Review Article

The role of nutrition related genes and nutrigenetics in understanding the pathogenesis of cancer

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A R T I C L E   I N F O

A B S T R A C T

Nutrition has a predominant and recognizable role in health management. Nutrigenetics is the science that identifies and characterizes gene variants associated with differential response to nutrients and relating this variation to variable disease states especially cancer. This arises from the epidemiological fact that cancer accounts for a high proportion of total morbidity and mortality in adults throughout the world. There is much evidence to support that genetic factors play a key role in the development of cancer; these genetic factors such as DNA instability and gene alterations are affected by nutrition. Nutrition may also lead to aberrant DNA methylation, which in turn contributes to carcinogenesis. The aim of this work is to clarify the basic knowledge about the vital role of nutrition-related genes in various disease states, especially cancer, and to identify nutrigenetics as a new concept that could highlight the relation between nutrition and gene expression. This may help to understand the mechanism and pathogenesis of cancer. The cause of cancer is a complex interplay mechanism of genetic and environmental factors. Dietary nutrient intake is an essential environmental factor and there is a marked variation in cancer development with the same dietary intake between individuals. This could be explained by the variation in their genetic polymorphisms, which leads to emergence of the concept of nutrigenomics and nutrigenetics.

Contents

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1. Nutrigenetics

1.1. Nutrition and health equation

It is an old fact that diet affects health. Hippocrates advised physicians in 400 BC: "Leave your drugs in the chemist's pot if you can heal your patient with food." Furthermore, it is well known that humans differ in their demands for particular nutrients [1].

Nutrition can contribute to disease pathogenesis or appearance either directly or indirectly. Nutrients and foods usually interact with genes in a *benign* manner, but sometimes this interaction can have fatal outcomes [2].

Humans are affected by both environmental and genetic factors; both factors must be considered equally to maintain normal health condition of the individual. Previous studies were mostly directed either to the effect of environmental factors alone or to genes only but not to both together. Recently, research has been designed to study gene–nutrition interaction [3].

Nutrition science investigates how nutrients can maintain normal and stable body homeostasis at the level of the cell, tissues, and organs. This science needs to understand the mechanism of nutrient-dependent interactions at the genetic molecular, protein production and metabolic profile levels [4,5]. Thus, nutrition research has proceeded from epidemiological and physiological aspects to molecular biology and genetics aspects as well as nutritional genomics [6,7].

1.2. Nutrition and genes (diet–gene interaction)

1.2.1. Nutrition and genetic variation

The Human Genome Project offers great help to the science of nutritional genomics. It helps scientists to discover multiple mutual relations between genes, nutrition, and diseases [8]. Sequencing of the human genome revealed significant genetic heterogeneity within human populations. Millions of single nucleotide polymorphisms (SNPs) have been found to have a relation to nutrition [9].

If SNPs are accommodated in the involved genes in metabolism of drugs, environmental agents, or dietary components, they may highly affect the individual response to exposure including diet [10].

1.2.2. Biological complexity of gene–diet interaction

The interaction between nutrition, metabolism, and gene expression is mandatory for maintenance of body homeostasis. Nutrition related or dependent disorders have been reported to be the result of a mixture of nutrients with multiple genes not with a single gene [11,12]. Genetic variation is the major basis for person-to-person divergence in response to diet. Understanding how genetic variation influences gene expression and recognizing genetic variants as risk factors for human nutrition dependent or related disorders is the focus of *nutrigenetics* [13].

1.2.2.1. Nutritional genomics (nutrigenomics and nutrigenetics).
There is an interacting two-way relationship between nutrition and the human genome. It defines and marks the gene expression and metabolic response. Then, it may affect the individual's health condition and susceptibility to disease [14]. Firstly, the genetic background of the individual can define the nutrient state, metabolic response, and susceptibility to diet-dependent or related health disorders [10]. Secondly, nutrients regulate the transcription factors that modify the gene expression, up or down, consequently, adjust the metabolic responses at the molecular level. The reason and result interplay between nutrition and the human genome has led to production of new sub-definitions, nutrigenetics, and nutrigenomics [7].

It is suggested that the science of nutrigenetics is involved in handling the mechanism by which genetic variations define the risk of individual to diseases, nutrient daily requirements, cellular metabolic response and behavior towards the bioactive dietary components or nutritional therapy, the main target of that is to clarify the impact of the gene variability on the interaction between nutrients and diseases. Nutrigenomics science is directed to review and study the genome-broad impact of nutrition. It is interested in the functional effect of various food components on the (-omes) branch of science including genome, transcriptome, proteome, and metabolome [14].

Nutrigenomics and nutrigenetics explore the interaction between nutrients and genes. However, they are clearly distinguished through the mechanisms of interactions between nutrients and genes that determine the risk of development of diseases. Nutrigenomics also

characterizes the impact of all aspects of nutrients as food-based, dietary limitation, or nutritional supplementary agents, on the gene expression. This will direct the interest to the genome-wide effects of nutrients on transcriptome, proteome, and metabolome in cells, tissues, or organisms. Moreover, it will characterize and confirm the genes that can influence the risk of diet-dependent diseases. It may also be useful in understanding how nutrients can affect the metabolic pathways and how these regulations can be inhibited in the early phase of diet-related and diet-dependent diseases [15].

Dolinoy and Jirtle [16] proposed the manner of gene expression, protein expression, and metabolite production as a result of exposure to nutrient and represented it by *dietary signature.* Therefore, nutrigenomics also aims to understand how these dietary signatures have an impact on the cellular function and the balance of the internal environment tissues within the whole body.

- **Principles of nutritional genomics**

    There are four principles of nutritional genomics [10]. (1) Diet is considered be a critical predisposing factor for many diseases in some individuals under particular conditions. (2) Diet ingredients change the gene structure and/or gene expression, and consequently, the human genome. (3) The variation of genotype between individuals can explain the equilibrium between health and disease. (4) Genes that are dependent on dietary factors in its regulation may have a role in the commencement, extent, advancement, and progression of chronic diseases.

- **Examples of nutrigenomics**

    Dietary cholesterol performs an inhibitory effect on the transcription β-hydroxy-β- methyl-glutaryl-CoA reductase gene. Dietary polyunsaturated fatty acids repress mRNA production of fatty acid synthase in hepatocytes through decreasing mRNA for lipogenic enzymes. This process depends on the degree of unsaturation of fatty acids [17].

    Phenylketonuria is an example of single gene mutation. Phenylketonuria patients should avoid phenylalanine-rich food. Many Asian populations have the problem of deficiency of the aldehyde dehydrogenase enzyme, which is responsible for metabolism of ethanol. This leads to an annoying manifestation in affected individuals after ingestion of alcohol. Galactosemia is a disease that results from an inherited genetic deficiency of one of the three enzymes that are implicated in the metabolism of galactose [18].

- **Examples of nutrigenetics**

    The methylenetetrahydrofolate reductase gene *(MTHFR)* is a well-defined example of a gene-nutrient interaction. MTHFR is involved in the metabolism of folic acid and maintenance of the normal blood level of homocysteine. A particular *MTHFR* gene SNP is associated with elevated homocysteine levels of in the blood of carriers, especially if there is a dietary deficiency of folic acid [19].

    Elevated homocysteine level is associated with cardiovascular diseases, and an increased predisposition of colon cancers, especially if there is also a marked dietary deficiency of folic acid compared to the recommended daily requirement [10].

1.2.2.2. Nutritional epigenomics. Many studies in the field of epigenetics have concentrated on the hereditary variation of DNA and consequently protein production, and how DNA and histones are linked, which could prompt certain changes in the chromatin structure without any change in the nucleotide sequence. Therefore alteration in the process of gene expression can result from epigenetic processing rather than change in DNA sequence such as cytosine methylation of DNA and histone acetylation as well as micro RNAs (miRNAs) and noncoding RNAs, which are also implicated in the epigenetic setting and regulation of the process of gene expression. Another study has reported that miRNAs have a role in chromatin remodeling: the mammalian genome carries the genetic code of many types of miRNAs that adjust gene expression through the modulating the mRNAs of interest [20]. These reported results and facts propose that DNA methylation, histone modification, and miRNAs could play integrated roles to regulate the process of gene expression [21].

Nutrients are involved in epigenetic changes that can alter and modify the intracellular signaling pathways [22]. The first concept concerning the role of dietary condition in epigenetic changes and the mechanism by which these epigenetic changes have an effect on the health consequence on long-dated amplitude were reported from the Dutch Famine Cohort. Results from these cohort studies showed that nutritional restriction during pregnancy could lead to an increased rate of predisposition of metabolic disorders in the progeny. The timing and the duration of maternal starvation are significant in determining the disease consequences; the first trimester of pregnancy showed more liability to incidence of diseases during the early adulthood stage. The results of the studies of the Dutch Famine Cohort concluded two important findings. First, there is a critical time during the embryonic development at which dietary behavior can stimulate and lead to epigenetic alterations; second, these epigenetic modifications are definitely transferred to progeny [20].

2. Nutrient gene–cancer interaction

2.1. Foods and cancer (diet-related carcinogenesis)

Diet is a mix of protective, carcinogenic, and mutagenic agents; most of them are metabolized by the enzymes of biotransformation process. Genetic polymorphisms that change protein expression or the function of these enzymes can modify the risk of developing cancer. More than 25,000 different bioactive food ingredients are proposed to be present in the foods ingested by humans [23].

More than 500 types of these bioactive food ingredients have been proved to be possible predisposing agents and have a role in cancer pathogenesis and others are not. It is thought that a diet containing protective micronutrients as well as carcinogens and mutagens may modulate the risk of cancer development, especially in genetically susceptible individuals [24].

Table 1

Dietary components, polymorphic genes and cancer Quoted from (Panagiotakos et al. [25]).

Dietary component	Polymorphic gene	Cancer site
Carcinogens		
Heterocyclic amines (red & processed meat)	NAT-2, NAT-1, CYP1A2	Colorectal, breast, other sites
Polycyclic hydrocarbons (red & processed meat)	CYP1A1, GSTM1	Gastrointestinal tract
Nitrosamines (fried potatoes)	CYP2E1	Nasopharyngeal, stomach
Alcohol	GSTM1, ADH (ALDH)	Colorectal
Aflatoxins (polluted grains)	CYP2E1	Liver
Anticarcinogens		
Cruciferous vegetables	CYP1A2, GST	Colorectal, other sites
Fruits & vegetables	CYP1A2, GST	Many sites
Calcium/vitamin D	Vitamin D receptor	Colorectal, prostate
Retinoids	Retinoic acid receptor	Variant acute promylocytic Leukemia, skin, others
Folate, methionine	MTHFR	Colorectal, cervix

ADH (ALDH) = alcohol dehydrogenase; CYP1A2 = cytochrome P450 1A2; CYP2E1 = cytochrome P450 2E1; GST = glutathione-S-transferase; MTHFR = methylene-tetrahydro-folate reductase; NAT-1, NAT-2 = N-acetyl transferase 1,2.

These bioactive food ingredients may emerge from different sources such as plants (phytonutrients) and animal sources (zoonutrients). These groups of dietary components may modulate the risk of cancer. They can be classified according to their interaction with specific genetic polymorphisms and how they affect each other into carcinogenic and anticarcinogenic (protective) foods [25]. Interactions between the different types of foods may affect the overall response [26]. Some examples of these two groups are presented in Table 1.

2.1.1. Molecular targets for bioactive food ingredients

Bioactive food ingredients and components function to maintain normal cellular activity, affect the neoplastic transition of normal cells to cancerous cells, and alter the biological behavior and attitude of the neoplasm. It is reported that these three states are important in modifying the risk and behavior of cancer but the pathophysiological mechanism is specific for each food type. Much evidence indicates the role of each foods to adjust Phase I and Phase II enzymes, which are xenobiotic metabolizing enzymes and consequently confirm maintenance of normal cells [27].

The modification of metabolism of carcinogens is one of the main possible mechanisms by which food components can minimize the risk of cancer. The gene expression of genes of the enzymes of Phase I xenobiotic metabolism, which usually activate most of the carcinogens, is established by xenobiotic sensing nuclear receptors such as AhR, PXR, and RXR. Then, the enzymes of Phase II catalyze the conjugations of these carcinogens and they are repeatedly regulated by different signaling pathways at the level of transcription [24].

The responses to dietary compounds that have a role in preventing cancer may be related to the diverse of the enzymes being processed and modified. For example, garlic is associated with autoactivation of CYP2E1, but does not affect other CYP450 isozymes by the same mechanism. Moreover, genetic polymorphisms in the regulatory regions of the genes of metabolizing enzymes and transporter proteins, such as AhR and PXR, may affect the total response to the bioactive food constituents [28].

Previous research [29–31] reported that many food components can modify the neoplastic progression as well as the programmed cell death (apoptosis). Key points in the cell cycle are regulated by different protein kinase complexes that are composed of cyclin and cyclin-dependent kinase molecules. Also, these cell cycle key points are affected by combined dietary components. It has been proved that the dietary factors either essential or nonessential can adjust and modify the cell cycle checkpoints, and consequently have a role in reducing the progression and proliferation tumor [29].

Apigenin (celery, parsley), curcumin (turmeric), epigallocatechin-3-gallate (green tea), resveratrol (red grape, peanuts, and berries), genistein (soybean), and allyl sulfur (garlic) have been reported to affect the cell cycle by different mechanisms. Some of these changes may be associated with the processing of synthesized proteins at the post-translational level. This modification includes shifts in the phosphorylation process of the main regulatory factors of cell division [30].

Another mechanism postulated by which other food ingredients can change the tumor behavior is by accelerating cell death and enhancing apoptosis. Apoptosis occur through two known pathways: the intrinsic, mitochondrial-mediated pathway; and the extrinsic, death receptor-mediated pathway. Dietary components can enhance or suppress the mechanism of apoptosis through its effect on mRNA transcription or expression and function of proteins. Some of these bioactive dietary components may also enhance apoptosis by stimulating free radical formation of reactive oxygen/nitrogen species and production in the cell [31].

2.1.2. Assessment of dietary intake

Precise and cheap methods for assessment of intake of particular essential and the nonessential bioactive nutrient components are essential to explore and resolve the relationship between different individuals' diet habits and possibility of cancer development. Food frequency questionnaires and 24-hour recall are considered to be the major data collection tools for assessing dietary

exposures, but they have some important limitations [24].

Absorption, metabolism, tissue distribution, and excretion of these bioactive food ingredients can affect the amount of these components that reach the target sites, so it is mandatory to find new methodologies that assess these variables and the effects at the cellular and molecular levels [12]. Integrating the assessments of diet intake with the analyses of concentrations of these bioactive food constituents or metabolites (metabolomic profile) in tissues and/or body fluids can provide special insights into the individual responsiveness to food and the possible prolonged duration of dietary exposures [32].

2.2. Factors affecting diet–gene–cancer interaction

2.2.1. Genetic variation among individuals

Interindividual variability of the genome structure and sequences makes understanding gene–nutrient interactions very complicated and difficult. SNPs and copy number variations are the most predominant structural variations in the human genome and consequently can participate in nutrient genetic heterogenecity [33]. For example, that variation in copy number of α-amylase and many cytochrome P450 genes has been reported. The increased copy number variation of α-amylase is related to a previous history of starchy food intake disorder [34].

2.2.2. Epigenetics affects the response to diet

The term *epigenetics* refers to the constant changes in DNA structure without mutating its sequence but can alter the expression of genetic information. The most commonly described and characterized epigenetic mechanism is chromatin remodeling through histone modification (and other chromatin proteins) and DNA methylation [35].

Epigenetic alterations could perform an important role during the development and pathogenesis of different disease including cancer. It has been proved in many studies that genes that are responsible for cell cycle regulation, DNA repair, angiogenesis, and apoptosis are suppressed and inactivated by epigenetic modification in the form of hypermethylation of their specific own CpG islands [23,25].

The main categories of these regulatory genes that are affected by DNA hypermethylation include the tumor suppressors cyclin dependent kinases, phosphatase, insulin-like growth factor-II, E-cadherin, and glutathione-S-transferase. DNA methylation patterns are proved to be influenced by the intake of multiple and/or combined food ingredients as vitamin A and zinc; even nonessential dietary components may have a role [36].

In 2007, Fang et al. [35] demonstrated that methyl deficient diets could lead to an evident alteration in the methylation patterns observed in the process of transformation of normal cells to cancerous cells. It is reported that genistein and related soy isoflavones through their possible direct regulatory effect on DNA methyltransferase enzyme can reactivate methylation-silenced genes. Genistein is a phytoestrogenic compound of the isoflavone class that is found in plants and has a structure similar to estrogen [35].

Table 2
Examples of genetic polymorphisms and related cancers Quoted from (Lampe and Chang [39]).

Polymorphic gene	Cancer
Cytochrome P450	Lung, breast, prostate, colon, liver, ovary
NAT2	Colon, bladder, breast
GSTs	Head and neck, lung, prostate
MTHFR	Colon
COMT	Breast
XRCC1	Colon, skin, lung, head and neck
XPD	Lung

COMT = catecholcatechol-O-methyltransferase; GSTs = glutathioneglutathione-S-transferases; MTHFR = methylenetetrahydrofolate reductase; NAT-2 = *N*-acetyl transferase-2; XPD: xeroderma pigmentosum gene group D; XRCC1: X-ray cross-complementing group 1 protein.

There are complex events of reversible modifications histone that could rule gene expression. These mechanisms include reversible histone acetylation, change of methylation pattern, phosphorylation/dephosphorylation, ubiquitination, and biotinylation. Modification of histone deacetylase has been documented as an important mechanism for adjustment of tumor behavior [1].

Many other food ingredients including butyrate, diallyl disulfide, and sulforaphane are considered to be weak ligands for the enzyme histone deacetylase and lead to change in its activity as observed in an *in vitro* study. Sulforaphane, an isothiocyanate compound that is found in some antioxidant rich vegetables, may lead to a marked increase in the global and local histone acetylation state of the promoter regions of the cellular senescence and proapoptotic genes, P21, and bax genes [37].

2.2.3. Timing and quantity of exposure to diet

Choosing the time and duration of nutrient exposure are important factors that determine the total response to foods or other nutrients supplements. In an experimental study, Virgili and Perozzi [38] concluded that the time of exposure of rats to dietary genistein is a very important factor in determining breast cancer risk.

2.3. Genes involved in cancer and interaction with diet

It is well known that humans show individual variability in the vulnerability to carcinogenesis. Genetic polymorphisms play an important role and contribute to explain the individual variation in cancer risk. Many studies have been carried out to compare the prevalence of different gene polymorphic forms in patients with cancer to the same gene polymorphism of normal unaffected control individuals [32].

Polymorphisms of gene variants that are involved in carcinogenesis are the most studied particularly those modifying the bioavailability, metabolism, affinity, and activity of several dietary constituents. Moreover, genes that have a role in influencing other mechanisms of carcinogenesis, such as those involved in DNA replication and repair as well as those involved in the production of sex hormones, have also been studied (Table 2) [39].

Genes involved in carcinogenesis are called candidate genes or susceptibility genes. The candidate gene is defined

Table 3
Examples of types of reaction and enzymes that participate in xenobiotic metabolism Quoted from (Brand et al. [42]).

Phase I reactions	
Oxidation	Ester hydrolysis
Xanthine oxidase	Carboxylesterase
Peroxidase	Amidases
Amine oxidase	Alcohol dehydrogenase
Monoamine oxidase	Aldehyde dehydrogenase
Dioxygenase	Superoxide dismutase
Reduction	
Cytochrome P450 dependent reductase	
Ketoreductase	
Glutathione peroxidase	
Hydration	
Epoxide hydrolase	
Phase II reactions	
Glucuronosyltransferase	Methylation
Sulfotransferase	O-Methyl transferase
Glutathione S transferase	N-Methyl transferase
Glucosyltransferase	S-Methyl transferase
Thiotransferase	Acetylation
Amide synthesis (transacylase)	N-acetyl transferase
	Acyltransferase
	Thiosulfate sulfutransferase

as the gene that is located in a chromosome region proposed of being involved in the expression in such disease. A candidate gene can also be identified by its association with its own specific phenotype and by linkage analysis to a specific region of the genome [40].

2.3.1. Carcinogen metabolism genes

The biotransformation and/or detoxification of a foreign compound or carcinogen is performed by Phase I and Phase II xenobiotic metabolizing enzymes. The aim of this process is to convert toxic, water insoluble, and not easily excretable compounds to compounds which are water soluble and more easily excreted. This is performed by conjugating the product of Phase I reaction to a carrier that facilitates their excretion in urine or bile. Phase I enzymes are responsible for the addition of one or more hydroxyl groups to the molecules, converting it into hydrophilic polar intermediates that can bind to a specific conjugating substrate that may enhance its reactivity to be complexed with DNA or protein. Sometimes this binding to DNA may produce carcinogens through enhancing the promutagenesis process [41].

The enzymes of Phase I reaction are cytochromes P450 that are coded by CYP gene family. Phase II or conjugating enzymes remove the polar intermediates from the body via their coupling to conjugating compounds such as glutathione, glutamine, and others. Several families of Phase II metabolizing enzymes had been recorded. They include glutathione-S-transferases, N-acetyltransferases, and UDP-glucoronosyl-transferases. Polymorphisms of the genes of Phase I and II enzymes may increase or decrease the possibility of cancer risk relying on the specific enzymatic activity that is being stimulated or inhibited as well as the substrate involved (Table 3) [41].

Cytochrome P450 is a family of enzymes that catalyze the oxidative transromation of several endogenous and exogenous substances as they are known to be monoxygenases or hydroxylases enzymes. They are expressed at different rates according to their tissue distribution. Therefore, these enzymes are important to explain the genetic susceptibility, carcinogen metabolism, and metabolism of chemopreventive agents [41].

2.3.2. Steroid hormone metabolism genes

Estrogens promote breast carcinogenesis through activation of genes involved in the modification of cell growth and proliferation. Estrogen biosynthesis and metabolism is a complex process and controlled by distinct and independent genetic variations. The genetic variation of the genes that are involved in the synthesis pathway of estrogens may alter their production. Genes in this pathway include CYP17 and CYP19. The bioavailability, half-life, and activity of estrogens are also influenced by other metabolizing enzymes such as catechol-O-methyltransferase enzyme (COMT) as it is involved in its biotransformation [42].

A 1931T-C polymorphism in the promoter region of CYP17 gene has been studied and characterized. CYP17 catalyzes the conversion of pregnolone to 17-hydroxypregnolone. The homozygous variant for this gene has a frequency rate of nearly 13% in the general population. Some studies have linked the increased risk of breast cancer with some CYP17 polymorphic forms [43].

COMT enzyme is a member of Phase II metabolizing enzymes that are involved in the inactivation of catechol estrogen molecules by conjugation with methyl group. Some polymorphisms of the COMT gene are associated with an amino acid substitution (Val158Met). This change is associated with diminished methylation process and consequently higher levels of estrogens. Many studies confirmed the higher incidence and prevalence of breast cancer in women who have COMT Val158Met polymorphism [44,45].

2.3.3. DNA repair genes

DNA repair corrects DNA damage induced endogenously or exogenously. DNA repair genes protect the genome from mutations. The capacity of DNA repair systems to prevent mutation can be influenced by polymorphisms of their genes. Polymorphisms of those genes are associated with altering their enzymatic activity and/or the ability of the protein product to bind their protein or receptor partner [45].

The best example of DNA repair mechanism is the X-ray cross-complementing Group 1 protein (XRCC1). This is involved in a mechanism called base excision mechanism. Three genetic polymorphisms in the coding region of the XRCC1 gene are specified at codons 194 (from Arg to Trp), 280 (from Arg to His), and 399 (from Arg to Gln). These genetic polymorphisms code for changes in amino acids that can change XRCC1 function [46].

Many studies have demonstrated that polymorphisms at codons 194 and 399 of XRCC1 are associated with high risk of developing of oropharyngeal squamous cell carcinoma, especially in individuals who are chronic users of tobacco and alcohol. Also, it shows an association with colorectal cancer among Egyptians, while, the variant 399Gln has been reported to be associated with a lower skin cancers risk [44].

The xeroderma pigmentosum group D (*XPD*) gene represents the genetic code of a DNA helicase enzyme that is concerned with nucleotide excision repair mechanism. Lys751Gln and Asp312Asn genetic polymorphisms in the XPD gene have been documented. These two genetic polymorphic forms have been reported to be associated with decreased nucleotide excision repair capacity. Asp312 XPD carriers were found to have nearly double the risk of development of lung cancer compared to noncarriers. By contrast, many studies have reported a relation between DNA damage (DNA adducts) and 751Gln XPD genotype even in apparently healthy individuals [47].

3. Conclusion

The etiology and pathogenesis of cancer is a complex interplay mechanism of genetic and environmental factors. Dietary intake and nutrient supplements are considered essential environmental factors, so scientists have reported that dietary and nutrients could play an important role in cancer development. Moreover, many studies have reported the intimate link of the quantity and quality of dietary nutrients with cancer incidence and pathogenesis.

There is a marked variation in cancer development with the same dietary intake between individuals. This could be explained by the variation in their genetic polymorphisms, which leads to the emerge of the concept of nutrigenomics and nutrigenetics. Nutrigenomics and nutrigenetics may explain the association of specific nutrient intake with genetic variations on cancer pathogenesis. Nowadays, nutrigenomics is widely used for discussing cancer development and pathogenesis or even the response to treatment as well as diet-related or dependent diseases. Subsequently, nutrigenomics may proceed in respect of the effective management of cancer in an individualized nutritional consultation according to the individuals' genetic profiles.

Conflicts of interest

All authors have no conflicts of interest to declare.

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