomics to personalize nutritional therapy based on an individual's genetic makeup. The genetic education of researchers, clinicians, public health professionals, and the general public is vital in this effort. As substantiation from nutrigenomics studies is growing, the healthcare professionals need an understanding of the integration of nutrition and genomics that may lead to genotype based personalized nutrition. So, nutrigenomics can play a vital role in understanding the genetic makeup and nutrition relationship which in turn promote a healthy life in individuals.

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