Invited Commentary

Abundant sunshine and vitamin D deficiency

(First published online 31 January 2008)

In this issue of the British Journal of Nutrition, Islam et al. (1) and Woo et al. (2) present data from Bangladesh and China showing a high prevalence of poor vitamin D status in women of childbearing age combined with a concurrent elevation in plasma parathyroid hormone (PTH) concentration. These reports add to the growing evidence of a high prevalence of vitamin D insufficiency in countries with abundant sunshine.(3–5).

Vitamin D can be acquired by cutaneous synthesis following exposure to sunlight and from the diet.(5–9) Vitamin D supply varies considerably worldwide, across population groups and between individuals, mainly because of differences in skin exposure to UVB radiation, the efficiency of cutaneous synthesis (for example, due to ageing and skin pigmentation) and in food fortification practices and supplement use.(3,4)

Vitamin D is an important determinant of bone health at all ages; deficiency causes rickets in children and osteomalacia in children and adults. A low vitamin D status has also been associated with increased bone loss and osteoporotic fracture risk in older people(6,7).

Markers of vitamin D status

Vitamin D is hydroxylated in the liver to 25-hydroxyvitamin D (25OHD). This circulates in plasma and serves as a reservoir for further hydroxylation to the biologically active metabolite 1,25-dihydroxyvitamin D. The plasma concentration of 25OHD is the most commonly used biomarker of vitamin D status because it has a long half-life and because it is not under tight homeostatic regulation and therefore reflects vitamin D supply and usage over a period of time.(8)

In the UK, a threshold concentration of 25 nmol/l (10 ng/ml) has traditionally been used to define a level above which there is little risk of vitamin D-deficiency rickets or osteomalacia.(6,7) More recently, links between vitamin D status and other health outcomes (for example, osteoporosis, cancer, CVD, diabetes, tuberculosis) have led to debates about what defines vitamin D sufficiency across the lifecycle and in different population groups(5,9). Proposed lower thresholds of 25OHD concentration range between 50 and 100 nmol/l(5,10). A complication in this debate is the lack of methodological standardisation of 25OHD measurements(11). Participation in the Vitamin D External Quality Assessment Scheme (www.deqas.org) helps to monitor assay and laboratory performance but it is not yet common practice to report these data in publications.

PTH has been proposed as a functional marker of vitamin D status because it is linked to 25OHD through the Ca–phosphate homeostatic system. An inverse relationship between the plasma concentrations of PTH and 25OHD has been reported in many cross-sectional and intervention studies(12–15) and an elevated plasma PTH concentration is considered to be a risk factor for osteoporosis(14,15). However, most studies have been conducted among older Caucasian people in Europe and the USA(12–15) and PTH is influenced by many factors other than vitamin D status, including stage of life, ethnic background, dietary Ca and phosphate intake, time of day and assay method(13,16–18).

Our studies in The Gambia and China, for example, have shown that plasma PTH concentration is elevated in populations with a low Ca intake, even when vitamin D status is good, and that the inverse correlations between plasma PTH concentration and bone health indices observed in Western countries are not found(5,19,20).

In older Caucasian populations bone mineral density (BMD) is a useful marker of fracture risk (www.ISCD.org), and has been used as a functional outcome measure in vitamin D studies. However, the use of BMD measures to predict fracture risk does not necessarily apply to younger people or non-Caucasians(21).

Therefore as we have recently argued, it is unlikely that a single biomarker of vitamin D status, threshold value of 25OHD concentration or outcome measure of vitamin D deficiency will be identified for use in all situations, and research is needed to refine existing biomarkers or to establish new indicators(17).

Cutaneous and dietary supply of vitamin D

In general people derive most of their vitamin D via exposure to sunlight, and official recommendations for vitamin D intake with respect to bone health are based on this assumption. Hence in the UK, for example, reference nutritional intakes are only set for stages of life where requirements are high (infants and children, pregnant and lactating women, older people), and those in vulnerable groups whose sun exposure might be limited(6,22).

Endogenous vitamin D production requires skin exposure to UVB radiation of wavelengths 290–315 nm, which can occur all year at tropical latitudes and in the summer months at temperate latitudes(23) (http://nadir.nilu.no/~oaleng/fastt/Vitamin D.html). It is not surprising therefore that in people living at temperate latitudes, for example, UK (50–62°N) and Beijing (39°N), there can be marked seasonal variation in plasma 25OHD concentration, i.e. lower in winter v. summer months, and that the contribution of dietary sources to vitamin D status is greatest during the winter(2,12,24).

What may come as a surprise is that in countries where...
theoretically vitamin D synthesis can occur all year round, there are reports of a high prevalence of low vitamin D status. The studies by Islam et al. (1) in Dhaka (23°N) and Woo et al. (2) in Hong Kong (22°N) illustrate the many factors that can limit cutaneous vitamin D synthesis, including environmental conditions such as pollution, time spent indoors because of living and working conditions, customary dress because of weather, culture and religion, skin pigmentation and use of sunscreen (5). Woo et al. (6) commented that 60% of women in Hong Kong indicated they did not like going out in the sun. Islam et al. (1) reported that >90% of their subjects used sunscreen.

**Vitamin D deficiency: a global perspective**

Although different methodologies were used, the studies by Woo et al. (2) and Islam et al. (1) both report a high number of women with values of 25OHD < 25 nmol/l (16% in Dhaka; 18% in Hong Kong and 40% in Beijing, measured in the spring time) and that >90% of women had 25OHD < 50 nmol/l (1,2). This is of concern because of potential adverse consequences for the women’s own health and because poor vitamin D status in pregnant women is associated with decreased fetal and childhood bone mineral accretion, and an increased risk of rickets in their infants (3). Furthermore, poor vitamin D status in women of childbearing age indicates that status is also likely to be poor in other age groups in the population. Based on the presence of rickets or a plasma 25OHD concentration < 25 nmol/l, current evidence shows that the prevalence of vitamin D deficiency is high in many parts of the world, for example, among elderly people in the UK, especially those living in residential care, among infants and pregnant women from ethnic minorities at northerly and southerly latitudes, in people living in or near the tropics who wear concealing clothing or who spend little time out of doors, those with a low Ca intake and among children from Asia and the Middle East (3–5).

Emerging research is leading to a substantial body of evidence on vitamin D and prevention or reduction of chronic and infectious diseases (6). This in turn is leading to calls to redefine deficiency and increase recommendations for intake. At the same time, frank vitamin D deficiency is a major public health problem in many parts of the world that requires urgent attention (7). It is important that policy solutions including the promotion of safe (with respect to melanoma risk) and adequate sun exposure for vitamin D synthesis are put in place. The current scientific debate about thresholds of 25-hydroxyvitamin D for multiple health outcomes (8) is an urgent attention (5). It is important that policy solutions include the promotion of safe (with respect to melanoma risk) and adequate sun exposure for vitamin D synthesis.

None of the authors have any conflicts of interest or sources of funding to report.

Inez Schoenmakers, Gail R. Goldberg and Ann Prentice

MRC Human Nutrition Research
Elsie Widdowson Laboratory
Fulbourn Road

References


