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Role of the Adrenal Glands, Repeated Matings and Monoamines in Lordosis Behavior of Rats[1,2]

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LARSSON, K., H. H. FEDER AND B. R. KOMISARUK. *Role of the adrenal glands, repeated matings and monoamines in lordosis behavior of rats.* PHARMAC. BIOCHEM. BEHAV. 2(5) 685–692, 1974. – Tetrabenazine, a potent monoamine depletor, was administered to estrogen-primed rats which were ovariectomized or ovariectomized and adrenalectomized. The drug treatment facilitated sexual receptivity in the nonadrenalectomized rats but had no such effects in adrenalectomized ones. Injection of saline, the solvent of tetrabenazine, in combination with repeated mating exerted the same facilitatory effect as the drug on the lordosis behavior. As in the case of tetrabenazine the effects only occurred in the presence of the adrenals. The facilitatory effect on the lordosis behavior was interpreted as due to adrenal steroids with progestational action which were secreted as a response to tetrabenazine, saline or/and repeated mating.

Sexual receptivity Lordosis behavior Tetrabenazine Progesterone Repeated matings Adrenals Adrenal steroids

OVARIECTOMIZED female rats may show sexual receptivity after treatment with estrogen alone [6], but the effects of estrogen are facilitated by subsequent injection of progesterone [3]. This synergistic action of estrogen and progesterone on sexual receptivity is probably a feature of the normal estrous cycle of rats because in this species elevated levels of systemic plasma progesterone are found to coincide with spontaneously occurring sexual receptivity [9]. The biochemical basis of the facilitatory effect of progesterone on the lordosis response is unknown.

Meyerson [20, 21, 22, 23] found that injection of reserpine (or other drugs which deplete brain monoamines) induced lordosis in estrogen-primed female rats. On the basis of these data he suggested the existence of a central monoaminergic mechanism inhibitory to lordosis which could be inactivated by a depletion of monoamines. Reserpine was thought to be a pharmacological mimic of progesterone in this respect.

Meyerson did not adequately consider an alternative explanation for these findings with reserpine. Evidence has accumulated that the adrenal cortex is involved in the facilitation of lordosis following reserpine administration. The adrenal gland of the female rat secretes progesterone and compounds with progestational activity [4, 15, 16, 17, 19]. Injection of ACTH into spayed rats results in increased concentration of systemic progesterone [16,17], and spayed estrogen-primed rats treated with ACTH display lordosis [8]. These findings, together with the fact that treatment of rats with reserpine produces increased secretion of ACTH [10, 11, 12], suggest that reserpine causes secretion of adrenal progesterone which, in turn, acts synergistically with previously injected estrogen to induce sexual receptivity. Indeed, direct evidence that adrenal secretions act synergistically with previously injected estrogen in facilitating the lordosis response has recently been obtained in mice [14,17] and rats [24]. Spayed estrogen-primed mice displayed lordosis after injection of reserpine but failed to do so after adrenalectomy. Similarly, injection of reserpine into spayed estrogen-primed rats resulted in exhibition of lordosis and increased secretion of systemic progesterone. Both these effects were suppressed by dexamethasone, a corticotropin suppressor.

The present study was aimed at investigating further Meyerson's hypothesis of monoamine depletion and induction of lordosis. For this purpose we administered tetrabenazine (TBZ), a potent monoamine depletor [25], to

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estrogen-primed rats which were ovariectomized or ovariectomized and adrenalectomized.

EXPERIMENT 1

Method

Animals. Sprague-Dawley female rats (50 days old) were purchased from CAMM-Laboratories, Wayne, N. J. They were housed individually in a temperature controlled room, at about 22° C, with lights off between 05.00–19.00. Food and water were available ad lib. Adrenalectomized animals (see below) were given 0.9% saline solution as drinking water. Experimental work began no sooner than 1 week after arrival of the animals at the laboratory. Adult, sexually experienced male rats of the Sprague-Dawley strain, about 5 months old, were used as stimulus males.

Procedure. All operations were performed under Equithesin anesthesia and the rats received Terramycin (2.5 mg) on the day of the operation. (In the adrenalectomized animals, ovariectomy was performed immediately after adrenalectomy). The animals were allowed 2 weeks to recover whereupon experimental treatment was begun.

Except for the group AONT animals (see Table 1), all females were injected with estradiol benzoate (EB, 3.3 μg/animal in 0.1 ml sesame oil). Depending upon the group assignment, the animals were injected 40 hr later with progesterone (P), tetrabenazine methanesulfonate (TBZ) or saline (S) in dosages indicated in Table 1. The hormones were injected intramuscularly in the hind legs and the drug and saline solutions were injected intraperitoneally. The TBZ dosage was selected for producing a significant lowering of monoamine levels [1].

Testing for lordosis posture (depression of the back, raising of the hind-legs and raising of the head) was performed in a circular Plexiglas cage, 53 cm in dia., 42 cm height, with the floor covered with wood shavings. The following procedures were used to elicit lordosis:

(a) Manual palpation of the flanks and the lower back (back-scratching).

(b) Manual palpation simultaneously of the lower back, flanks and the perineum (back-perineum-stimulation).

(c) Presentation to a male. The female was presented to a sexually vigorous male, who was permitted to perform 5 mounts with pelvic thrusting. The ratio of lordosis to mounts by the male (LQ) was used as an index of sexual receptivity.

(d) Cervical stimulation: a glass rod (1 cc syringe plunger) was inserted into the vagina until it contacted the cervix, while simultaneously the flanks, lower back, and perineum were palpated manually [18].

In assessing the effects of tetrabenazine (TBZ), progesterone (P) or saline (S), a positive lordosis response was recorded only if 2 observers could elicit lordosis by manual stimulation. In pretests performed to eliminate any rats which showed lordosis in response to estrogen alone, lordosis identified by only 1 observer was sufficient to eliminate that rat from further testing. All behavior tests were conducted with a double-blind procedure.

Testing with males was performed 3 hr after the injection of TBZ, P or S, and cervical probing was performed 4 hr after the injections. The animals were tested with back-scratching and back-perineum palpation immediately before the injections (pre-experimental tests) and at 1/2, 1, 2, 3, 4, 5, 6, 7 and 8 hr after the injections (experimental tests).

Experimental design. The animals were assigned to one of 7 groups and treated as indicated in Table 1. Each animal participated in 2 tests which were performed 1 week apart. In groups treated with TBZ and P, the animals first received either P or TBZ. On the second test the treatment of these 2 groups was reversed.

Statistical analysis. Animals in the three control groups (AOS, OS and AONT), which had been treated identically in the two series of tests, were selected randomly from the first and second series for the purpose of the statistical

TABLE 1

GROUPS AND TREATMENTS IN EXPERIMENT 1

Groups	N	Body weight M ± SE	Operation	Treatment
AOS	13	210 ± 8.25	Adrenalectomy + ovariectomy	3.3 μg/animal EB + 1.0 ml/kg saline i.p.
AOT	35	201 ± 4.38		3.3 μg/animal EB + 10 mg/kg tetrabenazine in 10 mg/ml saline i.p.
AOP	35			3.3 μg/animal EB + 0.6 mg/animal progesterone in 0.1 ml sesame oil i.m.
AONT	7	250 ± 3.54		No treatment
OS	10	222 ± 5.22	Ovariectomy	3.3 μg/animal EB + 1.0 ml/kg saline i.p.
OT	19	218 ± 3.05		3.3 μg/animal EB + 10 mg/kg tetrabenazine in 10 mg/ml saline i.p.
OP	19			3.3 μg/animal EB + 0.6 mg/animal progesterone in 0.1 ml sesame oil i.m.

AOS (Adrenalectomy–Ovariectomy–Saline); AOT (Adrenalectomy–Ovariectomy–Tetrabenazine); AOP (Adrenalectomy–Ovariectomy–Progesterone); AONT (Adrenalectomy–Ovariectomy–NonTreatment); OS (Ovariectomy–Saline); OT (Ovariectomy–Tetrabenazine); OP (Ovariectomy–Progesterone).

analysis. Statistical analysis was performed by the Mann-Whitney U test corrected for ties and the Wilcoxon matched-pairs signed-rank test. Since the hypothesis tested predicts unidirectional changes in the behavior (facilitation of lordosis) one-tailed tests were used throughout and probability values less than or equal to 0.05 were considered significant.

Results

The AOS group showed a significantly lower frequency of lordosis in response to back-perineum stimulation than the OS group (Tables 2, 3 and 4). In addition the AOS never displayed lordosis to back-scratching.

Inspection of Table 3 shows that in the OS group the lordosis frequency increased progressively during the course of testing until in the final tests half of the animals showed lordosis to back-perineum stimulation. A more detailed statistical analysis confirmed this trend. During the first 4 hr no systematic group differences occurred ($z = 0.72$, NS) but during the last 4 hr the OS group showed a markedly higher percentage of animals displaying lordosis to back-perineum stimulation than the corresponding adrenalectomized controls ($z = 2.90$, $p<0.002$). This suggests that adrenal secretions which were released by the procedure of repeated testing facilitated the display of lordosis.

Similar observations were made in the P-treated groups. During the first 4 hr no difference in the frequency of lordotic responses was recorded to back-perineum stimulation between AOP and OP groups ($z = 1.28$, NS). During the final 4 hr, the nonadrenalectomized P-treated groups exhibited a significantly higher proportion of females displaying lordosis ($z = 2.82$, $p<0.002$). In response to male mounting, no group differences were found in the LQ, but during the entire 8 hr testing period nonadrenalectomized rats showed a higher frequency of earwiggling than the corresponding adrenalectomized animals ($z = 1.84$, $p<0.03$).

TABLE 2

PERFORMANCES OF THE VARIOUS GROUPS IN EXPERIMENT 1

Groups	AOS	AOT	AOP	AONT	OS	OT	OP
N	13	30	27	7	10	17	14
Lordosis response to							
Male mounting*							
Percentage animals showing lordosis	20	13	88	0	30	41	100
LQ	10	15	81	0	20	18	86
Back-perineum stimulation†							
Percentage animals showing lordosis	23	40	70	0	60	41	100
Percentage tests with lordosis	13	14	37	0	29	22	61
Backscratching†							
Percentage animals showing lordosis	0	0	48	0	20	0	57
Percentage tests with lordosis	0	0	20	0	4	0	28
Earwiggling to backscratching							
Percentage animals showing earwiggling	0	0	67	0	30	0	86
Percentage tests with earwiggling	0	0	26	0	8	0	42
Lordosis to cervical probing with back-perineum palpation‡							
Percentage animals showing lordosis	100	100	100	100	100	100	100

*Based on performances during 1 test
†Based on performances during 9 tests
‡This test was performed at the end of the 8-hr testing procedure.

TABLE 3

PERCENTAGE OF ANIMALS SHOWING LORDOSIS TO BACKSCRATCHING (Bsc) AND TO BACK-PERINEUM STIMULATION (Bps) ON THE SUCCESSIVE TESTS

Groups		AOS	AOT	AOP	AONT	OS	OT	OP
N		13	30	27	7	10	17	14
Hours after injection								
1/2	Bsc	0	0	0	0	0	0	0
	Bps	23	33	4	0	10	24	0
1	Bsc	0	0	4	0	0	0	14
	Bps	23	33	11	0	0	24	29
2	Bsc	0	0	11	0	0	0	29
	Bps	8	13	30	0	10	24	57
3	Bsc	0	0	37	0	10	0	50
	Bps	23	13	63	0	33	24	71
4	Bsc	0	0	33	0	10	0	43
	Bps	8	3	70	0	33	24	64
5	Bsc	0	0	33	0	0	0	29
	Bps	8	7	52	0	33	29	71
6	Bsc	0	0	22	0	33	0	29
	Bps	8	7	59	0	50	24	93
7	Bsc	0	0	19	0	0	0	29
	Bps	8	0	56	0	50	12	93
8	Bsc	0	0	7	0	0	0	43
	Bps	8	7	33	0	40	12	71

Under the conditions of this experiment no facilitatory effects of TBZ on the display of lordosis were observed that could be attributed to the drug alone (Tables 2 and 3).

In contrast to the S- and P-treated animals, the TBZ-treated rats did not show any alterations in lordosis frequency as a consequence of repeated testing. Neither were systematic differences between adrenalectomized and non-adrenalectomized groups noted during the first 4 hr ($z = 0$, NS) or during the last 4 hr of testing ($z = 1.12$, NS). TBZ caused sedation, ptosis and piloerection in all rats.

The relatively high frequency of lordotic responses to back-perineum stimulation displayed by the nonadrenalectomized rats compared with the adrenalectomized ones, and the progressive increase in the lordosis frequency with repeated testing seen in the nonadrenalectomized rats give some support to the assumption that adrenal secretions may facilitate the elicitation of lordosis in estrogen-primed rats. The failure of TBZ, in the dosage applied to influence the occurrence of the lordotic response leaves the question unsolved of whether or not TBZ affects lordosis behavior via the adrenal gland. A second experiment was therefore performed in which Meyerson's dosage, route of injection, and schedule of testing were closely followed. However, the criteria for positive lordotic responses were the same as in our previous experiment.

EXPERIMENT 2

Method

Animals. A new group of animals used in this study was of the same strain as in Experiment 1 and was maintained under the same conditions.

Procedure. The animals were prior to the experimental treatment divided into 8 groups which were treated as indicated in Table 5. They were all injected with EB (10 μg/kg) intramuscularly and this was followed 50 hr later by S, TBZ or P.

The animals were tested with sexually active males and

TABLE 4

GROUP COMPARISONS OF THE FREQUENCY OF LORDOSIS PATTERNS DISPLAYED IN RESPONSE TO MALE MOUNTING (1 TEST) AND TO BACK-PERINEUM STIMULATION (9 TESTS)

Groups Compared	LORDOSIS TO Mount	p	Back-perineum Stimulation	p
AOS-AOT	z = 0.33	NS	z = 0.24	NS
AOS-AOP	z = 2.88	p <0.002	z = 2.59	p<0.005
AOT-AOP	T = 3	p <0.01	T = 9.5	p<0.01
OS-OT	z = 0.41	NS	z = 1.44	NS
OS-OP	z = 3.04	p <0.001	z = 2.79	p<0.003
OT-OP	T = 0	p <0.02	T = 0	p<0.01
AOS-OS	z = 0.47	NS	z = 2.02	p<0.02
AOT-OT	z = 1.15	NS	z = 0.18	NS
AOP-OP	z = 0.63	NS	z = 2.34	p<0.01

the criteria used for identifying lordosis in response to the male's mounting were the same as in Experiment 1. Testing was performed at 2, 4 and 6 hr after the injections of S, TBZ or P. In order to replicate the procedure used by Meyerson no pre-tests were performed. Neither were any attempts made to provoke lordosis behavior by back-scratching or back-perineum stimulation, because such stimulation was not used by him.

The handled controls were brought to the testing room in their home cages at the 2 and 4 hr testing periods and placed in an observation cage, which has previously been used for mating tests, for 5 min, after which they were placed in a second observation cage for 5 min. This procedure resulted in the rats being out of their home rack location for about 30 min during each test period. At the 6 hr testing period, they were placed into a similar cage with an active male and received 10 mounts. The non-handled controls were left in their home cages and racks until the 6 hr mating test.

Results

The LQ was significantly higher in the TBZ-injected nonadrenalectomized rats than in the TBZ-injected adrenalectomized rats in each test period (Tables 6 and 7). However, a similar difference was observed in the saline control group in the final test (i.e., a significantly higher LQ was observed in the nonadrenalectomized rats compared to the adrenalectomized rats).

In the nonadrenalectomized rats TBZ induced lordosis even in the first test. The possibility that TBZ induced lordosis by causing an early release of P cannot be excluded since injections of P also resulted in lordosis behavior even in the first test. Thus TBZ appears to be unable to facilitate receptivity in the absence of the adrenals, but it may release adrenal progestins which in turn facilitates lordosis. Progesterone was equally effective in inducing lordosis in the adrenalectomized-ovariectomized, and the ovariectomized groups. In the tests following the first one a tendency was observed for a steady increase in LQ in the nonadrenalectomized groups. Since no facilitation of lordosis was observed in either of the handled groups, it appears that repeated mounting and intromissions but not handling influence the adrenal-mediated effects on lordosis behavior.

DISCUSSION

Comparing corresponding data in the present two experiments, the nonadrenalectomized rats showed in the second experiment a higher LQ in response to TBZ than in the first experiment. While this difference presumably mainly reflects differences in the dosages of TBZ administered the possible importance of procedural deviations between the two experiments should not be overlooked.

Previous studies showed that TBZ in the dosages applied causes a marked reduction of the levels of monoamines and facilitates the display of lordosis in ovariectomized, estrogen-primed rats [1, 2, 20]. The present findings

TABLE 5

GROUPS, TREATMENT AND HOURS OF TESTING IN EXPERIMENT 2

Groups	N		Operation	Treatment	Hours tested		
AOS	12	259 ± 9.32	Adrenalectomy + ovariectomy	10 μg/kg EB + 2 ml/kg saline s.c.	2	4	6
AOT	12	253 ± 2.11		10 μg/kg EB + 40 mg/kg tetrabenazine s.c.	2	4	6
AOP	11	244 ± 1.55		10 μg/kg EB + 0.6 mg/animal progesterone i.m.	2	4	6
OS	10	268 ± 9.56	Ovariectomy	10 μg/kg EB + 2 ml/kg saline s.c.	2	4	6
OT	11	248 ± 6.81		10 μg/kg EB + 40 mg/kg tetrabenazine s.c.	2	4	6
OP	10	270 ± 9.29		10 μg/kg EB + 0.6 mg/animal progesterone i.m.	2	4	6
OS – Handled	10	237 ± 5.68		10 μg/kg EB + 2 mg/kg saline s.c.			6
OS – Nonhandled	10	265 ± 4.01		10 μg/kg EB + 2 mg/kg saline s.c.			6

TABLE 6

LQ SHOWN BY THE VARIOUS GROUPS IN TESTS WITH MALE RATS PERFORMED 2, 4 AND 6 HR AFTER INJECTION IN EXPERIMENT 2

Groups	2	4	6
AOS	11	22	18
AOT	3	8	8
AOP	56	73	71
OS	10	34	50
OT	36	44	53
OP	42	74	68
OS – Handled	–	–	12
OS – Nonhandled	–	–	12

suggest that these facilitatory effects, which were attributed to TBZ by Meyerson [20], may be due to release of adrenal steroids induced either by TBZ or by repeated behavioral testing, rather than to a direct action of TBZ on neural tissues regulating the expression of lordosis. The idea that monoamine depletors such as TBZ can facilitate lordosis by stimulating adrenocortical secretion is consonant with other experiments showing that reserpine does not facilitate lordosis unless the adrenal glands are present [24,27]. One of the ways in which these pharmacological agents may cause increases in adrenal cortical activity is by depleting brain catecholamines which normally exert a tonic inhibitory influence on the hypothalamic-pituitary-adrenal axis [28]. The writings of Meyerson favor the view that monoamine depletors such as reserpine or TBZ affect female behavior by acting directly on neural tissues mediating expression of lordosis rather than by acting indirectly to stimulate adrenocortical secretion. The experiments reported here suggest that additional controls and behavioral observations may have led to a different interpretation. In the present study we found that 7 of 11 ovariectomized, adrenalectomized rats displayed lordosis after being given only estrogen (10 μg/kg EB b.w.), and were therefore unsuitable for testing possible lordosis-facilitatory actions of TBZ. Meyerson did not state that he excluded such animals from his study. It is not certain, therefore, that the lordosis responses observed in ovariectomized, adrenalectomized, estrogen-treated (10 μg EB/kg b.w.) rats after reserpine administration were attributable to this drug [20].

A second finding which casts doubt on Meyerson's viewpoint is that the behavior displayed by our TBZ-treated rats differed from that exhibited by females in spontaneous estrus or estrogen-progesterone-induced estrus. TBZ-treated females rarely showed heightened locomotor activity, hopping, sniffing, presentation to the male or ear vibration. Rather than exhibiting these characteristic estrous behavior

TABLE 7

RESULTS OF STATISTICAL ANALYSIS IN EXPERIMENT 2 (MANN-WHITNEY U TEST CORRECTED FOR TIES)

Groups compared	Hour tested 2 z	2 p	4 z	4 p	6 z	6 p
AOS – AOT	1.13	NS	0.42	NS	0.80	NS
AOS – AOP	2.68	<0.004	2.65	<0.004	5.34	<0.001
AOS – OS	0.67	NS	0.76	NS	1.68	<0.05
AOS – OT	1.73	<0.04	0.09	NS	2.02	<0.02
AOS – OP	1.72	<0.04	2.69	<0.004	2.33	<0.01
AOT – AOP	3.34	<0.001	2.56	<0.005	3.25	<0.001
AOT – OS	0.29	NS	1.12	NS	2.57	<0.005
AOT – OT	2.35	<0.009	1.93	<0.03	2.80	<0.003
AOT – OP	2.49	<0.006	3.16	<0.001	2.81	<0.003
AOP – OS	2.81	<0.003	1.76	<0.04	1.50	NS
AOP – OT	1.11	NS	1.66	<0.05	0.82	NS
AOP – OP	0.87	NS	0.00	NS	0.27	NS
OS – OT	2.86	<0.002	0.46	NS	0.43	NS
OS – OP	1.92	<0.03	1.85	<0.03	1.46	NS
OS – OP	0.26	NS	1.68	<0.05	0.76	NS

patterns the TBZ-treated females generally remained motionless in a corner of the testing arena. The behavior seen in TBZ-treated females could not be accurately described as estrous. Furthermore, the TBZ-treated animals sometimes displayed a response to back-perineum stimulation which superficially resembled a lordosis. However, this was a hunching of the back with the rump and middle of the back held high (Fig. 1) while the head was depressed, rather than a true lordosis, in which the head and rump are elevated while the middle of the back is depressed. Nevertheless, it should be noted that all the rats were capable of showing the lordosis reflex when cervical probing was applied in conjunction with back-perineum palpation (Table 2).

A third factor not adequately considered by Meyerson involves the effects of repeated testing on female receptivity. Work in other laboratories has suggested that repeated testing causes an increase in lordosis frequency [5,13]. In the present study we found that in rats treated with S or TBZ repeated testing and mating facilitated the occurrence of lordosis (using criteria described in *Methods*) only in rats with intact adrenals. Testing was carried out in this study as described by Meyerson [20]. Interestingly, in Meyerson's study utilizing repeated testing, the proportion of animals showing lordosis increased from 8% at 2 hr after TBZ to 92% at 6 hr after TBZ. However, Meyerson interpreted these data as indicative of an effect of TBZ on lordosis, rather than an effect of repeated testing. This conclusion is put into question by our observation (Table 6) that the OS controls showed the same increase in lordosis frequency as the OT animals. Furthermore, Meyerson did not use a control group comparable to our AOS controls.

The criticisms of Meyerson's hypothesis that we have offered on the basis of data reported here do not preclude the possibility that monoamines are directly involved in facilitating lordosis [7,26]. Indeed, a recent report suggests that drugs which interfere with serotonergic activity (PCPA, methysergide) are effective in facilitating lordosis in ovariectomized, estrogen-primed rats whose adrenals were removed [29]. Although TBZ depletes serotonin, perhaps the reason for a failure to facilitate lordosis in the present study is that TBZ also depletes the other monoamines, adequate levels of which may be necessary for the lordosis response in the absence of adrenal secretions. If this is the case then we must somehow account for the occurrence of lordosis in rats with intact adrenals which received a comparable dosage of TBZ. Two possibilities are that (1) the presence of the adrenals counteracts general debilitating effects of TBZ and (2) steroids of adrenal origin may exert facilitatory effects on the lordosis response independent of the monoamines.

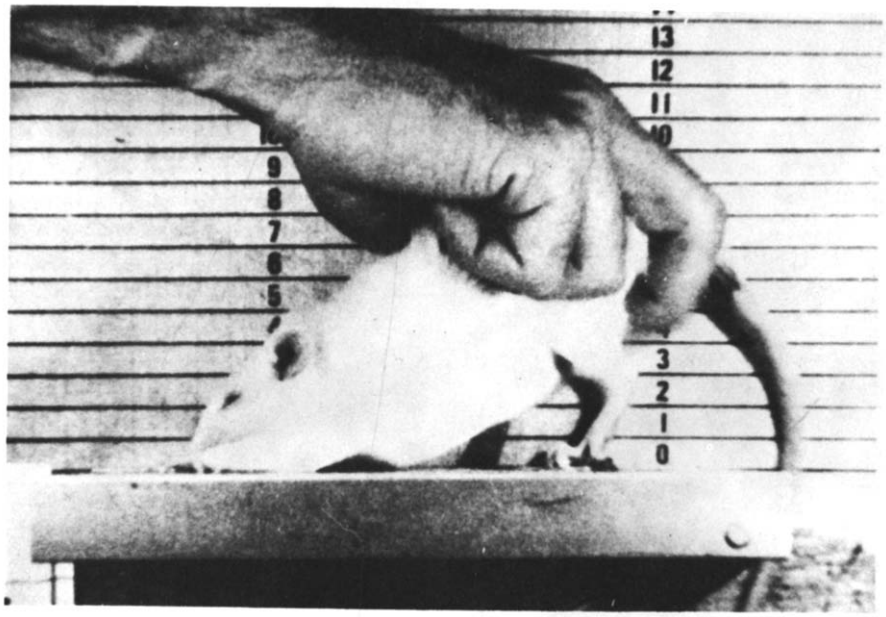

FIG. 1. A behavior pattern superficially resembling lordosis displayed by an AOT – rat in response to back-perineum stimulation. Note a hunching of the back with the rump and middle of the back held high while the head was depressed.

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