Cholecalciferol and 25-hydroxyvitamin D concentrations in adults with cystic fibrosis

Dear Sir:

The results obtained in the impressive study by Stephenson et al (1) are surprising and frustrating to desperate clinicians who have not succeeded in increasing serum 25-hydroxyvitamin D concentrations (SVDCs) in their cystic fibrosis (CF) patients. The failure of oral supplements to effect such is widely published (2–4). In search of alternatives, we undertook a retrospective investigation to clarify the relative importance of vitamin D sources and came to remarkably different conclusions (5).

SVDC data (n = 474 measurements) collected annually over 4 consecutive years (2001–2005) from 137 CF patients (mean age: 15.6 y; range: 0.5 mo to 42 y) were retrospectively evaluated. Plotted per month, these values depict an S-shaped curve convex from May to October (sunny) and concave in the following period (dark). Mean values in each period (sunny: 26.6 ng/mL; dark: 17.3 ng/mL) were significantly different from each other; however, this was not the case when values were compared with those of healthy controls during the same period (sunny: 28.6 ng/mL; dark: 18.8 ng/mL). A striking significant relation was found between SVDCs over the 4 y and the amount of sun hours in the preceding months. The SVDC curve constantly paralleled that indicating the hours of sunshine, with a delay of about 2 mo. It is interesting to note that all of these findings were valid in patients with exocrine pancreatic insufficiency and in those with pancreatic sufficiency, although the latter findings were valid in patients with exocrine pancreatic insufficiency and in those with pancreatic sufficiency, although the latter findings were valid in patients with exocrine pancreatic insufficiency.

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All of these findings suggest that endogenous vitamin D production in the skin, under the influence of sunlight, is an important source of this vitamin for CF patients. Sun exposure during the sunny months of the year should therefore be encouraged. In contrast, we found little evidence of an effect of oral supplements on SVDCs. These conclusions are not universally valid because the amount of sunlight largely depends on geographic latitude. However, under good conditions, sun exposure can be an inexpensive and convenient alternative to supplements for increasing SVDCs in CF patients.

Neither author had a personal or financial conflict of interest.

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REFERENCES

Reply to E Robberecht and S Vandewalle

Dear Sir:

We are grateful for Robberecht et al’s interest and comments on our article (1) and appreciate the opportunity to respond to their letter. We agree with the authors that vitamin D deficiency is an international problem and that normalizing serum 25-hydroxyvitamin D concentrations in patients with cystic fibrosis (CF) has been challenging. Boyle et al (2) showed that, even with high doses of ergocalciferol (vitamin D2, plant form), serum 25-hydroxyvitamin D [25(OH)D] concentrations do not increase as expected. Although our findings need to be confirmed in a randomized control trial, the results suggest that cholecalciferol (vitamin D3, animal form), at appropriate doses, can increase serum concentrations. Studies have shown that ergocalciferol does not increase serum 25(OH)D concentrations as efficiently as does cholecalciferol, which may explain these divergent results (3, 4). That is not to say that vitamin D3 is completely ineffective, and supplementation of milk with ergocalciferol in the United States has successfully eradicated rickets. However, the dose of vitamin D needed to prevent rickets is 100 IU/d in infants with little sun exposure. The dose of vitamin D needed to maintain nutritional adequacy on the basis of serum 25(OH)D concentrations is much higher. Robberecht et al raise an excellent point that sunlight may be the most effective and efficient way to normalize serum 25(OH)D concentrations in general and in CF patients in particular. This is undoubtedly the most “natural” way to make vitamin D. As stated in our article, Gronowitz et al (5) showed that 10 min of ultraviolet B exposure 3 times/wk increased serum 25(OH)D to concentrations as high as 124 nmol/L after 24 wk, which is much higher than that attained by supplementation with either cholecalciferol or ergocalciferol. It may be that such serum 25(OH)D concentrations can be achieved with higher
Is there a need for vitamin C supplementation of the normal diet? Effects of in vivo ascorbate depletion on adrenal function

Dear Sir:

In a recent article in the Journal, Padayatty et al (1) described a fast increase in the secretion of vitamin C from human adrenals in response to adrenocorticotropic hormone, preceding the rise in cortisol secretion. The function of the vitamin C released from the adrenals is not clear. The authors considered oral and intravenous vitamin C supplementation in the context of a putative paracrine function of adrenal vitamin C.

Our group has shown that vitamin C deprivation of guinea pigs for 15 d reduced the vitamin C content of their adrenals to <1/20 of the normal concentration in this tissue (2). These animals showed an impaired plasma aldosterone response to sodium depletion. Cells isolated from the adrenals of these vitamin C–deprived animals secreted less aldosterone than did cells isolated from the adrenals of animals without vitamin C depletion. A reduced conversion of labeled deoxycorticosterone to aldosterone by these cells seemed to indicate an impaired activity of CYP11B2 (2). Guinea pigs also have shown reduced in vivo and in vitro aldosterone responses after depletion of α-tocopherol (3). These findings are compatible with the need for antioxidants to protect the function of the adrenal cortex against reactive oxygen species generated by lipid peroxidation in the adrenal cortex (4). We did not see an impaired cortisol response after vitamin C or α-tocopherol depletion, perhaps because of the higher expression of the cortisol-synthesizing enzyme CYP11B1 than of the aldosterone-producing enzyme CYP11B2. In addition to these antioxidants, superoxide dismutase and glutathione peroxidase are highly concentrated in the adrenals, and they may add to the defense against reactive oxygen species. Our depletion experiments concerning the role of vitamin C in the function of the adrenal cortex do not support the necessity of vitamin C supplementation in addition to a normal diet.

None of the authors had a personal or financial conflict of interest.

Anne Stephenson

REFERENCES


Reply to V Bähr et al

Dear Sir:

Concerning our recent article in the Journal (1), Bähr et al wrote that we considered oral and intravenous vitamin C supplementation in the context of paracrine vitamin C function in the adrenal gland. The statement by Bähr et al takes what we wrote out of context. We did not consider supplementation but, rather, discussed supplementation only in the context of the data we presented.

Volker Bähr

REFERENCES


