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Recent insight into pathogenesis of inflammatory bowel disease for novel therapeutic strategies

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The activation of intestinal epithelial, innate and adaptive immune cells through Pattern Recognition Receptors (PRRs) are generally lead to defense mechanism or chronic inflammatory disorders and respond through secretion of pro-inflammatory cytokines and chemokine in host immune responses. The gastrointestinal tract is constantly exposed to wide range of antigens including enteric bacteria and foods but gut homeostasis is maintained and controlled by suppressing excessive immune responses to foreign antigens. In both innate and adaptive immunity, the disruption of regulatory mechanisms may result in inflammatory immune responses to enteric antigens and cause chronic intestinal inflammation such as Inflammatory Bowel Disease (IBD). Dysfunction of Toll-like receptors as a PRRs prototype is associated with infectious and inflammatory diseases, for which therapeutic management with PRPs antagonists demonstrate a promising drug strategy. IBD patients are mostly treated with anti-inflammatory and immunosuppressive drugs, antibiotics, and biological therapies and/or surgery. Oral medicinal plants-derived compound can be a future promising to the prevention and treatment of IBD. Better understanding of the etiopathology basis of IBD lead to the development of novel targeted therapeutics. Experimental animal models can be representative of human IBD and provide new insights into the role of the pathways, cells and molecules which is essential for intestinal homeostasis. Animal models of IBD (genetic, chemical or induced by immune cell transfer) are particularly useful to study the contribution of innate immune mechanisms of IBD and reveal the mechanisms of intestinal pathologies. Experimental animal models (gene targeting approaches and/or chemical models of colitis) of intestinal inflammation have studied the role of the epithelial barrier in intestinal homeostasis to demonstrate epithelial mechanisms deregulated in IBD. Association between IBD and genes that control immune pathways such as TLR, RAGE (Receptor of advance glycation end product), nucleotide oligomerization domain (NOD)-like receptor (NLRs) genes that regulate inflammatory response (NF-KB, AP1 and IRFs) and genes in the T helper cells pathway indicate the significant roles of host-microbe interactions in regulating intestinal immune hemostasis. Inflammatory cell infiltrates were dominated by abundant lymphoid follicles, large numbers of DC and granulocytes, CD4+ T cells and granulocytes and increased expression of inflammatory cytokines and chemokines (IL-6, IL-11, IL-12, IL-18, IFN-γ, COX-2) in colitis mice. Active compounds derived from natural compounds were also found with unique features as PRPs antagonists. Recent evidences encourage further research works on characterization for these compounds, which will become promising drug candidates in PRPs-based therapy in the future.

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