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Characterizing CXCR3 in inflammatory bowel disease: Small molecule inhibition of CXCR3 attenuates experimental model of Crohn's disease

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Inflammatory bowel disease (IBD) is a group of disorders characterized by idiopathic chronic inflammation of the intestine. Though IBD affects millions of individuals in the US and is responsible for billions of health care dollars, there is very limited treatment and no cure for the disease. Previous investigators have implicated the importance of the chemokine receptor CXCR3 in the propagation of IBD, as evidenced by the increased expression of its ligands in diseased tissue. Our work aims to discover the expression profile of CXCR3 and its ligands CXCL9, CXCL10, and CXCL11 and whether a small molecule inhibitor of CXCR3, AM487, can attenuate the murine model of Crohn's disease, a subset of IBD. Mice were treated with the CXCR3 small molecule inhibitor AM487 in order to evaluate the expression profile of CXCR3 and its ligands and the cytokine phenotype of the cells expressing CXCR3. CXCR3 is expressed preferentially by inflammatory T cells in the gut, and these CXCR3+ T cells, and its ligands are significantly increased in disease, at the site of inflammation. The small molecule inhibitor AM487 is capable of attenuating the severity of disease in the murine model of Crohn's disease. CXCR3+ T cells play an important role in potentiating inflammation in the gut. A better understanding of its expression profile will allow for more specific and effective methods of treating Crohn's disease. We show that small molecule inhibition of CXCR3 is capable of mitigating disease severity in our model of IBD.

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