transdermal loss of water and in our cases absorption of urea.

We hope that our cases and those of Dr Garty will remind our colleagues that skin is not an impervious barrier.

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Vitamin D metabolites in idiopathic infantile hypercalcaemia

Sir,

We have further investigated two of the infants with idiopathic infantile hypercalcaemia described by Martin *et al*¹ and confirmed the presence of appropriate suppression of 1 α hydroxylase activity in the presence of hypercalcaemia, suggesting intact feedback mechanisms.

As vitamin D supplements (400 IU/day) were withdrawn and a low calcium diet begun in case 1 the serum calcium concentration fell. Concentrations of 1,25-dihydroxycholecalciferol (1,25(OH)₂D) rose and 25-hydroxycholecalciferol (25-OHD) fell due to the withdrawal of supplements (Table 1).

Calcium load tests, modified from the protocol of Barr and Forfar,² showed an abnormal response when per-

formed at 7, 13, 18, and 24 months in case 1 and at 22 months in case 2. Furthermore, 1,25(OH)₂D concentrations performed during the most recent load tests showed appropriate suppression in the presence of hypercalcaemia (Table 2), suggesting intact homeostatic mechanisms.

Our assay is able to distinguish between 1,25(OH)₂D₂ and 1,25(OH)₂D₃ and it would seem that 1,25(OH)₂D₃ is more tightly controlled than 1,25(OH)₂D₂. The significance of this observation is unknown as these metabolites have not previously been measured in children with this condition. Further studies will be necessary to confirm our findings.

Dr Martin suggested that the low concentrations of 1,25(OH)₂D seen in the patients with hypercalcaemia could be due either to appropriate suppression of 1 α hydroxylase activity or to a reduced growth velocity. The results of the loading studies and the fact that the growth velocity of our first child did not alter with correction of the hypercalcaemia suggests that the former is a more likely explanation. Whether there is poor control of production of 1,25(OH)₂D₂ and whether this is important in the aetiology of the hypercalcaemia is an area for future study.

I thank Dr Barbara Mawer, Manchester University, for performing the assays.

References

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Table 1 Reaction of serum calcium and vitamin D concentrations to withdrawal of vitamin D supplements in case 1

	At diagnosis	Time after treatment		
		2 weeks	4 weeks	7 months
25-OHD (ng/ml)	48.6	53.5	40.6	30.7
1,25(OH) ₂ D (pg/ml)	31.0	38.0	47.0	69.0
Calcium (μ mol/l)	3.16	2.38	2.47	2.44

25-OHD=25-hydroxycholecalciferol; 1,25(OH)₂D=1,25-dihydroxycholecalciferol.

Table 2 Vitamin D metabolites during calcium loading in the two cases

	Fasting	Calcium loading (hours)					
		0.5	1	1.5	2	3	5
Case 1							
Calcium (μmol/l)	2.44	2.77	—	2.86	—	2.79	—
1.25(OH) ₂ D ₂ (pg/ml)	6	7	—	7	—	5	—
1.25(OH) ₂ D ₃ (pg/ml)	63	55	—	53	—	61	—
Case 2							
Calcium (μmol/l)	2.53	—	3.37	—	3.23	3.15	2.79
1.25(OH) ₂ D ₂ (pg/ml)	21	—	21	—	20	20	18
1.25(OH) ₂ D ₃ (pg/ml)	60	—	57	—	56	47	34

1,25(OH)₂D₃=1,25-dihydroxycholecalciferol.