

What's new in inflammatory bowel disease in 2008?

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Abstract

Ulcerative colitis and Crohn's disease represent the two major forms of inflammatory bowel disease. In this highlight topic series of articles we cover the latest developments in genetics and epidemiology, intestinal physiology, mucosal immunology, mechanisms of epithelial cell injury and restitution, current medical therapy, modern surgical management, important extra-intestinal complications such as primary sclerosing cholangitis, cholangiocellular carcinoma and autoimmune hepatitis as well as endoscopic and molecular screening, detection and prevention of small bowel and colorectal cancer.

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Key words: Crohn's disease; Ulcerative colitis; Inflammatory bowel disease; Immunology; Genetics; Epidemiology; Medical therapy; Surgery; Cancer; Extra-intestinal manifestations

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In this special issue of the *World Journal of Gastroenterology* we have put together a group of expert faculty from all over the world to cover inflammatory bowel disease as a highlight topic.

Ulcerative colitis and Crohn's disease represent the two major forms of inflammatory bowel disease. Crohn's disease was first observed by the German surgeon Wilhelm Fabry (aka Guilihelmus Fabricius Hildanus) in 1623, and was later described by and named after the New York physician Dr Burril B Crohn^[1,2]. Ulcerative colitis (UC) was first described

by the London physician Sir Samuel Wilks in 1859^[3].

Much has been learned since these early days about the etiology and particularly the genetic predisposition for these two disorders. The first paper in our series reviews the current knowledge about the etiology of inflammatory bowel disease. While all genome wide scans in the past have identified increased susceptibility genes, the most recently discovered susceptibility allele (IL23R) protects from Crohn's disease. The first article in our series puts this discovery in genetics in perspective with disease mechanisms and other etiological factors and its potential impact on the development of novel therapeutic strategies^[4].

The most widely accepted hypothesis for the cause of inflammatory bowel disease is a disturbed interaction of the host immune system with the commensal microflora and other luminal antigens. This interaction begins at the epithelial layer and extends below to a multitude of other immune cells of the mucosal interface. Ultimately, mature and activated antigen presenting cells, (i.e. dendritic cells) induce and probably perpetuate an imbalance of effector and regulatory T-cell. Crohn's disease is a mainly a Th1 and Th17 mediated process, while ulcerative colitis appears to be predominately mediated through Th2 and NK T-cells. Two papers review the effects of these components in the immunopathogenesis of IBD^[5,6].

Once the inflammation has occurred and is perpetuated by the dysfunctional immune system, the clinician is faced with distinct clinical phenotypes. Fortunately, the integrity of the gastrointestinal surface epithelium is rapidly reestablished even after extensive destruction. Rapid resealing of the epithelial barrier following injury is accomplished by a process termed epithelial restitution, followed by more delayed mechanisms of epithelial wound healing including increased epithelial cell proliferation and epithelial cell differentiation. Restitution of the intestinal surface epithelium is modulated by a range of highly divergent factors among them a broad spectrum of structurally distinct regulatory peptides, variously described as growth factors or cytokines^[7].

Recent advances in our understanding of the pathophysiology of inflammation and in bioengineering have led to new therapeutic concepts targeting almost every aspect of the inflammatory process and help with the restitution of mucosal integrity. This provides clinicians with the opportunity of selecting from and combining conventional compounds and concepts as well as modern biologics and novel approaches to regimens tailored to the individual patient's needs^[8].

Special issues in pediatric inflammatory bowel disease are discussed in a separate paper^[9].

Surgery has become an integral part of the management of inflammatory bowel disease and should not be considered a failure of medical management. Modern surgical techniques including minimally invasive procedures and current issues of surgical management are reviewed^[10].

Inflammatory bowel disease is a systemic illness, not limited to the gastrointestinal tract. A substantial fraction of patients develops extra-intestinal manifestations, which itself can cause additional morbidity and complications. Hepatic manifestations and especially primary sclerosing cholangitis are among the most common, sometimes overlap and cause diagnostic and therapeutic challenges^[11].

Finally, patients with chronic inflammatory conditions, including inflammatory bowel disease are prone to develop malignant complications. The final paper reviews current knowledge about the incidence and prevention of cancer^[12].

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