Human milk in the modern world

Sir,

In her recent review of our book Mavis Gunther (whose work we have admired greatly) seems to have cast us as a blend of brainless (if amiable) zealots, fundamental evangelists, and self-deluded visionaries.

Perhaps our strong secular advocacy is because our perspectives are very different from hers. Our main concerns are with disadvantaged communities (mainly in the third world), where breast feeding is vital for survival in early childhood, rather than ideal as in industrialised parts of the world.

Dr Gunther has misconstrued several concerns expressed by us. For example, the phrase 'statutory legislation for breast feeding' used in her review suggests forced breast feeding camps. In fact, in our book we discuss the legislation that is needed to make it easier for working women to breast feed should they wish to do so—scarcely wild-eyed evangelism, as this is already the practice in some parts of the world.

In essence, what may be called a Calcutta-eye view of infant feeding is very different from a London-eye view. In the third world breast feeding is one of the main components of primary health care in relation to nutrition, water supply, infection, economics, self sufficiency in food production, and child spacing¹ and as such it deserves activist advocacy.

Reference

- ¹ Jelliffe D B, Jelliffe E F P. Breast feeding and primary health care: a post-Alma Ata view. *Ecology of Food and Nutrition*, 1980; **9**: 189.

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Dr Gunther comments:

I hope that the Jelliffes can bring themselves to read my review again. My words were 'statutory powers to enable working mothers to feed their babies' which to me mean that the choice remains with the mother. Surely we are saying the same thing?

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Treatment of hypophosphataemic vitamin D-resistant rickets

Sir,

We read with interest the paper by Seino *et al.*¹ Our experience with 1- α -hydroxy-vitamin D₃ (1 α OHD₃) in the treatment of this disease is limited to one patient—a 2-year-old girl. The diagnosis was based on rickets, hypophosphataemia, raised alkaline phosphatase, normal

serum calcium, and normal serum parathyroid hormone. She has been treated for 2 months with daily administration of 0.5 μ g 1 α OHD₃ and about 2 g phosphate. Radiological examination already shows partial healing of bone (Figure, a and b).

Contrary to Seino *et al.* we have only used small doses of 1 α OHD₃ and we are astonished that the high doses used by them should be necessary, particularly as the risk of side effects and the cost of the treatment are increased.

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Reference

- ¹ Seino Y, Shimotsuji T, Ishii T, *et al.* Treatment of hypophosphataemic vitamin D-resistant rickets with massive doses of 1- α -hydroxy-vitamin D₃ during childhood. *Arch Dis Child* 1980; **55**: 49–53.

Figure Roentgenograms of right knee, showing the changes in bone structure. (a) Before treatment, and (b) after 2 months' treatment.

² Seino Y, Shimotsuji T, Ishida M, Ishii T, Yamaoka K, Yabuuchi H. Vitamin D metabolism in hypophosphatemic vitamin D-resistant rickets. *Contrib Nephrol* 1980; **22**: 101-6.

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Somatomedin-C in the Beckwith-Wiedemann syndrome

Sir,

We refer to the paper by Spencer *et al.*¹ We have obtained fasting sera for growth hormone and somatomedin-C on 3 unrelated patients with classical features of the Beckwith-Wiedemann syndrome. The results are shown in the Table.

Case	Age	Sex	Growth hormone	Somatomedin-C* (U/ml)	
				Patient	Normal
1	3 weeks	F	—	0.1	0.2-2.0
2	1 year	F	2	0.19	0.2-2.0
3	8 years	F	12	0.26	0.4-2.0

*By radioimmunoassay.

The growth hormone determinations were performed by Bio-Science Laboratories, Van Nuys, California, and the Somatomedin-C determinations by Nichols Institute, San Pedro, California, which also supplied the normal range. The very low values of Somatomedin-C in our patients do not support the concept proposed by Spencer *et al.*¹ No explanation for the conflicting findings is offered.

Reference

- ¹ Spencer G S G, Schabel F, Frisch H. Raised somatomedin associated with normal growth hormone. A cause of Beckwith-Wiedemann syndrome? *Arch Dis Child* 1980; **55**: 151-3.

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Dr Spencer comments:

The results presented by Robinow and Shafer are interesting but it is difficult to explain why they conflict with ours. However, since submitting our article,¹ I have been able to test somatomedin activity in another patient with Beckwith-Wiedemann syndrome. The patient was 7 years old and had a bioassayable plasma somatomedin activity of 1.6 U/ml. This value, although not as high as that in the patient we reported earlier, is still raised compared with normal age-matched children; our range

Dr Seino comments:

As previously stated, the mean plasma levels of $1,25(\text{OH})_2\text{D}$ are significantly low in hypophosphatemic vitamin D-resistant rickets and increase after treatment, but remain relatively low in spite of large doses of $1\alpha\text{OHD}_3$ ($0.4-2.0 \mu\text{g/kg}$ per day).¹⁻² These results suggest that the metabolism of $1,25(\text{OH})_2\text{D}_3$ is accelerated in such patients. The relatively low level of $1,25(\text{OH})_2\text{D}$, even after administration of a massive dose of $1\alpha\text{OHD}_3$, indicates that large doses are necessary in these patients. Moreover, the low level of $1,25(\text{OH})_2\text{D}$ before treatment showed a positive correlation with the low level of serum phosphate and TmP/GFR .² This finding suggests that massive doses of $1\alpha\text{OHD}_3$ are necessary in severe cases. I suppose that the case referred to by Kristiansen and Pedersen was mild, as commonly occurs in females. The risk of hypercalcaemia is always present during treatment with vitamin D metabolites. However, hypercalcaemia or hypercalciuria (urinary calcium/creatinine appears to be the best indicator) is easily controlled by reducing the dose of $1\alpha\text{OHD}_3$ because it has a short half-life.

References

- ¹ Seino Y, Shimotsuji T, Ishii T, *et al.* Treatment of hypophosphatemic vitamin D-resistant rickets with massive doses of 1α -hydroxy-vitamin D_3 during childhood. *Arch Dis Child* 1980; **55**: 49-53.