**Background:** *Faecalibacterium prausnitzii* may be a key protective bacterium, and useful therapeutically to attenuate inflammation and promote gut homeostasis in Crohn's disease (CD). However, the reasons for its variable persistence during active disease and recovery remain unknown. We hypothesise *F. prausnitzii* is constrained by dietary factors that might promote inflammation; and food additives have been implicated recently in microbial changes that promote inflammation. We have investigated how 8 common food additives affect the growth kinetics of 3 strains of *F. prausnitzii*.
**Methods:** *F. prausnitzii* A2-165, KLE1255, and AHMP21 were cultured using a habitat-simulating medium supplemented with 0.2% (wt/vol) glucose (M2G), or M2G prepared to contain 0.1% (wt/vol) of either sodium sulphite, aluminium silicate, carrageenan, carboxymethylcellulose, polysorbate 80, saccharin, sucralose, or aspartame, intended to approximate concentrations found in food. The 3 *F. prausnitzii* strains were also grown with M2G medium and once these cultures had reached mid-exponential phase of growth, either sodium sulfite or polysorbate 80 was added to the cultures to 0.1% (wt/vol). Growth was monitored by optical density measurements.
**Results:** Figure 1 shows all 3 strains were strongly inhibited by sodium sulfite and polysorbate 80. The growth rates of all 3 *F. prausnitzii* strains were not affected by the other food additives, with the exception of a small but significant decrease for strain KLE1255 in the presence of sucralose ($p < 0.05$). Cell yield of strain A2-165 was unaffected by the remaining food additives, whereas the cell yield of strain AHMP21 was reduced by saccharin ($p < 0.05$); and by sucralose and saccharin for strain KLE1255 ($p < 0.05$). Growth of all 3 *F. prausnitzii* strains was immediately arrested when sodium sulphite was added to mid-exponential phase cultures; the effects of polysorbate 80 were more variable and probably cell-density dependent.

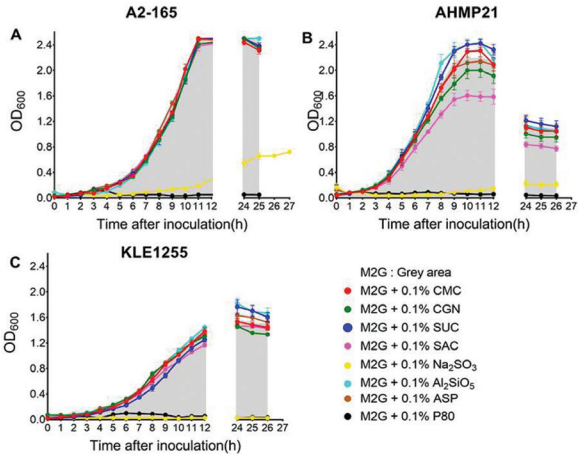


**Figure 1.** Effect of food additives (0.1% [wt/vol] final concentration) on the growth kinetics of F. prausnitzii strains A2-165 (A), AHMP21 (B), and KLE1255 (C). Data points are the mean ± SEM of optical density measurements at 600 nm ($n = 6$).

**Conclusions:** Sodium sulfite and polysorbate 80 have strong inhibitory effects on *F. prausnitzii* growth. Exclusion of such additives from the diet may be critical to improved Crohn's disease activity or prevention. This work is supported by The Leona M. and Harry B. Helmsley Charitable Trust.

## P838

## Effects of microbial metabolites on human intestinal epithelium

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**Background:** The intestinal epithelium is the interface between the microbiota and the underlying host mucosa. Intrinsic genetic as well as acquired defects in the epithelium have been described in inflammatory bowel diseases (IBDs) including Crohn's disease (CD). Bacterial metabolites, such as short chain fatty acids (SCFAs), are known to exert homeostatic, anti-inflammatory and anti-tumoural effects. However, the effects of SCFAs in the context of intestinal inflammation and specifically in CD, have not been extensively addressed. Results from our group reveal that the epithelial organoid system is a good tool to explore the impact of bacterial metabolites on the human epithelium. Our aim was to study the effect of faecal microbial SCFAs on the intestinal epithelium using organoid cultures from non-IBD controls and patients with CD.
**Methods:** SCFAs were extracted from faecal samples of non -IBD controls and active CD patients. The concentration of 13 SCFAs was measured in the derived faecal extracts (FEs) by HLPC. Organoid cultures, generated using biopsy samples from controls and CD patients, were incubated with faecal- SCFAs (1:50), or vehicle for 24 h. Total RNA was isolated from organoid cultures and the expression of genes associated with proliferation and other epithelial signalling pathways was analysed by qPCR.
**Results:** Despite the presence of active disease, faecal SCFA extracts from CD patients and controls showed comparable SCFA concentrations. Control FEs down-regulated KI67, CXCL1 and CLDN2 but their effect was significantly lower in healthy compared with CD organoids. MT1X was significantly increased by control FEs, however the effect was lower in CD-derived organoids. Remarkably, SCFA from control FEs reduced IL8 transcripts in control organoids, and did not affect IL8 expression in CD organoids. SCFAs derived from active CD patients showed a decreased ability to induce MT1X and to decrease CXCL1 in epithelial organoids from controls.
**Conclusions:** Both the ability of the CD epithelium to respond to SCFAs as well as the composition of the SCFAs from active CD patients show changes that suggest an altered microbial-epithelial interaction in CD. While SCFAs have potent effects on the epithelium, other metabolites and bacterial products may also be critical. Our current experiments include studying the effects of supernatants from specific gut commensal and pathogenic bacteria on human organoid cultures.

## P839 has been withdrawn.

## P840

## Dietary interventions rapidly alter metabolomics profile of patients with inflammatory bowel disease after pouch surgery

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