Bifidogenic effects of feeding infant formula containing galacto-oligosaccharides in healthy formula-fed infants
JEAC Napoli1, JC Brand-Miller1, P Conway3
1School of Molecular and Microbial Biosciences, University of Sydney, NSW 2006
2School of Biotechnology & Biomolecular Sciences, University of New South Wales, NSW 2052

Background - Human milk oligosaccharides are readily fermented in the infant colon where they selectively stimulate the growth of bifidobacteria. Bifidobacteria lower intestinal pH through the production of acetic and lactic acids which may suppress the growth of pathogenic bacteria.

Objective - To investigate the bifidogenic effects of a galacto-oligosaccharide (GOS) supplemented infant formula on the composition of the faecal microflora in formula-fed infants.

Design - Healthy full-term formula-fed infants were randomly assigned to receive standard infant formula supplemented with 0.7% GOS (n = 13) or the same formula supplemented with lactose as a control (n = 13) during a 21 day feeding trial. Twenty four breast-fed infants were also studied as a reference group. Faecal samples were collected on day 1, day 11 and day 21 and analysed for bacterial counts, pH and lactate concentrations.

Outcomes - GOS supplementation increased bifidobacteria counts 10-fold (P = 0.001) to the range of the reference group of breast-fed infants. The faecal pH after 21 days of feeding was significantly lower in the GOS formula group than in the control group (P = 0.004). Fecal lactate concentrations increased 4-fold in the GOS formula group from 1.5 ± 0.5 mmol/L on day 1 to 5.9 ± 1.4 mmol/L on day 21, but this increase was only marginally significant (P = 0.09).

Conclusions - This study showed that supplementation of infant formula with GOS stimulates the growth of bifidobacteria in the colon and results in lower fecal pH and increased fecal lactate concentrations.
Effect of experimental oligosaccharide on brain and body weight

B Wang1, A Staples1, A Hunter1, B Yu2, J Brand Miller1

Human Nutrition Unit, School of Molecular and Microbial Biosciences, G08 and Molecular Genetics Laboratory, Central Clinical School2, University of Sydney, NSW, Australia 2006

Background - The biological function of human milk oligosaccharides (HMO) is not fully understood. In addition to their ability to stimulate growth of Bifidobacteria and inhibit pathogens, there may be beneficial effects on brain development. Our hypothesis is that oral supplementation of certain oligosaccharides favourably influences brain growth and learning ability in an animal model. The piglet is the most appropriate as its brain growth closely parallels that of the human infant.

Objective - To investigate the effects of an experimental oligosaccharide found in human milk, on brain weight, total brain cortex cell number (estimated by DNA content) and body weight development in formula-fed piglets.

Design - Twelve 3-day-old male piglets were randomly allocated into a treatment (n = 6) or control group (n = 6). Piglets were fed a diet of soy/whey/casein sow’s milk replacer (55:9:36) for 32 days. The treatment group were fed the standard diet with 215 mg/kg oligosaccharide per day. Body weight was determined daily using electronic scales prior to the morning feed. The piglets were euthanased on day 36 and brain weight and frontal cortex DNA content were determined.

Outcomes – The rate of body weight gain (mean ± SD) did not differ between the groups: 217 ± 34 g/d in the treatment group and 216 ± 39 g/d in the controls. Brain weights were similar 54 ± 6 g vs 57 ± 4 g respectively, and cerebrum and cerebellum were 71% and 11% vs 69% and 11% respectively of total brain weight. Brain frontal cortex cell number was 24% higher (110.7 ± 14.2 x 10³ cells/mg tissue) in treatment than controls (89.3 ± 15.0 x 10³ cells/mg tissue) but the difference did not reach statistical significance (P=0.326).

Conclusions – This study showed that dietary supplementation of the experimental oligosaccharide had no significant effect on body weight gain, brain weight or frontal cortex cell number.