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Control of Aggression and Dominance in White-Throated Sparrows by Testosterone and Its Metabolites

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In three experiments, we investigated whether testosterone itself or its metabolites activate aggression and dominance in white-throated sparrows *Zonotrichia albicollis*. Groups of five to six sparrows, each treated with a different steroid implanted subcutaneously, were observed in outdoor aviaries during late winter to determine the birds' rates of aggression (supplantations and attacks scaled to the number of available subordinates) and dominance rankings with opponents not previously encountered. In Experiment 1, testosterone (T) had a greater effect on aggression and dominance than did androstenedione, 5α-dihydrotestosterone (D), androsterone, or estradiol (E). In Experiment 2, birds with T or D + E had higher aggression scores and dominance ranks than birds with either D or E alone. Birds with T and D + E did not differ. The testosterone metabolites, D and E, thus acted synergistically to determine rates of aggression and dominance ranks. To corroborate these results, in Experiment 3 we treated T-implanted birds with the following blocking agents: ATD, expected to reduce conversion of T to E (AT birds); progesterone, expected to reduce conversion of T to D (PT birds); or both (APT birds). The APT birds had lower aggression scores and dominance ranks than did AT or PT birds, despite having higher mean levels of circulating T than AT or PT birds or birds implanted with T alone. Cyproterone acetate also reduced aggression scores and dominance in T-implanted birds. We conclude that the hormonal control of aggression and dominance in these birds requires conversion of testosterone to both androgenic and estrogenic metabolites.

In recent years it has become apparent that the action of testosterone (T) in activating male reproductive behavior and aggression in birds is often mediated by two metabolites of testosterone: 5α-dihydroxytestosterone (5α-DHT) and 17β-estradiol. To a large degree, these steroids represent distinct metabolic alternatives: 5α-DHT apparently is not aromatized to form an estrogen; and estradiol is not reconverted into an androgen (Massa, 1984; Ottinger, Adkins-Regan, Buntin, Cheng, DeVoogd, Harding, and Opel, 1984).

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Male reproductive behavior and aggression are influenced by both of these metabolites of testosterone, although findings vary somewhat among species and for different components of behavior. 5α-DHT partially restores sexual behavior and aggression in castrated male birds (Adkins and Pniewski, 1978; Adkins, Boop, Koutnik, Morris, and Pniewski, 1980; Adkins-Regan, 1981; Cohen and Cheng, 1982; Deviche and Schumacher, 1982; Balthazart, Schumacher, and Malacarne, 1984), and testosterone and 5α-DHT both stimulate agonistic behavior (Young and Rogers, 1978; Schmedemann and Haase, 1984).

Estrogen also activates both sexual behavior and aggression in male birds (Guhl, 1949; Adkins, 1977; Adkins *et al.*, 1980; Steimer and Hutchison, 1981; Harding, 1983; and Harding, Sheridan, and Walters, 1983). Furthermore, accumulating evidence suggests that estrogens and 5α-DHT interact in controlling the behavior of male birds (Adkins, 1977; Adkins and Pniewski, 1978; Adkins *et al.*, 1980; Harding, 1983; and Harding *et al.*, 1983). In some cases, treatment with 5α-DHT and estrogen in combination has a greater effect than treatment with either alone (Harding, 1983; Harding *et al.*, 1983). This result suggests that these two metabolites of testosterone interact synergistically, rather than additively, in controlling behavior.

Studies of steroid blockers and enzyme inhibitors have provided further indications that both estrogenic and 5α-androgenic metabolites of testosterone activate male reproductive and aggressive behavior in birds. For instance, the effects of testosterone on sexual and aggressive behavior in male birds can be blocked by progesterone (Erickson, Bruder, Komisaruk, and Lehrman, 1967; Komisaruk, 1967; Meyer, 1972; Meyer and Angelo, 1975), apparently as a result of competition with testosterone for 5α- and 5β-reductases (Balthazart, Marielle, Sennap, and Schumacher, 1982; Bottoni, Lucini, and Massa, 1985). The effects of testosterone on sexual behavior can also be blocked by administration of anti-estrogens, such as 1,4,6-androstatrien-3,17-dione (ATD), an inhibitor of aromatase (Adkins and Nock, 1976; Adkins *et al.*, 1980; Adkins-Regan, Pickett, and Koutnik, 1982). Interpretation of experiments with these steroid competitors and enzyme inhibitors requires care, however, since these agents do not have highly specific effects on steroid metabolism (Alexandre and Balthazart, 1987). Cyproterone acetate has particularly diverse effects (Adkins and Mason, 1974; Silver, 1977). It is known to compete with testosterone for receptor sites on cells, but it acts as an anti-estrogen as well as an anti-androgen in castrated rats (Luttge, Hall, Wallis, and Campbell, 1975).

In view of these findings, we addressed three specific questions concerning the effects of the estrogenic and androgenic metabolites of testosterone on aggression and dominance rankings of white-throated sparrows *Zonotrichia albicollis*.

First, do aromatizable and nonaromatizable androgens differ in their effects? In Experiment 1, we compared testosterone and androstenedione (AE) (two aromatizable androgens), 5α-DHT and androsterone (A) (two nonaromatizable androgens), and 17-β-estradiol, an estrogenic metabolite of testosterone.

Second, is there any synergistic effect of 5α-DHT and estradiol on aggression and dominance in this species? In Experiment 2, we compared birds treated with 5α-DHT or estradiol alone or with a combination of 5α-DHT and estradiol.

Third, do the actions of competitors and inhibitors help to elucidate the effects of testosterone and its metabolites on aggression and dominance in this species? In Experiment 3, we compared three different steroid competitors and synthetic inhibitors: ATD, an aromatase inhibitor; progesterone, a competitor for reductase; and cyproterone acetate, an anti-androgen and/or anti-estrogen. While recognizing that these agents probably do not have distinct effects on endocrine function, we designed this experiment with the expectation that their effects on testosterone metabolism were at least partly complementary. Thus, if aggression and dominance are influenced by a combination of 5α-DHT and estradiol, testosterone-treated birds also implanted with both ATD and progesterone or with cyproterone acetate should show little aggression.

METHODS

Experimental Subjects and Study Sites

During October through March 1985–1986, white-throated sparrows *Z. albicollis* of all age and sex classes were captured at the Mason Farm Biological Reserve near Chapel Hill, North Carolina. In this species, as in related species, the gonads regress during the nonbreeding season (testes 1.0–1.5 mm in length, ovaries 4–6 mm in length with follicles $<$ 0.5 mm in diameter) and circulating titers of gonadal hormones fall to very low levels (testosterone $<$ 0.5 ng/ml, estradiol $<$ 20 pg/ml) (Wingfield and Farner, 1978a; Wingfeld, 1984a; Schlinger, 1987; Archawaranon, 1987). In experiments similar to the ones reported here (Archawaranon, 1987), we have found that variation in coloration of the plumage does not influence dominance rankings in this species (see also Schlinger 1987).

The birds were individually marked with colored bands and their weights and wing chords were measured. Sex was determined by laparotomy and age was determined by examining the pneumatization of the cranium (Hamel, Beacham, and Ross, 1983). Prior to observations, the birds were kept separately in individual cages in a room with a natural photoperiod. They were fed mixed seeds (sunflower seeds, millet, and hulled oats), mixed greens, and water with a vitamin mixture. Observations were conducted in 12 outdoor aviaries (each 2.4 $\times$ 2.4 $\times$ 2.6 m) at least 6 m

apart. On one side of each aviary, an enclosed observation chamber permitted close study of the birds through a window of one-way glass.

Hormone Implants

Birds were implanted subcutaneously on the upper back with 10 mm of crystalline steroid (Sigma Chemical Co.) in the center of a 20-mm length of Silastic tubing (internal diameter 1.50 mm, external diameter 1.99 mm, Dow Corning Silastic Medical Grade Tubing). By means of preliminary radioimmunoassays, we empirically determined how large an implant of testosterone was necessary to maintain circulating androgen at the levels normally seen in breeding males of related species (white-crowned sparrows *Z. leucophrys,* Wingfield and Farner, 1978a, b; song sparrows *Melospiza melodia,* Wingfield, 1984a,b). Both ends of the Silastic tubes were plugged with tips of wooden toothpicks and sealed with Dow Corning medical adhesive. For controls, 20-mm lengths of Silastic tubing were prepared in the same way, but with nothing inside them. Implants were checked periodically to make sure that they were still in place and that they contained hormones. We have not found differences between the sexes in responses to testosterone and estradiol in experiments similar to those reported here (Archawaranon, 1987). However, we made an effort to include equal numbers of males and females in each treatment whenever sufficient numbers of each sex were available.

Plan of Experiments

Each experiment consisted of several stages. At the beginning of each stage, birds were released simultaneously into aviaries. Between stages birds were caught, weighed, examined, and regrouped with different opponents not previously encountered during our experiments and released into a different aviary. During the regrouping process, the birds were held indoors in individual cages for 1–3 hr. Thus in each successive stage an individual encountered new opponents in a new location, a procedure that allowed us to average each subject's responses to hormonal treatment over replicated social situations.

Behavioral Observations

Observations were conducted between 0900 and 1200 EST following 60-min deprivation of food. Each bird was the subject of two focal–individual samples (Altmann, 1974) for 15-min periods on separate days during each stage of the experiment. Aggressive behavior included supplantations, in which one bird displaced another from a perch or from a water or food tray at the center of an aviary, and attacks, in which one bird charged and pursued the other. If one bird supplanted or attacked another in 75% or more of their interactions, it was considered dominant over the other.

Birds in our studies formed predominantly linear dominance hierarchies (Archawaranon, 1987; Archawaranon, 1987). When an aviary included a triangle (nontransitive triad), we determined the birds' ranks by arranging the dominance matrix to minimize the number of interactions below the principal diagonal.

Behavioral Analysis

In each stage of an experiment, an aggression score was calculated for each bird by dividing its total number of supplantations and attacks by the number of birds subordinate to it in the same aviary. An individual's aggression score thus represented the number of its aggressive actions per available recipient and consequently had no necessary relation to the bird's rank.

Each bird's dominance rank was scaled for the number of birds in its aviary. For example, if there were six birds in an aviary, the top-ranking bird was given a score of 1 and the others were given scores of 0.8, 0.6, 0.4, 0.2, an 0, respectively. If there were five birds in an aviary, they were given scores of 1, 0.75, 0.5, 0.25, and 0, respectively.

Dominance rank in white-throated sparrows in the field has been found to correlate significantly with sex and age, but not with wing length, brightness of plumage, or weight (Piper and Wiley, 1988). In studies of birds in aviaries it has also been reported that adult males tend to be the highest-ranking birds (Watt, 1986a,b; but see Schlinger, 1987). Our results from previous experiments with captive white-throated sparrows did not show significant effects of sex, brightness of plumage, or age on rank (Archawaranon, 1987). Nevertheless, in the three experiments reported here we included equal numbers of birds of each sex and age class in each treatment, provided the necessary birds were available.

Analysis of Hormones

After each experiment, blood was drawn for radioimmunoassay of steroids, and the birds were then released with numbered bands. Blood samples were taken from a dorsal branch of the jugular vein in heparinized hematocrit capillary tubes. After centrifugation, plasma was separated and frozen at −80°C until analyzed.

Radioimmunoassays, performed in the laboratory of the Endocrinology/Gerontology Section, U.S. Environmental Protection Agency, Research Trinagle Park, North Carolina, followed procedures described by Jackson and Franklin (1984) and Goldman, Cooper, Rehnberg, Hein, McElroy, and Gray (1986). Each plasma sample was first extracted with 1 ml:10 ml water:methylene chloride, the extract was filtered with phase separation filter paper (Whatman 1PS), and the filtrate was dried under nitrogen.

The residue was resuspended in 100 μl of 100 m*M* phosphate-buffered saline with 1% bovine serum albumin, pH 7.4.

Measurement of plasma levels of testosterone and estradiol employed solid-phase radioimmunoassay kits that measured both bound and unbound hormones (Coat-A-Count, Diagnostic Products Corp., Los Angeles, CA). Forty microliters of the resuspended extract was added to each sample tube. The standard curves were run in triplicate and the unknowns were run in duplicate.

According to literature from Diagnostic Products Corp., the radioimmunoassay for testosterone is highly specific; cross-reaction with 5α-DHT is less than 9% and with other natural androgens is less than 4%. The assay for estradiol cross-reacts with other steroids less than 1.5%. Inter- and intraassay variances in this study were 4 and 3%, respectively. Recovery of known amounts of these hormones from sparrow plasma was about 85%.

Statistical Analysis

We used nonparametric statistical tests, two-tailed Mann–Whitney *U* tests and two-tailed Wilcoxon matched-pairs signed-ranks tests. For each test, the aggression scores and dominance ranks of each individual were averaged across the relevant stages of an experiment, so each individual contributed only one value, or one matched pair of values, to each test.

Experiment 1

In this experiment, we investigated whether androgenic or estrogenic effects of testosterone or both influence aggression and dominance. Five hormone treatments were used: two aromatizable androgens, T and AE; two nonaromatizable androgens, 5α-DHT (D) and A; and an estrogen, estradiol (E).

Methods

This experiment, conducted from October–December 1985, included 12 groups of six birds each. In each group, five birds received respectively the five different hormones assigned at random. The sixth bird received an empty implant. The birds were then kept separately in individual, visually isolated cages in a room with a natural photoperiod for 1 week before each group was released in a separate aviary (stage 1). Observations of aggression and dominance were then obtained as described above.

Four times, at 2-week intervals (stages 2, 3, 4, and 5), birds were regrouped in new aviaries with new opponents that they had not encountered before. In each stage, observations of aggression and dominance were repeated as above.

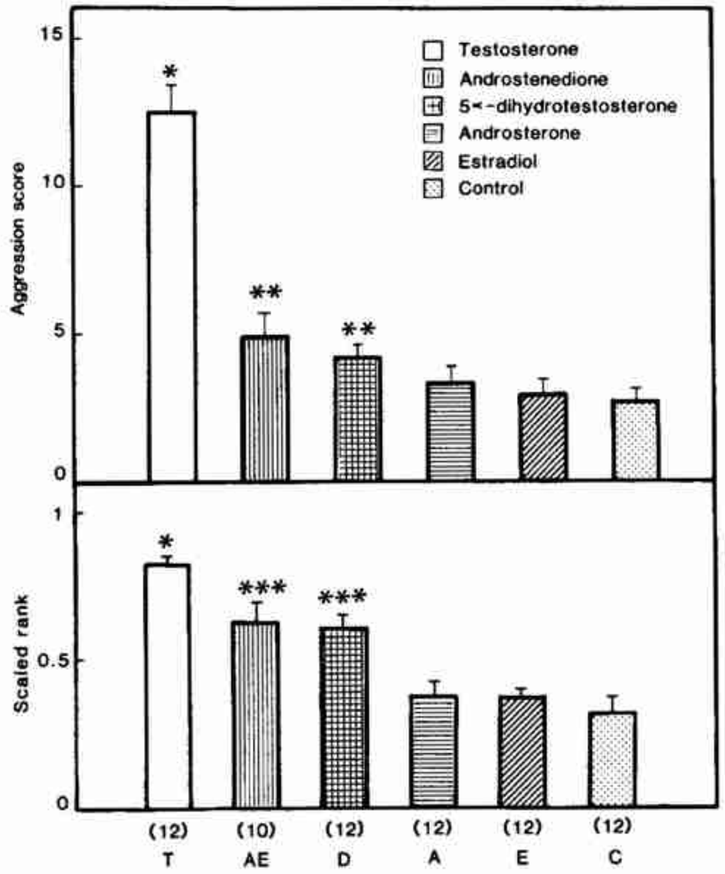

FIG. 1. Comparisons of the means of averaged aggression scores (upper) and scaled ranks (lower) of birds treated with different hormones in Experiment 1. Vertical lines indicate 1 SD; numbers of birds under each bar. *$P < 0.001$ compared with AE, D, A, E, and C birds; **$P = 0.004$ compared with A, E, and C birds; ***$P = 0.016$ compared with A, E, and C birds (two-tailed Mann–Whitney *U* tests).

Results

Individuals differed significantly in averaged aggression scores and dominance ranks depending on their treatment. T treatments produced significantly higher averaged aggression scores (AAS) and dominance ranks (DR) than other treatments ($n_1 = 12$, $n_2 = 58$: AAS, $U = 13$, $z = -5.22$, $P < 0.001$; DR, $U = 69$, $z = -4.348$, $P < 0.001$; Fig. 1). AE treatments did not have as great an effect as T treatments. Moreover, aggression scores and dominance ranks of birds with AE and D treatments did not differ ($n_1 = 10$, $n_2 = 12$: AAS, $U = 47$, $z = -0.86$, $P = 0.39$; DR, $U = 82$, $z = 0.99$, $P = 0.32$). Both produced significantly higher averaged aggression scores and dominance ranks than did A and E treatments and controls ($n_1 = 22$, $n_2 = 36$: AAS, $U = 218.5$, $z = -2.85$, $P = 0.004$; DR, $U = 259.5$, $z = -2.4$, $P = 0.016$). Aggression scores and dominance ranks of birds with A and E treatments were not different

TABLE 1
Average Testosterone Concentrations (ng/ml) for the Six Treatment Groups 6 Weeks after Implantation in Experiment 1 (mean ± SD, N in parentheses)

Treatment group	Sex	
	Male	Female
Testosterone (T)	11.95 ± 4.39[a,b] (3)	9.26 ± 2.89[a,b] (2)
Androstenedione (AE)	9.96 ± 0.25 (2)	— —
Androsterone (A)	0.54 ± 0.47 (2)	0.07 (1)
5 α-dihydrotestosterone (D)	0.28 ± 0.06 (5)	0.63 (1)
Estradiol (E)	0.19 ± 0.10 (2)	0.46 ± 0.21 (2)
Control (C)	0.35 ± 0.30 (2)	0.07 ± 0.01 (3)

[a] T and AE groups > A, D, and E groups (two-tailed Mann–Whitney U test, $U = 0$, $n_1 = 7$, $n_2 = 13$, $P < 0.002$).
[b] male and female (NS).

from control birds ($n_1 = 12$, $n_2 = 24$: AAS, $U = 150.5$, $z = 0.22$, $P = 0.826$; DR, $U = 147$, $z = 0.1$, $P = 0.920$). Thus testosterone had the greatest effect on aggression and dominance in this experiment.

There were no significant differences between stages of the experiment in the effects of any treatment on aggression and dominance.

Both T and AE treatments produced significantly higher plasma testosterone levels in both sexes than did the other treatments ($U = 0$, $n_1 = 7$, $n_2 = 13$, $P < 0.002$; Table 1). There was no significant difference between sexes either among birds with T treatments ($U = 3$, $n_1 = 2$, $n_2 = 3$, $P = 0.6$) or among controls ($U = 2$, $n_1 = 2$, $n_2 = 3$, $P = 0.4$).

Experiment 2

Because the results of Experiment 1 suggested that testosterone had a greater effect than did individual metabolites, a synergistic effect of 5α-DHT and estradiol was a possibility.

In this experiment, birds were implanted with both 5α-DHT and estradiol to imitate metabolized testosterone. If a combination of testosterone metabolites is needed to activate aggression and dominance, the effect of 5α-DHT together with estradiol should be similar to that of testosterone.

Methods

Twelve groups of five birds were used for this experiment during January through March 1986. Each bird in a group received one of five

treatments determined at random: T, D, E, a combination of 5α-DHT and estradiol (D + E), or an empty tube (C). The D + E birds received two 5-mm lengths of each hormone in Silastic tubes; others received 10-mm lengths of the hormone. After implantation, the birds were kept separately for a week in a room with a natural photoperiod before each group was released in a separate aviary (stage 1). Observations of aggression and dominance were obtained twice for each bird, as described above.

Twice, at 2-week intervals (stages 2 and 3), birds were regrouped in new aviaries with new opponents never met before and again observed. In each stage, each group consisted of five differently treated birds (T, D, E, D + E, and C).

Results

T and D + E treatments resulted in significantly higher averaged agression scores and dominance ranks than other treatments ($n_1 = 24$, $n_2 = 36$: AAS, $U = 88$, $z = -5.191$, $P < 0.001$; DR, $U = 186$, $z = -3.712$, $P < 0.001$; Fig. 2). Although birds with T treatments had higher averaged aggression scores and dominance ranks than D + E birds, the difference was not significant ($n_1 = 12$, $n_2 = 12$: AAS, $U = 45$, $z = -1.5$, $P = 0.14$; DR, $U = 55$, $z = -0.98$, $P = 0.326$). The D and E birds were more aggressive and had higher dominance ranks than the controls ($n_1 = 12$, $n_2 = 24$: AAS, $U = 266$, $z = 4.095$, $P < 0.001$; DR, $U = 259$, $z = 3.86$, $P < 0.001$).

Thus birds treated with T and D + E had significantly higher aggression scores and dominance ranks than the other birds. These two treatments had approximately equivalent effects in activating aggression and dominance in this experiment.

There were no significant differences between stages of the experiment in the effects of each treatment on aggression and dominance (Wilcoxon test).

Testosterone concentrations were significantly higher in birds with T treatments than in the other birds ($U = 0$, $n_1 = 12$, $n_2 = 16$, $P < 0.002$; Table 2). There were no differences between males and females in plasma testosterone levels in birds with T treatments ($U = 17$, $n_1 = 6$, $n_2 = 6$, $P = 0.469$). Birds with D + E and D treatments had significantly higher testosterone concentrations than did those with E treatments ($U = 8$, $n_1 = 6$, $n_2 = 10$, $P = 0.02$).

Estradiol levels were significantly higher in birds with E treatments than in others, with the exception of D + E birds ($U = 0$, $n_1 = 6$, $n_2 = 12$, $P < 0.002$). No significant differences were found between sexes in plasma estradiol levels in birds with E treatments ($U = 1$, $n_1 = 3$, $n_2 = 3$, $P = 0.1$).

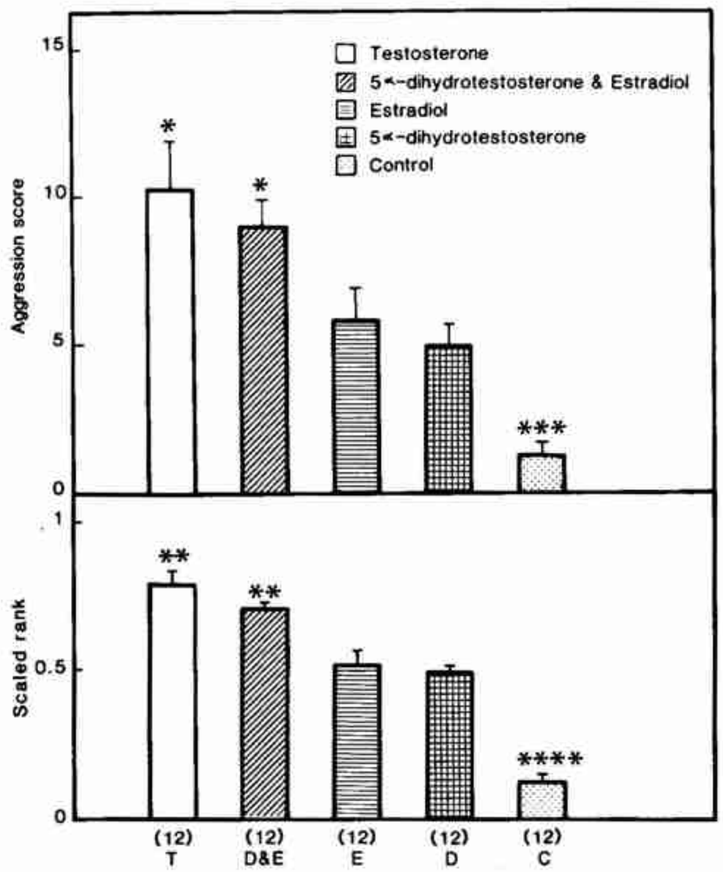

FIG. 2. Comparisons of the means of averaged aggression scores (upper) and scaled ranks (lower) of birds treated with different hormones in Experiment 2. Vertical lines indicate 1 SD; numbers of birds under each bar. $*P < 0.001$ compared with E, D, and C birds; $**P < 0.001$ compared with E, D, and C birds; $***P < 0.001$ compared with E and D birds; $****P < 0.001$ compared with E and D birds (two-tailed Mann–Whitney U tests).

Experiment 3

In Experiment 3, three different steroid blockers and synthetic inhibitors were compared: ATD (1,4,6-androstatrien-3,17-dione, Steraloids Inc.); progesterone (P; 4-pregnene-3,20-dione, Sigma Chemical Co.); and cyproterone acetate (CA; 17α-hydroxy-lα,2α-methylene-4,6-pregnadiene-3,20-dione-17-acetate, Schering). As all of the inhibitors were steroids, they were implanted in Silastic tubes like those used for the hormones.

Methods

Twelve groups of six differently treated birds were used in this experiment between March and May 1986. Four birds of each group, randomly chosen, were given subcutaneous implants of ATD, P, both ATD and P, and CA, respectively, and then kept in separate cages without visual contact in a room with a natural photoperiod for a week. Then these

TABLE 2
Average Hormone Concentrations for the Five Treatment Groups 6 Weeks after Implantation in Experiment 2

	Hormone concentration			
	Testosterone (ng/ml)		Estradiol (pg/ml)	
Treatment group	Male	Female	Male	Female
Testosterone (T)				
M	11.52[a,c]	11.84[a,c]	—	89.58
SD	1.08	1.41	—	65.60
N	6	6	—	2
5 α-dihydrotestosterone + estradiol (D + E)				
M	1.61[b]	1.67[b]	125.0	381.33
SD	1.22	0.88	—	—
N	2	2	1	1
5 α-dihydrotestosterone (D)				
M	1.57[b]	1.1[b]	32.82	38.23
SD	0.88	0.46	4.41	26.55
N	4	2	3	2
Estradiol (E)				
M	0.22	0.54	276.71[d,e]	417.66[d,e]
SD	0.16	0.43	16.32	39.92
N	3	3	3	3
Control (C)				
M	0.34	0.08	22.63	10.36
SD	0.21	0.02	6.04	1.84
N	3	3	3	2

Note. M, mean; SD, standard deviation; N = sample size. Two-tailed Mann–Whitney U tests: [a] T groups > D + E, D, and E groups ($U = 0$, $n_1 = 12$, $n_2 = 16$, $P < 0.002$). [b] D + E, D groups > E groups ($U = 8$, $n_1 = 6$, $n_2 = 10$, $P = 0.02$). [c] Male and female (NS). [d] E groups > T, D, and C groups ($U = 0$, $n_1 = 6$, $n_2 = 12$, $P < 0.002$). [e] Male and female (NS).

birds were given additional subcutaneous implants of testosterone (treatments AT, PT, APT, and CT, respectively). At this time, the fifth and sixth birds of each group were implanted with testosterone and empty tubes, respectively (treatments T and C). All 72 birds were then kept in individual cages for another week before the 12 groups of six differently treated birds were placed in separate aviaries (stage 1). Observations of aggression and dominance were obtained twice for each bird, as described above.

Twice, at 2-week intervals (stages 2 and 3), each bird was regrouped with new opponents in new aviaries. Each new grouping consisted of

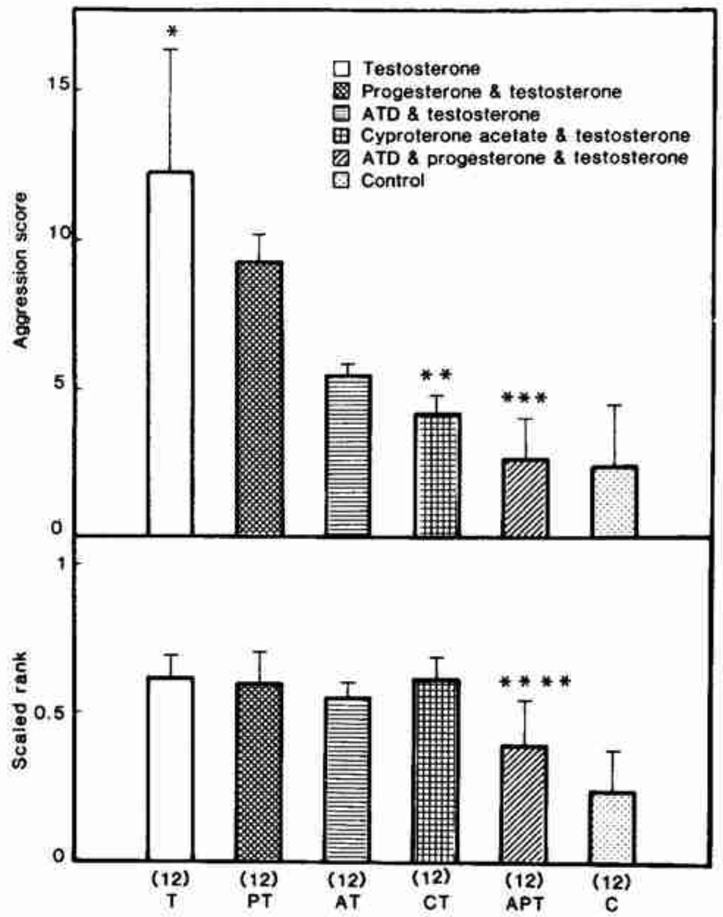

FIG. 3. Comparisons of the means of averaged aggression scores (upper) and scaled ranks (lower) of differently treated birds in Experiment 3. Vertical lines indicate 1 SD; numbers of birds under each bar. $*P < 0.001$ compared with PT and AT birds; $**P = 0.04$ compared with PT and AT birds; $***P = 0.04$ compared with PT and AT birds; $***P = 0.046$ compared with PT and AT birds (two-tailed Mann–Whitney U tests).

six differently treated birds (T, AT, PT, APT, CT, and C). In each stage, observations were repeated as described above.

Results

Comparison of treatments. Birds with APT treatments had significantly lower averaged agression scores and dominance ranks than birds with PT and AT treatments ($n_1 = 12$, $n_2 = 24$: AAS, $U = 205$, $z = 2.05$, $P = 0.04$; DR, $U = 203$, $z = 1.98$, $P = 0.046$; Fig 3). Although APT birds had higher averaged aggression scores and dominance ranks than controls, the differences were not significant ($n_1 = 12$, $n_2 = 12$: AAS, $U = 95$, $z = 1.32$, $P = 0.18$; DR, $U = 98.5$, $z = 1.53$, $P = 0.12$). Furthermore, APT birds had averaged aggression scores that did not differ from those of CT birds ($n_1 = 12$, $n_2 = 12$: AAS, $U = 71.5$, $z = 0.03$, $P = 0.976$), whereas CT birds had significantly higher dominance ranks than APT birds (DR, $U = 105.5$, $z = 1.94$, $P = 0.04$).

Despite receiving the same amount of testosterone as birds with T treatments, the PT and AT birds had significantly lower averaged aggression scores ($n_1 = 12$, $n_2 = 24$: AAS, $U = 34$, $z = -3.693$, $P < 0.001$), but dominance ranks were not different (DR, $U = 124$, $z = -0.671$, $P = 0.502$).

No significant differences were found in dominance ranks among PT, AT, and CT birds. However, CT birds had significantly lower averaged aggression scores than PT and AT birds ($U = 201$, $z = 1.91$, $n_1 = 12$, $n_2 = 24$, $P = 0.04$). Thus those agents predominantly blocking or inhibiting any one testosterone metabolite reduced aggression in these birds, but not dominance rankings.

Plasma hormone levels. No significant differences were found in testosterone concentration between sexes in T individuals ($U = 6$, $n_1 = 4$, $n_2 = 4$, $P = 0.343$; Table 3). Plasma levels of testosterone in AT, PT, and APT birds were significantly higher than in T individuals ($U = 23$, $n_1 = 8$, $n_2 = 13$, $P < 0.05$; Fig 4). Among birds treated with steroid blockers or synthetic inhibitors, plasma testosterone concentrations were in the following order: APT > AT > PT. APT birds had significantly higher plasma testosterone levels than AT and PT birds ($U = 0$, $n_1 = 2$, $n_2 = 4$, $P = 0.05$; $U = 1$, $n_1 = 2$, $n_2 = 7$, $P = 0.05$, respectively), but levels of plasma testosterone in AT birds were not significantly different from those in PT birds ($U = 8$, $n_1 = 4$, $n_2 = 7$, $P = 0.158$). Plasma testosterone levels in CT birds were not different from those in T individuals ($U = 23$, $n_1 = 6$, $n_2 = 8$, $P = 0.475$).

Estradiol concentrations did not differ in males and females with T treatments ($U = 8$, $n_1 = 4$ $n_2 = 4$, $P = 0.557$). Levels of plasma estradiol in the different groups were not significantly different from each other (T and C, $U = 14$, $n_1 = 5$, $n_2 = 8$, $P = 0.217$; T and AT, $U = 10$, $n_1 = 4$, $n_2 = 8$, $P = 0.184$; T and CT, $U = 23$, $n_1 = 7$, $n_2 = 8$, $P = 0.306$; PT and AT, $U = 7$, $n_1 = 4$, $n_2 = 8$, $P = 0.07$; and AT and C, $U = 10$, $n_1 = 4$, $n_2 = 5$, $P = 0.548$).

DISCUSSION

These experiments employed birds of both sexes captured during the winter when gonadal development and circulating gonadal hormones are at a minimum. Our results consistently failed to find any differences in behavior or circulating hormones of males and females within treatment groups, although sample sizes of each sex within treatments were small. Radioimmunoassays of free-living birds, both of closely related species and of white-throated sparrows in our population (Wingfield and Farner, 1984a,b; Archawaranon, 1987) have indicated that circulating gonadal hormones remain very low at least until migration begins in April. Our

TABLE 3
Average Hormone Concentrations for the Six Treatment Groups 4 Weeks after Implantation in Experiment 3

	Hormone concentration			
	Testosterone (ng/ml)		Estradiol (pg/ml)	
Treatment group	Male	Female	Male	Female
Testosterone (T)				
M	36.42[a,b]	31.62[a,b]	36.16[c]	37.62[c]
SD	8.27	10.0	21.44	16.68
N	4	4	4	4
ATD + testosterone (AT)				
M	61.75[d]	60.39[d]	22.41	10.82
SD	4.75	15.13	5.13	1.92
N	2	2	2	2
Progesterone + testosterone (PT)				
M	50.27[d]	53.42[d]	30.85	26.02
SD	15.27	25.50	6.86	8.79
N	4	3	5	3
ATD + progesterone + testosterone (APT)				
M	—	82.03[e]	18.74	28.22
SD	—	6.06	—	11.72
N	—	2	1	2
Cyproterone acetate + testosterone (CT)				
M	48.48[e]	25.35[e]	27.07	16.32
SD	20.76	—	7.48	—
N	5	1	6	1
Control (C)				
M	0.11	0.43	22.63	10.36
SD	0.03	0.3	6.04	1.84
N	4	3	3	2

Note. M, mean; SD, standard deviation; N = sample size. Two-tailed Mann–Whitney U tests: [a,c] Male and female (NS). [b] T groups < AT, PT, and APT groups ($U = 23$, $n_1 = 8$, $n_2 = 13$, $P < 0.05$). [e] APT > AT and PT groups ($U = 0$, $n_1 = 2$, $n_2 = 4$, $P = 0.05$ and $U = 1$, $n_1 = 2$, $n_2 = 7$, $P = 0.05$). [d] AT and PT (NS). [e] CT and T (NS).

Experiment 3, by extending into May, was possibly complicated by changes in endogenous gonadotropins or gonadal hormones. It is not clear, however, that seasonal changes explain the higher testosterone levels in this experiment in comparison to the previous two. That samples were taken 4 weeks after implantation of hormones, rather than 6 weeks as in the previous experiments, might also explain this difference. Note that the experimental birds had substantial doses of exogenous testosterone from March onward, presumably enough to inhibit seasonal increases in

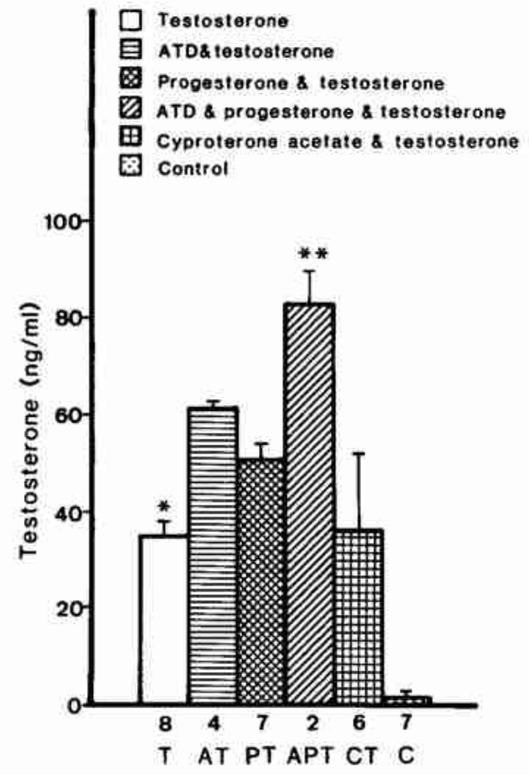

FIG. 4. Mean testosterone concentrations in birds with different treatments in Experiment 3. Vertical lines indicate 1 SD; numbers under each column are sample sizes. *$P < 0.05$ compared with AT, PT, and APT birds; **$P = 0.05$ compared with AT and PT birds (two-tailed Mann–Whitney U tests).

gonadotropin secretion. In addition, the results revealed no changes in behavior among stages of the experiment as the season progressed. Thus it seems likely that the behavioral differences observed in Experiment 3 resulted from the treatments, despite any effects of seasonal photostimulation.

The levels of circulating testosterone in our experimental birds approximated the maximal levels attained in males of related species during the breeding season (Wingfield and Farner, 1978a,b; Wingfield, 1984a,b). The behavior induced in our experiments is thus most appropriately compared with territoriality, the usual form of aggression in breeding males. Indeed, the highest-ranking birds in these studies often engaged in persistent chasing and supplantation of subordinate birds without regard to proximity to food, probably an expression of territorial behavior under conditions of captivity in which subordinates could not escape. On the other hand, the distinction between territoriality and dominance is not absolute (Waser and Wiley, 1979), so it remains to be seen whether or not the lower levels of circulating hormones during winter might also influence aggression and dominance.

Our results support the idea that aggression and dominance in white-throated sparrows are activated by the combined action of 5α-DHT and estradiol. The effect of these two metabolites of testosterone is synergistic, in the sense that their combination has a greater effect than either alone. The evidence for these conclusions was of three sorts.

First, testosterone which provided both androgenic and estrogenic metabolites consistently increased aggression and dominance in these experiments. In most studies of castrated male birds, exogenous testosterone restores aggressive behavior (Selinger and Bermant, 1967; Arnold, 1975; Hutchison, 1975; Adkins, 1977; and Harding *et al.*, 1983), although there is considerable variation among species in responsiveness to testosterone as opposed to androstenedione. For example, androstenedione is more effective than testosterone in restoring sexual behavior in pigeons and zebra finches (Pietras and Wenzel, 1974; Harding *et al.*, 1983, respectively), but testosterone is more effective in Japanese quail (Adkins, 1977) and chickens (Young and Rogers, 1978; Balthazart and Hirschberg, 1979). Studies of Japanese quail by Balthazart, Massa, and Negri-cesi (1979) found that aggressive behavior was correlated with individual differences in metabolism of testosterone to androstenedione in hyperstriatal or hypothalamic areas of the brain.

In white-throated sparrows, as indicated by Experiment 1, testosterone was more effective than androstenedione in activating aggression and dominance. Radioimmunoassays for testosterone in androstenedione-implanted birds in Experiment 1 showed that testosterone levels in these birds were nearly as high as in testosterone-implanted birds. Consequently, a limitation on the rate of conversion of androstenedione to testosterone is not likely to explain why androstenedione-implanted birds were less aggressive than testosterone-implanted birds.

Second, in Experiments 1 and 2, 5α-DHT or estradiol alone was less effective in eliciting aggression and dominance than testosterone alone or 5α-DHT in combination with estradiol. In other species as well, testosterone metabolites are important in activating behavior. In castrated male ring doves, aggressive courtship was induced by testosterone propionate or dihydrotestosterone propionate (Adkins-Regan, 1981). In castrated male zebra finches, hormone treatments that provided estrogenic metabolites induced more attacks on intruders than did any other treatment (Harding *et al.*, 1983). In male rats and mice, 5α-DHT and estradiol are ineffective if given alone, but male sexual behavior can be induced if 5α-DHT is administered in combination with low doses of estradiol (Baum and Vreeburg, 1973; Larsson, Sodersten, and Beyer, 1973, Larsson, Sodersten, Beyer, Morali, and Perez-Palacios, 1976; Feder, Naftolin, and Ryan, 1974; Luttge *et al.*, 1975; Wallis and Luttge, 1975). Finney and Erpino (1976) suggested that 5α-DHT and estradiol acted synergistically in inducing aggressive behavior in mice. In castrated mallard drakes as well, a combination of 5α-DHT and estradiol caused an increase in threatening, chest fighting, and chasing (Schmedemann and Hasse, 1984). In our experiments, the much reduced behavioral effects of 5α-DHT or estradiol when given alone differ from other studies of birds (Adkins-Regan, 1981; Ishii and Tsutsui, 1982; Harding *et al.*, 1983). These results suggest a synergism analogous to, although not so pronounced as, that in rodents.

The biochemical basis of this synergism remains unknown. It could be that 5α-DHT and estradiol act on different neuroanatomically separated mechanisms involved in behavioral activation (Balthazart, Schumacher, and Malacarne, 1985). Alternatively, 5α-DHT and estradiol could bind to the same receptor (Sheridan, 1983). It has also been suggested that estradiol could decrease the catabolism of 5α-DHT to diols which are relatively weak androgens. In this way, estradiol would permit the buildup in the brain of sufficient concentrations of 5α-DHT which would by themselves activate the behavior (Sodersten and Gustafsson, 1980). Finally, it is possible that 5α-DHT is aromatizable. Weniger and Zeis (1982) showed that it could be transformed into estradiol in the chicken ovary. If so, sufficient estradiol concentrations might activate behavior. Our data, however, provide no clear evidence that plasma levels of estradiol are higher in birds treated with 5α-DHT.

Third, the experiments with steroid blockers and synthetic inhibitors also suggest that metabolites activate aggression and dominance in white-throated sparrows. These blockers and inhibitors (in AT, PT, and APT birds) elevated testosterone concentrations in comparison to those in birds treated with testosterone alone. Presumably, in the presence of blockers or synthetic inhibitors, testosterone was not converted to metabolites within the target cells and, therefore, accumulated in the blood. APT birds (presumably no conversion of testosterone to 5α-DHT or estradiol) had significantly higher plasma testosterone levels than did AT (presumably no conversion of testosterone to estradiol) and PT birds (presumably no conversion of testosterone to 5α-DHT). It is also possible that these steroid blockers interfered with negative feedback loops through effects on hypothalamic response to circulating testosterone. Regardless of the mechanism involved, the high levels of plasma testosterone in the APT birds, in combination with their low levels of aggression and dominance, indicate that testosterone itself did not directly activate this behavior.

Since testosterone concentrations in CT birds were not different from those in testosterone-implanted birds, testosterone in these birds was presumably metabolized. The activity of the metabolites was apparently blocked by competition with cyproterone acetate. CT birds showed less aggression than birds treated with testosterone. Indeed, CT birds were

no more aggressive than APT birds and even less aggressive than either PT or AT birds.

In conclusion, these experiments provide evidence that aggression and dominance in white-throated sparrows are activated by an interaction of androgenic and estrogenic metabolites of testosterone. Testosterone is approximately as effective as 5α-DHT in combination with estradiol, whereas these metabolites are significantly less effective if given alone.

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