Concurrent Session 7: Gut Health

**Butyrylated starch decreases colonic smooth muscle contractility but does not affect colonocyte proliferation**

BH Bajka, GS Patten, DL Topping, MY Abeywardena and JM Clarke

CSIRO Human Nutrition, Adelaide, SA & Preventative Health National Research Flagship

**Background** – Non-digested carbohydrate is fermented by colonic bacteria to produce short chain fatty acids (SCFA). A major SCFA, butyrate, regulates gastrointestinal motility, the colonocyte cell cycle and may protect against colorectal cancer.

**Objective** – This study examined the effects of increased large bowel butyrate on colonocyte proliferation and receptor-independent and receptor-dependent contractility in isolated gut tissue sections.

**Design** – Sprague Dawley rats (n=8/group) were fed AIN-93G based diets supplemented with 0, 5, or 10% butyrylated low amylose maize starch (LAMSB) for 10 days. Digesta and hepatic portal venous butyrate levels were measured and colonocyte proliferation assessed using proliferating cell nuclear antigen. The contractility of colonic and ileal tissue were measured *in vitro* in response to electrical stimulation, KCl-induced depolarization-driven, and agonist stimulated muscarinic- and prostanoid-induced contractions.

**Outcomes** – LAMSB increased caecal (P<0.001) but not colonic tissue weight; increased caecal and distal colonic digesta weight and decreased digesta pH throughout the large bowel. Caecal and distal colonic butyrate pools and portal venous butyrate concentrations were higher in rats fed LAMSB (P<0.05). LAMSB at 10% decreased distal colonic crypt depth (P<0.05) but did not alter the rates of colonocyte proliferation. Further, the 10% LAMSB diet compared to control lowered electrically-stimulated (1.1 ± 0.2 vs 0.6 ± 0.1 V/g) and carbachol (8.7 ± 2.2 vs 5.8 ± 1.6 V/g) and PGE2 receptor-induced contractions (6.0 ± 0.5 vs 3.9 ± 0.8 V/g) of colon (P<0.05) but not ileum.

**Conclusion** – Increased large bowel butyrate does not affect proliferation of normal colonocytes but does reduce contractility of colonic smooth muscle.

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**Mandatory thiamin enrichment of bread making flour has improved the thiamin status of the population**

B Wood¹, M Riley², G Whelan³

¹ Victorian Local Governance Association, Carlton VIC 3053  
² Dairy Australia, Southbank VIC 3000  
³ Addiction Medicine, Monash University, Clayton VIC 3168

**Background** – In the late 1980s, the national estimate for Wernicke’s encephalopathy was considered to be about 500 cases per annum. Thiamin deficiency of the brain (acute Wernicke’s encephalopathy) in association with alcohol abuse had been reported in 32 cases over 33 months (Melbourne), and there was a relatively high rate at autopsy (acute, chronic). There was evidence (since 1941) that the thiamin supply was marginal in the national diet and there was sub-clinical thiamin depletion in many vulnerable groups. After many years of debate, legislation was enacted for mandatory thiamin enrichment to the level of 6.4 mg thiamin/kg bread making flour from January 1991.

**Objective** – To determine whether “mandatory thiamin enrichment of bread making flour has improved the thiamin status of the Australian population.”

**Design** – A comprehensive literature review was conducted, together with 24 key informant questionnaires (medical officers, nutritionists). The search included population thiamin supply and thiamin intake studies. Health outcomes associated with thiamin enrichment and thiamin status were evaluated from 1991 onwards. Evidence was sought for sub-clinical thiamin status, and clinical thiamin deficiency (acute Wernicke’s encephalopathy, beriberi heart disease). The Wernicke-Korsakoff Syndrome and Korsakoff’s psychosis were included.

**Outcomes** – There is now consensus evidence for improvement in thiamin intake, thiamin status, and consequent health of the population since 1991, with no known harmful effects. Acute Wernicke’s encephalopathy is now rare. Cases of the Wernicke-Korsakoff Syndrome have decreased significantly, together with other forms of alcohol related brain damage sometimes attributed (by association) to thiamin deficiency.

**Conclusions** – Maintenance of mandatory thiamin enrichment of all bread making flour is strongly supported. Extension of thiamin enrichment is recommended to all flours (including rye, wholegrain and pre-mixes used in bread making), and flour substitutes.