Concurrent Session 7: Gut Health

Resistant starch and targeted synbiotics can protect against colorectal cancer

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Background – Resistant starches (RS) are a class of prebiotics that have been shown to have physiological benefits mainly arising through their interaction with the colonic microflora. As the consumption of probiotics or live microorganisms becomes more widespread, ways are being sought to ensure and improve the delivery of the desired health benefits. The concept of using a specific type of RS that interacts with a particular probiotic microorganism as a "targeted synbiotic" to provide a distinct physiological outcome has recently been explored.
Objective – This study has evaluated in rats, the effect of a probiotic bacteria '*Bifidobacterium lactis*', the carbohydrate 'RS and their combination (synbiotic) on colorectal cancer development. *Bifidobacterium lactis* is a probiotic that has previously been shown to utilise RS as a substrate for fermentation.
Design – Sprague-Dawley rats (n = 180) were divided into 6 equal groups and fed semi-purified diets for 30 weeks. After 4 weeks on these nutritionally balanced diets, colonic neoplasms were induced by 2 weekly injections of azoxymethane (15 mg/kg B.W). Colons were resected after 30 weeks of feeding for evaluation of neoplasm formation, short chain fatty acids (SCFA), and epithelial biology. The experimental groups were as follows: Control, fed a diet containing no added dietary fibre or RS. There were two high amylose maize diets as the source of RS, namely Hi-maize 958 and Hi-maize 260. Also to each of these three diets lyophilized cultures ($1x10^{11}$ cfu/g) of *Bifidobacterium lactis* was added at a level of 1% by weight to give a total of six treatment groups.
Outcomes – Rats fed RS in combination with *Bifidobacterium lactis* significantly lowered the incidence and multiplicity of colonic neoplasms ($P<0.001$, using two-way factorial Poisson model) by over 50% compared to the Control group. Fermentation events (SCFA, pH) were altered by the inclusion of RS into the diet while the inclusion of *Bifidobacterium lactis* into the diet had no significant effect on the fermentation parameters.
Conclusion – The results of this study show that the synbiotic combination of RS and *Bifidobacterium lactis* significantly protected against the development of colorectal cancer.

Emu Oil improves aspects of colonic integrity in a rat model of ulcerative colitis

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Background – Ulcerative Colitis (UC) is a debilitating disease of the colon, characterised by inflammation and ulceration. The dextran sulphate sodium (DSS) model of colitis is highly reflective of human UC and is commonly used to assess novel therapies. Emu Oil (EO) is extracted from the fat of the Emu and is predominantly composed of fatty acids. Anecdotal evidence suggests EO contains anti-inflammatory and healing properties. In preliminary studies, we have demonstrated anti-inflammatory activity of EO in a model of chemotherapy-induced mucositis.
Objectives – To evaluate oral administration of EO for its potential to ameliorate DSS-induced UC in male rats.
Design – UC was induced in rats by consumption of 2%DSS (D) in drinking water from Day 5. Treatment groups (WW, W1EO, DW, D0.5EO and D1EO; n=8) were orally gavaged daily with either 0.5ml or 1mlEO, or 1ml water (W) from Day 0 to 11. Group 6 (D1EO(6)) commenced 1ml EO gavage on Day 6. Disease activity and metabolism parameters including bodyweight, food and water intake and urine and faecal output, were measured daily. Sections of proximal and distal colon were assessed histologically for measurements of crypt depth (CD).
Outcomes – Distal colon CD was significantly greater in all DSS-treated groups compared to healthy controls [DW (271±17 μm) compared to WW (202±6 μm) (26% greater; $P<0.05$)]. Furthermore, CD was significantly greater in D0.5EO, D1EO and D1EO(6) (352±22, 341±17, 409±16 μm, respectively) compared to DW (271±17 μm; $P<0.05$), and in D1EO(6) compared to D1EO (17% greater; $P<0.05$). Proximal colon CDs were significantly greater in D0.5EO, D1EO and D1EO(6) (339±16, 389±16 and 351±17 μm, respectively) compared to WW (260±11 μm) and in D0.5EO (373±18 μm) compared to DW (302±8 μm; $P<0.05$). There were no significant differences between treatment groups when comparing disease activity and metabolism parameters.
Conclusions – Emu Oil induced colonic crypt lengthening in a rat model of ulcerative colitis, suggesting an increase in the rate of repair in the large bowel. Further studies investigating the dose and timing of Emu Oil are indicated in order to optimise its therapeutic potential as a dietary supplement to augment conventional treatment approaches for ulcerative colitis.