Review

Lifestyle-Based Approaches to Cancer Prevention and Treatment: Diet, Physical Activity, and Integrative Strategies

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Lifestyle-Based Approaches to Cancer Prevention and Treatment: Diet, Physical Activity, and Integrative Strategies

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Abstract

Background/Objectives: Cancer remains a leading global cause of morbidity and mortality. Modifiable lifestyle factors, including diet, physical activity, weight management, tobacco and alcohol avoidance, and reduced exposure to carcinogens (e.g., ultraviolet radiation), play key roles in prevention and care. This narrative review synthesizes evidence on lifestyle-based interventions influencing cancer risk, treatment tolerance, and survivorship. **Methods**: A narrative review was performed in PubMed and Scopus, supplemented by manual screening via Google Scholar. Eligible publications (2001–2025) addressed diet, physical activity, obesity/weight control, carcinogen exposure (tobacco, alcohol, radiation), complementary therapies, or cancer outcomes. Priority was given to systematic reviews, randomized trials, and high-impact studies. **Results**: Evidence indicates that adherence to a predominantly plant-based diet, regular physical activity, and maintenance of a healthy body weight are consistently associated with reduced incidence of several cancers, including breast, colorectal, and liver cancer. Obesity and metabolic syndrome emerge as significant contributors to cancer onset and recurrence. Nutritional strategies, such as caloric restriction, ketogenic diets, and fasting-mimicking diets, have shown promise in enhancing treatment efficacy and improving quality of life. Robust epidemiology confirms that tobacco use markedly increases the risk of multiple cancers, while cessation reduces incidence and mortality; minimizing radiation exposure, particularly ultraviolet radiation through photoprotection and sun-avoidance behaviors, lowers skin-cancer risk. Complementary and mind–body therapies may alleviate treatment-related symptoms, although high-quality evidence on long-term safety and efficacy remains limited. **Conclusions**: Integrating lifestyle medicine into oncology offers a cost-effective, sustainable strategy to reduce cancer burden and enhance survivorship. Comprehensive programs combining dietary regulation, structured exercise, effective tobacco-cessation support, and pragmatic photoprotection/radiation-risk mitigation may extend healthspan, improve treatment tolerance, and help prevent recurrence.

Keywords: dietary interventions; exercise; weight management; tobacco cessation; radiation exposure; complementary therapies; healthspan

1. Introduction

Despite notable advances in cancer detection and treatment, completely eradicating the burden of cancer remains an unlikely outcome. A rational strategy for cancer control involves prioritizing primary prevention while also advancing new therapies and improving early detection. Promoting

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healthy lifestyle choices is central to effective primary prevention and lowering cancer risk. This includes adopting a predominantly plant-based diet, sustaining healthy body weight over time, engaging in consistent physical activity, avoiding or minimizing tobacco and alcohol use, and reducing exposure to radiation and carcinogenic substances. These preventive behaviors are expected to significantly lower the incidence of several cancers, such as melanoma, breast, colorectal, esophageal, liver, and head and neck cancers [1].

Specific lifestyle recommendations for preventing melanoma include consistent sunscreen use, wearing protective clothing, seeking shade, limiting sun exposure during peak hours (10 a.m. to 2 p.m.), and avoiding indoor tanning [2]. The role of carotenoid intake, particularly β-carotene and vitamin A, in cancer prevention has been explored, with findings indicating a notable inverse relationship between their consumption and cancer risk, including lung cancer [3]. Around 90% of lung cancer cases are attributed to smoking, and the combined use of tobacco and alcohol further elevates the risk of developing cancer. Globally, the use of smokeless tobacco is linked to approximately 400,000 cases of oral cancer, accounting for 4% of all cancers [4]. Therefore, tobacco cessation and alcohol moderation are likely to play a critical role in reducing cancer incidence.

There is a well-established connection between dietary habits, obesity, metabolic syndrome, and the development of various cancers. These factors are estimated to contribute to about 30–35% of cancer-related deaths, suggesting that many of these could be preventable through dietary interventions. Such approaches include fasting, fasting-mimicking diets, ketogenic diets, and calorie restriction [5].

Emerging research also highlights the synergistic potential of combining energy restriction — whether dietary or pharmacological— with regular physical activity to promote healthy aging and reduce cancer risk. These combined strategies have been shown to modulate key biological pathways involved in carcinogenesis, including inflammation and metabolic dysregulation [6].

This narrative review focused on lifestyle-based interventions aimed at improving cancer prevention and reducing associated risks, while also enhancing the overall health of cancer patients. Additionally, it explores the therapeutic potential of specific dietary strategies, nutritional supplements, and complementary and alternative medicine in cancer care.

2. Materials and Methods

This narrative review was conducted through a non-systematic search of the scientific literature in the electronic databases PubMed and Scopus, with additional manual search in Google Scholar to identify studies not indexed in the primary databases. The search was performed on 11 August 2025 and focused on peer-reviewed articles and reviews published within the last 25 years. The following Boolean string was used: ("cancer" OR "neoplasm" OR "tumor" OR "malignancy" OR "oncology" OR "cancer prevention" OR "cancer management") AND ("lifestyle" OR "lifestyle medicine" OR "behavioral interventions" OR "lifestyle interventions" OR "diet" OR "dietary pattern" OR "nutritional interventions" OR "nutrition" OR "physical activity" OR "exercise" OR "body weight" OR "obesity" OR "overweight" OR "micronutrients" OR "vitamins" OR "vitamin D" OR "vitamin B" OR "vitamin C" OR "vitamin E" OR "retinol" OR "beta-carotene" OR "carotenoids" OR "antioxidants" OR "dietary supplements" OR "nutraceuticals") AND ("complementary therapies" OR "complementary and alternative medicine" OR "CAM" OR "integrative oncology" OR "integrative medicine") AND ("carcinogen" OR "carcinogens" OR "tobacco" OR "smoking" OR "alcohol" OR "radiation") AND ("cancer risk" OR "cancer recurrence" OR "cancer outcomes" OR "survivorship") AND ("fasting" OR "intermittent fasting" OR "time-restricted feeding" OR "fasting-mimicking diet" OR "caloric restriction" OR "ketogenic diet") AND ("melanoma" OR "breast cancer" OR "colorectal cancer" OR "esophageal cancer" OR "liver cancer" OR "head and neck cancer" OR "pancreatic cancer" OR "prostate cancer" OR "lung cancer" OR "endometrial cancer"). Given the narrative design, no formal risk-of-bias appraisal was conducted; however, priority was given to recent systematic reviews, randomized trials, and studies in high-impact journals, with triangulation across multiple sources when available.

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2.1. Inclusion criteria

Studies were included if they met the following criteria:

- Peer-reviewed articles, systematic or narrative reviews, and meta-analyses.
- Published between 2001 and 2025.
- Focused on human subjects.
- Addressed at least one of the following themes: lifestyle interventions, dietary strategies, physical activity, obesity, complementary or alternative medicine, carcinogen exposure, or specific cancer types related to lifestyle factors.

2.2. Exclusion criteria

Studies were excluded if they:

- Were not written in English.
- Were published before 2001 unless deemed highly relevant.
- Focused exclusively on pediatric or veterinary populations.
- Reported preliminary data without peer review (e.g., preprints).
- Lacked relevance to the scope of lifestyle-based cancer prevention or management.

3. Intermittent and Periodic Energy Restriction (IF/FMD)

Fasting, traditionally associated with religious practices, involves the intentional avoidance of all food or certain types of food for a specified duration. In oncology, prolonged fasting—lasting several weeks or months—may not be advisable for cancer patients, as it can lead to unintended weight loss, which is often counterproductive. However, shorter-term fasting protocols, implemented over several weeks or months and consisting of fasting periods ranging from 12 to 72 hours followed by unrestricted food intake during specific eating windows, may be more feasible and suitable for individuals undergoing cancer treatment [7].

Intermittent fasting (IF), which involves alternating cycles of eating and fasting and typically includes calorie-restricted intake one to three days per week, is attracting growing global interest, particularly in oncology. This is due to its potential to modulate nutrient metabolism and energy homeostasis, support overall health, and possibly influence the development and progression of disease. A related but milder approach is the fasting-mimicking diet (FMD), which entails consuming a low-calorie, low-protein, plant-based diet for three to five days during each chemotherapy cycle [8].

The following sections will explore how nutrition influences cancer-related molecular pathways and how fasting regimens may affect tumor growth and treatment response.

3.1. Mechanistic Rationale: Cancer-Related Signaling and Nutritional Modulation

Cancer cells display a variety of traits that drive their malignant behavior. These include accelerated growth, impaired or lost tumor suppressor function, the ability to invade and spread to distant tissues and organs, stimulation of new blood vessel formation (angiogenesis), resistance to programmed cell death (apoptosis) and immune surveillance, as well as alterations in metabolic processes. The availability of nutrients such as sugars and amino acids influences several of these hallmarks, partly by modulating key signaling pathways, including insulin-like growth factor-1 (IGF-1), Ras, protein kinase A (PKA), and the PI3K–AKT pathway [9]. Persistent activation of these pathways can also result from mutations in oncogenes. To meet increased energy and biosynthetic needs, these signaling cascades often promote glycolysis, leading to elevated levels of glucose transporters and glycolytic enzymes in cancer cells. As a result, many tumor types become dependent on increased glucose intake or the supply of specific amino acids [10].

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Elevated blood glucose levels (hyperglycemia) have been associated with shorter progression-free and overall survival in cancer patients [11]. This underlines the importance of altered metabolic demands and glucose metabolism in cancer progression and patient outcomes, thereby supporting the potential role of nutritional strategies in regulating cancer cell growth and viability. In this context, fasting has demonstrated benefits for long-term health by decelerating the aging process, extending lifespan, reducing inflammation and oxidative damage, promoting cellular regeneration, and enhancing cardiovascular and cognitive functions [12]. Fasting also shows promise as a complementary strategy for cancer prevention and treatment by improving the efficacy and tolerability of anticancer therapies. Additionally, it may improve the quality of life in cancer patients through several adaptive biological responses triggered by the fasting state [13].

3.2. Fasting Regimens and Tumor Biology

Recent research has shown that altering the energy metabolism of cancer cells can suppress tumor development and enhance the immune system's capacity to combat tumors. For this purpose, there are several fasting regimes that need to be known; an overview is presented in Table 1.

Table 1. Fasting regimens: definitions and practical notes.

Fasting regimen	Definition	Reference
Fasting	Complete caloric abstention or exclusion of selected foods for a defined interval	[14]
Intermittent fasting	Alternation of eating and energy-restriction/water-only intervals on 1–3 days per week	[14, 15]
Periodic fasting	Severe energy restriction or water-only phases of ~48 h (up to a week in some protocols)	[18]
Short-term fasting	Time-limited abstention (≈12–72 h), e.g., alternate-day patterns	[19]
Fasting mimicking diet	Brief, hypocaloric, low-protein, plant-forward cycles aligned to treatment sessions	[17, 20]

Intermittent fasting (IF) has been found to decrease the adverse effects associated with chemotherapy and radiotherapy, while simultaneously increasing cancer cells' sensitivity to these standard treatments. As a result, IF is gaining recognition as a potentially valuable strategy in clinical oncology [14, 15].

Implementing fasting or a fasting-mimicking diet (FMD) can lead to reduced levels of blood glucose and insulin-like growth factor-1 (IGF-1). These metabolic changes affect several intracellular signaling pathways, such as PI3K-Akt, Ras, and the mammalian target of rapamycin (mTOR), thereby inhibiting cell growth and proliferation [16]. This is particularly significant because cancer cells are highly dependent on glucose metabolism, which makes them more vulnerable to chemotherapy when FMD is applied. This mechanism is referred to as differential stress sensitization (DSS), and it contributes to enhancing the therapeutic impact of anticancer treatments [21].

Fasting also activates a biological process known as differential stress resistance (DSR), through which healthy cells shift their focus toward protection and internal repair, potentially reducing the damage caused by chemotherapy. Recent preclinical findings suggest that fasting or FMD not only protects normal cells but may also support effector T-cell–mediated cytotoxicity, thereby strengthening the immune response against tumors [17].

Under fasting or FMD conditions, reduced insulin, leptin and IGF-1 levels down-modulate PI3K–Akt, Ras–PKA and mTOR–S6K signaling, decrease glucose transporter (GLUT) activity, and promote a metabolic shift toward oxidative phosphorylation (OxPhos) with increased reactive oxygen species (ROS) generation. These changes activate transcription factors such as FOXO, EGR-1 and p53 in normal tissues, enhancing stress-response and repair pathways that support treatment tolerance [21].

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3.3. Preclinical Evidence on Fasting

The benefits of incorporating fasting into cancer treatment have been strongly supported by preclinical research. Among these are the protection of healthy cells from the harmful side effects of chemotherapy, increased sensitivity of tumor cells to chemotherapy, and enhanced intratumoral infiltration of CD8+ T cells induced by chemotherapy, which collectively contribute to slowing disease progression [22]. In colon cancer mouse models, short-term fasting (STF) causes deficiencies in glucose and amino acids, triggering an anti-Warburg effect. This is marked by increased oxygen consumption without efficient ATP production, leading to elevated oxidative stress and programmed cell death (apoptosis) [23].

Recent mouse studies have demonstrated that intermittent fasting cycles—periods of 12 to 72 hours without food followed by refeeding—can have positive impacts on lifespan, age-associated diseases, physiological health markers, stress resistance, metabolic balance, and tissue regeneration [24, 25]. In xenograft mouse models of breast cancer, melanoma, and neuroblastoma, combining fasting with high-dose chemotherapy helped protect healthy cells from oxidative stress, DNA damage, and treatment-related endometrial hyperplasia [22]. Remarkably, findings from the same study suggest that two separate 48-hour fasting cycles alone were as effective as two rounds of chemotherapy in curbing tumor progression [22].

These effects are thought to result from reductions in blood glucose, insulin, IGF-1, and inflammatory markers, along with increases in IGF-1-binding proteins (IGFBPs) and ketone bodies [26]. Altogether, these findings underscore the potential of non-pharmacological dietary strategies, when used alongside conventional treatments, to significantly improve survival outcomes in certain types of cancer.

3.4. Clinical and Translational Evidence on Fasting

To date, only a few small-scale clinical trials have explored the impact of various intermittent fasting protocols used alongside chemotherapy, focusing on their effects on cancer progression and prognosis through the evaluation of metabolic and hormonal parameters. These clinical investigations have confirmed that integrating fasting into chemotherapy regimens is both safe and generally well-tolerated. Moreover, fasting may help reduce the toxicity commonly associated with chemotherapy [16, 27–29].

One clinical study examined the safety, immune-modulating, and metabolic outcomes of a five-day, cyclic fasting-mimicking diet (FMD) administered in combination with standard cancer therapies. In a cohort of 101 patients, the FMD was shown to be feasible and well-tolerated, leading to consistent reductions in blood glucose and growth factor levels. These metabolic shifts resemble those seen in preclinical studies and are believed to contribute to the tumor-suppressive properties of fasting and FMD. Additionally, the FMD alters systemic and tumor-specific immune responses, activating multiple immune mechanisms with antitumor effects [30].

In a broader clinical context, over 300 cancer patients have undergone FMD cycles alongside other therapies, and the occurrence of fasting-related adverse effects has been notably low [27,30,31]. Furthermore, clinical trials have revealed several beneficial outcomes of FMD in terms of cancer-related biological markers, including improved insulin sensitivity, reduced blood glucose, insulin, and leptin levels, and lower systemic inflammation [21].

Ongoing research is now focusing on developing short-term, controlled, and adaptable fasting protocols as cost-effective and powerful adjuncts to conventional cancer treatments. Preclinical models suggest that fasting implemented around chemotherapy or radiation sessions may reduce treatment-related side effects while enhancing therapeutic efficacy. Current human trials aim to evaluate safety, tolerability, and biological mechanisms underlying this promising strategy. In underweight or sarcopenic patients, fasting-like regimens should be avoided unless embedded in a supervised protocol with weight and albumin monitoring.

4. Ketogenic Strategies in Oncologic Metabolism (LC/KD)

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A ketogenic diet (KD) is characterized by an extremely low carbohydrate (LC) intake and is not primarily designed around calorie restrictions or fasting protocols. Typically, its macronutrient composition includes approximately 90% of caloric intake from fats, while carbohydrates and proteins account for only about 2% and 8%, respectively. These ratios align with the conventional KD formulations, typically represented by a fat-to-carbohydrate ratio of 4:1 and a fat-to-protein ratio of 3:1 [32].

KD is known to effectively stimulate the production of ketone bodies, such as β-hydroxybutyrate and acetoacetate, which can help suppress appetite and maintain low plasma glucose levels in non-cancer individuals [33]. The consistent high intake of fatty acids combined with minimal carbohydrate consumption drives a metabolic shift from glucose-based to fatty acid-based energy production. This increase in fatty acid oxidation leads to elevated concentrations of ketone bodies in the bloodstream [34].

The rationale behind KD in oncology stems from the Warburg effect—a metabolic hallmark of cancer cells where energy is primarily generated through glycolysis and lactate fermentation, even in the presence of oxygen. Cancer cells often exhibit increased glucose uptake to support their rapid growth and proliferation. Therefore, reducing carbohydrate intake through KD can help lower blood glucose, insulin, and IGF-1 levels. As many cancer cells depend on glucose for ATP production, carbohydrate restriction can starve tumor cells while allowing normal cells to rely on ketones for energy [35].

Among ketone bodies, β-hydroxybutyrate has been shown to possess anti-inflammatory effects. It achieves this by downregulating interleukin-1β (IL-1β) expression in bone marrow-derived macrophages and promoting the expression of mitochondrial uncoupling protein 2, thereby protecting against oxidative stress and supporting increased longevity [36]. Additionally, reduced levels of pro-inflammatory cytokines, such as tumor necrosis factor-α (TNF-α) and interferon-γ (IFN-γ), have been observed with KD, suggesting its potential to enhance cancer treatment outcomes through its anti-inflammatory actions [37].

Evidence also points to KD's role in hindering colon cancer progression by increasing oxidative stress within tumors. This process suppresses matrix metalloproteinase-9 (MMP-9) expression and promotes the conversion of tumor-associated macrophages (TAMs) from the M2 to the M1 phenotype, both of which are associated with anti-tumor activity [38]. MMPs are crucial contributors to tumor progression, metastasis, angiogenesis, and the degradation of the extracellular matrix [39].

The potential of low-carbohydrate diets like KD in cancer management is currently under investigation, particularly because of cancer cells' elevated glucose demands compared to normal cells. While preclinical studies in cellular and animal models have shown promising outcomes for KD in cancer prevention and therapy, clinical data on its safety and effectiveness remain limited and preliminary [40].

4.1. Preclinical Evidence on Ketogenic Diet

Multiple preclinical studies conducted on mouse models have highlighted the antitumor potential of ketogenic diets (KDs) when used as an adjunct to chemotherapy or radiotherapy. These studies have reported significant inhibitory effects against various types of cancer, including colorectal, gastric, prostate, head and neck, brain, and thyroid cancers [40].

In a recent investigation, the effectiveness of KDs in suppressing tumor development was demonstrated in autochthonous animal models of colorectal cancer (CRC). The antineoplastic effects of KDs were primarily attributed to the ketone body β-hydroxybutyrate (BHB), which was shown to inhibit the proliferation of colonic crypt cells and markedly reduce the progression of intestinal tumors. Further analysis suggested that BHB mediates its tumor-suppressive action via activation of the hydroxycarboxylic acid receptor 2 (HCAR2) and the transcriptional regulator homeodomain-only protein homeobox (HOPX), ultimately leading to gene expression changes and suppression of cell growth [41].

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Additionally, both BHB and acetone have been identified as modulators of N-methyl-D-aspartate (NMDA) signaling, a mechanism of physiological relevance given the widespread expression of NMDA receptors across various cancer types. Through these mechanisms, the ketogenic diet influences cellular redox balance by altering the ratio between nicotinamide adenine dinucleotide (NAD^+) and its reduced form (NADH), thereby modulating oxidative phosphorylation (OxPhos) and enhancing the production of reactive oxygen species (ROS) within tumor cells. This redox stress can promote apoptosis and sensitize tumors to cytotoxic therapies, while normal cells are generally protected through improved mitochondrial efficiency and adaptive metabolic responses [42,43].

Collectively, these preclinical findings suggest that ketogenic interventions may exert context-dependent anticancer effects through the regulation of oxidative stress and metabolic signaling pathways involving HCAR2, NMDA, and ROS.

4.2. Clinical and Translational Evidence on Ketogenic Diet

Several clinical trials have examined the effects of the ketogenic diet (KD) when used alongside chemotherapy, radiotherapy, or antiangiogenic therapies in cancer treatment, producing varied outcomes. Evidence from case reports and small-scale trials suggests that KD is generally safe and well-tolerated by cancer patients. For example, a four-week pilot study involving ten individuals with various types of cancer evaluated the safety and feasibility of KD. The findings showed that patients who experienced stable disease or partial remission had, on average, BHB levels three times higher than those whose disease progressed [44].

Similarly, Schmidt et al. conducted a pilot trial with 16 advanced-stage cancer patients, where 7 participants received a KD supplemented with oil-protein shakes. The study reported a statistically significant average weight reduction without significant changes in blood lipid or cholesterol levels. Some improvements in quality of life were also noted, and no serious adverse events occurred [45].

In a retrospective study of 53 patients undergoing treatment for high-grade glioma, six followed a KD composed of 77% fat, 8% carbohydrates, and 15% protein. These patients exhibited a substantial reduction in average blood glucose levels, dropping from 142.5 mg/dl before the diet to 84 mg/dl, compared to 122 mg/dl in patients on a standard diet [46].

Another randomized controlled trial involving breast cancer patients evaluated the effects of a medium-chain triglyceride (MCT)-based KD. Results showed reductions in fasting blood glucose, body weight, BMI, and body fat percentage, alongside increased blood ketone concentrations, indicating positive metabolic shifts in the intervention group [46].

Some studies also reported significant reductions in insulin levels among KD participants and an inverse relationship between BHB concentrations and IGF-1 levels [47]. A growing body of literature on adult cancer patients (aged 18 and over) with various malignancies—including glioblastoma, breast, liver, lung, pancreatic, colorectal, and head and neck cancers—has shown encouraging outcomes. These include improved survival, extended progression-free survival, better responses to conventional therapies, and enhanced quality of life [48].

Despite these promising findings, the limited scope and scale of existing studies underscore the need for more extensive research. Larger, controlled trials are necessary to thoroughly evaluate KD's efficacy in cancer treatment and to explore its potential in combination with other therapeutic strategies. KD feasibility hinges on adherence and safety checks; cap unintended weight loss (>5% over a cycle) and discontinue if fasting glucose persistently <70 mg/dL or BHB >4 mmol/L in symptomatic patients.

5. Vitamin D, Carotenoids and Signaling Axes

Dietary supplements refer to ingestible products, such as vitamins, minerals, herbal extracts, and other compounds, that are taken to enhance overall health and complement the regular diet. These supplements are widely used across the globe and encompass a diverse category of consumables that differ from standard foods and pharmaceutical drugs [49].

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5.1. Vitamins and Signaling

Numerous dietary elements, including specific vitamins, have been shown to interfere with key signaling pathways and molecular mechanisms involved in the progression of cancer. Vitamins such as B and D have been extensively studied over the years for their potential to lower cancer risk and mortality [50]. Researchers have concentrated on the preventive role of vitamin D against various cancer types. In the liver, both vitamin D2 and D3 are converted into the circulating form, 25-hydroxyvitamin D (25(OH)D or calcidiol), which is subsequently transformed into the biologically active form, 1,25-dihydroxyvitamin D ($1,25(OH)_2D$ or calcitriol), within the kidneys [49, 51, 52]. Numerous studies in vitro have demonstrated that vitamin D can inhibit the proliferation and induce apoptosis of various cancer cells, thereby impeding tumor development [53]. Similarly, B vitamins and their derivatives are vital to human physiology and have been recognized as important modulators of cancer risk [54]. Despite consistent observational links, results from high-dose supplementation trials are heterogeneous, supporting a 'target-to-sufficiency' rather than a pharmacologic approach.

5.1.1. Breast Cancer

Vitamin D plays a vital role in regulating the function of multiple organs, including the mammary glands. Its influence on mammary gland development is primarily mediated through the vitamin D receptor (VDR), which facilitates the vitamin's anti-proliferative, pro-apoptotic, and differentiation-inducing effects [55]. The pioneering work of Abe et al. and Colston et al. first identified the anti-cancer properties of vitamin D [56,57]. A range of clinical and observational studies have since supported vitamin D's role in modulating both the initiation and progression of cancer [58–60]. These findings underscore the involvement of VDR and associated proteins such as CYP27B1 and CYP24A1 in the development of breast cancer (BC) [61].

In particular, studies have indicated that individuals with triple-negative breast cancer (TNBC) exhibit the lowest vitamin D levels among all breast cancer subtypes, suggesting a possible protective function of vitamin D against TNBC [61]. Moreover, Bauer et al. conducted a meta-analysis of nine studies involving 5,206 patients and 6,450 controls, revealing a nonlinear inverse relationship between plasma vitamin D levels and breast cancer risk in postmenopausal women [61,62]. Additional in vitro and in vivo research has demonstrated that $1,25(OH)_2D_3$ and its analogues impact key cancer-related processes in breast tissue, including cell proliferation, apoptosis, differentiation, epithelial–mesenchymal transition, autophagy, metabolism, and the population of cancer stem cells [63].

Similarly, in a prostate cancer-related in vitro study comparing the MCF-7 breast cancer cell line with the non-tumorigenic MCF10A line, treatment with thiamine (1–2 mg/mL for 24 hours) significantly reduced proliferation in the MCF-7 cells only. This indicates a potential link between elevated levels of vitamin B12 and holohaptocorrin with increased cancer risk. Furthermore, a nested case–control study found a positive association between higher blood vitamin B12 levels and the overall risk of developing lung cancer [54].

5.1.2. Colorectal Cancer

Vitamin D has been extensively investigated for its potential protective role against colorectal cancer (CRC) [15–20]. Higher intake of vitamin D has been associated with a reduced risk of early-onset CRC and its precancerous lesions. Several mechanisms have been proposed to explain this inverse relationship, including vitamin D's ability to inhibit cell proliferation, migration, invasion, and angiogenesis in colon cancer cells, as well as its regulatory effects on intestinal immune cells [64]. Furthermore, colorectal cancer patients with elevated pre-diagnostic levels of 25-hydroxyvitamin D [25(OH)D] have demonstrated a lower risk of cancer-related mortality [65].

The COLON study found that patients with vitamin D levels exceeding 50 nmol/L, combined with higher magnesium intake (above the median), experienced reduced mortality rates from CRC

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[66, 67]. In a randomized trial led by Ng et al., serum 25(OH)D levels were measured in 515 patients undergoing chemotherapy for stage 4 CRC. The findings revealed that vitamin D deficiency (defined as <20–30 ng/mL) was highly prevalent in this population [66,68]. More recently, a large cohort study by Yuan et al. involving 1,041 patients with previously untreated metastatic or advanced CRC confirmed a widespread deficiency in vitamin D (less than 20–30 ng/mL) among this group [66, 69].

5.1.3. Lung Cancer

Over the past few decades, abnormalities in vitamin D levels or disruptions in vitamin D receptor (VDR) function have been linked to the development of various cancers, including lung cancer [53]. Epidemiological studies have highlighted the protective role of adequate vitamin D levels in lung cancer prevention. Several investigations have identified a correlation between serum vitamin D levels and the risk of developing lung cancer [53].

One study demonstrated that higher circulating vitamin D concentrations are associated with lower mortality rates and improved survival outcomes in lung cancer patients [70]. Additionally, a cohort study in Finland observed an inverse association between vitamin D levels and lung cancer incidence, particularly among women and younger individuals [53, 71].

Genetic variations in vitamin D signaling pathways may also influence lung cancer susceptibility and the effectiveness of therapeutic responses [53, 72]. Vitamin D has been shown to upregulate the expression of p53, a well-known tumor suppressor gene, while downregulating the expression of the pro-apoptotic gene Bcl-2 in A549 human lung carcinoma cells [53, 72].

Furthermore, vitamin D therapy has been reported to enhance the VDR-mediated suppression of the epidermal growth factor receptor (EGFR) gene. Studies have found that vitamin D significantly influences EGFR signaling in lung cancer cell lines, suggesting that vitamin D/VDR-based therapies could be a promising strategy for targeting lung cancer through EGFR modulation [53, 73, 74]. In addition, a nested case–control (NCC) study found a positive association between elevated blood levels of vitamin B12 and an increased overall risk of lung cancer [54].

5.1.4. Ovarian Cancer

Numerous studies examining sun exposure have identified an inverse relationship between vitamin D levels and the risk of ovarian cancer. Experimental research using cell lines, human xenograft models, and mouse models has provided compelling evidence that vitamin D contributes to reduced cell proliferation, increased apoptosis, and inhibited tumor progression in ovarian cancer. Additionally, Mendelian randomization studies have shown that genetically lower levels of 25-hydroxyvitamin D [25(OH)D] are significantly associated with an elevated risk of ovarian cancer, thereby reinforcing the potential protective role of vitamin D [51,75].

An Australian cohort study found that higher serum levels of 25D3 at diagnosis were associated with longer survival among women with invasive ovarian cancer [76]. Similarly, Mendelian randomization research conducted in European populations reported that genetically decreased 25D3 levels were inversely associated with ovarian cancer risk [77]. Evidence from genome-wide association studies (GWAS) also supports a protective role, suggesting that elevated 25D3 concentrations may lower the risk of epithelial ovarian cancer (EOC) [65,78].

In a case–control study involving 1,631 women with EOC, higher 25(OH)D levels were linked to improved survival outcomes [79]. Additional studies have explored variants in the vitamin D receptor (VDR) gene, which modulates the activity of 1,25-dihydroxyvitamin D, and have suggested that these genetic differences may influence both the risk of developing ovarian cancer and survival rates among patients [80–84].

To deepen our understanding of vitamin D's anti-cancer effects and to tailor VDR agonist therapies to individual patients, further research is needed. This should include integrated "omics" approaches—such as epigenomics, metabolomics, genomics, proteomics, and transcriptomics—to better predict individual sensitivities or susceptibilities to vitamin D-based interventions [50, 85].

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5.2. Carotenoids and Retinoid-Related Pathways

Carotenoids, which are antioxidant compounds found in fruits and vegetables, possess immuno-regulatory and anti-mutagenic properties. Numerous studies have reported associations between the intake of various carotenoids and a reduced risk of multiple cancer types. While these associations do not confirm direct causality, they highlight important patterns that suggest a protective effect against cancer development. Beyond their pigment-related aesthetic roles, carotenoids are recognized for their potent antioxidant functions and their role in the biosynthesis of essential nutrients such as vitamin A [86, 87].

Due to their antioxidant properties, specific carotenoids help mitigate the damaging effects of reactive oxygen species (ROS), which are implicated in the onset of various diseases including cancer, cardiovascular and neurodegenerative disorders, and aging. A broad range of scientific studies have also documented the role of carotenoids in regulating apoptosis, gene expression, angiogenesis, and immune function [87, 88]. Furthermore, dietary intake of carotenoids is believed to influence cellular pathways associated with antioxidant defense mechanisms [89].

5.2.1. Head and Neck Cancer

Head and neck cancer (HNC), which encompasses oral cavities, laryngeal, and pharyngeal cancers, has been linked to carotenoid intake due to compounds' antioxidant, anti-mutagenic, and immune-modulating effects. Although the exact biological mechanisms by which carotenoids may influence HNC risk remain to be fully elucidated, numerous studies have highlighted a potential protective role [88].

Two meta-analyses conducted by Leoncini et al. examined the association between dietary carotenoid intake and HNC risk [86, 90]. These analyses reviewed data from sixteen epidemiological studies and found that β-carotene consumption was associated with a significant reduction in HNC risk, including a 46% lower risk of oropharyngeal cancer (OPC) and a 57% lower risk of laryngeal cancer (LC). Similarly, β-cryptoxanthin and lycopene were associated with a 59% and 50% decrease in LC risk, respectively. Moreover, α-carotene, β-cryptoxanthin, and lycopene intake correlated with a 26% reduced risk of oral cavity (OCC) and pharyngeal (PC) cancers [86, 88].

In another pooled analysis of ten case–control studies conducted across North America, Japan, and Europe, involving 18,207 patients and 12,248 controls, researchers reported that higher overall carotenoid consumption was associated with a 39% reduction in the risk of OCC and PC, as well as a 39% lower risk of LC [90].

5.2.2. Breast Cancer

Research indicates that carotenoids may play a significant protective role against prostate and breast cancers. In a recent in vitro study, treatment with 1 μM of β-carotene was shown to suppress the expression of anti-apoptotic proteins such as PARP and Bcl-2. Similarly, studies using the MCF-7 breast cancer cell line demonstrated that lycopene concentrations of 2 μM or higher reduced cell proliferation and enhanced apoptosis, accompanied by increased mRNA expression of p53 and Bax—two key regulators of cell death [91, 92].

A meta-analysis conducted by Fulan Hu and colleagues examined the relationship between carotenoid intake and breast cancer risk, including dose–response patterns [93]. Their findings indicated that higher dietary intake of α-carotene was significantly associated with a reduced risk of breast cancer, though the associations involving dietary and total β-carotene intake remain inconclusive and require further exploration [93].

Another large case–control study involving 5,707 women with invasive breast cancer and 6,389 controls—comprising both pre- and postmenopausal women from Wisconsin, Massachusetts, and New Hampshire—investigated the links between vegetable, fruit, vitamin, and carotenoid consumption and breast cancer risk. The study found that high carotenoid intake was associated with a decreased breast cancer risk in premenopausal women, while no such protective effect was

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observed in postmenopausal participants, particularly among smokers [94]. Overall, these findings support the potential of carotenoids to inhibit breast cancer cell growth [91,92].

5.2.3. Colorectal Cancer

Numerous epidemiological and clinical studies have linked the risk of colorectal cancer (CRC) to both dietary and serum levels of carotenoids and retinol. In a cell culture study, lycopene at concentrations of 2 μM or higher significantly inhibited the proliferation of HT-29 human colorectal cancer cells. This antiproliferative effect is thought to involve suppression of the PI3K/Akt signaling pathway [95]. Additionally, lycopene has been shown to increase the expression of p21 and PCNA, while reducing levels of pro-inflammatory markers such as PGE2, COX-2, and phosphorylated ERK1/2 in animal models. Plasma concentrations of matrix metalloproteinase-9 (MMP-9) have also been inversely correlated with dietary lycopene intake [96].

A meta-analysis conducted by Xiaoyong Han and colleagues reviewed 22 studies examining the relationship between colorectal cancer risk and serum or dietary levels of carotenoids and retinol [97]. Their findings confirmed that higher levels of β-cryptoxanthin, lycopene, α-carotene, and β-carotene were significantly associated with a reduced risk of CRC. These results support the potential role of lycopene and other carotenoids in inhibiting tumor development and progression in colorectal cancer [96].

5.2.4. Lung Cancer

Carotenoids help protect the lungs from early stages of cancer development through their antioxidant properties, which are obtained at varying dietary concentrations. Multiple studies have reported a linear dose–response relationship between β-carotene intake and lung cancer risk, indicating a 2% reduction in risk for every additional milligram of β-carotene consumed daily. Likewise, a 1% decrease in lung cancer risk has been observed for every additional 10 μg of β-cryptoxanthin intake per day, and a 3% reduction for each extra milligram of lycopene consumed daily [98].

A recent case–control study conducted in Hawaii also reported an inverse association between lung cancer risk and the consumption of vegetables rich in carotenoids, suggesting a potential protective role of carotenoids against lung cancer development [99]. Furthermore, a random-effects meta-analysis by Gallicchio and colleagues—encompassing six clinical trials and 25 observational studies—found an overall inverse association between carotenoid intake and lung cancer risk. However, the observed reduction in risk was generally modest and did not reach statistical significance [98]. Beta-carotene supplementation in smokers has shown harm in legacy trials, underscoring that food-based carotenoids should not be conflated with high-dose single-nutrient pills.

5.2.5. Prostate Cancer

Due to their antioxidant properties, carotenoids may help reduce tumor-promoting inflammation. Among them, α-carotene, β-carotene, and lycopene have been extensively studied in relation to prostate cancer. Over seventy studies have investigated their associations, consistently finding that higher dietary intake and blood concentrations of lycopene are linked to a decreased risk of developing prostate cancer. Supporting this, two cell culture studies have shown that lycopene treatment significantly reduces cell proliferation and impairs cell cycle progression in prostate cancer cells [91, 100].

Additionally, a nested case–control study examined the relationship between prostate cancer risk and plasma levels of carotenoids such as cis-lycopene, trans-lutein, β-trans-carotene, and dihydrolycopene. The findings indicated that, particularly among African American men, trans β-carotene isomers were positively associated with increased prostate cancer risk, regardless of smoking habits or multivitamin use [101].

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Furthermore, a separate case–control study involving 65 prostate cancer patients and 132 control subjects assessed the influence of plasma levels of lycopene and other carotenoids and vitamins, including retinol and α- and γ-tocopherols. The study revealed a significant inverse association between prostate cancer and plasma levels of lycopene and zeaxanthin, while β-cryptoxanthin and lutein showed borderline associations with reduced risk [102].

6. Physical Activity and Sedentary Behavior

At present, smoking remains the leading modifiable risk factor for cancer. However, physical inactivity is rapidly emerging as the second-most significant preventable contributor to cancer development [103]. According to the World Health Organization (WHO), more than 1.4 billion adults—over a quarter of the global population—do not engage in sufficient physical activity [104]. With obesity now affecting individuals at younger ages, people are increasingly exposed to related health risks throughout their lives. High-income countries report rates of physical inactivity that are approximately twice as high as those in low-income nations. The global trend toward reduced physical activity has been steadily rising, posing negative consequences for environmental sustainability, economic productivity, quality of life, and the overall health of communities [105, 106].

Regular engagement in physical activity is widely recognized as a key preventive measure against non-communicable diseases such as cardiovascular disease, hypertension, type 2 diabetes, stroke, and numerous types of cancer [6, 107–114]. For decades, epidemiological research has consistently linked higher levels of physical activity with a lower risk of several common cancers. In 2012, it was estimated that the elevated body mass index (BMI) was responsible for 3.6% of all new cancer cases globally [115].

6.1. Prevention: Physical Activity and Cancer Risk

Any bodily movement driven by skeletal muscles that leads to energy expenditure qualifies as physical activity. While exercise is a subset of physical activity, it encompasses a wide range of intensities and forms, including occupational tasks, household chores, commuting, and recreational pursuits like walking, cycling, sports, and active play [116]. Engaging in physical activity positively influences the body's metabolic processes, making it more resistant to various types of cancer. The nature and intensity of the activity can impact metabolism and energy balance, thereby influencing how much food and nutrients can be consumed without leading to fat accumulation. When caloric intake exceeds energy expenditure, it results in a positive energy balance, promoting weight gain and increased body fatness [116].

Although physical activity is often emphasized for weight management, it also offers direct cancer-protective benefits through several biological mechanisms. These include modulation of the immune system [117–119], reduction in circulating estrogen [120], decreased oxidative stress [121], improved insulin sensitivity [122], suppression of inflammation [123], favorable epigenetic changes [124], reduced telomere shortening [125], and enhanced DNA repair [118]—all of which are associated with the initiation and progression of cancer.

Independent of its effects on obesity, physical activity has been inversely associated with colon and breast cancer, as demonstrated in a 2020 Mendelian randomization study using UK Biobank data. This inverse relationship was found to be independent of BMI [126].

Moreover, strength training, which builds skeletal muscle mass, is linked to lower risks of kidney, bladder, and colorectal cancers [127, 128]. Aerobic exercise also improves glucose metabolism and insulin sensitivity, potentially reducing the risk of cancers associated with insulin resistance, such as colon, liver, pancreatic, and endometrial cancers [129, 130]. While not all cancers are equally affected by physical activity, lifestyle interventions, including regular exercise, have shown potential to lower overall cancer risk [131]. For example, intense domestic activities like heavy housework may offer protective benefits against breast cancer. Exercising for three to five hours per week has been associated with a reduced risk of developing cancer [132].

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One study found that individuals who exercised had a 15–20% lower risk of breast cancer and a 24% reduced risk of colorectal cancer [133]. Exercise has been shown to reduce the risk of breast cancer in postmenopausal women and may help protect them from autonomic nervous system dysfunction [134, 135]. Among 126 studies on exercise and colorectal cancer (CRC), more active participants had a 19% lower risk of CRC [136]. Similarly, a meta-analysis on stomach cancer, encompassing 10 cohort and 12 case–control studies, reported a 19% risk reduction among those who exercised more frequently [137].

Given that obesity is a known risk factor for endometrial cancer, exercise-induced weight loss plays a protective role. According to a review of 33 studies, women who exercised regularly had a 20% lower risk of endometrial cancer [138]. A similar review on esophageal cancer found that those with the highest levels of physical activity had a 21% lower risk compared to the least active individuals [139]. In a large-scale study involving over 1 million participants, leisure-time exercise was associated with a 13% lower risk of bladder cancer and a 23% lower risk of kidney cancer [140]. A review of 25 studies found that while physical activity had little effect on lung cancer risk in non-smokers, it did lower the risk among smokers [141].

6.2. Physical Activity During and After Therapy

Beyond its role in cancer prevention, exercise has been shown to reduce the adverse effects associated with cancer treatments. Therapies such as chemotherapy and targeted treatments often produce side effects including fatigue, cognitive decline, depression, muscle and bone loss, and cardiac toxicity. These side effects can negatively impact treatment outcomes and overall quality of life [131]. In some cases, patients are unable to complete or tolerate therapy due to the severity of these side effects, potentially altering the progression of the disease. However, substantial evidence indicates that engaging in physical activity can help alleviate many of these treatment-related complications.

Exercise training influences both cancer-specific and physiological as well as psychological outcomes associated with treatment [107–111]. Not only can exercise reduce the side effects of therapy, but it may also enhance treatment efficacy, making it a valuable addition to the therapeutic process [142]. While some healthcare professionals express concern about whether physical activity could influence survival or prognosis, current research suggests that exercise may not significantly affect survival rates for cancers such as gastric cancer, non-Hodgkin lymphoma, and several others [143].

Exercise can also be leveraged as a form of cancer-suppressive therapy by disrupting cancer metabolism, particularly anaerobic glycolysis. The impact of physical activity on tumor metabolism depends on its frequency, duration, intensity, and type. By decreasing vasoconstriction in hypoxic environments, exercise improves tumor perfusion and reduces hypoxia, making the cancer microenvironment less aggressive and more susceptible to treatment [144, 145].

Additionally, regular physical activity helps counteract immunosenescence, the aging of the immune system, which is marked by decreased natural killer (NK) cell activity, chronic inflammation, impaired antigen presentation by dendritic and monocyte cells, and a reduction in naïve T cells capable of targeting emerging cancer cells. These changes contribute to the heightened cancer risk in older adults [146]. Exercise has been shown to slow immune aging by enhancing immune cell function [124], thereby playing a protective role against immune-related malignancies.

In summary, robust evidence supports that physical activity reduces cancer risk, curbs tumor growth and progression, mitigates the side effects of cancer treatments, improves treatment adherence, and enhances patient quality of life. A pragmatic mix of aerobic plus resistance exercise counters treatment-related sarcopenia and supports dose intensity, even when total weekly volume is modest.

7. Adiposity and Metabolic Health

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A body with overweight or obesity (defined as excessive fat accumulation) contains a higher proportion of fat compared to lean tissues such as muscle and bone. Carrying excess weight at unhealthy levels increases the risk of developing certain types of cancer and raises the likelihood of recurrence after treatment. According to the World Health Organization, in 2016, over 340 million children and adolescents and 1.9 billion adults were living with overweight or obesity, a number projected to rise further [147]. The global cancer burden is expected to continue growing due to ongoing trends of reduced physical activity and increased body fat, particularly in the context of an aging global population. If these trends persist, overweight and obesity may soon surpass smoking as the leading preventable cause of cancer.

7.1. Obesity and Cancer: Epidemiology and Mechanisms

Since the early 20th century, the relationship between body fat and cancer has been explored in scientific literature, with more focused discussions emerging since 1987 [148]. As the prevalence of overweight and obesity continues to rise, so does the incidence of cancers associated with excess body fat. This trend is expected to significantly increase the cost of future cancer treatments and place a growing financial burden on healthcare systems due to the long-term management of related comorbidities.

Maintaining a healthy weight is one of the most effective strategies for cancer prevention. Importantly, excess body fat during childhood has been linked to an increased risk of developing various diseases, including cancer, later in life. Hidayat et al. identified associations between early-life adiposity and the development of eight different types of cancer in adulthood [149]. Moreover, weight gain in adulthood is associated with a higher risk of overweight, obesity, and postmenopausal breast cancer, with a 6% increased risk for every 5 kg gained [150]. Despite growing evidence of the link between obesity and cancer in adults, the impact of weight gain throughout adulthood is often underestimated.

Body fatness contributes to cancer risk through several biological mechanisms. Obesity is associated with elevated fasting insulin levels and increased inflammatory mediators, both of which promote cell proliferation (Hallmark: sustained proliferative signaling) [151]. Additionally, obesity can impair apoptosis, the process by which damaged cells are eliminated, thereby allowing abnormal cells to survive (Hallmark: resisting cell death) [152]. Cancer cells can also spread through the bloodstream or lymphatic system (Hallmark: activating invasion and metastasis). These endocrine and metabolic disruptions, particularly involving insulin and inflammation, highlight the complex relationship between obesity and cancer development.

7.2. Clinical weight-loss strategies in oncology

Combined dietary energy restriction and progressive exercise represent first-line, scalable interventions to reduce adiposity, improve insulin resistance, and lower low-grade inflammation, mechanisms tightly linked to carcinogenesis and poorer outcomes [6, 122, 123]. Structured programs should favor protein-adequate, minimally processed diets and supervised training tailored to treatment phase and fatigue [107].

7.3. Survivorship and relapse risk

Post-treatment weight gain is common and associated with higher recurrence and mortality in several cancers [131, 150]. Survivorship care plans should embed long-term weight management with behavioral support, periodic monitoring, and referral pathways to exercise oncology services [131].

8. Carcinogenic Exposures: Tobacco and Ultraviolet (UV) Radiation

8.1. Tobacco as a Carcinogen

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Tobacco and tobacco smoke contain a complex mixture of over 9,500 chemical substances, many of which have been identified by regulatory authorities as harmful to human health. In 2012, the U.S. Food and Drug Administration (FDA) published a list of hazardous or potentially hazardous constituents found in both tobacco smoke and unburned tobacco, identifying 79 of them as carcinogenic [153]. Tobacco smoke increases cancer risk primarily due to its content of carcinogens such as nitrosamines, acrylamides, polycyclic aromatic hydrocarbons (PAHs), cadmium, and volatile organic compounds (VOCs) [154, 155].

There is a well-established causal link between smoking and lung cancer risk [154]. Extensive experimental and epidemiological evidence confirms that smoking is a major risk factor for several cancers, including those of the kidney, bladder, gastrointestinal tract, head and neck, colorectum, esophagus, pancreas, stomach, liver, cervix, and myeloid leukemia [153, 154, 156–158].

Animal studies have shown that several carcinogens in tobacco smoke can induce mammary tumors, and human research has demonstrated that various tobacco-derived compounds can reach breast tissue. Furthermore, the absorption and metabolic activation of carcinogens implicated in breast cancer—such as 4-aminobiphenyl and PAHs—are significantly higher in smokers than in non-smokers [157].

Head and neck squamous cell carcinoma (HNSCC) is a cancer type strongly associated with tobacco use. The risk of developing HNSCC is nearly ten times higher in smokers compared to non-smokers, and approximately 70–80% of newly diagnosed cases are linked to the combined use of tobacco and alcohol [158].

The carcinogenic effects of tobacco smoke are largely mediated by reactive oxygen species (ROS) and reactive nitrogen species (RNS), which damage key macromolecules such as DNA, lipids, and proteins. Scientific evidence indicates that smoking-induced oxidative stress plays a central role in both cancer development and inflammation. This oxidative stress triggers inflammatory responses, which in turn generate additional ROS, perpetuating a cycle of molecular damage that may initiate carcinogenesis [159].

8.2. Non-Ionizing Radiation as a Carcinogen

Human exposure to radiofrequency electromagnetic fields (RF-EMF) primarily arises from the use of personal electronic devices—such as mobile phones, Bluetooth headsets, cordless phones, and amateur radios—as well as from occupational and environmental sources. Occupational exposure includes equipment like induction heaters, high-frequency dielectric devices, and high-powered pulsed radars, while environmental sources involve medical technologies, broadcast antennas, and mobile phone base stations. Among the general population, the highest levels of exposure typically come from handheld devices used in close proximity to the body, particularly mobile phones. Additionally, numerous occupational groups are regularly exposed to RF radiation, including radar operators, military and security personnel using walkie-talkies, technicians servicing broadcast antennas, plastic welders, workers involved in RF-based testing or drying processes, and physiotherapists using diathermy equipment [160].

Stang et al. conducted both hospital- and population-based case–control studies investigating the development of uveal melanoma in relation to occupational RF radiation exposure and reported an increased risk among individuals exposed to radiofrequency-emitting devices, such as radios and mobile phones [161]. Similarly, Karipidis et al. carried out a case–control study across five major population centers in Victoria, Australia, identifying a potential link between occupational RF exposure and glioma risk [162].

Ultraviolet (UV) radiation, a non-visible component of the electromagnetic spectrum with shorter wavelengths than visible light, is also recognized as carcinogenic. While sunlight is the primary source of UV radiation, certain industrial activities—such as electric arc welding—can also emit significant UV levels. The International Commission on Non-Ionizing Radiation Protection (ICNIRP) has established a safety threshold for artificial UV exposure to the eyes and unprotected skin, recommending a limit of 30 J/m² over an 8-hour period [163]. UV radiation is a well-known risk

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factor for basal cell carcinoma (BCC), and prolonged occupational exposure to sunlight is believed to increase the risk of developing this form of skin cancer. A total of 19 case–control and five cohort studies have reported a significant association between occupational UV exposure and BCC incidence [164].

From a public-health perspective, UV avoidance and photoprotection remain the most actionable radiation-related strategies for cancer risk reduction in the general population.

9. Complementary and Mind–Body Approaches

The term complementary and alternative medicine (CAM) refers to a wide array of medical and health-related practices and products that fall outside the scope of conventional medicine. Among individuals with cancer, CAM is often used alongside standard treatments to help alleviate stress and anxiety related to the disease and its therapies, as well as to manage side effects such as nausea, pain, and fatigue. Common CAM approaches in oncology include herbal medicine, acupuncture, mind–body practices—such as meditation, yoga, and Tai Chi—nutritional supplements, energy-based therapies, and massage [165–168].

The use of CAM among cancer patients has been steadily increasing. It is estimated that between 33% and 47% of patients worldwide incorporate complementary, alternative, or integrative therapies into their care [169]. One of the most burdensome symptoms experienced by cancer patients is cancer-related fatigue (CRF), which significantly impairs quality of life by affecting daily functioning, social engagement, work performance, leisure activities, and interpersonal relationships. CRF can also hinder adherence to prescribed cancer treatments.

As a result, many patients turn to CAM in an effort to regain a sense of well-being and better manage their symptoms [170, 171]. While some of these therapies may offer symptom relief and contribute to improved quality of life, their therapeutic efficacy remains variable. Further research is needed to determine how best to integrate these modalities into evidence-based cancer care. Given heterogeneity in quality and potential drug–herb interactions, CAM use should be discussed with the oncology team and, where possible, evaluated within clinical protocols.

10. Limitations

Since this is a narrative review, a systematic study selection protocol was not followed (no protocol registration, no predefined eligibility with dual independent screening, and no formal risk-of-bias assessment). This may introduce a selection bias, as the inclusion of articles was partially based on the authors' criteria. However, this approach allowed for a deeper and more critical discussion of the available evidence, facilitating the formulation of practical recommendations based on the most relevant literature. Nonetheless, the non-systematic search strategy may have led to the omission of relevant studies, particularly those not indexed in the selected databases and to susceptibility to publication bias. The English-language focus may also have excluded pertinent non-English studies, and heterogeneity across study designs and populations limits generalizability. Some emerging or less-studied interventions may have been underrepresented due to the broad scope of the review. Accordingly, the findings should be interpreted as hypothesis-generating rather than causal. Future systematic reviews and meta-analyses are warranted to validate and expand upon the insights presented here.

11. Conclusions and Future Perspectives

Modifiable lifestyle factors such as physical activity, body weight regulation, dietary habits, tobacco and alcohol use, and exposure to environmental carcinogens play a decisive role in both the prevention and management of cancer. A growing body of epidemiological and mechanistic evidence confirms that adopting healthy behaviors significantly reduces cancer incidence, improves treatment tolerability, and enhances long-term survivorship outcomes. In particular, regular physical activity

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emerges as a key modulator of cancer risk and prognosis by influencing multiple biological pathways, including hormonal regulation, immune function, oxidative stress, and inflammation.

Excess body fat, increasingly prevalent across all age groups, is strongly associated with higher cancer risk and poorer post-diagnosis outcomes. The obesity epidemic, driven by sedentary lifestyles and high-calorie diets, poses a major public health challenge that may soon surpass tobacco as the leading preventable cause of cancer. Weight management through a combination of physical activity and dietary intervention should therefore be prioritized in cancer control strategies.

While the harmful effects of tobacco and radiation exposure are well established, the ongoing dissemination of new technologies and changing occupational environments calls for continued surveillance and updated safety standards. Population-wide interventions, such as taxation policies on tobacco and ultra-processed foods, stricter workplace protections, and educational campaigns, remain essential to mitigate carcinogenic exposures and promote sustainable prevention.

In addition to conventional medical approaches, the integration of evidence-based complementary and alternative medicine (CAM) into oncology care is gaining traction. CAM interventions—such as mind–body therapies, acupuncture, and herbal medicine—may help manage symptoms like fatigue and anxiety, although their effectiveness remains variable and often underexplored. Rigorous clinical and translational studies are still needed to establish safety profiles, mechanisms of action, and best practices for integrating CAM into evidence-based oncology.

From a clinical and public health standpoint, lifestyle interventions remain the most accessible, cost-effective, and impactful tools for cancer prevention and control. Future research should refine individualized prevention strategies, define dose–response relationships for diet and physical activity, and explore the interplay between lifestyle and genetic predisposition. Moreover, creating supportive environments through urban planning that promotes active living and school- or community-based health education will be critical to sustaining behavioral change across the lifespan.

Ultimately, lifestyle medicine should be regarded not as an adjunct but as a core pillar of modern oncology, integrating nutrition, exercise, and behavioral interventions within standard care pathways to improve outcomes and quality of life. Implementation priorities include embedding exercise oncology and nutrition services in cancer centers, developing pragmatic home- and community-based programs, and ensuring equitable access across healthcare systems.

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