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Crosstalk between Interleukins and Microbiota in Gut Immunity

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Introduction

The gastrointestinal tract serves as a central hub for immune regulation and microbial colonization, hosting trillions of commensal microbes and a dense network of immune cells. This intricate environment is governed by a dynamic interplay between the host immune system and the gut microbiota, ensuring a delicate balance between immune tolerance and defense. Among the most critical mediators of this immune-microbial dialogue are interleukins-a family of cytokines that regulate immune cell differentiation, activation, and communication. Interleukins play pivotal roles in shaping gut immunity by modulating epithelial barrier function, regulating inflammatory responses, and guiding T cell fate decisions. Conversely, the gut microbiota influences interleukin production and signaling, thus actively sculpting the immune landscape. Disruptions in this bidirectional communication are linked to a range of diseases, including inflammatory bowel disease, colorectal cancer, food allergies, and systemic autoimmune disorders [1].

Description

One of the most critical interleukins for maintaining gut homeostasis is IL-10, an anti-inflammatory cytokine produced by regulatory T cells, macrophages, and DCs. IL-10 suppresses excessive immune activation by inhibiting pro-inflammatory cytokine production, antigen presentation, and effector T cell responses. Mice deficient in IL-10 or its receptor develop spontaneous colitis due to uncontrolled microbial-induced inflammation. In humans, mutations in the IL-10 pathway are associated with early-onset IBD. The microbiota plays a pivotal role in driving IL-10 production. Certain bacterial species, such as Bacteroides fragilis and Clostridia clusters IV and XIVa, induce IL-10-secreting Tregs via microbial metabolites like short-chain fatty acids (SCFAs), including butyrate and propionate. These SCFAs enhance histone acetylation at the Foxp3 locus, promoting Treg differentiation and IL-10 expression, thereby reinforcing mucosal tolerance [2].

Microbiota-derived metabolites, including SCFAs, tryptophan catabolites, and secondary bile acids, modulate interleukin production and immune cell function. SCFAs, as mentioned, promote IL-10 production and Treg induction. Tryptophan-derived metabolites, sensed by the aryl hydrocarbon receptor (AhR), enhance IL-22 production by ILC3s and T cells, promoting epithelial repair and mucosal defense. Dysbiosis can lead to altered metabolic outputs, diminishing these protective interleukin responses and increasing susceptibility to infection and inflammation. Microbes also directly influence interleukin receptor expression. For example, SFB colonization upregulates IL-17RA and IL-22RA on intestinal epithelial cells, enhancing responsiveness to cytokine signaling. Pathogens may subvert this system by downregulating interleukin receptors or producing immunomodulatory molecules that antagonize cytokine signaling [3].

Modulating microbial communities or interleukin signaling holds promise for cancer prevention and therapy. In systemic autoimmune diseases such as Rheumatoid Arthritis (RA) and Systemic Lupus Erythematosus (SLE), gut dysbiosis alters cytokine production, skewing toward pro-inflammatory profiles. Mouse studies demonstrate that specific microbial signatures can induce IL-17 and IL-6 in the gut, contributing to systemic inflammation. Restoring microbial balance through probiotics or Fecal Microbiota Transplantation (FMT) has been shown to reduce disease severity by re-establishing homeostatic interleukin responses [4,5].

Conclusion

The intricate crosstalk between interleukins and the gut microbiota orchestrates the balance between immune tolerance and defense, playing a central role in gut health and disease. Interleukins such as IL-10, IL-17, IL-22, IL-6, and IL-23 act as key mediators in this bidirectional dialogue, responding to microbial cues and shaping immune outcomes. Disruption of this finely tuned communication can lead to chronic inflammation, infection, or malignancy, as seen in IBD, colorectal cancer, and systemic autoimmune diseases. As our understanding of the interleukin-microbiota axis deepens, novel therapeutic strategies are emerging that harness this interaction to restore mucosal homeostasis and treat disease. From biologics and dietary modulation to microbiota engineering, targeting the immune-microbial interface represents a promising frontier in precision medicine. Ultimately, appreciating the symbiotic relationship between our microbial residents and cytokine networks is essential to maintaining gut integrity and systemic immune balance.

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Conflict of Interest

None

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