Inflammatory bowel disease (IBD) includes ulcerative colitis (UC) and Crohn disease (CD), which are characterized by specific clinical, pathologic, and endoscopic features in the gut. Although the etiology of IBD is unclear, the involvement of immune deregulation and release of inflammatory mediators leading to intense inflammatory reactions is widely accepted. The mechanism by which immune deregulation initiates and perpetuates intestinal damage is unknown; however, a growing body of evidence suggests an association of chronic intestinal inflammation with enhanced production of reactive oxygen species (ROS) and oxidative stress in IBD [1,2].

5-Hydroxytryptamine (5-HT, serotonin) is a gut cytotoxin synthesized by serotonergic neurons and enterochromaffin cells (ECs). Despite its presence and regulatory action in various body parts, including the brain and blood vessels, approximately 95% of 5-HT is synthesized and localized to the intestines, where it maintains trans-epithelial resistance by regulation of tight-junction proteins in cultured human mammary epithelial cells [3]. Numerous studies have shown changes in 5-HT content in IBD patients. 5-HT-positive neuronal fibers and mucosal cryptand cryptoxyl hydroxylase-1, the rate-limiting enzyme in 5-HT biosynthesis, were significantly increased incolonies of patients with IBD [4-6]. There is also another report that rectal biopsy specimens obtained from patients with IBD exhibited significantly reduced 5-HT content and EC count compared to healthy controls [7]. Because the amount of normal tissue is reduced in IBD, especially in severe ulcerative colitis, it could well be that the measured 5-HT that is normally present in healthy cells is reduced. These findings seem to show correlation with the level of epithelial damage during...