Prebiotic β-galacto-oligosaccharide impact on clinical, inflammatory and microbiota outcomes in active ulcerative colitis: an open-label study

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Prebiotics may beneficially modulate innate immunity to promote immune homeostasis via both direct and indirect mechanisms. B-galacto-oligosaccharide (B-GOS) prebiotic supplementation beneficially influences markers of sub-clinical inflammation in humans[1]. The effect of B-GOS in active colonic inflammation has not previously been investigated. This study aimed to investigate the effect of 6-weeks B-GOS supplementation (containing active dose of 2.8 g/d β-GOS (Bimuno, Clasado, UK)) on clinical outcomes and faecal markers of inflammation, microbiota and microbial metabolites in patients with active ulcerative colitis (UC).

This open label study recruited 18 patients with active UC (Physician global assessment plus one of; endoscopic, faecal or serum markers of inflammation) to consume 2.8 g/d B-GOS for 6-weeks without altering their medication or habitual diet. Clinical outcomes were measured using the simple clinical colitis activity index (SCCAI), gastrointestinal symptom rating scale (GSRS) and Bristol stool form scale (BSFS). An SCCAI score of ≤2 was considered clinical remission. Faecal calprotectin (ELISA kit), microbiota (16S rRNA sequencing), short-chain fatty acids (gas liquid chromatography) and pH (pH probe) were measured. Follow-up (6-week) data were compared to baseline using Wilcoxon Signed Rank tests.

Following prebiotic, clinical scores (SCCAI), faecal calprotectin, microbiota, SCFA and pH were not significantly different from baseline. The proportion of normal stool (BSFS) following prebiotic (70%) was higher than baseline (49%) (p = 0.024) and there was reduced incidence (23% vs 46% p = 0.016) and severity (0.5 vs 0.7, p = 0.048) of loose stool (GSRS). Although there were no differences in SCCAI scores following prebiotic (2.8 (2.9)) compared to baseline (3.3 (2.2), p = 0.330), there were significantly lower SCCAI subscale scores for urgency (0.5 (0.8) vs 1.0 (0.7), p = 0.011). At baseline 46% had SCCAI ≤2 (despite objective evidence of inflammation) compared with 77% following prebiotic (p = 0.219). Sub-analysis of faecal microbiota proportions in patients with SCCAI ≤2 at baseline (n = 7), showed increased proportions of *Bifidobacterium* (baseline 1.65% (1.97), follow-up 3.99% (5.37), Z -1.992, p = 0.046) and *Christensenellaceae* (baseline 0.13% (0.33), follow-up 0.31% (0.76), Z -2.023, p = 0.043) between baseline and following prebiotic.

Six weeks supplementation with prebiotic β-GOS in active UC normalised stools but did not significantly lower clinical scores or markers of inflammation. Patients with less active disease had an increase in faecal *Bifidobacterium* and *Christensenellaceae* proportions, that did not occur in those with more active disease indicating the prebiotic effect may depend on disease activity. A controlled study is required to validate these observations.

1. Vulevic J, Juric A, Walton, GE *et al.* (2015) *Br. J. Nutr.* **114**(4), 586–595.