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The Involvement of Gonads and Gonadal Steroids in the Regulation of Food Intake, Body Weight and Adiposity in the White Leghorn Cock[1]

N. SNAPIR, B. ROBINZON AND B. SHALITA

Department of Animal Science, Faculty of Agriculture, Hebrew University of Jerusalem P.O.B. 12, Rehovot 76100, Israel

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SNAPIR, N., B. ROBINZON AND B. SHALITA. *The involvement of gonads and gonadal steroids in the regulation of food intake, body weight and adiposity in the white leghorn cock.* PHARMACOL BIOCHEM BEHAV 19(4) 617–624, 1983.—The effects of long-term injections of testosterone propionate (TP), diethylstilbestrol (DES) and TP+DES simultaneously to castrated and sham-operated White Leghorn cocks on feeding, weight gain, obesity, blood lipids and weight of various glands were studied. DES induced marked adiposity while TP reduced carcass fat content. Injections of TP+DES induced only moderate obesity. The responses of the castrated cocks to TP or DES were not always parallel to those of the sham-operated ones. In sham-operated cocks, TP induced permanent hypophagia and emaciation while in castrated cocks, although alleviating adiposity, it did not reduce the rate of weight gain and induced only a transient hypophagia. DES induced permanent hyperphagia and accelerated weight gain in sham-operated cocks while in those castrated, it induced only transient hyperphagia which later on changed into hypophagia. Although the latter cocks did not gain more weight than those castrated with no steroids supplementation, they were much more obese and had a fat content similar to that of the sham-operated ones treated with DES. The castration was found to alleviate the depressing effect of TP on adenohypophyseal and thyroidal weights. The results may suggest: (1) In the White Leghorn cocks, DES increases lipogenesis and food intake while TP results in the contrary. (2) Castration should not be considered as a lack of gonadal steroids only.

Chicken Food intake Obesity Castration Testosterone propionate Diethylstilbestrol

THE involvement of gonadal steroids in the regulation of food intake and body weight in mammals has been well-documented. In the normal cycling female rat, food intake and body weight are increased during the diestrus and reduced during proestrus and estrus [2, 6, 7, 11, 71, 73]. Parallel changes in food intake during the estrus cycle were observed in hamsters as well [15]. In monkeys, food intake is significantly higher during the luteal phase when progesterone level is high than during the follicular phase when estradiol level is increased [10]. Ovariectomy was found to increase food intake and body weight in rats [4, 9, 17, 18, 35, 37, 57], mice [29], hamsters [22,55], dogs [32] and monkeys [10].

The increased body weight of ovariectomized rats was reported to be associated with enlargement of body fat mass [46]. Estradiol administration either peripherally [47, 54, 66, 72, 78] or to the hypothalamus [34,79] abolished both hyperphagia and increased body weight following ovariectomy. Both estradiol and diethylstilbestrol (DES) reduced food consumption and body weight in hypothalamic-hyperphagic rats and mice [39, 52, 62].

Castration of male rats and Djungarian hamsters resulted in hypophagia and decreased weight gain [3,30]. Replacement therapy with testosterone propionate (TP) to castrated rats increased food intake and weight gain at low doses, but depressed both of them at higher doses [14, 21, 64]. It was suggested that the inhibition of feeding and weight gain by high doses of TP was the consequence of aromatization of testosterone into estrogen [28,58]. In accordance with this assumption, estradiol was found to suppress feeding and weight gain in normal [5,51] and orchidectomized male rats [14, 20, 51]. Estradiol was also found to antagonize the increase in weight gain and eating induced by TP [14]. Receptors for estradiol were found in the adipose tissue of both male and female rats [26,27] and estradiol was found to decrease lipoprotein lipase activity in adipose tissue of rats of both sexes [26]. Thus, it was suggested that the weight and appetite-reducing effects of injected estradiol and of estradiol born by aromatization of testosterone, are secondary to the reduction in lipogenesis in the adipose tissue [26].

From the above data, one may conclude that, in mammals, estrogens suppress feeding and adiposity while testos-

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terone enhances feeding and weight gain. However, there are some reports that conflict with this conclusion. Castration of male rats was reported to increase food intake and body weight while TP administration resulted in the contrary [41,42]. Similar results were obtained in male hamsters and gerbils [36, 49, 59, 85]. Orchidectomy was also found to increase the adiposity level obtained by ventromedial hypothalamic lesions in rats [38]. In contrast to the lipolytic effect suggested for estradiol, estrogen was found to enhance lipogenesis and weight gain in weanling male rats with hypothalamic obesity [24]. Obesity in man was found to be accompanied by a decrease in plasma levels of testosterone and an increase in estrogen [40]. The lipolytic activity and adiposity reducing effects of testosterone in the human have been well-documented [43, 44, 74, 75]. Estrogenic compounds were suggested to increase lipogenesis in the human [23]. Thus, the role of estrogens and androgens in regulation of food intake and adiposity in mammals is still controversial since data from different species, and sometimes even from the same species [3, 38, 41, 42] are not always in agreement.

The role of sex hormones in regulation of feeding and adiposity in aves is not clear as well. In migratory birds, such as the White-crowned sparrow and the White-throated sparrow, orchidectomy prevented the premigratory hyperphagia and obesity [50, 70, 80, 82] and replacement therapy with TP restored this premigratory fattening [50, 70, 80, 82]. In the intact male Red-winged blackbird maintained under short-day photoperiod, TP administration evoked hyperphagia and adiposity [63]. In male pigeons, castration was followed by decreased body weight and an initial decrease in food intake while replacement therapy with TP increased feeding and body weight [61]. However, in cockerels, TP was found to increase lipolysis [45]. In cockerels functionally castrated by hypothalamic lesions, replacement therapy with TP reduced both food intake and adiposity [68]. Estradiol was found to enhance lipogenesis in chickens [1, 25, 33, 48] and quails [60].

Since the role of androgens and estrogens in regulation of food intake and adiposity in aves, as in mammals, is not conclusively clear, the following study was designed. In this study, the long-term effects of TP and DES supplementation to sham-operated and to castrated White Leghorn cocks, on food intake, adiposity and some parameters of lipid metabolism were investigated.

METHOD

Animals

Fifty-six White Leghorn cocks, 2 months old, were kept in individual cages, fed a commercial breeder mash (16% Protein, 5% Fat, 63% NFE, 5% Fiber and 8% Ash) and watered ad lib. They were subjected to 14 hours of light daily, at an intensity of 5 lux at the height of the animals as radiated by incandescent bulbs (60 watts each).

Surgical and Experimental Procedures

The cocks were divided into two equal groups of 28 birds each. Complete orchidectomy was performed on all birds of one group while sham-operation involving opening of the abdominal wall only was carried out in all cocks of the other group. Following surgery, each group was divided again into 4 subgroups and the following injections procedure was maintained every second day for the entire experimental period:

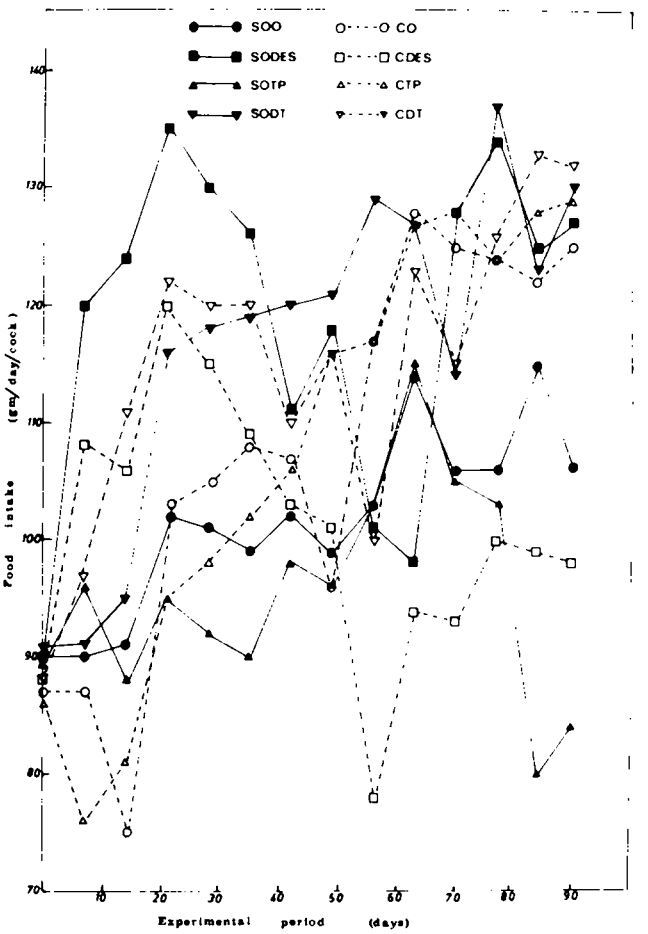

FIG. 1. Average daily food intake for the various experimental groups of cocks.

I. 0.2 ml corn oil
II. 1 mg DES (Teva Ltd., Israel)/0.2 ml corn oil
III. 1 mg TP (Assia Ltd., Israel)/0.2 ml corn oil
IV. 1 mg DES + 1 mg TP/0.2 ml corn oil.

All injections were IM. This procedure produced 8 experimental groups:

1. Sham-operated oil injected (SOO)
2. Sham-operated DES treated (SODES)
3. Sham-operated TP injected (STOP)
4. Sham-operated DES+TP treated (SODT)
5. Castrated oil injected (CO)
6. Castrated DES treated (CDES)
7. Castrated TP injected (CTP)
8. Castrated Supplemented with both DES an TP (CDT).

The experiment lasted for 90 days during which the following parameters were determined: individual daily food intake and body weight at 10 day intervals. Each tenth day, blood samples were drawn from the brachial vein for determination of hematocrit, plasma triglycerides [76] and plasma cholesterol [66].

Autopsy Procedure

The cocks were killed by decapitation. The following

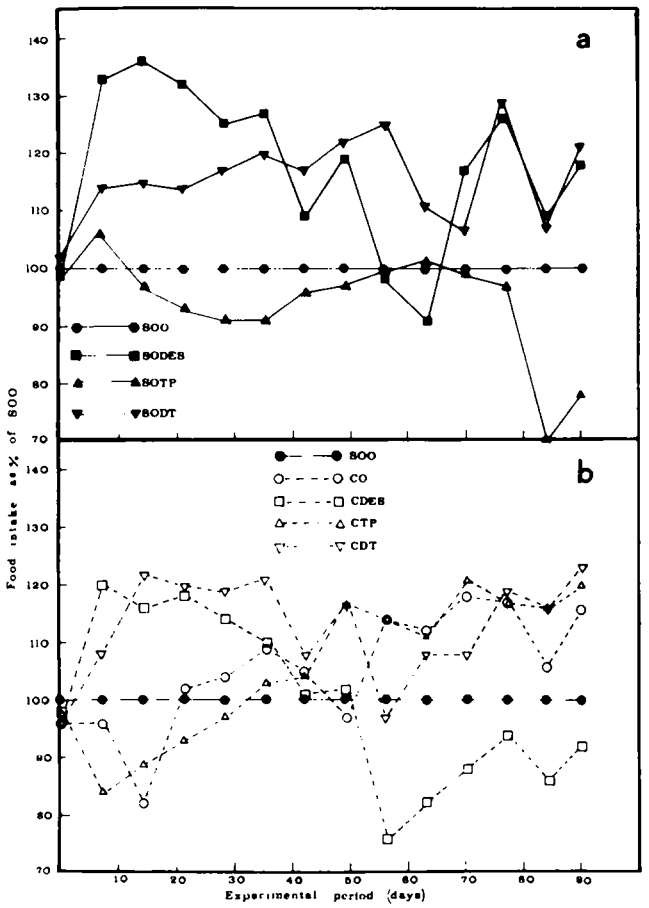

FIG. 2. Average daily food intake as percentage of that of the controls (SOO): (a) Food intake of the sham-operated cocks. (b) Food intake of the castrated cocks.

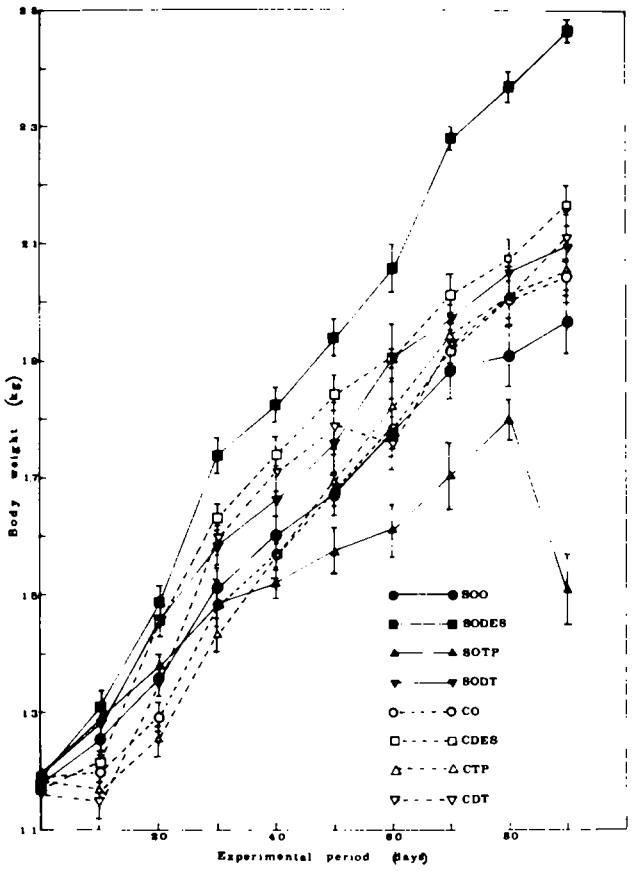

FIG. 3. Periodical changes in body weight of the various experimental groups of cocks. Vertical bars indicate the SE.

were immediately removed, cleaned from adhering tissues and weighed: abdominal adipose tissue, liver, testes, adrenals, thyroids and adenohypophysis. Samples of liver, abdominal adipose tissue and breast muscle were subjected to lipid extraction [16] and lipid percentage was determined by a gravimetric method. The testes were fixed in Bouin's Hollande, embedded in paraplast and serial section of 5 μm thickness each were prepared and stained with Hematoxylin Eosin. The seminiferous tubules diameter was measured in 5 sections from the middle of each testis. Statistical analysis of the data was carried out using ANOVA and Duncan's Multiple Range Test [8].

RESULTS

Castration of cocks (group CO) resulted in moderate hyperphagia (Figs. 1 and 2b) with no significant effect on weight gain (Fig. 3). In sham-operated cocks, supplementation of DES alone (SODES) and administration of both DES and TP (SODT) induced marked hyperphagia for most of the experimental period (Figs. 1 and 2a). However, while weight gain of the SODES cocks was significantly accelerated after 30 days, only non-significant elevation was observed in the SODT ones (Fig. 3). TP supplementation to sham-operated cocks (SOTP) depressed both food intake towards the end of the experiment, and weight gain, two months after start of treatment (Figs. 1, 2a and 3). In castrated cocks, these effects of the pharmacological manipulation were not always parallel to those in the sham-operated ones. DES administration to castrated cocks (CDES) induced hyperphagia only during the first 35 days of the experimental period and induced hypophagia for the last 5 weeks of the experiment (Figs. 1 and 2b). Although the CDES cocks were the heaviest among the castrated birds, they gained weight significantly less than the SODES ones for the last 60 days of the experiment (Fig. 3). In contrast to the TP effects in sham operated cocks, (SOTP), TP administration to castrated ones (CTP) depressed food intake only at the first 28 days of the experimental period and then had no noticeable effect on this parameter, so that both food intake and weight gain of the CO and the CTP cocks behaved similarly and were significantly higher than that of the SOTP birds during the last 35 days of the experimental period (Figs. 1, 2b and 3).

Supplementation of both DES and TP to castrated cocks (CDT) induced more eating and more rapid weight gain than castration alone (CO) in the first 5 weeks of the experiment (Figs. 1, 2b and 3), but toward termination of experiment, both food intake and weight gain of the CDT cocks did not differ from those of the CTP and CO ones (Figs. 1, 2b and 3).

Although the CDES cocks ate and gained weight less than

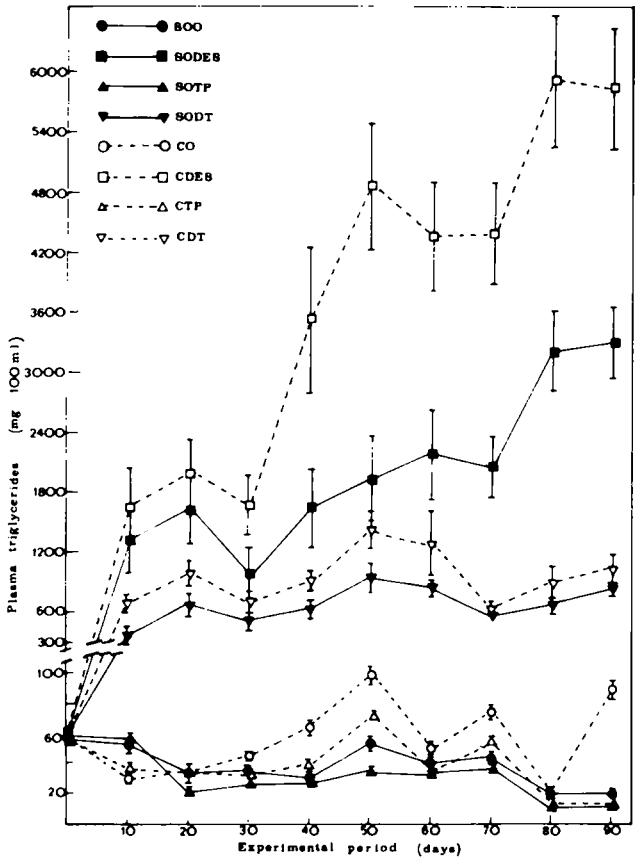

FIG. 4. Periodical changes in plasma triglycerides for the various expeimental groups of cocks. Vertical bars indicate the SE.

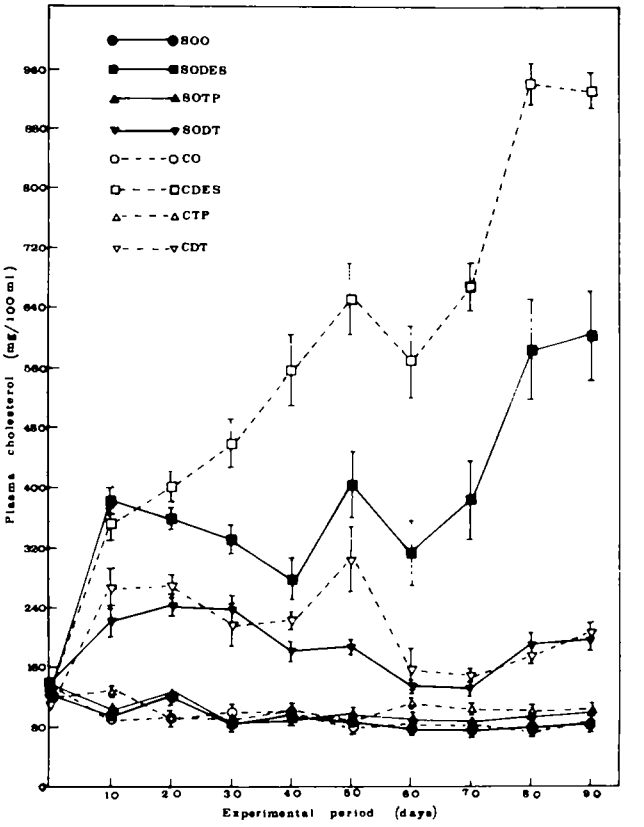

FIG. 5. Periodical changes in plasma cholesterol of the various experimental groups of cocks. Vertical bars indicate the SE.

the SODES ones, they had the highest plasma triglycerides and cholesterol levels during the last 60 days of the experimental period (Figs. 4 and 5). SODES cocks had higher plasma triglycerides and cholesterol than all other sham-operated cocks during most of the experimental period (Figs. 4 and 5). The combined supplementation of both DES and TP also increased plasma triglycerides and cholesterol in both sham-operated (SODT) and castrated (CDT) cocks during the whole experimental period, but significantly less than the administration of DES alone. Castration with no steroid administration (CO) induced only slight but significant increase in plasma triglycerides during the last 50 days of the experimental period but not in plasma cholesterol (Figs. 4 and 5). The increased plasma triglycerides in castrated cocks was suppressed by TP administration (CTP) so that at the end of the experiment, they had a similar level to that of the SOTP ones (Fig. 4).

The TP had a similar effect on hematocrit in castrated as in sham-operated cocks (Fig. 6). DES suppressed hematocrit value more in the CDES than in the SODES cocks. In both, the values were significantly below those of the CO, SODT and CDT ones during the last 60 days of the experimental period, and did not differ between them, but were significantly below the SOO value during the same period (Fig. 6).

At termination of experiment, the SODES had the highest body weight and the SOTP had the lowest while the rest of the experimentals did not differ significantly between them (Table 1). In accordance with the highest body weight, the SODES cocks had the highest amount of abdominal adipose tissue and the heaviest liver (Table 1). However, the CDES cocks that weighed significantly less than the SODES ones had only slightly less abdominal adipose tissue and lighter livers than those in the SODES group; both differences did not reach a level of significance (Table 1). The fat content in liver was even significantly higher in the CDES than in the SODES cocks (Table 1). The combined treatment of DES and TP also had an obesifying effect as manifested by the abdominal adipose tissue weights and by its fat content in both SODT and CDT cocks (Table 1). Castration alone also induced obesity that was manifested only in the weight of abdominal adipose tissue and its fat content (Table 1). TP administration to castrated cocks (CTP) reduced abdominal adipose tissue weight and the fat content in it and in the liver to levels similar to those of SOO ones so that they were leaner than the CO cocks (Table 1). However, TP supplementation to sham-operated cocks, although reducing body weight, did not induce significant reduction of lipids in abdominal adipose tissue or liver to below that of the SOO ones (Table 1). The total weight of liver for the SOTP cocks was the lowest of all the experimental groups (Table 1). No significant difference in lipid content in muscle were found between the experimental groups and the SOO group. However, the DES supplemented birds and the CO group had a significantly higher lipid content in muscle than those treated with TP.

DES or TP supplementation as well as the administration

of the two together to sham-operated cocks inhibited gonadal development as can be seen from the testicular weight and seminiferous tubules diameter of the SODES, SOTP and SODT cocks in comparison with those of the SOO birds (Table 2). This inhibition was most severe in the SODT cocks. These treatments caused some reduction also in thyroidal and adenohypophyseal weights that was most pronounced in the SOTP and the SODT cocks (Table 2). Castration induced a significant increase in adenohypophyseal weight and some elevation of thyroidal weight (CO, Table 2). These effects were alleviated in the CDES and the CDT groups (Table 2). However, TP supplementation to castrated cocks failed to reverse the increase in adenohypophyseal and thyroidal weights induced by castration (CTP, Table 2).

DISCUSSION

In general, results of the present experiment suggest that, in the White Leghorn male, castration or administration of estrogenic compound induces increased food intake and obesity development while testosterone administration reduces adiposity. These data are in agreement with previous reports for the chicken [1, 33, 45, 48, 60, 68], human being [40, 43, 44, 74, 75], male rat [24, 38, 41, 42], hamster [36,85] and gerbil [49,59]. However, these results are in conflict with the lipogenic role of testosterone and the lipolytic role of estrogen suggested in many other studies with mammals ([12, 13, 19, 65, 69, 77, 83] and birds [50, 61, 63, 70, 80, 82]).

There were some differences between the castrated and the sham-operated cocks in the effects of TP and DES. While in the sham-operated, TP reduced food intake for the whole experimental period and induced emaciation, it caused only temporary hypophagia in the castrated cocks and although it diminished the adiposity, it did not reduce the weight gain rate. The hyperphagia induced by the DES in the sham-operated cocks was permanent for most of the experimental period while in the castrated ones, it changed into hypophagia at the middle of the experimental period. Thus, the SODES cocks gained more weight than the CDES ones. However, the CDES cocks had the highest levels of plasma triglycerides and cholesterol and were at a similar level of adiposity as the SODES ones. It may be suggested that the more rapid increase in blood lipids may be involved in the reduction of food intake from hyperphagia into hypophagia in the CDES cocks. It is possible that the presence of the testes in the sham-operated cocks inhibited the rise in plasma lipids in these birds and thus enable the hyperphagia to continue until the end of the experimental period.

The absence or presence of the testes may be the cause for the difference in feeding behavior and growth pattern between the SOTP and the CTP cocks. One may suggest that in the presence of testes, there is more testosterone that increases the androgenic activity. However, considering the pharmacological dosage of TP administered and the similar values of hematocrit for the SOTP and the CTP cocks, this does not seem to be the case. Furthermore, in absence of the testes, TP administration did not reduce the weights of the adenohypophysis and the thyroids that were elevated due to castration. In the sham-operated cocks, TP did reduce the weights of these glands. These findings are in agreement with the inability of TP to reduce pituitary weight and its FSH content in caponized cocks [84] and with the reduction of the intensity of the negative feedback exhibited by testosterone on LH secretion in hemicastrated cocks [81]. Thus, it may be

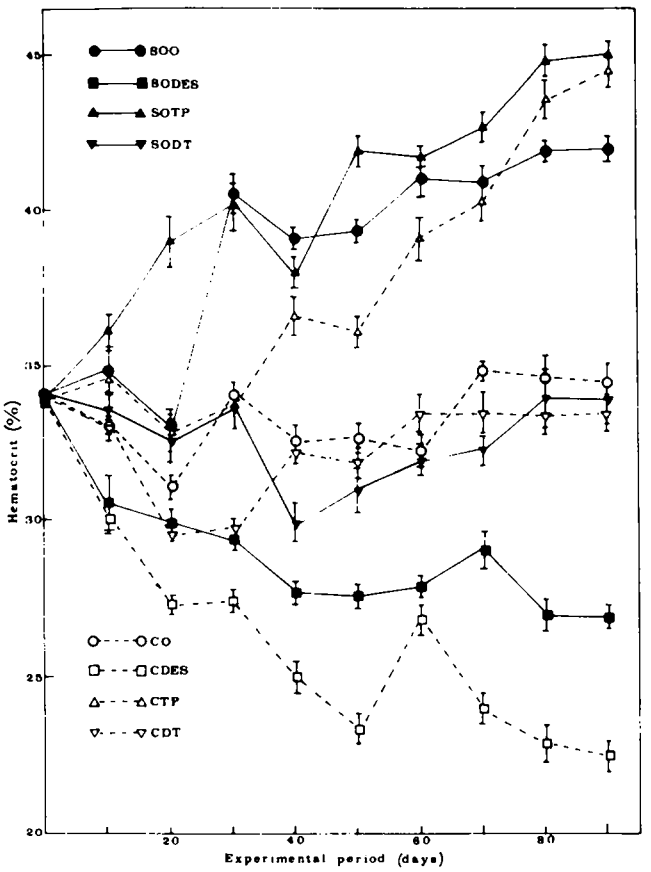

FIG. 6. Periodical changes in hematocrit values for the various experimental groups of cocks. Vertical bars indicate the SE.

suggested that the effects of the presence or absence of gonads are not solely mediated by the subsequent changes in gonadal steroids' levels and that some other gonadal factor may be involved. A gonadal factor that is non-steroid, the inhibin, was found to be involved in the negative feedback imposed by the gonads on gonadotropins' secretion [31,53]. The possible role of such gonadal factors in regulation of food intake and obesity should be studied.

The alterations in food intake and obesity after simultaneous administration of DES and TP seem to be the net results of the antagonism between the lipolytic effect of the androgenic compound and the lipogenic effect of the estrogenic one. Thus, the SODT and the CDT cocks were moderately hyperphagic and were less obese than the SODES and the CDES ones. The moderate elevation of plasma lipids in the cocks treated with these two compounds simultaneously may be the result of this antagonism.

The present study demonstrates that in the White Leghorn cock, androgen has a lipolytic effect and induces reduction in food intake while estrogen has the opposite effect. Furthermore, it suggests that the castrated cock does not always react to administration of gonadal steroids as does the normal cock and that castration should not always be considered just as a lack of gonadal steroids.

TABLE 1

SOME ADIPOSITY CRITERIA AT TIME OF AUTOPSY FOR THE VARIOUS GROUPS OF COCKS

Treatment	Body weight (g)	Abdominal adipose tissue weight (g)	Lipid in abdominal adipose tissue (%)	Liver weight (g)	Lipid in liver (%)	Lipid in muscle (%)
SOO n=7	1970 ± 54* (b)†	1.0 ± 0.3 (c)	40 ± 5 (c)	44.1 ± 2.7 (b)	4.2 ± 0.2 (c)	0.97 ± 0.06 (ab)
SODES n=7	2465 ± 18 (a)	104.5 ± 11.9 (a)	91 ± 2 (a)	70.2 ± 3.2 (a)	8.8 ± 0.4 (b)	1.31 ± 0.12 (a)
SOTP n=7	1510 ± 60 (c)	0.8 ± 0.2 (c)	39 ± 5 (c)	26.1 ± 1.8 (c)	3.6 ± 0.3 (c)	0.86 ± 0.07 (b)
SODT n=7	2099 ± 36 (b)	45.0 ± 4.9 (b)	76 ± 4 (b)	49.5 ± 3.2 (b)	5.7 ± 0.4 (c)	1.07 ± 0.08 (a)
CO n=7	2044 ± 32 (b)	30.1 ± 4.2 (b)	90 ± 3 (a)	37.8 ± 2.7 (b)	4.4 ± 0.3 (c)	1.07 ± 0.07 (a)
CDES n=7	2164 ± 38 (b)	82.3 ± 9.8 (a)	94 ± 2 (a)	57.2 ± 2.9 (a)	20.5 ± 1.0 (a)	1.19 ± 0.10 (a)
CTP n=7	2057 ± 55 (b)	0.9 ± 0.2 (c)	43 ± 5 (c)	36.9 ± 1.7 (b)	3.6 ± 0.3 (c)	0.88 ± 0.08 (b)
CDT n=7	2110 ± 84 (b)	65.4 ± 4.0 (a)	91 ± 3 (a)	46.1 ± 4.8 (b)	5.9 ± 0.4 (c)	1.05 ± 0.07 (a)

*Mean ± S.E.

†Figures not marked by the same letter are statistically different from each other ($p < 0.05$).

TABLE 2

WEIGHTS OF ADENOHYPOPHYSIS, ADRENALS, THYROIDS, TESTES AND SEMINIFEROUS TUBULES DIAMETER AT TIME OF AUTOPSY FOR VARIOUS EXPERIMENTAL GROUPS OF COCKS

Treatment	Adeno-hypophysis weight (mg)	Adrenal weight (mg)	Thyroid weight (mg)	Testes weight (g)	Seminiferous tubules diameter (μm)
SOO n=7	13.8 ± 0.9* (b)‡	184 ± 37 (a)	164 ± 14 (a)	21.6 ± 4.5 (a)	245 ± 22 (a)
SODES n=7	10.0 ± 0.8 (b)	215 ± 41 (a)	130 ± 15 (ab)	6.9 ± 0.9 (c)	145 ± 15 (b)
STOP n=7	9.4 ± 0.7 (b)	186 ± 38 (a)	117 ± 12 (b)	5.9 ± 0.8 (b)	150 ± 14 (b)
SODT n=7	9.9 ± 0.8 (b)	195 ± 45 (a)	114 ± 8 (b)	0.9 ± 0.2 (c)	65 ± 11 (c)
CO n=7	20.6 ± 1.2 (a)	166 ± 38 (a)	196 ± 17 (a)	—	—
CDES n=7	10.2 ± 0.8 (b)	198 ± 44 (a)	92 ± 8 (b)	—	—
CTP n=7	21.6 ± 1.7 (a)	185 ± 52 (a)	182 ± 13 (a)	—	—
CDT n=7	6.7 ± 0.6 (c)	185 ± 47 (a)	127 ± 16 (ab)	—	—

*Mean ± S.E.

‡Figures not marked by the same letter are statistically different from one another ($p < 0.05$).

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