size was 2.9±1.0 cm (range 1.0 to 5.0 cm), while HbA1c was 6.4±1.4%, AFP 399.2±1250.1 ng/mL, fucosylated AFP (AFP-L3) 10.7±19.1%, and protein induced by vitamin K absence or antagonist-II (PIVKA-II) 598.0±24,511.1 mAU/mL. Child-Pugh B classification was noted in 10 cases (6.0%). Univariate Cox hazard analysis for early recurrence beyond MC showed that Child-Pugh B (HR=3.53, 95% CI: 1.55-8.01; P=0.003), AFP-L3 (≥10%) (HR=2.08, 95% CI: 1.20-3.61; P=0.009), and PIVKA-II (≥100 mAU/mL) (HR=1.71, 95%CI: 1.00-2.9; P= 0.046) were significant risk factors, whereas AFP (≥100 ng/mL) was not (HR=1.14, 95% CI: 0.60-2.17; P=0.686). In multivariate analysis, Child-Pugh B (HR=3.82, 95% CI: 1.67-8.74; P=0.001) and AFP-L3 (≥10%) (HR=2.01, 95%CI: 1.14-3.54; P=0.016) were shown to be significant risk factors. The 1-, 3-, and 5-year recurrence rates for patients beyond MC and survival rates of patients without both risk factors were better than those of patients with 1 or more factors (5.4%, 29.1%, and 42.5% vs. 25.6%, 46.9%, and 66.2%, respectively, P=0.001; 97.2%, 90.1%, and 77.1% vs. 95.7%, 70.7%, and 47.1%, respectively, P=0.037). Conclusion: Child-Pugh B and AFP-L3 were found to be significant prognostic factors to predict early recurrence beyond MC after initial resection for small and single HCC. In patients with 1 or more factor, LT should be considered for obtaining a better prognosis.

Su1487

Bifobacteria Infantis Contributes Significantly to the Beneficial Effects of Oligofructose (Prebiotic) to Prevent Mucosal Inflammation, Metabolic Endotoxemia and Hyperphagia Induced by High Fat Feeding

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High fat feeding (HFD) leads to intestinal inflammation, metabolic endotoxemia, hyperphagia and obesity. This appears to be mediated by gut dysbiosis. It is conceivable these abnormalities may be corrected through the use of prebiotics or probiotics to alter gut microbiota. To test this, we performed studies on rat fed with HFD (53% kcal/g fat) with/without the fermentable non-digestible oligofructose (10%) (prebiotic). 454 pyrosequencing analysis of ileal mucosa following HFD showed a significant decrease in unclassified clostridiales (14% vs 70%), and significant increases in the Peptostreptococcaceae (27% vs 12%); Erysipelotrichaceae (24% vs 5%) and Clostridiaceae (24% vs 1%). Distal ileum showed inflammation characterized by increased expression of INFγ (2.1 fold), IL10 (3.7 fold) and IL1β (16.9 fold). HFD increased abnormal occludin distribution (0.95% vs 7.1%, P<0.05) resulting in a 19% decrease in transepithelial electrical resistance in the ileal mucosa. Dietary supplement with oligofructose enriched inulin altered gut microbiota composition by increasing Bifidobacteriaceae and unclassified Clostridiales and decreasing Clostridiaceae and Peptostreptococcaceae. Alteration in gut microbiota by oligofructose was accompanied by a marked reduction of intestinal inflammation, normalization of tight junction Occludin distribution (1.05% vs 7.1%, P<0.05), and a significant improvement of transepithelial electrical resistance in terminal ileum (320.6 Ohmes.7mm^2 vs 276 Ohmes.7mm^2, P<0.05). HFD increased plasma LPS levels from 0.35 to 1.61 EU/ml (P<0.05). Oligofructose prevented the development of endotoxemia and reduced energy intake (78 kcal/day from 86 kcal/day, P<0.05). Since oligofructose feeding caused a marked increase in Bifidobacteria, this bacteria (probiotic) may contribute to the beneficial effects of oligofructose feeding. Rats fed 2 wk HFD followed by Bifidobacteria infantis (109 U/day) for 2 wk caused a marked reduction in intestinal inflammation, normalization of tight junction Occludin distribution (1.05% vs 7.1%, P<0.05), an improvement of transepithelial electrical resistance in terminal ileum (301±10 vs 239±5 Ohmes 7mm^2, P<0.05), and 10% reduction energy intake (61.09 kcal/day from 67.95 kcal/day, P<0.05). In conclusion, we showed dietary oligofructose enriched inulin induces changes in gut microbiota, improves gut barrier function and prevents development of hyperphagia induced by HFD. These effects could be reproduced by Bifidobacteria infantis suggesting this probiotic contributes in a major manner to the beneficial effects of the prebiotic oligofructose to reduce gut inflammation, prevent metabolic endotoxemia and hyperphagia induced by HFD. These findings suggest that both oligofructose (prebiotic) and Bifidobacteria infantis (probiotic) may be useful in the prevention and/or treatment of metabolic disorders related to HFD.

Su1488

Effect of Native and Acetylated High Amylose Maize Starch on Fecal pH and Short Chain Fatty Acid Concentrations in a Cohort of Children in Southern India

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Background: Carbohydrates that resist digestion alter the gut microbiome and metabolome. The production of short chain fatty acids (SCFA) by the microbiome, in particular, influences many aspects of gastrointestinal health. We undertook a non-randomized sequential crossover feeding study to determine whether short term feeding with high amylose maize starch (HAMS), containing type 3 amylase-resistant starch, or HAMS esterified with acetate to the extent of 2.5% acetate (HAMSA) would alter fecal pH and SCFA concentrations in a cohort of children in southern India. Methods: Twenty children, aged 2 to 5 years, (ten stunted with height for age less than 2 standard deviations below the mean, and ten showing normal growth), were given cookies containing HAMS (10 g/day) every day for two weeks. After a two week washout period, they were then given HAMSA (10 g/day) cookies for two weeks. Thirteen stool samples were collected on days 0, 3, 7, 10, 15, 18, 22, 25, 29, 32, 36, 39 and 44. Fecal pH was measured and SCFA were measured by GC-MS. Informed consent was obtained from the parents. Results: Mean (SD) intake of HAMS was 8.2 (0.9) g/day, and of HAMSA was 8.3 (2.1) g/day. Fecal pH decreased and SCFA concentration increased (Table 1) within seven days of feeding either HAMS or HAMSA and returned to basal levels within a week during the washout period. No significant difference in the fecal response parameters was noted between healthy and stunted children. None of the children experienced any adverse effects during the study period. Conclusions: Both HAMS and HAMSA, when added to the usual diet, reduced fecal pH and increased fecal acetate concentrations in children aged 2-5 years in southern India. HAMS, but not HAMSA, also increased fecal concentrations of butyrate and propionate. The reduction in fecal pH and increase in fecal SCFA concentration induced by these poorly digested carbohydrates has potential implications for colonic nutrient absorption. We conclude that HAMS was fermented by the colonic microbiota to a mixture of SCFA, while the selective increase of fecal acetate upon HAMSA feeding suggests that esterified acetate was released, but that the esterification process (HAMS) somehow retarded microbial fermentation of the base starch thus resulting in insignificant increases in propionate and butyrate.

Table 1. Fecal pH and SCFA concentration (mmol/Kg feces) in relation to feeding intervention. Values shown are mean (SD).

	Day 0 (commencement)	Day 7-15 (2nd week of HAMS)	Days 22-29 (2nd week of washout)	Days 36-44 (2nd week of HAMSA)	P
Fecal pH	6.6 (0.72)	6.07 (0.83)*	6.71 (0.90)	6.15 (0.93)**	*P<0.001 vs day 0 **P<0.001 vs day 29
Acetate	61.2 (16.1)	79.1 (27.5)*	68.5 (21.2)	81.9 (32.5)**	*P=0.003 vs day 0 **P=0.049 vs day 29
Propionate	16.4 (6.7)	28.1 (16.4)*	18.5 (8.8)	23.9 (15.5)**	*P<0.001 vs day 0 **P=0.30 vs day 29
Butyrate	9.3 (4.7)	18.4 (13.3)*	12.3 (6.9)	16.2 (10.2)**	*P<0.001 vs day 0 **P=0.27 vs day 29

Su1489

Antioxidant Activity of Inulin and Its Ability to Prevent Human Colonic Muscle Cell Impairment Induced by Lipopolysaccharide Mucosal Exposure

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Background: Fructans, such as Inulin, are dietary fibers with pro-healthy properties which stimulate gastro-intestinal (GI) function acting as prebiotics. Lipopolysaccharide (LPS) is known to impair GI motility, following production of reactive oxygen species and other inflammatory mediators. The antioxidant activity of various fructans has been tested here and the protective effect of inulin on colonic muscle cell impairment, induced by exposure of human mucosa to LPS, was assessed in an ex vivo experimental model. Methods: The antioxidant capacity of fructans (inulins, agavins, and levans) was measured using the TEAC (Trolox Equivalent Antioxidant Capability) method. Cooking and digestion were simulated in an vitro system to test modifications in the antioxidant activity of inulin possibly caused by these processes. Human colonic mucosa and submucosa, obtained from disease-free margins of resected segments for cancer, were sealed between two chambers, with the mucosal side facing upwards and covered with 5 mL of Krebs solution with or without purified LPS from a pathogenic strain of Escherichia coli (O111:B4) and Inulin (Frutafit IQ®), and the submucosal side facing downwards into 25 mL of Krebs solution. The solution on the submucosal side was collected after 30 min of mucosal exposure to Krebs in the absence (N-undernatant) or presence of LPS (LPS-undernatant) and in the presence of LPS and Inulin (LPS+INU undernatant). Undernatants were tested for the antioxidant activity and their effects on isolated smooth muscle cells (SMCs). The inulin protective effect on the mucosa and submucosa were assessed measuring the protein oxidation level in all the experimental conditions analysed, as well as its ability to revert the LPS-induced impairment of SMC contraction. Results: Antioxidant activity of inulin was significantly higher compared to their constituent simple sugars and remained unaltered despite the cooking and digestive processes, simulated in vitro. Inulin treatment protected mucosa and submucosa against protein oxidation. Following thirty-minute exposure to LPS-undernatant a significant decrease in maximal Ach-induced contraction was observed when compared to the contraction induced in cells incubated with the N-undernatant (148.4±5.2 vs 98.1±9.0μm, p<0.05) and this effect was partially prevented by pre-incubation of LPS with Inulin (81.3±7.9μm of cell length following Ach exposure, p<0.005). Conclusions: Inulin, with an antioxidant activity resistant to cooking and digestion, protects the human colon mucosa from damage induced by LPS and this effect appears to be related to the scavenging activity of inulin during LPS-induced oxidative stress.

Su1490

Soluble Dextrin Fibers Modulate Intestinal Microbiota and Reduce Pro-Inflammatory Cytokine Secretion in IL-10 -/- Mice

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IL-10 deficient (IL-10-/-) mice spontaneously develop a patchy chronic colitis between the ages of 8 and 12 weeks. Severity of colitis is highly dependent upon the microbial composition. Non-digestible fermentable prebiotic fibers can stimulate the growth and activity of specific bacteria in the gut. The purpose of this study was to determine the effect of prebiotic fibers on intestinal microbiota and immune function in the IL-10-/- mouse. **Methods:** Prior to the development of colitis, 4 wk old 129S1/SvlmJ IL10-/- mice (n=8/group) were started on test diets; these included mouse chow (LabDiet 5001) with 4% cellulose as a control, new resistant starch (NRS), soluble fiber dextrin from tapioca (SFD-t), soluble fiber dextrin from corn (SFD-c), or soluble corn fiber (SCF70) (Tate & Lyle) for a total of 12 weeks. Effects of diet on animal growth, small intestinal permeability by lactulose-mannitol excretion in urine, histological injury, intestinal cytokine secretion using MesoScale Discovery Platform and microbiota composition by 16S rDNA pyrosequencing of stool were measured. ANOVA followed by Dunnett's multiple comparisons test was performed using GraphPad Prism. Microbiota data were compared using principal coordinate analysis. **Results:** There were no significant differences in animal growth, food eaten, intestinal weight, length, or permeability over the 12 week feeding period between the groups. However, all diets induced variable