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Testosterone and Adiposity in The Chicken: The Effect of Breed and Sex

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ROBINZON, B., I. ROZENBOIM, N. SAYAG, G. GVARYAHU, J. WAXLER AND N. SNAPIR. *Testosterone and adiposity in the chicken: The effect of breed and sex.* PHARMACOL BIOCHEM BEHAV 27(2) 223–226, 1987.—The leaning role, previously suggested for testosterone in chickens, was tested in males and females of fatty and lean breeds. The breeds used were the White Leghorn (WL), of which the male is very lean, and the White Giant (WG) and the White Cornish × White Rock (WCWR), in which both sexes accumulate fat. Castration of WL males induced adiposity and replacement therapy with testosterone propionate (TP) reduced the fat to normal. However, in males of the fatty breeds, neither castration nor TP administration had any effect on adiposity. Similarily, long-term supplementation of a high dose of TP had no effect on the level of adiposity in females of both the heavy and the light breeds. Thus, the leaning effect found for testosterone in the WL male is not a general phenomenon in chickens but rather a specific response of the WL male.

Chicken Adiposity Testosterone Castration Estradiol Breed Sex

IN the White Leghorn (WL) cockerels, castration resulted in adiposity, and TP administration reduced body fat [4,9]. In WL males of which the basomedial hypothalamus was bilaterally destructed, hyperphagia and adiposity develop, concomitant with involution of the testes, and reduction of plasma testosterone to almost nothing. TP administration to these functionally castrated cocks reduced food intake and adiposity, and alleviated the liver fatty hypertrophy [10]. However, the WL is a light breed which was bred to be lean, and in this characteristic, differs from the heavy breeds which were developed for meat production, and accumulate fat during growth. Force-feeding of chicks revealed a difference in the propensity to be overfed in these breeds. WL chicks could be overfed to 170% of the normal consumption, but in chicks of the heavy breeds the maximum excess was only 13%. Thus, the force-feeding induced more adiposity in the light breed than in the heavy one [6].

This report presents data collected in various experiments, carried out in our laboratory, during which a marked difference in the adiposity response to testosterone was found between the WL cock and the WL hen and between the WL male and both male and female of heavy breeds.

METHOD

Animals

The chickens used for the experiments below were either White Leghorns (WL) as the light breed, or White Cornish × White Rock cross bred chicks (WCWR) and White Giant (WG) as the heavy breeds, all supplied by the Isreali Chicken Breeders Union farms. All the birds were kept in individual cages, and subjected to 14 hours of light daily (at the intensity of 5 lux at the height of the animals) as radiated by 60 watt incandescent bulbs. They were fed a commercial starter mash until 5 weeks of age and thereafter with commercial breeder mash. Both food and water were provided ad lib.

Surgical and Experimental Procedures

Orcidectomy and sham-operation, involving opening of the abdominal wall only, were performed under pentobarbital anasthesia. Because the experiments of which the data collected were of different aims, the experimental procedures vary and will be described for each experiment separately. However, in all the experiments at autopsy the abdominal adipose tissue was removed, cleaned from adhering tissues, and weighed. Statistical analysis of data was carried out using ANOVA and Duncan's Multiple Range Test [2].

Experiment 1

Forty WL males were used. At 8 weeks of age, 32 were castrated and the rest were left intact and served as controls. At 40 weeks of age, 16 of the castrated ones were injected with 0.5 mg/day of testosterone propionate (TP) for the next six weeks. Then autopsy was carried out. This procedure produced three experimental groups: (1) Untreated controls, n=8; (2) Castrated, n=16; (3) Castrated which were injected with TP, n=16.

TABLE 1

BODY WEIGHT AND ITS CONTENT OF ABDOMINAL ADIPOSE TISSUE OF THE CHICKENS OF THE SIX EXPERIMENTS

Experiment	Breed	Sex	Treatment	n	Body Weight (g)	Abdominal Adipose Tissue (% of Body Weight)
1	WL	M	Control	8	1923 ± 50†	0.086 ± 0.043
1	WL	M	Castrated	16	2173 ± 127	2.686 ± 0.214*
1	WL	M	Castrated + TP	16	2086 ± 77	0.100 ± 0.051
2	WL	F	Control	20	1501 ± 56	1.940 ± 0.262
2	WL	F	TP	20	1586 ± 56	2.696 ± 0.484
3	WCWR	M	Control	15	3236 ± 53	1.518 ± 0.087
3	WCWR	M	Sham-Operated	15	3332 ± 192	1.668 ± 0.145
3	WCWR	M	Castrated	15	3008 ± 96	1.639 ± 0.111
4	WCWR	M	Control	15	2994 ± 80	1.377 ± 0.295
4	WCWR	M	Oil	15	3015 ± 101	1.370 ± 0.307
4	WCWR	M	TP	15	2938 ± 103	1.639 ± 0.229
5	WL	M	Control	6	2154 ± 115	0.076 ± 0.035
5	WL	M	Castrated	6	2208 ± 84	1.051 ± 0.184*
5	WL	M	Castrated + TP	6	2122 ± 67	0.094 ± 0.028
5	WCWR	M	Control	6	4651 ± 111	1.755 ± 0.651
5	WCWR	M	Castrated	6	4808 ± 77	1.582 ± 0.370
5	WCWR	M	Castrated + TP	6	4720 ± 204	1.654 ± 0.422
6	WG	M	Control	15	2406 ± 123	1.368 ± 0.243
6	WG	M	TP	15	2157 ± 252	1.710 ± 0.149
6	WG	F	Control	15	2014 ± 42	2.410 ± 0.034
6	WG	F	TP	15	1979 ± 77	2.376 ± 0.196
7	WCWR	M	Control	15	2923 ± 95	1.590 ± 0.149
7	WCWR	M	EB	15	3012 ± 89	3.039 ± 0.294*

*Significantly differs from the control ($p<0.05$).
†SEM.

Experiment 2

In this experiment, 40 seven month old WL laying hens were used. Laying hens were chosen because they are fattier then pullets. Twenty were injected with 0.5 mg/day TP for the next eight weeks and the remainder served as untreated controls. Autopsy was performed at the age of 48 weeks.

Experiment 3

Forty-five WCWR males were used. At the age of 5 weeks, 15 were castrated, 15 underwent sham-operation, and the rest served as controls. Autopsy was performed at 10 weeks of age.

Experiment 4

Forty-five WCWR males were used. At 4 weeks of age they were divided into 3 equal experimental groups as follow: (1) Untreated controls; (2) Cockerels injected with 0.5 ml/day of corn oil; (3) Cockerels injected with 1.0 mg/kg/day of TP dissolved in 0.5 ml of corn oil. Autopsy was made at the age of 10 weeks.

Experiment 5

Eighteen WL males and 18 WCWR cockerels were used. At 9 weeks of age 12 cockerels of each breed were castrated and six served as controls. At 19 weeks of age half of the castrated birds of each breed were injected with 3.0 mg/kg/day of TP for the next 12 weeks. Autopsy was made at 31 weeks of age.

Experiment 6

Thirty WG male and 30 WG females were used. At 4 weeks of age, birds of each sex were divided into 2 groups: one was injected with 1.0 mg/kg/day TP and the other was left untreated as controls. Autopsy was made at 8 weeks of age.

Experiment 7

Thirty WCWR males were used. At 4 weeks of age they were divided into two equal groups; one was injected with 1 mg/kg/day of estradiol benzoate (EB) and the other was untreated controls. Autopsy was made at 10 weeks of age.

RESULTS

The effects of the various treatments in the seven experiments on body weight and adiposity are presented in Table 1.

Castration of WL males induced adiposity (Experiments 1 and 5), while no such effect was observed in WCWR males (Experiments 3 and 5). While TP administration to castrated WL male nulified adiposity (Experiments 1 and 5), it had no effect on the adipose mass of castrated (Experiment 5), or intact (Experiments 4 and 6) males of the heavy breeds. TP had no effect on the fat mass of WL laying hens (Experiment 2), and of WG females (Experiment 6).

Estradiol benzoate did induce adiposity in WCWR males (Experiment 7).

DISCUSSION

In an earlier study, it was found that in the WL male testosterone reduces adiposity and prevents the fattening effect of castration when administered immediately following the orchidectomy [9]. In Experiment 1, TP administration to WL cockerels, which were already fat due to the castration, nullified their adiposity. Thus, in the WL male, where the removal of the testes induces a tremendous increase in the abdominal adipose tissue weight, TP replacement therapy not only prevents the development of adiposity but also alleviates obesity that was already instated.

In the WL, the male is very lean and thus the leaning effect of testosterone becomes evident mainly in the castrated ones. However, the WL laying hen is naturally fat and has low plasma testosterone and thus resembles the castrated male. But in contrast to the castrated WL cockerel, TP administration to WL hen did not reduce adiposity.

The effects of castration and TP administration in the WL male led to the conclusion that in the male chicken, testosterone has a leaning effect [4,9]. However, in the males of the heavy breeds of the present experiment, neither castration, nor testosterone administration, had any effect on adiposity. In this matter the females of the heavy breeds behave like the males. Thus, in these breeds, unlike the WL one, there is no sex difference in adiposity sensitivity toward testosterone, and both resemble the WL hen in their insensitivity.

Unlike their difference in adiposity responsiveness toward testosterone, the light and the heavy males did not differ in their response to estrogen. Estrogen was observed before to induce obesity in the WL male [9], and in the present study estradiol benzoate (EB) administered to WCWR cockerels increased adiposity as well. Thus, estrogen induces adiposity in both WL and WCWR cockerels, although only the WL ones are sensitive to the leaning effect of testosterone. This may suggest that a similarity between two breeds in their response to one gonadal steroid is not an indication for such a similarity in their response to another gonadal steroid. Furthermore, the antagonism in the androgenic and estrogenic effects on adiposity found in the WL cockerels [9] may evolve through different pathways.

The fattening role found for estradiol in both WL and WCWR cockerels is in agreement with previous reports for the chicken [1, 3, 5, 7, 9]. The increased adiposity in the castrated WL male and its alleviation with TP are in agreement with the leaning effect, previously suggested, for testosterone in the WL male [4, 9, 10]. This effect of testosterone was manifested in WL males, but not in WL females, nor in males and females of the heavy breed. One may suggest that the different goals for selection; for high fertility in the WL, and for rapid growth in the WCWR and the WG, preserved the sensitivity of the adipose tissue to testosterone in the males of the former and aborted it in those of the latter. However, in the WL females, testosterone did not reduce adiposity. Thus, even in this breed, females and males do not respond to testosterone in the same manner. It is possible that the ineffectiveness of testosterone in reduction of adiposity in the WL hen is the secondary result of circulating estrogens, because estrogen was found to antagonize the leaning effect of testosterone when both were administered simultaneously into WL males [19]. However, administration of such high doses of TP for such a long period was found to stop egg laying and to involute the ovary in WL females to a degree making estrogen secretion improbable. On the other hand, single injection of synthetic estrogen to newly hatched WL males resulted in a marked adiposity at maturity, 270 days later, although plasma testosterone was about four times higher than in the untreated control [8]. The obesity in the presence of high plasma testosterone may suggest that in those WL males that were exposed to a high level of estrogen, as neonates, a resistancy toward the leaning effect of testosterone had developed. If this is the case, then the inability of testosterone to reduce adiposity in hens of both the WL and the WCWR breeds may be the result of high estrogens in these birds at the embryonal and the neonatal stage. Exposure of WL male to a high level of testosterone as newly hatched resulted in a reduced abdominal adipose tissue weight as compared to the untreated controls, 270 days later [8]. Thus one may suggest that the exposure of the cockerel to testosterone at this early age increases the sensitivity to the leaning effect of testosterone at a later age. It is beyond the scope of this article to suggest what are the mechanisms that turned the cockerels of the light breed into sensitive, and those of the heavy breeds into insensitive, toward the leaning effect of testosterone. However, experiments are being conducted in our laboratory, to test the effects of embryonal and neonatal manipulations with gonadal steroids as well as with their competitive inhibitors on the level of adiposity at the later ages in both male and female chickens of the heavy and light breeds.

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