

Increased Adrenal Δ^5 - 3β -Hydroxysteroid Dehydrogenase in Vitamin A-Deficient Cockerels* (33567)

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Mildly vitamin A-deficient cockerels were shown in recent studies (1) to have increased

testicular size and earlier spermatozoan maturation than vitamin A-adequate controls.

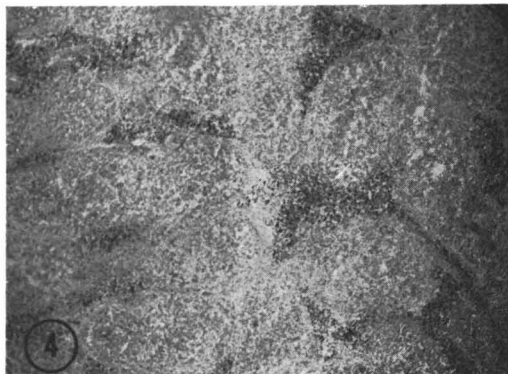
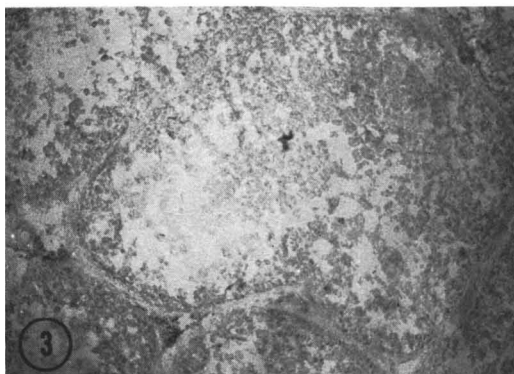
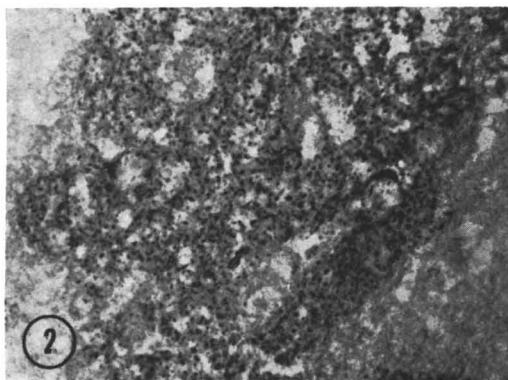
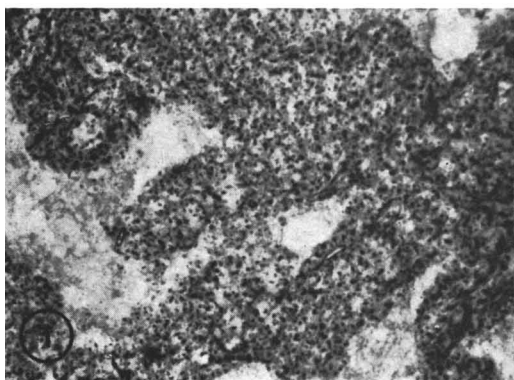


FIG. 1. Δ^5 - 3β -hydroxysteroid dehydrogenase activity in adrenal cortical area of a vitamin A-deficient cockerel, 340 $\times$.

FIG. 2. Δ^5 - 3β -hydroxysteroid dehydrogenase activity in adrenal cortical area of a vitamin A-adequate cockerel, 340 $\times$.

FIG. 3. Δ^5 - 3β -hydroxysteroid dehydrogenase activity in Leydig cells of the testis of a vitamin A-deficient cockerel, 136 $\times$.

FIG. 4. Δ^5 - 3β -hydroxysteroid dehydrogenase activity in Leydig cells of the testis of a vitamin A-adequate cockerel, 136 $\times$.

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Evidence was reported that adrenal corticosterone synthesis is inhibited in mild vitamin A deficiency (2-4). The hypothesis (1) was presented that corticosterone synthesis was possibly inhibited in mild vitamin A-deficient cockerels, and the resulting increased blood levels of adrenocorticotropin increased adrenal androgen production. The purpose of this investigation was to determine if the Δ^5 - 3β -hydroxysteroid dehydrogenase enzyme was increased in the adrenals and testes of mildly vitamin A-deficient cockerels.

Materials and Methods. Day-old single comb White Leghorn (SCWL) cockerels of a commercial strain were reared and fed as previously described (1). The chicks were randomly allotted to one of the following rations: (i) vitamin A-deficient basal, (ii) basal + 660 IU of vitamin A/kg, and (iii) basal + 6600 IU of vitamin A/kg. Hematocrits and weights of testes, combs, bursae, spleens, and adrenals were obtained on eight cockerels per treatment at 8 weeks of age. Immediately after the cockerels were sacrificed adrenals and testes were removed, frozen in dry ice, and Δ^5 - 3β -hydroxysteroid dehydrogenase was determined histochemically (5). Control tissues were run concurrently with those incubated in the substrate, dehydroepiandrosterone (DHA), postfixed in buffered formalin, and counterstained with nuclear fast red. Testes were also fixed in 10% buffered formalin and sections were stained with hematoxylin and eosin. Differences between A-adequate control and vitamin A-deficient treatment means were evaluated by the *t* test (6).

Results. The vitamin A-deficient cockerels grew well with the only major deficiency symptom being a drying of the mucosal surfaces of the mouth. In a number of the deficient chicks, limited ataxia was noticeable at 8 weeks of age when the birds were killed. Hematocrits, body weights, and organ weight responses of the cockerels are reported in Table I. Vitamin A-deficient cockerels demonstrated highly significant increases in testes weights, and significantly increased comb weights were recorded in the cockerels which received no vitamin A. Bursa size was

TABLE I. Effects of Vitamin A Deficiency on Certain Tissue and Organs of 8-Week-Old Cockerels.

Treatment (IU of vita- min A/kg)	Hemato- crit (%)	Body wt. (g)	Testes		Comb		Bursa		Spleen		Adrenal	
			Wt. (g)	% Body wt.	Wt. (g)	% Body wt.	Wt. (g)	% Body wt.	Wt. (g)	% Body wt.	Wt. (g)	% Body wt.
0	35.3	845	1.53 ^a	0.18 ^a	14.51 ^a	1.72 ^a	2.02 ^a	0.24	1.64	0.20	0.069	0.008
660	34.9	885	1.74 ^b	0.30 ^b	11.47	1.31	2.88	0.32	1.62	0.18	0.070	0.008
6600	33.2	872	0.63	0.07	9.96	1.15	2.91	0.34	2.04	0.24	0.062	0.007

^a Means significantly different ($p < 0.05$) from control.

^b Means significantly different ($p < 0.01$) from control.

reduced in the vitamin A-deficient cockerels.

Histochemically, adrenals from the vitamin A-deficient cockerels showed considerably more staining for the Δ^5 - 3β -hydroxysteroid dehydrogenase enzyme than did the controls (Fig. 1 and 2). However, the enzyme content of the Leydig cells from the A-deficient cockerels' testes was depressed (Fig. 3) when compared to the A-adequate cockerels' testes (Fig. 4). Histologically, the testes revealed a much more advanced stage of spermatogenesis in the A-deficient cockerels than the controls.

Discussion. Reactions catalyzed by Δ^5 - 3β -hydroxysteroid dehydrogenase leading to the synthesis of progesterone, 17α -hydroxyprogesterone, androstenedione, and testosterone in the adrenal and androgen synthesis in the testes have been well reviewed (7). The results of the present study indicate that mild vitamin A deficiency increases Δ^5 - 3β -hydroxysteroid dehydrogenase in the adrenal cortex with a concomitant decrease in the Leydig cells of the testis. The increased enzyme reaction in the adrenal probably increased adrenal androgen synthesis which resulted in increased testicular size and spermatogenesis. It is hypothesized that increased androgen synthesis by the adrenal suppressed pituitary intersti-

tial cell stimulating hormone which caused depression of Leydig cell steroidogenesis.

Summary. Mild vitamin A deficiency in SCWL cockerels increased Δ^5 - 3β -hydroxysteroid dehydrogenase in the adrenal cortices and decreased the enzyme in the Leydig cells of the testis. The vitamin A deficiency produced significantly larger combs and testes, increased spermatozoa maturation, and reduced bursa of Fabricius size. The increase in adrenal enzyme may account for the testicular effects observed.

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