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EDITORIAL

What's hot in inflammatory bowel disease in 2011?

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Abstract

Ulcerative colitis and Crohn's disease (CD) are the two major forms of inflammatory bowel disease (IBD). In this highlight topic series of articles, the most recent advances in the IBD field are reviewed, especially the newly described cytokines, including the therapeutic implications for their manipulation. In addition, the interplay between the intestinal microbiota and the host is reviewed, including the role of defensins and dysbiosis in CD pathogenesis. Finally, the importance of the non immune systems such as endothelial cells and the hemostatic system are highlighted as new players in IBD pathogenesis.

Key words: Crohn's disease; Ulcerative colitis; Inflammatory bowel disease; Immunology; Pathogenesis

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In this special issue of *World Journal of Gastroenterology* (*WJG*), we have selected an expert group that is actively involved in the investigation of inflammatory bowel disease (IBD) pathogenesis.

Crohn's disease (CD) and ulcerative colitis (UC) are the two major forms of IBD.

These diseases still pose major clinical and therapeutic challenges to the gastroenterological comunity.

It is now clear that CD and UC represent two distinct forms of chronic inflammation of the gastrointestinal tract and have different causes and pathogenic mechanisms. Still, the factors underlying the appearance of both CD and UC are roughly the same, and include a temporal association with progressive changes in the environment, an intrinsic genetic predisposition, the existence of a rich enteric flora, and an abnormal immune reactivity which is ultimately responsible for damaging the gut and causing clinical manifestations. Even though the categories of underlying factors are roughly the same, there are variations in each category as well as differences in how the underlying factors interact. The end result is two related but distinct disorders named CD and UC. In this special issue of *WJG*, differences and similarities of the etiopathogenic factors in each form of IBD will be illustrated and discussed in each review assessing the newly described cytokines[1], the interplay between the intestinal microbiota and the host[2], the role of defensins and dysbiosis[3] and the importance of extraluminal factors[4] and non immune systems such as endothelial cells[5] and the hemostatic system[6] as new players in IBD pathogenesis.

Since the recognition of IBD as a perplexing and challenging clinical entity, the investigation of its pathogenic mechanisms has gone through repeated cycles of new hopes, new knowledge, and new realities. Infectious, allergic, dietary, psychosocial, environmental, microbial, vascular, metabolic, immune and other basic theories have been put forward, most of them to be rebuked, if not ridiculed. At the moment, we appear to have settled down on a unifying but still wide-ranging hypothesis that IBD results from complex interactions between evolv-

ing environmental changes induced by society progress, a still undefined number of predisposing genetic mutations, an incredibly complex gut microbiota that may be constantly varying, and the intricacies of individual immune systems. The ability to integrate all these various components into a single cohesive and logical pathway of disease that explains all aspects of IBD appears still a bit distant at the moment. On the other hand, if we look back at where we stood only two or three decades ago, the progress achieved in our understanding of IBD pathogenesis and the way it has changed our approach to therapy is just short of spectacular.

Although we have made tremendous advances in disease pathogenesis, among the many diseases that exist, IBD is the one for which the exact etiology remains obscure and the mechanisms underlying tissue injury appear to be exceedingly complex. This certainly seems to be the case for the two main forms of IBD, namely CD and UC.

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