Body Water Kinetics in Vitamin A-Deficient Chickens[1] (37525)

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(Introduced by L. C. Faulkner)

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An essential function of vitamin A is to maintain membrane stability and permeability (1–3), and changes in the relative concentration of the vitamin in the membrane results in altered transport across it (4). Krause *et al.* (5) have suggested an effect of vitamin A on the turnover of phospholipids in membranes, which points to an important role of the vitamin in the maintenance of normal membranous structure and function.

De Luca and Wolf (6) proposed that the epithelial lining of the gastro-intestinal tract is the most sensitive tissue to the action of vitamin A and that the production of mucus-secreting (goblet) cells is adequate only in the presence of normal levels of the vitamin. They further indicated that without vitamin A these cells degenerate, and with deficient levels of the vitamin, intestinal epithelial cells lose their ability to differentiate to the mucus-secreting type. Vitamin A-deficient chickens appeared to have a higher water consumption rate and a more liquid excreta (unpublished observations). The present study was designed to study body water kinetics as a function of vitamin A intake.

Materials and Methods. Two groups of five-month-old ArborAcre × Indian River broiler pullets were used. They were maintained after hatching on the ration described by Nockels and Kienholz (7) supplemented with 6600 IU of vitamin A palmitate per kg of diet for the control group and with 300 IU of the vitamin/kg of diet for the A-deficient group. The latter group was considered to be mildly vitamin A-deficient as changes in body weight between the two groups were not significant.

Six pullets from each dietary regime were housed in wire bottom cages, three birds per cage, at 23 ± 2°. Humidity was not controlled. Feed and water were provided *ad libitum*. A three-day period for acclimatization was allowed before the experimental phase.

The birds were injected intravenously with 88.09 μCi of tritiated water in a single, 3-ml dose via the right or left brachial vein. Serial blood samples (3 ml) were collected from the brachial veins of all birds 24 hr after treatment to allow for complete equilibration of the tritiated water in all body compartments. Blood collection was repeated at 24-hr intervals for 6 days. Blood proteins were precipitated with 200 mg of dry trichloroacetic acid (TCA) immediately after collection. The samples were kept at room temperature throughout collection, and the protein-free supernatant was removed by centrifugation at 17,500*g* for 15 min. Duplicate 0.5 ml portions of the supernatant were transferred to vials containing 10 ml of "Aquasol" liquid scintillation fluid (New England Nuclear) and counted for 10 min in a Beckman 200 liquid scintillation counter (Beckman Instruments, Inc.). Base counting efficiency and quantification of the injected isotope were established with a primary tritiated toluene standard (New England Nuclear). A quench curve was prepared using known amounts of tritiated water added to varying ratios of blood and TCA. Counting efficiency ranged 30–36%. Radioactivity in the samples was converted to microcuries of tritium per unit volume. Regression curves were obtained by

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TABLE I. Body Water Kinetics in Normal and Vitamin A-Deficient Chickens.[a]

Parameters measured	Control pullets (6)[b]	Vitamin A-deficient pullets (6)[b]	Significance of comparison
Body weight (kg)	2.68 ± 0.67[c]	2.78 ± 0.44	Not significant
Body water (% body weight)	75.62 ± 2.05	71.6 ± 3.28	Not significant
^{3}H-water half life (hr)	145.2 ± 9.68	98.1 ± 6.93	$p < 0.01$
Body water flux (ml/hr/kg body weight)	3.66 ± 0.17	5.13 ± 0.28	$p < 0.01$
Body water flux (ml/hr/liter pool)	4.87 ± 0.29	7.26 ± 0.55	$p < 0.01$

[a] Five-month-old.
[b] Number of birds per group.
[c] Mean ± standard error.

correlation–regression analyses, and comparisons between mean values were established by paired Student's t tests.

Results and Discussion. The concentration of tritium in body water decreased exponentially with time. Individual values closely fit the calculated regression lines (correlation coefficients > 0.90). Kinetic parameters were derived as described by Chapman and Black (8) and are listed in Table I.

The half-life of tritiated body water was significantly shorter ($p < 0.01$) in the A-deficient birds than in the controls. Significant differences ($p < 0.01$) between the two groups were also observed when water turnover rates or flux through the body water pool were expressed as ml/hr/kg body weight and ml/hr/liter of body water pool, respectively (Table I). Water turnover rates, even when vitamin A is mildly deficient, are significantly altered.

Total body water, expressed as percent of body weight, was not significantly different between the two groups (Table I). Since body water half life and its rate of turnover were drastically altered in the A-deficient birds, this may be suggestive of an increased water intake by the deficient chickens to maintain total body water. This observation could not be confirmed since water consumption was not evaluated quantitatively.

In general, the observed results indicate that water turnover rates in the mildly vitamin A-deficient chicken are greatly increased, suggesting an alteration in normal water transport across the intestinal wall. Whether or not these apparent effects on transport are a direct consequence of the vitamin deficiency on the intestinal epithelial cell normal function, or represent an indirect result of impaired mechanisms such as that proposed by De Luca *et al.* (6, 9), clearly need further elucidation.

Summary. The effect of mild vitamin A deficiency on kinetic parameters associated with the body water pool was studied in five-month-old pullets by isotope dilution techniques, using tritium-labeled water.

The body water $T_{1/2}$ was significantly shorter ($p < 0.01$) and the flux through the body water pool greatly increased ($p < 0.01$) in the A-deficient chicks indicating that even under mild vitamin A deficiency, water turnover rates are significantly altered. These changes appear to be due to alterations in the stability and permeability of the cells involved in water transport in the A-deficient chicken, through mechanisms yet to be elucidated.

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