waiting time for transfer is usually less than two weeks.) Is this then the evidence of change? . . . Most general physicians, and Dr Leonard may be an exception, are only too glad for geriatricians to look after the elderly patient with multifactorial problems—it is not that they are incapable; it is just that they do not see the benefits of appropriate investigation and treatment in many of these elderly patients, despite the fact that about 70 out of 100 patients admitted will go home in three months. . . .

I wish Dr Leonard was right, but experience shows differently; the failure of general physicians in the proper assessment of elderly patients, the failure to accept the need for a multidisciplinary team approach to illness in the elderly, the neglect of the "uninteresting patient," and the failure to accept responsibility for the "longer-stay" patient have been rampant. I see no evidence of a change of attitude by the majority of general physicians and therefore I must wholly disagree with the theme of his paper.

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Breast-feeding: the immunological argument

SIR,—No article advocating breast-feeding for babies can be totally bad, and your leading article (15 May, p 1167) giving the immunological case makes some valid points, but aspects of it are so unbalanced and potentially misleading that comment is essential in view of the importance of the subject.

The assertion that "non-specific factors are of minor importance compared with the neutralising properties of specific IgA" shows a fundamental lack of understanding of immunology. Antibody has little effect on bacteria without activating a non-specific mechanism, and it is likely that the antibacterial effects of milk are the more important ones. Bullen *et al*[1] have shown that IgA antibody and lactoferrin have an extremely powerful synergistic action and I understand that they now have an extremely elegant explanation of this phenomenon which is still unpublished. To think of the two separately and say that one is important and the other is not is therefore unsound and also important, since, if Bullen is right, addition of iron will largely inactivate the IgA system.

Omission of any mention of the relevance of the buffering power of milk feeds so elegantly studied by Mrs Bullen[2] is another example of lack of appreciation of the really important work, since some children will undoubtedly be artificially fed and there is little doubt that the damaging effect would be far less if these factors were taken into account in the design of artificial infant feeds.

The fascinating work of Barlow *et al*[3] is quoted, but their more recent demonstration[4] that live cells in rat milk contribute to the protection mechanism contrasts with your discussion of cells only as a possible cause of disease. This has important implications for planning milk banks (standard heat treatment would inactivate many of the other despised non-specific factors as well as the cells).

You describe our prospective study, already confirmed elsewhere, of transient IgA deficiency preceding hypersensitivity diseases as "still largely hypothetical." This is far more true of most of the other work you quote, and attempts to establish the usefulness of these ideas are already encouraging.[5] It is supported by the demonstration that there are already at least five genetically determined systems of variation of immunity function within the normal range, and that a low level of one of these is associated with defective antigen handling or with allergy and infection.[6] The neonatal period is the most critical part of life from the immunological point of view, since the immune response is initiated following establishment of the intestinal flora. The important implication of this work is that management which may be appropriate for 80% of children may well not be appropriate for the remaining 20%. In this period, when immunity function is really stretched, a considerable minority may be vulnerable to damage.

The account of the vast and heterogeneous picture of cows' milk allergy is strangely selective, and I shall require further conviction that there are not problems of polio immunisation in the breast-fed.

These are only a few aspects of a vast subject of established short-term and probable long-term importance for the health of the child—and the adult. It deserves a more accurate and balanced account.

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[1] Bullen, J J, *et al*, *British Medical Journal*, 1972, **1**, 69.
[2] Bullen, C L, *et al*, *British Medical Journal*, 1971, **3**, 338.
[3] Barlow, B, *et al*, *Journal of Pediatric Surgery*, 1974, **9**, 587.
[4] Pitt, J, *et al*, *Pediatric Research*, 1976, **8**, 384.
[5] Taylor, B, *et al*, *Lancet*, 1973, **2**, 111.
[6] Soothill, J F, *Proceedings of the Royal Society of Medicine*, In press.

**There is in fact little basic disagreement between the opinions expressed in our leading article and those raised in Professor Soothill's letter; many of the apparent differences can be attributed to the terseness unavoidable in an article of limited length. The first point concerns the relative importance of specific and non-specific antimicrobial factors transferred in human breast milk. The importance of the latter was fully recognised in our article, despite its emphasis on "the immunological argument." Of course, antibody does not operate in isolation. Nevertheless, there is some precedent in the history of immunology for believing that specific antibody is of critical importance in host defence against infections.

The work of Mrs Catherine Bullen and her colleagues on the buffering power of human milk is well known but was not cited because the article was not designed to be exhaustive but to deal primarily with immunological mechanisms. We await with interest the publication of the as yet unpublished work to which Professor Soothill alludes.

A balanced leading article tries to take into account primarily those findings which have been adequately documented and discussed in published work. The more recent work of Barlow and her colleagues has been published only in abstract form and is therefore as yet impossible to assess. If this criterion for inclusion had been strictly applied it would have been inadmissible to make adequate comments about the work of Professor Soothill and his colleagues, who claim that transient IgA deficiency precedes hypersensitivity diseases. Little of this has been published since the first paper by Taylor *et al* appeared in 1973. Nevertheless, this is a concept of such fundamental importance and originality that it was mandatory to discuss it, albeit with the proviso that it must be considered hypothetical until firmer evidence is available. The paper to which Professor Soothill refers and which is still in the press may strengthen his argument.

We drew attention to manifestations of milk allergy which are admittedly unusual; as space did not permit any exhaustive list it seemed appropriate to indicate the wide range of disorders which may follow unwise feeding in infancy, something to which Professor Soothill surely cannot object. Finally, we simply stated that there is no conclusive evidence one way or the other that breast-feeding interferes with polio immunisation, a feeling with which Professor Soothill concurs.—ED, *BMJ*.

Establishment of lactation

SIR,—Dr E Eastham (and his colleagues (7 February, p 305)) provide useful information on the educational approaches needed to allow mothers to reach the right decision regarding breast-feeding their infants. In their sample 17 out of the 100 mothers who breast-fed their babies initially had ceased to do so by the time they went home from hospital. Figures were given regarding the 63 primiparae in the sample who initially breast-fed and the conclusion was drawn that "clearly those who were better informed [about breast-feeding] were more likely still to be breast-feeding at the time of discharge [from hospital]." A 3×2 contingency table can be drawn up using these data. A χ^2 test indicates that no significant difference is apparent between those mothers who were "well informed," "uninformed," or "intermediate" ($\chi^2 = 1{\cdot}590$ at 2 degrees of freedom). Clumping of the data into a 2×2 table still gives no significant difference. Perhaps another factor is more important.

The interviewers in this study assessed the reasons why 14 of the primiparae ceased breast-feeding before leaving hospital. In eight cases the interviewers thought this was due to either nipple damage, the baby's behaviour, or insufficient milk. In other words, over half these early lactation failures were blamed on anatomy, physiology, or the baby. Can this really be so? Cracked nipple is hardly ever a contraindication to breast-feeding[1]; the milk normally takes a day or two and sometimes longer to come down[2]; and how does a newborn baby indicate that it prefers the bottle to the breast?

Perhaps the statement that "breast-fed babies were given complementary feeds when their behaviour indicated it" provides a clue as to one of the reasons why breast-feeding stopped while some sample mothers were still in hospital. Stimulation of the nipple and areolar tissue and suckling trigger the let-down and prolactin reflexes,[2] the establishment and maintenance of which are essential for successful lactation.[3] Consequently anything that inhibits early attempts by the newborn to suckle will reduce the chances of successful breast-feeding being established. The administration of prelacteal or complementary feeds in hospital is such an inhibitor to suckling. Its effect will be compounded when the mother interprets it as a judgment by the nursing staff that she cannot provide adequate milk for her newborn child.

Double-blind studies to investigate the influence of prelacteal feeding in hospital on subsequent lactation performance and growth