

Review Article

Diet and Colorectal Cancer: A Comprehensive Systematic Review of Epidemiological Evidence

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Abstract

Colorectal cancer (CRC) ranks among the most prevalent cancers worldwide, with lifestyle factors including diet playing a crucial role in its development and progression. This systematic review aims to synthesize current epidemiological evidence on the associations between dietary factors and CRC risk, following PRISMA 2020 guidelines. A systematic literature search was conducted in PubMed/MEDLINE, Scopus, Web of Science, and Cochrane Library databases for articles published between January 2000 and December 2025. The search strategy combined terms related to colorectal cancer, dietary factors, and risk factors. Studies were included if they examined human populations, assessed dietary exposures, reported CRC outcomes, and were observational studies, interventional trials, systematic reviews, or meta-analyses. Two reviewers independently screened records, extracted data, and assessed quality using Newcastle-Ottawa Scale, Cochrane RoB 2.0, and AMSTAR-2 tools of 3,150 initially identified records, 90 studies met inclusion criteria and were included in the systematic synthesis. The reviewed evidence indicates that high consumption of red and processed meats (RR: +12-18% per 100g/day), saturated fats, ultra-processed foods, alcohol (dose-dependent), and tobacco (RR: 2-3× vs. non-smokers) is associated with increased CRC risk. Conversely, diets rich in fiber, fruits, vegetables, whole grains, fish, olive oil, dairy products, and specific micronutrients (vitamins D, B-group, C, E, selenium, zinc) show inverse associations with CRC risk (20-50% risk reduction). Epidemiological studies suggest that modifiable lifestyle factors may influence CRC risk by approximately 60-70%, though these estimates derive primarily from observational data with moderate to high heterogeneity. This systematic review highlights the potential for CRC prevention through dietary modifications. However, due to the observational nature of most available evidence, further well-designed prospective studies and randomized controlled trials are needed to establish causal relationships. The findings should be interpreted as associations rather than causal effects.

Keywords

Colorectal Cancer, Risk Factors, Lifestyle, Diet

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1. Introduction

In recent decades, the global scene remains dominated by the increase in malnutrition problems due to deficiency in the South and excess in the North, as well as chronic or non-communicable diseases related to diet. These chronic diseases are non-infectious pathologies. The most common are diabetes mellitus, obesity, metabolic syndrome, chronic kidney disease (CKD), cardiovascular (CV) diseases, cancer, and chronic respiratory diseases [1]. In addition, their incidence patterns are changing over time due to population aging, urbanization, increased economic development, and lifestyle changes [2]. According to the WHO, non-communicable diseases (NCDs) cause 41 million deaths per year, or 74% of deaths worldwide. Most deaths occur before the age of 70 and occur in low-income countries. Among these non-communicable diseases, cardiovascular diseases are the leading cause of death, followed by cancers, chronic respiratory diseases and diabetes [3].

Cancer is a complex and destructive condition that manifests itself by uncontrolled and abnormal growth of cells in the body [4, 5]. This disease can affect any organ or tissue, thus endangering the health and life of those affected. Despite advances in medical research, cancer remains a major challenge for health professionals who are looking for effective treatments, both preventive and curative. According to the WHO, cancer is a major cause of death worldwide, causing 9.7 million deaths in 2022; Among this constantly increasing prevalence, colorectal cancer (CRC) ranks second with 904,019 deaths, or 9.3% [6]. Several observations have shown that CRC presents both genetic and environmental risk factors estimated at between 60 and 70% [7]. Among these environmental factors, the most well-known for their associations with CRC are lifestyle, diet, smoking, alcohol consumption, sedentary lifestyle and obesity [8, 9].

This article presents a synthesis of recent studies on the impact of the consumption of different foods, nutrients, ingredients and beverages on the development of CRC, in order to

identify them as risk or protective factors. In addition to these risk and protective factors, it also highlights the influence of the Westernization of dietary practices on the spread of non-communicable diseases in the Global South. As this is a systematic review conducted according to PRISMA guidelines, the evidence presented reflects associations derived from a comprehensive and reproducible search strategy, with explicit acknowledgment of methodological limitations throughout.

2. Methodology

2.1. Search Strategy and Study Selection

A systematic literature search was conducted in PubMed/MEDLINE, Scopus, Web of Science, and Cochrane Library for articles published between January 2000 and December 2025. The search strategy combined terms related to colorectal cancer, dietary factors, and risk factors using Boolean operators.

2.2. Inclusion and Exclusion Criteria

Studies were included if they: (1) examined human populations; (2) assessed dietary factors or nutritional status; (3) reported colorectal cancer incidence, mortality, or related biomarkers; (4) were observational studies, interventional trials, systematic reviews, or meta-analyses; (5) were published in English.

2.3. Selection Process

Two reviewers independently screened titles, abstracts, and full texts. Disagreements were resolved by consensus. The selection process is summarized in a PRISMA flow diagram (Figure 1).

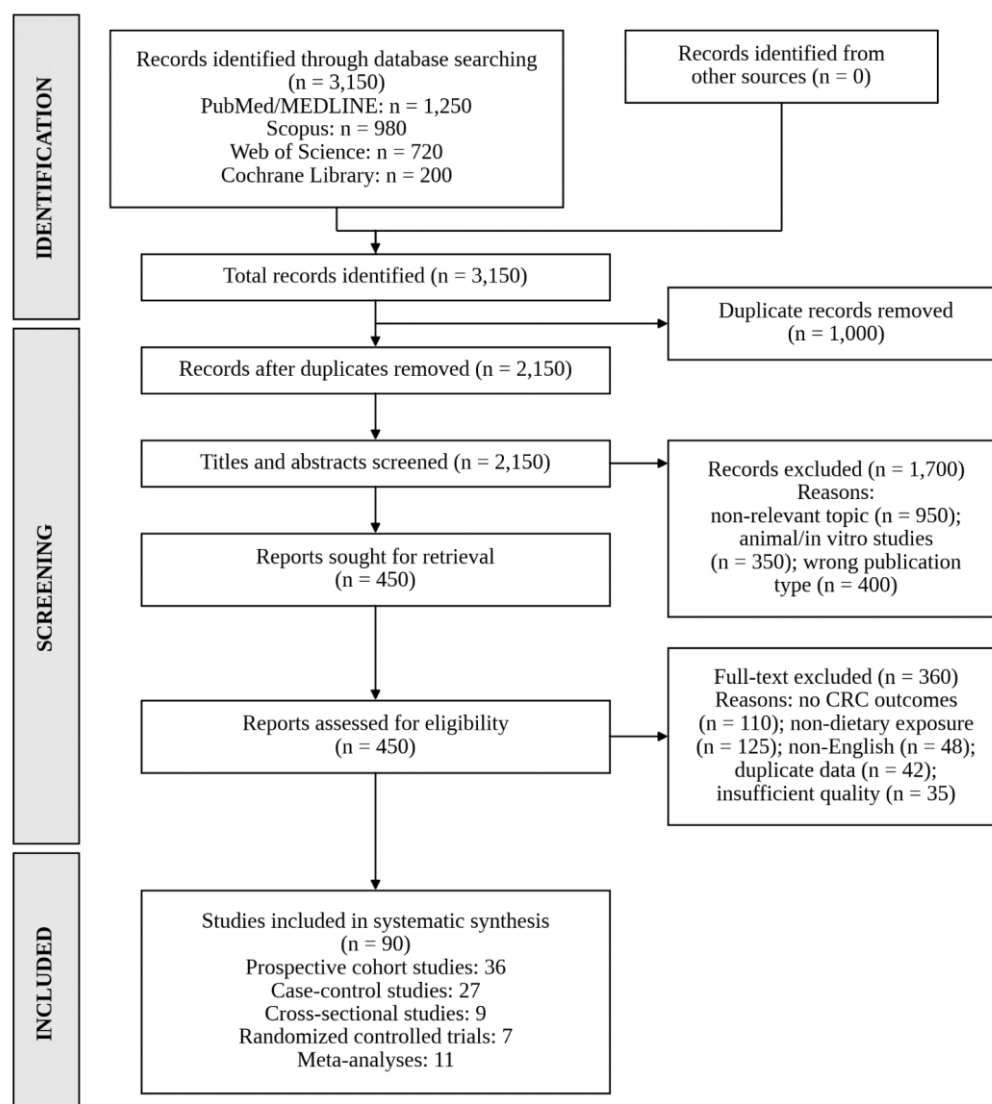


Figure 1. PRISMA flow diagram of study selection 2.4. Data Extraction.

Data were extracted using a standardized form. Quality was assessed using the Newcastle-Ottawa Scale for observational studies, Cochrane RoB 2.0 for trials, and AMSTAR-2 for reviews.

2.5. Data Synthesis

Due to heterogeneity in study designs and exposures, a systematic synthesis was conducted, organizing findings by dietary risk and protective factors.

Table 1. Characteristics of included studies (n=90).

Study design	n	Geographic distribution	Sample size	Key dietary factors	Quality
Prospective cohort	36	North America (16), Europe (13), Asia (5), Australia (2)	1,200 - 520,000	Red/processed meat, fiber, alcohol, fruits/vegetables	NOS: 28 high, 8 mod.
Case-control	27	Europe (11), Asia (9), North America (5), Africa (2)	120 - 5,400	Micronutrients, dietary patterns, meat	NOS: 18 high, 7 mod., 2 low
Cross-sectional	9	Europe (3), Asia (4), Africa (2)	250 - 3,800	Nutritional status, dietary habits	JB1: 6 high, 3 mod.

Study design	n	Geographic distribution	Sample size	Key dietary factors	Quality
RCTs	7	North America (3), Europe (2), Asia (2)	80 - 2,200	Supplementation, dietary inter-ventions	RoB 2.0: 3 low, 3 con-cerns, 1 high
Meta-analyses	11	Global	5-48 studies	Red meat, fiber, alcohol, dairy	AMSTAR-2: 7 high, 3 mod., 1 low
TOTAL	90	-	-	-	-

Abbreviations: NOS, Newcastle-Ottawa Scale; JBI, Joanna Briggs Institute; RoB 2.0, Risk of Bias 2.0; AMSTAR-2, A MeaSurement Tool to Assess systematic Reviews 2

3. Results

3.1. Anatomy of the Colon and Rectum

The large intestine is composed of the colon and the rectum, which is the last part of the digestive system [10]. The colon is a U-shaped muscular tube located below the stomach, while the rectum is a shorter tube that is connected to the colon [11-13]. Together, they are about 2 m long. They are surrounded by other organs such as the spleen, liver, pancreas, bladder, and reproductive organs [14]. The anatomy of the colon and rectum is of paramount importance in the digestion and elimination of bodily waste [15]. These organs have a vital role in the assimilation of nutrients, the production of stool, and the collection of unwanted waste [14]. An alteration in their homeostasis, such as the proliferation of cancerous tumors, can influence a person's overall digestive health [16].

3.1.1. Colon

The colon is located in the abdominal body, extending from the end of the small intestine to the rectosigmoid junction at the level of the third sacral vertebra [17]. It is approximately 1.5 meters long and has a diameter of 8 cm at its point of origin and 4 cm at its end. Its main function is to concentrate feces by reabsorption of water and salt. It is divided into four parts [18]: the ascending colon, the transverse colon, the descending colon, and the sigmoid colon.

3.1.2. Rectum

The rectum is the end of the large intestine that adjoins the sigmoid colon. Its primary function is to store stool before it is expelled through the anus [19]. The rectum is surrounded by muscles called sphincters, which regulate the movement of stool out of the body [18].

3.2. Pathophysiology

Colorectal cancer results from a set of genetic and molecular changes that prevent the normal growth and division of

cells in the intestinal mucosa. The pathophysiology of colorectal cancer is presented below [20]:

Initiation: Colorectal cancers usually begin with the formation of polyps, non-cancerous growths on the inner lining of the colon or rectum. Adenomas, which are some of these polyps, can become cancerous tumors. This transformation is frequently found and is linked to genetic mutations that alter the control of cell growth [21].

Promotion: Tumor cells that develop from adenomas continue to proliferate unchecked. The presence of environmental and risk factors, such as a diet high in saturated fat, excessive alcohol consumption, smoking, and obesity, can promote tumor growth [20].

Progression: As cancer cells multiply, they develop more and more mutations and characteristics that allow them to grow and spread more efficiently [22].

Metastasis: Metastasis is a process by which cancer cells can colonize surrounding tissues, enter blood or lymphatic vessels, and spread to other organs in the body [22]. In advanced stages, colorectal cancer can spread distantly through the lymphatic or blood system, metastasizing to organs such as the liver, lungs, or bones. Metastases complicate cancer treatment and may limit treatment options [20].

3.3. Epidemiology of Colorectal Cancer

3.3.1. Worldwide

Colorectal cancer (CRC) is one of the most common and deadliest cancers worldwide, with more than one million cases in 2022, representing 9.3% of all cancers. It is the third most frequent cancer in men (10.4%) and women (8.9%). CRC is the second leading cause of cancer death (9.3%) [23]. The geographical distribution of CRC is highest in Europe, North America, Australia, and East Asia, while it is lowest in sub-Saharan Africa and South Asia [6]. Risk factors include diets rich in red meat and saturated fats, smoking, alcohol consumption, obesity, and family history of CRC [24].

3.3.2. In Europe

In 2022, approximately 538,262 new CRC cases were diagnosed in Europe, making it the second most frequent cancer in women (after breast cancer) and the third in men (after prostate

and lung cancer) [6]. The highest incidence rates are observed in Western and Northern Europe (Hungary, Slovakia, Norway), while the lowest are in Southern and Eastern Europe [23].

3.3.3. In Asia

The epidemiology of colorectal cancer in Asia reveals distinct characteristics influenced by demographic, economic, and nutritional transitions. CRC is one of the most frequent cancers in Asia, with increasing incidence due to urbanization, obesity, and Westernized lifestyles. In China, the age-standardized incidence rate was 23.7/100,000 in 2022 [25]. In Japan and South Korea, it is the second or third most frequent cancer [6, 26]. In India, incidence remains low but is increasing in urban areas [27]. Despite early screening, South Korea has a high mortality rate, with a 2.4% increase in mortality between 2000 and 2020 [28]. Japan and South Korea have implemented early detection programs through fecal immunochemical testing (FIT) and colonoscopy, reducing mortality. However, in China and India, screening remains limited, leading to diagnosis at advanced stages [25]. Regional differences include East Asia (Japan, Korea, China) with high incidence, South Asia (India, Pakistan) with lower but increasing incidence, and Southeast Asia (Thailand, Malaysia) with rising rates linked to obesity and urban lifestyles [29].

3.3.4. In Africa

Colorectal cancer is less prevalent in Africa than in developed countries, with 70,428 cases (5.9%) of all cancers [23]. However, its incidence is increasing due to the adoption of Western lifestyles such as urbanization, processed diets, and sedentary behavior. CRC represents approximately 7.2% of cancers in men and 5% in women in Africa, and is responsible for 6% of cancer-related deaths. Regional variations in incidence rates are more pronounced in North Africa (Egypt and Tunisia) than in sub-Saharan Africa. Challenges include late diagnosis, limited access to healthcare, and lack of organized screening programs [30].

3.4. Risk Factors for Colorectal Cancer

Colorectal cancer is a chronic disease that affects many people, with an increased risk individuals with personal history of cancer, colorectal polyps, chronic intestinal inflammation, or diabetes mellitus. Lifestyle factors, such as obesity, physical inactivity, smoking, alcohol consumption, and poor dietary habits (excessive consumption of red and processed meat, excessive consumption of processed and fast foods, low intake of fiber from fruits and vegetables) contribute to the increased risk of colorectal cancer [9, 31].

3.4.1. Age and Gender

The rates of occurrence and death associated with colorectal cancer increase with age. In men, the rates are higher than in

women, the difference is visible after the age of 50 [32]. People over 65 are estimated to be three times more likely to develop colorectal cancer than people aged 50 to 64 and 30 times more likely than people aged 25 to 49 [33]. Age increases the risk of contracting cancer [34]. This is because over time, the amount of damage that can develop in cells increases. Thus, as we age, we increase the risk of one of our cells becoming abnormal and generating a tumor [35]. Across all ages and ethnic groups, men are approximately 1.5 times more likely to develop colorectal cancer than women [36]. According to data from the American Cancer Society, men have a 30% higher risk of developing colorectal cancer than women, with poorer prognosis and mortality rates [37]. Women have a higher susceptibility to right-sided colon cancer, often detected at a later stage [38]. Exposure factors, dietary habits, and sex hormones may explain the gender difference [37].

3.4.2. Medical, Family and Personal History

Family and genetic history

The risk of developing colorectal cancer increases when families have colorectal cancer, due to genetic predispositions and lifestyle factors. Factors that influence future cancer risk include the distance between generations of parents, age at first-degree cancer diagnosis, family history of cancer, the presence of other cancers, and relatives with cancer [39]. Studies have shown that individuals with affected parents have twice the risk of developing colorectal cancer compared to those with unaffected parents. The risk is significantly higher when a parent is affected before the age of 60, and this risk of disease also increases when the number of affected relatives is higher [40]. Hereditary non-polyposis colorectal cancer (HNPCC), also called Lynch syndrome, and familial adenomatous polyposis (FAP) are associated with 2 to 8% of colorectal cancers. These conditions are mainly autosomal dominant and are responsible for mismatch repair errors, affecting 20% and 80% of patients, respectively [41].

Chronic inflammatory bowel disease

Inflammatory Bowel Disease (IBD) is linked to colorectal cancer (CRC), accounting for 10-15% of IBD-associated deaths [42]. Patients with IBD are more likely to develop various intestinal malignancies, such as colorectal cancer, adenocarcinoma of the gastrointestinal tract, intestinal lymphoma, and anal cancer [43]. The probability of CRC is related to the duration of the disease and the severity of the underlying inflammation. For patients with IBD, the risk of cancer is approximately 2% after 10 years, 10% after 20 years, and 20% after 30 years [44]. Many extraintestinal cancers are also linked to IBD and treatments, such as cholangiocarcinoma, skin cancer, hematological malignancies, genitourinary cancer, uterine cancer, and prostate cancer [45]. Chronic intestinal inflammation is the third risk factor for developing colorectal cancer, after Hereditary non-polyposis colorectal cancer (HNPCC) and familial adenomatous polyposis (FAP), as it affects the gastrointestinal immune system and leads to persis-

tent inflammation, with a higher risk for symptomatic individuals [33, 46].

Colon polyps

Colon polyps, which are atypical tissues that grow and form a mucosal layer, are divided into a non-neoplastic category (hamartomatous, hyperplastic, and inflammatory polyps) and a neoplastic (adenomatous) category [20]. Among colorectal cancers, 95% are due to adenomatous polyps, and only 5% transform into colorectal cancer [47]. The transition time varies from 5 to 15 years, with increasing risk depending on the size of the polyp, the degree of dysplasia, and age. These polyps are essential elements for diagnosis in individuals over 50 years of age in order to be identified and removed [48].

Diabetes mellitus

Diabetes mellitus is a metabolic disease characterized by persistent hyperglycemia, due to dysfunctions in insulin production and/or action. According to epidemiological data, diabetes is an independent risk factor for various gastrointestinal cancers, including colorectal cancer [49]. Epidemiological research shows that individuals with diabetes are at high risk of developing cancer, especially solid tumors. A large retrospective study conducted in the United States revealed a significant risk of proximal colorectal cancer in diabetic men and women [50-54]. Type 2 diabetics have a two to three times higher risk of colorectal cancer than the non-diabetic population [55].

3.4.3. Lifestyle

Colorectal cancer is a complex disease that is influenced by different lifestyle factors, such as diet, obesity, physical exercise, smoking, and alcohol consumption [56].

Eating habits

Dietary habits have a critical impact on the progression of colorectal cancer. Diet can increase the risk of contracting this disease, while certain foods may help reduce CRC risk, though the evidence is primarily observational and associations should be interpreted cautiously [39, 57, 58].

Red meats and processed meats

According to several studies, there is an association between the consumption of red and processed meat, such as beef, pork, lamb, sausages and bacon, and an increased risk of colorectal cancer. The risk of colorectal cancer may also be increased by consuming processed foods, which contain saturated fats, added sugars, and additives [31, 59, 60]. "Red" meat generally includes pork, beef, lamb, and veal [31]. Processed meat refers to meat that has been preserved by curing, smoking, or adding chemical preservatives to improve flavor or extend shelf life [39]. According to various research and the World Cancer Research Fund (WCRF) reports, regular consumption of red and processed meat has been shown to be associated with increased risk for the development of colorectal cancer [33, 57, 58]. Approximately 12-18% increase in CRC risk may accumulate for every 100 grams of red and processed meat consumed per day [61, 62]. Cooking meat at high temperatures or over open flames (e.g., pan-frying, grilling, or

roasting) produces carcinogens such as heterocyclic amines (HAs), polycyclic aromatic hydrocarbons (PAHs), and N-nitroso compounds (NOCs) [39, 63]. During meat processing, dietary NOCs are produced externally from nitrogen oxides (nitrites or nitrates) and amines or amides. They are therefore found in some processed meats such as bacon, cold cuts, sausages, ham, smoked fish, and smoked cheeses [64]. NOCs are potent carcinogens that can react with DNA [39]. Heme, a porphyrin found in high amounts in red meat, is another factor that may promote malignant transformation of colon cells [39]; this increases the level of oxidative stress and causes lipid peroxidation in the intestines [65]. Reactive oxygen species play a role in DNA damage and genetic mutations [66]. Heme iron induces endogenous NOC synthesis and may cause fluctuation of the gut microbiome, resulting in dysbiosis [9].

Foods high in fat

An association has been documented between consumption of dietary fats, particularly of animal origin, and colorectal cancer incidence [67, 68]. This association involves multiple potential mechanisms. High-fat diets may promote increased bile acid production; primary bile acids can be converted by gut bacterial enzymes (alpha-dehydroxylase) into secondary bile acids, which may act as tumor promoters by inducing compensatory proliferation in colonic epithelial cells through protein kinase C (PKC) activation [69].

Recent research by Yang et al. (2022) demonstrated that high-fat diet promotes colorectal tumorigenesis through modulation of gut microbiota and metabolites in animal models, increasing the abundance of pro-inflammatory bacteria and altering microbial metabolite profiles [70]. Additionally, high-fat diets represent concentrated energy sources, contributing to weight gain, obesity, insulin resistance, and increased circulating concentrations of insulin-like growth factor 1 (IGF-1), which promotes cell proliferation and inhibits apoptosis [71, 72].

Ultra-processed foods

Ultra-processed foods are industrial formulations manufactured through extensive processing, typically containing additives, preservatives, and minimal whole foods [73]. These products include packaged snacks, sugary beverages, instant noodles, and reconstituted meat products. Recent meta-analyses have demonstrated significant associations between ultra-processed food consumption and cancer risk. High consumption of ultra-processed foods is associated with an increased risk of overall cancer, including colorectal cancer [74]. Mechanistically, ultra-processed foods may promote carcinogenesis through multiple pathways: high glycemic load leading to hyperinsulinemia, presence of additives with potential carcinogenic effects, contaminants from packaging materials, alterations in gut microbiota composition and function, and promotion of chronic inflammation [75, 76]. The displacement of nutrient-dense foods by ultra-processed alternatives may further compound these risks through inadequate intake of protective dietary components [77].

Ultra-processed foods are industrial products prepared for

consumption from food substances derived from whole foods, which are usually enhanced with flavors, colors, and other cosmetic additives. According to some research, these foods may increase the risk of cancer due to their obesogenic properties and exposure to potentially carcinogenic compounds such as certain food additives and neoformed contaminants from processing [75, 77]. In addition, some compounds, such as emulsifiers, sweeteners, colors, microparticles, and nanoparticles present in these foods, may affect various parameters, such as the gut microbiota, intestinal permeability, and inflammation [76]. The gut microbiota can convert factors from the gastrointestinal tract and diet into metabolites that may influence carcinogenesis [78].

Overweight and obesity

Nowadays, obesity is considered a major public health issue due to its frequency and rapid progression in many countries. According to the WHO, obesity is defined as "an abnormal or excessive accumulation of body fat that may be detrimental to health" [79]. It is a chronic disease of multifactorial origin that develops from the interaction of social, behavioral, psychological, metabolic, cellular and molecular factors [80]. The disease progresses by developing an inflammatory pathology affecting adipocytes and other elements of adipose tissue [81]. Adipose tissue is not only a storage site for triglycerides, but also a source of bioactive substances known as adipokines. These adipokines are involved in the synthesis of reactive oxygen species (ROS), which contributes to oxidative stress. The development of adiposity may reduce the activity of antioxidant enzymes and contribute to abnormalities, such as endothelial dysfunction and atherosclerosis [82]. Obesity has been linked to various types of cancer, including colorectal cancer, potentially through factors such as hyperinsulinemia, increased pro-inflammatory cytokines, estrogens, insulin-like growth factors, altered adipokine levels [24].

Observational studies suggest that obesity is associated with a 20-50% increase in CRC risk. This risk appears related to visceral obesity, which results from excessive accumulation of fat in the abdominal cavity, with estimates suggesting a 3-5% increase in risk with every 5 kg of weight gained [33, 83]. In addition, the relative risk of colon cancer appears higher than that of the rectum and may be more common in men than in women, with estimates of 50% and 20%, respectively, compared to normal-weight individuals [39].

Physical inactivity

Physical inactivity or a sedentary lifestyle is currently considered the fourth leading risk factor for mortality worldwide.

Its prevalence continues to increase in many countries, potentially influencing the prevalence of non-communicable diseases and the overall health of individuals [84]. According to several observational studies, lower CRC risk is associated with higher levels of physical activity. Physical activity is considered a major preventive factor for weight gain in adults [81, 85, 86]. It plays a vital role in public health by helping to reduce the prevalence of various diseases. Observational studies suggest that individuals who regularly practice physical activity throughout their lives may have a 40-50% lower risk of CRC [87]. Through different mechanisms, physical activity may help to slow or reduce tumor growth, modify sex hormone production and bioavailability, improve insulin sensitivity, and reduce inflammation [88]. Physical activity has been associated with preventive effects on colon cancer, with suggestion of dose-response relationships [89]. The WHO recommends moderate aerobic activity of 10 to 60 minutes, 2 to 3 times per week, for primary cancer prevention and 150 minutes for secondary and tertiary cancer prevention [84].

Smoking

Smoking is a risk factor for many diseases and is responsible for at least 13 cancers, including colorectal cancer [90]. Tobacco use is significantly associated with the incidence and mortality of colorectal cancer in observational studies [91]. According to meta-analyses, smokers have a 2-3 times higher risk of colorectal cancer than non-smokers, and this risk increases with dose and duration of exposure [92]. Tobacco contains various chemical compounds, many of which are carcinogenic, such as nicotine and N-nitrosamines, polycyclic aromatic hydrocarbons, aromatic amines, aldehydes, and metals that may directly damage the colorectal mucosa and induce genetic lesions or epigenetic modifications [93-95].

Alcohol consumption

Alcohol consumption has been recognized as a risk factor for developing colorectal cancer by several epidemiological studies. Alcohol may influence CRC risk through two different pathways. The alcohol metabolic pathway involves the oxidation of ethanol to acetaldehyde and then to acetate, exposing cells to potentially carcinogenic acetaldehyde [96]. Furthermore, alcohol may act as a folate antagonist, potentially contributing to folate deficiency, which could lead to DNA hypomethylation and impaired mitochondrial function [97]. Conversely, higher folate intake has been associated with decreased CRC risk in some observational studies, though these findings should be interpreted with caution [98].

Table 2. Association between lifestyle factors and colorectal cancer risk.

Risk Factor	Direction of Association	Estimated Amplitude	Proposed Mechanisms
Red/Processed Meat	↑ Risk	+12-18% per 100g/day	N-nitroso compounds, heterocyclic amines, PAHs, heme iron

Risk Factor	Direction of Association	Estimated Amplitude	Proposed Mechanisms
High-fat foods	↑ Risk	Not specified	Bile acid production, secondary bile acids, PKC activation, insulin resistance, IGF-1
Ultra-processed foods	↑ Risk	Not specified	Hyperinsulinemia, additives, gut microbiota alterations, chronic inflammation
Alcohol	↑ Risk	Dose-dependent	Acetaldehyde, folate antagonism
Smoking	↑ Risk	2-3× vs. non-smokers	Direct carcinogens, epigenetic lesions
Obesity	↑ Risk	+20-50%	Hyperinsulinemia, pro-inflammatory cytokines, adipokines
Physical Inactivity	↑ Risk	40-50% risk reduction with activity	↓ sex hormones, ↓ insulin resistance, ↓ inflammation
Dietary Fiber	↓ Risk	Protective (observational)	Carcinogen dilution, increased water content, bile acid binding, SCFA production
Fruits & Vegetables	↓ Risk	Protective (observational)	Antioxidants, anti-inflammatory compounds, protease inhibitors, fiber, vitamins, minerals
Calcium	↓ Risk	Protective (observational, via dairy)	Precipitation of secondary bile acids, ionized fatty acids, heme iron; inhibition of cell proliferation

Note: These associations are derived from observational studies and do not imply causation.

3.5. Diets in the Prevention and Treatment of Colorectal Cancer

3.5.1. Diet Rich in Fiber, Fruits and Vegetables

According to several observational studies, diets low in fiber, fruits, and vegetables may be associated with increased risk of several cancers: oral cavity, pharynx, larynx, esophagus, stomach, colorectum, pancreas, prostate, and lung [9, 57, 99]. Protease inhibitors, fiber, vitamins, minerals, folate, plant sterols, and antioxidants are abundant in fruits and vegetables. These compounds may help prevent DNA damage and have been associated with reduced CRC risk in observational studies, potentially due to their antioxidant and anti-inflammatory

properties [100]. They are essential for digestive health and may help prevent constipation, inflammation, and abnormal cell growth. The WHO recommends consuming at least 400 g, or five portions, of fruits and vegetables every day to reduce the risk of non-communicable diseases [101]. Additionally, starchy foods such as potatoes and rice have low energy density and may help prevent diabetes, obesity, and metabolic syndrome-related cancers such as postmenopausal breast, endometrial, and colorectal cancer in observational studies [99]. The potential protective effects of fiber consumption on CRC development suggested by observational studies include reduced transit time, increased stool bulk, dilution of carcinogens, binding of bile acids, and stimulation of beneficial gut microbiota, which ferment fiber to produce short-chain fatty acids that may suppress tumor growth [39].

Table 3. Protective components of fruits, vegetables, and whole grains and their mechanisms.

Component	Dietary Sources	Protective Mechanisms
Insoluble Fiber	Whole grains, vegetables	Carcinogen dilution, reduced transit time
Soluble Fiber	Oats, legumes, fruits	SCFA production (butyrate) via fermentation
Folate (Vitamin B9)	Green vegetables, legumes	DNA synthesis/repair, methylation
Vitamin B2, B6, B12	Various foods	Coenzymes in DNA replication and repair
Vitamin C	Citrus fruits, peppers, kiwi	Antioxidant, PDH regulation, epigenetic regulation
Vitamin E	Vegetable oils, nuts	Anti-proliferative, pro-apoptotic, anti-metastatic
Carotenoids	Tomatoes, carrots, spinach	Antioxidants, cell growth regulation, gene expression modulation

Component	Dietary Sources	Protective Mechanisms
Selenium	Brazil nuts, fish	Antioxidant, ↓ inflammation, selenoprotein expression
Zinc	Oysters, meat, legumes	Mucosal integrity, DNA repair, oxidative stress response
Calcium	Dairy products	Bile acid precipitation, inhibition of cell proliferation
Polyphenols	Olive oil, tea, berries	Modulation of MAPK, PI3K-Akt, Wnt/β-catenin pathways; apoptosis induction

Note: These mechanisms are based on experimental studies and may not directly translate to human cancer prevention.

3.5.2. Whole Grains

Worldwide, the majority of cereals such as wheat, rice, rye, barley, corn, and oats are intended for human consumption. They have several advantages and can be considered functional foods [102]. Consumption of whole grains, which are rich in fiber, vitamins, minerals, and antioxidant compounds, has been linked to reduced risk of type 2 diabetes, cardiovascular disease, and colorectal cancer in observational studies [103]. The presence of insoluble fiber in these cereals increases stool bulk, which may help dilute carcinogens in the colon and reduce transit time, potentially decreasing exposure of the colonic epithelium to harmful substances [104].

3.5.3. Fish

Fish consumption, especially oily fish rich in omega-3 fatty acids (eicosapentaenoic acid [EPA] and docosahexaenoic acid [DHA]), has been associated with reduced risk of colorectal cancer in some observational studies [105]. These fatty acids may influence cancer development through production of anti-inflammatory eicosanoids and specialized pro-resolving mediators such as resolvins, protectins, and maresins, which promote resolution of inflammation [106].

3.5.4. Olive Oil

Olive oil is an essential component of the Mediterranean diet, containing monounsaturated fatty acids (particularly oleic acid), squalene, tocopherols, and phenolic compounds [107]. These phenolic compounds, such as hydroxytyrosol, oleuropein, and oleocanthal, have demonstrated positive effects on free radicals, inflammation, gut microbiota, and colorectal cancer prevention in experimental studies [108, 109]. In experimental models, they decrease proliferation, migration, invasion, and angiogenesis by regulating several signaling pathways, such as the MAPK pathway, the PI3K-Akt pathway, and the Wnt/β-catenin pathway, while inducing apoptosis in various colorectal cancer models [109].

3.5.5. Diet Rich in Calcium, Vitamin D and Dairy Products

Milk consumption, by improving calcium and vitamin D in-

take, has been associated with reduced risk of colorectal cancer in some observational studies [110]. This association is largely attributed to calcium content [111]. Calcium may cause precipitation of secondary bile acids, ionized fatty acids, and heme iron in the colonic lumen, potentially reducing their carcinogenic effects on intestinal mucus. When calcium binds to calcium-sensing receptors in intestinal epithelia, intracellular signaling pathways may be activated to inhibit cell proliferation and migration and promote apoptosis [112]. Vitamin D plays a role in calcium homeostasis and intestinal absorption, and has been shown in experimental studies to regulate cell growth, proliferation and apoptosis, exhibit anti-inflammatory effects, and inhibit angiogenesis [113]. Some epidemiological studies have reported an inverse association between circulating 25-hydroxyvitamin D levels and colorectal cancer risk, suggesting that higher blood vitamin D levels may be associated with approximately 26% lower risk [114]. However, these findings are from observational studies and residual confounding cannot be excluded.

3.5.6. Micronutrients and Carotenoids

According to several studies, certain micronutrients, such as antioxidants such as carotenoids, trace elements, and vitamins (B9, B6, B2, B12, C, E) may play a role in cancer prevention [115-118]. It is important to note that while adequate intake of these micronutrients from dietary sources is associated with health benefits, high-dose supplementation may not confer additional benefits and could potentially have adverse effects in some cases.

Vitamins

B vitamins

Water-soluble B vitamins, including folate (vitamin B9), vitamin B2 (riboflavin), vitamin B6 (pyridoxine), and vitamin B12 (cobalamin), function as coenzymes in DNA replication, repair, and regulation of gene expression [115, 119]. Folate plays a crucial role in one-carbon metabolism, which is essential for nucleotide synthesis and methylation reactions. Folate deficiency may lead to uracil misincorporation into DNA, potentially causing chromosomal breaks and genomic instability [120]. Case-control studies have suggested that folate status and polymorphisms in folate metabolism genes (MTHFR, MTR) may modulate CRC risk in certain populations [120]. However, the relationship between folate and cancer risk may

be complex, with some evidence suggesting that high folate levels could potentially promote growth of existing neoplasms.

Vitamin C

Vitamin C (ascorbic acid) is essential for several key biological processes, such as antioxidant protection, transcription regulation, and epigenetic regulation of gene expression [121]. It is involved in regulation of pyruvate dehydrogenase (PDH) activity, modulating mitochondrial metabolism in KRAS-mutated colon cancer cells [122]. At pharmacological doses, vitamin C may exert pro-oxidant effects selective for cancer cells by generating hydrogen peroxide, while at physiological doses it acts primarily as an antioxidant protecting normal cells [118]. Evidence from supplementation trials has not consistently demonstrated cancer preventive effects, and high-dose supplementation should be approached with caution.

Vitamin E

Vitamin E plays an essential role as a fat-soluble micronutrient that has demonstrated significant antitumor activity in experimental cancer models, due to its antiproliferative, proapoptotic and metastasis-reducing effects on various cancer cells [123]. Tocotrienols, a form of vitamin E, have shown particular anticancer effects by targeting multiple signaling pathways involved in cancer cell growth and survival. However, large-scale randomized trials of vitamin E supplementation have not demonstrated consistent benefits for cancer prevention, and some studies have suggested potential harms with high-dose supplementation.

Carotenoids

Carotenoids are pigments that occur naturally in green vegetables, fruits, algae, and microorganisms [124] and are recognized for their antioxidant properties, attracting increasing interest in the context of cancer due to their effect on the regulation of cell growth, and modulation of gene expression [125]. β -Carotene, lycopene, lutein, β -cryptoxanthin, and α -carotene are the most abundant carotenoids in plasma [126]. Lycopene is a carotenoid with strong antioxidant activity that is a major component of tomato [127, 128]. A cell culture study showed that lycopene at concentrations of 2 μ M or higher reduced the proliferation of HT-29 cancer cells in the colon [128]. However, supplementation trials with β -carotene have shown unexpected adverse effects in smokers, highlighting the complexity of translating observational findings into supplementation recommendations.

Trace elements

Trace elements are essential minerals that are present in small amounts in the body, and are divided into essential and non-essential minerals. Essential trace elements play crucial roles in various biological functions, such as growth and development, fertility, antioxidant systems, and energy and nutrient metabolism [129].

Selenium (Se)

Selenium plays a crucial role as an antioxidant that can protect cells from oxidative damage. Some observational research suggests that adequate selenium status may be associated with

lower colorectal cancer risk, potentially by decreasing inflammation and promoting cellular health [130, 131]. Selenium deficiency has been associated with higher levels of inflammatory cytokines in a variety of tissues, including the gastrointestinal tract [132]. However, selenium supplementation trials have yielded mixed results, and excessive selenium intake can be toxic.

Zinc (Zn)

Zinc is involved in multiple biological activities, including the cellular response to oxidative stress, cell cycle progression, DNA replication and damage repair, and apoptosis [133]. Several studies have suggested that adequate zinc status may contribute to colorectal cancer prevention by helping to preserve the integrity of the intestinal mucosa [134]. Zinc deficiency may impair immune function and DNA repair mechanisms.

Copper (Cu)

Copper, a vital trace element and cofactor, plays a crucial role in maintaining biological functions, which is mainly attributed to its redox-active properties [135, 136]. Copper is generally present in small amounts in many foods, but the foods that contain it the most are organ meats, shellfish and it is distributed in the liver, muscles, eyes and brain [137]. Some research has suggested that copper may play a role in colorectal cancer prevention by inhibiting the growth of cancer cells in experimental models [135]. However, copper homeostasis is tightly regulated, and both deficiency and excess can be detrimental.

Iron (Fe)

Iron is an essential nutrient for almost all living organisms that has anti-inflammatory and antioxidant properties and plays a vital role in immune system functions [138, 139]. Iron has a complex relationship with colorectal cancer, being identified as both a potential risk factor and having protective effects depending on context [140]. However, iron deficiency may influence crucial mechanisms such as immune surveillance, gene regulation, and cell apoptosis, all of which are essential for host defense against malignant transformation and tumor growth [141]. Conversely, excess iron, particularly heme iron from red meat, may promote carcinogenesis through oxidative stress and NOC formation.

Manganese (Mn)

Manganese (Mn) is a trace element crucial for cell development and growth [142]. It acts as a cofactor for several enzymes involved in DNA repair and protection against free radicals, including mitochondrial superoxide dismutase (MnSOD), which converts superoxide radicals to hydrogen peroxide, protecting cells from oxidative damage that may contribute to carcinogenesis [143].

3.6. Nutritional Transition

The nutrition transition characterizes the changes in the dietary patterns of a population that move from an agricultural and subsistence economy to a more industrialized and service-

oriented economy [144]. With globalization, rapid urbanization and improving living standards, diets tend to become more Westernized, becoming richer in saturated fats, refined products, salt, and added sugars [145]. In developing countries, this change in diet is associated with an increase in the frequency of obesity, type 2 diabetes, cancers, cardiovascular diseases and other food-related health problems. There is often a corresponding decrease in the consumption of traditional, fresh and nutrient-rich foods in these countries, and an increase in the consumption of highly processed foods, rich in fat, sugar and salt [146]. This nutrition transition is happening rapidly in these developing countries, which now have to cope with the double burden of malnutrition. In most regions, hunger and malnutrition are the main risk factors for poor health; they represent major challenges and contribute to the incidence and prevalence of non-communicable diseases (NCDs) [147]. In order to address these challenges, it is essential to put in place comprehensive food system approaches, such as awareness campaigns for balanced diets, policies to promote healthy food choices, and initiatives to improve access to whole foods.

4. Discussion

This systematic review synthesized evidence from 90 studies on the association between dietary factors and colorectal cancer risk. The findings indicate that high consumption of red and processed meats, saturated fats, ultra-processed foods, and alcohol is consistently associated with increased CRC risk, while diets rich in fiber, fruits, vegetables, whole grains, fish, dairy products, and specific micronutrients are associated with reduced risk.

These findings are consistent with previous systematic reviews and meta-analyses, including the World Cancer Research Fund (WCRF) Continuous Update Project [58] and the IARC Monographs [33]. The magnitude of risk estimates observed in our review (12-18% increased risk per 100 g/day of red meat) aligns with previous pooled analyses [61, 62]. Similarly, the 20-50% risk reduction associated with high-fiber diets is consistent with the dose-response meta-analysis by Aune et al. [104].

The biological plausibility of these associations is supported by multiple mechanisms. Red and processed meats may promote carcinogenesis through the formation of N-nitroso compounds, heterocyclic amines, and polycyclic aromatic hydrocarbons during high-temperature cooking [39, 63, 64]. Heme iron, abundant in red meat, may catalyze lipid peroxidation and induce oxidative stress in the colonic mucosa [60]. Conversely, dietary fiber may exert protective effects through dilution of carcinogens, reduced transit time, binding of bile acids, and production of short-chain fatty acids by gut microbiota [39, 69].

Strengths of this review include the comprehensive search strategy across four major databases, adherence to PRISMA 2020 guidelines, rigorous quality assessment using validated

tools (NOS, RoB 2.0, AMSTAR-2), and inclusion of a large number of studies (n = 90) from diverse geographical regions.

However, several limitations must be acknowledged. First, the majority of included studies were observational, which precludes causal inference. Second, dietary assessment methods varied considerably across studies, introducing measurement error and heterogeneity. Third, residual confounding by socioeconomic status, physical activity, and other lifestyle factors cannot be excluded. Fourth, publication bias may have inflated the observed associations. Fifth, the predominance of studies from high-income countries limits generalizability to low- and middle-income settings. From a public health perspective, these findings support the promotion of dietary patterns rich in plant-based foods, whole grains, and fish, while limiting red and processed meat, ultra-processed foods, and alcohol. Such recommendations align with existing guidelines from the WCRF and WHO [58, 101].

5. Conclusion and Perspective

Colorectal cancer remains a major public health challenge worldwide. The evidence gathered in this systematic synthesis, derived primarily from observational epidemiological studies, suggests associations between various dietary and lifestyle factors and CRC risk.

Factors associated with increased CRC risk in observational studies: High consumption of red and processed meats, alcohol, saturated fats, and ultra-processed foods has been consistently associated with higher CRC risk across multiple studies.

Factors associated with decreased CRC risk in observational studies: Diets rich in fiber, fruits, vegetables, whole grains, fish, dairy products, and micronutrients (vitamins C, E, B-group, selenium, zinc) have demonstrated inverse associations with CRC risk.

These associations are supported by biologically plausible mechanisms identified in experimental studies, including modulation of inflammation, oxidative stress, insulin signaling, bile acid metabolism, gut microbiota composition, and direct effects on cellular proliferation and apoptosis.

However, important limitations must be acknowledged. The evidence derives primarily from observational studies, which can establish associations but not causal relationships. Residual confounding, measurement error in dietary assessment, publication bias, and heterogeneity in study designs limit the strength of conclusions. As a systematic synthesis, this article did not employ systematic review methodology, including formal quality assessment or meta-analysis, which limits quantitative synthesis of evidence. The estimates of risk reduction (e.g., 60-70% for lifestyle factors) should be interpreted cautiously as they derive from observational data and may overestimate true effects.

Future perspectives should focus on:

- 1) Conducting large-scale prospective cohort studies with standardized dietary assessment methods and objective

biomarkers to strengthen evidence on diet-CRC associations.

- 2) Implementing well-designed randomized controlled trials of specific dietary patterns for CRC prevention in high-risk populations, where feasible and ethical.
- 3) Exploring gene-diet and microbiota-diet interactions to better understand individual susceptibility and enable personalized nutrition approaches.
- 4) Investigating mechanisms underlying protective effects of specific nutrients using molecular and cellular approaches, with careful translation from experimental models to human applications.
- 5) Conducting longitudinal studies in developing countries to assess the impact of nutrition transition on CRC incidence.
- 6) Developing and validating biomarkers of dietary exposure and early carcinogenic changes.
- 7) Implementing public health policies aimed at reducing consumption of ultra-processed foods and red meats while improving access to affordable, nutrient-rich foods.

In conclusion, while definitive causal evidence from randomized trials is limited, the consistency of findings across diverse populations and the biological plausibility of observed associations support the potential for dietary modifications to contribute to CRC risk reduction. Public health strategies promoting balanced diets rich in plant-based foods and limiting consumption of processed meats, ultra-processed foods, and alcohol, combined with regular physical activity and weight management, represent prudent approaches supported by current evidence. Strengthening dietary education, integrating nutritional counseling into healthcare systems, and implementing policies to improve access to healthy foods will be essential steps in reducing the global burden of colorectal cancer.

Abbreviations

AMSTAR-2	A MeaSurement Tool to Assess Systematic Reviews, Version 2
CKD	Chronic Kidney Disease
CRC	Colorectal Cancer
CV	Cardiovascular
DHA	Docosahexaenoic Acid
DNA	Deoxyribonucleic Acid
EPA	Eicosapentaenoic Acid
FAO	Food And Agriculture Organization of The United Nations
FAP	Familial Adenomatous Polyposis
FIT	Fecal Immunochemical Test
HAS	Heterocyclic Amines
HNPCC	Hereditary Non-Polyposis Colorectal Cancer
IBD	Inflammatory Bowel Disease
IGF-1	Insulin-Like Growth Factor 1
JBI	Joanna Briggs Institute

KRAS	Kirsten Rat Sarcoma Viral Oncogene Homolog
MAPK	Mitogen-Activated Protein Kinase
MEDLINE	Medical Literature Analysis and Retrieval System Online
MnSOD	Manganese Superoxide Dismutase
mod	Moderate
MTHFR	Methylenetetrahydrofolate Reductase
MTR	Methionine Synthase
NCDs	Non-Communicable Diseases
NOCs	N-Nitroso Compounds
NOS	Newcastle-Ottawa Scale
PAHs	Polycyclic Aromatic Hydrocarbons
PDH	Pyruvate Dehydrogenase
PI3K-Akt	Phosphoinositide 3-Kinase / Protein Kinase B
PKC	Protein Kinase C
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
RCTs	Randomized Controlled Trials
RoB 2.0	Cochrane Risk of Bias Tool, Version 2
ROS	Reactive Oxygen Species
RR	Relative Risk
SCFA	Short-Chain Fatty Acids
WCRF	World Cancer Research Fund
WHO	World Health Organization

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Conflicts of Interest

The authors declare no conflicts of interest.

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