Background

Carbohydrates that resist digestion demonstrate a prebiotic effect by altering the colonic microbiota and the metabolome. The production of short chain fatty acids (SCFA) by the microbiota in particular, influences many aspects of gastrointestinal health.

Resistant starch is one category of prebiotic carbohydrates of which high amylose maize starch (HAMS) is an example. HAMS contains up to 70% amylose resistant starch that is resistant to pancreatic amylase and therefore enters the colon unabsorbed. There, it is rapidly fermented by colonic bacteria, yielding substantial amounts of SCFA including butyrate.

Another type of resistant starch is high amylose maize starch acetate (HAMSA) which is HAMS esterified with acetate; the latter is likely to be rapidly released by microbial hydrolases in the colon.

Methods

• Twenty children, aged 2 to 5 years, (ten stunted with height for age [HAZ] lower than 2 standard deviations below the mean, and ten showing normal growth).
• Informed written consent obtained from the parents.
• Fed cookies containing HAMS (10 g/daily) every day for two weeks (days 1-14).
• Two week washout period on their regular diet (days 15-28).
• Fed cookies containing HAMS (10 g/day) for two weeks (days 29-44).
• Thirteen stool samples collected - on days 0, 3, 7, 10, 15, 18, 22, 25, 29, 32, 36, 39 and 44.
• Fecal pH was measured.
• SCFA concentration quantitated by GC-MS.
• Microbiome characterisation was determined using shotgun metagenomic sequencing of faecal samples from 6 individuals. Sequencing downstream processes were performed as per Nielsen et al. 2014.

Results

P-values indicates significance for healthy versus stunted participants.

Figure 1

Effect of HAMS and HAMSA on fecal pH

![Figure 1](image1.png)

Figure 2

Effect of HAMS and HAMSA on fecal acetate

![Figure 2](image2.png)

Figure 3

Effect of HAMS and HAMSA on fecal propionate

![Figure 3](image3.png)

Figure 4

Effect of HAMS and HAMSA on fecal butyrate

![Figure 4](image4.png)

Figure 5

Characterisation of the faecal microbiome of a subset of 6 individuals from the study. Time points of each individual are indicated by acronym: Baseline (B), HAMS (H), Washout (W), and HAMSA (A).

A. Heatmap shows normalised read counts of the predominant microbial taxa detected using MetaPhlAn2. The normalised abundance is indicated by colour gradient (white, not detected; blue, most abundant).

B. Heatmap shows normalised read counts mapped to short-chain fatty acid biosynthesis genes from KEGG pathways. The normalised abundance is indicated by colour gradient (white, not detected; green, most abundant).

Conclusions

HAMS and HAMSA both lowered fecal pH with significantly lower pH being achieved in healthy children (Figure 1).

HAMS and HAMSA significantly increased acetate in stunted participants compared to baseline and day 29 respectively, but HAMSA produced a significantly greater effect in healthy children (Figure 2).

HAMS and HAMSA increased propionate in both healthy and stunted children compared to baseline but the rise was significantly greater in healthy children (Figure 3).

HAMS increased butyrate in healthy and stunted children (not significantly different) while HAMSA had no effect in stunted children while it increased butyrate significantly in healthy relative to stunted children (Figure 4).

There were significant differences between healthy and stunted children in the parameters of fermentation. Overall, colonic carbohydrate fermentation was significantly impaired in stunted children and HAMSA was less effective in inducing changes than was HAMS. However, neither resistant starch resulted in any significant changes in the microbiome of these children. The microbiota composition and functional genes encoding for short chain fatty acids biosynthesis appeared relatively stable throughout the feeding study for both healthy and stunted children (Figure 5).