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Functional Foods in Cancer Prevention: Mechanisms, Evidence, and Future Directions

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ABSTRACT

Cancer is a serious global health issue, responsible for millions of deaths each year despite breakthroughs in detection and treatment. The growing realization that up to one-third of cancers could be avoided via dietary and lifestyle changes has drawn attention to the importance of nutrition in cancer prevention. Functional foods, which contain bioactive components such as polyphenols, carotenoids, omega-3 fatty acids, glucosinolates, and probiotics, protect beyond simple nutrition. These components regulate oxidative stress, inflammation, carcinogen metabolism, apoptosis, angiogenesis, and epigenetic regulation, influencing several phases of carcinogenesis. A large number of experimental, epidemiological, and clinical studies show a negative relationship between plant-based, bioactive-rich diet consumption and cancer risk, although the quality of the evidence differs by cancer type and community. Despite promising molecular findings, translation into clinical recommendations is hindered by issues of bioavailability, dosage consistency, interindividual variability, and a paucity of large-scale human trials.

Future research should combine precision nutrition, improved delivery devices, and well-designed longitudinal studies to better understand dose-response correlations and processes in certain malignancies. Functional foods thus represent a potential, multi-targeted, and safe cancer prevention technique that combines nutrition and molecular oncology.

Keywords: *Functional foods, Cancer prevention, Bioactive compounds, Phytochemicals, Nutraceuticals, Epigenetics*

INTRODUCTION

Cancer constitutes a leading contributor to global morbidity and mortality, with approximately 20 million new diagnoses and 9.7 million deaths reported in 2022, figures projected to rise sharply in the coming decades due to population growth, aging, and lifestyle transitions (1) (2). Cancer incidence and mortality vary markedly across regions, with Asia accounting for nearly half of all new cases and more than half of cancer-related deaths, underscoring the regional disparities driven by environmental, dietary, and socioeconomic factors. Among the major cancers, lung, breast, colorectal, and prostate malignancies account for the largest proportion of the global burden, collectively responsible for over 40% of all cases (2). While inherited genetic predisposition plays a critical role, a substantial fraction of the global cancer burden, estimates range from 30% to 50%, is attributable to modifiable lifestyle and environmental factors (3). Among these, dietary exposures are particularly influential, capable of

modulating carcinogenic processes through both direct effects on cellular pathways and indirect effects mediated via obesity, metabolic dysregulation, and chronic inflammation.

Epidemiological evidence robustly supports the protective role of diets rich in plant-based foods, including fruits, vegetables, whole grains, legumes, nuts, and seeds, against several malignancies. Meta-analyses have demonstrated that each 200 g/day increase in fruit and vegetable consumption is associated with a 10–15% reduction in the risk of colorectal, gastric, and breast cancers (4) (5). Conversely, high intake of processed meats, sugar-sweetened beverages, ultra-processed foods, and alcohol has been consistently associated with increased cancer risk, with pooled relative risks ranging from 1.2 to 1.5 depending on cancer type (3). These findings underscore the dual potential of dietary modification as both a preventive and risk-reducing strategy.

Functional foods, defined as foods that provide health benefits beyond basic nutrition, have emerged as a particularly promising approach for cancer prevention. (6). Such foods are enriched with bioactive compounds capable of modulating diverse cellular and molecular pathways implicated in carcinogenesis. These include polyphenols, flavonoids, carotenoids, glucosinolates, phytosterols, and omega-3 polyunsaturated fatty acids. Mechanistic studies have demonstrated that these compounds exert pleiotropic effects, including scavenging of reactive oxygen species, attenuation of chronic inflammatory signaling via inhibition of NF-κB and COX-2, induction of apoptosis through intrinsic and extrinsic pathways, suppression of angiogenesis via VEGF downregulation, modulation of epigenetic marks, and enhancement of DNA repair capacity (7); (8) (9) (10).

Among functional food classes, polyphenols are perhaps the most extensively studied. Flavonoids, phenolic acids, and anthocyanins have been shown to inhibit tumor cell proliferation, induce apoptosis, modulate cell cycle progression, and attenuate inflammation. For example, epigallocatechin-3-gallate (EGCG) from green tea suppresses epidermal growth factor receptor (EGFR) signaling, inhibits matrix metalloproteinases, and downregulates anti-apoptotic proteins such as Bcl-2. Carotenoids, including β-carotene and lycopene, exert antioxidant and immune-modulatory effects and have been associated with reduced risk of prostate and gastric cancers. Glucosinolates from cruciferous vegetables are metabolized to isothiocyanates such as sulforaphane, which induce phase II detoxification enzymes, inhibit histone deacetylases, and modulate oncogenic signaling pathways, thereby reducing cellular transformation. Omega-3 fatty acids, particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), suppress inflammatory eicosanoid production, modulate membrane fluidity, and influence tumor microenvironment composition, collectively inhibiting cancer progression.

Preclinical studies provide strong mechanistic evidence for the anticancer properties of these compounds, whereas human observational and interventional studies offer complementary epidemiological support. Cohort studies have consistently reported inverse associations between intake of cruciferous vegetables, berries, and fatty fish and risk of colorectal, breast, and prostate cancers (11) (4). However, the translation of these findings into clinical practice faces challenges, including variability in bioavailability, differences in food matrices, individual metabolic responses, and a paucity of long-term, large-scale randomized controlled trials. Moreover, the synergistic effects of multiple bioactives consumed as part of whole dietary patterns are not fully elucidated, complicating recommendations for specific functional foods.

Despite these challenges, functional foods remain an attractive, low-risk strategy for cancer prevention that can be integrated into broader public health initiatives. Emerging

evidence also suggests a potential role for functional foods in modulating the gut microbiome, enhancing immune surveillance, and mitigating therapy-related toxicities, further expanding their translational relevance (10) (11). Accordingly, this review aims to provide a comprehensive synthesis of current knowledge regarding the role of functional foods in cancer prevention, integrating mechanistic, preclinical, and clinical evidence. Additionally, it identifies critical gaps in knowledge and outlines future research priorities to optimize dietary strategies for cancer prevention and support evidence-based recommendations in oncology nutrition.

Classification of Functional Foods

The conceptual framework for classifying functional foods can be delineated along two principal axes: (i) the dominant bioactive constituent that confers physiological functionality, and (ii) the natural food matrix or dietary source from which these bioactive are derived. This dual perspective is essential for elucidating both mechanistic underpinnings and translational applications in cancer prevention.

1. Classification by Bioactive Component

Functional foods may be categorized by their predominant classes of bioactive molecules, each characterized by distinct biochemical activities and cellular targets implicated in carcinogenesis.

1. Polyphenols represent one of the most extensively studied groups, encompassing flavonoids, phenolic acids, stilbenes, and lignans. These compounds orchestrate multifaceted protective mechanisms, including attenuation of oxidative stress through direct radical scavenging and Nrf2-mediated upregulation of endogenous antioxidant defenses. They further suppress chronic inflammation via inhibition of NF-κB, AP-1, and COX-2 signaling cascades, and can modulate epigenetic landscapes by influencing DNA methyltransferases and histone deacetylases (12) (13).
2. Carotenoids, a class of lipophilic tetraterpenoids such as β-carotene, lycopene, lutein, and zeaxanthin, contribute to redox homeostasis through singlet oxygen quenching and regulation of gap-junction intercellular communication. They influence transcriptional networks via interactions with nuclear receptors (PPARs, RARs, RXRs), thereby modulating cell differentiation and apoptosis pathways (14) (15).
3. Flavonoids, including subclasses like flavonols (quercetin), flavanones (naringenin), and isoflavones (genistein), exert anti-proliferative and anti-angiogenic effects through interference with PI3K/Akt and MAPK signaling. Isoflavones, in particular, act as selective estrogen receptor modulators, a mechanism central to their putative role in reducing hormone-dependent malignancies(16) (17) (18)
4. Glucosinolates, sulfur-rich metabolites of cruciferous vegetables, yield bioactive hydrolysis products such as sulforaphane and indole-3-carbinol. These molecules activate the Keap1–Nrf2 axis, induce phase II detoxifying enzymes (e.g., GST, NQO1), and inhibit histone deacetylases, collectively enhancing genomic stability and impeding neoplastic transformation (19) (20).
5. Omega-3 polyunsaturated fatty acids (PUFAs), notably eicosapentaenoic acid and docosahexaenoic acid, exhibit anti-inflammatory, anti-proliferative, and pro-resolving effects through suppression of pro-inflammatory eicosanoid synthesis, modulation of membrane lipid rafts, and activation of PPAR-γ and GPR120 signaling (21) (22).
6. Dietary fiber serves both as a metabolic substrate and as a structural modulator of the intestinal microenvironment. Its fermentation by commensal microbiota yields short-chain fatty acids, particularly butyrate, which function as histone

deacetylase inhibitors, promoting cell-cycle arrest and apoptosis in colonic epithelial cells while sustaining epithelial barrier integrity (23) (24).

7. Probiotics and prebiotics collectively regulate host–microbe symbiosis, enhancing mucosal immunity, competitive exclusion of pathogenic bacteria, and detoxification of procarcinogens. Specific strains such as *Lactobacillus rhamnosus* and *Bifidobacterium longum* have demonstrated the capacity to modulate cytokine production, reduce genotoxicity, and attenuate tumor-promoting inflammation (25) (26).

2. Classification by Food Source

A complementary framework classifies functional foods based on dietary matrices, recognizing that bioactive compounds rarely act in isolation. The food matrix exerts profound influence on stability, bioavailability, and synergistic biological interactions.

1. Fruits and vegetables constitute the most robustly validated sources of bioactives, offering a complex milieu of polyphenols, carotenoids, vitamins, and fibers that collectively modulate cellular redox balance, detoxification enzymes, and immune surveillance. Large-scale cohort studies consistently associate high fruit and vegetable intake with reduced incidence of gastrointestinal, breast, and lung cancers (27)
2. Whole grains and legumes deliver a spectrum of resistant starches, β-glucans, lignans, and micronutrients that sustain gut microbial diversity and lower systemic inflammation. The butyrogenic fermentation of their fibers is mechanistically linked to suppression of colonic neoplasia (28) (29)
3. Nuts and seeds are enriched with tocopherols, phytosterols, and unsaturated fatty acids that exert hypolipidemic and anti-inflammatory effects. Epidemiological evidence correlates regular nut consumption with decreased risk of colorectal and pancreatic cancers (30).
4. Herbs and spices, such as turmeric, garlic, ginger, and rosemary, contribute concentrated phytochemical pools, curcumin, allicin, gingerols, and rosmarinic acid, capable of targeting multiple oncogenic pathways, including STAT3, NF-κB, and COX-2, thereby exerting both chemopreventive and adjuvant therapeutic potential (31).

In sum, this bidirectional classification, by bioactive principle and dietary provenance, reflects the integrative nature of functional foods in modulating carcinogenic pathways. The synergy between complex phytochemical matrices, host metabolism, and the gut microbiome underscores the paradigm shift from single-nutrient interventions toward holistic dietary pattern–based cancer prevention strategies.

Mechanisms of Action of Functional Foods in Cancer Prevention

Functional foods exert chemopreventive and tumor-suppressive effects through an intricate network of molecular, cellular, and systemic mechanisms. Their bioactive constituents target multiple hallmarks of cancer, modulating pathways that govern oxidative stress, inflammation, cellular proliferation, apoptosis, epigenetic regulation, hormone signaling, and tumor-microenvironment interactions.

1. Antioxidant Mechanisms

Polyphenols, carotenoids, flavonoids, and other bioactives mitigate oxidative stress by scavenging reactive oxygen species (ROS), chelating transition metals, and potentiating endogenous antioxidant defenses via activation of the Nrf2/ARE pathway. This results in enhanced expression of glutathione peroxidase, superoxide dismutase, and catalase, thereby protecting nucleic acids, lipids, and proteins from oxidative damage and reducing mutagenic events that initiate carcinogenesis (32) (33).

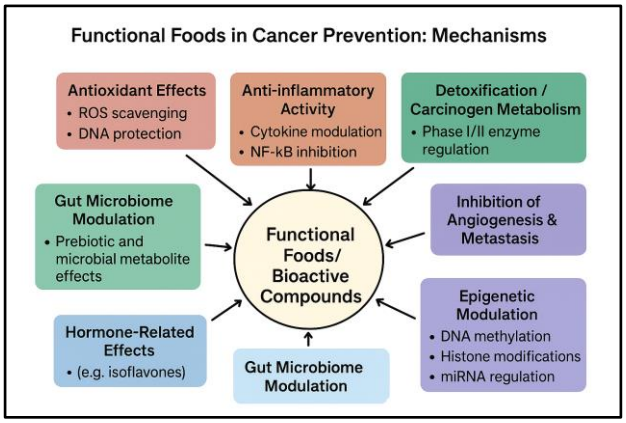
2. Anti-Inflammatory Modulation

Chronic, low-grade inflammation underpins tumorigenesis. Functional food bioactives suppress pro-inflammatory mediators, including TNF-α, IL-6, IL-1β, and prostaglandins, primarily by inhibition of NF-κB, AP-1, and COX-2 signaling pathways. Isoflavones, curcumin, and omega-3 PUFAs attenuate inflammasome activation and modulate immune cell polarization, disrupting the pro-tumorigenic inflammatory milieu (34).

3. **Modulation of Carcinogen Metabolism**
   Compounds such as glucosinolates, sulforaphane, and polyphenols regulate phase I (cytochrome P450) and phase II (e.g., GST, UGT) xenobiotic-metabolizing enzymes, facilitating detoxification of electrophilic carcinogens and preventing DNA adduct formation. This dual regulation enhances cellular resilience against genotoxic insults and attenuates initiation of malignant transformation (35) (36) (37) (38)
4. **Regulation of Cell Cycle, Apoptosis, and Autophagy**
   Functional bioactives induce cell cycle arrest at critical checkpoints (G1/S or G2/M) by modulating cyclins, cyclin-dependent kinases, and checkpoint regulators. Concurrently, they activate intrinsic and extrinsic apoptotic cascades via modulation of Bcl-2 family proteins, caspases, and p53 pathways. Certain polyphenols and isothiocyanates also promote autophagic flux, facilitating the removal of damaged organelles and contributing to tumor suppression (39) (40).
5. **Inhibition of Angiogenesis and Metastatic Progression**
   EGCG, curcumin, and resveratrol impede angiogenic signaling by downregulating vascular endothelial growth factor (VEGF) and its receptors while concurrently inhibiting matrix metalloproteinases (MMPs) responsible for extracellular matrix degradation. These effects collectively suppress neovascularization, invasion, and metastatic dissemination of malignant cells (41) (42)
6. **Modulation of the Tumor Microenvironment**
   Functional food components remodel the tumor microenvironment by influencing stromal cells, immune infiltrates, and extracellular matrix composition. Omega-3 PUFAs and polyphenols polarize tumor-associated macrophages toward an anti-tumorigenic M1 phenotype, enhance cytotoxic T-cell activity, and reduce myeloid-derived suppressor cell recruitment, collectively creating a milieu hostile to tumor proliferation (43) (44).
7. **Epigenetic Reprogramming**
   Bioactives exert profound epigenetic effects, modulating DNA methylation, histone acetylation/deacetylation, and microRNA expression. Sulforaphane, EGCG, and curcumin can reactivate silenced tumor suppressor genes and repress oncogene expression, thereby achieving long-term modulation of gene networks associated with carcinogenesis (45) (46).
8. **Hormonal and Endocrine Modulation**
   Phytoestrogens, particularly isoflavones, act as selective estrogen receptor modulators (SERMs), attenuating estrogen-driven proliferation in hormone-sensitive tissues. Through competitive receptor binding and downstream signaling modulation, these compounds reduce the risk of hormone-dependent malignancies, including breast and prostate cancer (47) (48)
9. **Gut Microbiome Modulation**
   Prebiotics, dietary fibers, and probiotics orchestrate gut microbial composition and function. Microbial fermentation produces short-chain fatty acids (SCFAs) such as butyrate, which possess anti-inflammatory, pro-apoptotic, and epigenetic-modulating properties. Additionally, gut microbiome modulation enhances systemic immune surveillance and strengthens mucosal

barrier integrity, indirectly attenuating tumorigenesis (49) (50).

In essence, the chemopreventive efficacy of functional foods derives from pleiotropic, synergistic actions that concurrently modulate oxidative stress, inflammatory signaling, detoxification pathways, cell fate, angiogenesis, epigenetic architecture, hormone signaling, and host–microbiome interactions. This integrative mechanism underscores their potential as adjunctive agents in dietary strategies for cancer prevention.



Challenges and Limitations

1. **Bioavailability and Metabolism**
   The therapeutic potential of bioactive compounds in functional foods is often constrained by poor bioavailability and rapid metabolism. Polyphenols such as curcumin and epigallocatechin gallate (EGCG) are extensively metabolized in the liver and intestine, leading to low plasma concentrations that may be insufficient to elicit anticancer effects (51), (52). Similarly, sulforaphane from cruciferous vegetables exhibits instability and rapid biotransformation, limiting systemic exposure (53). Overcoming these limitations requires formulation strategies such as nanoencapsulation or co-administration with absorption enhancers.
2. **Optimal Dose and Exposure Timing**
   Determining effective doses and exposure schedules for functional foods is challenging. The biological activity of bioactive compounds depends on concentration, frequency of intake, and interaction with food matrices. Additionally, the timing of consumption relative to meals or pharmacological treatments can alter absorption and efficacy. The lack of standardized dosing regimens in clinical studies complicates translation to dietary recommendations (54).
3. **Population Heterogeneity and Confounding Factors**
   Interindividual variability in genetics, microbiome composition, lifestyle, and environmental exposures can influence the effectiveness of functional foods. Such heterogeneity introduces confounding in epidemiological studies and can obscure causal relationships between dietary bioactive intake and cancer risk reduction. Accounting for these factors is essential for reliable interpretation of observational and interventional data.(55)
4. **Limited Large-Scale Randomized Controlled Trials (RCTs)**
   While preclinical studies provide mechanistic insight, human data are limited. Most clinical trials are small, short-term, or lack population diversity, restricting the generalizability of findings. Large-scale RCTs with standardized endpoints are needed to establish causality and inform evidence-based guidelines .(56)
5. **Safety Concerns and Interactions**

Although functional foods are generally considered safe, high-dose supplementation of bioactive compounds may pose adverse effects or interact with medications. For example, curcumin can modulate cytochrome P450 enzymes, potentially affecting drug metabolism. Comprehensive safety assessments and monitoring are required, especially in populations with comorbidities or polypharmacy.(57)

6. **Standardization and Regulatory Issues** Variability in source material, processing, storage, and formulation can lead to inconsistent bioactive content and activity. The lack of standardized quality control and regulatory oversight limits reproducibility across studies and complicates the integration of functional foods into public health recommendations. Establishing uniform standards for functional food production is critical for ensuring efficacy and safety.(58)

Future Perspectives

Future studies should focus on translating experimental findings into clinical evidence through well-designed human trials. Integrating omics-based tools and standardized assessment of bioactive compounds will help clarify mechanisms and optimize dosage. Exploring synergistic effects and incorporating functional foods into personalized nutrition models may enhance preventive potential. Collaborative, multidisciplinary research will be essential to establish functional foods as reliable, evidence-based tools in cancer prevention.

CONCLUSION

Functional foods play a significant role in cancer prevention by modulating oxidative stress, inflammation, and cellular signaling pathways. Although evidence from experimental and population studies is promising, consistent clinical validation is still lacking. Future research should focus on well-designed human trials and mechanistic studies to strengthen the causal link and guide the integration of functional foods into preventive nutrition strategies.

Declaration by Authors

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