Mating Behavior: Facilitation in the Female Rat after Cortical Application of Potassium Chloride

Abstract. *Mating behavior in ovariectomized female rats treated with estrogen can be greatly enhanced by subcutaneous injections of progesterone. Application of potassium chloride to the cortex of females previously treated with estrogen can also induce greatly increased sexual receptivity as indicated by the lordosis response. This facilitation of mating behavior by a treatment known to cause functional decortication suggests that mechanisms mediating female mating behavior are under tonic inhibition by an inhibitory system which involves the cerebral cortex.*

A central feature of sexual behavior in the female rat is the lordosis response. Occurrence of this postural response, which permits the male to achieve vaginal penetration (intromission), can be observed in spayed females treated with estrogen. However, if the estrogen-treated female is injected with progesterone several hours before testing, lordosis occurs with greater frequency (*1*). We here demonstrate that application of KCl to the cerebral cortex of estrogen-treated females also increases the frequency of lordosis. Such facilitation of lordosis frequency by a treatment which causes functional decortication (*2*) supports the hypothesis that the mechanisms which mediate the lordosis reflex are under some degree of tonic inhibition by a cortically involved inhibitory system. Recent reviews (*3*) have assembled evidence that (i) spinal and bulbo-spinal mechanisms which mediate reflexes related to coitus are under varying degrees of inhibition by more rostrally located neural circuits and (ii) that gonadal hormones promote coital behavior by modifying the inhibitory effects exerted by higher neural mechanisms.

In order to determine whether suppression of cortical activity by application of KCl would increase the incidence of lordosis, we implanted stainless steel cannulae bilaterally in two groups of ovariectomized adult female rats under Brevital anesthesia; the cannulae were implanted 5 to 7 days before the rats were tested. The cannulae rested on the dura overlying the parietal-occipital region of the neocortex in a manner described by Russel and Ochs (*4*). The shaft of each cannula was 1 mm long and had a lumen 1.3 mm in diameter. The cannulae were filled with a 15-percent KCl solution at the time of testing by dropping the solution into them with a syringe. One group of these implanted, spayed females was injected subcutaneously with 2 μg of estradiol benzoate in sesame oil daily for 2 days. The second group received no hormone injections. On the 3d day females in both groups were given a sex behavior test an hour before KCl was applied. After this preliminary test, KCl was applied to the cortex through the cannulae and each female was tested every 15 minutes during the next hour.

Animals were maintained on a reversed day–night light cycle of 14 hours of light and 10 hours of darkness. Testing began approximately 2 to 3 hours after the lights were turned off. A sex behavior test consisted of placing the female in a cage with a sexually active Long-Evans male. The female remained in the cage until the male mounted her 10 times or until 15 minutes elapsed. The number of lordosis responses shown by the female was divided by the number of mounts achieved by the male during the test. This ratio, multiplied by 100, constituted the female's score (lordosis quotient) for any particular test. Similar quotients have been used previously as a measure of the intensity of sexual receptivity (*1*). Recording only the presence or absence of lordosis does not provide a sensitive indication of changes in the receptivity within any particular animal.

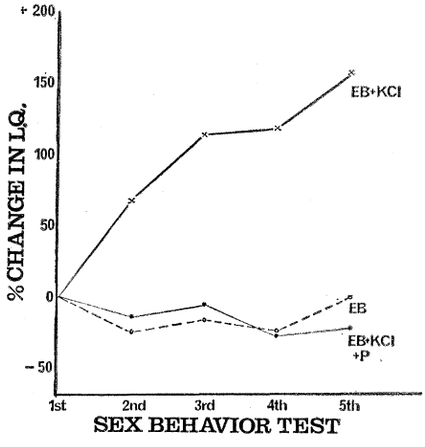

Fig. 1. Relative change in lordosis quotient (L.Q.) after application of KCl to groups 1 and 4, expressed as a percentage of the L.Q. obtained in the first sex behavior test of each group. The change in L.Q. for group 3 over the same time period is also shown. KCl was applied within 1 hour after the first test and 15 minutes before the second test. The remaining tests were conducted every 15 minutes.

The mean lordosis quotients (L.Q.) for these two groups, along with a summary of the treatments, are shown in Table 1. Cortical application of KCl in females previously tested with estrogen produced an increase in the mean L.Q. from 21 in the preliminary test to 54 in the fifth or last test. The difference between these scores was statistically significant ($P < .025$, Wilcoxon signed ranks, one-tailed test). Ovariectomized females not stimulated with estrogen failed to show even a single lordosis before or after application of KCl (group 2).

To control for the possibility that repeated testing of estrogen-treated females with sexually active males could increase the L.Q., a group of females (group 3, Table 1) that had received daily estrogen injections for 2 days were given sex behavior tests according to the same time schedule as the first two groups. In these rats, however, KCl was not applied to the cortex. The mean test scores for the five females in this group that showed a lordosis response in at least one of the behavior tests are shown in Table 1. Repeated testing of estrogen-treated females in the absence of KCl application failed to alter the L.Q. significantly.

Close observation of females treated with KCl indicated that some postural impairments were present, especially when the females were mounted. The most noticeable impairment was a splaying out of the female's rear legs when the male mounted. In such instances it was impossible to be certain whether the female was or was not showing lordosis and in most cases the females had to be scored negative for lordosis.

To ascertain the extent to which KCl treatment and the resultant motor debilitation interfered with the lordosis response, a group of already highly receptive females were tested with KCl. A high incidence of lordosis is routinely achieved in females treated with estrogen by administering subcutaneous progesterone 4 to 6 hours

before the animals are tested (*1*). Therefore, rats of group 4, which had been implanted with cannulae 5 to 7 days before, were treated with estrogen, as was group 3. On day 3 each female received 0.5 mg of progesterone 4 hours before the first sex behavior test. After the first test KCl was applied to the cortex and the females were tested every 15 minutes for the next hour. The mean lordosis quotients for the five tests appear in Table 1. The preliminary test score of 71 is well within the range normally reported for animals treated with estrogen and progesterone (*1*). After KCl treatment the mean L.Q. dropped to 55 by the fifth test. While this decrease in L.Q. is not statistically significant (Wilcoxon signed ranks), motor and postural impairments were obvious during those tests after KCl was applied. The mean lordosis quotients of the estrogen-KCl group (group 1) and the estrogen-progesterone-KCl group (group 4) 60 minutes after KCl was applied were nearly identical (54 and 55, respectively). While KCl facilitates the occurrence of lordosis in the estrogen-treated female (group 1), it also interferes with the expression of this reaction (group 4). This interference appears to result from impairment of the female's postural responses when she is mounted by the male. Larsson (*5*) has also concluded that the failure of females treated with estrogen and progesterone to show lordosis after application of 25-percent KCl was the result of motor impairment and not inhibition of the lordosis response per se.

The relative changes in L.Q. for groups 1, 3, and 4 are shown in Fig. 1 as percent change from the first or control sex behavior test. In spite of the debilitating motor impairments, application of KCl to the cortex of females treated only with estrogen led to a 157 percent increase in mean L.Q.

After surgical destruction of 97 to 100 percent of the neocortex, Beach (*6*) observed that while the display of lordosis in females treated with estrogen and progesterone became more variable, lordosis was displayed in response to stimuli which had not elicited this reaction before they were decorticated (for example, pipette in the vagina, grasp of the experimenter's hand, preliminary investigation by the male). Also, several of the decorticate females frequently maintained the lordosis posture for a long period after the male had dismounted. While data are not available to determine the level of lordosis responding in surgically decorticate, estrogen-treated females not treated with progesterone, these observations indicate a facilitation of the lordosis reaction subsequent to elimination of cortical function.

Table 1. Summary of treatments and the mean lordosis quotients for females that showed lordosis in at least one of the five tests. EB refers to subcutaneous injections of 2 μg estradiol benzoate, and P indicates subcutaneous injection of 0.5 mg progesterone.

Group	No.	Treatment at (hours)				No. showing lordosis	Mean lordosis quotient for each test*				
		0	24	44	48		1st	2nd	3rd	4th	5th
1	12	EB	EB		KCl	9	21	35	45	46	54
2	12				KCl	0	0	0	0	0	0
3	12	EB	EB			5	26	20	22	20	26
4	11†	EB	EB	P	KCl	10	71	62	67	52	55

* If a female showed a lordosis in any test, all scores for that female were used. † One animal died following cannula implantation leaving a total of 11 females for this group.

Surgically decorticate females also showed an increase over preoperative scores in other aspects of the female mating pattern such as ear wiggling and hopping. These reactions were absent in the females treated with KCl in this study as well as in others (*5*).

Facilitation of female mating responses by decortication is in marked contrast to the effect of decortication on male mating behavior. Large surgical lesions of neocortex in male rats abolish their mounting (*7*). Treatment with exogenous testosterone failed to restore this behavior pattern in the decorticate male. Sexually receptive females, if tested with another receptive female, often show mounting behavior. Decortication abolished this mounting pattern in the female (*8*). Thus, while mounting behavior is dependent upon cortical function in both sexes, elimination of the neocortex in the female enhances the frequency of lordosis. Whether this enhancement results from removal of some cortically involved inhibitory system or whether it renders the lordosis response mechanism(s) more sensitive to sexual stimuli, or both, is not clear.

Our finding that cortical application of KCl produces a facilitation of lordosis frequency in estrogen-treated spayed females suggests that progesterone may facilitate mating responses by suppressing some form of cortically involved inhibitory activity. The notion that progesterone facilitates lordosis by overcoming the influence of an inhibitory neural system has also been suggested by recent pharmacological studies (*9*). While the site of progesterone action is undefined, several studies show that cortical electrical activity is suppressed after progesterone treatment (*10*). On the other hand, progesterone has also been shown to lower the cortical arousal threshold to reticular formation stimulation in rabbits (*11*).

Cortical application of KCl has been shown to induce the release of adenohypophyseal lutenizing hormone (*12*). From these studies, Taleisnik has suggested that the cortex may inhibit hypophyseal gonadotrophin release, an inhibition which is removed by spreading depression induced by application of KCl. These findings suggest that the intimate relationship between sexual behavior and ovulation in the rat may be in part regulated by similar cortically involved mechanisms.

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