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Sunlight or diet: what is the answer for providing sufficient vitamin D in the UK?

Madam,

The recent paper by Diffey [1] casts doubt on current opinion that summer sunlight is the major contributor to vitamin D status in the UK [2] . His model of seasonal changes in serum 25-hydroxyvitamin D (25(OH)D) agrees well with measured data from a UK population [3] and confirms that the increase in 25(OH)D (16 nmol/L) is small compared to wintertime circulating 25(OH)D (41 nmol/L). The model is helpful for monitoring the relationship between sunlight exposure and 25(OH)D. He goes on to state that the model predicts that sun exposure in the UK accounts for 50% (5-8 nmol/L) of 25(OH)D in summer and 20% of the 25(OH)D in winter. We believe that this interpretation might be misleading. Cutaneous synthesis of vitamin D is almost absent in winter, so a figure of 20% might encourage people to spend more time outside believing that it can provide 40% of the vitamin D that could be made in the summer, while in practise this would impact very little on vitamin D status. Excess sunlight in winter may even decrease circulating 25(OH)D [4].

The other source of vitamin D is the diet, but usual intakes in the UK are low (around 3.5 ug [140 IU]) [2]. The increase in 25(OH)D per µg [40 IU] dietary vitamin D has been estimated, during winter-time daily supplementation of oral vitamin D for 5-6 months, as 0.70 nmol/L [5] or 1.96 nmol/L [6]. The discrepancy between the two estimates serves to highlight the problems encountered when comparing studies where 25(OH)D has been measured in

different laboratories using different methodology. Using Cashman's data there is a 5-fold difference between his estimate of 7.0 nmol/L 25(OH)D, for 3.6 ug vitamin D a day, and winter-end circulating levels of 37 nmol/L seen in the untreated group. We believe that vitamin D stores could provide an explanation for the difference, and Diffey agrees that this could be a possible contributory factor. Heaney *et al.* estimated that the equivalent of 96 µg vitamin D in total was required each day to maintain the end-of-summer circulating 25(OH)D of 70 nmol/L; and that tissue stores could account for 81- 85% of winter needs together with a daily dietary intake of 12.5 µg [5]. Although circulating 25(OH)D has a half life of 3-4 weeks, it is suggested that vitamin D may be stored in tissues for months or even years [7]. We need to question whether 25(OH)D is fully reflecting vitamin D stores, as is generally assumed.

Assuming there is a significant vitamin D store, is the major contributor sunlight or diet? It is estimated that full body exposure to sunlight can produce 25,000 IU vitamin D in a single day [8]. In Manchester, 6 weeks exposure of 1.3 SED three times a week in a laboratory setting wearing t-shirt and shorts, increased mean 25(OH)D by 26 nmol/L (from 44 nmol/L to 70 nmol/L) [9]. As summer sunlight consistently increases 25(OH)D in the population above that made from usual diet, it is clearly an important source of vitamin D, but it is unclear how much is stored and whether circulating 25(OH)D fully reflects tissue stores. Our own longitudinal data from Aberdeen (figure) shows consistent early spring 25(OH)D concentrations of 40 nmol/L across 3 years, even after a poor summer in 2007. In addition we found striking consistency in 25(OH)D measured for this study and those done in 1998/2000, independent of seasonality and method of analysis, which again suggests that there are other factors besides diet and sunlight that affect circulating 25(OH)D.

Diffey argues that it would be better to rely on dietary sources for vitamin D, because the amount of sun exposure required to make an impact on vitamin D status would increase cancer risk. However, the oral route may not be without its risks and problems. Few foods contain vitamin D, and boosting dietary intakes of vitamin D would require large-scale supplementation or further food fortification, which poses practical difficulties. One advantage of the sunlight route is that it does not allow excess production of vitamin D, but vitamin D may be toxic when too much is given orally. The conclusion that oral vitamin D is not as toxic as once thought was based on short-term studies and small numbers of healthy subjects [10]. It is likely that the vitamin D stores of many people in the UK are depleted or sub-optimum because of insufficient sunlight exposure and low dietary intakes, and that short-term high dose supplementation with vitamin D would be effective in repleting them. However, we do not know the health repercussions of continued high-dose vitamin D supplementation, particularly if these stores are replete. We would urge caution on this course of action for the general population until we can be certain about the relationship between 25(OH)D and stored vitamin D.

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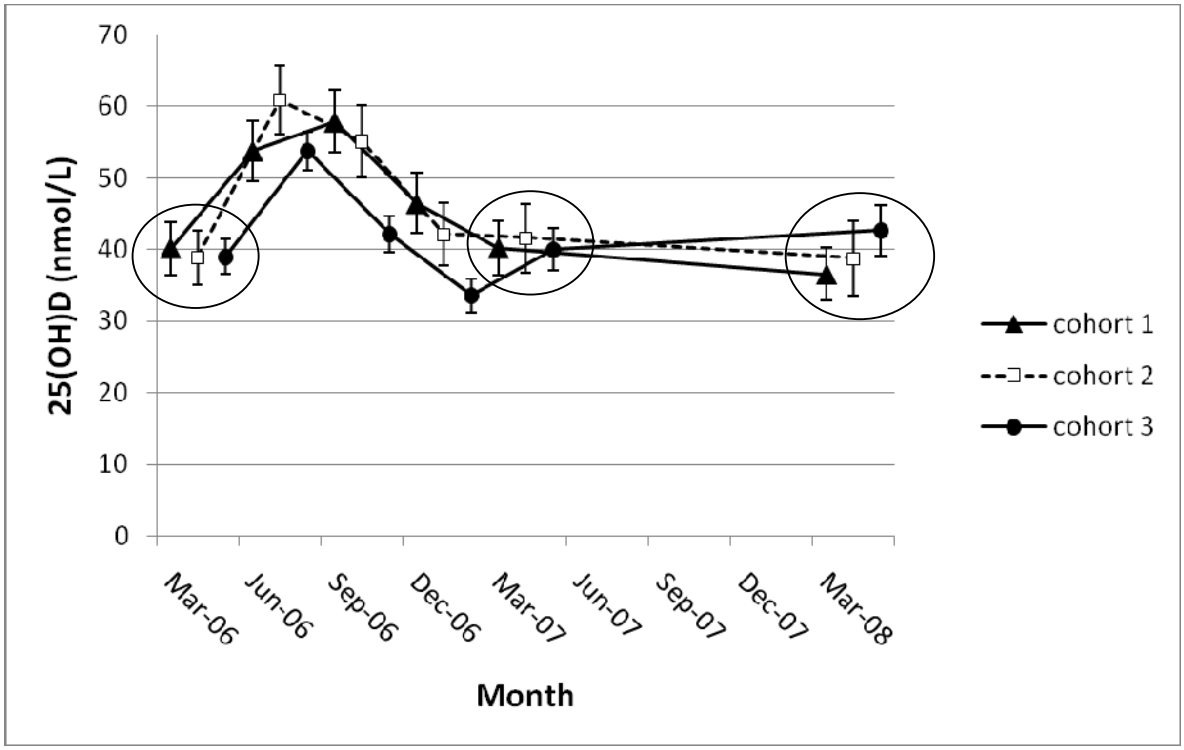
Figure. Longitudinal measurements of 25-hydroxyvitamin D from 2006 to 2008 in Aberdeen



Serum 25-hydroxyvitamin D [25(OH)D] (mean and 95% CI) was measured by immunoassay in women aged 60-65 years, living in Aberdeen, Scotland. Each cohort was seen at 3-monthly intervals from spring 2006 to spring 2007 inclusive, with an additional visit in spring 2008. Cohort 1 started in March 2006 (n=120), cohort 2 started in April 2006 (n=109) and cohort 3 started in May 2006 (n=159). Circled points indicate the spring measurements for each year.