The Role of IL-23 in Crohn's Disease

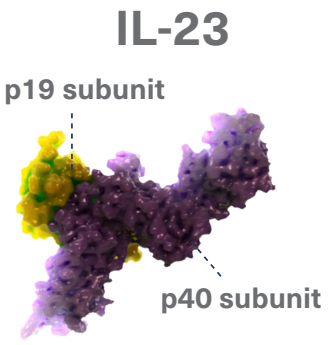
About Interleukin-23

Interleukin-23 (IL-23) is a key signaling molecule (also known as a cytokine) involved in inflammatory processes.[1] It is thought to be linked to a number of chronic immune-mediated diseases, including Crohn's disease.[1,2]

As a member of the IL-12 family of cytokines, IL-23 has pro-inflammatory properties[2]

IL-23 is composed of two subunits – p40, which is shared with IL-12, and p19, which is unique to IL-23[1]

IL-23 is produced by immune cells that survey the intestinal environment, such as dendritic cells and macrophages, in response to signals in the gut[1,2]



About IL-23 and Immune Functions

IL-23 is produced by a network of immune cells in the gut.[1,2]

When IL-23 binds to its receptor, it signals through the JAK-STAT pathway via STAT3 and activates inflammation-inducing functions of T helper 17 (Th17) cells.[1,2]

Once activated, Th17 cells release pro-inflammatory cytokines, including IL-17A and IL-17F, TNFα, IL-22, IL-26, and interferon gamma, which trigger inflammation.[2–4]

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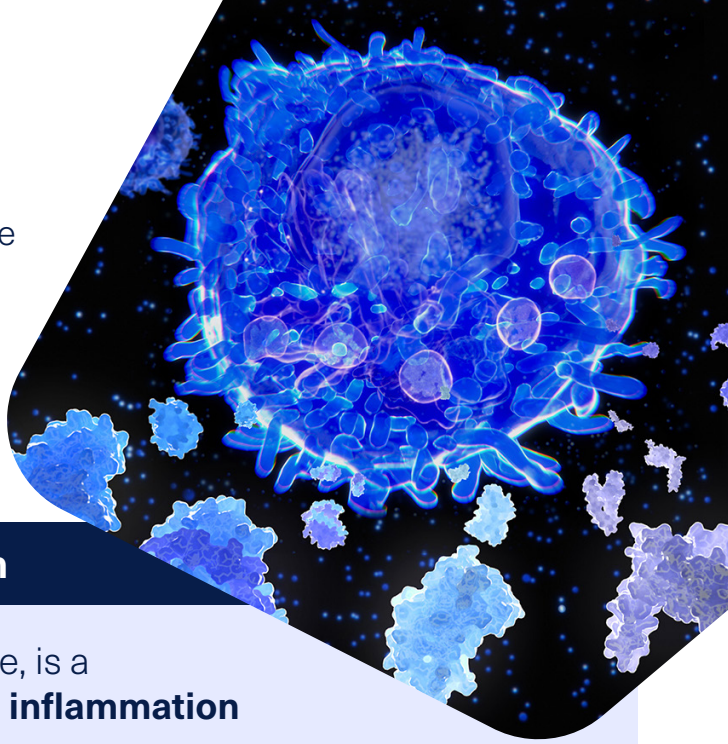
Targeting IL-23 to Inhibit Inflammatory Signaling

Selectively targeting the IL-23 pathway by inhibiting the p19 subunit can reduce inflammation-causing Th17 cells, thereby decreasing inflammatory cytokines involved in the development and progression of Crohn's disease, reducing symptoms and potentially helping patients reach remission.[2–4]



Crohn's Disease and the Immune System

Crohn's disease, an inflammatory bowel disease, is a **chronic, systemic disease that manifests as inflammation in the gastrointestinal tract.**[5,6]

Levels of IL-23 are higher in people with Crohn's disease, especially during flares, and people with mutations in the IL-23 receptor are more likely to develop Crohn's disease.[1,2,7]

Increased IL-23 signaling **drives chronic inflammation, as activated Th17 cells promote inflammation** and are poorly responsive to anti-inflammatory signals, and IL-23 suppress T-cells which typically regulate inflammation.[1,2]

References

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