

Up-regulatory impact of boron on vitamin D function -- does it reflect inhibition of 24-hydroxylase?

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Abstract

Nutritional intakes of boron have been shown to lessen the adverse consequences of vitamin D deficiency in rodents. Pilot clinical studies suggest that this effect may be mediated, in whole or in part, by an increase in serum 25-hydroxyvitamin D. We propose that, in concentrations achievable with good diets, boron suppresses the activity of the microsomal enzyme 24-hydroxylase, chiefly responsible for catabolism of this steroid. This inhibition may reflect a direct interaction with the enzyme, or perhaps boron's ability to form a covalent complex with the product of its activity, 24,25-dihydroxyvitamin D. An up-regulatory impact of boron on 25-hydroxyvitamin D is potentially beneficial in light of the fact that the vitamin D status of many individuals is poor during winter months, and traditional supplemental doses of this vitamin are often too low to correct this problem. There is growing evidence that good vitamin D status -- as reflected by 25-hydroxyvitamin D levels -- may reduce risk for a host of prominent disorders; thus, boron may have the ability to potentiate this protection. Clinical studies also suggest that nutritional boron can up-regulate 17beta-estradiol levels in women, including postmenopausal women receiving hormone replacement therapy. The catabolism of this hormone is achieved by microsomal enzymes catalyzing vicinal hydroxylations -- a description that also applies to 24-hydroxylase. This suggests the more general hypothesis that nutritional boron can inhibit a range of microsomal enzymes which insert hydroxyl groups vicinal to existing hydroxyls in steroids -- including the enzymes which catabolize estradiol and 25-hydroxyvitamin D.

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