Incidence of symptomatic vitamin D deficiency
A L Callaghan, R J D Moy, I W Booth, G Debelle, N J Shaw

There was an overall incidence of 7.5 per 100 000 children, with those of south Asian ethnic origin having an incidence of 38 per 100 000, and children of black African or African-Caribbean ethnic origin having an incidence of 95 per 100 000. Only one child of white ethnic origin was reported, giving an incidence of 0.4 per 100 000.

DISCUSSION
This study has provided useful quantitative evidence for the incidence of asymptomatic vitamin D deficiency in young children in the United Kingdom in the early part of the 21st century. The only comparable study in a child health clinic in Manchester between May 2001 and July 2002 identified two of 124 ethnic minority children with rickets between the ages of 6 and 36 months, giving a prevalence of 1.6%. The results of our survey are likely to be applicable to other areas of the UK with large ethnic minority populations.

The figures we identified are probably an underestimate as nearly a quarter of the paediatricians did not respond, and a separate survey of children under the age of 16 years presenting to three Birmingham hospitals between June 2001 and June 2003 identified 65 individuals. In addition we recognise the limitations of case ascertainment using a single method of reporting in determining the true incidence of a condition. This survey has highlighted three modes of presentation in this age group. Although the majority presented with bowed legs as toddlers, a quarter of the children presented with hypocalcaemic convulsions under the age of 6 months. In this latter group, who were all south Asian, maternal vitamin D deficiency was identified as an aetiological factor in many cases. This observation calls into question the recent NICE guideline on antenatal care which does not recommend routine vitamin D supplementation for pregnant women. The third mode of presentation was with gross motor delay, particularly delay in walking. All these children were black African or African-Caribbean in whom prolonged and often exclusive breast feeding was present. Breast milk is a poor source of vitamin D, containing only 40 IU/litre. Such children often have a significant proximal myopathy and it is conceivable that the

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Figure 1 Mode of presentation of symptomatic vitamin D deficiency.
child who died may have had a cardiomyopathy, which is a potential consequence of severe vitamin D deficiency.5
This survey highlights that the recommendations of the Department of Health for vitamin D supplementation are being ignored.6 Although the majority of the children were being breast fed it is pertinent to note that 50% of those who presented with hypocalcaemic convulsions were formula fed. This suggests that the vitamin D content of infant formulas is inadequate to compensate for the impact of maternal vitamin D deficiency during pregnancy.

It is our intention to repeat this survey in the West Midlands in a few years time following a renewed public health campaign to ensure appropriate vitamin D supplementation to vulnerable groups, including pregnant mothers, in order to reduce the incidence of this preventable condition.

ACKNOWLEDGEMENTS
We acknowledge the Department of Rheumatology for providing a list of consultant paediatricians and the West Midlands Public Health Observatory for the 2001 census data.

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REFERENCES

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