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Review Article

Cancer and Food-Related Risks: A Review

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Abstract: This review presents the most current scientific evidence related with the interaction between diet and cancer risk. For the purpose of ensuring the food's microbiological and chemical safety, as well as improving its palatability, numerous food processing techniques have been utilized. Innovations and advancements in food processing are being driven by the growing demand for food that are not only nutritious but also convenient and healthy. When new processes and compounds are discovered as a result of improvements in analytical capabilities, it is necessary to conduct a thorough analysis of the potential effects on human health. In this review, the most recent scientific findings concerning the influence of the foods we eat and the risk of developing cancer are presented. Therefore, there is a pressing need for ongoing research as well as the development of comprehensive strategies to address these preventable causes of cancer and to encourage healthier lifestyle choices. This review presents an analysis of the most recent scientific data concerning the correlation between dietary habits and the likelihood of developing cancer. In addition to an increase in the consumption of refined sugars and foods that are high in carbohydrates, these patterns also demonstrates a decrease in the consumption of macronutrients and micronutrients. In addition to this, this review investigates the impact of certain dietary regimens, such as the Mediterranean diets, as well as the intake of meat and dairy items, on the likelihood of developing cancer. Epidemiological studies, molecular mechanisms, and emerging perspectives in the field of personalized nutrition were also been reviewed. It is the objective of this review to present a comprehensive analysis of relevant studies, offering the latest scientific insights regarding the correlation between diet and the likelihood of cancer development

Key words: Cancer, Cancer Risk Factors, Comprehensive Review, Dietary Patterns, Food-Related Risks

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1. INTRODUCTION

A significant contribution is made by diet, which accounts for thirty to thirty-five percent of the risk factors associated with the onset of cancer [1]. Although the available epidemiological data are not consistently precise and clear for many different types of foods, there have been associations between a variety of foods and dietary patterns and the increased risk of various cancers [2]. Tobacco is a widely consumed addictive food (chewable or as smoke) and its use is undeniably the primary contributor to the onset of lung and mouth cancer, being accountable for the overwhelming majority of diagnosed cases. The incidence of lung cancer continues to be high, despite the extensive anti-smoking campaigns; therefore, it is necessary to investigate other potential risk factors influencing cancer. In Western countries, dietary factors are a significant contributor to the incidence of cancer [3,4]. As a consequence, dietary factors stand as the second most significant preventable contributor to cancer, following closely behind tobacco. A significant contribution of vitamins that prevent cancer, need to be established with a focus on the necessity of conducting controlled human intervention trials to validate the efficacy of vitamin supplementation [5,6]. All factors influencing the concentration of acetaldehyde in the saliva or intracolonic fluid are deemed crucial in the context of cancer probabilities. The role of acetaldehyde becomes more complicated when it interacts with other carcinogens in the digestive tract. This interaction results in synergistic effects, which may increase the overall potential for carcinogenicity [7]. Acetaldehyde has been linked to the promotion of inflammation, which is a significant contributor to the development of cancer. An inflammatory response in the gastrointestinal tract can be induced by prolonged exposure to acetaldehyde, which can increase the likelihood of developing cancer [8]. When it comes to designing interventions that target this particular pathway, having an understanding of how acetaldehyde contributes to inflammation provides valuable insights. Tobacco use and dietary factors are both significant contributors to the likelihood of developing cancer. The leading factor contributing to lung cancer is tobacco usage; nonetheless, dietary habits also contribute significantly to the onset of different types of cancer. Therefore, there is a pressing need for ongoing research as well as the development of comprehensive strategies to address these preventable causes of cancer and to encourage healthier lifestyle choices. It is the objective of this review to present a comprehensive analysis of epidemiological studies, offering the latest scientific insights regarding the correlation between diet and the likelihood of cancer development.

1.1 Red Meat

The occurrence of heme iron in red meat is one of the potential reason for the association between consuming red meat and an elevated likelihood of cancer development [9]. The protein responsible for transporting oxygen within red blood cells, hemoglobin, includes heme iron as one of its components [10]. When people consume heme iron, it has the potential to interact with other substances in the gastrointestinal tract, which can result in the formation of N-nitroso compounds, which are known to be carcinogenic [11]. Moreover, heme iron has the capacity to foster the proliferation of detrimental bacteria in the digestive tract, a factor associated with an elevated susceptibility to cancer [12]. While the evidence remains inconclusive, several reputable entities, such as the World Health Organization and the American Cancer Society, recommend limiting the consumption of red and processed meats to reduce the risk of cancer development [13]. The correlation between the intake of red meat and the onset of breast cancer is closely tied to the presence of heme iron. This element is known to trigger oxidative reactions, leading to the formation of tumors. Consuming red meat has been demonstrated to elevate the occurrence of Helicobacter pylori infections, subsequently resulting in heightened expression of the CagA gene and the secretion of pro-inflammatory cytokines [14]. As a result, red meat consumption is a significant contributor to the development of gastric cancer. The consumption of red meat, containing aromatic hydrocarbons, heterocyclic amines, and heme iron, has been shown to be associated with the beginning of the development of tumors, according to a substantial body of evidence. In many parts of the world, meat is considered to be a primary food group. It is a rich source of protein and fats, in addition to essential vitamins and nutrients such as zinc (Zn), vitamin A, and vitamin B. Meat holds a prominent position as a primary food group. In the United States, a discernible rise in the consumption of lamb and goat has been observed, alongside with an increased demand for red meats such as beef, as well as processed meats like hot dogs and sausages. These types of meats make up a significant portion of the overall meat consumption. The intake of processed meat has been linked to an elevated likelihood of developing rectal cancer, colon cancer, breast cancer, and colorectal cancer. Moreover, it is also regarded as an undisclosed risk factor for stomach cancer. This is particularly applicable to women who have undergone menopause [15].

1.2 Processed Meat

The majority of processed meat is sourced from preserved pork or beef, utilizing methods beyond freezing. Processed meat undergoes treatment to enhance preservation, to improve the quality of carcass cuts, and to modify flavor. There exists a diverse range of processed meat products, making classification into distinct categories. Examples of processed meat include ham, heated sausages, bacon, raw sausages, bologna, liver paté, hot dogs, blood sausage, other patés, luncheon meat, spread meat, canned meat, cold cuts, and corned

beef [16]. In Europe, processed meat intake was reported at 27 grams per day among females and 48 grams per day among males [17]. Among individuals of European heritage, the consumption of recently obtained red meat equaled 36 grams daily for females and 60 grams daily for males [18]. In a case-control study conducted in Bethesda, involving individuals with a median age of 58 years, participants consumed an average of 12 grams of processed meat and 36 grams of red meat daily [19]. These figures may be conservative as they are derived from data obtained through food-frequency questionnaires, and the participants were older than the general population. Meat is a staple food in the human diet, and while it is a source of high-quality nutrients, is also a significant contributor to cholesterol and saturated fatty acids globally. Meat is one of the staple diet. The majority of epidemiological investigations suggest that a substantial intake of meat, especially processed meat, is associated with an elevated likelihood of developing colon cancer. Some substances inherent to meat are generated during its processing and cooking may contribute to the correlation between high meat consumption and an elevated likelihood of developing colon cancer. It can be inferred from the existing body of research that there is a substantial epidemiological evidence associating the intake of processed meat with an increased susceptibility to colorectal cancer. Several carcinogenic chemicals, such as N-nitroso compounds, polycyclic aromatic hydrocarbons and heterocyclic aromatic amines, are found in processed meat [20]. The quantity of these chemicals found in meat products is influenced by the processing and preparation of the meat, and there are suspicions that these compounds may contribute to the onset of colorectal cancer. In addition to the link attributed to mutagens, the correlation between consuming red or processed red meat and the susceptibility to colorectal cancer may be influenced by recent findings indicating that, the intestinal microbiota, including organisms like bacteroides, could contribute to the development of colorectal cancer. On the other hand, there is a need for additional research for the interactions and possible mechanisms elucidation that exist between dietary factors and gut microbiota in relation to the risk of colorectal cancer [21].

1.3 Food Processing

The deliberate incorporation of compounds as additives or the inadvertent inclusion of contaminants during food processing might lead to the alterations of constituents in food products, potentially carrying implications for the risk of developing cancer. While scant evidence supports the notion that trans fatty acids increases cancer risk, research has established their adverse effects on blood lipid profiles and the susceptibility to heart disease. The hydrogenation process of vegetable oils leads to the formation of trans fats, a type of fatty acid that is predominantly absent in natural sources. During the processing of meat, compounds that have the potential to increase the carcinogenic potential of these foods can be accidently added that includes preservatives such as salt or sodium nitrite to deter bacterial contamination, or smoking for preservation purposes and to amplify color and flavor. The elevated intake of processed meats has been associated with an elevated risk of colorectal cancer, as indicated by epidemiologic studies [22]. This correlation might arise from the presence of nitrites, which are commonly included in a range of processed meats, such as hams, luncheon meats, and various types of hot dogs and processed meats. Some food processing techniques, like freezing and canning vegetables and fruit, can indeed offer benefits by aiding in the conservation of vitamins and other bioactive food components [23]. This preservation may potentially contribute to lowering the risk of cancer development. In the course of human history, various techniques of food processing have been employed, mainly with the aim of substantiating the microbiological and chemical safety of foods and improving their palatability. Enhancements and emerging advancements in food processing are propelled significantly by increasing consumer demand for food that is not only nutritious but also convenient and healthy. Emerging procedures or recently identified compounds, often revealed due to enhanced analytical capabilities, require a thorough assessment of their potential effects on human health. These risk assessments are examples of two traditional approaches to evaluating the safety of food. In the existence of lipid and chloride, 3-MCPD is generated in a diverse array of foods manufactured through both industrial and household processes [24]. As a consequence of the fact that 3-MCPD is identified as a non-genotoxic carcinogen, it is presupposed that there exists a threshold of impact, and ample data to determine an appropriate level of consumption. Given that acrylamide, aside from its neurotoxic and reproductive toxicity, is also acknowledged as a genotoxic carcinogen either in domestic or industrial settings, has raised significant concerns [25]. Substances recognized as genotoxic carcinogens are undesirable in food and are typically addressed using the ALARA (As low as reasonably achievable) principle [26]. Conversely, for unavoidable substances, a quantitative risk assessment might offer risk managers more insightful guidance on appropriate actions. Currently, there is a dearth of adequate dose–response relationships and mechanistic information regarding acrylamide's carcinogenicity.

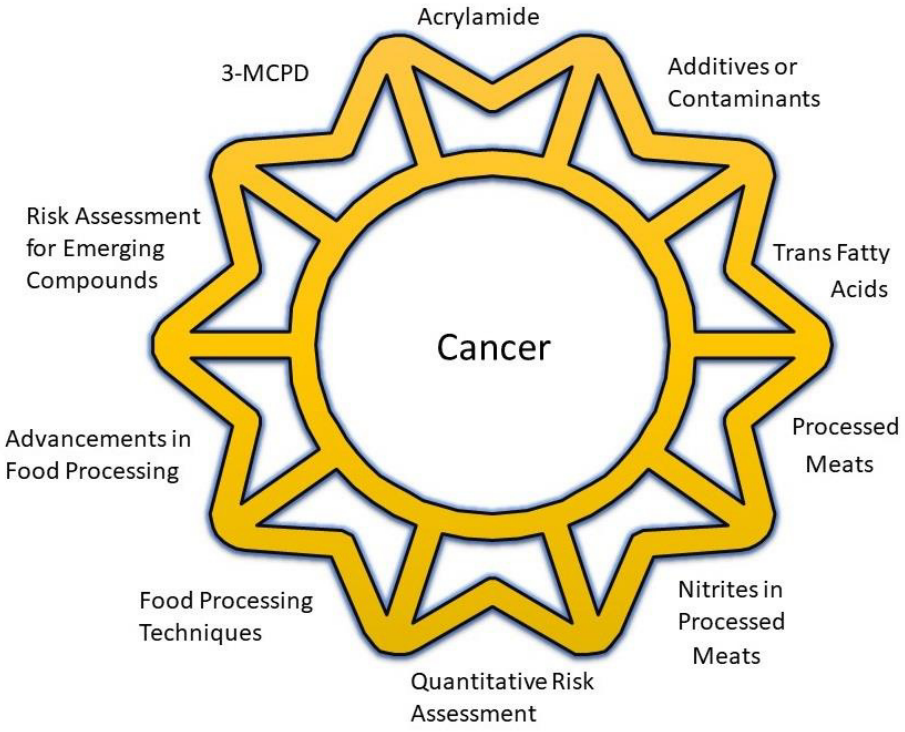

Figure 1 : Factors of food processing that influences cancer

1.4 ultra-processed foods, (UPF)

Excess body weight, were recognized as a substantial risk element for several cancers, such as postmenopausal breast, pancreas, stomach, colorectal, liver, esophagus, endometrium, gallbladder, ovary, kidney, and (advanced) prostate cancers, as well as hematological malignancies. Body fatness is responsible for 16% of the post-menopausal burden of breast cancer. In studies examining the percentage of total energy intake from Ultra-Processed Foods (UPF) in adults, a Malaysian study reported the lowest proportion (22%) [27]. Nevertheless, a recent comprehensive analysis unveiled a slight association between dietary acrylamide and the incidence of kidney and endometrial cancer in nonsmoking individuals. Countries such as Spain (24.5%), Lebanon (27.2%), Brazil (20–29.7%), France (29.8–35.7%), Canada (45.2–51.8%), and the United Kingdom (52–54.2%) reported higher levels of UPF consumption [28]. The peak concentrations were documented in the United States of America (55.5–56.1%) [29]. Adult populations daily servings or the frequency of Ultra-Processed Food (UPF) consumption in the United States, reported to be an average intake of four times per day. Meanwhile, in Spain, another study reported a range of 1–5 servings per day, covering the spectrum from the least to the most significant quartile of UPF intake [30]. The proportion of total energy intake from UPF varied, with reported values between 41.8 and 43.4% in children and adolescents (solely in Brazil) aged 3 to 4 years, 47.7 and 49.2% in those aged 6 to 8 years, and 50.6% in adolescents [31]. Specific nutrients, foods, and exposures to dietary patterns are all examples of dietary risk factors that play a role in the development of UPF. The presence of high added sugar, trans fat, saturated fat and levels of sodium are all examples of nutrient exposures [32]. Cancer influencing dietary factors encompass consuming a low quantity of whole grains, sugar-sweetened beverages, fruit, nuts and seeds, as well as fish and vegetables, and consuming a high quantity of red meat, potato chips, and processed meat [33]. UPFs are characterized by the presence of ingredients that are not commonly used in the culinary industry. These include protein, sugar, and petroleum by-products (such as high-fructose corn syrup, protein isolates, maltodextrin, and hydrogenated oil), as well as cosmetic enhancements designed to make the final product more appealing [34]. There were two prospective cohort studies from Spain, conducted over a median duration of 8 years, individuals who consumed the highest quartile of UPF faced a 26% increased likelihood of developing overweight compared to those with lower UPF consumption []. When accounting for participants who were not followed up and those with repeated exposure measurements at the 10-year follow-up, the risk persisted at 24% and 19%, respectively [35]. Individuals who were not overweight at the beginning of the study demonstrated a twenty percent increase of developing excess weight compared to those in the lowest quartile. While there was initially an elevated risk of developing obesity for individuals initially classified as overweight (BMI 25–30) in the crude analysis, this significance did not persist in the fully adjusted baseline

model BMI [36,37]. Although no associations with UPF and explored outcomes were identified, studies on adults and adolescents suggested that a higher intake of MPF-PCI was linked to a reduced likelihood of being overweight or obese [38]. Possible explanations include an insufficient nutritional composition, as Ultra-Processed Foods (UPFs) act as carriers for additional sodium, sugars, and trans-fats, displacing Minimally Processed Foods (MPFs) in the diet. Potential factors include the formation of carcinogens during high-temperature cooking, such as acrylamide in carbohydrate-rich foods, as well as inflammatory responses associated with acellular nutrients and industrial food additives. Other considerations involve disruptions in gut microflora balance and increased intestinal permeability. Certain characteristics of UPFs, such as their palatability and quasi-addictiveness, widespread availability, convenience, and intensive marketing practices employed to promote purchasing and consumption, particularly among children and adolescents, may contribute to excessive consumption [39].

1.5 Deep-fried foods

The consumption of deep-fried foods has not been specifically studied in relation to cancer; however, there have been reports of heightened risks of certain types of cancers being correlated with the intake of deep-fried foods. Consumption of food that has undergone deep-frying has also been associated with an elevated risk of developing pancreatic cancers [40,41]. The intake of fried foods in general has been associated with the onset of oral and pharyngeal cancers, as well as esophageal malignancies and laryngeal conditions. There is a high concentration of advanced glycation end products (AGEs) in foods that are cooked at high temperatures [42]. AGEs that are derived from food have been associated with higher oxidative stress and inflammatory responses [43]. Additionally, it has been demonstrated that a diet low in AGEs (obtained by avoiding cooking methods that involve high heat) can reduce indicators of oxidative stress and inflammation [44]. The foods that are deep-fried has the highest amount of AGE. As an example, a chicken breast that has been immersed in hot oil for twenty minutes contains over nine times the quantity of AGEs compared to a chicken breast that has been simmered for one hour. Research with prostate cancer cell lines indicates that the interplay between AGEs and the receptor for AGE (RAGE) might contribute to the progression of prostate cancer [45]. During the process of deep-frying common foods, especially carbohydrate-rich items such as potatoes, the production of acrylamide, a known carcinogen, occurs in substantial quantities [46]. To explore the connection between acrylamide and prostate cancer risk, researchers have conducted two case–control studies and four prospective cohort studies. The carcinogenicity of deep-fried foods is not solely attributed to acrylamide but additional potential mechanisms include heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons (PAHs), formed when cooking muscle meats like fish and chicken at high temperatures [47]. Surprisingly, the elevated risk of prostate cancer linked to the consumption of french fries and doughnuts was comparable to that associated with fish and chicken, indicating the involvement of agents beyond HCAs and PAHs in the development of prostate cancer. The process of deep-frying induces changes in the chemical structure of oils through oxidation and hydrogenation, resulting in a decrease in unsaturated fats and an increase in trans fatty acids [48]. Mutagenic compounds, such as aldehydes, formed during deep-frying remain in the oil, get incorporated into fried food, and undergo metabolism in the gut [49]. Reusing oil and prolonging frying time further amplifies the production of toxic compounds. Deep-fried foods, like fried chicken, french fries, and fried fish, are popular choices in restaurants, especially fast-food establishments, contributing to a potential higher consumption of fast food overall. Apart from exposure to high temperatures, fast food often contains elevated levels of refined carbohydrates, sugars, salt, and additives like sodium phosphate and sodium nitrite/nitrate. Over the past few decades, there has been a marked surge in fast food consumption, leading to a growing proportion of total energy intake [50].

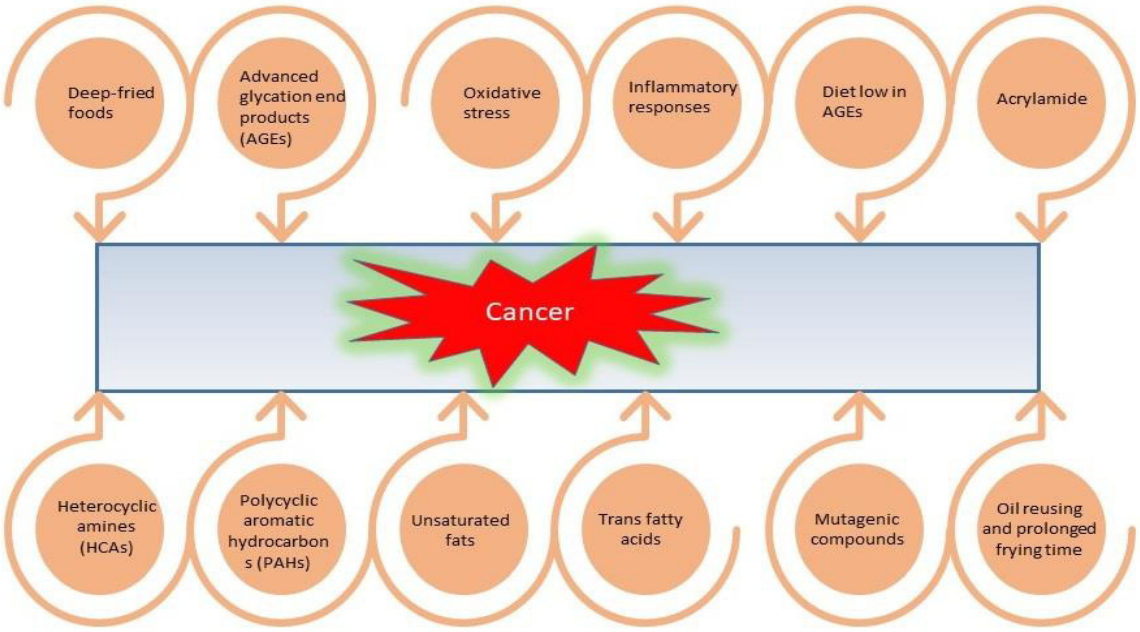

Figure 2 : Important factors of fast foods that influence cancer

1.6 Sugar as well as refined carbohydrates

Consuming foods abundant in sugar and refined carbohydrates not only increases the likelihood of developing conditions such as type 2 diabetes and obesity but also indirectly influences the risk of cancer [51]. Inflammation and oxidative stress are key components in the intricate connection between a diet high in sugar and and the risk of cancer development [52]. The intake of an elevated amount of sugar and refined carbohydrates can lead to persistent inflammation in the body [53]. Moreover, this inflammation is linked to an elevated probability of cancer development. Studies conducted in 2020 indicated that obesity and type 2 diabetes, both linked to a diet rich in sugary and starchy foods, contributing to chronic inflammation [54]. Prolonged inflammation sets the stage for an environment within the body conducive to the growth and progression of cancer cells [55]. Insulin resistance, a condition where cells inadequately respond to insulin, leading to elevated blood sugar levels, is associated with a high sugar intake. Insulin resistance not only poses a risk for diabetes but also appears to play a role in cancer development [56]. The condition is linked to an excess of insulin, which may stimulate the growth of certain types of tumors. Sugary foods and refined carbohydrates typically boast high calorie content but offer low nutritional value [57]. Regular consumption of these foods can contribute to weight gain and obesity, recognized risk factors for various types of cancer [58]. Adipose tissue, or fat cells, can produce hormones and substances that contribute to inflammation, potentially playing a role in cancer development [59]. Imbalances in hormone levels, influenced by diets high in sugary foods, may affect the development of hormone-sensitive cancers [60]. In summary, inflammation, oxidative stress, insulin resistance, weight gain, and hormonal imbalances collectively contribute to the relationship between a diet rich in sugary foods and refined carbohydrates and the risk of developing cancer [61]. Adopting a nutrient-dense and balanced diet, prioritizing whole foods, can help mitigate these risks and promote overall health [62].

1.7 Fast Food

A category of cuisine that is mass-produced and crafted for commercial resale as fast food places a considerable emphasis in influencing cancer. It is a term used in the business world to refer to food that is sold in a restaurant or store and are packaged for takeout or takeaway. The food may contain ingredients that have been frozen, preheated, or precooked. The potential link between the intake of fast food and the onset of cancer, particularly colorectal cancer (CRC), has been explored, indicating that fast food might play a role in the initiation of cancer [63]. The growing prevalence of obesity and the adoption of Western dietary patterns, which are characterized by the consumption of high-calorie dense foods like fast food, may be associated with an elevated likelihood of colorectal cancer [64]. There is a correlation between particular fast food items and an increased risk of colorectal cancer such as regularly indulging in potato and corn chips, along with frequent consumption of fried potatoes, has been demonstrated to correlate with an elevated risk of colorectal cancer (CRC) [65]. In addition, the study indicated a significant association between the

intake of falafel and a higher susceptibility to colorectal cancer (CRC), emphasizing the potential impact that specific types of fast food might have on cancer development. Jordanian population study on fast food consumption offers valuable insights into the dietary patterns of the region and the potential impact of those patterns over colorectal cancer [66]. The findings highlighted the necessity of addressing the implications of fast food consumption on cancer risk within populations that are either newly developed or in the process of economic transition. The urgent need for comprehensive public health initiatives aims to promote healthier dietary choices and raise awareness about the potential health risks associated with frequent consumption of fast food from fast food restaurants [67]. There is a complex relationship consuming fast food and the likelihood of developing colorectal cancer sheds light. Exploring the nutritional elements, including the intake of fast food, influence on the development of colorectal cancer is a crucial step toward formulating preventive measures and potential interventions [68]. Colorectal cancer is a serious health concern and public health initiatives should not only focus on dietary choices but should also address broader lifestyle factors that contribute to the risk of colorectal cancer [69]. A few examples of this would be encouraging regular physical activity, discouraging smoking and excessive alcohol consumption, and advocating for routine screenings which are extremely important in the prevention of cancer [70].

1.8 Tobacco

A distinct correlation has been identified between tobacco smoking and an increased risk of cancer across different organs [71]. These organs encompass the lungs, excretory system, upper respiratory tract (comprising the oral cavity, pharynx, larynx, and esophagus), pancreatic gland, nose cavity, paranasal sinuses, nasopharynx, stomach, liver, kidney, uterine cervix, and myeloid leukemia. The relative risks linked to tobacco smoking and cancer development vary significantly among different cancer sites, ranging from 1.5 to 30.0 [72]. These findings stress the substantial impact of tobacco smoking on cancer risk, emphasizing the crucial role of public health interventions in curbing tobacco use and addressing associated health risks. Various types of tobacco consumption, including cigars, pipes, bidis, along with cigarette smoking, contributed to an elevated cancer risk, particularly affecting the lungs and specific regions of the upper aero digestive tract [73]. Moreover, exposure to environmental tobacco smoke, or involuntary smoking, is recognized as a substantial contributor to the development of lung cancer, supported by a meta-analysis of over fifty studies on non-smokers. The complex composition of tobacco smoke, containing numerous carcinogens, poses a threat by causing DNA damage in lung cells [74]. Cumulative exposure to tobacco over time, rather than the act of smoking alone, significantly influences the risk of lung cancer, emphasizing the higher risk for individuals engaged in substantial or long-term smoking practices. Current indications suggest a potential linkage between higher alcohol intake and an increased probability of developing cancer in various regions of the gastrointestinal (GI) tract [75]. Although ethanol itself is not inherently carcinogenic, the exact mechanism responsible for alcohol-related cancers remains elusive [76]. Acetaldehyde, the initial metabolite formed during ethanol oxidation, has been associated with carcinogenic effects based on research involving animal and cell cultures [77]. Recent studies propose that acetaldehyde, generated by microorganisms in the gastrointestinal tract or originating from salivary glands during alcohol consumption, may act as a localized carcinogen in humans [78]. Moreover, excessive alcohol use may contribute to indirect effects that promote tumor growth, including nutritional deficiencies, higher exposure to other carcinogens, metabolic activation of procarcinogens, and local effects of potent alcoholic beverages [79]. Alcohol abuse appears to exacerbate these effects. Importantly, the risk of cancer escalates with increased alcohol consumption, particularly when combined with smoking, amplifying synergistic effects [80]. Alcohol consumption is recognized as a contributing factor to various cancers, such as breast, colorectal, esophageal, and liver cancers. Even moderate alcohol intake is linked to an increased cancer risk, that are directly proportional to the quantity consumed [81]. Despite the fact that the exact mechanism behind the increased risk of developing cancer due to alcohol consumption is not entirely comprehended, it is believed to be linked to the body's metabolism of alcohol. The breakdown of alcohol produces acetaldehyde, a toxic substance capable of damaging DNA and cellular components, ultimately leading to cancer [82]. In addition to its direct effects, alcohol consumption can increase the probability of cancer by contributing to the development of other risk factors, such as obesity or smoking [83]. For instance, alcohol consumption may contribute to weight gain, a recognized factor associated with various cancers like breast and colorectal cancer. The oral cavity, where saliva-borne enzymes initiate ethanol conversion into acetaldehyde, is a crucial site in the metabolic transformation of alcohol [84]. The higher concentration of acetaldehyde in saliva, associated with increased alcohol consumption, exposes the oral mucosa and upper digestive tract to the carcinogenic effects of this compound. Acetaldehyde continues its journey through the digestive tract until it reaches the colon, with its levels influenced by factors such as alcohol quantity and frequency of consumption [85]. Persistent exposure to heightened intracolonic acetaldehyde levels is associated with an increased susceptibility to cancer development in the digestive tract [86].

2. CONCLUSION

It is widely recognized that a link exists between the intake of unhealthy foods and an elevated likelihood of developing tumors and cancer. As a result, a key preventive factor against tumors is the maintenance of a favorable nutritional status through the consumption of a diet that is well-balanced. In spite of this, the findings of epidemiologic studies are inconsistent and inconclusive, which makes it difficult to obtain evidence that is both clear and consistent regarding the connection between food and the risk of developing cancer. In the upcoming years, additional research in the field of public health will be necessary to offer further clarification on this matter in order to address the uncertainties that have been raised.

3. AUTHOR CONTRIBUTION STATEMENT

Dr. Nisar Ahmed conceived the study and was responsible for the overall direction, analysis, and planning. Dr. Biswajit Dash Scarried out the implementation. Dr. Somenath Ghosh took the lead in writing the manuscript.

4. CONFLICT OF INTEREST

Conflict of interest declared none.

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