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Breast feeding and atopic eczema

Generally mild and transient, atopic eczema may be one of the most agonising of all diseases. Its incidence appears to be rising alarmingly in Britain: 12·3% of a group of 12 555 children born in a single week in 1970 were reported by their parents to have had atopic eczema by the age of 5 years,[1] more than twice the proportion reported in a similar study 12 years previously.

The pathogenesis of atopic eczema has not been established, though dietary factors play an important causal part, at least in early childhood.[2] The mechanism by which foods can exacerbate the disease is unknown, but allergy is widely believed to be the most likely explanation. Alternatively it may be an entirely metabolic disorder, the associated immunological disturbances being merely epiphenomena.

Certainly susceptibility to the disease is, to a great extent, inherited. Current data suggest that a history of any atopic disease in one parent roughly doubles a child's risk, and such a history in both parents doubles it again.[3] The concordance rate of 15% for monozygotic twins compared with just 4% for dizygotic twins both confirms the genetic contribution and highlights the crucial role of environmental influences.[4] Genes appear to determine an infant's predisposition to atopic eczema, but the presence or absence of critical environmental factors probably determines whether or not the disease occurs.

Three quarters of all those who will develop atopic eczema do so within the first year of life. The apparent rarity of the disease in the neonatal period contrasts with its peak incidence during the next five months, implying that the most important environmental factors must be operating during these early months. Interest has naturally focused on the possible importance of what the baby eats, and in particular on whether the disease is more likely to occur in infants fed cows' milk rather than breast milk. The frequently beneficial effect of avoidance of cows' milk in the established disease[2] heightens the suspicion that infant feeding practices may have a preeminent role in its initiation. The first large scale survey along this line of inquiry was undertaken as long ago as 1936,[5] showing a sevenfold greater incidence of atopic eczema in infants fed wholly on cows' milk formulas compared with those exclusively breast fed, and a twofold greater incidence in those partially breast fed. Since that time similar studies have been reported with steadily increasing frequency and with very variable findings.[6-13]

Problems in the design of these studies may explain this inconsistency, at least partly. The methods used to diagnose the disease have differed widely; some studies relied on parental diagnosis alone, whereas in others eczema was diagnosed by general practitioners, paediatricians, or dermatologists—yet it may be exceedingly difficult, if not impossible, to distinguish primary irritant napkin dermatitis from atopic eczema. Since the two conditions are equally common clearly agreed criteria to attempt their separation are a prerequisite in this type of survey. In prospective studies parents had generally (and correctly) been left to select the method of feeding, rather than having it imposed by random allocation. Apart from the inevitable loss of "blindness," those with a family history of atopic eczema are almost certainly more likely to select to breast feed because of their awareness of the claimed protective effect—and this trend is likely to be particularly definite in mothers of higher socioeconomic class. Since a positive family history and high socioeconomic class are both associated with an increased risk of atopic eczema,[1] this might easily introduce a bias towards more eczema in the breast fed, which would in its turn tend to obscure any real protective effect.

In the face of such inconsistent data many doctors have been tempted to conclude that breast feeding is of no—or even negative—prophylactic value. Careful review of the published data shows, however, that prospective studies that have considered infants genetically at risk as a separate group and in which exclusive breast feeding has been continued for at least 12 weeks have fairly consistently shown a protective effect. This protective effect, however, is only relative; many exclusively breast fed infants develop atopic eczema. How long the protection lasts is also not clear, though two of the studies demonstrating this effect appeared to show continuing protection many months after stopping exclusive breast feeding.[8 9]

If, then, breast feeding is protective, what are the underlying mechanisms? The relative scarcity of food antigen in human milk has been widely assumed to be the principal reason for the less frequent development of eczema by breast fed infants. This theory explains the occasional development of eczema in such infants either as a result of early supplementation with cows' milk during the immediate perinatal period[14] or by the transmission of maternally derived food antigens to the infant in her milk. The importance of early supplementation with cows' milk has proved impossible to assess because of the difficulty of ascertaining whether this has occurred in the individual case. Since, however, early supplementation is likely to prejudice the successful establishment of breast feeding most good obstetric units have now abandoned it. Clearly some transmission of food antigens occurs

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in breast milk,[15] though the amounts appear to be very small indeed; some doubt must remain whether they could sensitise, though they can produce allergic symptoms in infants who are already sensitised.[16 17] The possibility cannot be excluded that initial sensitisation in such cases occurred in utero. Some babies have been shown to have circulating IgE antibodies to food antigens at birth,[18 19] and haemagglutinating antibodies to food antigen may be found in the amniotic fluid surrounding babies who develop eczema in infancy associated with clinical allergy to the same antigen.[20]

Other mechanisms might contribute to a protective effect from human milk. Infant feeding has been established as an important factor in experimental allergy in rats,[21] though the protective factor appears to be IgG antibody and is therefore probably irrelevant in man. Human milk does, however, contain secretory IgA, which reduces the entry of antigens at mucosal surfaces.[22] Human milk also contains a factor which accelerates the production of secretory IgA by the neonate.[23] Both are absent from cows' milk formulas. The potent adjuvant effect of bacterial toxin in the development of allergic responses in experimental animals[21] raises the possibility that toxin derived from the infants' microflora may play an important part in allergic sensitisation—and breast fed infants have a much less toxigenic gut flora than those fed cows' milk formulas.[24] Previous experience of an antigen causes lactating rats to transmit a factor in their milk which suppresses the development of an allergic response to the same antigen in their offspring[21]—though this finding is of questionable relevance for human disease. Nevertheless, further experiments on the same strain of rats have shown that early exposure to cows' milk also heightens subsequent allergic responsiveness to non-cows' milk antigens.[25] The responses of human neonates have not been shown to be analogous, but the concept that feeding cows' milk might predispose to allergy in an antigen non-specific way is obviously important.

What can be said at this stage is that the mechanisms of the protective effect of breast milk against development of atopic eczema and, conversely, of the provocative effect of cows' milk formulas are likely to be extremely complex. One question often raised is whether other infant feeds might provoke less eczema than cows' milk formulas. Particular interest has centred on soya "milks." One study designed to answer this question suggested that soya was superior in this respect to cows' milk[6] but a more recent study gave the opposite result,[26] and the question remains open. Infant feeds are now available based on hypoallergenic casein hydrolysate, and if the antigen load in the feed were the crucial factor these products might turn out to be effective in preventing atopic eczema—but the necessary studies have not been performed.

No firm, scientifically based answers are yet possible to the many questions that have been raised on this subject, but the clinician is still faced with the problem of providing advice to his patients. For the present, mothers-to-be of infants at risk (that is, where either parent or a sibling has a history of atopic disease) are best advised to plan to breast feed exclusively for four months and to continue to breast feed after weaning until the end of the first year. Lactating mothers often ask whether they themselves should avoid certain foods, in particular cows' milk and eggs. In the absence of reliable data it seems sensible to advise mothers of infants at risk to cut down their intake of these two highly allergenic foods, though not necessarily eliminating them obsessionally. If immunologically important amounts of food antigen reach the infant through human milk there is probably a threshold for maternal intake. In that case eggs as such should be avoided, though small amounts in cake and other foods could possibly be ignored. Similarly cows' milk should not perhaps be drunk raw, but small quantities in tea might be allowed. Any mother who wishes to manipulate her diet in this way should seek nutritional advice from a dietitian. At the very least, she will require supplementary calcium, of which a good source is a daily 3 g tablet of effervescent calcium lactate. If, for any of a variety of reasons, a supplementary or an alternative feed is required for the infant at high risk one based on casein hydrolysate (Pregestimil) may be best on theoretical grounds—though no clear beneficial effect has been shown in practice, such feeds are expensive, and they cannot at present be prescribed for this indication on an EC10.

DAVID J ATHERTON

Consultant Dermatologist,
Hospital for Sick Children,
Great Ormond Street,
London WC1N 1EH

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