




Human study

Glutathione levels in chronic inflammatory disorders of the human colon

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Abstract

Glutathione depletion has been described in tissues obtained from several chronic diseases. Increased free radical production by inflammatory cells occurs in inflammatory bowel disease. We hypothesized that this could induce depletion of gut antioxidants. In this study, we examined the potential relationship between chronic inflammation and colonic glutathione levels. Using a validated assay, glutathione levels were determined in the mucosal-submucosal layer and the muscularis externa layer in surgical colonic specimens from 26 patients with ulcerative colitis, 14 patients with Crohn's colitis, and 10 patients who underwent partial colectomy for non-obstructive neoplasia. Inflammation was graded histologically. Glutathione levels were decreased in the muscularis externa and in the mucosal-submucosal layers from both ulcerative colitis and Crohn's colitis (both $p < 0.05$). There were parallel declines of glutathione levels in the muscularis externa layer compared to the mucosal-submucosal layer from individual colonic specimens. In ulcerative colitis, glutathione levels were reduced in histologically active disease compared to inactive disease (in the mucosal-submucosal layers: Mean \pm SEM were 214 ± 68 nmol/g wet tissue and 808 ± 30 , respectively; in the muscularis externa layers: 333 ± 97 and 890 ± 340 ; both $p < 0.05$). In Crohn's colitis, there were no significant differences between histologically active and inactive disease (in the mucosal-submucosal layers: 114 ± 53 and 461 ± 206 ; in the muscularis externa layers: 105 ± 59 and 553 ± 211 ; both $p > 0.05$). This study provides evidence that chronic inflammatory disorders of the colon are associated with glutathione depletion. In ulcerative colitis, there was a relationship between the severity of inflammation and glutathione depletion. By contrast, this relationship was not significant in Crohn's colitis. The results suggest that there could be a primary defect in glutathione production in Crohn's colitis, or a difference in the relative levels of free radical production by inflammatory cells present in these two disorders of colonic inflammation.

