# Autacoids in Inẑammatory Bowel Diseases

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## Editorial

Inẑammatory bowel diseases (IBD), including Crohn's disease and ulcerative colitis (UC), are complex disorders characterized by chronic, local and systemic inẑammation and spontaneously relapsing course. The causes of these diseases are unknown, however they display genetic and environmental components and appear to be immunologically mediated in part by enteric microbiota [1]. The immune system cells activation may lead to activation of plasma proteolytic cascades [1,2], as well as to release of inẑammatory mediators in inẑamed intestine. There are convincing evidences that IBD are diseases of immunological hyperresponsiveness within the mucosa. Immunological reactions may be directed against luminal bacteria and their products normally present in the intestine [2]. Both innate and adaptive immune response play a role in IBD pathogenesis and possibly in etiology. Autacoids, a local hormones e.g., histamine, serotonin, kinins, angiotensin, eicosanoids, neurotensin, nitric oxide, endothelins may be produced and act locally in IBD – inẑamed intestine, and play a role in inẑammatory response and progression in both Crohn's disease and UC. A role of eicosanoids and nitric oxide in IBD was relatively well delineated. Much less attention has been paid to other autacoids, although a signiẐcance of the intestinal tissue kallikrein – kinin system in IBD has been investigated in the late 1990s – 2000s. Histamine,

area and intracellularly in inẑammatory tissue. In contrast, B1R protein is found in the basal area of enterocytes in normal intestine, but in the apical portion of enterocytes in inẑamed tissue. B1R protein is signiẐcantly increased in both active UC and CD intestines compared to controls. In patients with active UC, B1R mRNA is signiẐcantly higher than B2R mRNA. However, in inactive UC patients, the B1R and B2R mRNA did not diwer signiẐcantly. Hxus, bradykinin receptors in IBD may reẑect intestinal inẑammation. Increased B1R gene and protein expression in active IBD provides a structural basis of the important role of bradykinin in chronic inẑammation. Latter Hara et al. [16] shown that upregulation of B1R in the trinitrobenzene sulphonic acid (TNBS) – induced colitis model is in part dependent on NF- κ β activation. Kininogen has been demonstrated in both normal and inẑamed human colon, thus, ITK can generate kinins. In addition, kallistatin, a major tissue kallikrein inhibitor and kininase II, a kinin inactivator, have been demonstrated in intestinal tissue [11]. Our data from human study showing alteration in distribution of BR1 and signiẐcantly increased its levels in the patients with IBD tend to corroborate with experimental Hara et al. results suggesting that selective B1R receptor antagonist may have potential in therapeutic trial. It should be noted that kinins are implicated in the regulation of blood pressure, sodium homeostasis and the cardioprotective ewect of preconditioning. Angiotensin-converting enzyme (kininase II) inhibition increase blood levels of bradykinin and kallidin peptides [17]. Hxus,