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Study of histone 3 acetylation in patients with Crohn's disease

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There was very little study on understanding fibrotic intestinal pathology in patients with Crohn's disease since most studies focused mainly on inflammatory pathway. In murine colitis study, it had been showed that the elevation of transforming growth factor (TGFβ) can lead to increased activation of fibroblast and increased secretion of extracellular matrix protein (ECM). Epigenetic mechanisms involving histone modification is proven to play an important role in intestinal inflammation. Increased in histone deacetylase activity (HDAC) was found in many inflammatory conditions such as arthritis and cancer. There are numbers of HDACis (histone deacetylase inhibitors), which result in acetylation of cell, which is essential for gene expression. Few recent studies showed that in murine model of inflammatory colitis, HDAC inhibitor can reduce the overall inflammatory symptoms. The data is lacking in human and hence, this study was performed based on the hypothesis that histone acetylation will be low in the mucosa overlying a stricture area of CD patients compared to a non-stricture area in the same patients. The first aim was to measure and compare the level of histone acetylation in the stricture mucosal area of CD patients compared to a non-stricture mucosal area. The second aim was to see whether or not HDACi can reduce the expression of collagen gene of mRNA in the intestinal fibroblast. Hypothesis is that histone acetylation will be low in the mucosa overlying a stricture area of CD patients compared to a non-stricture area in the same patients. The results supported the hypothesis and were consistent with previous experiment done in murine studies. We are getting closer to achieve our goal of understanding histone acetylation in the CD bowel and this could lead potentially to novel therapeutic strategies for IBD.

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