1,2-Fucosyllactose Does Not Improve Intestinal Function or Prevent Escherichia coli F18 Diarrhea in Newborn Pigs

Objectives: Infectious diarrhea, a leading cause of morbidity and deaths, is less prevalent in breastfed infants compared with infants fed infant formula. The dominant human milk oligosaccharide (HMO), α-1,2-fucosyllactose (2′-FL), has structural homology to bacterial adhesion sites in the intestine and may in part explain the protective effects of human milk. We hypothesized that 2′-FL prevents diarrhea via competitive inhibition of pathogen adhesion in a pig model for sensitive newborn infants. Methods: Intestinal cell studies were coupled with studies on cesarean-delivered newborn pigs (n=24) without (control) or with inoculation of enterotoxigenic Escherichia coli F18 (7.5×1010/day for 8 days) fed either no (F18) or 10 g/L 2′-FL (2FL-F18). Results: In vitro studies revealed decreased pathogen adhesion to intestinal epithelial cells with 2′-FL (5 g/L; P<0.001). F18 pigs showed more diarrhea than control pigs (P<0.01). Administration of 2′-FL to F18 pigs failed to prevent diarrhea, although the relative weight loss tended to be reduced (~19 vs ~124 g/kg, P=0.12), higher villi were observed in the distal small intestine (P<0.05), and a trend toward increased proportion of mucosa and activities of some brush border enzymes in the proximal small intestine. In situ abundance of α-1,2-fucose and E coli was similar between groups, whereas sequencing showed higher abundance of Enterobacteriaceae in F18, Enterococcus in control and Lachnospiraceae in 2FL-F18 pigs. Conclusions: 2′-FL inhibited in vitro adhesion of E coli F18 to epithelial cells, but had limited effects on diarrhea and mucosal health in newborn pigs challenged with E coli F18.