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Testosterone Dose-Dependency of Sexual and Nonsexual Behaviors in The Gonadotropin-Releasing Hormone Antagonist-Treated Male Rat

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The testosterone dose-dependency of several mating and nonmating behaviors was examined in the male rat, chemically castrated with a GnRH antagonist analog. Graded doses of testosterone enanthate (TE) were given to male rats to reinstate behaviors abolished by GnRH antagonist treatment. GnRH antagonist treatment alone markedly lowered serum LH, FSH and T concentrations and ventral prostate and testis weights. Open field behaviors were not significantly affected by GnRH antagonist treatment or castration. Scent-marking behavior was markedly suppressed by both castration and GnRH antagonist and restored by the lowest dose of TE (0.05 mg). All measures of male sexual behavior were impaired by GnRH antagonist treatment and castration and restored by the lowest dose of TE (0.05 mg). The doses of TE required to restore normal ventral prostate weights and testis weights were higher than those required to maintain scent marking and mating behaviors. No direct behavioral effects of the GnRH antagonist, other than those that can be explained by GnRH antagonist-induced suppression of testosterone were observed. The finding that sexual and nonsexual behaviors in the male rat have different testosterone requirements from those maintaining spermatogenesis and fertility may have significant implications for contraception.

Key words: Testosterone, sexual behaviors, nonsexual behaviors, scent-marking, open field behaviors, GnRH antagonist

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The influence of circulating testosterone (T) levels on the expression of several reproductive and nonreproductive sexually dimorphic behaviors in the adult rat is well established (Feder, 1984). Various components of male sexual behavior, such as mounting and ejaculation (Davidson, 1966; Damassa et al, 1977), as well as other behaviors such as aggression (Barfield et al, 1972) and territorial scent-marking (Price, 1975; Brown, 1978), are activated by T.

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The behavioral effects of T in the estrogen-treated male rat have been reported previously (Ewing et al, 1979). In this study, we employed a unique model system to study the androgen dose-dependency of several behaviors in the male rat. A potent, long-acting antagonist analog of GnRH was used to suppress the release of LH and thereby reduce T production to castrate levels. Graded replacement doses of testosterone enanthate (TE) were then given in an attempt to reinstate behaviors abolished by the GnRH antagonist treatment.

GnRH analogs have been studied extensively in recent years as potential male contraceptives. We recently demonstrated the ability of a GnRH anatagonist to suppress T production and spermatogenesis in male rats (Bhasin et al, 1988). In that study we showed that normal mating behavior could be maintained in antagonist-treated animals by a dose of TE that was lower than that required to maintain spermatogenesis. In the present study, we used the same hormone treatment regimen to address several unanswered issues. Primarily, we wished to examine whether mating behavior and a nonsexual androgen-dependent behavior (territorial scent-marking) are activated by the same dose of T. We also wished to investigate the possibility that the observed effects were generalized, perhaps due to changes in arousal, rather than specific to androgen-dependent behaviors. We therefore included a control behavior, activity in the open field, believed to be largely dependent upon organizational rather than activational effects of circulating steroids (Beatty, 1979). We also modified the mating behavior protocol to control better for differences in arousal level among experimental animals. Finally, we included two additional groups (castrate controls, and antagonist-treated castrates) to see whether antagonist treatment has direct effects other than those caused by a decrease in serum T levels.

Materials and Methods

Experimental Animals

Sixty-day-old sexually naive male Wistar rats were purchased from Charles-River (Wilmington, MA), housed three per cage, and maintained on a reversed light cycle (lights off at 10 A.M., on at 10 P.M.). Standard lab chow and water were available *ad libitum*, and body weights were recorded weekly. Prior to any hormone treatment or testing, the rats were handled daily for 2 weeks. During the following week, each rat was exposed to sexual experience with receptive females in three 20-minute sessions at 2-day intervals. After this habituation period, animals were screened for normal mating behavior, and those failing to ejaculate in at least two out of three trials were eliminated from the study. The remaining 48 animals were habituated to the other test situations (scent-marking and open-field), and then tested on all behaviors to obtain baseline values. Rats were then assigned to eight treatment groups of six animals each as follows, with all groups having similar average baseline test performances and similar average weights:

Group	Intact/ Castrate	Antagonist dose	Testosterone dose
I	Intact	ethylene glycol: saline	sesame oil
II	Intact	250 μg/day	sesame oil
III	Intact	250 μg/day	0.05 mg/day
IV	Intact	250 μg/day	0.15 mg/day
V	Intact	250 μg/day	0.50 mg/day
VI	Intact	250 μg/day	1.50 mg/day
VII	Castrate	ethylene glycol: saline	sesame oil
VIII	Castrate	250 μg/day	sesame oil

Hormone Treatment

The GnRH antagonist (Ac-D(2)Nal_1,4Cl-D-Phe_2,D-Trp_3,D-Arg_6, D-Ala_{10}-GnRH-HOAc) was provided by Marvin J. Karten, Ph.D., of the Contraceptive Development Branch of the National Institutes of Health. The antagonist, dissolved in a solution of ethylene glycol in normal saline, was administered by subcutaneous injections once a day at about 9 A.M.

Testosterone enanthate (17-[(1-oxoheptyl)oxy]-androst-4-en-3-one) was dissolved in sesame oil to concentrations of 0.5, 1.5, 5 and 15 mg/ml and stored at room temperature. Subcutaneous injections of 0.1 ml were given once a day immediately after the antagonist injections. All TE injections were delivered in the hindquarters to prevent contamination of the trunk blood collected at the time of decapitation.

The treatment period lasted 60 days. All rats were sacrificed by decapitation on day 61 at 178 days of age; organ weights were recorded and sera frozen for hormone measurements.

Behavior Testing

Each behavior was tested once before initiating treatment and at 2-week intervals during treatment for a total of five tests per behavior. All tests were performed at least 2 hours after hormone injections and after lights were turned off. Light sufficient to observe behavior was provided by three 15-watt red bulbs.

Mating behavior was tested using a modification of the method of Yahr and Gerling (Yahr and Gerling, 1978). Testing took place in cylindrical plexiglass arenas 45 cm in diameter and 37.5 cm tall. Males were placed individually in the arenas for a 5-minute habituation period, and a sexually receptive female was then introduced. (The females were previously ovariectomized and rendered receptive by priming with a 10-μg subcutaneous injection of estradiol benzoate 3 days prior to testing, and a 0.5-mg injection of progesterone on the morning of testing).

Observations began at the moment the female was introduced. After 10 minutes, the female was removed and another introduced unless the male had already ejaculated. This was done to control for variation in compatibility between male-female pairs. If a female failed to display lordosis when mounted, or attacked the male, she was considered unreceptive and replaced immediately.

If the male did not achieve intromission within 20 minutes of the introduction of the first female, the test was terminated. If intromission was achieved, the male was given 20 minutes after the intromission to achieve ejaculation. This "resetting the clock" method differs from the protocol we used in our earlier studies, where animals were given a total of 20 minutes to achieve ejaculation. This change was made to control for natural variations in arousal among the animals. Specifically, some males are initially slow to respond to the presence of the female, but once they initiate mating behavior, they are likely to respond as do other animals (Yahr and Gerling, 1978).

Information recorded during the session included mount frequency, intromission frequency, mount latency (latency to the first mount or intromission, whichever occurred first), intromission latency and ejaculation latency (the interval between first intromission and ejaculation). After ejaculation, the time to the next intromission (post-ejaculatory interval) was recorded, and the test terminated.

Scent-marking behavior was tested using a modification of the published method (Price, 1975; 1977). Testing was done in the mating behavior arenas, each equipped with clean bedding and a threaded steel rod (0.47 cm diameter) suspended 2.5 cm above the floor. Males were placed individually in the arenas for 5 minutes. The arenas and floor were wiped with an ethanal/water/acetic acid solution and both the bedding and the rods were changed before another rat was introduced to the arena. The rods were examined under ultraviolet light in order to enhance the visibility of the fluorescent urine. A ruler was placed alongside the rod, and the number of 0.5-cm segments marked by urine was recorded. Rods were washed in an Alconox detergent solution immediately after each determination.

Open-field behavior was measured in a circular arena 4 feet in diameter, segmented by white lines into 49 sectors of equal area, and enclosed by a 60-cm-high black plexiglass wall. Dimensions were those described previously (Latane, 1969). Males were introduced individually into the center of the arena and left for 5 minutes, during which time the following data were recorded: time taken by the rat to enter a sector adjacent to the wall, total number of sectors entered (Squares; a rat was considered to have entered a sector when he placed both front paws in that sector), number of rears (# Rears; lifting both front paws off the floor), number of grooming actions (# Grooms), and number of fecal boli deposited in the arena (# Boli). After each test, fecal boli were removed and the floor was washed with an ethanol/water/acetic acid solution (50:50:1) and thoroughly dried before introducing the next rat.

Physiologic Measurements

All rats were sacrificed by decapitation 16 to 24 hours after the last hormone injection. Trunk blood was collected and the sera separated, and each seminal vesicle, ventral prostate, and testis was removed and weighed.

Serum LH and FSH concentrations were determined by direct radioimmunoassay using reagents supplied by the National Pituitary Agency. Assay sensitivity was 0.05 ng/ml of RP-2 for rLH, and 0.75 ng/ml of RP-2 for rFSH. Intra-assay and interassay coefficients of variation were 3.1% and 11%, respectively, for rLH, and 2.4% and 12.1%, respectively, for rFSH.

Data Analysis

Statistical analyses were performed on a Digital VMS/VAX computer using the BMDP statistical package (Dixon, 1985). Physiologic parameters were analyzed using one-way analysis of variance (ANOVA). Open-field and scent-marking behaviors were analyzed using a one-way ANOVA with repeated measures, as were those mating behavior measures that were parametrically distributed (mount frequency and intromission frequency). The data were log transformed whenever variances were not equal (e.g. mount and intromission frequencies). If an overall significant effect was seen, *post hoc* testing was performed using t-matrix analyses. Measures of latency and post-ejaculatory intervals were analyzed using a non-parametric ANOVA (Kruskal-Wallace), and *post-hoc* testing was performed with the Wilcoxon rank sum (Mann-Whitney) test (only if an overall effect was observed).

Results

Physiologic Measurements

Weight gain in hormone-treated animals over the course of the study was not significantly different from controls, except in the group receiving the highest dose of TE (Group VI, 1.5 mg TE) (data not shown). This group showed a reduced rate of weight gain after the 11th week of treatment, but actual weight loss was not observed. Testis weights were markedly suppressed by GnRH antagonist treatment to less than 20% of control values, and increased in a dose-dependent fashion with graded increases in TE (Table 1). However, even the maximum dose of TE (1.5 mg) only increased testis weight to 88% of control values ($p < 0.05$). Ventral prostate weights were also profoundly suppressed by GnRH-antagonist treatment, and were identical to those seen in castrates (Table 1). Prostate weights did not regress as much in animals treated with GnRH antagonist plus 0.05 mg TE as in GnRH antagonist-treated animals without TE, while a 0.15-mg dose of TE maintained prostate weights at levels not significantly different from the intact controls. Superphysiologic doses of TE (0.50 and 1.50 mg) increased prostate weights above control levels (Table 1).

Serum LH was suppressed below the lower limit of detection (< 0 .05 ng/ml) for all GnRH antagonist

TABLE 1. Effects of GnRH Antagonist with and without Testosterone on Organ Weights and Reproductive Hormones*

Group	Testis (g)	Porstate (g)	LH (ng/ml)	FSH (ng/ml)
Control	3.62 ± 0.07	0.750 ± 0.10	0.32 ± 0.08	5.42 ± 0.46†
GnRH-Ant	0.65 ± 0.01†	0.050 ± 0.001†	0.06 ± 0.008†	1.34 ± 0.06†
GnRH-Ant+TE 0.05 mg	0.87 ± 0.03†	0.400 ± 0.020†	< 0.05†	1.78 ± 0.07†
GnRH-Ant+TE 0.15 mg	1.08 ± 0.08†	0.720 ± 0.030	< 0.05†	2.39 ± 0.09†
GnRH-Ant+TE 0.50 mg	2.51 ± 0.08†	1.150 ± 0.140†	< 0.05†	3.60 ± 0.13†
GnRH-Ant+TE 1.50 mg	3.18 ± 0.09†	1.040 ± 0.100†	< 0.05†	4.38 ± 0.24†
Castrated	----------	0.050 ± 0.005†	6.24 ± 0.81†	29.06 ± 4.43†
Castrate+GnRH-Ant	----------	0.050 ± 0.003†	< 0.05†	2.27 ± 0.19†

*Data are mean ± SEM, n = 6 per group; GnRH-Ant = GnRH antagonist.
†vs Control $p < 0.05$.

treatment groups, and serum FSH concentrations were markedly decreased in antagonist-treated rats (Table 1). As found in our previous study, the addition of graded doses of TE led to a dose-dependent increase in serum FSH (Bhasin et al, 1988).

Behavior Measurements

As expected, no consistent pattern of differences was found among the groups for the five open-field measures (Table 2). In contrast, scent-marking behavior was markedly suppressed by both castration and antagonist treatment, and this suppression was evident by the first posttreatment test (Table 3). Scent-marking behavior was maintained at the lowest dose of T (0.05 mg, Group III) and higher doses produced results not significantly different from the controls.

GnRH antagonist was very effective in suppressing all measures of male sexual behavior (Table 4). All measures of mating behavior tested in this experiment were maintained by the lowest dose of TE (0.05 mg). Both mount latency and intromission latency were significantly higher in antagonist-treated animals (Group II) than in controls, and this effect was seen by the second post-treatment test.

TABLE 2. Effects of GnRH Antagonist and Testosterone on Open Field Behavior in the Male Rat*

	Group							
Activity	Control	GnRH-Ant	GnRH-Ant +0.05 mg TE	GnRH-Ant +0.15 mg TE	GnRH-Ant +0.5 mg TE	GnRH-Ant +1.5 mg TE	Castrate	Castrate + GnRH-Ant
Squares	139.2 ± 6.4	158.7 ± 16.5	85.3 ± 28.5	138.4 ± 16.1	160.7 ± 17.0	161.4 ± 19.3	140.2 ± 41.7	207.4 ± 8.9
TTW	4.5 ± 0.9	4.2 ± 1.1	3.6 ± 0.5	4.7 ± 1.3	3.5 ± 0.3	4.2 ± 0.4	3.1 ± 0.5	3.0 ± 0.3
Rears	19.1 ± 3.5	14.2 ± 2.1	6.0 ± 3.0†	10.3 ± 2.3	14.0 ± 2.5	10.8 ± 1.8	11.3 ± 3.7	25.4 ± 2.9
Grooms	3.7 ± 0.6	3.2 ± 1.2	2.4 ± 0.9	3.1 ± 0.6	2.1 ± 0.4	1.7 ± 0.6	2.9 ± 0.5	3.0 ± 0.6
Boli	1.3 ± 0.6	2.6 ± 0.9	2.4 ± 0.7	2.9 ± 0.3	1.9 ± 0.3	1.1 ± 0.5	2.0 ± 0.7	1.2 ± 0.3
N =	6	6	4	6	6	6	5	5

*TTW: Time to wall in seconds; Square: Number of squares entered; Rears: Number of times animal raises front feet up; Grooms: Number of times animal grooms self; Boli: Number of fecal boli deposited in arena. These behaviors were studied before initiation of treatment and every 2 weeks (total of 5 times) during the 60-day treatment period. The values obtained over the four posttreatment tests were averaged for each animal. The mean values were than averaged across animals within each treatment group. Data are mean ± SEM, n = 6 per group. GnRH Ant = GnRH Antagonist.
† $p < 0.05$ vs Control.

TABLE 3. Effects of GnRH Antagonist and Testosterone on Scent Marking Behavior*

	Test 1	Test 2	Test 3	Test 4	Test 5
Control	31.8 ± 7.5	51.0 ± 6.9	4.10 ± 6.2	46.5 ± 5.2	40.3 ± 6.2
GnRH-Ant	50.7 ± 3.4	17.9 ± 5.5†	21.5 ± 6.6†	16.3 ± 6.2†	20.9 ± 4.3†
GnRH-Ant +TE, 0.05 mg	37.2 ± 8.5	28.4 ± 12.7	13.4 ± 9.1†	27.2 ± 9.6	32.4 ± 11.6
GnRH-Ant + TE, 0.15 mg	40.8 ± 6.0	39.3 ± 6.9	41.0 ± 2.9	40.4 ± 5.7	43.2 ± 4.5
GnRH-Ant + TE, 0.50 mg	32.8 ± 6.1	57.8 ± 5.0	51.9 ± 6.0	42.6 ± 11.4	53.3 ± 4.8
GnRH-Ant + TE, 1.50 mg	34.8 ± 9.5	54.2 ± 6.5	45.6 ± 8.9	31.6 ± 8.0	43.5 ± 9.1
Castrate	48.1 ± 4.5	0.50 ± 0.50†	7.3 ± 5.0†	7.9 ± 4.5†	7.5 ± 5.9†
Castrate + GnRH-Ant	55.1 ± 6.1	19.8 ± 10.2†	14.1 ± 6.0†	14.7 ± 5.9†	19.6 ± 8.2†

*Number of 1-cm units marked by urine were recorded for each animal on each of the 5 test days. Data are mean ± SEM, n = 6 per group; GnRH-Ant = GnRH antagonist.
†vs control $p < 0.05$.

No groups receiving TE had mount latency significantly dfferent from the controls or from other TE-treated groups in any tests. Ejaculation latency, intromission latency and number of animals achieving ejaculation were suppressed in Group II animals (Table 4), but the effect was not seen until the third posttreatment test. No significant antagonist effect was seen for mount frequency, or for the number of animals displaying mounting behavior. As with mount latency and intromission latency, the lowest dose of TE was effective in maintaining ejaculation latency and intromission frequency at control levels. No further change in these measures was observed with increasing doses of TE. All animals that ejaculated, regardless of group, had similar postejaculatory intervals (Table 4).

Castration alone (Group VII) and castration plus antagonist treatment (Group VIII) produced essentially the same effects as did antagonist treatment of intact animals (Group II) with two exceptions: first, the number of animals displaying mounting behavior was significantly suppressed in both castrate groups (VII and VIII) in the third and fourth posttreatment tests, whereas antagonist treatment alone did not produce significant changes in these measures. Secondly, although Groups II, VII and VIII all showed significant increases in mount latency, the increase was significantly less in the antagonist-alone group ($p < 0.05$). The data therefore do not support any direct effects of the antagonist analog on these behaviors other than those that can be explained by the antagonist-induced suppression of T.

Discussion

This study confirms earlier observations that 1) suppression of T production in intact animals with an antagonistic analog of GnRH produces a suppression of T-dependent behaviors similar to that reported for surgical castration (Davidson, 1966; Damassa et al, 1977); 2) accessory sex organ weights are also suppressed by antagonist treatment, and 3) these effects can be reversed by concomitant treatment with an appropriate dose of T. In the present study, these findings have been extended to nonsexual as well as sexual behavior, lending further support to the validity of the antagonist-treated animal as a model for investigating the steroid dose-dependencies of a variety of reproductive and nonreproductive behaviors. Such an approach may be preferable when surgical gonadectomy is undesirable, as was the case with our studies of concomitant effects on spermatogenesis (Bhasin et al, 1988). The fact that no treatment effects were seen for open-field behaviors indicates that the effects of antagonist treatment in adulthood are specific to behaviors that respond to the activational effects of androgens.

Our findings indicate that different androgen-dependent behaviors and other secondary sex characteristics are not equally sensitive to T. The dose of TE that maintained normal mating behavior (0.05 mg/day) was the same as that required to maintain normal scent-marking behavior. The 0.15-mg dose was required to maintain normal prostate and seminal vesicle weight. In contrast, testis weights were significantly below normal even at the highest dose of TE (1.50 mg). We have previously

shown that spermatogenesis and fertility in antagonist-treated rats are restored only at the highest 1.50-mg dose of TE (Bhasin, et al, 1988).

It is significant that relatively low doses of TE maintain sexual behavior, but addition of larger amounts do not appear to further enhance sexual behavior in the male rat. Even at doses of TE (0.50 and 1.50 mg) that produce superphysiologic serum T levels (Bhasin et al, 1988) and higher than normal accessory organ weights, mating behaviors are not significantly different from controls. This gives support to observations by other investigators that

TABLE 4. Effect of GnRH Antagonist and Testosterone on Mating Behavior in the Male Rat*

		Group						
Behavior	Control	GnRH-Ant	GnRH-Ant +0.05 mg TE	GnRH-Ant +0.15 mg TE	GnRH-Ant +0.5 mg TE	GnRH-Ant +1.5 mg TE	Castrate	Castrate + GnRH-Ant
Mount frequency	13.5 ± 4.5	6.1 ± 3.8[e]†	12.8 ± 9.4	12.8 ± 2.3	9.2 ± 2.5	9.7 ± 3.8	0.2 ± 0.2[e]†	3.7 ± 3.5[h]†
Intromission frequency	9.2 ± 1.1	3.5 ± 2.2[b]†	6.8 ± 0.5	10.5 ± 2.2	7.3 ± 1.2	11.8 ± 2.9	0[i]†	0.2 ± 0.2[g]†
Mount latency (sec)‡								
median	5.0	39.5[a]	12.0	4.0	5.0	3.0	> 1200.0[e]†	> 1200[h]†
range	2-48	1-> 1200	4-285	2-90	1-15	1-12	805- > 1200	115- > 1200
Intromission latency (sec)‡								
median	16.5	> 1200[b]†	17.0	29.5	41.0	12.0	> 1200[f]†	> 1200[g]†
range	5-48	25- > 1200	13-804	5-165	4-760	5-30	> 1200- > 1200	410- > 1200
Ejaculation latency (sec)								
median	989.5[d]	> 2400[c]†	491.5[d]	945.5[d]	593.0	272.5†	> 2400[f]†	> 2400[i]†
range	235- > 2400	1395- > 2400	323- > 2400	230- > 2400	135- 1018	165- 825	> 2400- > 2400	> 2400- > 2400
Postejaculatory interval (sec)								
median	437.0	> 1200[c]†	617.0	465.0	435.0	425.5	[i]†	[i]†
range	420- 635	> 1200	427- 670	356- > 1200	397- 586	356- 470	[i]†	[i]†

*Mating behavior was studied once before initiating treatment and every 2 weeks during the treatment period. The values shown represent data from the last posttreatment tests. The data for mount frequency and intromission frequency were parametrically distributed and are expressed as Mean ± SEM, n = 6 per group. Otherwise, the median and range are shown. For calculation of means, only responding animals were included. GnRH Ant = GnRH Antagonist.

†Significantly different from Control $p < 0.05$.

a. Two animals in this group did not mount.

b. Four animals inthis group did not intromit.

c. Five animals in this group did not ejaculate.

d. One animal in this group did not ejaculate.

e. Four animals in this group did not mount.

f. None of the animals in this group had intromission or ejaculation.

g. Only one animal in this group had intromission.

h. Only two animals in this group mounted.

i. None of the animals ejacualted.

‡Latency to first mount or intromission, whichever occurred first.

maintenance of male sexual behavior requires relatively low levels of androgen (Damassa et al, 1977; Feder, 1984). These data confirm and extend the work of Damassa et al, 1977 and Davidson et al, 1978, who reported similar relationships between androgen levels, sexual behavior and sexual reflexes in the male rat.

It is also significant that many GnRH antagonist-treated animals continued to mount and even in surgically castrated rats, mounting response was very slow in responding to castration. Although mounting is a sexually dimorphic behavior, and is androgen-dependent, it is conceivable that the time-course of this response to hormonal changes is much slower than that for other behaviors. These data are consistent with observations by other investigators that some males, including humans, maintain certain aspects of sexual behavior after withdrawal of T (Davidson et al, 1982). There is also some evidence that hormones other than steroids can affect the expression of sexual behaviors. It has been reported that lordosis behavior in estrone-primed ovariectomized female rats is enhanced by administration of GnRH, whereas the effect is not observed with estrone alone, or with estrone in combination with LH, FSH or TSH (Moss et al, 1975). Our data do not demonstrate any direct effects of the GnRH antagonist on mating behavior other than those mediated by a decrease in serum T.

In summary, we have demonstrated the appropriateness of the antagonist-treated rat as a model for studying the dose-dependency of both sexual and nonsexual androgen-dependent behaviors. We have also shown that the dose of T required to support normal sexual behavior is lower than that required to support testis weight and spermatogenesis. Furthermore, various androgen-dependent behaviors show different time-courses in response to alteration of hormone levels.

Whether these findings can be extrapolated to human behavior is an unanswered question. Human sexual behavior has been shown to be responsive to activational effects of steroid hormones (Bancroft, 1980; Sanders and Bancroft, 1982; Kwan et al, 1983) and there is some evidence for androgen effects on nonreproductive behavior as well (Komnenich et al, 1978; Houser, 1979). However, the dose-dependent relationships of those behaviors have not been well elucidated. The finding that reproductive and nonreproductive sexually dimorphic behaviors in the male rat have different testosterone requirements from those for maintaining spermatogenesis and fertility may have significant implications, such as in contraception.

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