days. There were 58 breast-fed and 20 bottle-fed infants. The latter were fed a formula of humanized cows' milk (Similac-20). Though the lactose content of this formula is almost identical to that of human milk (7.2 vs. 7.0 g/100 ml, respectively), as is shown in Tables 1 and 2,

Table 1 Reducing substances in stool (Clinitest)

Reducing substances (%)	<0.5	0.5-2.0	Total
Breast-fed	8	50	58
Similac-20	16	4	20
Total	24	54	78

$\chi^2 = 31.02$, $P < 0.001$.

Table 2 Stool pH

Stool pH	4.4-6.0	6.1-7.2	Total
Breast-fed	52	6	58
Similac-20	7	13	20
Total	59	19	78

$\chi^2 = 23.89$, $P < 0.001$.

significantly more breast-fed neonates had 0.5% or more reducing substances in their stool as well as acid stool (pH between 4.4 and 6.0). As was observed by Counahan and Walker-Smith, in our series also estimation of stool pH was an unreliable index for lactose absorption. The caloric content of human milk is identical to that of Similac-20 (67 cal/100 ml). Since the average weight gain during the first 2 weeks of life of the formula-fed infants exceeded that of the breast-fed infants, food consumption, and therefore lactose intake of the former might have been even greater. Thus, in our study lactose intake was not a determinant in the trend for higher levels of reducing substances and lower pH in the stools of breast-fed neonates. Further investigations will be required in order to determine the exact contribution of the different gut flora in breast-fed infants to the observed differences in stool sugar content and pH.

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Dr. Walker-Smith comments:

I was pleased to see the letter of Drs. Nitzan and Rosenfeld confirming our finding that breast-fed infants have increased stool reducing substances. This observation supports the view that breast-fed babies have a temporary 'physiological malabsorption' of lactose. I agree that there is not a higher concentration of lactose in breast milk as compared to the artificial feeding formula used,

and so this cannot account for the greater proportion of breast-fed infants who have increased levels of stool reducing substances. Some other explanation must be sought, including the contribution of gut flora, but the inter-relationships between faecal sugar, pH, and flora is likely to be very complex. Whether the primary event in this inter-relationship is unabsorbed lactose entering the colon of breast-fed infants, influencing their colonic pH and flora, or whether small intestinal flora in such infants modifies the absorption of lactose is unknown and warrants investigation.

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Haemolytic uraemic syndrome in sibs

Sir,

The occurrence of multiple cases of a disease in some families does not by itself provide irrefutable evidence for genetic predisposition for that disease. This appears to be the case in the haemolytic-uraemic syndrome (HUS), which has been reported to affect multiple sibs in some families (Kaplan *et al.*, 1975). The reports of simultaneous occurrence of the disease in unrelated adopted sibs (Chan *et al.*, 1969) and the presence of asymptomatic mild cases in family contacts (Tune *et al.*, 1974), along with a well-recognized predilection of the disease to appear in endemic (Gianantonio *et al.*, 1964) and epidemic (McLean *et al.*, 1966) forms in various parts of the world, however, appear to implicate environmental agents, namely infections, as causative factors.

In a review of 41 sibships with multiple cases of HUS, Kaplan and associates (1975) could distinguish two groups of families on the basis of (1) the interval between onset of disease in affected sibs, (2) geographical location (endemic versus nonendemic) of the families and, (3) severity of the syndrome as indicated by mortality. The authors speculated that the milder disease occurring simultaneously in sibs in families living in endemic areas could be attributed to environmental agents, while genetic factors may have played an important role in severe form of the disease occurring in sibs after longer intervals in families living in nonendemic areas.

We have seen a New York City family in which 2 male infants were affected with HUS with an interval of 2 years between the cases. The parents were healthy non-consanguineous black Americans. Both parents and 2 sisters (7- and 1-year-old, respectively) were in good health. The first infant was born in December 1970 and died in May 1971, the second was born in February 1973 and died in August 1973. The clinical picture included gastroenteritis, haemolytic anaemia, disseminated intravascular coagulopathy, hypertension, and renal failure. Necropsy was performed on the second infant only, and showed widespread fibrin thrombi in arterioles and capillaries throughout both kidneys.