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Roles of foods and related components: an overview on cancer causatives, and plausible preventions

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ABSTRACT

Cancer, a complex and frequently fatal disease category, is recognized as the leading cause of death worldwide. Growing evidence suggests that nutrition plays an important bidirectional role in cancer prognosis. With food components acting as both carcinogenic initiators and preventative agents, the relationship between diet and carcinogenesis involves complex and multifaceted interactions in the biochemical makeup of living things. Diets high in processed red meat, refined sugars, and harmful fats, particularly those from Western countries, have been linked to an increase in cancer. Diets high in whole grains, fruits, vegetables, and healthy fats, on the other hand, have been shown to be cancer-protective. The interaction of nutritional components in foods has an impact on fundamental physiological and biochemical processes such as oxidative stress, immune response, and inflammation. These molecular physio-biochemical interactions, as part of the critical biomechanistic dynamics, shift or create complex pathways that either promote or inhibit cancer development. The current review examines the contrasting roles of foods in cancer biology, specifically the mechanisms by which food-related carcinogens and anticarcinogens function. It also examines the impact of specific dietary components on cancer risk and suggests practical dietary patterns for cancer prevention and management. Understanding the dynamic relationships between foods and diseases allows for more informed decision-making and implementation at both the individual and population levels, potentially lowering cancer risk, management costs, and the burden on healthcare systems. As a result, evidence-based nutritional interventions and strategies can help to prevent and treat cancer.

1. Food items and cancer: an introductory outlook

The link between diet and cancer has been the focus of much debate, discussion, and scientific research. According to mounting evidence, certain foods, eating habits, and dietary patterns have the potential to play a critical role in both cancer initiation and prevention. Although no single meal pattern has been shown to guarantee cancer prevention, consuming a nutritionally well-balanced diet rich in recommended and required key nutrients has played a significant role in cancer incidence reduction (Kamal et al., 2022). Cancer development is influenced by a variety of factors, including genetic predisposition, environmental exposure, and lifestyle choices (Marino et al., 2024). Among these, nutrition stands out as a controllable risk factor (Clinton et al., 2020). Several food components have been discovered to have potential carcinogenic properties, and their roles have been documented using epidemiological research, in vitro investigations, and animal models.

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Traditional healers are well aware of food's role in cancer prevention, occurrence, and treatment. Strong antioxidants, which primarily counteract harmful free radicals, are among the dietary components that reduce the incidence of cancer (Raman et al., 2016). Food fibers play an important role in the removal of potentially cancer-causing substances from the digestive system (Ma et al., 2021). Omega-3 fatty acids, one of the most prominent and sought-after food components, are well known for their anti-inflammatory properties (Aderinola et al., 2024), a condition that is closely related to cell disease. Among other food components, plant-derived phytochemicals with various biological functions that help to prevent carcinogenesis are worth mentioning. Various fruits, vegetables, whole grains, and legumes have played important roles in cancer prevention diets because they contain beneficial components in large quantities and have the necessary properties and dietary characteristics to contribute to human nutrition and health (Li et al., 2017). A wide range of berries, cruciferous vegetables, citrus fruits, allium family vegetables, spinach, and grass family foods have been extensively studied to confirm their potential anticancer properties (Hossain et al., 2022). Furthermore, certain herbs, spices, and beverages, such as green tea, turmeric, ginger, fermented liquids, and so on, have shown promising results in vitro and in vivo laboratory tests, with some of their roles confirmed through epidemiological observations (Hossain et al., 2022). Examining modern foods and their ingredients, along with genetic, environmental, and other factors that cause cancer, is considered crucial for finding solutions to cancer prediction issues. This includes looking at diets and their components, food pollutants and their byproducts, and cancer-causing substances in food from different sources.

2. Foods, its components and carcinogenesis

Various cancer types have been linked to dietary components, food contaminants, xenobiotic substances in foods and their metabolites, and food-associated carcinogens in cancer etiology studies (Bartoszek & Holota, 2023). The individual's genetic composition, familial background, susceptibility, and physiological and biochemical factors that support triggers, as part of the body's system compatibility, all play important roles (Rahman et al., 2018). Chemical and biochemical issues related to the environment, the workplace, and the habitat all have an impact on eating habits (Lowden et al., 2010). Diets that contain artificial substances, pesticides, nematicides, residues from natural and synthetic fertilizers, chemical metabolites, and food processing components, as well as packaging materials, stabilizers, storage compounds, coloring and flavoring agents, emulsifiers, taste enhancers, and both alcoholic and non-alcoholic beverages, require special attention (Rane et al., 2023). The roles and mechanisms of epigenetic and genetic factors are also considered cancer causatives, as they are thought to activate incorrect gene(s) and biochemical pathways that lead to disease onset (Sadikovic et al., 2008). Understanding these interactions is crucial for developing effective prevention strategies and promoting healthier eating habits. By focusing on whole, minimally processed foods and reducing exposure to harmful substances, individuals can potentially mitigate their risk of cancer and other diet-related diseases.

2.1. Epigenetic mechanisms for food-based carcinogenesis

The gene activity alterations occurring without any modifications in the primary DNA are termed the epigenetic fall-out (Portela & Esteller, 2010). These alterations are caused by the mutations of the primary DNA as well as its involved proteins, particularly histones, which are known to have vital functions in controlling the expressions of different genes. The epigenetic changes involve the DNA and histone modifications, as well as the control manipulations of the non-coding RNAs (Prandi et al., 2022). These modifications exert substantial influence on gene expression and are triggered by diverse environmental and lifestyle factors, including the often practiced dietary choices, or simply the diet patterns, lack of physical exercise, and consistent exposures to harmful substances causing malignancies and advancements of cancer(s) (Lorenzo et al., 2022).

The field of nutriepigenomics, which explores the interface between nutrition and epigenetics, has revealed that bioactive compounds found in various foods can induce epigenetic modifications affecting their cellular processes (Kaur & Kumar, 2020). Primarily, these modifications occur through three major mechanisms, namely, DNA methylation, histone modifications, and non-coding RNA-mediated gene regulation. In this context, several bioactive compounds present in food have been shown to have detrimental effects by modulating epigenetic mechanisms. For example, polyphenols, abundant in fruits, vegetables, and tea, have shown the capacity to alter DNA methylation patterns as well as histone modifications (Beetch et al., 2020). Polyphenols, with their inherent ability to counteract negative epigenetic regulations through epigenetic marker modification, cause the reactivation of advantageous genes, such as tumor suppressor, antioxidant, and DNA repair genes, as well as the deactivation of damaging genes, such as oncogenes involved in inflammation, cell cycle progression, cell growth, invasion, blood vessel formation, and cancer spread (Rajendran et al., 2022). Epigallocatechin-3-gallate (EGCG), a catechin-based compound found in green tea, has been shown to inhibit DNA methyltransferases and histone deacetylases, potentially leading to the reactivation of cancer cells' tumor suppressor genes (Rajavelu et al., 2011; Nalla et al., 2025). Curcumin, the primary component of turmeric (Curcuma longa), has been shown to regulate histone acetylation and DNA methylation, influencing the expression of genes associated with inflammation and cancer progression (Link et al., 2013). For the TRAMP animal model of prostate cancer, the curcumin prevented tumor development by reversing the Nrf2 promoter methylation gene and by suppressing the H3K4me3 epigenetic mark in the experimental LNCaP cells (Jiang et al., 2022). Curcumin also decreased methylation through the DLEC1 promoter gene toward colony formation in HT29 colon cancer cell lines (Albani et al., 2010). Resveratrol, a potent molecule present in grapes and red wine, has been shown to modify the miRNA expression profile, possibly leading to its anti-inflammatory and anticancer characteristics (Mohammed et al., 2025). The role of blueberries was also discovered to be involved in modifying the DNA methylation patterns of genes related to insulin signaling and inflammation in individuals with type 2 diabetes, thereby indicating the potential influence of blueberries in preventing type 2 diabetes through epigenetic mechanisms (Ma et al., 2018). Additionally, under the in vitro conditions, the applications of resveratrol downregulated the metastasis-associated protein 1 by acetylating the p53 factor in the PCa cell lines (Soltani et al., 2016).

Cruciferous vegetables, which contain significant quantities of isothiocyanate, such as sulforaphane, have undergone a thorough investigation for their impact on epigenetics (Hudlikar et al., 2021). Sulforaphane has demonstrated the ability to hinder histone deacetylases and prompt alterations in miRNA expression, which may contribute to its chemo-preventive characteristics (Pan et al., 2018). Omega-3 fatty acids, which are present in higher proportions in certain fish and specific plant oils, have been linked to the regulation of DNA methylation patterns and histone modification (Frankhouser et al., 2022). Omega-3 fatty acid ingestion may also lead to epigenetic modifications that contribute to anti-inflammatory and cardioprotective benefits (Sunagawa et al., 2022). The epigenetic impacts of meals extend beyond the plant-derived materials. The probiotics and fermented foods, which include supportive bacteria, have been demonstrated to affect the epigenetics of the host by producing short-chain fatty acids and other metabolites. Butyrate, a short-chain fatty acid synthesized by the bacteria in the gut, is a powerful inhibitor of histone deacetylase and has been linked to positive effects on gut health and also in reducing inflammation (Modoux et al., 2022).

The ability of foods to induce epigenetic changes has important implications for the prevention and treatment of a wide range of diseases, including cancer. The ability of bioactive substances to reverse specific changes has provided predictive opportunities for the prevention and treatment of various cancer types. Specific components in foods have been shown to demethylate DNA and inhibit HDAC, reactivating tumor suppressor genes (Stefanska et al., 2012). Cardiovascular diseases have also been studied in the context of nutriepigenomics, and epigenetic modifications caused by certain functional foods have been shown to influence the expression of genes involved in lipid metabolism, inflammation, and endothelial function, all of which contribute to better cardiovascular health (Evans & Ferguson, 2018). The Neurodegenerative diseases, such as Alzheimer's and Parkinson's, have been linked to epigenetic dysregulation, and neuroprotective foods, such as those high in polyphenols and omega-3 fatty acids, are known to exert positive effects via epigenetic mechanisms, influencing genes involved in neuronal survival and plasticity (Açar et al., 2023; Atlante et al., 2020). Nonetheless, encouraging discoveries have been made, but numerous obstacles remain in the way of overcoming the shortcomings in the field of nutriepigenomics to positively regulate gene function to our advantage in cancer prevention. Because of the complex composition of food matrices and the presence of numerous bioactive substances, it remains difficult to attribute specific epigenetic effects to individual food components. Furthermore, there is a lack of understanding of the specific quantity, timing, and duration of consumption of these foods that may appear to be required to cause long-term epigenetic changes that cause or prevent cancer genesis.

2.2. Carcinogenic foods

Carcinogenic food refers to the food components, intrinsic and external, that upon consumption increase the risks of cancer (Sugimura, 2002). The concept is derived from epidemiological inputs, toxicological investigations, and molecular and cellular biology research that established the connection between the specific food components and the incidences of cancers. The potential of food to cause cancers may emerge from various sources, including naturally existing substances, natural compounds, pollutants introduced during food production and processing, and chemicals generated during heating and preservation cycles (Rather et al., 2017). How these foods and their specific components contribute to the development of cancer is varied and intricate. These processes can directly harm DNA, change how genes are expressed, disrupt cell communication, and lead to long-lasting inflammation, which can eventually result in cancer (Ghazi et al., 2020). A number of food-borne carcinogens have the capability to operate as genetic change initiators, thereby directly inducing genetic alterations in the DNA (Ghazi et al., 2020). On the other hand, certain carcinogens function as promoters, thereupon increasing the proliferation and survival of the cells that are already in a precancerous state. These promoters can facilitate the progression of cancer by enhancing the growth signals within these cells, making them more resistant to apoptosis (Nosrati et al., 2017). Consequently, understanding the roles of both initiators and promoters is crucial for developing effective strategies to reduce cancer risk associated with dietary factors.

2.2.1. Red and processed meat

The carcinogenicity of red and processed meat has received a great deal of attention after the International Agency for Research on Cancer, a World Health Organization agency, issued a press release (#240) revealing the implications of red and processed meat in developing different cancers, especially the alimentary canal area cancers (Domingo & Nadal, 2016). The red meat, reddish in color as compared to the chicken and fish, encompasses the meat of larger animals, i.e., buffalo, cows, veal, pork, lamb, horse, and goat. Processed meat refers to the items that are typically prepared from red meat and undergo a set of procedures, such as curing, salting, freezing, and smoking. Examples include bacon or gammon, which sometimes contain significant quantities of minced fatty tissues, and sausages. Red meat consumption has been categorized as "likely to cause cancer in humans," while processed meat has been categorized as "known to cause cancer in humans" (Farvid et al., 2021a). Processed and cooked red meats are believed to contain several kinds of carcinogens that may lead to cancer, specifically colorectal cancer (Fig. 1) (Turesky, 2018). N-nitroso compounds, which are found in cured meats, are examples of such compounds. Heterocyclic aromatic amines are also produced in well-cooked meats and poultry products. Polycyclic aromatic hydrocarbons (PAHs) are produced when meat is smoked or cooked over a flame. Furthermore, heme, a blood component, can accelerate the nitrosation of naturally occurring secondary amines in the animal body and cause oxidative damage by catalyzing lipid peroxidation in the digestive system (Santarelli et al., 2008). Each of these compounds found in animal meats has demonstrated the ability to form DNA adducts. If these adducts are not neutralized by enzymatic systems, certain DNA adducts can cause mutations during cell division, potentially leading to cancer development. The consumption of carcinogenic compounds found in processed meat has significantly interfered with and altered human physiology, resulting in cancer risks in a dose-dependent manner. Consuming 50 g of processed meat per day increases the risk of colorectal cancer by 18 %. This corresponds to nitrosamine exposure of 0.005–0.05 μg/day and heme iron intake of 0.5–2.5 mg/day (Aykan, 2015). Regular consumers ($\geq$3 servings/day) accumulate PHAs, particularly benzo[a]pyrene, at levels of 0.03–1.5 μg/day, while high-temperature cooking methods increase exposure to heterocyclic amines in the range of 0.1–10 μg/day, raising cancer risks (Cheng et al., 2021). Raising cancer risks emphasizes the importance of understanding dietary choices and their long-term health implications. Reducing the consumption of processed meats and adopting healthier cooking methods may help mitigate these risks and promote better overall health (Qian et al., 2020; Godfray et al., 2018). In that context, Han et al., systematic review and meta-analysis indicated that the consumption of processed meat is associated with a modest yet significant increase in both cancer mortality and incidence (Qian et al., 2020). Reducing consumption by three servings per week did not appear to decrease the overall cancer mortality rate or the mortality rate for specific cancers, such as colorectal and esophageal cancers (Qian et al., 2020). The International Agency for Research on Cancer has stated that processed meat may induce cancer in humans, indicating its potential carcinogenic effects. Although the evidence is not particularly robust, it appears that reducing the consumption of processed meat may be an effective strategy to diminish cancer risk.

When red meat is consumed, the heme iron enters the human digestive system and forms N-nitroso compounds in both the stomach and the meat (Sasso & Latella, 2018). These products, along with reactive oxygen species (ROS), cause DNA damage. Red meat thermal treatment produces heterocyclic aromatic amines (HAAs), while cooking processes produce polycyclic aromatic hydrocarbons (PAHs) (Das et al., 2023). Both HAAs and PAHs produced during meat processing contribute to DNA damage in tissues, which is a critical step in cancer development (Bukowska et al., 2023). Fig. 1 depicts the progression of these harmful aromatic compounds through the human digestive tract and their potential carcinogenic effects. 'Several epidemiological studies have confirmed these findings for PAHs and their derivatives. Several surveys investigated the potential role of red and processed meat as risk factors for cancer development, as well as the public health implications of various red and processed meat-borne cancers. A total of 640 articles were analyzed, and the findings revealed that eating more red meat increases the risk of developing esophageal cancer (Choi et al., 2013). Another case-control study of 2101 primary lung cancer patients in the Lombardy region of Italy found that both red and processed meats were associated with adenocarcinomas and squamous cell carcinoma of the lungs (Lam et al., 2009). Similarly, consumption of both red and processed meats was found to be strongly associated with an increased risk of developing colorectal cancer (18 % higher) (Farvid et al., 2021a).

2.2.2. Highly processed food (UPFs)

Highly processed or ultra-processed foods (UPFs) are typically made up of food-derived ingredients and additives, as well as ingredients like

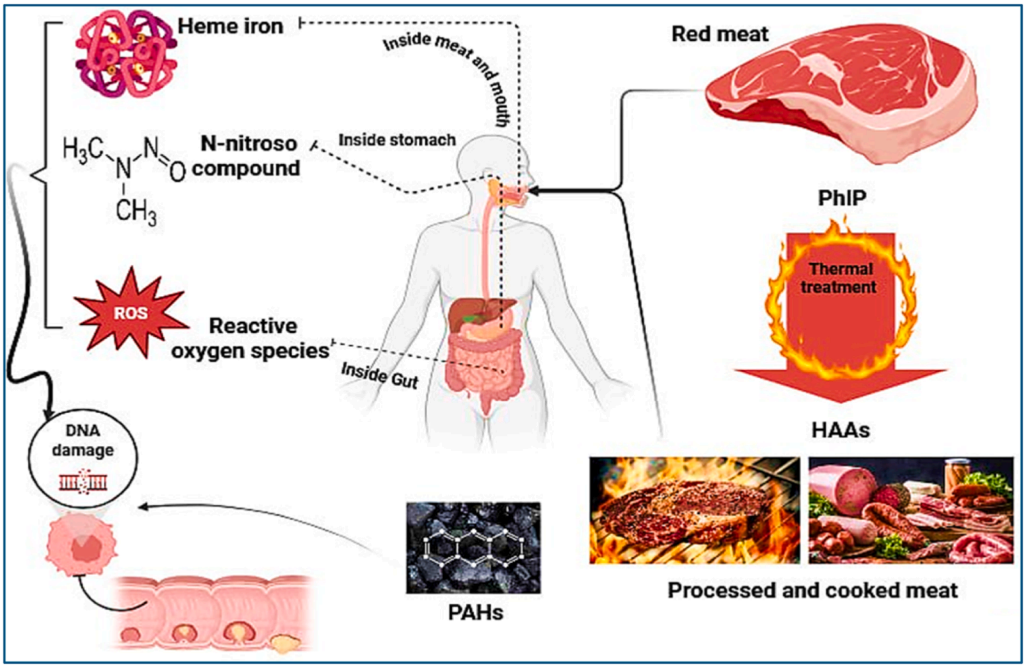

Fig. 1. Carcinogenic pathway associated with red meat consumption and processing. The heme iron in red meat enters the digestive tract and generates N-nitroso compounds both in the meat and the stomach. These chemicals, in conjunction with reactive oxygen species, induce DNA damage. The thermal processing of red meat results in the formation of heterocyclic aromatic amines (HAAs), whereas cooking methods yield polycyclic aromatic hydrocarbons (PAHs). The primary picture depicts the advancement of these chemicals through the human digestive system and its possible carcinogenic implications.

coloring agents, flavor molecules, sweeteners, solubilizers, and emulsifiers. These constituents add little to no value to the whole-food content. This description applies to sugar-sweetened beverages, confectionery, ice cream, chocolates, savory snacks, burgers, processed meat, frozen foods, and, to a lesser extent, canned foods (Pagliai et al., 2021). UPFs are known to increase total and saturated fats, sugar, and salt levels in food while decreasing fiber and vitamin content (Luiten et al., 2016). Despite certain nutritional values, UPFs have been found to contain neo-formed pollutants as a result of the Maillard reaction. These contaminants include acrylamide, heterocyclic amines, and PHAs, all of which have high carcinogenic potential (Chain, 2015). Furthermore, the packaging of ultra-processed foods contains elements that have prolonged contact with the container. The materials have also been suggested to have carcinogenic and endocrine-disrupting properties. Bisphenol A is one of the harmful substances found in ultra-processed foods (Muncke, 2011). Epidemiological studies have found a link between high UPF intake and an increased risk of cancer, including breast (Shu et al., 2023), colorectal (Wang et al., 2022), and prostate (Isaksen & Dankel, 2023). Advanced glycation end-products (AGEs), which are produced by the glycation of proteins and lipids, interact with cell surface receptors and activate signaling pathways, increasing tumor proliferation (Eva et al., 2022). AGEs also altered the gut microbiome. They are also held responsible for obesity-causing conditions as well as the progenitor of other disruptive content, which is among the causes and consequences of high UPF consumption and is significantly associated with a high incidence of cancers with varying degrees of severity, spread, and speed of the disease (Mao et al., 2021). UPFs also disrupt gut microbiota, increase intestinal inflammation, compromise barrier integrity, and alter metabolite production. These events promote carcinogenesis by increasing inflammatory cytokines, producing genotoxic metabolites, and causing immune dysregulation (Anastasiou et al., 2025). This dysregulation can lead to an environment conducive to tumor growth and progression, further highlighting the importance of dietary choices in cancer prevention. Consequently, reducing the intake of ultra-processed foods may serve as a critical strategy for improving overall health and mitigating cancer risk (Babalola et al., 2025). In collusion, ultra-processed foods (UPFs) are laden with detrimental fats, sugars, and additives, significantly contributing to the onset of cancer via mechanisms such as systemic inflammation, dyslipidemia, and oxidative stress.

2.2.3. Alcoholic beverages

Alcoholic beverages are widely acknowledged as probable contributors towards multiple cancer triggers, which have been supported by strong epidemiological evidence that links their intake to a higher risk of malignancies. Since 1988, the International Agency for Research on Cancer (IARC) has categorized the consumption of alcoholic beverages as carcinogenic to humans (Pflaum et al., 2016). The carcinogenicity of alcoholic beverages is associated with the presence of several materials in their formulation substances and the processes of liquor making. The constituents, ethanol and its metabolite, acetaldehyde, are the primary ingredients that have been held as the primary cause of cancers (Kokkinakis et al., 2020). Mechanistically, ethanol is predominantly broken down by alcohol dehydrogenase into acetaldehyde, which is further converted into acetate by the enzyme aldehyde dehydrogenase. Acetate can then be utilized by the body for energy or converted into fatty acids. However, the accumulation of acetaldehyde, a toxic compound, can lead to cellular damage and has been linked to various forms of cancer, including those of the mouth, throat, and esophagus (Zakhari, 2006).

Acetaldehyde is categorized under Group 1 carcinogens by the IARC owing to its capacity to induce DNA adducts, causing DNA cross-linking, and its ability to produce reactive oxygen species (ROS). These steps result in genomic instability and mutagenesis (Mizumoto et al., 2017). In addition to ethanol and acetaldehyde, alcoholic beverages also contain a variety of other potentially cancer-causing chemicals, including acrylamide, ethyl carbamate, polycyclic aromatic hydrocarbons, and furan (Hernandes et al., 2020). Meta-analysis-based results suggested that the daily consumption of one alcoholic drink is linked to an 11 % increased risk of breast cancer, as compared to the individuals who did not consume alcohol (Longnecker, 1994). Heavy drinking, 3 or more drinks/day, is highly associated with an increased risk of colorectal

(McNabb et al., 2020) and liver cancer development (Turati et al., 2014). Additionally, studies have shown that the risk associated with heavy alcohol consumption can be exacerbated by other lifestyle factors, such as poor diet, smoking, and lack of physical activity (Noble et al., 2015; Åberg et al., 2023). As a result, it is crucial for individuals to be mindful of their drinking habits and consider the cumulative impact on their overall health and cancer risk.

2.2.4. Contaminated foods

Contamination of foodstuffs originating either from agri-production, storage, packaging, derivatization, and processing or from microbiological contamination sources is the prime contributor to cancer genesis. The food processing industries' roles in inducing various chemicals during various processes of food preparation, packaging, and storage often introduce carcinogenic substances into the diet (Nerín et al., 2016), which have the ability to cause cancer owing to their intricate interactions with the human biological systems. The chemicals-based contaminants of particular concern include mycotoxins, aflatoxins, pesticide residues, toxic heavy metals, e.g., cadmium, lead, PAHs, and other persistent organic pollutants (Li et al., 2021). The aflatoxins, which are synthesized by the specific fungi, *Aspergillus flavus*, and *A. parasiticus* (Al-Ouqaili et al., 2018), are powerful carcinogenic agents working against the liver (Liu et al., 2012). Pesticide residues, although now subject to governmental and institutional regulations, may also build up in the food chain, and be the cause of certain types of cancer (Ashraf et al., 2023). However, the data on this relationship is inconclusive, but the effects are known and are influenced by the in-take density of the exposure. For example, endosulfan, the most prevalent organochlorine pesticide, was at high levels of presence at 0.214 mg/kg in the serum analysis of the cancer patients, as compared to the control (non-exposed), and mean levels in subjects at 0.166 mg/kg, indicated the endosulfan role in cancer incidences owing to its high percentage in the blood of the affected subjects (Attaullah et al., 2018). The 4,4-DDE, a metabolite of DDT, showed a 6.89-fold increased serum concentration of the metabolite in cancer patients as compared to the control in the analysis (Mengistu et al., 2025). Biological contaminants, such as hepatitis B and C viruses in relation to liver cancer (Moudgil et al., 2013), and bacteria, such as Helicobacter pylori and gastric cancer, have been linked to carcinogenesis due to chronic infection and inflammation (Salman & Hawezy, 2020; Yang et al., 2021). These contaminants cause cancer via a variety of mechanisms, including direct DNA damage, epigenetic changes, oxidative stress induction, hormone disruption, and chronic inflammation. The intricate nature of these interactions, as well as individual heterogeneity in terms of cancer vulnerability, severity levels of contaminant exposure, and potential to cause problems in the human system, have been debated for their roles in carcinogenesis and risk probability.

2.2.5. Sugary foods and non-alcoholic beverages

Sugary soft drinks, desserts, and other carbonated and non-alcoholic drinks have been known to cause hyperinsulinemia and insulin resistance, which in turn have been implicated as crucial factors for initiating numerous types of cancers (Almahri et al., 2024). Regular consumption of sugary soft drinks and desserts is known to trigger rapid glucose absorption and thereupon subsequent hyperinsulinemia. Chronic hyperinsulinemia provokes insulin resistance (cells being less responsive to insulin signaling despite elevated insulin levels), and mechanistically, this insulin dysregulation is directly implicated in carcinogenesis. The insulin dysregulation stimulates the cell proliferation and apoptosis inhibition through the insulin and IGF-1 receptors that are substantially expressed in cancer cells, thereupon, in effect, causing the cancer.

Additionally, insulin resistance introduces a proinflammatory microenvironment, which is conducive to tumor initiation and progression (Chang et al., 2021). Although there is no precise quantity of consumption that definitively causes cancer, epidemiological studies have shown that a daily intake of sugary beverages ($\geq$ serving/day ~330 mL) may potentiate increased cancer risks in several organs, e.g., breast, colorectal area, pancreas, and gastric region (Farvid et al., 2021b; Feng et al., 2023; Davis et al., 2023). Also, among the most common fallout of insulin resistance and hyperinsulinemia is obesity (Zhang et al., 2021). Foods laden with extra, unnatural, processed, and concentrated sugars and beverages, are known to trigger genetic obesity, which, in certain instances, can potentiate the risks of getting cancers through disruption of different biochemical and other involved physiological pathways (Qi et al., 2012). Obesity, insulin resistance, and chronic inflammation may also result from diets heavy in sugar and refined carbohydrates. These disorders also contribute to the prognosis of cancers. Additionally, high blood sugar can encourage the growth of cancer cells (Chandran et al., 2014), and a recent study correlated the impact of certain beverages with cancers. Regular consumption of sweetened beverages has the potential to create cancers of the pancreas, esophagus, and breasts (Milajerdi et al., 2019; Chen et al., 2015; Romanos-Nanclares et al., 2021).

2.2.6. Food additives

Artificial colors, flavor agents, synthetic and artificial additives in foods, and chemical preservatives that are added to food packages and fast food preparations to improve their taste and shelf life (Awuchi et al., 2020), have been pinpointed for their health risks (Ramesh & Muthuraman, 2018). An awareness of the health hazards associated with fragrance and flavor molecules and synthetic dyes is essential since these contaminants are abundant in food products now available in the markets. Strictly following the food additives regulation can improve the overall alarming situation with the unprecedented use and risks of these food contaminants over time. Animal studies have, to a larger extent, established the connections among cancers and several food contaminants (Singh, 2018).

Organizations have recommended prevention procedures for microbial entities such as fungi, bacteria, and viruses in order to prevent food contamination hazards from spreading (Flynn et al., 2019). Food storage and transportation require the use of food preservatives, the presence of microbial entities, microbial byproduct testing, control, and containment. Food preservatives are a necessary ingredient for packaged food items, since without them, the foodstuff would not last long. These preservatives are intended to maintain food's appearance as well as its flavor and odor while extending its shelf life. These products are becoming an everyday necessity as well as critical for food transportation. Any control regime for microbial and synthetic food contaminant control and regulation needs to be forcefully implemented to prevent these cancer-causing ingredients. The recommended procedures may keep the food from spoiling for a very long time upon storage (Dey & Nagababu, 2022). However, it is essential to balance the use of preservatives with consumer health concerns. Increasing awareness about the potential risks associated with certain additives may lead to a demand for more natural alternatives in food preservation (Chauhan & Rao, 2024). In that context, animal model studies have shown that several preservatives can cause cancer. Any long-term exposure to these substances, such as butylated hydroxyanisole (BHA) and butylated hydroxytoluene (BHT), is cause for concern (Mizobuchi et al., 2022) due to their carcinogenic properties. Nitrates and nitrites used as meat preservatives are also classified as potent carcinogens, as they produce nitrosamines, another potent carcinogen (Zhang et al., 2023). Another chemical class, sulfites, and certain other individual chemical entities, i.e., sulfur dioxide, sodium bisulfite, and potassium bisulfite, are also classified as preservatives and form part of the winery. They are used to stop browning and control the taste, as well as to keep the contents from spoiling, and are under scrutiny for their harmful effects and potential carcinogenic effects. Sulfites are also used to preserve dried fruits, certain processed foods, and, in some cases, meat products (Kilic-Akyilmaz & Gulsunoglu, 2015). Sulfites may cause unwanted allergies in asthmatic patients (Witkowski et al., 2022). Although no direct carcinogenic effects have been established for this class of preservatives, the substances may cause oxidative stress and DNA damage (Oshimo et al., 2021). All types of additives, as well as several synthetic and harmful chemicals, including preservatives, should be avoided, whether as preservatives or in non-preservative roles (Javanmardi et al., 2019). This caution extends to both processed and packaged foods, where hidden additives can significantly impact health. Consumers are encouraged to read labels carefully and opt for fresh, whole foods whenever possible to minimize exposure to these potentially harmful substances (Temple, 2020).

2.2.7. Agri-based food pesticides and herbicides

Agriculture commonly uses pesticides, which are synthetic inorganic complexes. Pesticides are also organic and natural in origin, have a wide range of structural compositions and chemical diversity, and have been used as a single- or multi-component mixture of substances in agricultural crop production. Pesticides are used to stop, remove, eradicate, and repel pests, as well as other disease-carrying insects, airborne and buoyant winged creatures that affect humans and livestock, small animals, pets, and unwanted plant and animal species. The materials have also been combined with other materials. These products and their mixture combinations for pest and unwanted herb and weed control have been recommended as being avoided or used in moderation within safe limits. They also interfere with food production, processing, storage, and transportation (Saravi & Shokrzadeh; Rao & Madhulety, 2005). Pesticides and herbicides, as well as their naturally sourced equivalents, particularly chemical-based pesticides and pyrethrins, have been shown to leave traces of carcinogens. Nonetheless, organic farming and integrated pest management controls with herbal products are intended to reduce pesticide and herbicide risks (Costa et al., 2023). For example, the intake of chlorpyrifos (CPS), an organophosphate pesticide, was found to be 0.01 mg/kg/day via inhalation, while a reference dose level of 0.0003 mg/kg/day was established as the safe limit with no significant human health risks (Wołejko et al., 2022). These findings highlight the importance of monitoring pesticide exposure and adhering to safety guidelines to protect public health. Additionally, ongoing research into alternative pest management strategies continues to be essential for minimizing potential health risks associated with chemical pesticides (Pathak et al., 2022). Table 1 provides a summary of carcinogenic foods, their sources and contaminants, the biomechanistic involved, and the risks associated with various cancer types.

2.3. Food processing-based carcinogens

Raw food processing involving high temperatures of cooking, frying, boiling, grilling, and broiling also adds to the conversion of various food-ingrained substances into probable and potent carcinogens, especially HCAs and PAHs (Adeyeye, 2020; Bulanda & Janoszka, 2022). These materials are the byproducts of sugar and amino acids, both of which are present in the foods, and are formed under high-temperature processing of food materials (Ahmad Kamal et al., 2018). The reuse of cooking media, the denatured oils, also elevates the levels of harmful trans fats and acrylamide, a known carcinogen in foodstuffs (Başaran & Turk, 2021). The chances of lipid oxidation/peroxidation from the heated oils are also high enough to be a cause of concern, which has been related to increased cancer risks (Min & Boff, 2002). The processing cycle and improper storage conditions are known to yield molds and aflatoxins, also categorized as food carcinogens (Negash, 2018; Lehmane et al., 2022). These harmful substances can lead to serious health issues, prompting the need for stricter regulations and guidelines in food preparation and storage. Additionally, educating consumers about the risks associated with reused cooking oils and the importance of proper food handling could significantly reduce the incidence of foodborne illnesses and related cancers (Losasso et al., 2012).

In summary, significant reactive oxygen species (ROS) production

Table 1
Summary of carcinogenic foods, their sources and contaminants, biomechanistic involved, and risks of various cancer types.

Foods Type	Carcinogenic Contaminants	Foods Sources	Biomechanistic and symptoms	Probable Cancers Risk	References
Red and Processed Meat	N-nitroso compounds, Heterocyclic amines, PAHs, Heme iron	Smoked, Cured, or well-cooked red meat (beef, pork, lamb, *etc.*)	DNA Adduct formation, oxidative stress, inflammation, lipid peroxidation, nitrosation	Colorectal, esophageal, lungs	(Domingo & Nadal, 2016; Farvid et al., 2021; Turesky, 2018; Santarelli et al., 2008; Aykan, 2015; Cheng et al., 2021; Choi et al., 2013; Lam et al., 2009)
Ultra-Processed Foods (UPFs)	Acrylamide, Heterocyclic amines, PAHs, Bisphenol A, AGEs	Sugary drinks, fat-lazed and fat-free snacks, frozen meals, processed meat	Inflammation, gut dysbiosis, DNA damage, endocrine disruption	Mammary glands, colorectal, prostate	(Pagliai et al., 2021; Luiten et al., 2016; Chain, 2015; Muncke, 2011; Shu et al., 2023; Wang et al., 2022; Isaksen & Dankel, 2023; Eva et al., 2022; Mao et al., 2021; Anastasiou et al., 2025)
Alcoholic Beverages	Ethanol, Acetaldehyde, Acrylamide, Ethyl carbamate, PAHs	Wine, beer, spirits, and other hard drinks	DNA adducts, oxidative stress, genomic instability	Mammary glands, colorectal, hepatic	(Pflaum et al., 2016; Kokkinakis et al., 2020; Mizumoto et al., 2017; Hernandes et al., 2020; Longnecker, 1994; McNabb et al., 2020; Turati et al., 2014)
Contaminated Foods	Aflatoxins, Pesticides, Heavy metals (Cd, Pb), Viruses, Bacteria	Spoiled food, fungus-infected crops, polluted produce	DNA damage, chronic inflammation, oxidative stress	Liver, gastric	(Nerín et al., 2016; Li et al., 2021; Al-Ouqaili et al., 2018; Liu et al., 2012; Ashraf et al., 2023; Moudgil et al., 2013; Salman & Hawezy, 2020; Yang et al., 2021)
Sugary Foods Carbonated and Non-Alcoholic Beverages	High glucose/ fructose, insulin dysregulation	Soft and carbonated drinks, desserts, processed sweets	Hyperinsulinemia, obesity, proinflammatory state	Mammary glands, colorectal, pancreatic, gastric	(Almahri et al., 2024; Chang et al., 2021; Farvid et al., 2021; Feng et al., 2023; Davis et al., 2023; Zhang et al., 2021; Qi et al., 2012; Chandran et al., 2014; Milajerdi et al., 2019; Chen et al., 2015; Romanos-Nanclares et al., 2021)
Food Additives	BHA, BHT, Nitrites/ Nitrates, Sulphites	Preserved meats, packaged and fast foods, dried fruits	Oxidative stress, DNA damage, nitrosamine formation	Colorectal, others (based on animal studies)	(Awuchi et al., 2020; Ramesh & Muthuraman, 2018; Singh, 2018; Dey & Nagababu, 2022; Mizobuchi et al., 2022; Zhang et al., 2023; Kilic-Akyilmaz & Gulsunoglu, 2015; Witkowski et al., 2022; Oshimo et al., 2021; Javanmardi et al., 2019)
Pesticides and Herbicides	Chlorpyrifos, Endosulfan, DDT metabolites (e.g., 4,4-DDE)	Conventionally grown crops, contaminated soil/water-based agri-products	Hormone disruption, DNA damage, oxidative stress	Various (general cancer risk)	(Saravi and, Shokrzadeh; Rao & Madhulety, 2005; Costa et al., 2023; Wołejko et al., 2022)

and inadequate neutralization, diminished antioxidant efficacy, persistent inflammation, hormonal dysregulation due to dietary practices or prolonged intake of certain foods, direct exposure to diverse carcinogens from various food sources, irregular gene expression, gut health, and the body's incapacity to neutralize and mitigate the effects of carcinogens are principal factors in the onset and advancement of cancer. Nonetheless, various additional causative factors, both collectively and independently, contribute, including individuals' physical activity and nutritional support, a compromised immune system, hormonal imbalances resulting from the absence of specific food-derived substances, such as estrogen-mimicking compounds in the diet, and sleep disorders associated with stress, chronic infections, excessive sun exposure, and the consumption of tobacco and other deleterious substances.

3. Cancer preventive roles of various food types

A range of fruits, vegetables, whole grains, beans, legumes, and oils, together with minerals, vitamins, and phytochemicals, have demonstrated cancer-preventive and antithetical effects (Chakraborty et al., 2020). The foods have been known to lower risks for several cancer types and malignancies. The plant-based foods have especially shown the potential to fight cancer. Several fruits are known and named exclusively as containing cancer-fighting components, especially high-end antioxidants (Mohammed et al., 2023; Donaldson, 2004). In this context, it is important to highlight both traditional opinions and solid evidence regarding the food compounds that promote anticancer properties. Food components belonging to flavonoids, polyphenolics, anthocyanins, and other strong antioxidants, including vitamin C, resveratrol, and quercetin, are well documented (Pandey & Rizvi, 2009; Mohammed & Khan, 2022). Additionally, experts believe that certain food components, due to their diverse structures and reported bioactivity, function as cancer-preventive agents. Certain other food categories from different sources and traditionally classified are included and discussed in detail based on the literature and the concurrent information available. This discussion encompasses a variety of food sources, which are rich in these bioactive compounds. The synergistic effects of these foods, when consumed as part of a balanced diet, may enhance their anticancer properties and contribute to overall health and well-being (Rathod et al., 2023).

3.1. Fruits and vegetables

Fruits and vegetables are the cornerstone foods for cancer prevention. These products are rich in antioxidants, vitamins, carotenoids, polyphenols, vitamin C, folates, fibers, and various phytochemicals that have experimentally been demonstrated as having anticancer properties. There is consistent evidence supporting the idea that consuming more vegetables and fruits has a preventive impact against various types of cancers, including stomach, esophageal, lung, oral cavity, pharyngeal, endometrial, pancreatic, and colorectal. The positive impact of cruciferous vegetables on several types of malignancies may be attributed to their abundant concentration of various antioxidants and other phytochemicals (Talalay & Fahey, 2001). These compounds work synergistically to reduce inflammation, enhance detoxification, and protect cells from oxidative stress, all of which contribute to their potential cancer-fighting properties (Aslam et al., 2024). Incorporating a variety of these vegetables into one's diet may not only improve overall health but also serve as a vital strategy in cancer prevention.

Recent systematic reviews indicated that citrus-class (citrus-fruit-based) juices and their extracts possess significant anticancer properties. Some of these reviews specifically examined both preclinical and clinical evidence. Numerous other studies have also indicated that citrus fruits, particularly orange and grapefruit juices can inhibit the proliferation of cancer cell lines and may also retard tumor growth in animals. Flavonoids, vitamin C, and other significant bioactive compounds facilitate these effects by functioning as antioxidants, modulating the inflammatory pathways, and inducing apoptosis. Observational studies have also indicated that the consumption of citrus juices reduces the risk of oral and pancreatic cancers. These observations emphasize the necessity for more stringent clinical trials to validate these claims and explore the potential therapeutic advantages of citrus-derived produce in cancer prevention and treatment (Cirmi et al., 2017). Sulforaphane, a recognized isothiocyanate present in cruciferous vegetables, such as broccoli, cabbage, and cauliflower, is clinically reported to induce apoptosis in prostate cancer cell lines, irrespective of the presence of androgens, by activating tumor suppressor genes and enhancing the activity of the phase II detoxifying enzymes. Sulforaphane diminishes the levels of apoptosis-inhibiting proteins, such as cyclin D2 and survivin, while enhancing the activity of the Nrf2 genes. These actions induce cell cycle arrest, disrupt mitotic processes, and activate caspases. Earlier reports endorse the inclusion of cruciferous vegetables in the diet, as they may inhibit or halt cancer proliferation and also facilitate the destruction of tumor cells by natural apoptotic processes (Paulo et al., 2018). Additionally, cruciferous vegetables also have significant quantities of glucosinolates, which, under in vitro and animal model studies, have been demonstrated to possess potent anticarcinogenic capabilities, especially against digestive tract, liver, lung, and breast cancer. The intake of these vegetables has been correlated with a decreased risk of prostate cancer. The overall relative risk (RR) of these vegetables at the highest intake was 0.87 with a significant linear trend ($p = 0.002$), with a combined RR of 0.955 (95 % CI: 0.928–0.982) for every 15 g of cruciferous vegetables per day (Long et al., 2023). A meta-analysis involving 13 different epidemiological studies indicated that high cruciferous content consumption considerably reduced the risk of breast cancer with a relative risk (RR) factor of 0.85 (95 % CI = 0.77–0.94) (Liu & Lv, 2013).

Moreover, berries, such as strawberries, blueberries, raspberries, and blackberries, that are rich in ellagic acid and anthocyanins, have demonstrated their ability to inhibit tumor growth and stimulate the self-destruction of abnormal cells (apoptosis). Protection from free radicals (Denev et al., 2012) and suppression of inflammation by 50 μM and 100 μM of blackberry anthocyanin formulation are also known. The use of pro-anthocyanidins, 150 μg/ml and 500 μg/ml, in lipopolysaccharide-induced inflammation (LPS, 1 μg/mL, 24 h) (Land Lail et al., 2021) has induced cell cycle arrest, apoptosis, and angiogenesis inhibition with a reduction in cell proliferation by 16.7 % ($p < 0.01$) and 87.7 % ($p < 0.01$), respectively (Wang et al., 2021). The strong prophylactic effects of berries have worked against cancer cells, particularly colon cancer cell lines (Fig. 2).

The berry polyphenols enhance cell adhesion protein expression (E-cadherin, P-cadherin, ICAM-1, VCAM-1) and stabilize the cellular junctions (Mauray et al., 2012). Berry antioxidants also counteract oxidative stress by regulating ROS, SOD, CAT, and GPx bioactivities. The berry compounds are known to suppress inflammatory mediators (TNF-α, IL-1β, IL-6, IL-15) and downregulate the NF-κB, COX-2, and iNOS signaling (Mohammed & Khan, 2022; Hameed et al., 2020; Huang et al., 2025; Kukula-Koch et al., 2024). At the cellular level, the berry's phytochemicals induce cell cycle arrest at the G1/S checkpoint and inhibit proliferation pathways (PCNA, Ki-67, cyclins, CDKs, MAPKs) to promote apoptosis through modulation of Fas, Bcl-2/Bax ratio, caspase activation, and PARP cleavage (Farghadani & Naidu, 2023). Additional preventive mechanisms include suppression of angiogenesis (VEGF, CD31, vWF) and inhibition of metastasis (Tsakiroglou et al., 2019). These effects collectively contribute to the berry compounds' potential as a natural therapeutic option in cancer prevention and treatment.

Additionally, select berries and citrus fruits, abundant in flavonoids and limonoids, particularly those found in Citrus species such as oranges, lemons, and grapefruits, have been shown to reduce the incidence of several cancers, including colon and lung cancers. A pooled analysis of 17 studies revealed that individuals who frequently consumed citrus fruits exhibited a 50 % reduction in the incidence of oral cavity and pharyngeal cancers compared to those who consumed minimal amounts

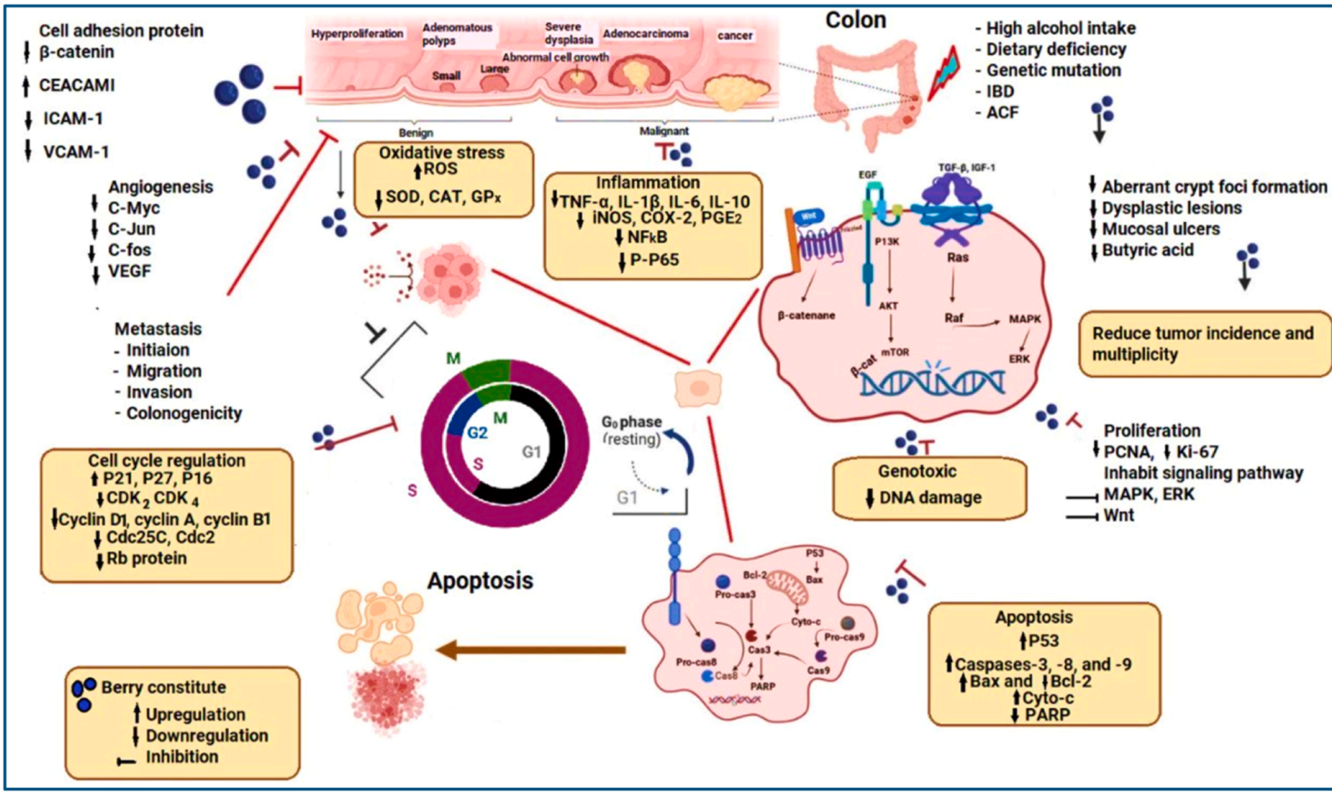

Fig. 2. Molecular mechanistic pathways of various mechanisms of colon cancer's plausible prevention by in-take of berries. Berry polyphenols increase the expression of cell adhesion proteins (E-cadherin, P-cadherin, ICAM-1, and VCAM-1) in the colon epithelium, which helps to stabilize cellular connections. Berry antioxidants combat oxidative stress by neutralizing ROS levels and modulating ROS, SOD, CAT, and GPx activities. Berries include anti-inflammatory chemicals that reduce TNF-α, IL-1β, IL-6, and IL-15 levels while also inhibiting NF-κB, COX-2, and iNOS signaling. Berry phytochemicals cause cell cycle arrest at the G1/S checkpoint. Berry components suppress proliferation pathways (PCNA, Ki-67, cyclins, CDKs, and MAPKs) and induce apoptosis via regulating Fas, the Bcl-2/Bax ratio, caspase activation, and PARP cleavage. Additional preventative measures include angiogenesis suppression (VEGF, CD31, vWF) and metastatic inhibition.

of citrus fruits (odds ratio: 0.50; 95 % confidence interval 0.43–0.59) (Cirmi et al., 2018). The flavonoids in citrus fruit peels have exhibited potential anticancer properties and cancer prevention by inhibiting cell growth, regulating the cell cycle, and preventing metastasis. Koolaji et al. published a comprehensive investigation on the bioactivity of specific flavonoids in citrus fruits (Koolaji et al., 2020). Koolaji et al. highlighted that compounds such as hesperidin and naringin play significant roles in these protective effects, underscoring the importance of incorporating citrus fruits into a balanced diet for enhanced health benefits.

3.2. Whole grains

The use of whole grains (WG) as part of diets has become vital in cancer prevention strategies. The unprocessed, unrefined grains are a great source of fiber, vitamins, minerals, and phytochemicals since they have their bran, germ, and endosperms intact (Chen et al., 2016). Advocacy for increased consumption of whole grains has continuously been sought in a number of epidemiological works for their benefits of lowering the chances of several diseases, including colorectal cancer. Certain processes, such as altered gut microbiota, decreased inflammation, and improved removal of possible carcinogens, are considered to be responsible for the protective benefits of whole grains (Andersen et al., 2021). Bioactive substances, i.e., phenolics, lignans, and phytic acids, have antioxidant and anticarcinogenic properties. The high fiber content also shortens the foods' colon transit time, thereby limiting the exposure of the food to potentially hazardous food-laden chemicals and thus avoiding the chances of cancer initiation (Yang et al., 2019). Compared to low whole grain intake, individuals with high whole grain intakes were inversely associated with colorectal, esophageal, and gastric cancers (Zhang et al., 2020). The meta-analyses indicated that whole grains, e.g., wheat, rice, barley, and sorghum, contained phenolic acids, lignans, and β-glucans, which can reduce the risk of breast cancer, particularly in postmenopausal individuals. Whole grains also enhance the efficacy of the treatments through proper nutrition and may reduce the adverse effects, thereby rendering the nutritional aspect a promising component of the cancer treatment regimens (Xie et al., 2019). Certain reports indicated that increasing the consumption of whole grains may reduce the risk of breast cancer. The study also recommended formulating foods aimed at mitigating cancer risk (Xie et al., 2019). In another meta-analysis, it was again confirmed that WG intakes are highly associated with reduced risk of cancers (Gaesser, 2020). This suggests that incorporating whole grains into the diet could be a strategic approach in cancer prevention.

3.3. Legumes

Various legumes, e.g., beans, lentils, and peas, have garnered significant attention in cancer prevention nutritional schemes owing to their rich nutrient profile and potential anticancer properties. The legumes are also rich in fibers, vitamins, proteins, minerals, and bioactive components. The legumes are characterized by the presence of polyphenols, saponins, and phytosterols, a class of compounds. Some of the meta-analysis studies have conjoined higher legume consumption with reduced risk of cancers and cancer mortality, particularly for colorectal and prostate cancers (Papandreou et al., 2019). The anticancer properties of legume constituents are attributed to multiple mechanisms, including antioxidant potential (Xu & Chang, 2012), modulation of the cell signaling pathways (Thompson et al., 2012), and regulation of gene expression (Campos-Vega et al., 2013). In particular, the soybean isoflavones have been shown to possess anti-estrogenic properties, which may have an impact on hormone-dependent malignancies owing to their similarity in structure with the human female estrogen hormone, 17-β-estradiol, which binds to alpha and beta estrogen receptors and mimics the activity of estrogens in target organs (Fig. 3). According to plasma concentration observance through different time passes, the areas under the curve (AUC) for each chemical, which showed absorption, were around 2.94 lg/ml hr for daidzein and 4.54 lg/ml hr for genistein and glycosides genistin and daidzin, the estrogen-mimicking aglycone components (Vitale et al., 2013). Certain peptides from legumes also induced molecular mechanisms against cancer cells through interactions with matrix metalloproteinases, DNA damage, and reduced mitochondrial potential, the actions associated with the anticancer effects (Luna-Vital & González de Mejía, 2018). The high fiber content of legumes may also aid in reducing inflammation and enhancing gut health. These two aspects are becoming widely acknowledged for their protective actions against a number of cancer causatives (Awika et al., 2018). These protective actions highlight the importance of incorporating legumes into a balanced diet, as their bioactive compounds, along with their fiber content, may play a significant role in cancer prevention.

The isoflavones exhibit strong antioxidant properties that neutralize the free radicals in malignant cells, thereby mitigating oxidative damage. Secondly, the isoflavones competitively bind to the androgen receptors, thereby interfering with the androgen-mediated cell proliferation pathways (Mahmoud et al., 2014). Third, the isoflavones interact with the estrogen receptors through two distinct mechanisms, i.e., direct binding to the β-estrogen receptors, which decreases the cell proliferation while simultaneously increasing the apoptosis (Xu et al., 2009). The competitive inhibition of estrogen receptors reduces the cell proliferation triggered by endogenous estrogens (Fig. 3). The structural homology between the isoflavones and estradiol enabled these compounds to modulate hormone-dependent cancer pathways, potentially explaining their chemopreventive effects, as observed in epidemiological studies of populations with high soy consumption (Tanwar et al., 2021). These findings suggest that isoflavones could serve as a viable strategy for reducing the risk of hormone-dependent cancers, further emphasizing the importance of dietary choices in cancer prevention.

3.4. Fishes

Fishes, high in omega-3 fatty acids, especially docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) (Yi et al., 2023), have been extensively studied for their important role in cancer prevention. These polyunsaturated fatty acids (PUFAs) alter the cell signaling pathways linked to the development of cancer. The products have also shown anti-inflammatory properties (VanderSluis et al., 2017). Several mechanisms have been suggested for their protective action against cancers, including the inhibition of angiogenesis, stimulation of apoptosis in cancer cells, and the suppression of pro-inflammatory eicosanoids (Fig. 4) (Kansal et al., 2014). The inhibitory effects of omega-3 fatty acids on tumor growth directly affect the cancer cells and indirectly affect the host immune system, specifically through anti-inflammatory mechanisms, and this phenomenon has been considered for its perceived anticancer bioactivity. In prostate cancer, omega-3 fatty acids are known to regulate several complex metabolic processes, including β-oxidation, the release of lipids from glycerophospholipids, direct activation of nuclear receptors, and gene transcription, all of which may influence the growth and progression of prostate cancers (Gu et al., 2013). A correlation between cancer risks among fish consumers indicated a lower incidence of colon cancer. Preclinical studies identified multiple mechanisms of actions of fish components for their significant role in reducing breast and colon cancer risks (SRR 0.94, 95 % CI 0.89–0.99) (Caini et al., 2022; Nindrea et al., 2019). Fish-sourced vitamin D and selenium have positive effects on preventing cancer occurrences (Janoušek et al., 2022; Guo et al., 2021). Additionally, omega-3 fatty acids found in fish have been associated with anti-inflammatory effects, which may further contribute to cancer prevention (Wall et al., 2010). These combined benefits highlight the importance of incorporating fish into the diet as a potential strategy for reducing the risk of various cancers.

A known, multifaceted anticancer mechanism of omega-3 polyunsaturated fatty acids (PUFAs), including alpha-linolenic acid, eicosapentaenoic acid, and docosahexaenoic acid is well established (Montecillo-Aguado et al., 2023). The bioactive lipids generate specialized pro-resolving mediators (resolvins, protectins, maresins) that exert multiple beneficial effects (Irún et al., 2019). At the cellular

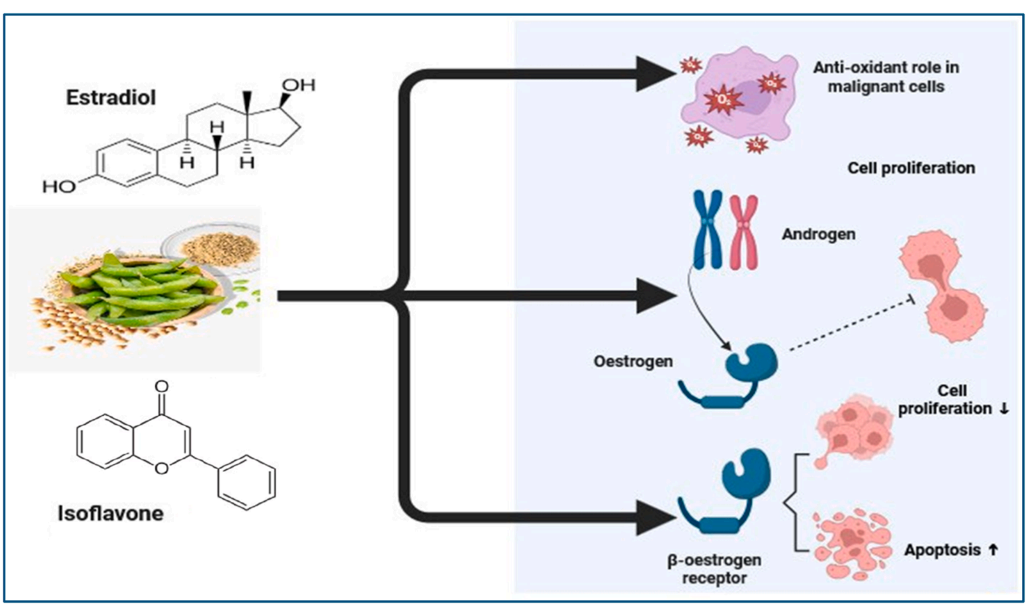

Fig. 3. Potential mode of action of isoflavones (derived from soybeans, *Glycine* max, and other legumes) in cancer prevention. Isoflavones have antioxidant capabilities that neutralize free radicals in cancer cells, reducing oxidative damage. Isoflavones can competitively attach to androgen receptors, disrupting androgen-mediated cellular growth pathways. Its engage with estrogen receptors via two distinct mechanisms: direct binding to β-estrogen receptors, which diminishes cell proliferation while concurrently enhancing apoptosis (programmed cell death), and competitive inhibition at estrogen receptors, which attenuates cell proliferation induced by endogenous estrogens.

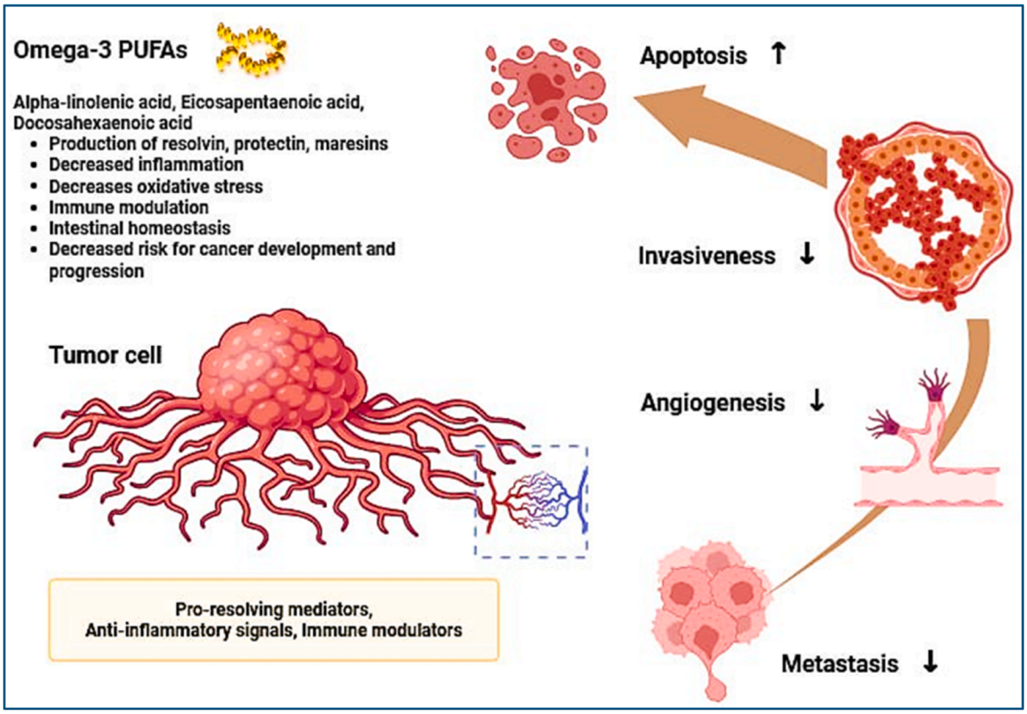

Fig. 4. An illustration of the effects of omega-3 acids on tumor cells. PUFAs induce apoptosis in cancer cells while concurrently diminishing tumor invasion. They also impede angiogenesis and reduce metastasis. The anti-inflammatory characteristics of PUFAs reduce oxidative stress, boost immunological regulation, and promote intestinal equilibrium.

levels, omega-3 PUFAs promote apoptosis in cancer cells, while simultaneously they reduce tumor invasiveness. The PUFAs also inhibit angiogenesis (formation of new blood vessels) and suppress cancer's further spread (Spencer et al., 2009). The anti-inflammatory properties of omega-3 PUFAs also contribute to decreased oxidative stress, enhanced immune modulation, and improved intestinal homeostasis. Improved intestinal homeostasis plays a crucial role in maintaining overall health and preventing various diseases. By supporting a balanced gut microbiome, omega-3 PUFAs can further enhance metabolic functions and promote a robust immune response, ultimately contributing to the body's resilience against chronic conditions (Sanz et al., 2025).

3.5. Genus Allium vegetables

The plant genus *Allium* includes families of garlic, onions, leeks, and chives, which have received considerable attention in restricting cancer development (Asemani et al., 2019). These vegetables contain high levels of organosulfur compounds, specifically allicin, diallyl sulfide, and S-allyl cysteine, which have been shown to possess anticarcinogenic properties (Alam et al., 2023). Concurrent epidemiological data indicated that increased intake of allium-class vegetables suppressed several types of cancers, particularly stomach, prostate, and colorectal cancers (Zhang et al., 2022). *Allium* compounds work through a variety of processes that contribute to the perceived anticancer properties. The involved mechanisms include antioxidant action, regulation of carcinogen metabolism, reduction of cell growth, activation of apoptosis, and suppression of the formation of new blood vessels, the angiogenesis (Patiño-Morales et al., 2021). Preliminary research conducted in laboratory settings, and on animal models has demonstrated that orally taken garlic extract prevents cancers and suppresses the development of tumors, as well as adds to the effectiveness of standard cancer therapies using the T24 BC BALB/C-nude mouse xenograft animal model. The garlic extract showed significant differences in tumor volume and tumor weight in the animal groups fed with 20 mg/kg ($p < 0.05$) extract as compared to the control group (Kim et al., 2017). An intake of total *Allium* vegetables was reported to be significantly contributing to gastric cancer prevention based on the meta-analysis of the study involving 17 epidemiological observations (Dalmartello et al., 2022). Additionally, the *Allium* vegetables also exhibited immunomodulatory properties that potentially enhanced the anticancer effects (Marefati et al., 2021).

3.6. Nuts and seeds

A number of seeds, including nuts, have been observed to be working towards preventing cancer occurrence. Walnuts, hazelnuts, almonds, pecans, pistachios, and flaxseeds are sources of unsaturated fatty acids, proteins, fibers, vitamins, minerals, and various phytomedicinal chemicals, including polyphenols, phytosterols, α-tocopherol, and lignans, which have pronounced antioxidant properties (Gonçalves et al., 2023). A dose-preventive response relationship against cancer was observed in the population consuming the diet with 7 seeds and nut items together (Hoshiyama & Sasaba, 1992). A potential protective effect of nuts against colorectal cancer was demonstrated. In Taiwan, a prospective trial was carried out in women, which enrolled ~25,000 participants and followed them up every year for ten years to ascertain the preventive anticancer effects (Yeh et al., 2006). A case-control study involving 923 female colorectal cancer patients in Korea also revealed that a high intake of nuts was responsible for the lower incidence of colorectal cancers (Lee et al., 2018). Recent research also indicated that an intake of 28 g/day of nuts can cause an 11 % decrease in risk of cancer death, as compared with non-nut-consuming people (Balakrishna et al., 2022). Nuts and seeds are thought to prevent cancer through a variety of methods, including the presence of selenium and vitamin E, which are known to help prevent oxidative stress and neutralize the body-generated free radicals (Askarpour et al., 2020). Omega-3 fatty acids and phytosterols, known anti-inflammatory agents, also help reduce chronic inflammation linked to the development of cancers (Nabavi et al., 2015). Furthermore, several of the phytochemicals found in nuts and seeds have been shown to have the capacity to control gene expression, affect cell signaling pathways, and encourage cancer cells to undergo apoptosis (Khani & Meshkini, 2021). Walnuts are rich in ellagitannins, which are metabolized to urolithins, compounds with potential anti-estrogenic effects (Sánchez-González et al., 2017). The flaxseeds contain high levels of lignans and phytoestrogens that are suggested to influence hormone-dependent cancers (De Silva & Alcorn, 2019). These compounds may help regulate estrogen levels in the body, potentially reducing the risk of certain cancers, particularly breast and prostate cancer. Incorporating a variety of nuts and seeds into the diet can therefore be a beneficial strategy for enhancing overall health and potentially mitigating cancer risks.

3.7. Herbs and spices

Herbs and spices have been used for millennia in traditional medicine and everyday cuisine. These highly valued consumptive commodities not only add flavor to the foods but also promote good health. The health benefits of using spices may be due to their strong antioxidant properties and presence of minerals and vitamins, while their biological effects may be attributed to their ability to cause changes in various cellular processes, such as metabolism, cell division, apoptosis, cell differentiation, and immunocompetence (Zheng et al., 2016). Common herbs and spices include significant quantities of bioactive compounds, including alkaloids, flavones, polyphenols, triterpenes, and saponins. These compounds have the potential to operate as medicinal agents, capable of preventing chronic metabolic illnesses and cancers (Gao et al., 2021). Numerous herbs and spices, e.g., turmeric and curcumin, ginger, and rosemary, have shown promise in inhibiting cancer cell proliferation, inducing apoptosis, and modulating the signaling pathways involved in carcinogenesis. For instance, ginger leaf extract was tested at doses (50, 100, and 200 μg/ml for 24 and 48 h), and it interacted with the cAMP-responsive element-binding (CREB) site and activated the transcription factor 3, ATF3. Sesame leaf extract (250 g/ml and 500 g/ml) also recorded HCT116 cell cycle death during their G2/M phase (Hossain et al., 2022). The bioactive component, 6-gingerol, from ginger has been proven to inhibit cell proliferation, and push apoptosis in colorectal cancer cells through inhibition of the ERK1/2/JNK/AP-1 signaling pathway (Radhakrishnan et al., 2014). The black pepper and its active components possess the ability to scavenge free radicals, which is also thought to potentially aid in the prevention of cancers and regulation of tumor growth (Butt et al., 2013). The extracts of rosemary were identified to inhibit the viability of prostate and colon cancer cells, as well as induce cell cycle arrest at the G_2/M stage with the ability to reduce cancer cell migration (Chan et al., 2022). Finally, due to the lower toxicity and anti-inflammatory properties, together with the positive outcomes of consuming curcumin in preventing several cancers, curcumin's frequent use is highly recommended by nutritionists (Howells et al., 2021).

3.8. Fermented foods and beverages

Fermented foods and beverages typically undergo enzymatic conversions of their components, which involve the presence of a number of microbial organisms. These foods include high levels of probiotics, bioactive peptides, and other metabolites, which also sustain the body and provide health benefits (Baruah et al., 2022). Fermented foods possess cancer-preventive qualities that can be attributed to various factors and their chemical components. As reported, the concentration of lactose and other sugars, that are prone to fermentation, are chemically reduced in fermented foods, while the comparative concentrations of the phenolic compounds that possess antioxidant properties are raised after fermentation, which helps to prevent cancer incidence and progression (Kok & Hutkins, 2018). As part of fermented foods, probiotics have several advantages, including their ability to prevent mutations, fight against harmful microorganisms, reduce the risks of cancer incidences, alleviate diarrhea, improve lactose intolerance, lower cholesterol levels, boost the immune system, and suppress the *Helicobacter pylori* infection. Tasdemir, S.S. and N. Sanlier (Tasdemir & Sanlier, 2020), presented a detailed study showing the relationship between fermented foods and cancers, including cancers of the colon, colorectal, gastric, breast, and lungs. Probiotic cultures contained in foods are also believed to decrease exposure to chemical carcinogens (Średnicka et al., 2021). By altering the intestinal environment and decreasing metabolic activity by the bacteria that may produce cancer-causing compounds, the probiotic cultures can be regulated/restrained for their beneficial effects (Lili et al., 2018). This also can add to detoxifying carcinogens and allow metabolic products, like butyrate, to be produced, which enhances the ability to induce cell death and stimulate the immune system to combat the cancer cells' growth (Djaldetti & Bessler, 2017). The proapoptotic potential of *Propionibacterium freudenreichii* in fermented milk has been demonstrated in human gastric cancer cell lines. This effect is associated with the formation of short-chain fatty acids (SCFAs), specifically propionate and acetate types. In addition, the fermented milk enhanced the cytotoxic effect of camptothecin, a chemotherapeutic medication used for treating stomach cancer (Cousin et al., 2012). Studies have also demonstrated that consuming fermented soybeans and black and green beans may hinder the proliferation of human breast cancer cells as well as prostate adenocarcinoma cells, including triggering programmed cell death. Exposing breast cancer cells to fermented beans has led to activation of calpain and caspase 8, 9, and 3. These observations indicated that apoptosis is triggered by pathways involving mitochondria and endoplasmic reticulum (Chia et al., 2012). These findings suggest that incorporating fermented soybeans and various types of beans into the diet could potentially serve as a complementary approach in cancer treatment, enhancing the effectiveness of conventional therapies.

3.9. Olive oil

Olive oil, a cornerstone of the Mediterranean diet, has garnered significant attention for its role in cancer prevention. The food is rich in monounsaturated fatty acids, primarily oleic acid, and contains numerous bioactive compounds, including phenolics, squalene, and tocopherols (Farràs et al., 2021). The ability of olive oil to prevent cancers is due to its intricate phytochemical compositions' bioactivity, which possesses antioxidant, anti-inflammatory, and anti-proliferative capabilities. Both the phenolic and lipid components of olive oil contain a diverse range of antioxidant and anticancer components that may protect against colorectal cancer (Memmola et al., 2022). Multiple epidemiological and preclinical studies have provided evidence for the health benefits and the significance of extra virgin olive oil (EVOO), the first cold-pressed sole oil, in preventing cancers. In a recent case analysis of 13,800 patients under study and 23,340 controls in nineteen observational studies, a correlation between higher consumption of olive oil and lower odds of developing any type of cancer (log OR −0.41, 95 % CI: −0.53, −0.29), including breast cancer (log OR −0.45, 95 % CI: −0.78, −0.12) and digestive system cancer (log OR −0.36, 95 % CI: −0.50, −0.21), was observed (Grosso et al., 2013; Schwingshackl & Hoffmann, 2015). The phenolic compounds in EVOO, for instance, oleocanthal, tyrosol, and oleuropein, have been reported to modulate gut microbiomes that are reported to be directly linked to metabolic and inflammatory diseases and gut cancers (Cheng et al., 2020). Olive oil has been proposed as a prophylactic agent as well as a therapeutic material to inhibit the growth of colon cancers by triggering apoptosis. Additionally, the EVOO has also been found to decrease the production of cyclooxygenase 2 (COX-2) and Bcl-2 proteins, which play significant roles in the development of colorectal cancers (Pelucchi et al., 2011). Furthermore, studies have indicated that the bioactive compounds present in extra virgin olive oil, such as phenolic compounds, may enhance gut health by promoting beneficial microbial growth and reducing inflammation (Gavahian et al., 2019). These effects highlight the potential of olive oil not only as a dietary staple but also as a strategic component in cancer prevention and management.

4. Conclusion and prospects

Diet significantly influences both individual and public health, impacting disease prevention and treatment. Certain food types and dietary practices have been associated with various diseases, including cancer. Nutritional knowledge has evolved over time due to meticulous scientific research and observations. This highlights foods that can either induce or inhibit cancer. No single food is recognized as the most effective means to prevent or treat cancer. A balanced diet comprising a diverse array of foods, including fruits, leafy greens, whole grains, legumes, fish, and healthy fats such as olive oil, is recommended. The synergistic effects of these ingredients, particularly when enhanced by combining turmeric with black pepper to increase curcumin bioavailability, demonstrate the potential of dietary strategies in cancer treatment. Future research should concentrate on developing a scientific methodology to standardize food components through isolation, identification, and testing in vitro and in vivo. Establishing a balanced dietary regimen tailored to the prevention and treatment of cancer will be essential. To achieve optimal results, it is recommended that food items be methodically permuted and combined according to their established roles. Moreover, clinical trials are essential to validate these dietary strategies and to enhance understanding of how nutrition can combat cancer.

Ethical statement

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CRediT authorship contribution statement

Hamdoon A. Mohammed: Writing – review & editing, Writing – original draft, Conceptualization. **Ghassan M. Sulaiman:** Writing – review & editing, Writing – original draft, Conceptualization. **Ali Z. Al-Saffar:** Writing – review & editing, Writing – original draft. **Mosleh M. Abomughaid:** Writing – review & editing, Writing – original draft. **Nehia N. Hussein:** Writing – review & editing. **Zeina T. Salih:** Writing – review & editing, Writing – original draft. **Noora A. Hadi:** Writing – review & editing, Writing – original draft. **Mayyadah H. Mohsin:** Writing – original draft, Software. **Hayder M. Al-kuraishy:** Writing – review & editing. **Riaz A. Khan:** Writing – review & editing, Writing – original draft, Conceptualization. **Ahmed Ismail:** Writing – review & editing, Writing – original draft.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

No data was used for the research described in the article.

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