Supplementation of the Breastfed Baby
"Just One Bottle Won't Hurt"---or Will It?

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Background

- The gastrointestinal (GI) tract of a normal fetus is sterile
- the type of delivery has an effect on the development of the intestinal microbiota
    - vaginally born infants are colonized with their mother's bacteria
    - cesarean born infants' initial exposure is more likely to environmental microbes from the air, other infants, and the nursing staff which serves as vectors for transfer
- babies at highest risk of colonization by undesirable microbes or when transfer from maternal sources cannot occur are cesarean-delivered babies, preterm infants, full term infants requiring intensive care, or infants separated from their mother
- breastfed and formula-fed infants have different gut flora
    - breastfed babies have a lower gut pH (acidic environment) of approximately 5.1-5.4 throughout the first six weeks that is dominated by bifidobacteria with reduced pathogenic (disease-causing) microbes such as *E coli, bacteroides, clostridia, and streptococci*
    - babies fed formula have a high gut pH of approximately 5.9-7.3 with a variety of putrefactive bacterial species
    - in infants fed breast milk and formula supplements the mean pH is approximately 5.7-6.0 during the first four weeks, falling to 5.45 by the sixth week
    - when formula supplements are given to breastfed babies during the first seven days of life, the production of a strongly acidic environment is delayed and its full potential may never be reached
    - breastfed infants who receive supplements develop gut flora and behavior like formula-fed infants
- The neonatal GI tract undergoes rapid growth and maturational change following birth
    - Infants have a functionally immature and immunonaive gut at birth
    - Tight junctions of the GI mucosa take many weeks to mature and close the gut to whole proteins and pathogens
    - Open junctions and immaturity play a role in the acquisition of NEC, diarrheal disease, and allergy
    - sIgA from colostrum and breast milk coats the gut, passively providing immunity during the time of reduced neonatal gut immune function
    - mothers' sIgA is antigen specific. The antibodies are targeted against pathogens in the baby's immediate surroundings
    - the mother synthesizes antibodies when she ingests, inhales, or otherwise comes in contact with a disease-causing microbe
    - these antibodies ignore useful bacteria normally found in the gut and ward off disease without causing inflammation

- infant formula should not be given to a breastfed baby before gut closure occurs
    - once dietary supplementation begins, the bacterial profile of breastfed infants resembles that of formula-fed infants in which bifidobacteria are

no longer dominant and the development of obligate anaerobic bacterial populations occurs (Mackie, Sghir, Gaskins, 1999)
    - relatively small amounts of formula supplementation of breastfed infants (one supplement per 24 hours) will result in shifts from a breastfed to a formula-fed gut flora pattern (Bullen, Tearle, Stewart, 1977)
    - the introduction of solid food to the breastfed infant causes a major perturbation in the gut ecosystem, with a rapid rise in the number of enterobacteria and enterococci, followed by a progressive colonization by bacteroides, clostridia, and anaerobic streptococci (Stark & Lee, 1982)
    - with the introduction of supplementary formula, the gut flora in a breastfed baby becomes almost indistinguishable from normal adult flora within 24 hours (Gerstley, Howell, Nagel, 1932)
    - if breast milk were again given exclusively, it would take 2-4 weeks for the intestinal environment to return again to a state favoring the gram-positive flora (Brown & Bosworth, 1922; Gerstley, Howell, Nagel, 1932)
- in susceptible families, breastfed babies can be sensitized to cow's milk protein by the giving of just one bottle, (inadvertent supplementation, unnecessary supplementation, or planned supplements), in the newborn nursery during the first three days of life (Host, Husby, Osterballe, 1988; Host, 1991)
    - infants at high risk of developing atopic disease has been calculated at 37% if one parent has atopic disease, 62-85% if both parents are affected and dependant on whether the parents have similar or dissimilar clinical disease, and those infants showing elevated levels of IgE in cord blood irrespective of family history (Chandra, 2000)
    - in breastfed infants at risk, hypoallergenic formulas can be used to supplement breastfeeding; solid foods should not be introduced until 6 months of age, dairy products delayed until 1 year of age, and the mother should consider eliminating peanuts, tree nuts, cow's milk, eggs, and fish from her diet (AAP, 2000)
- in susceptible families, early exposure to cow's milk proteins can increase the risk of the infant or child developing insulin dependent diabetes mellitus (IDDM) (Mayer et al, 1988; Karjalainen, et al, 1992)
    - the avoidance of cow's milk protein for the first several months of life may reduce the later development of IDDM or delay its onset in susceptible individuals (AAP, 1994)
    - sensitization and development of immune memory to cow's milk protein is the initial step in the etiology of IDDM (Kostraba, et al, 1993)
        - sensitization can occur with very early exposure to cow's milk before gut cellular tight junction closure
        - sensitization can occur with exposure to cow's milk during an infection-caused gastrointestinal alteration when the mucosal barrier is compromised allowing antigens to cross and initiate immune reactions
        - sensitization can occur if the presence of cow's milk protein in the gut damages the mucosal barrier, inflames the gut, destroys binding components of cellular junctions, or other early insult with cow's milk protein leads to sensitization (Savilahti, et al, 1993)

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