**Maternal FUT2 genotype in relation to risk of acute infections and rotavirus vaccine shedding in infancy**

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**Abstract:**

The human *FUT2* gene codes fucosyltransferase2, an enzyme that catalyzes the production and secretion of H-antigen in bodily fluids and tissues. *FUT2* status has been associated with risk of infection for some pathogens. In addition, mothers who are homozygous positive or heterozygous for the *FUT2* gene produce a class of oligosaccharides in their milk that can act as decoy receptors for some selected pathogens. The link between maternal *FUT2* status and illnesses in breast-fed infants is not clear.

The Pediatric Respiratory & Enteric Virus Acquisition and Immunogenesis Longitudinal (PREVAIL) Cohort study of mother-infant pairs in Cincinnati, OH assessed whether an epidemiologic association may exist between maternal FUT2 genotype and incidence of acute gastroenteritis (AGE), acute respiratory infection (ARI), and rotavirus vaccine shedding in their infants. We expected that the exclusively breastfed children of *FUT2+* mothers would have lower incidences of infection due to the unique breastmilk oligosaccharide composition.

We included all 75 mother-infant pairs who exclusively breastfed their infants, which provided a total of 1,243 child-weeks of follow-up from birth to 6 months. All mothers were genotyped for *FUT2* by PCR. Of these, 40 (53%) infants were vaccinated against rotavirus and had ≥1 weekly stool sample collected within 8 weeks after vaccination and tested by real time RT-PCR assays to determine vaccine shedding. Infant infection data came from weekly maternal reports of AGE and ARI that were collected by using an automated text messaging system.

Among the exclusively breastfed, infants of *FUT2+* mothers were found to have longer median duration of rotavirus vaccine shedding (27 days for RV1 vaccine and 18 days for RV5 vaccine) post-vaccination than babies of *FUT2-* mothers (14 days) (p=0.007 for RV1, p=0.021 for RV5 vaccine). Additionally, infants of *FUT2+* mothers had more AGE symptomatic study weeks (5.6%) than children of *FUT2-* mothers, who had no symptomatic AGE weeks (p<0.001). Similarly, infants of *FUT2+* mothers had ARI with fever for approximately 2.7% of study weeks, whereas babies of *FUT2-* mothers had no cases of ARI with fever (p=0.006). In this ongoing study, we plan to continue our analysis to include the infant’s own FUT2 status and medically-attended illness.