ISSN 2744-1636
Bioengineering Studies *Review Article*
Vol. 4, No. 1, June 2023, pp.16-27
https://doi.org/10.37868/bes.v4i1.id248

Precision nutrition impact on cancer development and prevention

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Received May 3, 2023
Revised May 25, 2023
Accepted Jun. 1, 2023

Abstract

The main nutritional influences on human DNA and its tenderness towards cancer development and prevention are reviewed in this paper. Nutrigenomics research has established dietary patterns that can alter gene expression, influence metabolic pathways, and change health outcomes. Considering the fact that numerous dietary elements can either encourage or slow down carcinogenesis, nutrition has a remarkable influence on cancer development and progress. Precision nutrition is an ascending subject that recognizes the distinctiveness of genomics and environmental conditions and looks for customized nutritional therapies. The complex interplay between the human genome and nutrition, just like consequences and its effects on cancer progression and prevention are presented in this paper. These discoveries have important consequences for the health of the public and cancer patients.

Keywords: Nutrigenomics, Nutrition epigenetics, Cancer, Precision nutrition, Human genome

1. Introduction

Nutrition is a vital life component; our bodies require intake of micro and macronutrients on a daily basis to satisfy the needs of complex processes within an organism. The intake of those molecules will affect the genome, since some of the molecules affect the activities of chromatin modifiers and transcription factors [1]. The key of nutrigenomics is the influence of nutrition and epigenomics on their complex correlation. The expression of genes and that complex correlation is evident in the metabolic organs of humans, which also affects the immune system and proper brain functioning. Needed nutrients to maintain the right function on a cellular level has a great impact on homeostasis in our bodies. Correct intake of nutrients on a daily basis, a diet, can play an important role in preventing diseases like cancer, diabetes, cardiovascular diseases and also managing the symptoms of already existing chronic diseases within the body [2-3]. Many factors, including nutrition and food intake, may influence how genes are expressed. These might potentially modify gene expression by serving like a ligand for transcriptional regulators. One of the ways that nutrients can affect gene expression is by changing the enzymes in different pathways that eventually end up in a different gene expression [4-6]. This goes even further; enzymes can interfere with signaling pathways and alter gene expression. One of the pathways responsible for changes within gene expression is transduction pathways, and nutrients might directly influence these exact pathways [7-10]. The way genes are expressed might affect the organism and the functions of certain organ systems. Therefore, nutrient- gene correlation can increase or decrease progression of disease in the body, by simply choosing what kind of nutrient we will have [11].

Genetic mutations are merely to blame for 5–10% of malignancies. Environmental variables, which are largely influenced by lifestyle, account for 90–95% of cancer cases [12]. Nearly 40% of all malignancies have dietary and nutritional causes. One or more of the characteristics of cancer are impacted by bioactives from diet or signaling pathways activated by the nutritional condition of the patients. In this review article, I will examine using bioactives in food and nutrition as part of an approach to avoid and cure cancer and highlight recent studies on the relationship between diet, nutrition, and bioactives in several cancer hallmarks [13].

Nutritional epigenetics is another subfield of the bio phenomenon known as epigenetics. Studying changes in gene expression without changing the DNA sequence is known as epigenetics. Contrary to popular belief, which holds that our genes dictate our fate, epigenetics provides some evidence that environmental variables can alter how genes are expressed. For instance, nutritional epigenetics seeks to explain how diet influences gene expression, and this research outlines several of those mechanisms [14]. Precision nutrition has been made possible by improvements in our understanding of the effects of diet on gene expression, specific diseases, particularly cancer, complicated biological processes, and the relationships between those two. Using data about a person's own genes or proteins, precession nutrition is a type of medicine that can be used to treat, diagnose, or prevent illness. Precision medicine is used to diagnose cancer, design a patient's course of treatment, assess the effectiveness of that treatment, and determine the patient's prognosis [15]. Some instances of precision medicine include the use of targeted treatments to treat particular cancer cell types, such as breast cancer cells that are HER2-positive, or the use of tumor marker tests to assist in the diagnosis of cancer. This article looks at the most recent advancements in our knowledge of how diet can affect how genes are expressed, providing fresh insight into how precision nutrition can help treat the debilitating condition [16].

2. The role of nutrigenomics

The topic of how nutrients affect DNA stability, repair, and numerous gene expression pathways has recently received more attention in nutritional studies [14]. Numerous food ingredients can alter genetic and epigenetic processes, which in turn has an impact on health [17]. The field of nutrigenomics studies how genes interact with nutrients or dietary bio actives and how this affects human health. It aims to determine how different foods, such as macronutrients and micronutrients, affect the genome. The study of how nutrients influence gene expression, transcriptional activity, and the various responses of gene variants is referred to as "nutrigenomics" as well [10, 18].

In order to comprehend how dietary components, affect metabolic pathways and homeostatic regulation, the word "nutrigenomics" also implies the study of biological systems utilizing functional genomic techniques. This area of science will identify the perfect diet form inside a series of nutritional alterations, in opposed to Nutrigenetics that will produce vitally important information that aids physicians in determining the ideal diet for a specific individual or tailored nutrition. [18]. Transcriptomics, proteomics, and metabolomics are among the technological tools employed in nutrigenomics research. By influencing signal transduction, chromatin structure, protein function, and gene regulation, diet has been proven in numerous studies to alter how genes are expressed [12].

Among the nutrition-related pathologies which are polygenic and multifactorial, which indicates that their onset and progression are influenced by an assortment of genes and their variants in addition to a plenty of environmental factors, especially diet, are obesity, metabolic syndromes, type 2 diabetes, CVD, and some kinds of cancer [13,19]. Transcription factors (TFs) are a crucial molecule that enables nutrients to alter the expression of genes. One of the most important families of nutrition sensors is the PPAR TF family, which has 48 members in the human genome. The majority of the receptors in this superfamily bind nutrients and their metabolites and also influence the expression of specific genes involved in a number of metabolic processes in the liver, such as fatty acid oxidation, ketogenesis, gluconeogenesis, amino acid metabolism, cellular proliferation, and acute-phase response [13, 20]. Furthermore, dietary components can alter signal transduction pathways right away. For instance, the polyphenol 11-epigallocatechin-3-gallate (EGCG) in green tea inhibits the tyrosine phosphorylation of the Her-2/neu receptor and the epidermal growth factor receptor, hence reducing signaling through the phosphatidyl inositol 3-kinase (PI-3)-AKT kinase-NF-kB pathway. Activation of the NF-kB

pathway has been associated to some types of breast cancer [14, 21, 22]. Nutritional deficiencies are more serious than radiation because they are caused by continuous exposure to environmental variables that encourage DNA damage. For instance, due to the substantial inclusion of uracil (4 million uracil/cell) in human DNA, a shortage of folate leads chromosomes to break [15, 18].

3. Dietary patterns and gene expression

Given that dietary components are regularly consumed and involved both directly and indirectly in the regulation of gene expression, it follows that a portion of the genes regulated by food must be involved in the start, progression, and severity of disease [16, 23]. The discovery of the galactose operon in bacteria was a crucial discovery for the exploration of the regulation of metabolism. This study revealed how bacteria may adjust the degree of expression of specific enzymes to alter their metabolism to meet their nutritional needs. It also established the first connection between differences in enzymatic activity and the transcriptional regulation of gene expression [17, 24]. Modifications to the mechanisms that control gene expression (from transcription to translation) may result in metabolic diseases. Therefore, studying how foods affect gene expression may improve our comprehension of metabolic diseases and maybe result in the creation of cutting-edge therapeutic approaches based on dietary changes and individual genetic profiles [25].

The number of times the DNA replicates determines the length of the telomere, which is composed of DNA tandem repetitions TTAGG that protect the ends of chromosomes together with bound proteins [26]. Telomeres give cells the ability to distinguish between chromosomal ends and double-strand breaks, prohibiting end-to-end fusion, recombination, and chromosome annihilation. Telomeres stop the breakdown of genomic DNA at the ends of linear chromosomes, protecting their structural integrity. According to published studies, telomerase, the enzyme that preserves telomeric ends, is catalytically controlled by histone acetylation and methylation, two epigenetic changes [27]. Diets rich in fruits, vegetables, healthy fats, and fiber result in the production of longer telomere segments [28]. Given the link between oxidative stress and telomere erosion, eating foods strong in antioxidants may have considerable health benefits. Bioactive compounds that help maintain TL length include carotenoids, fiber, omega-3 fatty acids, vitamins A, C, D, and E, polyphenols, and others. On the other hand, pro-inflammatory diets high in processed meat, sweet drinks, and saturated fats were linked to telomere shortening [29].

Dietary chemicals may have an impact on non-coding RNAs (microRNA and lnc-RNA), which control the epigenome. They interact with DNA and chromatin-modifying complexes to regulate gene expression at several levels, including transcription, and have been shown to affect the local epigenetic landscape [30]. Small non-coding RNA molecules called microRNAs (miRNAs), which have a length of 19 to 14 nucleotides, are essential for the post-transcriptional control of gene expression [31]. They work by inhibiting translation or degrading the transcript by inducing exonuclease action, decamping, or deadenylating the poly (A) tail. A single miRNA can regulate several mRNAs. MiRNAs control several biological processes in animals, including cell growth, hormone signaling, immunology, and metabolism. Cell death, division, and proliferation are some of these processes [32]. Many diseases, including cancer, can develop as a result of miRNA malfunction [33].

Methyltransferases can result in heritable alterations to DNA methylation. A methyltransferase known as DNMT3 is responsible for DNA methylation during embryogenesis. The mother's food and environmental factors may have an impact on the methylation level during embryogenesis [34, 35] Protein limitation is a typical illustration of maternal malnutrition. Pregnancy low-protein diets changed DNA methylation generally or at particular sites [36]. Studies on humans also show that offspring of mothers who have previously been malnourished are more likely to experience metabolic abnormalities connected to epigenetic changes that occurred during fetal life. A diet high in carbohydrates and low in protein causes both hypomethylation and hypermethylation at specific loci in children.The regulation of those pathways involves the transcriptional regulation of the genes that encode essential enzymes for the processes of metabolism involved in glucose homeostasis. The mechanism by which carbohydrates regulate the transcription of these genes was finally discovered through the purification and characterization of the carbohydrate-responsive element binding protein (ChREBP), alongside to the transcriptional regulation exercised by insulin and glucagon and their signaling

cascade. In response to glucose and fructose, this protein forms a heterodimer with its partner Mlx, which then binds and activates the transcription of target genes with ChoRE motifs (such as glucokinase, pyruvate kinase, ATP citrate lyase, acetyl CoA carboxylase, and fatty acid synthase) [37].

After lipids and carbohydrates, proteins are the 3rd most important category of macromolecules that should be included in the diet. Despite the fact that some amino acids can be produced by higher species, a subset of them, known as essential amino acids, can only be found in food. A diet that is both healthy and balanced must provide all of the essential amino acids that an organism needs, as well as a variety of sources and amounts of proteins. This is due to the fact that the body normally is unable to manufacture all of the other amino acids. The body's supply of amino acids comes from the conflicting processes of protein synthesis and protein breakdown. When the amount of amino acids declines, especially those that are essential, the so-called amino acid response occurs [38]. The kinase is stimulated by the eukaryotic initiation factor 2 alpha (eIF2) when the integrated stress response is activated, and as a result, mRNA synthesis is slowed or interrupted because of decreased eIF2B protein complex activity [39,40] . On the other hand, the activating transcription factor 4 (ATF4) activates a group of genes associated with starvation adaptation through binding to CCAAT enhancer-binding protein (C/EBP)-ATF response elements (CARE) [41]. Activation of the AAR controls a variety of functions, including the beginning of translation, chromatin structure, transcription start site, transcription rates, mRNA splicing, RNA export, and RNA turnover [42] .

Vitamins (vitamin D, vitamin A, vitamin E), polyphenols (resveratrol, quercetin, catechins, curcumin), fatty acids (omega-3 and omega-6), and minerals (selenium, zinc) are examples of bioactive dietary components that can affect the expression of miRNA, which in turn affects gene expression and the phenotype impact [43].

4. The impact of nutrition on cancer

Cancer, that is still the second-leading cause of death in the US, is thought to be decreased by maintaining a healthy diet, getting enough exercise, and maintaining a regular body regimen [44,45]. According to recent estimates, a balanced diet alone might avert 80,000 cases of cancer each year [46]. Population-based research have attempted to correlate food patterns with cancer incidence and mortality during the past few decades. Numerous studies have shown that leading a healthy lifestyle generally lowers the risk of getting cancer by 10% to 60%. This involves giving up smoking and maintaining an appropriate weight through physical activity and a balanced diet [45, 47]. The increased consumption of fruit, vegetables, and whole grains is what the scientists attribute to the significant risk decreases in colorectal, breast, stomach, liver, head and neck, and prostate cancers. The Mediterranean diet's observance was likewise inversely correlated with cancer mortality [48]. However, there is conflicting evidence linking vegetarian diets to cancer mortality and incidence. An extensive review of 96 studies on vegetarianism and veganism found that whereas cancer incidence significantly decreased, cancer death did not [49].

Diet is hypothesized to affect carcinogenesis through inflammatory mechanisms and metabolic pathways. In fact, deregulated cellular energetics is a recently recognized hallmark of cancer [50]. For increased cell development, rapid tumor division requires significant energetic and anabolic inputs. In contrast to normal physiologic cellular environments, where glucose is primarily taken up through glycolysis to the mitochondria for aerobic respiration to produce adenosine triphosphate, data show that tumors move into anerobic glycolysis even in the presence of oxygen instead of diverting glucose to lactate [51].

Metabolic syndrome and obesity are linked to a number of cancers, suggesting a potential dietary etiology. greater BMI scores, elevated glucose levels, and insulin resistance are linked to greater cancer incidence and mortality rates.7,8 Metabolic dysregulation brought on by obesity has a profile in common with cancer's modified cellular energy signature. Breast, colorectal, and lung cancer risk are all raised in humans with persistent IGF-1 elevation. The prolonged stimulation of the insulin/IGF-1 pathway, which activates PI3k, causes this rise in glycolytic flux [52, 53]. Calorie restriction (CR) is commonly used to describe a daily decrease in energy intake of 20% to 60% [51] . A recent investigation on primates randomly allocated 72 rhesus monkeys to conventional feed or a 30% calorie restriction. The results revealed a significant fall in the onset of age-related illnesses, notably a 50% lifelong decline in the incidence of cancer [54]. IGF-1 appears to play a role in

this pathway, at least in part, by inhibiting proto-oncogene signaling downstream of PI3k [55]. In fact, just 48 to 72 hours of brief fasting led to a 70% reduction in the level of IGF-1 in the blood and shielded mice from lethal chemotherapy doses [56]. Fasting cycles have been proven to be effective at reducing the toxicities of chemotherapy, delaying the onset of cancer in a number of tumor mouse models, and enhancing long-term survival [57, 58].

Restricting certain nutrients is the aim of the numerous dietary restrictions being explored for cancer. Protein and carbs are both restricted on the ketogenic diet (KD), which is distinguished by the high level of ketone bodies in the plasma. The initial iteration of the KD was created in the 1920s due to intractable pediatric epilepsy [59]. When fatty acids are consumed at extremely low carbohydrate levels, such as during fasting, the liver produces beta-hydroxybutyrate. The blood-brain barrier can be crossed by this ketone body, providing the brain with more energy. The KD may have a selective advantage against cancer cells since beta-hydroxybutyrate avoids the Warburg metabolism while giving adequate energy via the tricarboxylic acid cycle in typical tissues. The KD also leads to significant reductions in insulin and IGF-1 due to signaling molecules that inhibit histone deacetylase and gene expression [55, 60, 61]. Preclinical studies showed that the KD markedly retarded tumor growth, increased tumor cell susceptibility to chemotherapy and radiation therapy, decreased cachexia, and increased survival [62].

The relationship between nutritional status and oncologic outcomes and treatment-related toxicities has been demonstrated to be significant before, during, and after definitive therapy for different cancer locales. For instance, a lot of study has been done on how malnutrition can negatively affect morbidity, quality of life, and cancer-specific mortality in patients with head and neck malignancies. In fact, nutritional deficiencies in patients with head and neck cancer are frequently made worse by curative chemoradiation due to side effects like mucositis, dysphagia, xerostomia, nausea/vomiting, and other acute toxicities. Patients with head and neck cancer are frequently considered undernourished before starting any definitive treatment. [63, 64].

In addition to lung or head and neck tumors, nutritional deficiencies have also been linked to prognosis in cases of breast, gynecologic, gastrointestinal, and genitourinary malignancies [65].Keeping up a healthy diet might help cancer survivors manage many of the problems they usually run across. Not only is nutrition crucial for enduring effective treatment at certain disease locations, but it may also be crucial for the eventual cure. Patients are more likely to develop comorbid conditions like diabetes, cardiovascular disease, and osteoporosis as aging is often accelerated by cancer therapy. Physical weakness and weariness, two common long-term side effects of cancer treatment, make cancer survivors less active and lead to muscle mass loss, which aids in the development of sarcopenic obesity [66, 67]. Therefore, maintaining a healthy diet is crucial throughout cancer treatment, particularly among cancer survivors who received curative therapy [62].

Table 1. Four cancer related nutritional facts

1	Obesity and alcohol use, which increase the risk of many cancers, are the most important dietary variables contributing to the global burden of cancer.
2	Products can include mutagens that may trigger cancer, specific kinds of salted fish can induce nasopharyngeal cancer, and meals tainted with alpha toxin can cause liver cancer.
3	Fruits and vegetables aren't definitely linked to cancer risk, despite extremely low intakes having the potential to increase the risk for various cancers, especially aerodigestive and others.
4	The proof is not yet sufficient to be definite, but there may be additional nutritional factors that raise the risk of cancer.

5. The power of precision nutrition

The nomenclature of precision nutrition reflects the fact that it is a relatively recent field of study. The terms "precision nutrition" and "personalized nutrition" are not understood by all people [68]. One thing that can be stated about these methods, though, is that they aim to use information relevant to an individual or a group to offer nutritional recommendations that, in principle, would be better suited than general advice. Be aware that PN can occur at the group level yet still be considered personalized if the teams are formed based on fundamental standards that guarantee the same nutritional advice is provided to each participant of the same group. From an individual standpoint, this is a step up from PN and is known as stratification [69]. The degree of detail that PN

can achieve will ultimately depend on how much an individual difference within the identical stratified group affects the final prediction result, the extent to which this difference can be recognized by the technology utilized, and the cost-effectiveness compromise between these two factors. In reality, it seems that when these criteria are considered, stratification will probably prevail [70].

The concept of unique biological variation in reaction to nutrition forms the basis of PN [69]. The intended outcome variable can therefore be expected, and this can be utilized to create dietary advice, if the causes causing this variance and its effect on the intended outcome variable can be recognized. So, exactly what are these variables? The answer to this question depends on the anticipated outcome variable. No set of constant variables will ever produce a particular output. Instead, each event chooses which factors are thought to be important for predicting the result. This could, in certain cases, involve a considerable number of distinctive characteristics. They might be separated into groups, which we'll call PN elements. One PN component that is typical is genetics. It is thought that many observable variations, including eye color and hair color, have genetic roots. This idea also applies to how the body responds to nutrition [71]. Another vital factor in PN is nutrigenomics, which is an investigation of how genes relate to nutrition. Genetic variation influences how food is metabolized, but food also regulates gene expression and signaling [72]. The accuracy of PN models will undoubtedly be impacted by neglecting to account for this interaction because PN techniques routinely collect nutritional data. When novel gene-diet associations for various chronic conditions are discovered, PN techniques that consider nutrigenomics can be enhanced [73].

Metabolomics will have a role in PN when this discipline matures and these methods are applied more frequently, for example by generating phenotypes and examining how various people metabolize different diets [69]. However, clinically important biochemical values that are assessed with traditional methods (i.e., not taken into account from a metabolomics perspective) tend to occur more frequently and serve as a representative of the class of clinical biochemical parameters. These include typical clinical tests like blood sugar, hormone levels, blood counts, as well as additional signals thought relevant for a particular PN intervention. The microbiome is one of the additional PN traits because of its expanding importance for health and link to dietary consumption [74].

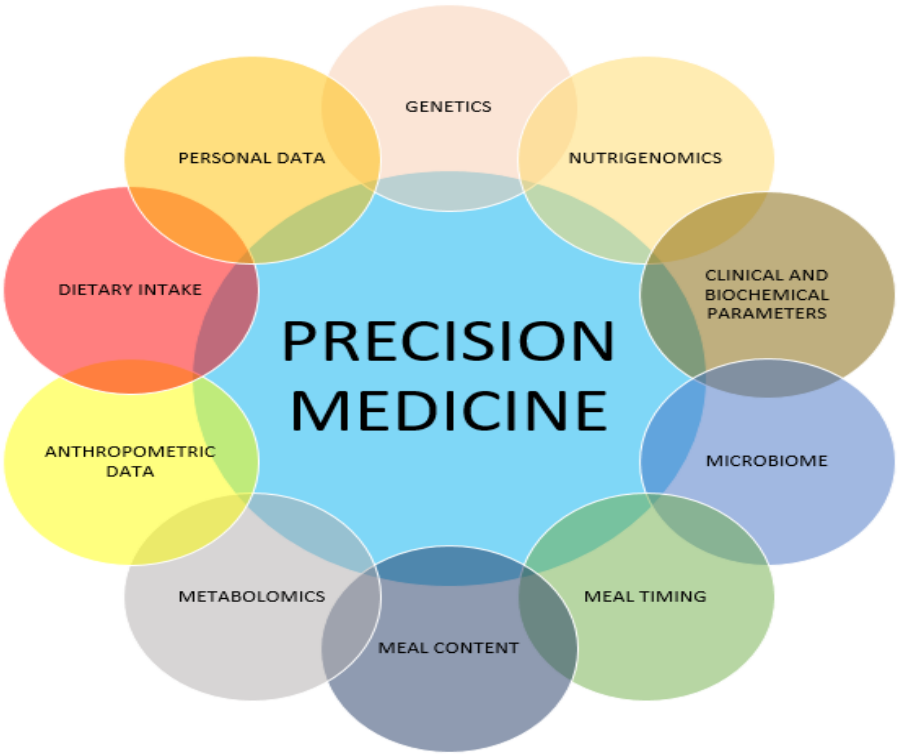

Figure 1. Precision nutrition factors

5.1. Precision nutrition: a tool for cancer fight

Numerous epidemiological investigations have been made into the connection between diet and cancer over the years, either as a preventive strategy or by demonstrating a link amongst the intake of specific food products and the emergence and growth of tumors. Contrarily, the field of study known as precision nutrition is still in its infancy and draws on well-established components like the microbiome and genetic and epigenetic variation. Precision medicine development is happening at the same time as this development [75]. Recent studies have shown that various bioactive foods can change the physiological properties of lines of human cells depending on their ability to influence the expression of specific genes. The use of nutritional therapy as a supplemental form of treatment for cancer is accepted on a global scale due to its advantages of lesser toxicity and more patient acceptance [76]. Epidemiological findings inspired more research into molecular pathways, and when phototherapy was applied in a setting of precision nutrition, this greatly boosted the efficacy of the treatment [77]. Recent research on the relationship between cancer prevention, treatment, and recurrence points to the value of examining the connection between particular food constituents and particular health outcomes [78]. Because extremely specific treatment targets must be addressed, precision nutrition must be based on special foods with well-established genetic mechanisms of action in terms of modifying gene expression and signaling pathways associated with proliferation, invasion, angiogenesis, metastasis, or death [29]. For example, it has been shown that nutritional strategies that block proliferative signals, dampen oncogenic metabolism, and lower inflammation can counteract the genetic instability associated with cancer [79]. Traditional foods like extracts of rosemary were proposed as potential ingredients of precise dietary supplements in cancer treatment, revealing molecular pathways linked to cancer progression and connections with already existing anticancer medications [80]. Cancer risk is strongly correlated with body fatness. The association between obesity and cancer is supported by some of the strongest evidence to date. Up to 33% of colon, breast, kidney, and digestive tract malignancies are associated with obesity. Overweight body fat alters the hormonal milieu of the organism because adipose (fat) tissue is hormonally active. Specific chemical cues secreted by body fat change how cells normally function. Certain hormones decrease when we maintain a slender body, which may explain why danger is decreased. The main factors in our food and way of life that contribute to the promotion of cancer are oxidation, inflammation, immune-suppression, blood sugar regulation, and stress. Diet can be responsible for 25–35% of cancer cases [81]. In this cancer-metabolic environment, there are more and more instances of pathways related to metabolism that could be engaged in precision nutrition. Modifications in lipid metabolism also have an impact on how cancer grows metabolically for use in precision nutrition, metabolic genes implicated in the synthesis of fatty acids are being studied in CRC [15]. At 2010 we saw the examination of 392 SNPs and 43 fatty acid metabolism-related genes in 1225 CRC cases and 2032 controls from the European Perspective Investigation into Cancer and Nutrition study (EPIC cohort) [82] . Recent genetic study of 57 SNPs located in 7 lipid-metabolism-related genes in CRC patients was done to ascertain when any genetic variation might be connected to the up-regulation of these enzymes and so serve as a biomarker of alterations linked to lipid metabolism. [83]. It is crucial to lower additional medical conditions that put life at hazards, like diabetes and cardiovascular disease, because cancer survival rates are rising. Unfortunately, compared to the general population, cancer patients significantly more frequently experience both of these illnesses [84]. Therefore, diet is a factor that can be changed, is disproportionately accessible, and is supported by growing data. This element might enhance cancer patient outcomes from the point of diagnosis via medication and into the post-survivorship phase [85].

6. Conclusion

The processes of DNA integrity, gene expression, and reparation are greatly impacted by nutrients. The goal of nutrigenomics is to understand how various diets alter the genome, while highlighting the interaction between genes and nutrients. Diet affects gene expression thanks to multiple mechanisms, like signal transduction, and may lead to pathologies and damage the DNA if it’s not executed properly. Dietary elements have a significant purpose in regulating gene expression and are directly engaged in the development or deterioration of diseases.

Nutritional components may determine the length of telomeres, DNA and RNA methylation, as well as crucial metabolic processes. Fusion of bioactive elements existing in fatty acids, minerals and vitamins can alter expression of miRNA. Including a healthy diet and continual physical activity leading to a more balanced lifestyle, is the ultimate prevention of numerous cancer cases, causing deaths of thousands each year. The correlation between cancer and nutrition lies in metabolic pathways and inflammatory responses, usually including calorie restriction and fasting in the daily diet as intervention gives positive results. Furthermore, the necessity of maintaining a high-quality diet during and post cancer treatment increases survivorship of cancer patients. Lately, an emerging field of therapeutic approach is precision nutrition. Precision nutrition is a type of personalized nutrition, tailored according to the needs of the patient, providing the individual with an opportunity to satisfy all the nutritional needs of their body. Comparatively to other fields, precision nutrition is relatively new and it will need broader understanding. For now, it embodies elements like genetics, metabolomics and nutrigenomics. These elements help to identify individual responses to various nutritional components and improve their reliability while making dietary models. Factors like obesity and metabolic diseases are firmly connected to cancer development, while diet plays a remarkable role in adjusting these factors. Applying dietary interventions as a component of the precision nutrition movement, can have a powerful impact on outcomes for cancer patients and their recovery path.

Declaration of competing interest

The authors declare that they have no known financial or non-financial competing interests in any material discussed in this paper.

Funding information

No funding was received from any financial organization to conduct this research.

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