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LETTERS

VITAMIN D DEFICIENCY

Authors' reply to Nussey

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We read Professor Nussey's response to our position statement with interest.[1 2] We are not a committee but a current comprehensive group of clinicians who manage children with bone disease in the UK. Our opinions are based on our combined clinical experience of vitamin D deficiency in infants, children, and adolescents across the UK. Our statement was a concise expression of our position rather than an exposition of the evidence. However, careful consideration of the extant literature underpins our statement (although we acknowledge the paucity of studies that examine clinical outcomes in relation to serum 25-hydroxyvitamin D_3 (25-(OH)D_3) concentrations).

Our cut-off value for 25-(OH)D_3 (25 nmol/L) is that used by the National Institute for Health and Clinical Excellence for deficiency. It is based on systematic reviews undertaken by the Cochrane Collaboration, the Scientific Advisory Committee on Nutrition, the Institute of Medicine, and the Department of Health for "low vitamin D status" (endorsed in the recent letter from the UK chief medical officers).[3] Nonetheless, rather than this value (which depends partly on the laboratory method used) alone determining the development of rickets, other crucial factors need to be taken into account, including low serum phosphate or low dietary intake of calcium resulting in an increase in serum parathyroid hormone. In contrast to the adult autopsy data cited, Edouard and colleagues found no evidence of defects of bone mineralisation in bone biopsies from 37 children with serum 25-(OH)D_3 concentrations of 13-50 nmol/L.[4] It is true that we have not looked at the important question of whether current recommended doses of vitamin D raise serum 25-(OH)D_3 concentrations adequately, but there is not yet an agreed definition of "adequate."

Nussey provides no references to support the statement that a few children with rickets have "several" fractures. The evidence suggests that, if fracture occurs in a mobile child with rickets, a single fracture is the norm. The national diet and nutrition survey showed that up to 30% of South Asian toddlers in the UK have a 25-(OH)D_3 concentration less than 25 nmol/L.[5] Vitamin D status in infants in this group is probably the same or even lower. If serum 25-(OH)D_3 less than 25 nmol/L were associated with fragility fractures, we would expect to see hundreds of cases of fractures of ribs or proximal humeruses captured on the thousands of chest radiographs performed for indications such as bronchiolitis each year. This is not our experience. We also know that the prevalence of clinical signs of rickets is far lower than the incidence of deficiency (<1/1000).[6]

We stand by our original statement and trust that the guidance provided will be useful for clinicians managing children with vitamin D related problems.

Competing interests: None declared.

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2 Nussey SS. Position statements on vitamin D deficiency are no substitute for well designed studies. *BMJ* 2013;346:f956.
3 Department of Health. Vitamin D—advice on supplements for at risk groups. Letter from chief medical officers of the United Kingdom. 2012. www.scotland.gov.uk/Resource/0038/00386921.pdf.
4 Edouard T, Glorieux FH, Rauch F. Relationship between vitamin D status and bone mineralization, mass, and metabolism in children with osteogenesis imperfecta: histomorphometric study. *J Bone Miner Res* 2011;26:2245-51.
5 Lawson M, Thomas M. Vitamin D concentrations in Asian children aged 2 years living in England: population survey. *BMJ* 1999;318:28.
6 Callaghan AL, Moy RJ, Booth IW, Debelle G, Shaw NJ. Incidence of symptomatic vitamin D deficiency. *Arch Dis Child* 2006;91:606-7.

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