

Effect of Mammalian Growth Hormone and Prolactin on the Growth of Hypophysectomized Chickens (42328)

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Abstract. Body weight gain and shank-toe growth during a 26-day treatment period following hypophysectomy were 55 and 46%, respectively, of control values, but the body weight gain was unaffected and bone growth only slightly reduced when the hypophysectomized chickens were fed a low dose of corticosterone (5 ppm). Bovine growth hormone (0.5 mg GH/kg body wt/day for 18 days) enhanced body weight gain and shank-toe length increase (an estimate of bone growth) by 46 and 33%, respectively, compared to the growth of hypophysectomized chickens receiving only corticosterone. These same endpoints were increased approximately 24% after ovine growth hormone treatment in hypophysectomized chickens not receiving corticosterone. Body weight gain during 18 days of treatment with bovine prolactin (0.5 mg PRL/kg/day) was 27% greater than the value for corticosterone-treated hypophysectomized chickens, but bone growth was unaffected. The mammalian GH preparations increased heart weight of the hypophysectomized chickens (25-29%), but pectoralis muscle weight was unaffected. GH treatment enhanced thymal weights by 71% in corticosterone-treated hypophysectomized chickens, and by 93% in hypophysectomized animals not receiving corticosterone. GH had no significant effect on bursal weights, and PRL had no effect on either of these lymphoid organ weights in corticosterone-treated hypophysectomized chickens. GH increased liver and adipose tissue weights considerably more than the large increases that followed treatment of hypophysectomized chickens with corticosterone alone (69 and 126% greater, respectively), but had no effect on these endpoints in hypophysectomized chickens not receiving corticosterone. PRL also greatly increased liver and adipose tissue weights in corticosterone-treated hypophysectomized chickens (79 and 75%, respectively). These results provide evidence that mammalian GH enhances body weight gain, bone growth, and the growth of several organs in the hypophysectomized chicken. Mammalian PRL increased body weight gain, liver weight, and adipose tissue weight in corticosterone-treated hypophysectomized chickens, but did not influence bone growth or the weights of the heart, pectoralis, thymi, or bursa. © 1986 Society for Experimental Biology and Medicine.

There is inadequate information regarding the effect of mammalian growth hormone (GH) on body and organ growth of birds. Nitrogen retention and body, liver, heart, spleen, bursa of Fabricius, and thymus weights were not significantly altered following the administration of mammalian GH to intact domestic fowl (1-7). Mammalian GH slightly increased body weight (8) and tibial growth (9) in chick embryos which have no detectable plasma concentrations of chicken GH (10) and in slow-growing autosomal dwarf chickens (11). GH enhanced body growth to a greater extent in growth-depressed, cortisol-treated chick embryos (12), but the data are difficult to interpret because a statistical analysis was not provided. In addition, bovine growth hormone (bGH) that had been subjected to partial trypsin digestion increased somewhat the feed

conversion and carcass protein in intact broilers, and tended to increase body weight gain, but this latter response was not statistically significant (13). Bovine GH reduced the weight loss that follows hypophysectomy of the pigeon, and increased body and organ weights of hypophysectomized pigeons when administered in combination with other hormones (14).

Body weight gain, bone growth, and the growth of several organs are reduced following hypophysectomy of the chicken (15, 16; unpublished observations, C. Scanes, D. Duyka, T. Lauterio, S. Bowen, L. Huybrechts, W. Bacon, and D. King). Thyroxine (T₄) and triiodothyronine (T₃) partially restore the lesser growth observed in young hypophysectomized chickens (15; unpublished observations, C. Scanes *et al.*), but the influence of other hor-

mones on the growth of the slow-growing hypophysectomized chicken has not been adequately studied. A fairly low dose of chicken GH increased their growth only slightly (unpublished observations, C. Scanes *et al.*). The purpose of the present studies was to ascertain whether mammalian GH and prolactin (PRL) enhance body and organ growth of young hypophysectomized chickens. Since prednisone (along with T₄) enhanced the responsiveness of hypophysectomized pigeons to mammalian GH and PRL (14), and corticosterone enhanced GH effectiveness in altering plasma free fatty acids, amino acids, and insulin levels of hypophysectomized ducks (17), we have ascertained the effects of bGH and bPRL on the growth of hypophysectomized chickens receiving a low dose of corticosterone. The corticosterone dosage used had no effect on body weight of intact chickens (18). The effect of ovine growth hormone (oGH) on body growth of hypophysectomized chickens not receiving corticosterone was also assessed.

Materials and Methods. *Animals.* White Leghorn cockerels (provided on the day of hatching by Greider Farms, Elizabethtown, Pa.) were housed in brooder units (Petersime Co., Ohio) in a well-ventilated animal room maintained at 23–26°C with lights on from 0700 to 2200 hr. All birds had access to small brood chambers (32–35°C), food (Pacemaker Starter, Agway, Inc., Syracuse, N.Y.), and water *ad libitum*. Chickens were hypophysectomized or subjected to a sham operation at 21–23 days of age (15). The shams were the same as hypophysectomies except for slitting the dura and removing the adenohypophysis.

Experimental design. Control and hypophysectomized chickens were divided into experimental groups at 24 days of age in Study 1 and 27 days of age in Studies 2–4, and assigned to treatment groups on the basis of body weight and date of surgery. Controls had the same body weights as hypophysectomized chickens just prior to surgery. Increases in body weight and shank-toe length (the combined length of the metatarsus and longest toe which estimates bone growth; see (15)) were determined for the treatment duration. The birds were decapitated 24 hr after the last hormone injection and the following organs or tissues were weighed: heart, superficial pectoralis muscle, thymi, bursa of Fabricius, liver,

and abdominal adipose tissue. The pituitary region of hypophysectomized chickens was carefully dissected and examined using a dissecting microscope (×10 magnification). Only those birds lacking visible remnants were used. The percentages of hypophysectomized chickens that survived for the duration of the study and lacked visible remnants of adenohypophysial tissue were 86, 58, 53, and 38% for Studies 1, 2, 3, and 4, respectively.

Hormone treatments. In Study 1, hypophysectomized chickens were fed a standard diet containing corticosterone (5 ppm) for 26 days while intact, sham-operated, and hypophysectomized controls received only the standard diet. The following treatments for Studies 2–4 were administered for 18 or 19 days: (Study 2) intact + vehicle, sham-operated + vehicle, hypophysectomized + corticosterone (5 ppm in the feed), hypophysectomized + corticosterone (5 ppm) + bovine growth hormone (bGH; 0.5 mg/kg body wt/day); (Study 3) intact + vehicle, hypophysectomized + corticosterone (5 ppm), hypophysectomized + corticosterone (5 ppm) + bovine prolactin (bPRL; 0.5 mg/kg body wt/day); (Study 4) intact + vehicle, hypophysectomized + vehicle, hypophysectomized + ovine growth hormone (oGH; 0.5 mg/kg body wt/day). The bGH (USDA-bGH-B1), bPRL (NIAMDD-bPRL-6), and oGH (NIAMDD-oGH-12) were dissolved in alkaline saline (adjusted to pH 8.5) and injected subcutaneously under the wing at 0900 to 1000 hr each day. Vehicle-treated chickens were injected with alkaline saline (pH 8.5). The dosage of corticosterone administered in the feed was selected because it maintained normal body weight in intact chickens (18).

Statistical analysis. Data were analyzed by analysis of variance followed by the Newman-Keuls group comparison or the *t* test (for thymal and bursal weights where there were large differences between intact/sham values and hypophysectomy values).

Results. *Body weight and bone growth.* The effects of hypophysectomy and hormone replacement on body growth are summarized in Table I. Final body weight and body weight gain of hypophysectomized chickens during a 26-day treatment period were approximately 69 and 55%, respectively, of intact/sham values. Final shank-toe length and shank-toe

TABLE I. EFFECTS OF CORTICOSTERONE (B), GROWTH HORMONE (GH), AND PROLACTIN (PRL) ON THE GROWTH OF HYPOPHYSECTOMIZED (Hx) CHICKENS

Treatment	Final body weight (g)	Body weight gain (g)	Shank-toe length (mm)	Shank-toe increase (mm)
Study 1				
Intact (10)	718 ± 12 ^A	471 ± 10 ^A	136 ± 1 ^A	48 ± 1 ^A
Sham (10)	670 ± 14 ^B	440 ± 12 ^A	131 ± 1 ^B	44 ± 1 ^B
Hx (17)	481 ± 12 ^C	252 ± 11 ^B	108 ± 1 ^C	21 ± 1 ^C
Hx + B (15)	473 ± 12 ^C	250 ± 11 ^B	104 ± 1 ^D	18 ± 1 ^D
Study 2				
Intact (9)	634 ± 13 ^A	331 ± 10 ^A	127 ± 1 ^A	32 ± 1 ^A
Sham (10)	601 ± 6 ^A	306 ± 4 ^A	125 ± 1 ^A	31 ± 1 ^A
Hx + B (10)	439 ± 12 ^B	181 ± 10 ^B	105 ± 1 ^B	15 ± 1 ^A
Hx + B + bGH (11)	524 ± 15 ^C	264 ± 12 ^C	110 ± 1 ^C	20 ± 1 ^C
Study 3				
Intact (10)	638 ± 7 ^A	330 ± 4 ^A	128 ± 1 ^A	34 ± 1 ^A
Hx + B (9)	444 ± 10 ^B	173 ± 5 ^B	107 ± 1 ^B	16 ± 1 ^B
Hx + B + PRL (9)	492 ± 13 ^C	220 ± 11 ^C	108 ± 1 ^B	17 ± 1 ^B
Study 4				
Intact (7)	647 ± 19 ^A	362 ± 13 ^A	130 ± 2 ^A	36 ± 1 ^A
Hx (7)	460 ± 17 ^B	201 ± 13 ^B	109 ± 1 ^B	17 ± 1 ^B
Hx + oGH (7)	522 ± 26 ^C	247 ± 18 ^C	115 ± 2 ^C	21 ± 1 ^C

Note. Data ± SEM (N); SEM values < 1.5 were listed as 1; corticosterone (B) was administered in the feed (5 ppm); bGH (bovine growth hormone) and oGH (ovine growth hormone) were injected (0.5 mg/kg/day); mean values within a study are different if there are no common superscript letters ($P < 0.05$).

growth during the same treatment period for the hypophysectomized animals were approximately 81 and 46% of the control values. Sham operations had only a very slight effect on body growth (see Table I, Studies 1 and 2). Final body weight and body weight gain of the hypophysectomized cockerels were unaffected by the corticosterone treatment, but there was a slight reduction in shank-toe growth (Study 1). Final body weight, body weight gain, final shank-toe length and increase in shank-toe length after 18 days of treatment with bGH and corticosterone were increased 19, 46, 5, and 33%, respectively, compared to the values for hypophysectomized chickens receiving corticosterone alone (Study 2). These same endpoints were increased 14, 23, 6, and 24%, respectively, after 19 days of treatment with oGH in hypophysectomized chickens not receiving corticosterone (Study 4). Final body weight and body weight gain after 18 days of treatment with bPRL and corticosterone were increased 11 and 27% compared to the values for corticosterone-treated hypophysectomized chickens (Study 3), but bone growth was unaffected by the PRL.

Organ and tissue weights. The effects of hypophysectomy and hormone replacement on organ and tissue weights are summarized in Tables II and III. The sham operation had little or no effect on organ growth.

Heart and pectoralis. Heart and pectoralis weights of hypophysectomized chickens were approximately 52 and 70%, respectively, of intact/sham values (Study 1), but were unaffected by corticosterone treatment. Bovine GH increased heart weight by 25% in corticosterone-treated hypophysectomized chickens (Study 2), but pectoralis weight was unaffected. Ovine GH increased heart weight by 29% in hypophysectomized chickens not receiving corticosterone (Study 4), and pectoralis was again unaltered. PRL had no effect on heart or pectoralis growth.

Thymus and bursa. Thymal and bursal weights of hypophysectomized chickens were approximately 20 and 51%, respectively, of intact/sham values (Study 1), and corticosterone treatment further reduced their weights (46–62% of values for hypophysectomy alone; Study 1). GH treatment enhanced thymal weights by 71% in corticosterone-treated hy-

TABLE II. EFFECTS OF CORTICOSTERONE (B), GROWTH HORMONE (GH), AND PROLACTIN (PRL) ON HEART, PECTORALIS, THYMIC, AND BURSAL WEIGHTS IN HYPOPHYSECTOMIZED (Hx) CHICKENS

Treatment	Heart (g)	Pectoralis (g)	Thymi (g)	Bursa (g)
Study 1				
Intact (10)	4.28 ± 0.19 ^A	27.75 ± 0.62 ^A	5.74 ± 0.38 ^A	3.58 ± 0.28 ^A
Sham (10)	3.92 ± 0.12 ^A	25.65 ± 0.74 ^B	4.87 ± 0.15 ^B	3.84 ± 0.16 ^A
Hx (17)	2.15 ± 0.10 ^B	18.73 ± 0.59 ^C	1.05 ± 0.08 ^C	1.90 ± 0.13 ^B
Hx + B (15)	2.45 ± 0.10 ^B	18.39 ± 0.45 ^C	0.48 ± 0.02 ^D	1.17 ± 0.08 ^C
Study 2				
Intact (9)	4.03 ± 0.08 ^A	22.65 ± 0.47 ^A	4.66 ± 0.27 ^A	3.49 ± 0.22 ^A
Sham (10)	3.78 ± 0.12 ^A	23.09 ± 0.46 ^A	4.44 ± 0.22 ^A	3.18 ± 0.16 ^A
Hx + B (10)	2.06 ± 0.09 ^B	15.46 ± 0.73 ^B	0.55 ± 0.04 ^B	1.42 ± 0.08 ^B
Hx + B + bGH (11)	2.57 ± 0.12 ^C	16.10 ± 0.51 ^B	0.94 ± 0.07 ^C	1.65 ± 0.10 ^B
Study 3				
Intact (10)	3.15 ± 0.08 ^A	21.67 ± 0.41 ^A	4.97 ± 0.32 ^A	3.78 ± 0.19 ^A
Hx + B (9)	1.96 ± 0.06 ^B	16.11 ± 0.47 ^B	0.59 ± 0.10 ^B	1.46 ± 0.14 ^B
Hx + B + PRL (9)	1.92 ± 0.09 ^B	16.99 ± 0.42 ^B	0.64 ± 0.06 ^B	1.45 ± 0.11 ^B
Study 4				
Intact (7)	3.58 ± 0.12 ^A	31.92 ± 0.99 ^A	5.40 ± 0.44 ^A	3.38 ± 0.21 ^A
Hx (7)	1.71 ± 0.11 ^B	21.80 ± 1.10 ^B	1.32 ± 0.04 ^B	1.72 ± 0.19 ^B
Hx + oGH (7)	2.20 ± 0.20 ^C	24.28 ± 1.20 ^B	2.55 ± 0.24 ^C	2.23 ± 0.16 ^B

Note. Data ± SEM (N); corticosterone (B) was administered in the feed (5 ppm); bGH (bovine growth hormone) and oGH (ovine growth hormone) were injected (0.5 mg/kg/day); mean values within a study are different if there are no common superscript letters ($P < 0.05$).

hypophysectomized chickens, and by 93% in hypophysectomized animals not receiving corticosterone. GH treatment did not significantly affect bursal weights of hypophysectomized chickens with or without concurrent corticosterone treatment. PRL had no effect on the lymphoid organ weights in corticosterone-treated hypophysectomized chickens.

Liver and adipose tissue. Hypophysectomy reduced liver weight (57% in Study 1; 67% in Study 4), but had no effect on abdominal adipose tissue weight. Corticosterone treatment increased liver and adipose tissue weights by 51 and 458% of the values for hypophysectomized cockerels (Study 1). GH further increased liver and adipose tissue weights in the corticosterone-treated birds (69 and 126%, respectively, Study 2), but did not affect these endpoints in hypophysectomized chickens not receiving corticosterone (Study 4). PRL also greatly increased liver and adipose tissue weights in corticosterone-treated hypophysectomized chickens (79 and 75%, respectively, Study 3).

Discussion. The reduction in body and organ growth following hypophysectomy in the present studies is comparable to that which we have observed previously (15; unpublished

observations, C. Scanes *et al.*). Mammalian GH enhanced body weight gain and bone growth of hypophysectomized chickens, whereas PRL increased body weight to a lesser degree and had no effect on bone growth. The growth-promoting effect of mammalian GH in hypophysectomized chickens confirms our previous unpublished observations several years ago with a less purified porcine GH. More recently we observed only a slight increase in body growth following administration of chicken GH to hypophysectomized chickens, but the dosage was $\frac{1}{5}$ that used in the present studies (unpublished observations, C. Scanes *et al.*). Mammalian GH and PRL had only a small effect on the body weight gain of hypophysectomized pigeons except when the hormones were administered in combination with prednisone and T_4 (14). Administration of oGH to hypophysectomized rat pups (1 mg/kg/day from 7 to 15 days of age) increased body weight gain by 32%, but had no significant effect on tail length (19). A higher dose of GH (fourfold) increased body weight gain and tail length by 68 and 27%, indicating a greater effect on body weight gain than bone growth. We observed a greater effect of GH on body weight gain than bone growth (shank-

TABLE III. EFFECTS OF CORTICOSTERONE (B), GROWTH HORMONE (GH), AND PROLACTIN (PRL) ON LIVER AND ABDOMINAL ADIPOSE TISSUE WEIGHTS IN HYPOPHYSECTOMIZED (Hx) CHICKENS

Treatment	Liver (g)	Abdominal adipose tissue (g)
Study 1		
Intact (10)	15.1 ± 0.2 ^A	
Sham (10)	14.7 ± 0.4 ^A	2.1 ± 0.4 ^A
Hx (17)	8.5 ± 0.4 ^B	1.5 ± 0.1 ^A
Hx + B (15)	12.8 ± 1.1 ^A	8.5 ± 1.1 ^B
Study 2		
Intact (9)	14.2 ± 0.4 ^A	7.1 ± 0.5 ^A
Sham (10)	13.3 ± 0.4 ^A	4.4 ± 0.4 ^A
Hx + B (10)	9.8 ± 1.3 ^B	5.3 ± 1.0 ^A
Hx + B + bGH (11)	16.6 ± 1.4 ^A	12.0 ± 1.7 ^B
Study 3		
Intact (10)	13.7 ± 0.3 ^A	5.2 ± 0.7 ^A
Hx + B (9)	9.6 ± 0.9 ^B	7.1 ± 1.0 ^A
Hx + B + PRL (9)	17.2 ± 1.6 ^A	12.4 ± 1.0 ^B
Study 4		
Intact (7)	14.8 ± 0.5 ^A	2.6 ± 0.8 ^A
Hx (7)	9.9 ± 0.5 ^B	1.6 ± 0.2 ^A
Hx + oGH (7)	11.2 ± 0.7 ^B	2.8 ± 0.9 ^A

Note. Data ± SEM (N); corticosterone (B) was administered in the feed (5 ppm); bGH (bovine growth hormone) and oGH (ovine growth hormone) were injected (0.5 mg/kg/day); mean values within a study are different if there are no common superscript letters ($P < 0.05$).

toe length increase) in corticosterone-treated hypophysectomized chickens (46% vs 33% increase), but the two endpoints of growth showed the same responsiveness (23% vs 24%) to GH in hypophysectomized cockerels not receiving corticosterone (Table I). Ovine PRL alone did not enhance body weight gain or tail length in hypophysectomized rat pups, but it antagonized somewhat the growth-promoting effects of exogenous GH (19). PRL increased the body weight of hypopituitary dwarf mice, but it was less effective in promoting growth than GH or T₄ (20).

Several studies have shown that plasma GH levels in various avian species tend to be high during the early rapid phase of growth and lower in later stages of growth and in adults, but there are also many examples where plasma concentrations of GH do not correlate well with the rate of body growth (for review see (5, 21)). The present paper provides evidence that mammalian GH enhances body weight and bone growth in the hypophysec-

tomized chicken. These observations indicate that further study of GH influence on the growth of birds is warranted. Since mammalian GH is also biologically active on several metabolic endpoints in the chicken (5, 21, 22), elaboration of its effect on the growth of hypophysectomized chickens would seem justified.

Mammalian GH enhanced heart weight of our hypophysectomized chickens to about the same degree as body weight when the hormone was administered with corticosterone and to a somewhat greater degree than body weight when administered in the absence of corticosterone. Pectoralis muscle weight, on the other hand, was unaffected by GH treatment either in the presence or absence of corticosterone. Growth hormone treatment of hypophysectomized rats for a longer period (59 days) than in our studies increased both heart and skeletal muscle weights, but to a lesser extent than it stimulated body weight gain (23). Skeletal muscle weights were increased more than heart weight following GH treatment of the hypophysectomized rats (23). Growth hormone treatment of hypophysectomized rats for as little as 7 days increased the weight and DNA content of several skeletal muscles, including the pectoralis (24, 25). Growth hormone treatment also caused small increases in rat muscle protein and RNA synthesis, and large increases in the efficiency of protein synthesis (26). Further studies are needed to clarify the apparent lack of responsiveness of skeletal muscle to GH in our hypophysectomized chickens. This would seem particularly important to our understanding of body growth, because muscle constitutes such a large proportion of the body mass.

Since lymphoid organ weights, especially at the age of the birds in our study, are not sensitive indicators of the immune response (for review see (27)), it would seem inappropriate to discuss the functional significance of the lymphoid organ weight changes observed in the present study. It should be noted, however, that the immunological competence of rats is impaired by hypophysectomy and restored by either PRL or GH (28). Since lymphoid organ weight changes in the hypophysectomized chicken have not been reported previously, we have discussed them in the context of the limited information available. Both thymal and bursal weights were greatly reduced in the hy-

pophysectomized chickens, but the thymus was affected more than the bursa (see Table II). Bursal weight was unaffected 10 days after hypophysectomy of 6-week-old pigeons (14), but thymal weight was significantly less as early as 13 days after hypophysectomy of the rat (29). It is known that glucocorticoid administration depresses thymal and bursal weights of intact chickens (2, 3, 18, 30), but the further reduction in these lymphoid organ weights in corticosterone-treated hypophysectomized chickens observed in the present studies has not been reported previously. Thymal weight increased substantially in the GH-treated hypophysectomized chickens, but GH had no significant effect on bursal weight. Ovine GH in the absence of corticosterone enhanced thymal weight to a greater extent than bGH in corticosterone-treated hypophysectomized chickens. Since oGH was not more effective on other endpoints of growth, it would appear that GH stimulation of the thymus is greater in the absence of exogenous glucocorticoid. Although mammalian GH did not significantly increase thymal and bursal weights of intact chickens (2, 3), there is evidence that these organs are responsive to GH. The administration of a low dose of chicken GH enhanced thymal weight of hypophysectomized chickens and further elevated bursal weight when administered in combination with T₃ (unpublished observations, C. Scanes *et al.*). Bovine GH administration partially reversed the decrease in bursal weight induced by glucocorticoid treatment of intact chickens (2). Growth hormone alone stimulated a small increase in bursal weight of the hypophysectomized pigeon, whereas bursal weight was doubled when GH was administered in combination with PRL and T₄ (14). Both primary antibody response and bursal weight were increased following the administration of mammalian GH to sex-linked dwarf chickens (11).

Our data on liver and adipose tissue weights are difficult to interpret without concurrent data on related metabolic endpoints. Growth hormone alone had no effect on these organ weights in hypophysectomized chickens, but both GH and PRL increased their weights substantially when the hormones were administered in combination with corticosterone. Liver and adipose tissue weights of hypophysectomized pigeons were increased following GH and PRL administration, and the

response was greatly enhanced by prednisone and T₄ (14). It has been suggested that PRL plays an important role in the regulation of fattening in migratory birds (31). Since the data from several studies (for review see (5, 21)) are consistent with GH having a lipolytic and an antilipogenic effect in birds, an increase in adipose tissue weight following GH administration would not be expected. Our observation that adipose tissue weight was unaltered following hypophysectomy was also unexpected. Carcass fat increases substantially following removal of the pituitary in older chickens (16, 32, 33). Since corticosterone greatly enhanced adipose tissue weight of young hypophysectomized chickens in the present study, as well as in somewhat older hypophysectomized chickens (34), it might be speculated that differences in glucocorticoid production following hypophysectomy at different ages are at least partly responsible for differences in adiposity. But this does not seem to be the case because plasma corticosterone levels in chickens hypophysectomized in our laboratory at 3 weeks of age were 53% of control values at 24 days after hypophysectomy (35), which is nearly identical to the 50% reduction reported for older birds following hypophysectomy (see 36).

In conclusion, these results demonstrate that mammalian GH augments body weight gain, bone growth and the growth of several organs in hypophysectomized chickens. Mammalian PRL enhanced body weight gain, liver weight, and adipose tissue weight in corticosterone-treated hypophysectomized chickens, but did not influence bone growth or the weights of the heart, pectoralis, thymi, or bursa.

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