



Microbiota-derived short-chain fatty acids promote Th1 cell IL-10 production to maintain intestinal homeostasis

The gut microbiota has been shown to play important roles in maintaining intestinal homeostasis. However, inappropriate immune responses against these bacteria can spur IBD, including crohn's disease and ulcerative colitis. Whether these bacteria elicit immunopathology depends on the T cell responses induced by the bacteria. Great efforts have been made in understanding the development of those T-cells and the mechanisms involved. However, how pathogenic T cells are regulated following differentiation is relatively unclear.

Using a mouse model of colitis Dr. Yingzi Cong and his colleagues from The University of Texas determined that short-chain fatty acids (SCFAs) produced by gut bacteria as fermentation products of dietary fiber, promoted Th1 cell production of IL-10 mediated by G-protein coupled receptor 43 (GPR43).

To confirm this finding the authors fed mice SCFAs which promoted intestinal T cell IL-10 production and inhibited colitis development. More importantly, SCFAs also promoted IL-10 production of human T cells from healthy individuals as well as from IBD patients, which highlights the translational potential for SCFAs as therapeutic target in treatment of IBD.

The authors used Bio X Cell's anti-mouse IL-10R antibody (clone 1B1.3A) to block IL-10 signaling *in vivo* and show that SCFA treated Th1 cells limit intestinal inflammation through increased IL-10 production.

Read the full article in Nature Communications: <https://www.nature.com/articles/s41467-018-05901-2>