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The Molecular and Cellular Basis for Inflammatory Bowel Diseases (IBD)

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Message from the Guest Editors

In the last few years, substantial progress has been made in understanding the pathogenetic mechanisms of inflammatory bowel disease (IBD), specifically Crohn's disease (CD) and ulcerative colitis (UC). Recent works have examined the concept that IBD could result from dysregulation of the intestinal barrier and a pathologic activation of the intestinal immune response toward several bacterial or viral antigens. This has been translated into newer, more effective therapies—biologic and molecular therapies—that have decreased the occurrence of flares, led to remission in more patients, and improved patients' quality of life.

To date, several factors have been proposed to be involved in the pathogenesis of IBD, including antigen presentation and balance between the different T-cell subpopulations, altered microbiota, anomalies of immune regulation, and phagocytosis; nevertheless, new cellular and molecular targets are under investigation and novel therapeutic approaches have been developed accordingly.



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Special Issue



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