Response to Vitamin D Intake: From the Antarctic to the Institute of Medicine

Dear Editor:

We commend you and the Committee to Review Dietary Reference Intakes for Vitamin D and Calcium for your outstanding summary of the committee's work and the detailed reports on its reevaluation of the DRI for calcium and vitamin D (1,2). We are honored that some of the data from our Antarctic research (3) was included in this effort. As you are aware, with the publication of our second Antarctic study (4), we have extended our initial findings and documented that BMI, among other factors, has a significant effect on response to vitamin D intake and supplementation.

Given this information, and knowing that the committee did not have all of the individual data available to them, we analyzed these data in light of the 2011 report (2). Study details have been previously published (3,4). We combined the data from the 2 studies in a fashion matching the Institute of Medicine (IOM) committee report; i.e. mean dietary intake data for the 3 sessions were calculated, with the intake from the vitamin D supplement added (for supplemented groups) to yield a total vitamin D intake. This analysis was done using the results of content analysis of the vitamin D pills used in these studies (Covance), which was reported in both of our Antarctic papers. A total of 95 participants, 64 men and 31 women, were included because they had provided complete diet records. Fourteen participants were in the "no study supplement" groups, meaning they were allowed to consume whatever supplements they wished, as long as these were reported. Supplemented participants received [target dose (analyzed content)]: 400 (416) IU/d, 1000 (1380) IU/d, 2000 (2760 in the first Antarctic study and 2230 in the second Antarctic study) IU/d, or 10,000 IU/wk (10,300) (1 IU = 0.025 μg). Because of the impact of participant BMI on the vitamin D response, we split the data into 2 groups: data from participants having BMI $\geq$ 30 (obese) and from those having BMI < 30 (nonobese). Data were fit to an ordinary least-squares regression analysis consistent with the methods reported in the IOM report, where achieved serum 25-hydroxyvitamin D was regressed on the natural log of total vitamin D intake, with no y-intercept and with the addition of a dichotomous predictor indicating whether participants were obese or not. This enabled us to estimate the effects of obesity. Model diagnostics were favorable with the elimination of a single influential outlier in our dataset. The model, predicting achieved serum 25-hydroxyvitamin D concentration from total vitamin D intake, revealed a significantly different response when the results for obese participants were compared with those for nonobese participants (Fig. 1). A lower predicted CI of $\sim$50 nmol/L was associated with a vitamin D intake of 270 IU/d for nonobese and 1130 IU/d for obese participants (Fig. 1). Each of these is considerably different from the 600 IU/d reported by the IOM to predict the lower CI selected with the intention of "overshooting" the target serum value of 40–50 nmol/L. If indeed serum 25-hydroxyvitamin D should be 40–50 nmol/L to maximally protect bone, then individuals considered to be obese (BMI $\geq$ 30) may require more vitamin D than nonobese individuals.

In conclusion, when combined, these data fit the model derived by the IOM Committee, even though all individual data were condensed into the 3 published points. Given the apparent weighting of these data to the overall curve [Fig. 5–4 in the complete report (2)], similar findings based on all of the data, as shown here, provides confidence in the DRI model. Nonetheless, when viewed in light of our recent findings, these data provide evidence that in an environment with minimal sun exposure (4), the recommended 600 IU vitamin D/d may be too low for about one-third of the country [based on CDC estimates that $\sim$30% of people in the United States are obese (5)].

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Literature Cited

1 Supported by the National Aeronautics and Space Administration Human Research Program Human Health and Countermeasures Element.
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