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REVIEW ARTICLE

Diet, weight status, and physical activity in cancer prevention

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Abstract

Background: Cancer is a multifactorial disease involving individual, behavioral and environmental factors that can contribute to its onset. More than 40% of cancers are estimated to be attributable to avoidable risk factors, particularly diet, physical activity, weight status, and alcohol consumption. **Aim:** To provide insight into the role of weight status, diet, and physical activity in cancer causation and prevention, and to highlight evidence-based recommendations. **Material and methods:** A literature review of published studies, particularly recent systematic reviews, meta-analyses, and large prospective studies was conducted using PubMed/Medline, ScienceDirect, and Google Scholar databases. **Results:** There is evidence that diets high in red and processed meats, fat and refined carbohydrates, and low in plant foods such as vegetables, fruits, and whole grains, high consumption of alcohol, overweight/obesity, and physical inactivity are associated with increased risk of tumor development and progression. Therefore, the current recommendations for cancer prevention are based on: (1) eating at least five portions (400g) of vegetables (3 portions) and fruits (2 portions); (2) limiting the consumption of alcohol and unhealthy foods such as red and processed meats, energy-dense foods, and trans-fatty acids; (3) maintaining a healthy weight status; and (4) being at least moderately physically active. **Conclusion:** While a balanced diet, as defined by the recommendations, can help reduce the risk of certain cancers, no single food can prevent the development of this pathology. The term "anticancer" associated with diet is therefore misleading. Several nutritional factors are associated with an increase or a decrease in the risk of certain cancers. Adopting a healthy and plant-based diets, avoiding overweight/obesity, and being physically active are considered as cornerstones of preventive strategies against cancer. Further studies are needed to elucidate the associations between dietary and lifestyle patterns and cancer.

Keywords: Cancer, Diet, Weight status, Overweight, Obesity, Physical activity.

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1 Introduction

Cancer is one of the major non-communicable diseases worldwide. In 2018, there were an estimated 18 million cancer cases and 9.6 million cancer-related deaths [1]. The most common cancers are lung and breast cancers (each represents 12.3% of the total number of newly diagnosed cases), followed by colorectal and prostate cancers (10.6% and 7.5%, respectively) [2]. The lifetime risk for developing cancer is one in five for men and one in six for women, and approximately one in eight men and one in eleven women die from this disease which remains the second leading cause of global deaths [1].

Data from the first cancer incidence registers in populations from several countries [3] indicated that in the 1960s, colon and breast cancers were 5 to 10 times more frequent in the United States and Western Europe than in Japan and Southeast Asia. In contrast, stomach cancer was 15 times less common in the United States than in Japan or China. These differences cannot be explained exclusively by the carcinogenic factors known or suspected at the time (tobacco, alcohol, radiation, chemical, and physical carcinogens); the role of other factors related to lifestyle and diet has been hypothesized [4]. Moreover, a systematic review and meta-analysis study reported an increased risk of gastric cancer among immigrant populations from two high-incidence regions (the former Soviet Union and Japan) to low-incidence regions (including Western Europe, Australia, Brazil, Canada, Israel, and the United States) and suggested that an assessment of immigrant generation along with other risk factors might help identify high-risk populations for prevention and therapeutic interventions [5].

Since the beginning of the 1970s, numerous fundamental, clinical and epidemiological research studies have sought to identify and clarify the role of certain nutritional factors that may be involved as risk or protective factors in the development of cancers. The collective expertise of this work has made it possible to highlight the relationships between nutrition and cancer with varying degrees of certainty.

Current evidence shows that of all cancer-related deaths about 25-30% are caused by tobacco use, 30-35% are attributed to unhealthy diet, 15-20% are related to carcinogenic infections including Helicobacter pylori, Human papillomavirus, Hepatitis B virus, Hepatitis C virus, and Epstein-Barr virus, and the remaining 15-30% are due to other factors such as radiation, lifestyle habits, stress, and environmental pollutants [6,7]. The genetic predisposition may also contribute to the onset of cancer, but the majority (90-95%) of human cancers is attributed to exogenous factors [7].

The economic cost of cancer poses a significant burden to patients, families, communities, and governments throughout

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the world, particularly in low- and middle-income countries. This is due to population aging and globalization of economies as well as increasing adoption of cancer-causing behaviors such as cigarette smoking, having a sedentary lifestyle, and eating unhealthy foods [8]. Thus, cancer prevention is currently one of the major public health issues at national and global levels.

According to the World Health Organization (WHO), between 30-50% of all cancer cases could be prevented through implementing existing evidence-based practices in cancer prevention, and by avoiding behavioral and dietary risk factors such as low fruit and vegetable intake, being overweight or obese, and insufficient physical activity [1,8,9].

In this paper, we provide an overview of the associations between cancer and diet, weight status, and physical activity synopsizing the main mechanisms underlying these associations as well as highlighting evidence-based recommendations to prevent tumor development and progression. Such information may help to raise awareness among regulators, physicians, patients, and caregivers, and may also inform the development of cancer prevention and control strategies.

2 Material and Methods

We conducted a literature review of published studies, particularly recent systematic reviews, meta-analyses, and large prospective studies, in peer-reviewed journals available in PubMed, ScienceDirect, and Google Scholar databases. These databases were searched between 16 March and 31 August 2020. The key search terms "cancer", "tumor" and "cancer types" were paired with "nutrition OR diet OR diet components OR food OR bioactive compounds OR red and processed meats OR carbohydrates OR alcohol OR alcoholic beverages OR fat OR fatty acids OR physical activity OR lifestyle OR body mass index (BMI) OR overweight OR obesity OR prevention OR causation OR development OR progression OR risk factors OR recommendations". Only human studies in English language and articles that had full text available were included. All selected full-text articles were examined for citations of relevant studies.

3 Results

3.1 Relationship between diet and cancer

Diet is a double-edged sword involved in both causation and prevention of human cancers [8]. On one hand, there are unhealthy diets, that are rich in red and processed meats, refined grains, potato chips, sugary beverages, sweets, and high-fat dairy[10-13], and drinker pattern (diet with a high content of wines, beers, and liquors) [14], which may be related to an increased risk for cancer. On the other hand, there are healthy diets, characterized by high intakes of vegetables, fruit, fish, poultry, whole grains, and low-fat dairy, which are associated with a reduced risk of cancer [10-12]. For instance, systematic reviews have shown that the Western diet, defined as high dietary intake of saturated fats, sucrose, red meat, processed meat, pre-packaged foods, and low intake of fiber, represents a growing risk of various cancers including breast and colorectal cancer [10,15,16]. Conversely, adherence to the Mediterranean diet, which is characterized by high consumption of fruits, vegetables, and fish, and low consumption of red meat, was found to be associated with a reduced risk of several types of cancer including breast, stomach, liver, and colorectal cancers [15-18].

3.1.1 Diet components which probably reduce cancer risk

Evidence-based recommendations released by various international organizations suggest that intake of primarily plant-based foods such as vegetables, fruits, pulses, and whole grains plays an important role in cancer prevention [1,13]. Several meta-analyses have shown significant associations between higher vegetable consumption and reduced risk of many cancers, including oral cavity, esophagus, pharynx, and larynx cancers [13], as well as breast [10], ovarian [19], colorectal [20], pancreatic [21], and liver cancers [22]. For instance, cruciferous vegetables such as broccoli, cauliflower, and Brussels sprouts [23], vegetables rich in carotenoids such as carrots, sweet potatoes, and green leafy vegetables [24,25], onions, and garlic [26] are well known for their anti-cancer effects.

Increased fruit intake has also been shown in meta-analyses to be associated with a lower risk of colorectal [20], stomach, esophageal, lung, [13], breast [10], pancreatic cancers [21]. For example, a systematic review conducted by Aune et al. revealed inverse associations between the intake of citrus fruits, dried fruits, tinned fruits, and fruit juice, and cancer risk [27].

In addition, several studies have observed that increased consumption of dietary whole grains (such as brown rice, quinoa, oats, wheat, and rye) has protective effects against colorectal, pancreatic, and gastric cancers [9,13].

Plant-based foods are high in dietary fibers, vitamins, minerals, and other phytochemicals, including carotenoids, phenolics, and flavonoids that may help to prevent cancer [28]. It has been demonstrated that the consumption of sufficient amounts of plant-derived bioactive compounds has carcinogenesis-preventive effects [29]. Potential mechanisms explaining these associations include antioxidant effects, increased detoxification enzyme activity, favorable action of micronutrients, prevention of nutrient deficiencies, and stimulation of the immune system[13,30]. These natural bioactive compounds are also well known for their anti-cancer effects due to their ability to scavenge free radicals, their anti-mutagenic and anti-proliferative properties as well as their beneficial role in modulating hormonal concentration and hormonal metabolism [31].

Furthermore, fibers of non-starchy vegetables and fruit were found to be associated with increased stool bulk which may reduce the intestinal transit time of foods and therefore lower the absorption of carcinogens in the colon [32]. The inverse association between cancer risk and diets rich in whole grains and dietary fibers may be mediated by intestinal microbiota as well [11]. Additionally, the health-promoting effects of fruits, vegetables, whole grains, and other plant-derived foods may be attributable to synergic interactions between bioactive compounds and other nutrients in the whole diet [29].

According to the WHO, of the global burden of disease

attributable to insufficient fruit and vegetable intake, about 15% was from cancers [33]. It has been suggested that the worldwide estimates of the preventable fraction of cancer cases due to low fruit and vegetable consumption can range from 5-12% up to 20-30% for upper gastrointestinal tract cancers [34]. For this reason, it is recommended to consume at least five portions or servings (at least 400 g) of a variety of non-starchy vegetables and fruits every day, and at least 30 g per day of fibers from food sources [13]. The Finnish experience shows the feasibility and effectiveness of cancer prevention programs targeting certain risk factors carried out at the population level. This efficiency is linked in particular to changes in the food supply in Finland, for example concerning access to plant products [35].

3.1.2 Diet components that may increase cancer risk

Red and processed meats

Meat contains on average between 20% and 24% protein (when raw), provides all essential amino acids (lysine, threonine, methionine, phenylalanine, tryptophan, leucine, isoleucine, and valine) and appropriate amounts of various bioavailable micronutrients such as iron, zinc, selenium, vitamins B6, B12, and D, that are required for general health and wellbeing [36]. However, several studies suggest that high consumption of red and processed meats is associated with increased colorectal cancer risk, and provide some evidence for other cancer sites such as the esophagus, lung, and stomach [37-40].

The association between cancer and intake of red and processed meats may be explained by several mechanisms of action including (1) increased fat intake together with meat intake, which leads to an increase in insulin resistance and higher production of bile acids, and therefore could promote carcinogenesis; (2) cooking meats at high temperatures (e.g. roasting) and/or for prolonged times (e.g. stewing) that may result in the formation of carcinogenic heterocyclic amines and polycyclic aromatic hydrocarbons; (3) carcinogenic N-nitroso compounds (NOC), produced inside meat per se or by endogenous metabolic processes due to redox reactions of nitrogen oxides, nitrites and nitrates with secondary amines, namely N-alkyl amines; (4) heme iron in red meat, that acts as a nitrosylation agent forming NOC, and can promote carcinogenesis through increasing cell proliferation in the mucosa, lipid-peroxidation and/or cytotoxicity of fecal water [38].

Cancer risk associated with meat intake may be reduced by decreasing, but not eliminating, meat intake and modifying food preparation/cooking methods [39]. Moreover, as red meat is an important source of micronutrients with anticancer effects, including selenium, vitamin B6 and B12, and vitamin D, adjusting the balance between meat and other dietary components may be critical to protecting against potential cancer risks [39].

Dietary carbohydrates

Some meta-analyses that included several large cohort studies in Europe and North America have suggested an association between low carbohydrate intake and increased mortality [34,42]. However, a large prospective study of individuals from 18 countries across five continents reported that high carbohydrate intake was associated with an increased risk of mortality [43].

Many studies have investigated the association between diets high in carbohydrates, glycemic index or glycemic load, and risk of various cancers; however, the results have been inconsistent [44-48]. Systematic reviews and meta-analyses of observational studies found that high glycemic index and high glycemic load are related to increased risk of cancer at several common sites[34,45,49]. However, other meta-analysis studies do not support an association between diets high in glycemic index, glycemic load, total carbohydrates, and some cancers including endometrial [46], pancreatic [47], and prostate cancer [48].

Consumption of high glycemic load foods, such as grains, potato products, and added sugars, was found to have causal relationships with obesity, diabetes, cardiovascular diseases, and some cancers; while non-starchy vegetables, whole fruits, legumes, and whole-kernel grains appeared to have protective effects [50]. Diets with a low glycemic index/glycemic load were found to be associated with reduced concentrations of fasting blood glucose, glycosylated proteins, and insulin, and therefore decreased the risk of developing some chronic diseases, including cancer [51].

Nevertheless, the biological effect of high-glycemic-index carbohydrates may depend on various factors related to insulin sensitivity, glucose intolerance, and other genetic or acquired physiological predispositions [50]. For instance, several lines of evidence indicate that insulin resistance may play a role in the etiology of pancreatic cancer. Likewise, some potential risk factors for pancreatic cancer such as overweight and obesity, low physical activity, and type 2 diabetes are associated with insulin resistance [47,52].

Alcoholic beverages

There is evidence that consumption of alcoholic beverages (beer, wine, or spirits) is strongly linked to the risk of developing cancer of many organs, such as mouth, pharynx, larynx, esophagus, liver, colorectum, and breast [9,13]. Regardless of the type of alcohol consumed, the International Agency for Research on Cancer has classified its content, ethanol in itself, as a primary carcinogenic component [53].

The main mechanisms by which excess ethanol intake may promote carcinogenesis are: (1) formation of acetaldehyde that exerts mutagenic and carcinogenic effects; (2) expression of P450 2E1 cytochrome and associated oxidative stress and conversion of pro-carcinogens to carcinogens; (3) depletion of S-adenosylmethionine that leads to a global DNA hypomethylation; (4) induction of increased production of inhibitory guanine nucleotide-binding regulatory proteins and components of extracellular signal-regulated kinase–mitogen-activated protein kinase signaling; (5) accumulation of iron and associated oxidative stress; (6) inhibition of the tumor suppressor gene BRCA1 and increased estrogen responsiveness (mainly in the breast); and (7) alteration of retinoic acid metabolism [54].

When examining the impact of intensity and duration of alcohol intake, it has been suggested that relatively heavy

drinkers (>60 g/d) are three times more likely to develop cancer compared to non-drinkers, but the risk starts increasing already at moderate intakes, particularly among women. Regarding the effects of alcohol cessation, the estimated time for normalization of the risk is more than 10 years [55].

Although relatively small amounts of ethanol seem to be safe for some organs, they may be harmful to other organs and tissues. Thus, alcohol consumption should be limited to two drinks per day for men and one drink per day for women [56].

Dietary fat

Traditionally, high dietary fat intake has been considered detrimental to health, and the current dietary guidelines recommend avoiding a high-fat diet and encourage low consumption of saturated fat in particular [57]. Several prospective cohort studies have examined the association between dietary fat intake and mortality from all causes including cancer [43,58,59]. One of these studies conducted among a population in 18 countries from 5 continents found that fats including saturated and unsaturated fatty acids were associated with a lower risk of total mortality [43], and another study reported a positive association between trans-fat intake and all-cause mortality [58].

A systematic review and meta-analysis of prospective cohort studies reported that a diet high in saturated fat was positively associated with higher mortality from cancer, while a diet high in polyunsaturated fat was associated with lower mortality from cancer [60]. However, a recent meta-analysis from prospective cohort studies and randomized controlled trials found no significant association between dietary total fat and fatty acids and the risk of cancer [61].

During the last decades, omega-3 and omega-6 polyunsaturated fatty acids have been a subject of extensive research, because of their nutritional role and their possible relation with the development of certain types of tumors. These essential polyunsaturated fatty acids are derived from the diet. Fish such as tuna, sardine, salmon, and trout, and nuts are rich in omega-3 fatty acids, while food such as eggs, avocado, and most vegetable oils are good sources of omega-6 fatty acids. Omega-6 fatty acids are converted by COX-2 enzyme into prostaglandin E2, a pro-inflammatory cytokine involved in the development of several cancers [62], whereas omega-3 fatty acids compete against omega-6 fatty acids as a substrate for COX-2 enzyme resulting in the production of prostaglandin E3, which does not possess mutagenic properties [63].

Although high consumption of omega-3 fatty acids and low omega-6 fatty acids/omega-3 fatty acids ratio was found to be associated with a low risk of breast, ovarian, prostate, pancreatic, colorectal, and upper aero-digestive tract cancers [13,64-68], there is no enough evidence to suggest a significant relationship between the consumption of omega-3 fatty acids and cancer risk [69,70].

Despite the controversial findings of previous studies, there is some evidence that low-fat diets may reduce the incidence of some common cancers [16,20,71,72]. The World Cancer Research Fund (WCRF) also recommends limiting the consumption of foods high in fat to prevent weight gain, overweight, and obesity, and therefore prevent obesity-related cancers [13].

3.2 Relationship between weight status and cancer

Weight status is determined using body mass index (BMI), calculated by dividing body weight in kilograms by height in meters squared. The healthy or normal range of BMI for adults is 18.5-24.9 kg/m^2; overweight corresponds to BMI ≥ 25 kg/m^2 and obesity to a BMI ≥ 30 kg/m^2 [73]. The healthy range of BMI for children and adolescents varies with age [74].

Obesity and overweight are now recognized as risk factors for various cancers, including mouth, pharynx, larynx, esophageal, stomach, pancreatic, gallbladder, liver, colorectal, breast, ovarian, endometrial, prostate, kidney cancers [13,75].

Mechanisms linking overweight/obesity to cancer are complex and not fully understood. They include obesity-related factors affecting cancer cell promotion and progression such as (1) insulin resistance/hyperinsulinemia and abnormalities of the insulin-like growth factors (IGFs) system and signaling; (2) abnormality in sex hormones biosynthesis and pathway; (3) chronic low-grade inflammation and increased oxidative stress; (4) altered level and action of adipocytokines; (5) factors associated with excessive ectopic fat accumulation; (6) favorable microenvironment for tumors development and cellular perturbations, including vascular perturbations; (7) combined risk factors of obesity and cancer such as disruption of circadian rhythm and unhealthy dietary patterns; (8) disruption of the intestinal microbiome [76].

Furthermore, epidemiologic studies published throughout the 20th and 21st centuries associate high-calorie diets and obesity with the incidence of many types of cancer. Morbidities of obesity, including insulin resistance and diabetes mellitus type 2, are both independently recognized to increase cancer risk[77,78].

There is growing evidence that intentional weight loss may result in a reduced incidence of cancer, particularly obesity-related cancers in women [79], and weight gain is associated with increased cancer risk, especially esophageal adenocarcinoma, colorectal (particularly in men), pancreatic, liver, gallbladder (in women), renal, postmenopausal breast, endometrial, ovarian, and prostate cancers [80,81].

Thus, maintaining BMI within the healthy range (18-24.9 kg/m^2) and waist circumference below 94 cm for men and 80 cm for women [82] as well as avoiding weight gain (measured as body weight or waist circumference) throughout the life span are recommended for cancer prevention [9,13]. This recommendation may be best achieved through maintaining energy balance by i) eating a diet rich in whole grains, fruit, vegetables, and beans; ii) being physically active; iii) limiting the consumption of fast foods and other foods high in fat and sugars; and iv) limiting intake of sugar-sweetened drinks [13]. It should be noted that higher levels of physical activity (46-61 minutes of moderate-intensity physical activity per day) are required to have a significant effect on weight control [83].

3.3 Physical activity

There is strong evidence that regular physical activity, the main determinant of energy expenditure, contributes to the primary prevention of multiple chronic diseases (e.g., cardiovascular diseases, diabetes, cancer, hypertension, obesity, depression, and osteoporosis) and premature mortality [84].

The beneficial effects of physical activity on cancer risk can be attributed to several mechanisms that have been linked to tumor development at various anatomic sites. Physical activity reduces levels of reactive oxygen species, enhances immune function, decreases body fatness and inflammation, and improves insulin sensitivity [85,86]. It has also been reported that moderate physical activity induces gene expression of antioxidant enzymes such as superoxide dismutase, glutathione peroxidase, and catalase, known for their protective effects against oxidative DNA damage and carcinogenesis [87,88]. Moreover, physical activity affects the metabolic profile of estrogens which results in reduced hormonal activity and increased anti-proliferative properties in cancer cells [89].

Regular physical activity is associated with a lower risk of most common malignancies, including lung, breast, hepatobiliary tract, endometrial, colon, oropharyngeal, kidney, and bladder cancers [89-91]. For example, physically active men and women were found to have about a 30%-40% reduction in the relative risk of developing colon cancer compared to their inactive peers. Similarly, physically active women exhibited a 20%-30% reduction in the relative risk of breast cancer compared to inactive women [92]. Several studies have also reported that higher levels of leisure-time physical activity were associated with more than a 20% reduced risk for 7 cancers (esophageal adenocarcinoma, cancers of the liver, lung, kidney, gastric cardia, endometrium, and myeloid leukemia) [93]. However, physical activity of a moderate to vigorous intensity was found to be positively associated with a higher risk of melanoma, a serious form of skin cancer, and prostate cancer [93,94].

Thus, physical activity is becoming an increasingly important intervention for reducing cancer risk. Observational studies have confirmed the protective effect of physical activity against cancer, and support moderate physical activity (>4.5 MET) more than light physical activities (<4.5 MET) [93]. Evidence-based recommendations for cancer prevention suggest being at least moderately physically active across the lifespan (taking part throughout each week in at least 150 minutes of moderate-intensity physical activity) [13].

4 Discussion

There is a wide range of factors related to diet, weight status, and physical activity that can influence the biological processes underpinning the development and progression of cancer. Many studies have shown that diets, characterized by high intakes of vegetables, fruit, and whole grains may have protective effects against cancer. Conversely, diets that are rich in red and processed meats and foods high in fat and sugars as well as alcohol consumption were found to be associated with increased risk of various types of cancers including breast, colorectal, and esophageal cancer [10,11]. However, there is no enough evidence to suggest a significant relationship between cancer risk and various diet components. For instance, a systematic review and meta-analysis study found no association between red and processed meat and pancreatic cancer risk [95]. Similarly, polyunsaturated fatty acid intake was found to have little or no effect on lung cancer risk [69].

Despite scientific advances worldwide and the large body of research published over the last decades, the overall contribution of diet and nutrition to cancer is not well understood [8,32]. This is due to several factors such as measurement error, strong correlations between different foods and nutrients, and other potential confounding factors that should be considered when interpreting the results of studies dealing with diet and cancer [32]. Indeed, observed associations between diet and cancer risk could be confounded by other risk factors, such as physical inactivity for colorectal cancer, human papillomavirus for cervical cancer, Helicobacter pylori for stomach cancer, and smoking for lung cancer [32]. Also, other dietary factors including breastfeeding and fasting, rather than a diet, could be protective factors against cancer and many metabolic, inflammatory, and immune diseases[96,97]. Further studies are needed to fully understand the complex association between diet and carcinogenesis, particularly the effects of certain dietary patterns instead of individual food or nutrient that could be positively or negatively related to the risk of cancer development.

Overweight and obesity are associated with an increased risk of common and less common cancers [13]. However, for some cancer types, associations differ between sexes and populations of different ethnic origins [75]. Furthermore, excess body weight may have protective effects on certain cancers including premenopausal breast cancer and lung cancer [98]. The obesity paradox in cancer may be partially explained by methodological approaches related to using BMI as a surrogate measure of general adiposity, research limitations such as inadequate adjustment for confounding risk factors, selection, stratification and detection biases, and potential confounders including age, tobacco smoking, and physical inactivity [76].

Given the multifactorial etiology of obesity involving genetic, behavioral, epidemiologic, and metabolic factors, a transdisciplinary research approach to investigate the relationship between overweight/obesity and cancer is warranted. Moreover, sarcopenia (low muscle mass), a prevalent condition in obese cancer patients, may need further body composition assessments using sophisticated tools to explore the obesity paradox in cancer and the potential mechanisms that could explain it.

Previous studies have demonstrated that people who engage in moderate to vigorous levels of physical activity are at a lower risk of developing several cancers including lung, breast, hepatobiliary tract, colon, and oropharyngeal cancers [13,90]. Nevertheless, for some cancers, this risk reduction may be independent of the direct effect of physical activity. It has been reported that physical activity helps prevent unhealthy weight gain and physical exercise decreases levels of cancer biomarkers including estradiol, mainly as a result of weight loss [99]. Thus,

the reduced cancer risk associated with physical activity may be primarily due to lowering body weight.

In addition, observational studies have shown that low 25-hydroxyvitamin D levels, in women with early-stage breast cancer, are associated with an increased recurrence risk [100]. Since outdoor physical activity can increase 25-hydroxyvitamin D levels, this could be another mechanism linking physical activity to cancer [90].

Although there is irrefutable evidence that physical activity plays an important role in preventing many cancers, the type, intensity, duration, and frequency of physical activity that have optimal effects for cancer prevention remain unclear [95]. Further studies including controlled clinical randomized trials are required to address this issue.

5 Conclusion

Our findings suggest that adopting a diet high in plant foods such as vegetables, fruits, and whole grains and low in red and processed meats, fat and refined carbohydrates, limiting alcohol consumption, maintaining a healthy weight, and regular physical activity are the cornerstones of cancer prevention interventions. Further studies are needed to elucidate the associations between diet, physical activity, overweight/obesity and cancer for the development of effective preventive strategies aimed at reducing the cancer burden.

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