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The Role of Intestinal Flora and Immune System in the Development of Ulcerative Colitis: Insights from Animal Experiments and Molecular Pathways

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Description

Ulcerative colitis (UC) is a chronic inflammatory disease of the intestine that is characterized by recurrent symptoms. Its pathogenesis is attributed to environmental factors, genetic susceptibility, dysbiosis of the intestinal flora and autoimmunity. Recent advances in high-throughput and macrogenetic sequencing technologies have led to significant breakthroughs in understanding the role of intestinal flora in the pathogenesis and progression of UC. Intestinal flora performs various vital functions, such as secretion, nutrient synthesis and absorption, metabolism and maintenance of intestinal barrier integrity. Animal experiments have demonstrated that mice do not develop UC under germ-free conditions; however, transplantation of microbiota leads to colitis in mice, suggesting the significance of intestinal flora in UC pathogenesis. A balanced intestinal ecosystem is crucial for maintaining immune system function, including the gut-associated lymphoid tissue (GALT), which houses dendritic cells that activate the NF-κB signaling pathway, leading to a cascade reaction that affects UC development. Research indicates that activation of NF-κB and STAT3 triggers the release of inflammatory factors, such as IL-6 and TNF-α, that are closely associated with UC gut inflammation.

The intestinal flora and immune system are closely intertwined and play essential roles in maintaining human health. The intestine is home to a vast and diverse ecosystem of microorganisms collectively referred to as the intestinal flora or microbiota. This microbiota interacts with the intestinal mucosa and immune cells to promote the development and maintenance of the immune system. The intestinal flora is involved in the production of short-chain fatty acids (SCFAs), which can modulate the differentiation and function of immune cells, such as regulatory T cells (Tregs), promoting a healthy immune response. The microbiota can also stimulate the production of immunoglobulin A (IgA), an antibody that helps to prevent pathogens from adhering to the intestinal mucosa.

The gut-associated lymphoid tissue (GALT) is a key component of the immune system that protects the body against harmful pathogens. Dendritic cells in the GALT recognize and process antigens from the microbiota and present them to T cells, leading to the activation of the immune system. This process helps to maintain immune homeostasis and prevent the development of inflammatory diseases, such as inflammatory bowel disease (IBD). Alterations in the intestinal flora, such as dysbiosis, have been linked to the development of various autoimmune and inflammatory diseases. For example, in IBD, dysbiosis can lead to an imbalance between pro-inflammatory and anti-inflammatory immune responses, leading to chronic inflammation in the gut. Overall, the relationship between the intestinal flora and immune system is a complex and dynamic interplay that plays a vital role in maintaining human health.

Ulcerative colitis (UC) is a chronic inflammatory bowel disease characterized by ulceration and inflammation of the colon and rectum. It affects millions of people worldwide and its incidence is increasing in many countries. While the exact cause of UC is unknown, it is believed to be a complex interplay between genetic, environmental and immune factors. In recent years, researchers have begun to focus on the role of intestinal flora and the immune system in the development of UC, using animal experiments and molecular pathways to gain insight into this complex disease. The human gut is home to trillions of microorganisms collectively known as the intestinal flora or microbiota. These microorganisms play an essential role in maintaining gut health, including aiding digestion, producing vitamins and providing a barrier against pathogens. Disruption of the gut microbiota has been linked to various diseases, including inflammatory bowel diseases such as UC.

Animal experiments have been instrumental in revealing the complex interplay between intestinal flora and UC. For example, a study in mice found that an imbalance in the gut microbiota could lead to UC-like symptoms. Specifically, when researchers disrupted the balance of gut bacteria in mice by introducing pathogenic bacteria called Helicobacter hepaticus, the mice developed UC-like symptoms, including colonic inflammation and ulceration. Another study in rats found that treatment with antibiotics that targeted specific gut bacteria prevented the development of UC-like symptoms, providing further evidence of the role of intestinal flora in UC. The immune system also plays a critical role in the development of UC. The immune system is responsible for identifying and eliminating foreign pathogens and maintaining homeostasis in the gut. However, in UC, the immune system mistakenly attacks the gut lining, leading to inflammation and ulceration. Studies have shown that specific immune cells, such as T-cells, play a role in the development of UC. For example, a study in mice found that T-cells that target specific gut bacteria could lead to UC-like symptoms.

Molecular pathways have also provided insights into the role of intestinal flora and the immune system in UC. For example, studies have identified various signaling pathways that are dysregulated in UC. One such pathway is the nuclear factor kappa B (NF-κB) pathway, which is involved in inflammation and immune response. Studies have shown that dysregulation of the NF-κB pathway can lead to the development of UC-like symptoms. Another pathway that has been implicated in UC is the Toll-like receptor (TLR) pathway. TLRs are a type of protein that plays a role in recognizing pathogens and activating the immune response. Dysregulation of the TLR pathway has been linked to the development of UC. For example, a study in mice found that activation of the TLR pathway could lead to UC-like symptoms. Animal experiments and molecular pathways have provided valuable insights into the role of intestinal flora and the immune system in the development of UC. Disruption of the gut microbiota and dysregulation of immune pathways can lead to inflammation and ulceration in the gut, leading to UC-like symptoms. Further research in this area is needed to better understand the complex interplay between genetic, environmental and immune factors that contribute to the development of UC [1-5].

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Conflict of Interest

None.

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