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Immunology of Inflammatory Gastrointestinal Bowel Disease

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Introduction

Approximately 6 to 8 million people worldwide suffer with IBD, which includes Crohn's disease (CD) and ulcerative colitis (UC). IBD, which affects patients' quality of life and daily activities, is a chronic, progressive, relapsing, or remitting intestinal condition that raises healthcare costs. Although the specific pathophysiology of IBD is still largely unknown, it is widely acknowledged that it is brought on by an aberrant immune response against microbes in those with genetic susceptibility to it.

Untargeted treatments (such amino salicylates, glucocorticoids, and immunomodulators) and targeted biologic treatments (like anti-TNF, anti-IL-12/IL-23, and anti-47 integrin) are currently available for IBD. While up to 30% of patients do not respond to first treatment and up to 50% of patients lose their response over time, biologic medicines are beneficial in many people. A serious consequence for people with long-term IBD is intestinal fibrosis. Effective antifibrotic treatments are currently lacking, and the precise molecular mechanisms and pathways involved in the onset of intestinal fibrogenesis remain largely unknown [1].

About the Study

In order to better understand the relationship between symbiotic flora, intestinal epithelial cells (IECs), and the immune system as well as to highlight the immunological pathogenesis of inflammatory bowel disease (IBD), we set out to summarise this information. We also wanted to clarify how the intestinal immune system functions. We also studied the immunological pathophysiology involved in intestinal fibrogenesis and offered fresh anti-fibrotic immunotherapies for inflammatory bowel disease (IBD) [2].

The distal ileum and colon are home to the majority of the trillions of microorganisms that make up the human gut microbiota, which also includes fungus, protozoa, viruses, and archaea. By controlling innate immune system activation, affecting host energy metabolism, immunological homeostasis and development, and maintaining mucosal integrity, the gut microbiota plays a critical role in the aetiology of IBD. For example, Clostridium difficile can cause dendritic cells (DCs) and goblet cells to release TGF- and IL-10, producing enough signals to increase the Treg population. Additionally, in order to combat colitis, Bacteroides fragilis can increase the levels of anti-inflammatory cytokines and the Treg population. Additionally, by competing with the invaded pathogens for nutrients and space, the gut microbiota can create vital substances like vitamin K and short-chain fatty acids (SCFAs) and interfere with their growth. Loss of beneficial species, reduced microbial variety, and excessive development are the three categories under which microbiota dysbiosis can be categorised [3].

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The composition of the gut microbiota is altered in IBD patients, according to mounting data. Pseudomonas-like bacteria are identified in the tissues of CD patients; IBD patients have an altered gut micro biota. Additionally, Escherichia coli, a pathogenic bacteria, is more prevalent in the gut, has the ability to survive and reproduce in macrophages, and causes IBD by inducing the release of tumour necrosis factor (TNF-), which triggers an inflammatory response. Additionally, Faecalibacterium prausnitzii, a probiotic bacterium, can induce DCs to release the anti-inflammatory cytokine IL-10 and block the production of IL-12 and interferon (INF-), both of which were noticeably reduced in the guts of IBD patients. These researches have offered potential beneficial IBD treatments, even if they were unable to definitively show the connections between microbial dysbiosis and IBD. Meanwhile, Britton has observed that mice receiving faeces from IBD donors are more likely to develop colitis than mice receiving faeces from healthy donors. This finding was made after Britton colonised germ-free mice with intestinal microbiota from healthy and IBD donors. Since it is unknown what exactly is in donor faeces, the effectiveness of faecal microbiota transplantation (FMT) for the treatment of IBD has not been verified in clinical trials.

Immune cells

There are two types of intestinal immune cells: innate immune cells and adaptive immune cells, both of which have a significant impact on the immunological responses in IBD. The interaction of innate immune cells, including macrophages, dendritic cells (DCs), neutrophils, natural killer (NK) cells, and innate lymphoid cells (ILCs), results in the production of cytokines, chemokines, and antimicrobial agents that cause inflammation and activate the adaptive immune system through phagocytosis and antigen presentation [4].

Macrophages, DCs, neutrophils, NKT cells, and ILCs constitute the initial line of defence in the mucosal innate immune system. Innate immune receptors (PRRs), including Toll-like receptors (TLRs) and NOD-like receptors (NLRs), are present on both macrophages and dendritic cells (DCs) and are crucial for encouraging wound healing and establishing tolerance to specific infections. Certain pathogen-associated molecular patterns (PAMPs) of pathogens bind to these receptors, activating a number of signalling pathways and triggering the synthesis of pro-inflammatory cytokines, chemokines, and antimicrobial peptides. Additionally, they function as antigen-presenting cells (APCs), which connect innate immunity and adaptive immunity by secreting cytokines and presenting antigens to T cells. Healthy gut resident macrophages exhibit reduced responsiveness, proliferation, and chemotactic activity because they do not express CD14 [5].

Unlike innate immune cells, which lack high specificities and immunological memory capacities, adaptive immune cells work in concert with one another to neutralise invasive infections. T cells are important members of the adaptive immune response. The naive T cells are activated and differentiated into different subsets, such as effector, regulatory, and memory T cells with up-regulated specific homing receptors, such as chemokine receptors (CCR9 in the small intestine and CCR10 in the colon) and integrins like L2, 41, 47, and E7, when stimulated by antigens in the gut-associated lymphoid tissue Leukocyte migration to the inflamed gut is made possible by the binding of these receptors to cellular adhesion molecules (CAMs) expressed on endothelial cells of blood vessels.

Conclusion

IBD genetics has advanced significantly, and multiple related molecular and cellular pathways have been identified. Future therapies for IBD may be

based on changes at particular gene loci. Additionally, it is anticipated that FMT, drugs with a natural origin or derivation, new antibodies or inhibitors, combined therapy plans, and multifactor blockers would all help to alleviate the IBD therapeutic bottleneck. Therapeutic approaches that combine the use of medications with anti-inflammatory and medications with antifibrotic effects will also offer important insights into how IBD is currently treated.

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