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Boosting the gut barrier offers hope for Crohn's disease

Researchers at the University of Gothenburg, Sweden, have uncovered a critical mechanism that may pave the way for innovative treatments for Crohn's disease. Their findings center on repairing the protective barrier of the gut to slow disease progression.

The research is based on tissue samples from adult patients with Crohn's disease as well as experiments conducted on mice. The results provide a fresh perspective into the early mechanisms underlying Crohn's disease.

"By reinforcing the gut's protective barrier, we might prevent bacteria from invading the epithelial cells that line the intestine, which could halt both the onset and progression of the disease," says Thaher Pelaseyed, Associate Professor at Sahlgrenska Academy, University of Gothenburg.

A barrier against bacteria

Epithelial cells that line the small intestine are protected by a carbohydrate-rich layer called the glycocalyx, which prevents bacteria from coming in direct contact with the cells. A key component of this barrier is the transmembrane mucin MUC17, which forms a dense protective network on the surface of the epithelial cell.

The results, published in Journal of Clinical Investigation Insight, propose a novel approach to treat Crohn's disease by strengthening the gut's natural defenses rather than merely treating symptoms.

"Understanding how MUC17 is transported within intestinal epithelial cells to ultimately form the protective glycocalyx on the cell surface allows us to identify new drug targets that repair the glycocalyx barrier in Crohn's disease. This approach could reduce the need for traditional anti-inflammatory treatments, which often have limited efficacy and carry a risk of resistance," explains Thaher Pelaseyed.

Transport Disruption Weakens the Barrier

The study shows that patients with Crohn's disease have reduced levels of MUC17 on the surface of the epithelial cells, leaving cells vulnerable to bacterial attacks. Experiments on mice lacking the equivalent protein, showed that bacteria—both harmful and commensal— could more easily reach intestinal cells, causing cell death and inflammation.

Further investigations identified that the perturbation in MUC17 levels is not due to reduced production of the protein but rather to disrupted transport and stability within the intestinal epithelial cells. In a complementary study, published in Biochemical Journal, the team identified three key proteins—MYO1B, MYO5B, and SNX27—that are essential for positioning and stabilizing MUC17 on the surface of the epithelial cells. When these processes fail, the glycocalyx is impaired, allowing bacteria to invade intestinal tissue more easily.

Facts about Crohn’s Disease

Crohn’s disease is a chronic inflammatory bowel disease that commonly affects the small intestine and colon. Symptoms often include bloody diarrhea, abdominal pain, and weight loss, though complications outside the intestine can also occur.

Globally, Crohn's disease is estimated to affect up to 300 per 100,000 people, with the highest prevalence in North America and Europe.

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(Biochemical Journal): The MYO1B and MYO5B motor proteins and the sorting nexin SNX27 regulate apical targeting of membrane mucin MUC17 in enterocytes, https://doi.org/10.1042/BCJ20240204

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photo: Malin Arnesson