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# Review of the role of dietary fiber in the prevention of colorectal cancer

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**Abstract:**

Colorectal cancer (CRC) is one of the most prevalent types of cancers, whose etiology has been highly related to diet. High intake of dietary fiber has been found to favor the reduction of CRC risk, as deduced from a lot of studies. The aim of this review is to explore the role of fiber in CRC chemoprevention and to provide insight into the mechanisms underlying the reduced risk of this neoplasia associated with the consumption of fiber. In this work, the authors describe the state of the literature regarding the association between intake and CRC risk through presenting data from epidemiological investigations, clinical intervention studies, and molecular mechanisms. Data were assembled from studies like the European Prospective Investigation into Cancer and Nutrition (EPIC) and fiber supplementation studies to assess the role of dietary fiber on CRC. The analysis indicated that dietary fiber intake, particularly from fruit, vegetable, and whole grain sources, is inversely related to CRC risk. Most studies, such as meta-analyses and large cohort studies, presented the significantly reduced CRC incidence of the highest intake of fiber. The favorable benefits of fiber are attributed to various modes of mechanisms, including the modulation of gut microbiota, bile acid metabolism, and a reduction in oxidative stress. Dietary fiber might play a vital role in this regard and lower the risk of CRC through its effects on different biological pathways. Promotion of dietary fiber intake could be a key strategy for the prevention of CRC, especially via a balanced diet rich in fruits, vegetables, and whole grains. Public health initiatives that aim at increasing fiber intake should start early in life to prevent not only metabolic but also neoplastic diseases more influenced by dietary factors. However, fiber supplementation appears promising, although further large-scale studies will be required to establish its definite role in CRC chemoprevention.

**Keywords**: Colorectal cancer; dietary fiber; chemoprevention; gut microbiota; polyphenols

## Introduction:

CRC is one of the leading causes of cancer-related mortality worldwide and represents a major public healthcare challenge. It is the third most commonly diagnosed cancer in the world, with increasing incidence attributed to dietary patterns and lifestyle factors. Of these, diet plays a paramount role in modulating colorectal cancer risk, with special focus on the protective effects of dietary fiber. Evidence suggests that dietary fiber, in addition to promoting intestinal health, may also play a role in the observed reduction in risk of colorectal cancer by affecting various biological mechanisms. These include increased intestinal motility, regulation of intestinal microflora, and the production of short-chain fatty acids (SCFAs), which are reported to have anti-inflammatory and anti-cancer effects.

Despite voluminous research, the precise role of dietary fiber in the prevention of colorectal cancer remains an area of active investigation. Studies point out the differences in protective effects between different types of fiber and their sources, as well as the influence of individual dietary patterns. More recently, the interaction between dietary fiber and the gut microbiota in maintaining colon health and preventing carcinogenesis has been of interest.

This article tries to give an overview of the possible protective mechanisms of dietary fiber by critically reviewing available epidemiological studies, mechanistic studies, and clinical trials to identify the gaps in current knowledge.

## Methods

A systematic review of the literature was conducted to examine the role of dietary fiber in colorectal cancer prevention. Articles were identified through electronic databases, including PubMed, NCBI, using search terms such as for example dietary fiber, colorectal cancer prevention. Several studies published in English were included, with a focus on epidemiological studies, randomized controlled trials, and mechanistic research.

Inclusion criteria were studies that:

- evaluated the relationship between dietary fiber intake and colorectal cancer risk,
- investigated the biological mechanisms underlying the protective effects of fiber,
- focused on human populations or used human-relevant models.

Exclusion criteria included studies that primarily addressed other dietary components, such as fats or proteins, or did not directly relate to colorectal cancer prevention.

## Epidemiology and molecular mechanisms of colorectal cancer

In general, molecular alterations in colorectal cancer can be put into two broad categories: oncogenic mutations resulting in hyperactivity or gain of new function, and mutations that result in reduction or loss of function of tumor-suppressor genes. More interestingly, most oncogenic and tumor-suppressing mutations in colorectal cancer are somatic, i.e., they occur in cells after a person is born; they are not passed from parent to offspring [1, 2, 3]. In 2020, an estimated 1.9 million people were diagnosed with colorectal cancer. That number is expected to grow to 3.2 million by 2040, driven by increasing numbers of the population and changes in lifestyle habits [4]. The incidence of colorectal cancer varies strikingly across different regions of the world; this is almost always increasing, often with economic development and the adoption of Western dietary habits combined with changes in lifestyle [5, 6].

Colorectal cancer refers to malignancies that arise in the colon or rectum, parts of the digestive system. Together they make up the large intestine; the colon absorbs water and nutrients from food, while the rectum stores waste until it is excreted. The colon is divided into the right side, which includes the cecum, ascending colon, and most of the transverse colon, and the left side, which covers the remaining transverse colon, descending colon, and sigmoid colon. Colorectal cancer begins with the uncontrolled growth of glandular epithelial cells lining the colon or rectum. Often, the site of origin is related to the development of adenomatous polyps that can progress to adenocarcinoma [7].

The cells of colorectal cancers have a step-by-step development of morphological alterations from epithelial hyperplasia to atypical hyperplasia, the formation of adenomas, carcinoma in situ, and finally, invasive carcinoma. The classical theory of two-hit hypothesis could be explained by two major models of carcinogenesis, namely the classical or the suppressor pathway and the alternative or the mutator pathway. The cancer has been classified, according to the origin of mutations, into sporadic, hereditary, or familial colorectal cancer. Sporadic cases account for about 70% of all cases and are generally believed to arise from the adenoma-carcinoma sequence due to a specific set of genetic alterations [8].

Chromosomal instability (CIN) in CRC harbors genetic alterations that are not only characteristic for the hereditary forms but also occur in sporadic cases. Among the critical pathways affected are the Wnt/β-catenin, TGF-β receptor, Notch, and Hedgehog signaling. This is principally caused by mutations in the APC gene, which results in the aberrant translocation

of β-catenin to the cell nucleus. The APC gene encodes a tumor suppressor protein, part of a destruction complex that also includes axin, a cyclin-dependent kinase inhibitor (CKI), and glycogen synthase kinase 3 (GSK-3). This complex keeps levels of β-catenin in check and mediates normal cellular function [9].

The destruction complex allows β-catenin to be recognized by β-TrCP, a protein involved in the regulation of the cell cycle. In turn, this requires that a conserved Ser/Thr-rich sequence proximal to the amino terminus of β-catenin be phosphorylated. Axin acts as an essential scaffold, coordinating interactions between both kinases and β-catenin, to achieve this ordered phosphorylation. Thus ubiquitinated, β-catenin is targeted to the proteasome for degradation. However, many details of the destruction complex's activity, especially the function of APC, are still not well understood [10].

The β-catenin synthesis is under more than one level of controls from the interaction between the Wnt protein and its' transmembrane receptor called Frizzled, Fz. After accumulating at the nucleus, β–catenin is bound by a diverse group of transcription factors known to control mitosis or cell proliferation. Mutations on the APC gene disturbed said regulatory events, allowing in their place an unregulated accumulation of β-catenin inside the nucleus; this resulted in the rampant activation of Wnt targeted genes that caused tumor cell over-proliferation. The TGF-β/SMAD signaling pathway is involved in the major regulation of cell growth, differentiation, and apoptosis. The binding of TGF-β to type II TGF-β receptors induces the phosphorylation of SMAD transcription factors—specifically, SMAD2 and SMAD3—by the receptor. The phosphorylated SMAD2/3 then complexes with SMAD4 and translocates into the nucleus, where it regulates genes responsible for cyclin production and apoptosis. Although TGF-β normally functions as a tumor suppressor, mutations in the genes for TGF-β receptors can lead to uncontrolled growth of tumor cells. Other critical regulators of the gut epithelium proliferation are the Notch and Hedgehog pathways. In the Notch pathway, ligand-receptor interactions result in the translocation of the receptors into the nucleus, where they exert their effects on cell fate, differentiation, and oncogenesis. In cancer, mutations may result in continuous activation of this pathway, thereby setting off uncontrolled cell division. The Hedgehog pathway has protein ligands, Indian, Sonic, and Desert, which bind to the Patched receptor. This normally inhibits the activity of Smoothened—a G protein-coupled receptor. Dysregulation of the Hedgehog pathway, through abnormal activation rather than silencing, is more frequently implicated in colorectal cancers through altered expression of target genes [11].

## High fiber dietary

Among other environmental factors, it may be said that dietary fibers can influence the risk of colorectal cancer. The way the dietary fibers are processed can significantly alter the complex process involving the development of colorectal cancer. Dietary fiber refers to that component of plant foods-particularly vegetables-that cannot be digested by enzymes. They include various types of non-digestible carbohydrates, and an example is lignin and polysaccharides. The important forms of dietary fibers are non-starch polysaccharides, pectins, hemicelluloses, cellulose hydrocolloids, and fructo-oligosaccharides. The above polysaccharides can be further sub-classified based on the molecular structure into either a linear or branched form [12].

Functional fibers are a type of dietary fiber that includes isolated, non-digestible carbohydrates, such as inulin and oligofructose, which offer beneficial physiological effects for the human body. Another way to classify dietary fibers is by their solubility in water. Soluble fibers, found in fruits and vegetables, dissolve in water, while insoluble fibers, more commonly found in cereals, do not. The gut microbiota is very important in the fermentation of dietary fiber, with the soluble ones being fermented faster than the insoluble ones [13, 14].

For this, it is said that adults need approximately 30–35 grams a day for men and 25–32 grams a day for women. The latter will provide adequate nutritional requirements to keep the gut microbiome healthy and improve metabolic functions, thus lowering the chances of cardiovascular diseases and colon cancer [15].

The main dietary sources of fiber are cereals, vegetables, fruits, and legumes. Fiber from these various foods differs in chemical composition, physical properties, and solubility, indicating that each might have specific effects on cancer prevention. However, studies of dietary fiber intake from different sources in relation to the risk of colorectal cancer or adenocarcinoma have yielded inconsistent results. Moreover, not much is known about how these associations develop and which sources of fiber are most important [3].

Dietary fiber can also affect the makeup of the gut microbiota, which is the diverse microbial community found in the human digestive system. Using advanced tools like metagenomics, meta-transcriptomics, and bioinformatics, researchers can analyze and track the microbial population, observe changes in its composition, and explore how these microbial communities interact with the gut environment. In a healthy adult, the gut microbiota consists of approximately $10^{13}$ microorganisms from various species, with the most abundant groups being the Firmicutes and Bacteroidetes phyla [16].

## The relationship between fiber and the gut microbiota

Dietary habits can alter the composition, diversity, and richness of the gut microbiota. Dietary fiber acts as a substrate for microbial fermentation processes in various bacterial species with such enzymes that can degrade these complex carbohydrates. In essence, bacteria depend on dietary fibers for energy, metabolizing them through fermentation. This process is mediated through the microbiota of the gut via a biochemical process called fermentation and results in the production of short-chain fatty acids-SDFAs like butyrate, acetate, and propionate. This lower colonic pH and prevent the transformation of bile acid metabolites into their more harmful and toxic forms. Butyrate has anti-cancer properties, with reduced cell proliferation and the induction of apoptosis-programmed cell death [17, 18].

In a study where pigs were fed three different fiber-rich diets, replacing corn, soybean meal, and soybean oil with 20% sugar beet pulp (SBP), defatted rice bran (DFRB), or soybean hull (SBH), notable changes were observed in the gut microbiota. Analysis of fresh feces showed that the microbial communities of DFRB and SBH diets varied significantly over time. As the pigs adapted to the diets over 7-21 days, the count of bacteria responsible for degradation of cellulose and SCFAs production increased with fiber-rich diets, whereas the basal diet showed an increase in the number of pathogenic bacteria. Besides, the gut microbiota of pigs adapted more quickly to the SBP diet than to the DFRB diet, reflected in higher levels of beneficial SCFAs, such as propionate, butyrate, and isovalerate [19].

## Role of butyrate in preventing colon cancer

Butyrate is not directly a part of dietary fiber but is one of the products formed during the fermentation of fiber in the gut. Specifically, soluble fiber fractions are fermented by the gut microbiota, leading to the production of short-chain fatty acids (SCFAs), including butyrate. This SCFA plays a crucial role in supporting gut health, as it serves as the primary energy source for healthy colon cells. Numerous studies have shown that butyrate has protective effects on colonocytes, promoting apoptosis (programmed cell death) and inhibiting the proliferation and differentiation of colon cancer cells, potentially preventing the progression of cancer [20].

In the context of colorectal cancer, tumor cells more often rely on the Warburg effect-a process in which tumors favor glycolysis, rather than oxidative phosphorylation, for energy production. Therefore, even low doses of butyrate never enter the mitochondria of cancerous colon cells and accumulate in the nucleus, thereby acting as a histone deacetylase (HDAC) inhibitor and inducing apoptosis. In addition, butyrate has also been shown to have anti-

inflammatory properties that are important for colorectal cancer prevention. SCFAs, including butyrate, may be bound by a family of G-protein-coupled receptors that generally regulate various biological pathways. More precisely, butyrate interacts with the GPR43 receptor on T cells, leading to an anti-inflammatory response which may be advantageous in colorectal cancer prevention [20].

In one experiment, Kang et al. used a mouse model of CRC induced by azoxymethane and dextran sodium sulfate to test a role of butyrate in the regulation of CRC and further intestinal microecological balance. And it was revealed that butyrate relieved weight loss, improved survival rates, and repressed tumor growth and progression. Further research showed that butyrate treatment lowered the relative abundance of opportunistic pathogenic bacteria, while that of opportunistic probiotic bacteria, including Actinobacteriota, Bifidobacteriales, and Muribaculacea, was increased, confirmed further by 16S rDNA sequencing. This study identified butyrate as a molecule with the potential to improve CRC outcomes by correcting gut microbiota imbalance and hence providing useful evidence for possible use as a prophylactic agent in offsetting tumor growth and regulating tumor-associated microbiome [21].

The following study by Alvandi et al. observes the association of SCFA levels with CRC risk and incidence. The analysis in this paper falls into two outcomes: CRC risk and CRC incidence. A combined estimate for acetic, propionic, and butyric acid revealed that CRC subjects with high risk had considerably lower concentration of the above-named SCFAs: Standardized Mean Difference-SMD = 2.02, 95% CI 0.31 to 3.74, p = 0.02. Moreover, lower SCFA was associated with higher incidence of CRC, SMD = 0.45, 95% CI 0.19 to 0.72, p = 0.0009, compared to healthy subjects. Qualitative analysis showed that in 70.4% of these studies, fecal concentrations of acetic, propionic, and butyric acids, or total SCFAs, were lower for the individuals with a higher risk of CRC, while 66.7% of them showed significant decreases in the levels of acetic and butyric acid in CRC patients compared with their healthy counterparts. In a nutshell, these three major SCFAs when present in lower fecal concentrations are associated with enhanced risks of CRC and higher incidence [22].

Shuwen et al. tried to confirm at the animal level that the NAB inhibited OXA. They constructed a murine model in which the implantation of CRC cells was performed subcutaneously, intestinal bacteria were detected by 16S sequencing technology, and metabolites in mouse stools were detected by GC-MS. NAB was a differential metabolite that affected the effectiveness of OXA. Results revealed that NAB and oxaliplatin synergistically inhibited the proliferation, migration, and invasion and induced the apoptosis in cell lines;

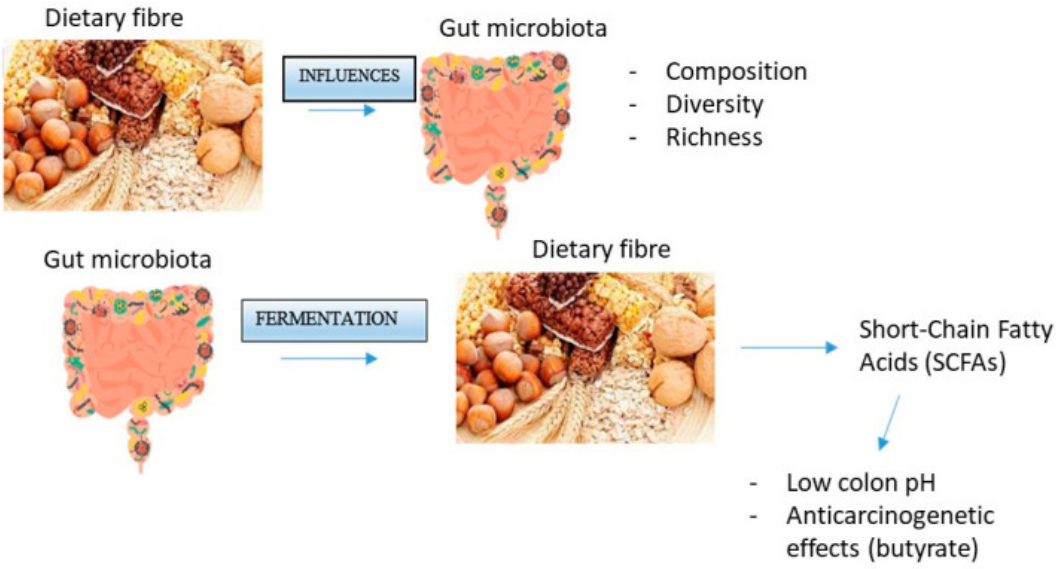
animal experiments confirmed that oxaliplatin in combination with sodium butyrate was able to inhibit the tumors of mice. Besides this, intestinal microbe detection and microbial metabolite detection in the fecal samples from mice showed increased butyrate-producing species, especially the increase of Bacteroides bacteria when the combination of NAB and OXA was used [23].

### Fiber interaction with bile acids

Dietary fibers play a very important role in the prevention of colorectal cancer by interacting with bile acids. These are amphiphilic molecules derived from cholesterol and produced in the liver. They favor the absorption of lipids in the small intestine and form micelles that dissolve cholesterol. The metabolic processing of bile acids occurs in the large intestine; there, the intestinal microbiota promotes the deconjugation and dehydroxylation of primary bile acids to form secondary bile acids. Secondary bile acids are implicated in the pathogenesis of colon cancer. Studies both in humans and animals have indeed demonstrated that toxic bile acids exert proliferating effects on colon cells, thereby supporting a carcinogenic role [24].

Dietary fiber intake can help counteract the negative effects of bile acids on colon health. Fiber promotes increased bowel movement, which plays a crucial role in reducing the risk factors associated with colon cancer. Additionally, fiber binds to bile acids, influencing the gut-liver axis. This binding process helps lower cholesterol levels, which in turn reduces the likelihood of colorectal cancer development [18].

From a molecular point of view, fibers assist in the retention of polyphenols, thereby enhancing their availability for metabolic activity by gut bacteria. Gut microbiota either promotes or reduces oxidative stress depending on the breakdown of fibers into phenolic compounds. These, upon absorption through the intestine and entering the bloodstream, act as antioxidants and thus contribute to the body's defense against oxidative damage [18].




**Ryc. 1**. How fibre reacts with microbiota [25]

**How Fibre helps prevent colorectal cancer**

The hypothesis that dietary fiber protects against colorectal cancer was first put forward in 1971 by Burkitt. He reported that the incidence of colorectal cancer is very low among rural African populations. Colorectal cancer incidence of 3.5/100,000 in the 35- to 64-year-old men versus 51.8 /100,000 in the same age group in Connecticut, USA in Kampala, Uganda, was the recorded result. This was attributed to the significant difference in the African diet, which included minimal amounts of meat but was rich in fiber from fruits, grains, and vegetables [26].

The EPIC study, which took place from 1992 to 2015, aimed to investigate the relationship between dietary and environmental factors and the development of cancer. The results showed that the risk of colorectal cancer was 40% lower among participants with the highest intake of dietary fiber compared to those with the lowest intake [11].

The PrebiotiCa study assessed the impact of six types of soluble prebiotic fibers, including nystose, kestose, 1F-β-fructofuranosylnystose, raffinose, and stachyose, on colorectal cancer prevention. Of these, raffinose and stachyose have shown the most significant protection, with an odds ratio of 0.73 and 0.64, respectively, for the highest intake category compared with the lowest. Of these, stachyose had the highest inverse association with colon cancer risk (OR = 0.74) compared with rectal cancer. Finally, a ten-year follow-up study of people with Lynch syndrome, a genetic condition that predisposes carriers to colorectal cancer, investigated the effects of consuming 30 grams of resistant starch daily for up to four years versus placebo [27].

It has been suggested that the conflicting results reported by studies performed in the United States and Europe may relate to differences in the primary source of dietary fiber; cereal sources, more frequently consumed in the US diet than fruit and vegetable sources, represent the dominant sources of dietary fiber in European diets. Fiber intakes are also generally lower overall in US populations. Other evidence suggests whole grains intake also lowers colorectal cancer risk [28].

A 2017 update of the Cochrane review evaluated the effects of dietary fiber on the recurrence of adenomatous polyps and the incidence of colorectal cancer in participants with a history of adenomas that had been cleared, leaving a polyp-free colon at the beginning of the study. The results showed no difference in the occurrence of developing at least one adenoma, multiple adenomas, or adenomas of 1 cm or larger between the fiber intervention and control groups over three to four years. Additionally, there was no apparent variation in single or multiple adenoma recurrence after eight years of intensive dietary alteration. Yet the authors did point out that it should be interpreted with great caution because of a considerable percentage dropout rate and the fact that adenomas are surrogate rather than final endpoints, which would be colorectal cancers themselves [29].

The PPT was a large, randomized clinical trial that examined the hypothesis that a diet low in fat and high in fiber and fruits and vegetables would reduce the recurrence of adenomatous polyps of the colon within a period of 4 years. The results showed no significant reduction in the risk of recurrence compared with the control group. The PPT-CFS extended the observation period for 4 additional years after the intervention ended. The results also revealed no meaningful differences between the intervention and control groups regarding the likelihood of advanced or multiple adenoma recurrence [30].

A 2018 meta-analysis conducted by Yu Ma et al. examined dietary fiber intake in relation to site-specific colorectal cancer. The findings indicated that higher intakes of fiber were inversely related to the risk of developing proximal and distal colon cancer. The risks in the highest category of fiber intake were lower: for proximal colon cancer, the RR=0.86 and distal colon, 0.79, representing a risk of 21% lower than the low quartile of intake [31].

## Conclusions

Several pathways relate to the intake of fiber and a reduced risk of CRC. It helps in increasing stool bulk and promoting regular bowel movements, thereby reducing the time that

potentially harmful substances, such as bile acids, stay in contact with the colon. Further, fiber is a prebiotic that supports the proliferation of intestinal microflora able to produce short-chain fatty acids with anti-inflammatory properties, which exert a protective role in the colon. Moreover, fiber may modulate cholesterol levels, reducing the risk of CRC further. The exact mechanisms remain under study; however, the evidence to support dietary fiber in CRC prevention is impressive.

However, despite the solid evidence supporting the role of fiber in CRC prevention, the implementation of dietary fiber supplementation as a chemoprevention strategy remains inconclusive. While supplementation with fiber has been widely encouraged, large-scale population studies have not yet definitively proven its role in preventing CRC recurrence or reducing the incidence of new cases. This further underlines the complex nature of CRC prevention, where there is an interplay between genetic predisposition, lifestyle modification, and environmental influences in ways not yet fully understood.

A key factor in the fight against CRC is early nutritional education. Indeed, a very positive change in dietary behavior among subjects at high risk for CRC might be obtained by education on dietary patterns, starting from a young age, rich in fruits, vegetables, and whole cereals. Moreover, dietary education could help avoid not only CRC but also the overall burden of cancer by preventing metabolic diseases that are well-known risk factors for CRC, such as obesity and type 2 diabetes. It is important that this education be incorporated into public health initiatives and policies so that individuals can make better, healthier dietary choices throughout their lives.

Furthermore, dietary fiber itself, while important, should be put into a broader context regarding CRC prevention. Equally important is a healthy lifestyle that includes regular physical activity and weight management, with avoidance of smoking and excessive intake of alcohol in reducing the risk of CRC. A combination of these lifestyle factors, added to a high intake of dietary fiber, provides an integrated approach to CRC prevention.

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