Management to prevent the structural weakening of the colonic mucus barrier in gastro-digestive diseases.

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Introduction

Despite the well-established health advantages of dietary fibre consumption, little is known about the processes by which fibre deficiency affects the gut microbiota and changes disease risk. We investigated the functional interactions between dietary fibre, the gut microbiota, and the colonic mucus barrier, which serves as a primary defence against enteric pathogens, using a gnotobiotic mouse model in which animals were colonised with a synthetic human gut microbiota composed of fully sequenced commensal bacteria. The gut bacteria turns to host-secreted mucus glycoproteins as a nutrition source during chronic or intermittent dietary fibre shortage, causing the colonic mucus barrier to erode. Dietary fibre deficiency, combined with a fiber-deficient, mucus-eroding microbiota, promotes mucosal pathogen epithelial access and fatal colitis and *citrobacter rodentium* [1]. Our findings highlight complex relationships between nutrition, the gut microbiome, and intestinal barrier dysfunction, which could be used to enhance health through dietary therapies.

Goblet cells create the intestinal mucus layer, which is a key component of epithelial protection. Goblet cells of the intestine are thought to be a homogenous cell type. However, in this investigation, we found many distinct goblet cell populations that follow two separate differentiation trajectories, as well as their individual gene and protein expression profiles. Intercrypt goblet cells (icGCs), which are found on the luminal surface of the colon, secrete mucus that differs from the mucus secreted by crypt-residing goblet cells. Mice with faulty icGCs were more sensitive to chemically induced colitis and developed spontaneous colitis as they grew older. In addition, patients with both active and remissive ulcerative colitis had mucus changes and fewer icGCs, highlighting the relevance of icGCs in sustaining functional colitis protection of the epithelium [2].

The inner mucus layer of the colon protects us from infections and commensal-induced inflammation, and it has been proven to be faulty in those with active UC. The goal of this study was to figure out what was causing the compositional changes, what was causing them, and how they might be contributing to UC pathogenesis. Sigmoid colon biopsies were taken from individuals with UC with continuous inflammation (n=36) or in remission (n=28) and 47 patients without colonic illness in this single-center case-control research. Ex vivo mucus samples were taken from biopsies, and the protein content was determined using nanoliquid chromatography-tandem mass spectrometry. A subset of individuals had their mucus penetrability and goblet cell responses to microbial stimulation tested [3].

A small group of 29 secreted/transmembrane proteins were discovered to make up the core mucus proteome. Major structural mucus components, notably the mucin MUC2, were reduced in active UC, even in non-inflamed segments (p0.0001). Active UC was linked to a reduction in sentinel goblet cell populations and a weakening of the secretory response of goblet cells to microbial challenge. In a sample of UC patients (12/40; 30%), abnormal penetrability of the inner mucus layer was noted. The SLC26A3 apical membrane anion exchanger, which delivers bicarbonate necessary for colonic mucin barrier production, was found to be reduced in penetrable mucus samples. In active UC, structural components of the core mucus were diminished [3]. These changes were linked to a reduction in the secretory response of goblet cells to microbial challenge, although they occurred independently of local inflammation. Mucus abnormalities are thus expected to play a role in UC aetiology.

Gut bacteria can influence the colonic mucus layer, a physical barrier that separates trillions of gut bacteria from the host, and diet has a big impact on gut microbiota composition. The relationship between a WSD, gut microbial composition, and the intestinal mucus layer, on the other hand, is less obvious. The colonic microbiota composition of mice given a WSD is altered, resulting in enhanced penetrability and a slower growth rate of the inner mucus layer. Microbiota from chow-fed mice can be transplanted to prevent both barrier abnormalities. Furthermore, we discovered that giving WSD-fed mice *Bifidobacterium longum* was enough to restore mucus growth, whereas giving them the fibre inulin prevented enhanced mucus penetrability [4].

Conclusion

The presence of specific bacteria, we believe, is critical for appropriate mucus function. These discoveries, if verified in humans, could help researchers better understand disorders involving a damaged mucus layer, such as ulcerative colitis.

References

1. Desai MS, Seekatz AM, Koropatkin NM, et al. A dietary fiber-deprived gut microbiota degrades the colonic mucus barrier and enhances pathogen susceptibility. Cell. 2016;167(5):1339-53.e21.

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