

EFFECTS OF DIETARY CALCIUM ON VITAMIN D METABOLISM IN CHICKENS

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The clearance rate of [^3H]-25-hydroxyvitamin D₃ (25(OH)D₃) from the circulation is increased in calcium-deficient rats due to an enhanced hepatic destruction of 25(OH)D₃ which may lead to rapid vitamin D deficiency (Clements et al. 1987). Experiments were carried out to determine whether vitamin D deficiency could be induced in chickens by a similar mechanism and whether such deficiency could explain disorders of skeletal development in commercial chicken flocks.

Chickens were raised on a low calcium diet (0.3% calcium) or a control diet (1.0% calcium) and were given oral doses of vitamin D₃ (5-40 ug, 3 doses/week). Plasma 25(OH)D₃ levels in chickens on the low calcium diet (16.2-32.8 nmol/l) were 28-44% lower than in control chickens (28.2-45.8 nmol/l). Plasma 1,25-dihydroxyvitamin D₃ (1,25(OH)₂D₃) levels were appropriately elevated in chickens on the low calcium diet (700 pmol/l) compared to controls (240 pmol/l) and this may account for their reduced 25(OH)D₃ status.

Plasma clearance rates of 25(OH)D₃ were determined by measurement of the elimination half-time ($t_{1/2}$) of a tracer amount of ^3H -25(OH)D₃. In contrast to rats, there was no significant difference in $t_{1/2}$ between calcium-deficient chickens ($t_{1/2}$ =6.07 days) and the control chickens ($t_{1/2}$ =5.79 days). In both cases, however, clearance rates were faster than those reported for rats ($t_{1/2}$ =15.4 days). The clearance rate of 1,25(OH)₂D₃ determined in control chickens was also faster ($t_{1/2}$ =10-30 min) compared to rats ($t_{1/2}$ =8 hr, Paulson and Kenny 1985) or humans ($t_{1/2}$ =11 hr, Wiesner et al. 1980). Measurement of the binding affinity of 25(OH)D₃ for vitamin D binding protein (DBP) in chick and rat plasma showed that 25(OH)D₃ has a four-fold lower affinity for chicken DBP ($K_A=1.17 \times 10^9 \text{M}^{-1}$) than for rat DBP ($K_A=5.16 \times 10^9 \text{M}^{-1}$). This difference may account for the shorter $t_{1/2}$ of 25(OH)D₃ and 1,25(OH)₂D₃ in chickens.

Calcium-deficient and control chickens were injected with a tracer amount of (4- ^{14}C , 1,2- ^3H)vitamin D₃ to determine the quantity of vitamin D metabolites in the bile which were derived from 1,25(OH)₂D₃. Comparison between bile $^{14}\text{C}/^3\text{H}$ and plasma $^{14}\text{C}/^3\text{H}$ showed that metabolites derived from 1,25(OH)₂D₃ was two-fold higher in bile from calcium-deficient chickens. This represents the increased catabolism of 1,25(OH)₂D₃ due to calcium deficiency. There was no significant difference between calcium-deficient chickens and controls in the quantity of biliary metabolites not derived from 1,25(OH)₂D₃. These results are in marked contrast to calcium-deficient rats in which the amount of biliary metabolites not derived from 1,25(OH)₂D₃ was 63% greater than controls.

Calcium deficiency in chickens increases the rate of utilization of 25(OH)D₃ only by increased conversion to 1,25(OH)₂D₃ and not by enhanced hepatic degradation of 25(OH)D₃.

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