FEEDING DONOR HUMAN MILK COMPARED TO INFANT FORMULA IMPACTS THE EXPRESSION OF LIPID AND CAROTENOID UPTAKE AND METABOLISM-RELATED GENES IN PRETERM PIG INTESTINE

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Background: Infants fed human milk typically have greater blood and tissue carotenoid concentrations than infants fed formula, even when formula carotenoid content is matched to that of human milk. This suggests that carotenoids from human milk are better absorbed than from formula, but absorption differences have not been explained by differences in physical attributes. Here we test the hypothesis that expression of genes implicated in carotenoid absorption and metabolism differs in the small intestinal tissue of donor human milk (DHM)- vs. formula fed-piglets.

Materials/Methods: Preterm piglets were fed either pasteurized DHM (Prolacta, n=7) or premature infant formula (Enfamil Premature) (n=7) from 2 to 7 days of age. Healthy jejunal mRNA expression of scavenger receptor class B member 1 (Scarb1), low-density lipoprotein receptor (Ldlr), 3 ATP binding cassette transporters (Abca1, Abcb1, Abcg2, Abcg5, Abcg8), beta-carotene oxygenase 1 (Bco1), cluster determinant 36 (Cd36), intestine specific homeobox (Isx),and beta-carotene oxygenase 2 (Bco2), and Niemann-Pick C 1 Like 1 (Npc1l1) analyzed by qRT-PCR using pre-validated porcine Taqman expression assays. The target gene relative expression was normalized to ribosomal protein L4 (Rpl4) (dCt method), formula-fed piglet expression was analyzed relative to DHM-fed piglets (ddCt method), and group differences were determined by one-way ANOVA (alpha=0.05).

Results: Expression of all 12 target genes was detectable. Expression of Ldlr was greater in formula-fed piglets (1.57±0.39-fold, P=0.018), while expression of Cd36 was lower with formula feeding (0.747±0.20-fold, P=0.034) than DHM-feeding. Expression of the other genes did not significantly differ by diet treatments.

Conclusions: This study indicates that expression of Cd36 and Ldlr differ by DHM- vs. formula-feeding after only 5 days. Cd36 has been shown to mediate apical absorption of beta-carotene, and therefore the lower expression in the formula-fed piglets may provide a partial mechanistic explanation by which carotenoid absorption is lower in formula-fed versus DHM-fed infants. Ldlr plays a role in cholesterol homeostasis, and expression changes may indicate a shift in intestinal lipid metabolism resulting from different cholesterol contents of DHM vs. formula. Future studies should examine the effects of HDM vs. formula on piglet carotenoid absorption, serum carotenoid concentrations, and gene expression at different intestinal locations.

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