small numbers. It is perhaps debatable whether hypothyroidism should be classed as a congenital malformation. If this case is excluded then the figures are $\chi^2 = 7.98$, $P = 0.005$.

Our data on head circumferences have been misinterpreted by Campbell and Sullivan. We did not claim that there was any statistical difference in head circumference of babies of non-drinkers compared with those of all drinkers. But there was a statistical difference when comparing the infants of mothers drinking over 20 ml alcohol daily at the gestational ages stated. The small numbers do invite caution in interpretation of the results, and this we expressed in the discussion in our paper.

We do not claim that there is a proved association between alcohol consumption and congenital malformation, but our data make us highly suspicious that this may be so. Similar studies of larger numbers of pregnancies are required.

Plasma 25-hydroxyvitamin D and rickets in low birthweight babies

Sir,

Dr McIntosh and his colleagues have recently drawn attention to the high incidence of rickets in babies weighing <1 kg at birth.¹ Eight of the 15 survivors in their unit in 1981 developed radiological rickets at 2–3 months when fed on breast milk, even though they were given 2000 units of vitamin D a day. They conclude that lack of vitamin D is unlikely to be the cause of these bone changes, and comment on the possible role of calcium and phosphate deficiency. We were surprised, therefore, that Dr McIntosh¹ and Dr Brooke² have not commented on the evidence in favour of calcium supplements published by Day *et al*³ in 1975 and Steichen *et al*⁴ in 1980.

We have not seen metaphyseal cupping of the type described by McIntosh in this hospital in the past 6 years, but we have had pathological fractures in 5 babies associated with marked osteoporosis⁵ 6 to 9 weeks after birth. All babies of birthweight <1 kg in this hospital have been fed on Gold Cap SMA since 1977 and all have had a daily supplement of 400 units of vitamin D. Four of the 10 survivors born between 1977 and 1980 suffered multiple pathological rib fractures on this diet, and 2 of these babies also had limb fractures that healed with appreciable angulation.

Because of this additional oral calcium supplements were introduced at the end of 1980 (total calcium intake 26 mmol/100 ml of milk). There have been no other changes in feeding policy in the past 6 years. We have not seen a single pathological fracture in any of the 10 very low birthweight survivors born since then. Nor have we detected any radiological or biochemical evidence of rickets. While there must be a 10% possibility that this change occurred by chance, it has certainly encouraged us to continue offering calcium supplements. It would be valuable to have these findings confirmed by another unit that cares for more of these babies.

It should perhaps be pointed out that there is only 1 commercial milk preparation on sale in the United Kingdom that provides as much calcium as was used in

the experimental studies of Day and Steichen, and that this contains too little sodium to be entirely satisfactory for babies of very low birthweight. We therefore use the same experimental milk formula for babies of <1.3 kg as was used in the studies reported by Day *et al*,³ and make this up as required from SMA milk concentrate and a mineral concentrate provided by the hospital pharmacy.

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Dr McIntosh and co-workers comment:

The conclusion of our paper on plasma 25-hydroxyvitamin D¹ in rickets in infants less than 1000 g was indeed that substrate insufficiency was the likely cause. We are aware of the data of Day *et al*² and of Steichen *et al*³ suggesting that calcium insufficiency is important, but are also aware of the case reported by Rowe *et al* of a breast fed infant who responded to additional phosphorus intake.⁴ We think that whether rickets of prematurity is due to calcium or phosphorus deficiency, or both, is still open to question. Our concern in our paper was to disprove that it was due to insufficiency of vitamin D.

In our paper we stated that 8 of the 15 survivors of <1000 g birthweight over a 1 year period developed radiological rickets. We did no formal balance studies on these infants, but if the enteral intakes of calcium and phosphorus in the 8 babies developing rickets are compared with those of the 7 babies who did not, there is a significantly lower intake of both these minerals in the group developing rickets at 27 and 28 weeks' postconception (see Figs. 1 and 2). Allowing for the enteral fat malabsorption in infants of this gestational age, the accumulation rate for both calcium and phosphorus must be orders of magnitude less than the in utero accumulation rate given by Shaw.⁵ We are embarking on more formal balance studies on this extremely high risk group of babies that we would still prefer to feed on breast milk. We propose, like McCowan and Hey, to add calcium and also phosphate as Senterre and his associates (personal communication, 1983) have shown that the addition of calcium alone predisposes to nephrocalcinosis.