Effect of Hypervitaminosis D₃ on Magnesium Metabolism of the Chick

JAMES E. H. STAFFORD and NORMAN A. EDWARDS

Department of Physiology and Biochemistry, University of Reading, Reading RG6 2AJ, Berks., U.K.

A fall in the concentration of plasma Mg²⁺ is induced in rats by excess of vitamin D (Hanna, 1961). This has been attributed to a deposition of Mg²⁺ in the bone and a diminished binding of Mg²⁺ to plasma proteins (Richardson & Welt, 1965). In chicks, high concentrations of vitamin D₃ produce changes in plasma Ca²⁺ and phosphate concentrations similar to those observed in rats (Taylor et al., 1968), but the effects on Mg²⁺ concentration have not been studied.

Daily intramuscular injections of vitamin D₃ for 1 week in two groups of 3-week-old male chicks produced a significant dose-dependent decrease in the plasma, femur and skeletal-muscle concentrations of Mg²⁺ compared with a control group. The decreases caused by 70000 and 140000 i.u. respectively of vitamin D₃ were as follows: in plasma, 13 and 39%; in bone, 10 and 18%; in muscle, 13 and 19%. The vitamin D₃ produced an increase in the Ca²⁺ concentration of the liver and skeletal muscle and a small decrease in the percentage bone ash. Neither the liver Mg²⁺ concentration nor the percentage binding of Mg²⁺ to the plasma proteins was affected by the treatments.

At both dosages of vitamin D similar degrees of hypercalcaemia and hypophosphataemia occurred. The hypomagnesaemia appeared to be independent of the changes in plasma Ca²⁺ and phosphate concentrations.

The results were consistent with a renal loss of Mg²⁺ (Liftshitz et al., 1967), resulting in a depletion of the total body Mg²⁺ that superficially resembled the effect of a dietary magnesium deficiency (Stafford & Edwards, 1972).

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Magnesium Status and Thyroid Activity

H. P. HUMPHRAY and F. W. HEATON

Department of Biological Sciences, University of Lancaster, Bailrigg, Lancaster, U.K.

The availability of Mg affects thyroid activity (Humphray & Heaton, 1972), a positive correlation between the Mg concentration in serum and the (protein-bound iodine)/(total serum iodine) ratio being observed in rats subjected to specific dietary deficiency or loading with magnesium salts. The action of thyrotrophin on the thyroid gland appears to be mediated by the formation of cyclic AMP (Schell-Frederick & Dumont, 1970; Robison et al., 1971), and as Mg activates the membrane-bound adenylate cyclase of the gland in vitro (Pastan & Katzen, 1967; Burke, 1970; Wolff & Jones, 1971) we investigated the effects of deficiency and loading with magnesium salts on the concentrations of cyclic AMP within the thyroid gland, to determine whether this was the mechanism by which Mg produced its effect in vivo.

Three groups of male Wistar albino rats, initially weighing 60-70g, were fed with magnesium-deficient (0.4mg/100g), control (75mg/100g) and Mg-loaded (380mg/100g) diets for 12-14 days. All rats received an amount of food equal to that consumed by the deficient animals and distilled water was provided ad libitum. Each rat was injected intraperitoneally with 10μCi of carrier-free Na¹²⁵I in saline (0.9% NaCl) and killed by exsanguination 24h later. The thyroid glands were excised immediately and

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