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Nutrition: The key to rehabilitation of colon cancer

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Abstract
The second most destructive and third most common kind of cancer nationwide is colorectal cancer, often known as colon cancer. A lower-end of the digestive system cancer that affects the colon or rectum. The two main variables that increase the chance of developing a disease are lifestyle and eating habits. Consuming fruits and vegetables, which mostly include food hydrocolloids, can assist in lowering the risk of this kind of neoplasia (unregulated and unstable cell development). Various dietary polysaccharides have implications in anti-tumoral therapy, including as a coadjuvant to common medications, as carriers, or as direct influences on cancer cells. Inulin, -glucans, pectins, fucoidans, alginates, mucilages, and gums are a few groups. Discussing colorectal cancer processes and the functions of various polysaccharides in maintaining intestinal health is crucial. It has previously been investigated how altered mechanisms in colorectal cancer are influenced by genetic, environmental, and immunological factors. In-depth research has been done on the methods by which dietary fibre sources affect a healthy gut environment, including microbial diversity, byproduct synthesis (mainly short-chain fatty acids), inflammatory profile modulation, and tumoral mutant pathways regulation.

Keywords: Colon cancer, dietary fiber, nutraceutical, anti-cancer

Introduction
The second leading cause of mortality in the world, colorectal cancer or colon cancer is very deadly. A major global burden is the prevalence of colon cancer, the third most prevalent cause of cancer-related deaths globally and the fourth most often diagnosed malignancy (Labianca *et al*., 2010) [1]. 10% of the newly reported cases for 2017 were fatalities. Nevertheless, because to the quick development of screening technologies and better treatment choices, the overall colon cancer survival rate is rising. The demand for developing efficient methods of medical intervention increased as a result (Markowitz *et al*., 2002) [2]. Additionally, there are four phases of colon cancer. Phase 1 of cancer's progression comprises development through the mucosa, invasion of the muscles, and expansion through the colon or rectum wall without infiltration into lymph nodes or surrounding tissue. Phase 2 cancer has spread into surrounding tissues after infiltrating through the colon or rectum wall. Colon cancer that has reached phase 3 has metastasized to four or more lymph nodes and perhaps to nearby organs. Eventually, phase 4 cancer has disseminated to one or even more distant organs and may reach the peritoneum (a covering of smooth tissue that encircles your abdominal organs and covers your abdominopelvic cavity) (Ulanja *et al*., 2019) [3].
Physiological, familial, and psychological lifestyle factors all contribute to the development of colon cancer (Blalock *et al*., 1990) [4]. These social determinants include eating patterns such as the intake of red/processed meat and alcohol, which can be connected to developing nations adopting a western influence lifestyle, a low level of exercise, smoking, elderly, and overweight, as well as eating fast food and high sugar beverages drink. Additionally, avoiding or limiting the consumption of red/processed meat, alcohol, and tobacco may help to prevent or lower the risk of colon cancer. In other circumstances, nutrition from either plant or animal sources, whether directly or indirectly, is a major factor in the development of colorectal cancer (Slattery *et al*., 2003) [5].
Surgery may be the only treatment for phase 1 colon cancer, according to the American Cancer Society (El-shami *et al*., 2015) [6]. In the majority of cases, this is achieved using a colonoscope to remove the adenoma or the malignant area. A piece of the colon must always be eliminated, nevertheless, if the cancer is too large to be managed locally (partial colectomy). But on the other contrary, surgery is a good choice for treating malignant tissue and nearby lymph nodes in phase 2 of colon cancer and could be the only necessary intervention. If the cancer has a high chance of recurrence, postoperative treatment is also advised following surgery (Kennedy *et al*., 2011) [7].

The three most often used chemotherapy regimens are capecitabine, oxaliplatin, and 5-fluorouracil with leucovorin. Furthermore, more configurations may be used. Phase 3 cancer is often treated with a partial colectomy, which removes the cancerous portion of the colon as well as any nearby lymph nodes, preceded by adjuvant chemotherapy. The most popular adjuvant chemotherapy regimens are FOLFOX (5-fluorouracil, leucovorin, and oxaliplatin) and CapeOx (capecitabine and oxaliplatin). Therefore, certain patients could be able to receive 5-Flourouracil alone or in combination with leucovorin or capecitabine, based on their age and health (Schrag *et al*., 2001) [8].

Colon cancer usually travels to the liver in the last stages, but it may also reach the peritoneum (the membrane of the abdominal cavity), the lungs, the brain, or distant lymph nodes. Many malignancies are often improbable to be cured by surgery. Surgery will be required to remove the cancerous colonic tissue, any nearby lymph nodes, and any metastatic cancerous sites (Fleshman *et al*., 1996) [9]. Chemotherapy is often given after that. In certain cases, hepatic artery infusion may be used if the cancer has spread to the liver. Before surgery, chemotherapy may be used if the metastasis cannot be removed because the tumorous tissues are too large or numerous. Surgery to remove the tumours can be tried if they start to recede (Bosset *et al*., 2006) [10]. After surgery, chemotherapy may be delivered once more. Two further methods for eliminating liver tumours are embolization and ablation. Additionally, if the disease has progressed too far for surgery to be helpful, chemotherapy is the main treatment option. The majority of phase 4 cancer patients receive chemotherapy and/or targeted therapies to treat their disease, including FOLFOX (leucovorin, 5-fluorouracil, and oxaliplatin, "Eloxatin"), FOLFIRI (leucovorin, 5-fluorouracil, and irinotecan, "Camptosar"), CAPEOX or CAPOX (capecitabine (Xeloda) and oxalip (leucovorin, 5-Fluorouracil, oxaliplatin, and irinotecan) (Watanabe *et al*., 2001) [11].

Causative agents of colon cancer

The majority of colon cancers often start as dysplastic adenomatous polyps. Small bowel epithelium is continuously replaced. Progressive degeneration throughout this transformation phase typically results in adenomatous polyps, followed by dysplasia and aggressive malignancy (Ajouz *et al*., 2014) [12]. Study results including such frequent early carcinoma detection in large adenomatous polyps, identification of adenomas in patient populations 10 years prior to cancer across both sporadic and familial cases, and decrease of colon cancer mortality rate by the discharge of polyps in randomized studies confirm the hypothesis that Colon cancer is a direct consequence of an adenoma-carcinoma pattern (Aquina *et al*., 2017) [13].

Epigenetic and genetic alterations accumulate over time to cause colon cancer (Grady and Carethers, 2008) [14]. These modifications turn cancer from normal glandular epithelium. Individuals with hereditary types of colon cancer are conceived with defective genes. This indicates that a second hit is necessary since the mutant gene is present in one allele in the zygote from the beginning (germ-line mutation). The most well-known forms of hereditary colon cancer are familial adenomatous polyposis (FAP) and hereditary non-polyposis colorectal cancer (HNPCC). Somatic mutations, which originate after birth as a result of environmental conditions, can lead to sporadic malignancies (Armaghany *et al*., 2012) [15].

Oncogenic mutations can result in overexpression of a gene or process, whereas tumour suppressor gene alterations might result in the removal of an inhibitory signal (Zoratto *et al*., 2014) [16]. Every person's case of colon cancer is unique and heterogeneous, and these modifications that produce it have an impact on the disease's phenotype, prognosis, and therapeutic response. The three main molecular pathways that contribute to the development of colon cancer are chromosomal instability, microsatellite instability, and the methylator phenotype. Numerous paths may contribute to the emergence of colon cancer, and each one has distinct properties (Hong, 2018) [17].

Vulnerability Considerations of colon cancer

The incidence of colorectal cancer is significantly influenced by genetic, environmental, and aging factors (Marley and Nan, 2016) [18]. Lynch Syndrome (Hereditary Nonpolyposis Colorectal Cancer), Familial Adenomatous Polyposis (FAP), and MUTYH-Associated Polyposis are hereditary colorectal cancer disorders (MAP). Only around 5% of all cases of colorectal cancer are hereditary, with the majority of cases being caused by Lynch syndrome and familial adenomatous polyposis (Al-sukhni *et al*., 2008) [19].

Despite the absence of the aforementioned hereditary colon cancer syndromes, having first-degree relatives with a family history of the disease increases the chance of developing colorectal cancer in roughly 20% of cases. When compared to the general population, the risk multiplies more than double if first-degree relatives have a history of colorectal cancer.

Inflammatory bowel disease - Ulcerative colitis occurs more frequently than Crohn's disease—male sex, African American ethnicity, and male sex are other factors that are well-known to be associated with colorectal cancer. Red meat, sedentary lifestyles, and obesity. cigarette usage, alcohol use, history of abdominal radiation, acromegaly, kidney transplantation with immunosuppressive therapy, diabetes mellitus and insulin resistance, androgen deprivation therapy, cholecystectomy, coronary artery disease, and ureterocolic anastomosis are all risk factors (Amersi *et al*., 2005) [21].

The positive impact of nutrition in colon cancer

Regular physical activity, a way of eating high in fruits and vegetables, fibre, folate, calcium, dairy products, vitamin D, vitamin B6, magnesium intake, fish consumption, garlic, regular aspirin use, and non-steroidal anti-inflammatory drugs are important mechanisms that have been linked to a decrease in the frequency of Colon cancer (Xu *et al*., 2016) [22].

Dietary fibre: Dietary fibres are considered complex carbohydrates that are present in plants (Reddy, 1999) [23]. They pass through the small intestine undigested but are fermented by colonic bacteria. Non-starchy polysaccharides, such as those in fruits, vegetables, whole grains, cereals, legumes (including beans and lentils), plantains, and tubers, make up this fibre. Pectin, guar, and oat bran dietary fibres are highly fermentable, but wheat bran and cellulose dietary fibres are not. Short chain fatty acids, including such butyrate and propionate, are produced during the colonic microbiota's fermentation of dietary fibres. These fatty acids

have been shown to have anti-proliferative potential by inducing apoptosis, arresting cell cycle progression, and inhibiting chronic inflammatory processes. In addition to increasing stool weight and frequency, dietary fibres can also raise faecal bulk, which may lessen the capacity of faecal mutagens to interact with mucosa cells. These include the insoluble fibres found in foods like almonds, wheat bran, whole-wheat flour, legumes, and vegetables like potatoes, cauliflower, and green beans. Additionally, dietary fibres may shorten intestinal transit times, lower levels of secondary bile acid synthesis, and lessen insulin sensitivity (Zeng *et al*., 2014) [24].

Whole grains: Brown rice, whole-wheat bread, whole-grain cornmeal, cracked wheat, and oatmeal are just a few examples of the whole grains that are very effective in preventing colon cancer (McCullough *et al*., 2003) [25]. Wholegrain fluctuate in their physicochemical and structural qualities, as well as their physiological impacts, according to the composition of their polysaccharides and the amount and diversity of dietary fibres they contain. Energy, proteins, and some other primary and secondary metabolites, which include phytochemicals (phenolic compounds), phytoestrogens, and other bioactive substances that can guard against or prevent colon cancer, are all found in whole grains. These include vitamins (especially B vitamins, including folate), minerals, phytochemicals (phenolic compounds), and phytoestrogens. Additionally, whole grains are a good source of dietary fibre, oligosaccharides, and resistant starch, all of which can affect the environment in the gut (more explanation under dietary fibre) (Slavin, 2000) [26].

Calcium supplementation and milk products: Calcium and the gut microbiota, particularly the bacteria that create lactic acid, have a role in yogurt's ability to lower the risk of developing colon cancer which result in the decrease of nitoreductase, soluble faecal bile acids, and fecal-activated bacterial enzymes (Sorenson *et al*., 1988) [27]. Free fatty acids and unconjugated bile acids can be bound by calcium, which lessens the toxicity of these substances on the colon and rectum. In order to have an effect, calcium must promote cell differentiation and death, suppress cell growth, stop colonic K-ras alterations, and stop haem-induced colorectal carcinogenesis. This is significantly constrained by research linking a high-calcium diet to prostate cancer (Lipkin and Newmark, 1995) [28].
Given this, caution should be exercised while consuming dairy products, especially those that are high in calcium, even though there are several other bioactive components in dairy products that may also play a role in their ability to lower the danger of colon cancer (Pence, 1993) [29].

Fish and fish-related items: According to several meta-analyses, consuming a lot of fish, especially fresh fish like freshwater fish and sea fish, may lower your chance of developing colon cancer. Long-chain polyunsaturated fatty acids (PUFAs), primarily the n-3 fatty acids found in fish, such as eicosapentaenoic and docosahexaenoic acids, are recognized to suppress the development of colorectal cancer. Consuming processed fish, such as salted, dried, smoked, and barbecued fish, should have been done with caution, since there may be a link to an increased risk of colon cancer. This happens because fish processed at high temperatures create heterocyclic amines, which are carcinogenic, and dried or salted fish contains N-nitrosamines (Engest *et al*., 2007) [30].

Methodology: Fish is well acknowledged to be an excellent source of vitamin D, and vitamin D inhibits the development and spread of colon cancer by altering gene expression directly through the vitamin D receptor and inducing cell differentiation and death. Selenium, which can change the metabolism of carcinogens, prevent or repair oxidative DNA damage, and control immunological response, is also found in fish. A strong n-3 fatty acid consumption decreases the expression of nuclear transcription factor B (NF-Kb), inducible nitric oxide synthase, and the formation of the arachidonic acid metabolites prostaglandin E2 and leukotriene B4 (iNOS). These procedures can all prevent the development of colorectal cancer (Wu *et al*., 2012) [31].

Fruits and vegetables without starch: High fruit and non-starchy vegetable intake have been linked to a lower risk of colon cancer. This is because folate and other minerals and compounds, including carotenoids, tocopherols, ascorbic acid, alkaloids, and phenolic compounds, are consumed. These phytochemicals also have anti-inflammatory, anti-cancer, and antioxidant capabilities. By having antioxidant effects, these substances reduce the impact of ROS and prevent cellular damage and carcinogenic insults (Koushik *et al*., 2007) [33].

Phytochemical compounds and health supplements (Nutraceutical): Nutraceutical, also referred to as functional foods, are bioactive substances that come from natural sources like secondary plant metabolites, dietary supplements, herbal products made from fruits, vegetables, and plants, as well as microorganisms or marine organisms. These substances have the ability to prevent, treat, and manage a range of illnesses, such as colon cancer. Powerful antioxidant and anti-proliferative properties are found in phytochemicals, which are mostly found in fruits and vegetables. When these chemicals are combined, they have a synergistic impact against a variety of malignancies (Kuppusmay *et al*., 2014) [34].
The phytochemicals astaxanthin, cryoptoxanthin, xanthophyll, and zeaxanthin metabolites, as well as the carotenoids (alpha) and carotene (beta) from carrots, lycopene from grapes, papaya, and tomatoes, halocynthiaxanthin from a marine organism called Halocynthia roretzi, play important roles as free radical fighters (Sharma *et al*., 2014) [36]. Intestinal bacteria transform polyphenols, which also are divided into flavonoids and non-flavonoids, into simple phenolic acids, which are then absorbed in the small intestine and reduce the risk of colon cancer. Green tea, grapes, turmeric, ginger, and other plants, as well as marine algae, seaweeds, and microbes, all contain polyphenols that act as anti - inflammatory agents and have a substantial impact on the development of colorectal cancer. Dietary polyphenols called flavonoids, which naturally present in plants and drinks such fruits, vegetables, and juices, have been linked to a lower risk of colon cancer (Espin *et al*., 2007) [35].
The incidence of colon cancer has been shown to be decreased by dietary supplements such as omega-3 fatty acids, vitamins (vitamin D, folate, and vitamin B complex),

and eugenol from honey, balm, cinnamon, clove oil, and citrus. As a single or combined preventative or therapeutic approach for colon cancer, herbs and herb preparations have been employed. Several therapeutic plants have been investigated using various experimental models (either as extracts, liquids, or diet enriched) (Haidari *et al*., 2020) [37].

Fibre and Its Significance

To evaluate the part that fibre plays in the development of colon cancer, many epidemiological studies and two randomised control trials have been conducted. While several epidemiological studies have revealed a negative correlation between fibre consumption and colorectal adenomas and carcinomas, two prospective trials showed no such correlation. Additionally, three randomised controlled clinical trials were unable to show that fibre protects against colon cancer (Rose *et al*., 2007) [38].

Even though a pooled analysis of several prospective studies showed an inverse link between dietary fibre consumption and colon cancer, it did not result in a benefit when it was corrected for other dietary risk variables. Cereal fibre and whole grains had the greatest protective effect against colon cancer, according to a meta-analysis. According to a cohort study research, a sensible diet high in whole grains and dietary fibre was linked to a lower risk of developing colon cancer that was Fusobacterium nucleatum positive but not Fusobacterium nucleatum negative. The role of resistant starch in preventing colorectal cancer is still being researched. After fermentation, resistant starch produces Short Chain Fatty Acids, butyrate being one of them. Butyrate has been demonstrated to have anti-neoplastic characteristics in the colon via controlling immunological response and preventing histone deacetylation, providing further prevention towards colon cancer. Nevertheless, a clinical study found no advantage in the establishment of adenoma or carcinomas in Lynch Syndrome patients when resistant starch-Novolose 30 g daily was administered (Ferguson and Harris, 1996) [39].

A recent study demonstrated that fibre consumption changes the gut microbiota in healthy people. In a recent prospective trial comprising 1575 patients with colorectal cancer in phases 1 to 3, greater fibre intake, particularly from cereals, was linked to lower overall and colorectal cancer-specific mortality (Song *et al*., 2018) [40].

The negative impact of nutrition in colon cancer

Studies have identified sugar, animal fats, red and processed meats as the key dietary ingredients that produce inflammation in the body and may raise the risk of colon cancer. Colon cancer frequency has been associated with a high dairy product intake, including milk, yogurt, and cheese. The inclusion of calcium and other substances including casein, lactose, lactoferrin, and butyrate in these products, which can potentially boost calcium bioavailability, has been linked to this decrease (Johnson and Lund, 2007) [41].

High-sugar beverages and instant snacks: Colon cancer risk may be enhanced by eating a diet high in sugar, fat, and fast food and by drinking beverages sweetened with sugar. Fast food and other processed meals, such as snacks, bakery items, and sweets, are commonly consumed in big numbers because they are easily accessible and high in energy (Slattery *et al*., 1997) [42]. Colon cancer risks can be raised by adding free sugars such high fructose corn syrup, sucrose, and sugars found naturally in fruit juices, syrups, and honey to beverages in order to make them sweet-tasting (Bostick *et al*., 1994) [43].

Accordingly, it is advisable to cut back on, avoid, or switch to sugar-free beverages or liquids sweetened with artificial sweeteners if you regularly consume beverages such as sweetened water, sodas, energy drinks, barley water, sports drinks, or tea-based beverages.

Pasteurized and red meat: An increased risk of colon cancer is associated with a high diet of red or processed meat. Processed meat includes red meat that has been preserved by smoking, grilling, cooking, frying, salting, and curing as well as red meat such as beef, hog, veal, and lamb (Corpet, 2011) [44].

Methodologies - The development of heterocyclic amines and polycyclic aromatic hydrocarbons during processing at high temperatures may contribute to the greater risk of colon cancer linked with diets heavy in red/processed meat (related to poor intakes of fruits, vegetables, and fibre). By stimulating the endogenous synthesis of powerful carcinogens, N-nitroso compounds, and cytotoxic alkenals from lipid peroxidation, haem, which is found in large intakes of processed/red meat, may promote the development of colorectal cancer (Kruger and Zhou, 2018) [45].

Smoking forms of tobacco: Smoke from cigarettes contains carcinogens such acetaldehyde, aromatic amines, benzopyrene, N-nitrosamines, aromatic amines, and polycyclic aromatic hydrocarbons. It is well known that tobacco smoke contains nicotine and nicotine-derived nitrosamine, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone, which accelerate CRC metastasis by encouraging cell migration and epithelial-mesenchyma transition. These substances might potentially interact with DNA to generate DNA adducts that would mutate genes or cause dysbiosis in the gut microbiota, which would result in colon cancer (Slattery *et al*., 2004) [46].

Drinking alcohol: There is evidence from several research, including meta-analyses, cohort studies, experimental studies, and many more that long-term alcohol consumption is associated with an increased risk of colon cancer. Alcohol use that is high or moderate (>12.5 gram per day) is linked to an elevated risk of colon cancer and associated deaths. However, there are variations amongst different groups due to variations in genetic variables, body composition, and other dietary factors, such as folate consumption (Na and Lee, 2017) [47].

Methodologies: One of the main organs for distributing alcohol consumed orally is the colon, which causes the intracolonic level of ethanol to be identical to the blood level. The colorectum's cytochrome P450 2E1 (CYP 2E1) converts ethanol to acetaldehyde, a recognised carcinogen, at increased levels since these tissues also express CYP 2E1's functionality. This carcinogen, which the International Agency for Research on Cancer (IARC) has grouped as a phase 1 human carcinogen, causes oxidative stress by increasing the generation of reactive oxygen species (ROS),

which works against the body's antioxidant copying strategy. Reactive oxygen species may result in lipid peroxidation, protein modification, or the formation of carcinogenic adducts when they attach to DNA. As a result, DNA synthesis and repair processes may be inhibited, and glutathione's structure and function may be altered. Consequently, they could promote the growth of colonic mucosal (Pederson *et al*., 2003) [48].

Bacterial alcohol dehydrogenase and catalase, which are secreted in the colon by the colonic microbiota, also decompose ethanol to create acetaldehyde in the colon or rectum. Additionally, alcohol can function as a solvent for other dietary or environmental carcinogens to enter the mucosal cells, decreasing the formation of prostaglandins, lipid peroxidation, and hormone metabolism (Meyer and White, 1993) [49].

Intracolonic ethanol is transitioned to acetaldehyde in the colorectum by cytochrome P450 2E1 (CYP2E1), alcohol dehydrogenase (ADH), and catalase. Acetaldehyde is then transferred to acetate by aldehyde dehydrogenase (ALDH), and its accumulation promotes the development of colorectal cancer by causing mutagenic DNA adducts, lipid (Cho *et al*., 2012) [50].

Animals fats: Consuming animal fats has been associated with a small number of studies on CRC risk. An excessive intake of animal fats alters the colon's flora, which causes intestinal inflammation and raises the risk of colon cancer. A excessive dose of animal fats may enhance the synthesis of primary bile acids in the colon, which are then degraded by anaerobic bacteria in the large bowel and produce secondary bile acids that are carcinogenic, such as deoxycholic and lithocholic acids. Elevated concentrations of these substances may promote colonocyte proliferation and ROS generation, which raises the possibility of mutation and malignant conversion. Colon cancer susceptibility may be decreased by limiting or avoiding consumption of red/processed meat, sugary diets, fast food, sugar-sweetened beverages, alcohol, smoking, and animal fats.

Individuals with colon cancer and the impact of diet

In Hong Kong, a hospital-based case-control study of Chinese populations found that current, regular, heavy alcohol drinkers and cigarette smokers had an elevated risk of colon cancer, and that abstaining from these behaviours for a significant amount of time reversed the risk (Khan *et al*., 2010) [51]. A significant prospective cohort research with extensive patient screening revealed that individuals ingesting dietary fibre, particularly from cereal and fruit, had a lower chance of developing an adenoma. Additionally, advancement in a diet rich in refined grains and high fibre intake, as well as a decrease in red and processed meat, were observed in a theory-driven cognitive and behavioral dietary treatment program undertaken on Chinese colon cancer patients. No dietary deficiency or dietary-related anaemia was found, which could be due to the use of alternative protein sources (poultry, seafood and tofu). The likelihood of patients surviving after this improvement in the dietary intervention was connected to knowledge of the part food plays in colon cancer prevention and therapy (Slattery, 2000) [52].

In the UK (Biobank research), low meat eaters (those who consumed processed/red meat or poultry fewer than five times a week) had a decreased risk of colon cancer than regular meat eaters (those who consumed processed/red meat or fowl more than five times a week) (Wattling *et al*., 2022) [53]. This demonstrates that a high intake of processed meat and red meat on a regular basis is linked to a high risk of colon cancer. A high intake of processed meat was linked to a higher risk of death in individuals with inflammatory bowel illness, which in turn elevated the risk of colon cancer, in another large-scale cohort study.

According to studies conducted on colon cancer patients in China, a limited diet of chicken, shellfish, and processed or unprocessed red meat might lower the chance of developing the disease. There is, however, no consensus about the role that eating a lot of white meat (poultry and fish) plays in lowering the risk of colon cancer because different findings have been recorded. According to a UK Biobank research, people who consumed an average of 76 g of red/processed meat per day were at an elevated risk of colon cancer. The UK recommends consuming no more than 90 g of red/processed meat per day (Parra-sotto *et al*., 2022) [54].

There are few clinical trials examining the role of food in colon cancer patients after surgery. However, studies have shown that a diet high in red/processed meat, refined grains, sweets, and high alcohol consumption were linked to an increased risk of colon cancer occurrences, whereas increased coffee consumption, dietary fibre, and vegetables, particularly those that were light and low-fat, were linked to a lower risk of colon cancer death. Additionally, the 2011 Alternate Healthy Eating Index (high intake of whole grains, fruits, vegetables, legumes, nuts, and long chain omega-3 fatty acids, and low intake of salt, saturated fat, and red/processed meat), moderate alcohol consumption, and lower intake of beverages and juices with added sugar were all linked to a reduced risk of colon cancer mortality in women (Yusof *et al*., 2012) [55]. As the majority of dietary information is now focused on colon cancer prevention, further research is generally recommended to examine the impact of nutrition on colon cancer survival (post-diagnosis) (Bradbury *et al*., 2020) [56].

Conclusion

Consideration should be given to both a plant-based diet with a high consumption of dietary fibre, whole grains, fruits, and vegetables, as well as an animal-based diet with foods like fish and dairy. Additionally, diets low in or free of animal fats, red and processed meat/fish, alcohol, smoking, a diet high in sugar/fat, fast food, and beverages with added sugar are suggested. These are said to typically play important roles in avoiding colon cancer and serving as the patients' post-diagnosis and post-treatment nutritional needs, hence reducing the overall risk of colon cancer and its related mortality.

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