

Research may provide new targets for IBD therapies

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Modifying the small white blood cells that protect against disease might help treat immune disorders, according to a study¹ published in *Cellular and Molecular Gastroenterology and Hepatology*, the basic science journal of the American Gastroenterological Association. Specifically, researchers found that modulation of B lymphocyte function may be a means of regulating T lymphocyte function to treat immune-mediated disorders, including inflammatory bowel diseases (IBD).

Researchers uncovered the following pathway: [gut bacteria](#) stimulate intestinal B lymphocytes to release interleukin (IL)-10 that, in turn, induces development of regulatory T lymphocytes that prevent excessive inflammatory responses and limit immune-mediated disease. This signaling depends, in part, on IL-27, a member of the IL-12 cytokine family that has been linked to IBD.

"Our study elucidates previously unexplored intercellular signals by which gut microbiota regulate the mucosal immune system to prevent disease," said lead study author Yoshiyuki Mishima, MD, PhD, University of North Carolina, Chapel Hill. "These findings potentially could be exploited to treat patients with IBD."

The role of B lymphocytes in producing protective antibodies that are secreted into the intestine is well-recognized. However, the contributions of B lymphocytes and their secreted products (other than antibodies) are not well understood. This mouse study shows that IL-10 and IL-27, which are secreted by B lymphocytes, regulate development of regulatory T lymphocytes.

"The work provides new insight into mechanisms by which gut bacteria drive mucosal immune homeostasis," added Jerrold R. Turner, MD, PhD, AGAF, editor-in-chief, *Cellular and Molecular Gastroenterology and Hepatology*.

More information: Mishima, Yoshiyuki, et al. Resident Bacteria-Stimulated Interleukin-10-Secreting B Cells Ameliorate T-Cell-Mediated Colitis by Inducing T-Regulatory-1 Cells That Require Interleukin-27 Signaling, *Cellular and Molecular Gastroenterology and Hepatology* 2015; 1(3): 295-310, www.cmghjournal.org/article/S2009-00036-3/fulltext

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