Correspondence

Archives of Disease in Childhood, 1976, **51**, 159.

Fat absorption from two infant milks

Sir,

On the general principle of stimulating a healthy competitive spirit among the infant milk firms, we changed from SMA Ready-to-feed to Cow and Gate V-formula in July 1974, for the routine feeding of bottle-fed infants in this hospital. I was interested to read the report from Milner *et al.* (*Archives*, 1975, **50**, 654) that they had shown fat malabsorption in infants on V-formula compared with SMA, since this tended to confirm the feelings of some of our staff that babies thrive better on the latter.

However, an investigation of the rates of weight gain of babies fed with the two milks does not confirm our fears about the suitability of V-formula for feeding small infants, nor does it suggest that net energy ingestion is significantly less in V-formula-fed babies. I have examined the weight gains in all immature appropriately-grown babies admitted to our Special Care Baby Unit during the periods June 1973-June 1974 (SMA), and July 1974-July 1975 (V-formula). There were no other changes in the routine management of healthy immature infants during this time. Weight gains were calculated by the method of least squares during two periods in each infant, unless these periods overlapped by more than 50%, in which case the first was used: (1) days 15-28 inclusive, (2) the period between the attainment of 2000 g body weight and 2300 g body weight (the usual discharge weight). Results are given in the Table and show no significant difference in weight gain on the two milks during the first period, and a significantly more rapid weight gain on V-formula during the second period.

It is difficult to sustain the view that there is any long-term disadvantage in the use of V-formula in comparison with SMA. I suggest that the fat malabsorption observed by Milner and his colleagues during V-formula feeding in their infants may have been the result of carrying out the fat balances too soon after changing milks. It is conceivable that malabsorption of saturated fats such as C16:0 may be temporary, and the infants may need several days of exposure to the increased level of intake to optimize intraluminal or mucosal digestion. Studies on high fat diets in preterm infants which I am carrying out in this unit tend to support this, since net energy ingestion increases in most infants during the whole of a 10-day period on high-fat feeding.

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Prof. R. D. G. Milner comments:

Thank you for giving me the opportunity to respond to the letter by Dr. O. G. Brooke.

We agree with Dr. Brooke that infants fed Cow and Gate V-formula gain weight as fast as those fed SMA Ready-to-feed formula. The Editors will recollect that evidence supporting this viewpoint was part of the original manuscript submitted for publication but was finally omitted in the light of the referee's report.

I think that Dr. Brooke's suggestion that fat malabsorption may have been the result of carrying out the fat balances too soon after changing milks is unlikely

TABLE
Results of weight gain

	SMA	V-formula
No. of infants	26	33
Birthweight (g $\pm$ SD)	1724* ($\pm$ 359)	1698* ($\pm$ 246)
Gestational age (w $\pm$ SD)	33.4* ($\pm$ 2.9)	33.7* ($\pm$ 2.0)
Days 15-28		
Intake (ml/kg per d)	207* ($\pm$ 16.4)	206* ($\pm$ 14.1)
Daily weight gain (g/kg per 100 ml feed)	7.5* ($\pm$ 1.7)	8.1* ($\pm$ 1.7)
2000-2300g bodyweight		
Intake (ml/kg per d)	199* ($\pm$ 11.3)	194* ($\pm$ 8.9)
Daily weight gain (g/kg per 100 ml feed)	7.4† ($\pm$ 1.5)	8.3† ($\pm$ 1.5)

*NS

† t = 2.25, P < 0.05.

to be correct. The intestinal transit time of newborn infants is short enough to make 3 days a satisfactory period to establish equilibrium. Furthermore, our experimental design took account of this possibility in that half the babies were fed one milk first and the remainder in the reverse order. Cow and Gate V-formula produced more steatorrhoea when given before or after SMA Ready-to-feed formula.

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Peripheral gangrene in hypernatraemic dehydration of infancy

Sir,

In their article (*Archives*, 1975, 50, 616) on peripheral gangrene in hypernatraemic dehydration of infancy, Drs. Comay and Karabus state, 'Therapy . . . should be directed primarily towards eliminating the cause', and suggest 'treatment of dehydration'. Surely the cause is the dehydration, and elimination of this cause by preventing dehydration occurring would be a more effective form of therapy. Drs. Comay and Karabus state that 36 000 dehydrated children were admitted to their hospital over a 3-year period, and this can only mean that the standard of primary medical care and child health services around their hospital are very poor. If they wish to eliminate peripheral gangrene in hypernatraemic dehydration of infancy, should they not start by raising the standards of service which is being offered in their community?

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Congenital myotonic dystrophy

Sir,

The articles on congenital myotonic dystrophy by Harper (1975) and by Simpson (1975) describe many interesting signs of this disease. However, they fail to mention whether thin ribs were observed on chest x-rays. We (Fried *et al.*, 1975) have found thin ribs as a very helpful sign in the diagnosis of neonatal myotonic dystrophy but more data are needed. In Simpson's case 'Chest x-ray showed normal lung fields', and in Harper's report 33 cases had 'respiratory problems' in the neonatal period. It is hoped that at least some of the original chest x-ray films are still available and will be published in order to get some estimate on the frequency of thin ribs in neonatal myotonic dystrophy. Furthermore, in those patients who survived the neonatal period and had this sign, we may learn at what age this sign disappears.

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Harper, P. S. (1975). Congenital myotonic dystrophy in Britain. I. Clinical aspects. *Archives of Disease in Childhood*, 50, 505.
Simpson, K. (1975). Neonatal respiratory failure due to myotonic dystrophy. *Archives of Disease in Childhood*, 50, 596.

Dr. P. S. Harper comments:

Thin ribs were neither observed nor reported on in chest radiography of patients in the series, but the sign was not specifically sought and the films will be reviewed. Dr. Fried's observation is of interest and is another factor supporting the prenatal onset of abnormalities in most of these infants. An explanation of its occurrence could be hypoplasia of the intercostal muscles, comparable to the diaphragmatic hypoplasia observed in some of our patients, and to the reduced fetal swallowing probably responsible for the high incidence of hydramnios. It would seem more likely that the thin ribs are a secondary aspect of this widespread intrauterine muscle hypoplasia, rather than a primary abnormality of bone.

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