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Monoaminergic Mediation of Masculine and Feminine Copulatory Behavior in Female Rats

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RODRIGUEZ-SIERRA, J. F., A. N. NAGGAR AND B. R. KOMISARUK. *Monoaminergic mediation of masculine and feminine copulatory behavior in female rats.* PHARMAC. BIOCHEM. BEHAV. 5(4) 457–463, 1976. – Ovariectomized rats treated with testosterone propionate (TP; 100 μg/kg × 6 days) and para-chlorophenylalanine (pCPA; 100 mg/kg × 3 days), a serotonin synthesis inhibitor, showed more masculine copulatory behavior (including the ejaculatory pattern) than did females receiving either TP or pCPA alone. The facilitatory effect of pCPA on the masculine copulatory behavior in females was not potentiated by pargyline (50 or 100 mg/kg), a monoamine oxidase inhibitor; instead, pargyline antagonized the effect of pCPA. Apomorphine (100 μg/kg), a dopamine receptor stimulant, did not increase masculine copulatory behavior in TP treated females. A dopaminergic facilitatory effect was therefore not demonstrated. These results suggest a serotonin-mediated inhibition of masculine copulatory behavior in female rats. When feminine copulatory behavior was tested, females receiving TP plus pCPA plus pargyline, TP plus pargyline, or TP plus apomorphine displayed lordosis in response to mounting by male rats. Lordosis did not occur after administration of TP, pCPA, or pargyline, individually or in any other combination. The responses in pargyline groups are consistent with the hypothesis of a noradrenergic facilitatory system for lordotic behavior. The responses in the apomorphine group are discussed in terms of a possible role for low level dopaminergic stimulation in facilitating lordosis.

Pargyline pCPA Apomorphine Masculine copulatory behavior Lordosis Monoamines

MASCULINE copulatory behavior (MCB: clasping, mounting, intromitting and/or ejaculating) in male rats is apparently under the inhibitory control of serotonergic neurons. When treated with para-chlorophenylalanine (pCPA), which inhibits serotonin synthesis [15], male rats show increases in MCB [18, 23, 28, 29, 30, 32]. Castration seems to prevent the pCPA induced enhancement, and the pCPA enhancement effect can be restored by testosterone [6, 11, 34]. The effects of pCPA on MCB in males may be blocked by administration of the serotonin (5-Hydroxytryptamine, 5-HT) precursor, 5-Hydroxytryptophan [29,32].

Pargyline, an inhibitor of monoamine oxidase [12], reduces MCB in males when given along [18,33]. However, pargyline potentiates the increase in MCB in males induced by pCPA [23,26]. This suggests that an increase in catecholamines can also facilitate the homotypical copulatory behavior of males. Support for this view comes from studies which demonstrate an increase in MCB of male rats after treatment with l-DOPA, the catecholamine precursor [19,35]. Dopamine has been implicated as the critical catecholamine because dopamine receptor stimulation by apomorphine enhances, while dopamine receptor blockade by haloperidol or pimozide suppresses the display of MCB in males [12, 19, 35].

Genetic female rats can, under appropriate conditions [3, 7, 8, 16], show the motor components of clasping, mounting, intromission, and ejaculation, indicating that the neural mechanisms underlying these behavior patterns exist in females. However, because the occurrence of these patterns in the normal female is rare, relative to the occurrence in male rats, the term masculine copulatory behavior has been applied [3, 5, 8]. In the present study, we analyze whether the monoamines influence MCB (i.e., heterotypical copulatory behavior) in female rats. Sheard [28] observed that female rats treated with pCPA showed increased mounting. Singer [31] also reported increases in mounting, but not in intromissions in females given pCPA after a nonreported amount of testosterone. We attempted to confirm these findings and also to test the role of the catecholamines in the control of MCB in female rats. Previous studies have indicated that 5-HT and dopamine systems inhibit the display of lordosis [8, 9, 19, 20, 21, 36]. In the present study we also analyzed the role of these monoamines in the control of the homotypical copulatory behavior of the same females.

METHOD

Animals

Sprague-Dawley female rats (200–250 g) were purchased from Charles River Co., North Wilmington, Mass. They were housed individually in a temperature controlled room, at about 22°C, with lights off from 10:00–20:00.

TABLE 1

SCHEDULE OF INJECTIONS STARTING IMMEDIATELY AFTER THE PRETEST. DAYS 1-6 INJECTIONS WERE GIVEN 2-3 HR INTO THE DARK PHASE OF THE CYCLE

		Injection Schedule Day No.				Time Prior to Test Day 7	
GROUPS	N	1–3	4	5	6	3 hr	15 min
O + S	10	O	O + S	O + S	O + S	S	S
Parg (100)	10	O	O + S	O + S	O + S	Parg	S
Parg (50)	10	O	O + S	O + S	O + S	Parg	S
pCPA + Parg (100)	10	O	O + pCPA	O + pCPA	O + pCPA	Parg	S
pCPA + Parg (50)	10	O	O + pCPA	O + pCPA	O + pCPA	Parg	S
TP + pCPA + Parg (100)	10	TP	TP + pCPA	TP + pCPA	TP + pCPA	Parg	S
TP + pCPA + Parg (50)	10	TP	TP + pCPA	TP + pCPA	TP + pCPA	Parg	S
TP + Parg (100)	10	TP	TP + S	TP + S	TP + S	Parg	S
TP + Parg (50)	10	TP	TP + S	TP + S	TP + S	Parg	S
TP + APO	10	TP	TP + S	TP + S	TP + S	S	APO
pCPA	10	O	O + pCPA	O + pCPA	O + pCPA	S	S
TP	10	TP	TP + S	TP + S	TP + S	S	S
TP + pCPA	10	TP	TP + pCPA	TP + pCPA	TP + pCPA	S	S

S = Saline; O = Sesame oil; TP = Testosterone propionate (100 ug/kg); Parg = Pargyline (50 mg/kg or 100 mg/kg); APO = Apomorphine HCl (100 ug/kg, dose expressed as salt); pCPA = para-Chlorophenylalanine methyl ester HCl (100 mg/kg, dose expressed as salt).

Food and water were available ad lib. Stimulus females were ovariectomized adults of the Sprague-Dawley strain, injected 2 days before testing with 10 μg estradiol benzoate and 4 hr before testing with 1 mg progesterone. All were sexually receptive to a male. Stimulus males were adult vigorous copulators of the same strain.

Procedure

Animals were ovariectomized under Equithesin anesthesia (3 cc/kg) and allowed one week for recovery. Non-hormonal pretests were then begun.

Testing for masculine copulatory behavior. Testing was performed in dimly lit rooms in the middle of the dark phase of the day–night cycle in circular Plexiglas arenas (55.9 cm in dia., 40.9 cm high). All females received a pretest for MCB.

After the experimental female was arena-adapted for 10 min, a sexually receptive stimulus female was introduced. The pair remained together for 10 min during which the following behavioral measures were recorded (a) number of clasps (experimental female mounts stimulus female from behind and clasps the flanks, but does not exhibit pelvic thrusting). (b) number of mounts (clasps with pelvic thrusting). (c) number of intromission patterns (mounts with springing dismount). (d) number of fights initiated by the experimental female).

Occasionally, a stimulus female mounted the experimental female; however the test female never showed lordosis in response to this stimulation.

Testing for feminine copulatory behavior. Immediately following this pretest the experimental female was placed in a similar arena containing a male and removed after being mounted 10 times, during which the following behavioral measures were recorded: (a) occurrence of lordosis; Lordosis Quotients were then computed (LQ; lordosis/mount × 100); (b) occurrence of hopping or darting; (c) occurrence of ear wiggling; and, (d) number of rejections (female kicks male or rolls onto her back when male attempts to mount). One experimental female showed lordosis to the male during the pretest and was excluded from the experiment.

Following these pretests, about half of the animals received daily injections of testosterone propionate (TP, 100 μg/kg) for 6 consecutive days, while the other animals received daily injections of the sesame oil vehicle. Each of these subgroups was further subdivided as shown in Table 1. Saline, pargyline, apomorphine and pCPA were administered intraperitoneally, while the TP and oil were administered subcutaneously. Two hr, 50 min after pargyline injection; and 5 min after apomorphine injection, the 10 min arena-adaptation was begun. Testing the followed the same procedure as the pretreatment tests. Thus, testing began 3 hr after pargyline and 15 min after apomorphine. Weighted scores were computed by subtracting the pretest individual score from the posttreatment scores.

RESULTS

Masculine Copulatory Behavior

The results from the pretest and test for heterotypical copulatory behavior (i.e., MCB) are shown in Tables 2–5. Only 7 of the 13 groups are shown individually; the other six groups differed minimally from pretest to test or from corresponding oil/TP controls, and are combined as all other groups.

On the pretest, scores of MCB showed no significant differences among groups on one-way analysis of variance. On the test scores, one-way analysis of variance showed significant differences among groups in the number of intromission patterns ($F = 7.28$, $p<0.001$), number of mounts ($F = 5.28$, $p<0.01$), number of clasps ($F = 7.28$,

TABLE 2

SUMMARY OF INTROMISSION PATTERN DATA FROM THE MCB TEST

Groups	No. of Responders		Mean No. of Intromission patterns (± SEM)	
	Pretest	Test	Pretest	Test*
TP + pCPA	0	6†	0	5.8 ± 2.2‡
pCPA	0	1	0	0.8 ± 0.8
TP	0	3	0	0.8 ± 0.5
O + S	0	1	0	0.1 ± 0.1
TP + APO	0	1	0	0.1 ± 0.1
TP + pCPA + Parg (50)	0	1	0	0.2 ± 0.2
TP + pCPA + Parg (100)	0	0	0	0
All Other groups§	<0.5	0	<0.1	0

*Analysis of variance of all 13 groups, F = 5.57, $p<0.001$.
†Compared to pretest, McNemar's test, $p<0.02$.
‡TP + pCPA group significantly different from all other groups (Newman-Keuls, $p<0.01$).
§Average no. responders and incidents of the intromission pattern.

$p<0.001$), and number of fights (F = 2.68, $p<0.005$). Using the weighted scores, one-way analysis of variance also showed significant differences in the number of intromission patterns (F = 5.26, $p<0.001$), number of mounts (F = 2.82, $p<0.005$), number of clasps (F = 6.41, $p<0.001$), and number of fights (F = 2.39, $p<0.05$).

Because of variability between groups during the pretest and the low number of responses during the test, weighted scores were calculated. The results of the analysis of variance and the post-hoc Newman-Keuls tests on the weighted scores were in complete agreement with the results of these tests on the raw scores.

Intromission pattern. The number of animals showing the intromission pattern when treated with TP + pCPA increased significantly, from 0–6 (see Table 2). Group TP + pCPA also showed a greater number of intromission patterns than all other groups (Table 2). Post-hoc Newman-Keuls tests on either the raw or weighed scores showed that the TP + pCPA group was significantly different from all other groups ($p<0.01$). Furthermore, three of the TP + pCPA animals exhibited the ejaculatory pattern: two within one min after the end of the test and the other when retested an hour after the test. Post-ejaculatory intervals, noted for two of the three animals, were 4.5 and 3.5 min.

Mounting. After treatment with TP + pCPA or pCPA, the proportion of rats showing mounts increased significantly from pretest levels (Table 3). The number of pCPA animals mounting was also significantly greater than oil treated animals. Newman-Keuls tests showed that only the TP + pCPA group differed significantly from all other groups ($p<0.05$).

Although 6 or 10 pCPA animals showed mounting, a significant increase from the pretest, the mount frequency was not high enough to differ from other control groups. The TP + APO group's raw or weighted scores did not differ significantly from the TP control group.

Clasping. The number of females showing clasps in the TP + pCPA or pCPA groups was significantly greater than their respective pretest scores (Table 4). The TP + pCPA and pCPA groups also differed significantly from all other groups, but not from each other (Newman-Keuls, $p<0.05$).

Fighting. TP + pCPA, pCPA, TP, and TP + APO groups all increased the mean number of fights compared to pretest levels, the facilitation in the TP + pCPA group being significantly greater than in the other groups (Table 5).

The TP + pCPA group showed a higher absolute proportion of fights, but due to a higher baseline, this was not significant.

Feminine Copulatory Behavior

Females in only four of the groups displayed lordosis, and their LQ's did not differ significantly from each other (F = 0.42, $p>0.05$; Table 6). The number of responders in the TP + APO or TP + pCPA + Parg (100) groups was significantly higher than for the TP control (Fisher's exact probability = 0.04). The level of rejection behavior directed to the stimulus male was not significantly different among the groups (F = 1.16, $p>0.05$).

DISCUSSION

Masculine Copulatory Behavior

MCB was enhanced when female rats were treated with TP + pCPA. This is clearly seen in the higher incidence of intromission patterns and the occurrence of the ejaculatory pattern in 3 of the animals in this group. This finding suggests that these patterns are under the inhibitory control of a serotonergic system in females as well as in males. We found that pCPA alone also increased the proportion of females mounting. This is different from findings by others with male rats in which testosterone is necessary for pCPA to increase any of the measures of MCB [34].

Until recently, it had been assumed that female rats lack the potential to exhibit the ejaculatory pattern [2], unless they are treated with androgens perinatally [38]. However, recent observations have shown that perinatally untreated female rats implanted subcutaneously with testosterone and subjected to peripheral electric shock in adulthood, can be induced to show the ejaculatory pattern [16]. Subcutaneous implants of estrogen for 7 months have also resulted in the occurrence of the ejaculatory pattern in ovariectomized rats [8]. Animals not showing the ejaculatory pattern after 7 months of estrogen were then

TABLE 3

SUMMARY OF MOUNTING DATA FROM THE MCB TEST

Groups	No. of Responders Pretest	No. of Responders Test	Mean No. of Mounts (± SEM) Pretest	Mean No. of Mounts (± SEM) Test*
TP + pCPA	2	8†	1.4 ± 1.0	4.9 ± 1.7§
pCPA	0	6†‡	0	2.1 ± 0.6
TP	1	4	1.5 ± 1.5	1.5 ± 0.7
O + S	0	1	0	0.2 ± 0.2
TP + APO	0	3	0	0.4 ± 0.2
TP + pCPA + Parg (50)	1	2	0.1 ± 0.1	0.1 ± 0.9
TP + pCPA + Parg (100)	2	0	0.6 ± 0.4	0
All other groups‖	<1	<1	<0.5	<0.5

*Analysis of variance of all 13 groups, $F = 5.28$, $p<0.01$.
†Compared to pretest, McNemar's test, $p<0.02$.
‡Compared to saline control, Fisher's probability test, $p = 0.02$.
§TP + pCPA groups significantly different from all other groups (Newman-Keuls, $p<0.01$).
‖Average no. responders and incidents of mounting.

TABLE 4

SUMMARY OF CLASP DATA FROM THE MCB TEST

Groups	No. of Responders Pretest	No. of Responders Test	Mean No. of Clasps (± SEM) Pretest	Mean No. of Clasps (± SEM) Test*
TP + pCPA	5	10†‡	1.9 ± 1.1	5.9 ± 1.8§
pCPA	1	7†	0.2 ± 0.2	4.9 ± 1.5§
TP	5	5	1.3 ± 0.6	2.1 ± 0.8
O + S	2	3	0.5 ± 0.4	0.5 ± 0.3
TP + APO	1	4	0.1 ± 0.1	1.0 ± 0.4
TP + pCPA + Parg (50)	4	3	1.1 ± 0.5	0.8 ± 0.5
TP + pCPA +Parg (100)	3	0	1.1 ± 0.6	0
All other groups‖	<4	<1	<1	<1

*Analysis of variance of all 13 groups, $F = 7.28$, $p<0.001$.
†Compared to pretest, McNemar's test, $p<0.01$.
‡Compared to TP control, Fisher's probability test, $p<0.001$.
§TP + pCPA and pCPA groups significantly different from all other groups, but don't differ from each other (Newman-Keuls, $p<0.05$).
‖Average no. responders and incidents of clasping.

TABLE 5

SUMMARY OF AGONISTIC BEHAVIOR DATA FROM THE MCB TEST

Groups	No. of Responders Pretest	No. of Responders Test	Mean No. of Fights (± SEM) Pretest	Mean No. of Fights (± SEM) Test*
TP + pCPA	3	6	0.8 ± 0.4	5.9 ± 2.8†
pCPA	1	4	0.1 ± 0.1	3.2 ± 1.7†
TP	1	3	1.0 ± 1.0	2.9 ± 1.9†
O + S	2	3	0.9 ± 0.8	0.8 ± 0.5
TP + APO	0	4‡	0	2.0 ± 1.4†
TP + pCPA + Parg (50)	1	1	1.0 ± 0.1	0.2 ± 0.2
TP + pCPA + Parg (100)	1	0	0.1 ± 0.1	0
All other groups§	<1	<1	<0.5	<0.5

*Analysis of variance of all 13 groups, $F = 2.68$, $p<0.005$.
†Newman-Keuls test shows that TP + pCPA group did not differ from the pCPA, TP or TP + APO groups, but was significantly different from all other groups ($p<0.05$).
‡Compared to pretest, McNemar's test, $p<0.05$.
§Average no. responders and incidents of fighting.

TABLE 6

FEMININE COPULATORY BEHAVIOR

Groups	Mean LQ*	No. Responders	Responders Mean LQ†	No. Animals Hop	Dart	Ear Wiggle
TP + APO	12 ± 5.5	4/10‡	30 ± 7.1	1	1	0
TP + Parg (50)	15 ± 10.0	3/10	50 ± 25.2	2	2	1
TP + pCPA + Parg (50)	18 ± 9.6	3/10	60 ± 11.5	4	0	0
TP + pCPA + Parg (100)	26 ± 11.1	4/10‡	65 ± 8.7	4	5	3
All other groups	0	0/90	—	0	0	0

*Group mean LQ ± SEM.
†Responders mean LQ ± SEM.
‡Compared to TP control, Fishers's exact probability = 0.04.

injected with pCPA for 4 days, at which point they all achieved the ejaculatory pattern. These findings demonstrate that the neural organization for the ejaculatory pattern is present in the female as well as in the male rat.

In preliminary studies we have observed an increase in the likelihood of occurrence of the intromission pattern in adrenalectomized-ovariectomized females treated with TP – pCPA. The ejaculatory pattern, however, did not occur in these rats. Absence of the ejaculatory pattern has been reported in adrenalectomized castrated male rats treated with TP + pCPA [19]. This may be of interest in view of the recent demonstration of the involvement of the adrenals in the ejaculatory pattern induced by estrogen in males [13]. Although adrenal androgens were invoked in that study as possible mediators, both in [19] and our pilot studies the animals received exogenous androgens. It is possible that other factors are responsible for the absence of the ejaculatory pattern after adrenalectomy.

Pargyline, at a dose which is effective in enhancing the TP + pCPA increase in MCB in males [10,31], did not enhance MCB in our females. Rather than potentiating the TP + pCPA effect on MCB, pargyline suppressed it. This may have been due to a general debilitating effect of pargyline. Our pargyline treated animals in the high dose group looked sedated and slightly cataleptic, they squeaked when touched, and their fur was wet from urination. When we then reduced the dose by half, our animals were not evidently debilitated, but the pargyline still blocked rather than facilitating MCB.

Another contrasting result was obtained with apomorphine. A dose which is effective in increasing MCB in TP primed males [12, 19, 35], failed to do so in our TP primed females.

There are several ways in which these differences can be accounted for. For the pargyline groups, remaining stores of 5-HT could have been elevated, producing a suppression of MCB. For apomorphine, a higher dose is unlikely to induce MCB, since in preliminary observations, increasing the apomorphine dose led to stereotypic behavior which competes with MCB, as measured by mounts per minute [19]. It is possible, however, that an enhancement of heterotypical patterns could have occurred if apomorphine were given to females primed with a higher dose of TP. Since drug metabolism and utilization can differ between the sexes [14], it is possible that this is true of the drugs used in our study. Still another possible explanation is that male and female rats differ in involvement of catecholamines in MCB. The resolution of this problem must await further study.

Agonistic behavior showed a parallel with those of MCB. Those groups which exhibited enhanced MCB also showed increases in the number of fights. The literature linking masculine behavior and agonistic behavior is extensive [24]. Apomorphine, however, increased the number of animals engaging in fights without affecting the MCB of females.

Feminine Copulatory Behavior

When the animals were tested for feminine copulatory behavior, it was found that the TP + APO, TP + Parg (50), TP + pCPA + Parg (50), and TP + pCPA + Parg (100), but not the TP + Parg (100) group, exhibited lordosis. This exception is likely to be due to strong debilitation observed in this group. In TP primed females, pCPA did not facilitate lordosis responding when administered by itself; combined treatment with pargyline was necessary. Perhaps pCPA is less effective with TP than with estrogen, and an increase in catecholamine tone increases this effect still more. Another possibility is that pCPA acts differentially when given chronically versus acutely, since previous studies have shown a difference in lordosis responding based on whether pCPA is administered chronically [27], as in this study, or acutely [9,21].

Pargyline and apomorphine have been shown to block lordosis responding in female rats [10,20]. However, those findings do not necessarily contradict the present findings. In those experiments in which inhibition of lordosis was found, the animals had been pretreated with estrogen and progesterone and were already responding maximally. Thus, possible facilitatory effects could not appear. In our study, females maintained on TP were not showing lordosis, so a facilitation by the drugs could be seen. It is likely that the aromatization of testosterone to estrogen [4,22] produced subthreshold estrogenic action, allowing the facilitatory effects of pargyline and apomorphine to appear.

There is evidence that norepinephrine facilitates lordosis [10, 37, 38]. The facilitation of lordosis in our pargyline groups is consistent with a noradrenergic facilitatory system. However, the facilitation of lordosis in our apomorphine group contrasts with findings by others of an inhibitory role for dopamine on lordosis [10,20]. A possible explanation for the latter findings is that high level dopamine receptor stimulation produces stereotypy [26]

which interferes with lordosis, a behavior pattern which in rats requires immobilization [17]. On the other hand, low level dopamine receptor stimulation, as in our study, could enhance the transmission of sensory information [1,36], and thus previously ineffective stimuli would become adequate to elicit lordosis. It is also possible that the facilitatory effect of apomorphine on lordosis is mediated by the adrenals. This appears unlikely, however, since in a preliminary study adrenalectomized-ovariectomized rats treated with TP + APO displayed lordosis.

In conclusion, 5-HT synthesis inhibition by pCPA enhances MCB in female rats as well as in male rats. This is a potent effect, for even the ejaculatory pattern occurred in some of our females. The role of catecholamines in MCB in females remains unclear.

The present findings suggest some similarities and some possible differences between the sexes. pCPA can enhance mounting behavior in females even without TP priming, but TP is necessary for this effect in males. Dopaminergic stimulation seems to be ineffective on MCB in females while it enhances MCB in males. Our results also indicate a dopaminergic role in the facilitation of lordosis.

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REFERENCES

1. Antelman, S. M., H. Szechtman, P. Chin and A. E. Fisher. Tail pinch induced eating, gnawing, and licking behavior in rats: Dependence in the nigrostriatal dopamine system. *Brain Res.*, in press.
2. Beach, F. A. Hormonal factors controlling the differentiation, development and display of copulatory behavior in the ramstergig and related species. In: *The Biopsychology of Development*, edited by E. Tobach, L. R. Aronson and E. Shaw. New York: Academic Press, 1971.
3. Beach, F. A. and P. Rasquin. Masculine copulatory behavior in intact and castrated female rats. *Endocrinology* **31**: 393–409, 1942.
4. Beyer, C., N. Vidal and A. Mijares. Probable role of aromatization in the induction of estrous behavior by androgens in the ovariectomized rabbit. *Endocrinology* 87: 1386–1389, 1970.
5. Crowley, W. R. and O. B. Ward. Male copulatory behavior induced in female rats by peripheral electric shock. *Physiol. Behav.* **13**: 129–131, 1974.
6. Del Fiaco, M., W. Fratta, G. L. Gessa and A. Tagliamonte. Lack of copulatory behaviour in male castrated rats after p-Chlorophenylalanine. *Brit. J. Pharmac.* **51**: 249–251, 1974.
7. Emery, D. E. and B. D. Sachs. Ejaculatory pattern in female rats treated with estrogen and para-Chlorophenylalanine. Abstract of paper presented at New England Endocrinology Conference. Storrs, Conn. Dec. 5–6, 1974.
8. Emery, D. E. and B. D. Sachs. Ejaculatory patterns in female rats without androgen treatment. *Science*, in press.
9. Everitt, B. J., K. Fuxe and T. Hokfelt. Inhibitory role of dopamine and 5-hydroxytryptamine in the sexual behavior of female rats. *Eur. J. Pharmac.* **29**: 187–195, 1974.
10. Everitt, B. J., K. Fuxe, T. Hokfelt and G. Johsson. Pharmacological and biochemical studies on the role of monoamines in the control by hormones of sexual receptivity in the female rat. *J. comp. physiol. Psychol.* 89: 556–572, 1975.
11. Gessa, G. L., A. Tagliamonte, P. Tagliamonte and B. B. Brodie. Essential role of testosterone in the sexual stimulation induced by p-Chlorophenylalanine in male animals. *Nature* **227**: 616–617, 1970.
12. Gessa, G. L. and A. Tagliamonte. Possible role of brain serotonin and dopamine in controlling male sexual behavior. *Adv. Biochem. Psychopharmac.* **11**: 217–228, 1974.
13. Gorzalka, B. B., D. L. Rezek and R. E. Whalen. Adrenal mediation of estrogen-induced ejaculatory behavior in the male rat. *Physiol. Behav.* **14**: 373–376, 1975.
14. Kato, R. Sex-related differences in drug metabolism. *Drug Metabolism Rev.* **3**: 1–32, 1974.
15. Koe, B. K. and A. Weissman. *p*-Chlorophenylalanine: a specific depletor of brain serotonin. *J. Phamrac. exp. Ther.* **154**: 499–516, 1966.
16. Kreiger, M. S. The effect of peripheral electrical shock on male copulatory behavior exhibited by female rats. Paper presented at the Eastern Regional Conference on Reproductive Behavior. Nags Head, N.C., May 18–21, 1975.
17. Komisaruk, B. R. Strategies in neuroendocrine neurophysiology. *Am. Zool.* **11**: 741–754, 1971.
18. Malmnas, C. P. and B. J. Meyerson. *p*-Chlorophenylalanine and copulatory behavior in the male rat. *Nature* **232**: 398–400, 1971.
19. Malmnas, C. O. Monominergic influence on testosterone activated copulatory behavior in the castrated male rat. *Acta physiol. scand. Suppl.* **395**: 1–127, 1973.
20. Meyerson, B. J. Central nervous monoamines and hormone induced estrous behaviour in the spayed rat. *Acta physiol. scand. Suppl.* **241**: 1–32, 1964.
21. Meyerson, B. J. and T. Lewander. Serotonin synthesis inhibition and estrous behavior in female rats. *Life Sci.* 9: 661–671, 1970.
22. Meyerson, B. J., H. Carrer and M. Eliasson. 5-Hydroxytryptamine and sexual behavior in the female rat. *Adv. Biochem. Psychopharmac.* **11**: 229–242, 1974.
23. Mittler, M. M., B. Morden, S. Levine and W. Dement. The effects of para-Chlorophenylalanine on the mating behavior of male rats. *Physiol. Behav.* 8: 1147–1150, 1972.
24. Moyer, K. E. Sex differences in aggression. In: *Sex Differences in Behavior*, edited by R. C. Friedman, R. M. Richart and R. L. Vande Wiele. New York: John Wiley and Sons, 1974.
25. Naftolin, F. K., J. Ryan and Z. Petro. Aromatization of androstenedione by the diencephalon. *J. clin. Endocr.* 33: 368–370, 1971.
26. Puech, A.-J., P. Simon, R. Chermat et J.-R. Biosser. Profil neuropsychopharmacologique de l'apomorphine. *J. Pharmacologie (Paris)* **5**: 241–251, 1974.
27. Segal, D. S. and R. E. Whalen. Effects of chronic administration of *p*-Chlorophenylalanine on sexual receptivity of the female rat. *Psychopharmacologia* **16**: 434–438, 1970.
28. Sheard, M. H. The effect of *p*-Chlorophenylalanine on behavior in rats: relation to brain serotonin and 5-hydroxyindoleacetic acid. *Brain Res.* **15**: 524–528, 1969.
29. Shillito, E. E. The effect of paraChlorophenylalanine on social interactions of male rats. *Br. J. Pharmac.* 38: 305–315, 1970.
30. Silas, P. J. and D. A. Dewsbury. *p*-Chlorophenylalanine facilitates copulatory behavior in male rats. *Nature* **232**: 400–401, 1971.
31. Singer, J. J. Effects of *p*-Chlorophenylalanine on the male and female sexual behavior of female rats. *Psychol. Rep.* **30**: 891–893, 1972.
32. Tagliamonte, A., P. Tagliamonte, G. L. Gessa and B. B. Brodie. Compulsive sexual activity induced by *p*-Chlorophenylalanine in normal and pinealectomized male rats. *Science* **166**: 1433–1435, 1969.

33. Tagliamonte, A., P. Tagliamonte and G. L. Gessa. Reversal of pargyline-induced inhibition of sexual behavior in male rats by *p*-Chlorophenylalanine. *Nature* **230:** 244–245, 1971.
34. Tagliamonte, A., W. Fratta, G. Mercuro and G. L. Gessa. Failure of *p*-Chlorophenylalanine (PCPA) to restore copulatory behavior in male castrated rats. *Rev. Farmac. Terapia* **3:** 399–403, 1972.
35. Tagliamonte, A., W. Fratta, M. Del Fiaco and G. L. Gessa. Possible stimulatory role of brain dopamine in the copulatory behavior of male rats. *Pharmac. Biochem. Behav.* **2:** 257–260, 1974.
36. Ungerstedt, U. Brain dopamine neurons and behavior. In: *The Neurosciences.* Third study program, edited by F. O. Schmitt and F. G. Worden. Cambridge: The M. I. T. Press, 1974.
37. Ward, I. L., W. R. Crowley, F. P. Zemlan and D. L. Margules. Monoaminergic mediation of female sexual behavior. *J. comp. physiol. Psychol.* 88: 53–61, 1975.
38. Ward, I. L. and F. J. Renz. Consequences of prenatal hormone manipulations on the adult sexual behavior of female rats. *J. comp. physiol. Psychol.*78: 349–355, 1972.
39. Zemlan, F. P., I. L. Ward, W. R. Crowley and D. L. Margules. Activation of lordotic responding in female rats by suppression of serotonergic activity. *Science* **179:** 1010–1011, 1973.