Format: Abstract

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Dietary boron decreases peak pancreatic in situ insulin release in chicks and plasma insulin concentrations in rats regardless of vitamin D or magnesium status.

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Abstract

Because dietary boron deprivation induces hyperinsulinemia in vitamin D-deprived rats, the influence of dietary boron on insulin metabolism as modified by nutritional stressors was examined in two animal models. Male weanling Sprague-Dawley rats were assigned to each of four (Experiment 1) or 8 (Experiment 2) dietary groups for 35 d: the basal diet (< 0.2 mg B; <1.0 mg Mg/kg) was supplemented with boron (as orthoboric acid) to contain <0.2 or 2.0 (a physiologic amount) mg B/kg; with magnesium (as magnesium acetate), at 100 (inadequate) or 360-400 (adequate) mg/kg; and with cholecalciferol [vitamin D-3; 25 microg/kg for study length (Experiment 2), or, depleted for 16-17 d then repleted until end of experiment (Experiments 1 and 2)]. In the rat model, boron reduced plasma insulin (Experiment 1, $P < 0.002$; Experiment 2, $P < 0.03$), but did not change glucose concentrations regardless of vitamin D-3 or magnesium status. Cockerels (1 d old) were fed a ground corn, high protein casein and corn oil-based basal diet (low boron; 0.3 mg B/kg) supplemented with boron as orthoboric acid to contain 0.3 or 1.65 mg/kg (a physiologic amount) and vitamin D-3 at 3.13 (inadequate) or 15.60 (adequate) microg/kg. In the chick model, boron decreased ($P < 0.045$) in situ peak pancreatic insulin release at 26-37 d of age regardless of vitamin D-3 nutriture. These results suggest that physiologic amounts of boron may help reduce the amount of insulin required to maintain plasma glucose.

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