STRONG CHILDREN'S RESEARCH CENTER

Summer Research Scholar

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ABSTRACT

Title: Maternal perinatal colonization of *Bifidobacterium longum* subsp. *infantis* and fecal IgA in traditional farming and urban communities

Background: The prevalence of allergic diseases has dramatically increased over the last 50 years, particularly in high-income Westernized countries. More than 1 in 4 U.S. children reported having an atopic disease in 2021; however, the majority of primary prevention strategies have been unsuccessful to date[7]. Maternal prenatal exposure to traditional farming lifestyle has been shown to be protective against atopy in early life, but it is unclear how this protection is conferred to the infant[1,4,5]. The maternal perinatal gut microbiome can influence early immune development by various mechanisms such as transplacental transfer of bacterial metabolites, seeding of infant microbiome during delivery, and maternal-infant immune alignment during pregnancy and lactation[6]. Given the diverse microbial exposure on farms, the maternal microbiome during pregnancy may play a role in protection against allergy.

We have identified high colonization and abundance of keystone species, *Bifidobacterium longum* subspecies *infantis* (*B. infantis*) in infants from the Old Order Mennonites (OOM), a traditional farming community at low risk of atopic disease relative to infants from urban Rochester (ROC) who are at high-risk for allergy[2,3]. *B. infantis* is traditionally high in non-industrialized communities with low rates of atopic disease, but the route of colonization of *B. infantis* in infants remains unclear. Our lab has also shown that the gut microbiome composition of OOM women during the perinatal period is distinct from that of ROC women. OOM women also harbor a higher capacity for short-chain fatty acid production, bacterial metabolites known to induce IgA production. However, how the maternal gut microbiome during the perinatal period influences *B. infantis* colonization and further influences maternal mucosal immunity has not been investigated in traditional farming communities.

Objective: To examine the maternal gut microbiome as a potential reservoir for infant-associated *B. infantis* and assess maternal fecal IgA and its relationship with maternal lifestyle and atopic disease.

Methods: The abundance of *B. infantis* was measured via qPCR in 152 maternal stool samples from 2nd trimester to 8 weeks postpartum (OOM, n=78; ROC, n=74) and 10 sibling stool samples (OOM, n=5; ROC, n=5). Protein was extracted from a subset of maternal fecal samples, and an ELISA was used to measure the levels of fecal IgA1 and IgA2.

Results: Of the 152 maternal fecal DNA samples analyzed via qPCR, only 3 (1 OOM, 2 ROC) had detectable levels of *B. infantis* (1.97%). Further, out of the sibling samples, 3/10 (2 OOM, 1 ROC) were colonized with *B. infantis* (30%). In terms of mucosal immune response, fecal IgA1 did not differ between OOM and ROC mothers, but fecal IgA2 concentration was higher in ROC relative to OOM women. Concentrations of neither IgA subclass differed in samples collected pre- versus postnatally.

Conclusion: The broad lack of detectable levels of *B. infantis* in the maternal stool samples suggests that infants may be colonized via a route other than vertical transmission during delivery. Moreover, the higher rate of *B. infantis* abundance in sibling samples supports other individuals in the home as potential sources/carriers of *B. infantis*. These results could have implications for timing of probiotic supplementation for mothers and infants. Fecal IgA2 results further suggest that different lifestyles modify mucosal immunes responses, perhaps due to differences in the gut microbiome, with potential implications for both maternal health and early fetal/infant immune development.

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