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Gut Microbiota Imbalance Recovery in Colorectal Cancer Therapy

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Abstract: *The gut microbiota, regarded as the "eighth organ" of the human body, plays a crucial role in maintaining the intestinal barrier function, participating in immune regulation, facilitating substance metabolism and absorption, as well as cholesterol degradation. Colorectal cancer, a common malignant tumor of the digestive tract, has the dysbiosis of the gut microbiota as a significant contributing factor to its development. Through various approaches such as the intake of probiotics, prebiotics, synbiotics, and the implementation of microbiota transplantation, the disruption of the gut microbiota can be rectified, and its balance can be sustained. This offers more alternatives in preventing the incidence of colorectal cancer, retarding tumor progression, reducing postoperative complications of colorectal cancer, enhancing the efficacy of anti-tumor drugs, and mitigating their side effects.*

Keywords: *Dysbiosis; Microbiota Transplantation; Probiotics; Prebiotics; Colorectal Cancer*

1. Introduction

Colorectal cancer is a prevalent malignant tumor of the digestive tract, posing a significant threat to human life and health. Globally, the incidence and mortality of colorectal cancer rank third and second, respectively, among all malignant tumors[1]. Intestinal microbiota dysbiosis is a crucial factor in the initiation and progression of colorectal cancer[2]. This paper provides a comprehensive review of the physiological characteristics of the intestinal microbiota and the association between microbiota dysbiosis and colorectal cancer.

2. Gut microbiota and its related physiological characteristics

A large and complex microbial community resides in the human gut, among which bacteria occupy the main position. The number of bacteria, varying in species and quantity, can reach up to \(1 \times 10^{14} \), with over 1000 distinct species[3].The intestinal microbiota can generally be categorized into three groups: beneficial bacteria (such as Bifidobacterium and Lactobacillus spp.), pathogenic bacteria (such as Helicobacter pylori and toxigenic Bacteroides fragilis), and opportunistic pathogenic bacteria (such as Escherichia coli)[4]. Given that the internal structures and pH values differ across various segments of the intestine, the distribution of the intestinal microbiota also varies accordingly. It is worth noting that even within the same segment and at the same plane, the microbiota distribution is heterogeneous. Vertically, the bacteria in the intestine can be divided into three layers: the luminal bacteria layer (predominantly Escherichia coli and Enterococcus), the intermediate bacteria layer (mainly Peptostreptococcus and Faecalibacterium prausnitzii), and the mucosal bacteria layer (chiefly Lactobacillus and Bifidobacterium)[5].Normally, the intestinal microbiota maintains a dynamic balance with the external environment and the host. The resident bacteria in the intestine typically adhere closely to the intestinal mucosal epithelial cells, thereby preventing the adhesion of other bacteria. Moreover, these resident bacteria can secrete bacteriocin-like substances, which not only inhibit the proliferation of other bacteria but may also eliminate them. This process enhances the immune response of the intestinal mucosa and boosts the host's immune defense against exogenous bacteria.The normal intestinal microbiota is capable of synthesizing a variety of vitamins essential for the human body, including vitamins B1, B2, B6, B12, folic acid, and biotin[6]. It also plays a crucial role in maintaining the intestinal barrier function, facilitating substance metabolism and absorption, and contributing to cholesterol degradation[7].The intestinal microbiota is actively involved in immune metabolism, particularly through the actions of metabolites such as short-chain fatty acids, bile acids, and tryptophan

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metabolites[8]. Additionally, the intestinal microbiota has demonstrated a remarkable impact in the comprehensive treatment of cancer. It not only enhances the efficacy of anti-cancer drugs but also mitigates their toxic and side effects[9].

3. Distribution of Intestinal Microbiota in Colorectal Cancer Patients

Kostic et al.[10] and Castellarin et al. [11]reported an increased load of Fusobacterium in colon cancer tissues. Wu N et al.[12] used culture - independent pyrosequencing and RT-qPCR methods to compared the composition of the human intestinal microbiota between CRC patients and healthy subjects.This investigation confirmed a significant elevation in the abundances of Bacteroides and Fusobacterium in colorectal cancer patients.Moreover, the research findings further indicated that colorectal cancer patients harbored a greater number of other specific potential pathogens compared to the control group. These pathogens included Enterococcaceae and Campylobacter, among others.

4. Ways of Gut Microbiota Imbalance

Under normal circumstances, the distribution of microorganisms in the gut remains relatively stable. Due to external environmental or host factors, the types and quantities of normal microorganisms may change. The possible ways that can cause gut microbiota imbalance are summarized and analyzed as follows:

4.1 Overuse of antibiotics

The irrational use of antibiotics is the most prevalent cause in clinical settings, particularly the long - term application of broad - spectrum antibiotics. Liu Chonghai et al. [13] established an animal model of intestinal microbiota dysbiosis and discovered that after treatment with cephalosporin antibiotics, the intestinal microbiota of mice was significantly disrupted. Further investigations showed that when the normal intestinal microbiota was affected by antibiotics, it typically led to a decrease in the expression of relevant recognition receptors (Toll - like receptors) in innate immunity, consequently resulting in a decline in the body's immune function.Due to the abuse of antibiotics,the quantity and diversity of colonizing bacteria decline, while the proliferation of certain fungi increases. This subsequently leads to secondary infections, further exacerbating the imbalance of the intestinal microbiota.

4.2 Translocation of Intestinal Bacteria

Generally, the distribution of microbiota in different parts of the digestive tract remains relatively stable. However, when the intestinal mucosa is damaged, the permeability of intestinal mucosal epithelial cells increases. As a result, intestinal bacteria and their metabolites can penetrate the intestinal mucosal barrier and enter other tissues, organs, or be absorbed into the bloodstream, thereby causing microbiota dysbiosis. The body may experience shock due to trauma or various other factors, triggering corresponding stress - induced protective responses. To ensure the blood supply to vital organs such as the heart and brain, the blood supply to the intestinal mucosa is reduced, which can lead to necrosis and shedding of the intestinal mucosa. This increases its permeability, creating conditions for the translocation of microbiota. Related research has indicated that under chronic stress conditions, the neuroendocrine system is also in a state of stress. Under the regulation of the brain - gut axis, the permeability of the intestinal mucosa increases, thereby inducing microbiota translocation .

5. The Role of Microbiota Dysbiosis in the Onset and Progression of Colorectal Cancer and Associated Mechanisms

As previously mentioned, compared to healthy individuals, patients with colorectal cancer exhibit a significant imbalance in their intestinal microbiota. The quantity and variety of normally colonized beneficial bacteria decrease, while the number of pathogenic bacteria increases. Holtmeier et al. [14] employed quantitative polymerase chain reaction (qPCR) to detect the expression of Fusobacterium nucleatum in colorectal cancer tissues and adjacent non-cancerous tissues. The results indicated that its expression level was notably elevated in tumor tissues and was positively correlated with lymph node metastasis.Some scholars have pointed out that in the feces of colorectal cancer patients, the ratio of Bifidobacterium to Escherichia coli declines. Since Bifidobacterium can enhance the body's anti-tumor

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immunity, a reduction in its numbers increases the likelihood of cancer cell immune escape[15]. Research has shown that certain bacteria that promote colorectal cancer (such as Fusobacterium nucleatum, Escherichia coli, and Bacteroides fragilis) and protective bacteria (such as Clostridium butyricum, Streptococcus thermophilus, and Lactobacillus paracasei), along with their corresponding metabolites, increase, facilitating the initiation and development of colorectal cancer [16]. Evidently, microbiota dysbiosis plays a pivotal role in the progression of colorectal cancer, tumor immune escape, and poor patient prognosis. The relevant mechanisms are classified and analyzed below.

5.1 Carcinogenic Effects of Bacterial Cells, Secretions, and Metabolites

Numerous studies have demonstrated that multiple bacterial species, including Streptococcus bovis, Enterococcus faecalis, Fusobacterium nucleatum, Bacteroides fragilis, and Escherichia coli, are enriched in the intestines of colorectal cancer patients. These bacteria regulate the proliferation, invasion, metabolism, and immune function of tumor cells and are involved in the development of colorectal cancer [17].Deng et al. [18] conducted animal experiments and found that Streptococcus bovis can induce immunosuppression by recruiting tumor-infiltrating $CD11b^{+}TLR\text{-}4^{+}$ cells, thereby promoting the development of colorectal cancer. Additionally, some researchers have reported that a specific antigen extracted from the cell wall of Streptococcus bovis can upregulate cyclooxygenase-2 expression, promoting the carcinogenesis of human adenocarcinoma cells[19].Rubinstein et al.[20]discovered that Fusobacterium nucleatum can bind to epithelial cadherin through surface adhesion proteins, followed by the activation of β-catenin, thereby altering the intracellular signaling pathways of intestinal mucosal epithelial cells and promoting the development of intestinal cancer.Research has also shown that the toxin secreted by Bacillus fragilis can induce the formation of colonic tumors in mice by activating transcription factor 3 and the signal transducer and activator of transcription in the T helper 17 response[21].Moreover, Campylobacter jejuni can produce cytolethal distending toxin with DNase activity, which causes DNA double-strand breaks and promotes the proliferation of tumor cells[22].In addition to the carcinogenic effects of bacterial cells and their associated cytotoxins, numerous studies have shown that the metabolic products of some intestinal bacteria and the related enzymes they synthesize play a crucial role in tumorigenesis and tumor progression. Metabolic products such as hydrogen sulfide gas and secondary bile acids produced by intestinal microbiota can induce DNA damage, increase the incidence of tumors, and promote the progression of colorectal cancer. Meanwhile, substances such as spermine oxidase, nitroreductase, β-glucuronidase, and azoreductase, synthesized by certain bacteria, can also cause DNA damage and increase the probability of gene mutations, significantly elevating the risk of tumor development[23][24].

5.2 Activation of Inflammatory Factors and Immune Imbalance

Microbiota dysbiosis in the intestine leads to a decline in the function of the intestinal mucosal barrier, which further facilitates the translocation of bacteria. The translocated bacteria can trigger a chronic inflammatory response, releasing a substantial amount of chemokines and cytokines (such as TNF-α, IL-6, IL-17, and IL-23), thereby promoting the proliferation and survival of tumor cells[25].Furthermore, these cytokines can activate signaling pathways such as TGF-β, Notch, and Wnt, reducing the self-renewal capacity of intestinal mucosal epithelial cells and further driving the progression of colorectal cancer[26].Microbiota dysbiosis activates transcription factors NF-κB and signal transducer and activator of transcription 3 (STAT3), leading to abnormal tissue repair and immune imbalance in the colorectal region. Simultaneously, it can also activate pathways such as MAPK and Akt/PKB, influencing the survival and mitosis of colorectal cells and thus promoting the development of colorectal cancer[26].

6. Approaches to Restore Dysbiotic Intestinal Microbiota

Numerous studies to date have demonstrated that probiotics, prebiotics, synbiotics, and microbiota transplantation play a crucial role in restoring the microbiota after dysbiosis. Among them, the treatment of microbiota dysbiosis with probiotics is the most common approach currently, and microbiota transplantation has also become a research focus in recent years.

6.1 Probiotics, Prebiotics, and Synbiotics

Probiotics are live microorganisms that colonize the body and confer beneficial effects on the host,

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such as Clostridium butyricum, Lactobacillus, Bifidobacterium, Actinomyces, and Yeast. Prebiotics are substances that the human body cannot absorb (mostly oligosaccharides). These substances can be utilized by the normal intestinal microbiota to enhance their activity or promote their growth. Synbiotics are a combination of probiotics and prebiotics, to which some vitamins and trace elements can be added. These three types of substances play a significant role in regulating the host's mucosal and systemic immune functions and maintaining the balance of the intestinal microbiota.Studies have shown that long-term use of broad-spectrum antibiotics can lead to intestinal microbiota disorders, increased intestinal mucosal permeability, decreased resistance and protective ability of the intestinal mucosa, and is prone to cause secondary infections. Oral administration of probiotics, prebiotics, or synbiotics can correct the microbiota dysbiosis caused by antibiotics, reduce intestinal inflammatory responses, and restore the role of the normal intestinal microbiota in intestinal immune regulation[27]. Cao et al. [28] found through mouse and dog models that Bifidobacterium longum can scavenge elevated reactive oxygen species, reduce inflammatory factors, reshape the intestinal barrier function, and restore the regulatory and immune functions of the intestinal microbiota.

6.2 Microbiota Transplantation

Microbiota transplantation, also known as fecal microbiota transplantation, involves transplanting the functional intestinal microbiota from the feces of healthy individuals into the intestines of patients through various methods to re-establish a new intestinal microbiota. In the 4th century in China, fecal suspensions were used to treat food poisoning. During World War II, German soldiers used fresh camel feces to treat bacillary dysentery. In 1958, American doctors used microbiota transplantation to treat pseudomembranous colitis. In 1983, microbiota transplantation successfully cured Clostridium difficile infections[29]. The use of microbiota transplantation to treat microbiota dysbiosis caused by various reasons has currently become a research focus among domestic and international researchers.Studies have found that the colonization and infection of Clostridium difficile are influenced by the human intestinal microbiota. Imbalance of the intestinal microbiota leads to an increase in some bacteria, which can create conditions conducive to the germination, proliferation, and production of Clostridium difficile toxins. These toxins, in turn, will alter the integrity of the intestinal mucosa, further exacerbating Clostridium difficile infections. Adults and neonates infected with Clostridium difficile can correct microbiota dysbiosis through microbiota transplantation, inhibit the growth of Clostridium difficile, reduce the inflammatory response, and restore the integrity of the intestinal mucosa [30]. Intestinal microbiota disorders are an important cause of ulcerative colitis. Li et al. [31]found through animal experiments that microbiota transplantation can reshape the composition of the intestinal microbiota in ulcerative colitis and downregulate the NF-κB signaling pathway to improve intestinal inflammation in mice with ulcerative colitis.

6.3 Phage Therapy

Phages are viruses that infect bacteria and can be used for antibacterial purposes (as an alternative to antibiotics) or to regulate the composition of the microbial community. In addition, genetically modified phages can be used as "gene vectors" for the biosynthesis and degradation of nutrients and the genetic regulation of the intestinal microbiota [32]. Currently, phage therapy is mainly applied in Eastern Europe and Russia, has not been widely used yet, and its efficacy awaits further confirmation [33].

7. Applications of Restoring Dysbiotic Microbiota in the Treatment of Colorectal Cancer

7.1 Applications of Probiotics, Prebiotics, and Synbiotics

Chen et al.[34] intervened in male C57BL/6 mice with colorectal cancer induced by 1,2 - dimethylhydrazine (DMH) using two probiotics (Clostridium butyricum and Bacillus subtilis). By comparing the tumor incidence rate and detecting inflammation - and immunity - related markers, they found that Clostridium butyricum and Bacillus subtilis could inhibit the proliferation of colorectal cancer cells, cause cell cycle arrest, and promote apoptosis.Some research shows that Lactobacillus helveticus NS8 can regulate intestinal microbiota dysbiosis by promoting beneficial symbiotic microorganisms while inhibiting cancer - related microorganisms. NS8 can significantly inhibit the proliferation of early intestinal epithelial cells in colorectal cancer, increase the level of apoptosis, and also inhibit the activation of NF - κB and upregulate the anti - inflammatory cytokine IL - 10, thereby suppressing the inflammatory response [35].Other studies have found that Bifidobacterium longum can regulate certain

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oncogenic and tumor - suppressor miRNAs, thus inhibiting the proliferation, invasion, apoptosis, and cell cycle of tumor cells[36].Kotzampassi et al.[37] detected the efficacy of four probiotics (Lactobacillus acidophilus, Lactobacillus plantarum, Bifidobacterium lactis, and Saccharomyces boulardii) in preventing postoperative complications of colorectal cancer through a randomized clinical trial. The results showed that all four probiotics could significantly reduce the incidence of infections and anastomotic fistulas and significantly shorten the duration of mechanical ventilation.Existing studies have shown that probiotics, prebiotics, and synbiotics play important roles in maintaining colonic barrier function, metabolism, immune regulation, and inhibiting the proliferation of host tumor cells. Moreover, they can not only reduce the adverse reactions of chemotherapy drugs but also enhance the anti - tumor effects of chemotherapy drugs[38].

7.2 Applications of Microbiota Transplantation

Yu et al. [39] established a mouse model of colorectal cancer with microbiota dysbiosis and transferred the intestinal microbiota of healthy mice into the colorectal cancer mice. The results showed that the diameter and number of tumor lesions in the colorectal cancer mice were significantly reduced, and the survival period of the mice was significantly prolonged. This study indicates that microbiota transplantation can inhibit the development of colorectal cancer by reversing intestinal microbiota disorders, improving intestinal inflammation, and enhancing the anti - cancer immune response.Research has found that microbiota transplantation and anti - PD - 1 therapy have a synergistic effect in the treatment of colorectal cancer. The combined treatment of the two can better control the development of tumors and significantly prolong the survival period of mice [40].

8. Probiotics, Microbiota Transplantation and Prevention of Colorectal Cancer

Dong et al. [41] discovered through tumor animal models that Lactobacillus salivarius has anti - proliferative and pro - apoptotic effects. It can significantly inhibit tumor formation. Its metabolites can suppress the growth of tumor cells, arrest the cell cycle, and induce apoptosis, showing a significant preventive effect against colorectal cancer induced by dimethylhydrazine.Rosshart et al.[42] found through animal experiments that transplanting the intestinal microbiota of wild mice into experimental mice enabled the reconstituted experimental mice to better resist colon carcinogenesis induced by mutations or inflammatory effects.

9. Conclusion and Prospect

A large number of current studies have proven that intestinal microbiota dysbiosis is an important cause of colorectal cancer. Multiple methods such as the intake of probiotics, prebiotics, synbiotics, and microbiota transplantation can correct the disorder of the intestinal microbiota and maintain its balance. The most common probiotic therapy plays an important role in preventing the occurrence of colorectal cancer, delaying tumor progression, reducing postoperative complications of colorectal cancer, enhancing the efficacy of anti - tumor drugs and reducing their side effects. Future research can focus on the administration method, dosage of probiotic therapy, combination of multiple probiotics, and combined treatment with anti - tumor drugs. With the progress of research, microbiota transplantation is no longer just a simple alternative therapy. It has gradually become a biologically reasonable treatment method and is being accepted step by step. It has currently become a research focus for many researchers. Future research will focus on donor screening, microbiota preservation, administration methods and routes.

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