Fig. Changes in serum calcium, phosphorus, alkaline phosphatase, and 25-hydroxy-vitamin D during the consumption of vitamin D-fortified chupatti flour.

effectiveness in flour and that a substantial proportion survives the cooking process involved in preparing chupatties. Storing the flour for up to 16 months before use did not appear to affect its potency.

Chupatti flour is consumed by the bulk of the Asian population of the United Kingdom and its use begins soon after the introduction of mixed feeds in early childhood. It provides an excellent vehicle for the selective fortification of a foodstuff consumed by the population at risk. The minority of Asians who are predominantly rice-eating or who adhere to a European diet do not in our experience develop rickets or osteomalacia outside infancy.

We have recently completed a trial of vitamin D-fortified butter in Asian subjects. Fortification to 'margarine' levels (100 units/oz) over a period of a year did not raise the serum 25-hydroxy-vitamin D levels of those consuming it; butter fortified to 400 units/oz was effective but this level of fortification is unlikely to be acceptable in a foodstuff consumed by the whole population. Milk remains an alternative, and so far untried, vehicle in the

UK but, as with butter, fortification would affect the whole population and the necessary enabling legislation permitting fortification might therefore be open to more objection and delay.

On balance, the fortification of chupatti flour with vitamin D offers the best hope of eliminating rickets and osteomalacia from the Asian population of the UK outside infancy; the procedure is cost-effective and technically feasible. The implementation of this measure for a limited period of years, with monitoring at regular intervals of serum 25-hydroxy-vitamin D levels in selected consumers seems to us worthy of trial without undue delay.

We are indebted to Spillers Ltd. and especially Mr. B. Hartley for providing vitamin D-fortified chupatti flour.

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Congenital thrombocytopenia and milk allergy

Sir,

Further to the article by Whitfield and Barr regarding thrombocytopenia and milk allergy in a child with an absent radius (TAR) (*Archives*, 1976, **51**, 337), the following account of a newborn with congenital thrombocytopenic purpura and milk allergy, who was otherwise physically normal, may be of interest.

In October 1973 a term, healthy boy was born in this hospital to a mother who had mild toxæmia and long-standing idiopathic thrombocytopenic purpura. Scattered petechiae were present over his body at birth and cord

blood examination showed a Hb of 14.8 g/dl and platelets of $11\,000/\text{mm}^3$ ($11.0 \times 10^9/\text{l}$). Treatment was started immediately with steroids (IM hydrocortisone with oral prednisone initially and subsequently prednisone alone). A few hours after birth the baby became extremely irritable and developed a high pitched cry with an accompanying increase in petechiae and a further fall in platelets. One unit of platelet concentrate was administered.

The course over the following 3 weeks was unremitting. Platelet levels varied from 3000 to 6000 ($3.0\text{--}6.0 \times 10^9/\text{l}$) and there was corresponding fluctuation in the number of petechiae and ecchymoses present. Steroids were continued and occasional units of platelet concentrate given when there was evidence of complications, such as melaena. During the fourth week of life the platelet level stabilized and gradually steroids were reduced and finally discontinued on the fifth week. Feeding was started at birth with Cow & Gate formula which he had continued to tolerate well.

From the fifth to the eleventh week, the platelet level rose to between 10 000 to 17 000 ($10.0\text{--}17.0 \times 10^9/\text{l}$) though there were occasional drops to about 5000 and there continued to be fresh crops of petechiae. On 23 December 1973, aged 11 weeks, he developed a mild fever and loose stools. Milk feeds were discontinued and he was given clear fluids. The following day the platelet level was 11 000 ($11.0 \times 10^9/\text{l}$). Because of the diarrhoea (later found to be due to *E. coli*), he was kept on clear fluids for 3 days and then gradually started on half-strength milk feeds. Platelet levels were determined 5 days from the onset of this illness and the level had risen to 135 000 ($135 \times 10^9/\text{l}$) which was almost 120 000 higher than any that had previously been recorded. This was repeated the following day and was 150 000. By this time, the infant's diarrhoea had subsided and normal feeding had been resumed. Platelet levels were determined during the following 10 days and there was a continuous fall to 30 000. The significant and dramatic rise in the level of platelets that had occurred during the period on a milk-free diet and the fall which occurred after the reintroduction of normal milk feeds raised the possibility that the thrombocytopenia was aggravated by milk. Consequently milk was completely withdrawn from his diet. There followed a gradual increase in the platelet level, which 3 weeks later had reached 200 000 ($200 \times 10^9/\text{l}$).

Fig. Haematological response to milk-free diet.

The child was subsequently reviewed in the outpatient department but had no recurrence of purpura and his general development appeared normal. When seen aged 1 year, the platelets were 300 000 and his mother was advised to reintroduce milk gradually to his diet. He returned for a further review 3 months later, when his mother reported that she had attempted on two occasions to reintroduce milk but on both occasions he had developed severe wheezing and bruising.

There is evidence, therefore, that the withdrawal of milk in this case produced an improvement in idiopathic thrombocytopenic purpura and reintroduction of milk haematological relapse on 2 occasions. On two further occasions reintroduction of milk produced wheezing and bruising (no platelet levels were determined on either occasion). It is possible, therefore, that even in the absence of any specific manifestations of milk allergy (at least initially) in this case of idiopathic thrombocytopenic purpura, milk may have exacerbated the haematological changes and clinical picture.

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