Best-feeding the baby

Human infants should be fed their own mothers’ breast-milk. Where this is unavailable, replacement feeding becomes necessary. Through the ages and right up to the present, human milk has been supplied by other lactating women within or from outside the family. Donated breast-milk has been used extensively in milk banks, and numerous examples are known of women successfully initiating breast-feeding of unrelated infants despite not having recently been pregnant.

Nevertheless, genuine replacement feeding is frequently required. This should aim to totally satisfy the infant’s nutritional requirements for at least the first 4 - 6 months of life. Initially milk from a variety of domestic animals had to be used, but from the early 20th century advances in food processing technology allowed the progressive modification of cow’s milk for the development of modern infant formulas (which now have to comply with the standards set in the Codex Alimentarius since 1981).

Together with the introduction of the feeding bottle and teat, which enabled easy artificial feeding, this resulted in a rapid decline of breast-feeding by the mid-20th century. Although advances in knowledge about the real advantages of human breast-milk above formula have now led to breast-feeding once again being the chosen method of feeding for most educated and affluent women in developed countries, breast-feeding rates are still declining in many areas.

Even though modern formulas have a gross composition similar to that of human milk, numerous differences remain. Their complete evaluation now requires assurance of nutritional, biochemical and physiological function far beyond mere growth and weight gain.

Breast-fed babies grow more slowly than bottle-fed infants within the first year of life, but this appears to have no long-term influence on growth or final height, and may indeed be protective in the longer term. Accumulating evidence points to nutritional programming, with long-term beneficial effects of breast-feeding in the reduction of risk for adult disease including obesity, hypertension and coronary vascular disease.

The reported protective effects of breast-milk extend far beyond nutritional aspects to neurodevelopment and learning, gut mucosal ontogeny, allergies, and immunity and prevention of infection.

A large number of humoral, cellular and anti-inflammatory factors combine in colostrum and mature breast-milk to promote development, maturation and protection of the gut mucosa and its immune system as well as the resident microflora. These include sIgA, lactoferrin, lysozyme, lactoperoxidase, monoglycerides and non-esterified fatty acids, long-chain polyunsaturated fatty acids, nucleotides, trophic factors and hormones, as well as numerous complex oligosaccharides, glycoproteins and mucins.

These numerous protective effects have led to the recognition that breast-milk is in fact a solution of biologically active protective agents with nutritional effect. Indeed, recent modifications in infant formulas have been aimed at making longer-term functional outcomes in babies fed formula more comparable to those in babies fed breast-milk.

The reported protection afforded by breast-feeding against gut, urinary, respiratory and ear infections is based primarily on the above immune protective factors but also on the avoidance of unsafe alternative feeds.

The recent attention drawn to outbreaks of neonatal infection caused by Enterobacter sakazakii from contaminated infant formulas has again highlighted the fact that formula production is not a microbiologically sterile process. Bacterial contamination of feeds can occur through contaminated formula powder, during unhygienic reconstitution or mixing with unsafe water, after failure to clean the feeding utensils properly and if formula is left to stand in bottles after reconstitution or after feeding.

The article by Joosten and Lardeau (p. 87) in this issue describes deliberate bacterial contamination of commercially available formula feeds in an imitation of the last scenario quoted above, and shows the bacteriostatic effect of a low pH, particularly when incubated at 37°C.

This finding is not unexpected but is clinically applicable only in the circumstances described. Feed contamination leads to infection, but has to survive gastric acidity to do so. All normal infants, including premature ones, are able to maintain an intragastric pH below 4 from the first day of life as one of the important barriers against infection. Where pathogens do traverse the stomach, buffering in the duodenum tends to negate pH-mediated protection. Heyland et al. showed that acidification of enteral feeds to pH 3.5 in critically ill patients was followed by significantly less gastric colonisation, but could not demonstrate improved clinical outcome.
Manufacturers will continue to modify and improve infant formulas, but cannot hope to emulate the individualised protection and unique biological advantages of the infant’s own mother’s breast-milk. Breast-feeding will remain best feeding.17

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The article by Joosten and Lardeau in this issue of SAJCN (p. 87) looks at the microbiological safety of acidified infant formula feeds as tested in vitro. The authors rightly point to diarrhoea as being one of the leading causes of morbidity and mortality among infants in developing countries, especially those under the age of 5 years. Such diarrhoea is often associated with poor hygiene conditions and subsequent contamination of made-up infant feeds with potential enterobacterial pathogens. It is known that breast-feeding reduces exposure to these potential pathogens as well as supplying the infant with appropriate nutrients and protective antibodies, so decreasing the incidence of diarrhoea. Breast-feeding remains the method of choice. However, where this is not possible (and there may be good reasons for this), formula feeding may be necessary. If formula feeding is to be undertaken, it is advisable that measures be put in place to minimise contamination of the made up feed from potential enteropathogens. In the real world, boiling of the water used to reconstitute such feeds and disinfection of feed bottles is not always possible or easy.

The authors point to acidification of the feed artificially as a possible way to prevent contaminating organisms from proliferating. This is achieved by the addition of lactic acid to such formulas. The in vitro testing carried out to corroborate such a supposition pointed to an acid pH of less than 5 as being bacteriostatic for most enteropathogens and even bactericidal for some. It was interesting to note that the organisms used in the in vitro test procedures were all common potential pathogens of the gastro-intestinal tract and included members of the Enterobacteriaceae as well as Staphylococcus aureus, Bacillus cereus, the yeast Candida albicans and a rotavirus.

After the inoculation into appropriate media, these were then incubated at temperatures of 4, 25 and 37°C. Growth or inactivation of the various organisms by the lowered pH was then assessed. Anti-rotavirus activity was determined via an alpha-type neutralisation test against two rotavirus serotypes.

The important observation of the lowered pH on organism growth and inactivation may provide a safe alternative to the usual aseptic techniques in preparing infant formula feeds, where such precautionary measures are not possible. However, as correctly indicated by the authors, further work will be necessary to confirm the clinical relevance of such in vitro findings and whether the formula ingredients are in any way adversely affected by the low pH.

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Further reading

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