Proceedings of the 3rd International Conference on Environmental Geoscience and Earth Ecology
DOI: 10.54254/2753-8818/93/2025.22053

The Role of Gut Microbiota in the Development of Colorectal Cancer (CRC)

Weicheng Zhang

Shanghai Experimental Foreign Language School Puxi Campus, Shanghai, China
noforgive55@qq.com

Abstract: In recent years, CRC has gradually become one of the most common and deadliest types of cancer worldwide. Numerous studies in modern biology have explored the gut microbiota, revealing an inseparable relationship between microbial composition and CRC development. Currently, multiple articles suggest that microbial products can help promote or inhibit the formation of tumor microenvironment, while different dietary can help reshape the distribution of gut microbiota. This retrospective article aims to summarize previous experimental findings on how gut microbiota and dietary affect tumor progression through microbial products and propose possible future research directions as well. Short chain fatty acids and secondary bile acids mainly used to regulate the tumor microenvironment and has been widely studied. Additionally, research indicates that high protein and high dietary fiber diet with Omega-3 fatty acids contributes to the formation of SCFAs, thus reduced intestinal inflammatory response and reduces the risk of CRC induction.

Keywords: CRC, gut microbiota, microbial products, dietary

1. Introduction

The World Health Organization reports that colorectal cancer (CRC) is the second greatest cause of cancer-related deaths worldwide and the third most frequent cancer worldwide, accounting for around 10% of all cancer cases. It mainly affects the old, with the majority of cases occurring in people aged 50 and above. Resent studies suggest that the gut microbiota is closely related to colorectal cancer through mice experiments. Gut microbiota can guide the production of many organic compounds, such as short chain fatty acids (SCFAs), secondary bile acids which may promote or inhibit the formation of tumor microenvironment. Dietary helps the changes in distribution of gut microbiota, further leading to production of microbial products. However, there remains a lack of clinical studies between CRC patients and the general population, and further investigations are needed to determine the precise impact of gut microbiota on the tumor microenvironment in the human body. Additionally, the molecular mechanisms by which derivatives of gut microbiota promote or inhibit tumor progression are not yet clear. This study aims to discuss the mechanisms of tumor induction by microbial products and gut microbiota remodeling through diet.

2. Microbial product

Studying the microbial products by different types of bacteria and their induction of tumors progression and signaling pathways detects whether people are more likely to trigger CRC by testing

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concentration of microbial markers. The following section will discuss in detail how SCFAs and secondary bile acids affect tumor progression via different mechanisms.

2.1. Short chain fatty acids

The routes of short chain fatty acid (SCFA) generation are very well understood and have recently been detailed. SCFAs are primarily formed by saccharolytic fermentation of carbohydrates that evade digestion and absorption in the small intestine. The major SCFAs include formate, acetate, propionate and butyrate. Among them, butyrate accounts for 40% of the entire production process, and its impact on promoting the generation of the tumor microenvironment has been the most extensively studied.

The Warburg effect, a process in which cancer cells exhibit increased glucose uptake and lactate production even in the presence of oxygen and functional mitochondria, has been widely investigated. In a multicellular setting, the Warburg Effect can offer a favorable environment for cell proliferation. According to the acid-mediated invasion theory, H+ ions released by cancer cells may permeate into the surrounding environment and change the extracellular matrix, making the cells more invasive [1]. Since glucose is the primary energy source for malignant colonocytes due to the Warburg effect, butyrate accumulated and served as an N-butyrate inhibitor of histone deacetylase. Therefore, butyrate hindered the proliferation of cancerous colonocytes experiencing the Warburg effect while stimulating the growth of both normal and diseased colonocytes when the Warburg effect was prevented [2].

Butyrate binds to G-protein-coupled receptor 109a on dendritic cells and macrophages to regulate the differentiation of CD4+ T cells. Additionally, it results in a decrease in T helper 17 (Th17) cells, which promote chronic inflammation and the aggravation of inflammatory diseases, and an increase in regulatory T (Treg) cells, which inhibit chronic inflammation and the aggravation of inflammatory diseases, and interleukin (IL)-10, which gives Tregs the ability to suppress Th17 cell-mediated inflammation, as well as CD4+ T cells. To sum up, butyrate reduces colonic inflammation and slows the spread of colon cancer [3].

SCFAs interact with FFAR2 to improve anti-tumor immunity in addition to inhibiting tumor growth by triggering anti-inflammatory responses. When FFAR2 is not present, dendritic cells become overactivated and generate IL-27, which further compromises their immune response to colorectal cancers by reducing CD8+T cells [4].

Despite their anticancer properties, SCFAs may accelerate the growth of cancer under some conditions. For example, when germ-free mice's intestinal tracts are colonized by the common oral microorganism Fusobacterium nucleatum strain Fn 7-1, the SCFAs that Fn 7-1 produces raise the risk of intestinal cancer by binding to FFAR2 and promoting the expression of IL-17, which favors the occurrence of an inflammatory environment before tumor formation. (F. nucleatum and its metabolites, in particular its high production of SCFA like acetate and butyrate) [5]. Therefore, while SCFAs generally exhibit protective effects against CRC, their role is context-dependent and may vary based on microbial composition and host immune responses.

2.2. Secondary bile acids

Bile salt hydrolases, which have been isolated from a variety of gut microbiota bacteria, including as Bifidobacterium, Lactobacillus, Listeria monocytogenes, Bacteroides vulgatus, Clostridium perfringens, and B. fragilis, catalyze the hydrolysis of the conjugated primary bile acids. Additionally, dehydroxylation is necessary for the production of secondary bile acids, which is facilitated by intestinal bacterial enzymes [6].

Gut microbes can convert primary bile acids including deoxycholic acid (DCA), ursodeoxycholic acid (UDCA), and lithocholic acid (LCA) into secondary bile acids.

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2.2.1. DCA

Clostridium, a bacterium commonly found in the human gut microbiota, produces DCA, a key secondary bile acid. DCA instigates a complex cascade of cellular events that culminate in genomic instability. For instance, it spurs the production of reactive oxygen species (ROS), which directly attack the DNA. ROS can break the DNA strands, modify nucleotide bases, and disrupt the hydrogen bonds that maintain DNA's double - helix structure. This oxidative DNA damage is a significant contributor to genomic instability. DCA also impairs the endoplasmic reticulum and mitochondria. In the endoplasmic reticulum, it disrupts protein folding, leading to the build - up of misfolded proteins and activation of the unfolded protein response (UPR). Prolonged UPR activation can trigger cell death if normal function isn't restored. In mitochondria, DCA interferes with the electron transport chain, reducing ATP production and increasing ROS levels. Moreover, DCA promotes micronucleus formation. Micronuclei, which contain fragmented or whole chromosomes, indicate chromosomal instability. During cell division, they can cause incorrect genetic material segregation, resulting in chromosomal aneuploidy, a characteristic of cancer cells. Long - term exposure to high - dose DCA accumulates DNA damage in cells. Mutations in genes regulating cell growth, division, and apoptosis occur over time, creating a favorable environment for cancer cell growth, particularly in the colon and rectum, thus significantly raising the risk of colorectal cancer [7].

DCA accelerates CRC development through various means. High expression of miR - 199a - 5p suppresses colorectal tumor cell proliferation, migration, and invasion by regulating the cell cycle - related protein CAC1. However, DCA upregulates CAC1 expression, counteracting miR - 199a - 5p's inhibitory effects and accelerating tumor growth [8]. An academic study showed that DCA inhibits the P53 gene, a crucial tumor suppressor, by activating protein kinases that degrade the P53 protein [9]. Another study demonstrated that DCA induces L197 colorectal carcinoma cells to over - express the c - fos and cox - 2 genes [10]. The c - fos gene, involved in cell growth and differentiation, when over - expressed, can lead to abnormal cell proliferation. The cox - 2 gene, which encodes an enzyme for prostaglandin production, promotes a pro - inflammatory microenvironment conducive to tumor growth, angiogenesis, and metastasis when upregulated.

2.2.2. LCA

LCA, a secondary bile acid, is predominantly generated by Clostridium and functions as an endogenous promoter of colorectal cancer (CRC). Its impact within the tumor microenvironment (TME) is rather complex and contradictory.

On one hand, LCA exerts a promotional effect on colorectal tumor progression. It triggers endothelial cell proliferation and the formation of tube - like structures by activating the Erk1/2 signaling pathway. This activation leads to a subsequent reduction in STAT3 phosphorylation within HCT116 cells. As a result, the production of IL - 8 is increased. IL - 8 is a key cytokine, and high levels of it, along with its associated receptors, C - X - C motif chemokine receptors 1 (CXCR1) and 2, are frequently detected in the TME of colorectal carcinoma cells. This elevated IL - 8 and its receptor system can enhance tumor cell migration, angiogenesis, and immune evasion, all of which contribute to the growth and spread of colorectal tumors [11, 12].

Conversely, certain LCA compounds, namely 3 - oxoLCA and isoalloLCA, possess anticancer properties. 3 - OxoLCA binds directly to the crucial transcription factor retinoid - related orphan receptor - γt (RORγt). By doing so, it effectively halts the differentiation of TH17 cells, which are known to promote inflammation and tumor growth. In contrast, isoalloLCA promotes Treg cell differentiation. It achieves this by generating mitochondrial reactive oxygen species (mitoROS). These mitoROS then stimulate an increase in FOXP3 expression. FOXP3 is a master regulator of

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Treg cell function, and enhanced Treg cell differentiation can suppress the overactive immune response that may otherwise support tumor growth, thus exerting an anticancer effect [13].

2.2.3. UDAC

Parabacteroides distasonis is responsible for the production of UDCA, a secondary bile acid that stands as the 7β - hydroxy isomer of chenodeoxycholic acid. Significantly, UDCA has emerged as a remarkable player in the fight against colon cancer. It has been observed to effectively reduce chemically induced tumor progression. In patients suffering from primary sclerosing cholangitis, who are at an elevated risk of developing colon cancer, UDCA showcases chemopreventive effects [14].

The underlying mechanism of UDCA's action lies in its activation of the membrane G - protein - coupled bile acid receptor, TGR5. Once activated, TGR5 initiates a cascade of events that lead to the inhibition of YAP signaling. Specifically, TGR5 exerts its influence by predominantly regulating the cAMP/PKA signaling pathway. This regulation is crucial as it leads to a reduction in RhoA activation. RhoA is a protein that plays a key role in various cellular processes related to tumor growth and spread. By reducing YAP and Ki67 expression, UDCA demonstrates its anti - tumor capabilities. YAP is a transcriptional co - activator associated with cell proliferation and survival, while Ki67 is a marker of cell proliferation. In the AOM/DSS - induced CRC model, UDCA suppresses tumor growth in a concentration - dependent manner. The higher the concentration of UDCA, the more pronounced the inhibition of tumor growth. This unique function of UDCA in controlling YAP signaling and CRC progression highlights the significance of maintaining normal intestinal bile acid metabolism in cancer patients [15].

3. The relationship between gut microbiota and dietary

Our dietary habits are the result of a specific mixture of different nutrients with different amounts, continuously digested in our gut. By evaluating and improving existing dietary patterns, it is possible to modulate gut microbiota composition, thereby reducing the risk of colorectal cancer (CRC). At the same time, one study indicates that the enterotype distribution is continuous rather than discrete, implying that changes in dietary and gut microbiota may have an impact on the entire gut environment [16].

3.1. Effect of Animal-Based Diet on Gut Microbiota

According to a recent mouse experiment, mice fed a high-fat diet showed a decrease in Bacteroidetes and an increase in Firmicutes, indicating that lipopolysaccharide (LPS) is an early component causing metabolic disorders generated by a high-fat diet [17]. The distribution of gut microbiota can be impacted by a high-fat diet, as several research have shown: Firmicutes were up and Bacteroidetes were down [18-20].

Conversely, the ketogenic diet had the opposite effect on the gut microbiota, in which Bacteroidetes getting increased and Firmicutes getting decreased [21]. When tested on humans who fed the modified Mediterranean ketogenic diet, similar results were obtained, while the modified Mediterranean ketogenic diet also reduced lactate and acetate in the subjects' feces, and increased butyrate and propionate [22]. In addition, the gut microbiota linked to high-fat ketogenic diets lowers intestinal pro-inflammatory Th17 cell counts, thus reducing intestinal inflammatory response [23]. According to these results, a ketogenic diet might help reduce the incidence of colorectal cancer.

DOI: 10.54254/2753-8818/93/2025.22053

3.2. Effect of Plant-Based Diet on Gut Microbiome

A recent study indicated that a plant - based diet is associated with a relatively high abundance of Prevotella species [24]. Another study compared the dietary habits of European and African children and their impacts on the gut tumor microenvironment. European children consumed a low - fiber diet, whereas African children had a high - fiber diet. And the researchers found that the African children's intestines contain more SCFAs than the European children [25]. Another study suggests that Porphyromonadaceae and Erysipelotrichaceae significantly decreased as dietary fiber increased, while Bacteroides and Alloprevotella significantly increased, thus the plant-based dietary could remarkably change the distribution of gut microbiota [26]. Furthermore, a human dietary intervention study examining gut bacterial composition based on self-reported dietary habits found that vegetarians exhibited an enrichment in the Prevotella enterotype.

3.3. Effect of Omega-3 polyunsaturated fatty acids (Omega-3 PUFA) on Gut Microbiota

Omega-3 PUFA supplementation has been shown in a recent study to boost the abundance of butyrate-producing bacterial taxa [27]. Additionally, it seems that the amounts of Firmicutes and Bacteroidetes are decreased by omega-3 PUFAs from flaxseed and fish oil, respectively [28].

Additional research has repeatedly demonstrated that supplementing with ω-3 increases the abundance of bacteria that produce butyric acid, such as Lactococcus, Coprococcus, and Eubacterium [29]. Furthermore, bacterial species of the families Lachnospiraceae, Ruminiclostridium, Ruminococcaceae UCG-014, Enterobacter, and Veillonella parvula were found in greater abundances in diets supplemented with Omega-3 PUFAs [30].

3.4. Effect of Fiber on Gut Microbiota

The intact, naturally occurring lignin and carbohydrates found in plants that are neither absorbed or broken down in the small intestine are referred to as dietary fiber. There are two types of dietary fiber: soluble and insoluble. Intestinal bacteria break down soluble fiber to create SCFAs [31].

An open-label, non-randomized research that examined a prebiotic fiber intervention in a small group of individuals with Parkinson's disease suggests that prebiotic fibers may increase SCFAs. It also pointed out that each type of fiber has an own metabolic profile, with resistant starch being highly butyrogenic, resistant maltodextrin being highly propiogenic, and rice bran and inulin increasing the synthesis of all three SCFAs [32]. Furthermore, a stool fermentation investigation revealed that each fiber enhanced different bacterial species and that there was minimal overlap between the fibers. The bacteria enriched by the prebiotic fibers included the genus Prevotella and the families Lachnospiraceae and Ruminococaceae (supported by resistant starch, Cluster 1), the genera Ruminoccocus, Dorea, and Bacteroides (supported by rice bran, Cluster 2), the genera Blautia, Anaerostipes, and Bifidobacterium (supported by inulin, Cluster 3), and the genus Parabacteroides (supported by resistant maltodextrin, Cluster 4) [32]. Furthermore, a different study revealed that the group that consumed a high-fiber diet with probiotics produced more SCFAs, particularly acetate and butyrate [33].

4. Conclusion

Based on the results of multiple studies, a correlation exists between the tumor microenvironment of CRC and the gut microbiota and their microbial products, yet the specific molecular mechanisms need more systematic academic and clinical research to better promote medical guidelines for CRC prevention and control, as well as clinical risk estimation and analysis of CRC. Since dietary patterns are closely related to the formation of gut microbiota and microbial products, more detailed

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comparative studies of regional diets may lead to the proposal of more reasonable dietary structures considering both gut microbiota and the tumor microenvironment. The standardized application of gut microbiota in the treatment and prevention of CRC is becoming increasingly important, such as obtaining the appropriate distribution range of gut microbiota through CRC risk analysis and mouse experiments, preventing excessive supplementation of gut microbiota or microbial products from causing pro - inflammatory or ineffective effects, and providing more diverse and better - prognosis CRC treatment options like combining traditional radiotherapy and chemotherapy with prebiotic supplementation. The study's conclusions may enable the proposal of more innovative CRC treatment plans, and under the basic mechanisms background, genetic engineering might change the DNA base sequence to prevent tumor progression. Additionally, this paper puts forward several questions as possible future research directions, including how to ensure the balance of gut microbiota when using Fecal Microbiota Transplantation, how breast - feeding affects the formation of healthy gut microbiota in children, and how the gut microbiota in the child of a mother with CRC differs from that of an ordinary child; these questions lack academic and systematic discussions, and their results may offer a new perspective on CRC.

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