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Potassium Chloride–Induced Lordosis Behavior in Rats Is Mediated by the Adrenal Glands

Abstract. *Potassium chloride applied topically to the neocortex facilitates lordosis behavior in estrogen-primed ovariectomized rats. This effect is absent in rats that are also adrenalectomized. Potassium chloride fails to facilitate lordosis in estrogen-primed rats, with intact adrenals, which are pretreated with dexamethasone, an inhibitor of the release of adrenocorticotropic hormone. Adrenalectomized rats that have been treated with dexamethasone are capable of responding to progesterone with the display of lordosis. The results suggest that application of potassium chloride to the neocortex acts as a stressor that causes the release of adrenal progestin.*

During the past few years the hypothesis has been developed that the lordosis posture that characterizes sexual receptivity in the rat is normally under tonic neocortical inhibition and that gonadal hormones, progesterone in particular, function to reduce that inhibition (*1*). Consistent with this hypothesis are observations that the application of potassium chloride to the cerebral cortex and electrical stimulation of the cerebral cortex facilitate lordosis behavior in rats stimulated with estrogen (*2*). The present report gives evidence that the effects on lordosis behavior of the application of KCl are mediated by the adrenal gland and do not represent the effects of a functional neodecortication. Rather, the present studies suggest that the application of KCl to the neocortex results in the secretion of adrenocorticotropic hormone (ACTH) and adrenal hormones, presumably including progesterone.

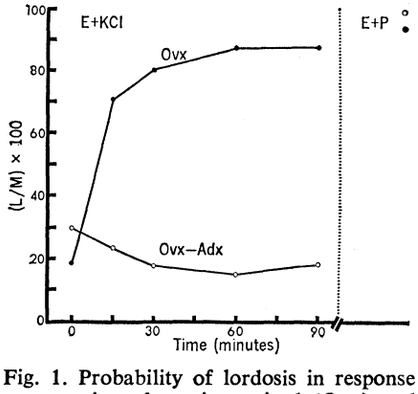

Fig. 1. Probability of lordosis in response to mounting of ovariectomized (Ovx) and ovariectomized-adrenalectomized (Ovx-Adx) female rats prior to time 0 and 15, 30, 60, and 90 minutes after the application of potassium chloride to the neocortex. The rats were estrogen primed at the time of treatment. The E + P (estrogen plus progesterone) test occurred 1 week after the KCl test and was preceded by estrogen and progesterone treatment.

In our studies mature Sprague-Dawley female rats were maintained in individual cages on a reversed light-dark cycle of 12 hours each with food and water freely available. Adrenalectomized rats were provided with 0.9 percent saline. All animals were ovariectomized and were implanted with bilateral 15-gauge aluminum cannulas fixed to the skull over the parietal-occipital cortex in the manner described by Russel and Ochs (*3*). The tip of each cannula rested on the dura.

Behavior tests consisted of placing each female with a sexually vigorous male. Testing began 2 to 3 hours after the onset of the dark phase of the lighting cycle. The male was allowed to mount the female ten times. The occurrence of each lordotic response was recorded and a lordosis quotient (L.Q.) was determined [L.Q. is (No. of lordosis responses/mounts with thrusting) × 100]. All females were screened for their response to estrogen alone by administering 5 μg of estradiol benzoate subcutaneously daily for 2 days, followed by behavioral testing on day 3. Rats that obtained an L.Q. score of 40 or higher on the screening test were eliminated.

In experiment 1 nine rats were ovariectomized and eight were ovariectomized and adrenalectomized. Testing began 2 to 3 weeks after surgery. Each rat was administered 5 μg of estradiol benzoate subcutaneously daily for 2 days. On day 3 they were given 500 μg of progesterone and were tested 3 hours later. One week later these animals were given estrogen daily on days 1 and 2; on day 3 they were tested for the display of lordosis behavior. Following this test the cannulas were filled with 15 percent KCl, and the animals were retested 15, 30, 60, and 90 minutes later. The next week the animals were again given estrogen and progesterone and were tested for lordosis behavior.

On the first test with estrogen and progesterone the L.Q. scores were 61.1

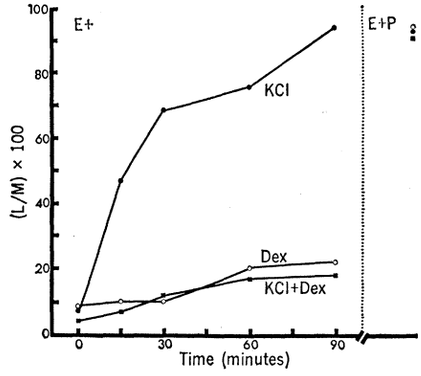

Fig. 2. Probability of lordosis in response to mounting of estrogen-primed ovariectomized rats. All animals were tested prior to treatment (time = 0). The KCl animals then had potassium chloride applied topically to the neocortex. The KCl + Dex animals received cortical application of KCl 1 hour after a subcutaneous injection of dexamethasone (Dex). The Dex animals received cortical application of saline 1 hour after a subcutaneous injection of dexamethasone. All animals were tested 15, 30, 60, and 90 minutes after either KCl or saline treatment. Following the final test all animals received progesterone and were retested 3 hours later.

for the ovariectomized rats and 82.5 for the ovariectomized-adrenalectomized ones. These scores were not significantly different.

Figure 1 shows the effects of KCl application; KCl applied to the neocortex facilitated lordosis behavior in the rats with intact adrenal glands, but not in the adrenalectomized rats. Analysis of variance revealed a significant effect of adrenalectomy ($F = 221.5$; d.f. = 1,88; and $P < .001$), a significant time of testing effect ($F = 34.2$; d.f. = 4,325; and $P < .001$), and a significant interaction ($F = 85.6$; d.f. = 4,352; and $P < .001$).

Lordosis behavior on the estrogen-progesterone test which followed the KCl test is also shown in Fig. 1. Both groups showed frequent lordosis responses and the two groups did not differ significantly in this regard.

The results of experiment 1 suggested that the KCl facilitation of lordosis is mediated by the adrenal gland, possibly by adrenal progesterone. This is consistent with the findings of Feder and Ruf and of Resko (*4*), who showed that ACTH can stimulate adrenal progesterone secretion and can induce receptive behavior in estrogen-primed rats. To obtain further confirmation of this possibility, experiment 2 was designed to examine KCl-induced lordosis behavior in rats in which ACTH secretion was blocked by the synthetic glucocorticoid dexamethasone. Female rats were ovariectomized, implanted with cannulas, and screened for their response to estrogen as in experiment 1. All received 2 μg of estradiol benzoate daily for 2 days. On day 3, 15 animals received a subcutaneous injection of 500 μg of dexamethasone (9α-fluro-11β,17α,21-trihydroxy-16α-methyl-1,4-pregnadiene-3,20-one) per kilogram of body weight dissolved in propylene glycol in a concentration of 1.25 mg/ml according to the procedures of Paris *et al.* (*5*); six control animals received an injection of propylene glycol. One hour later all animals were tested for lordosis behavior. Eight dexamethasone-treated and six vehicle-treated animals were then administered 15 percent KCl through the cannulas; seven dexamethasone-treated animals received saline through the cannulas at the same time. All animals were then retested for behavior 15, 30, 60, and 90 minutes later. Following the last test all animals were given a subcutaneous injection of 500 μg of progesterone and were tested again 3 hours later.

As is shown in Fig. 2, KCl facilitated lordosis in estrogen-primed rats, but not in those pretreated with dexamethasone. Dexamethasone by itself had little effect on lordosis behavior. Analyses of variance indicated that the groups differed significantly ($F = 29.6$; d.f. = 2,18; and $P < .001$), that the group treated with KCl plus dexamethasone did not differ from the dexamethasone group ($F < 1.0$, not significant), and that the KCl plus dexamethasone group was significantly lower than the group treated with KCl only ($F = 50.0$; d.f. = 1,18; and $P < .001$). On the final test, after progesterone treatment, all groups showed high levels of lordosis behavior, and the groups did not differ significantly from each other.

In contrast to previous findings (*2*), the KCl-treated animals in the present studies showed only very slight motor impairment. The results of these studies suggest that the KCl facilitation of lordosis behavior is mediated by adrenal gland secretions rather than by the induction of a suppression of some neocortical inhibitory system. The data thus require a reconsideration of the currently popular model which postulates that progesterone acts by releasing neocortical inhibition of lordosis behavior. Based on the present conclusion, the only compelling data for the possibility of a neocortical inhibitory system derive from Beach's observation (*6*) of exaggerated lordosis behavior in some female rats that had sustained neocortical lesions.

The most reasonable interpretation of the present findings would seem to be that topical application of KCl to the neocortex acts as a stressor causing the release of ACTH and of adrenal steroids. The most likely adrenal hormone to be active is progesterone, since neither corticosterone nor a wide variety of metabolites of progesterone are as effective as progesterone in inducing lordosis in estrogen-primed rats (*7*). Moreover, it is known that a variety of agents, such as reserpine and Metopirone, can induce lordosis in estrogen-primed rats and do so concomitant with a rise in plasma progesterone levels and that both the behavioral and secretory processes can be blocked by dexamethasone (*5*). Presumably, KCl treatment is acting in the same way.

The present findings do not negate the possibility that lordosis behavior is under neocortical inhibition. The data only cause one to raise questions about the interpretation of experiments regarded as providing support for that hypothesis.

RICHARD E. WHALEN
BLAKE L. NEUBAUER
BORIS B. GORZALKA
Department of Psychobiology,
University of California,
Irvine 92664

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Electronic and Catalytic Properties of Tungsten Carbide

Levy and Boudart (*1*) reported that tungsten carbide is much like platinum in its catalytic activity, whereas pure tungsten is not, and suggested that formation of the compound made the electron distribution of tungsten platinum-like. Subsequently, x-ray photoemission (*2*) and soft x-ray appearance potential (*3*) experiments on tungsten, platinum, and tungsten carbide were independently reported. Apparently conflicting conclusions were drawn in the reports of these experiments. Our purpose in this comment is to note that the two sets of experimental data are not inconsistent with one another and that they are both consistent with the essential point of Levy and Boudart's suggestion as we read it.

The photoemission study probed the occupied conduction bands of the three materials below the Fermi level, E_F. Because of transition probability weights, the *d*-like component of these bands is most heavily weighted in the spectra (*4*). The spectrum of tungsten carbide in the vicinity of E_F indicated a high density of *d*-like electron states at E_F, and in this respect the compound