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In vitro fermentation of sugar beet arabino-oligosaccharides by fecal microbiota obtained from patients with ulcerative colitis to selectively stimulate the growth of *Bifidobacterium* spp. and *Lactobacillus* spp.

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The commensal bacteria found in the human gut are important for host health, and an unfavorable composition of the gut microbiota can affect the synergistic interaction that exists between microbes and their host. An altered microbial composition is suggested to play a pivotal role in the pathogenesis of ulcerative colitis (UC), an inflammatory bowel disease, and compositional changes have been observed in the colonic microbiota by us as well as by other research groups¹⁻³. Since bifidobacteria and lactobacilli may exert anti-inflammatory effects, a reduced level of these commensal bacteria may compromise the colon health and favor intestinal inflammation.

In this study, selective stimulation of fecal bifidobacteria and lactobacilli from healthy subjects and UC patients in remission or with active disease were investigated using arabino-oligosaccharides (AOS; DP2-10) derived from sugar beet pulp. The fermentative-induced changes were compared to those for fructo-oligosaccharides (FOS), which are known to have a prebiotic effect. The fermentation studies were carried out using a validated small-scale static batch system, and changes in the fecal microbial communities and metabolites were monitored after 24 h by quantitative real-time PCR and short-chain fatty acid analysis. With a few minor exceptions, AOS affected the communities similarly to what was seen for FOS. Quantitative real-time PCR revealed that *Bifidobacterium* spp. and *Lactobacillus* spp. were selectively increased after fermentation of AOS or FOS by fecal microbiota derived from UC patients. The stimulation of growth of *Lactobacillus* spp. and *Bifidobacterium* spp. was accompanied by a high production of acetate and hence a decrease of pH. The fermentation of AOS may thus help improve the inflammatory conditions in UC patients through stimulation of bacteria eliciting anti-inflammatory responses and through production of acetate.

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