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Effects of Dexamethasone, Corticosterone, and ACTH on Lordosis in Ovariectomized and Adrenalectomized-Ovariectomized Rats[1]

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DE CATANZARO, D. AND B. B. GORZALKA. *Effects of dexamethasone, corticosterone, and ACTH on lordosis in ovariectomized and adrenalectomized-ovariectomized rats.* PHARMAC. BIOCHEM. BEHAV. 12(2) 201–206, 1980.—The involvement of the pituitary-adrenocortical axis in the control of the lordosis reflex was investigated. In Experiment 1, estrogen-primed ovariectomized (ovx) and adrenalectomized-ovariectomized (adx-ovx) females were treated chronically with dexamethasone, a compound blocking ACTH release from the pituitary. Dexamethasone inhibited lordosis, effectively blocking an adrenalectomy-induced facilitation of the reflex. In Experiment 2, corticosterone was similarly administered chronically; this compound also inhibited lordosis in adx-ovx females. In Experiment 3, acute peripheral administration of synthetic ACTH caused a marked increase in lordosis in ovx females. The results suggest that in the adrenally intact animal, ACTH may exert its effect through adrenal steroids. An acute elevation of adrenal steroids may increase lordosis, whereas a chronic elevation may decrease it.

Dexamethasone Corticosterone ACTH Lordosis Adrenal Adrenalectomy Ovariectomy Female sexual behavior

THERE is a growing literature on the relationship of the pituitary-adrenocortical system to sexual behavior [12,15]. Some of the evidence supporting such a relationship comes from studies comparing adrenalectomized and non-adrenalectomized rats. One study [8] reports that adrenalectomized-ovariectomized (adx-ovx) females show substantially higher levels of lordosis behavior after estrogen priming, and when presented to sexually active males, than do adrenally-intact ovariectomized (ovx) females. Some additional studies [6, 9, 14], although not extensively comparing adx-ovx and ovx females, contain indications corroborating a facilitation of lordosis by adrenalectomy. The mechanism underlying this effect is unknown. One possible explanation is that the sustained presence of some adrenal steroids or catecholamines could in some manner inhibit lordosis in ovx females, whereas the absence of adrenal hormones might produce a disinhibition in adx-ovx females. An alternative, but possible related, explanation is that higher levels of endogenous adrenocorticotropic hormone (ACTH), which accompany adrenalectomy [5,11], might facilitate lordosis through extra-adrenal action.

There is also some evidence that exogenous ACTH may affect female receptivity. Acute peripheral administration of this hormone to estrogen-primed ovx rats facilitates lordosis [10]. This facilitation of lordosis is temporally correlated with a release of steroids from the adrenal cortex. This temporal correlation, however, does not prove a causal link between adrenal steroid levels and the facilitation of lordosis by ACTH. In a recent review of the literature on ACTH [7] it was argued that most of the behavioral effects of this peptide result from its direct action on the brain. Furthermore, there is evidence that acute administration of ACTH directly into the ventricular system of the brain facilitates lordosis in female rabbits [2]. Thus, research is required to clarify whether facilitation of lordosis in rats through acute peripheral ACTH administration relates to adrenal or extra-adrenal effects of the hormone.

To shed further light on these issues, the present series of experiments examined the relationship between pituitary-adrenocortical hormones and sexual receptivity in both ovx and adx-ovx female rats. These experiments involved administration of dexamethasone, a synthetic steroid that blocks ACTH release; corticosterone, the major adrenal steroid in the species; and cosyntropin zinc hydroxide, a synthetic ACTH preparation. The comparison of effects of these compounds in ovx and adx-ovx females should provide informa-

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tion about the mediation of adrenalectomy-facilitated lordosis, adrenal steroid effects on lordosis, and adrenal versus extra-adrenal effects of ACTH.

EXPERIMENT 1

The synthetic steroid, dexamethasone, is known to have powerful effects on hypothalamic and pituitary glucocorticoid receptors, markedly reducing pituitary output and plasma levels of ACTH [17,19]. It thus provides a useful tool for examination of pituitary-adrenal involvement in behavior. For example, if ACTH is involved in the control of estrogen-induced lordosis, facilitating this behavior, the administration of dexamethasone might reduce lordosis quotients. Furthermore, if elevation of ACTH is responsible for facilitation of lordosis by adrenalectomy, dexamethasone might produce in lordosis in adx-ovx females through extra-adrenal mechanisms. Alternatively, since blocking ACTH release also blocks the output of adrenal steroids, dexamethasone could effect a functional adrenalectomy. Accordingly, if an absence of adrenal steroids accounts for adrenalectomy-facilitated lordosis, dexamethasone might raise the performance of ovx females but not affect adx-ovx females.

There have been a few previous studies of the effects of dexamethasone on lordosis, but these have yielded conflicting and incomplete results. One study by Lisk and Reuter [17] reports a significant facilitating effect of chronic dexamethasone treatment on lordosis in estrogen-primed ovx females. However, the baseline level of performance in their study was low and the facilitation was quite small. Another study [3] indicates that an acute dose of dexamethasone may prevent a brief period of estrus occurring in recently ovx females, this estrus being attributable to ACTH-induced release of adrenal sex steroids. This latter study also found that acute dexamethasone may delay but not prevent estrus when administered in proestrus to intact females. In the present experiment, both adx-ovx and ovx females were examined after estrogen priming and chronic treatment with dexamethasone.

METHOD

Subjects and Surgery

Forty-eight Sprague-Dawley female rats were obtained from Canadian Breeding Farms, Montreal, at 70 days of age. At about 80 days of age, 26 of these females were bilaterally adrenalectomized and overiectomized and the remainder bilaterally ovariectomized under 40 mg/kg sodium pentobarbital (Nembutal, Abbott) anaesthetic supplemented where necessary with ether. All surgery was performed via bilateral lumbar incisions. Adx-ovx animals were given continuous access to 0.9% saline solution in lieu of water immediately following surgery and throughout the course of the experiment. Animals were housed in groups of 5–7 in triple wiremesh cages in a room maintained under a reversed 12-hr dark/12 hr light cycle at 21 ± 1°C. Four adx-ovx animals died prior to the first test of behavior. Body weights of females were within a range of 205–225 g at the time of the first estrogen injection.

Procedure

The first test of lordosis behavior occurred 3 weeks following surgery. One week, 48 hr, and 24 hr prior to testing, each female was given 10 μg estradiol benzoate in 0.05 cc peanut oil SC. Half of each of the ovx and adx-ovx animals were also each given 0.5 mg/kg dexamethasone (Steraloids) dissolved 2 mg/ml in propylene glycol, SC daily for 5 consecutive days, with the last injection occurring 1 hr prior to behavioral testing. The remaining, control ovx and adx-ovx females each received the propylene glycol vehicle alone according to the same schedule. This dexamethasone injection regimen is identical to that employed by Lisk and Reuter [17]. Beginning 3 weeks following lordosis testing, this whole procedure was repeated with the same subjects; however, the daily dose of dexamethasone was raised to 2 mg/kg in a 8 mg/ml propylene glycol solution.

Lordosis testing

Behavioral testing involved presentation of females individually to stud male rats in cylindrical pyrex testing jars measuring 45 cm high with a diameter of 29 cm. Stud males were given brief access to fully receptive females (each given 10 μg estradiol benzoate 48 hr and 500 μg progesterone 6 hr before presentation) just prior to sessions with experimental females. Each experimental female was placed with a single male until ten mounts accompanied by pelvic thrusting occurred. If a male did not mount, the female was placed in another jar containing a different male. The female's response to a mount was categorized as either a lordosis response, consisting of a full arching of the back [13], or no lordosis, consisting of any other response or no response. A lordosis quotient was calculated as the percentage of mounts resulting in a lordosis response.

RESULTS AND DISCUSSION

Table 1 presents the results of lordosis tests. Among animals treated with the vehicle alone, adx-ovx animals showed higher lordosis quotients than did ovx animals. Lordosis quotients were slightly reduced in both adx-ovx and ovx animals by the 0.5 mg/kg dexamethasone dose. Lordosis was very frequent in all animals treated with 2 mg/kg dexamethasone; most of these animals showed lordosis quotients of zero. A 2×2×2 analysis of variance, treating the repeated measure at a different dose as a within subjects factor, showed a significant surgery (adx-ovx vs ovx) by drug (dexamethasone vs vehicle) interaction $F(1,37)=6.96$, $p=0.012$. Subsequent Newman Keuls comparisons ($p<0.05$) examining this interaction indicated that the adx-ovx vehicle group exceeded all other groups, and that these other groups did not differ from one another. There were also main effects in the analysis of variance of surgery, $F(1,37)=11.30$, $p=0.002$; drug, $F(1,37)=13.25$, $p=0.001$; and dose regimen, $F(1,37)=8.20$, $p=0.007$.

The present results confirm previous findings [8] that adrenalectomy facilitates lordosis in estrogen-primed ovx females. Dexamethasone apparently failed to effect a functional adrenalectomy in ovx females, since it did not raise performance in ovx females to levels seen in untreated adx-ovx females. Rather, it almost eliminated lordosis behavior in adx-ovx females and produced a small reduction in lordosis in ovx females.

EXPERIMENT 2

The results of Experiment 1 indicate that a synthetic corticosteroid may suppress lordosis behavior in estrogen-primed females. Furthermore, they demonstrate that females with normal levels of endogenous corticosteroids (adrenally

TABLE 1

MEAN LORDOSIS QUOTIENTS (± SE) FOR ESTROGEN-PRIMED OVX AND ADX-OVX FEMALES GIVEN DEXAMETHASONE OR PROPYLENE GLYCOL VEHICLE IN EXPERIMENT 1

Treatment	Dose 0.5 mg/kg or vehicle for 5 days	Dose 2 mg/kg or vehicle for 5 days
Ovx-Vehicle	27.27 ± 11.21	18.18 ± 6.30
Ovx-Dexamethasone	22.00 ± 7.72	11.00 ± 6.57
Adx-Ovx-Vehicle	67.00 ± 7.62	53.00 ± 9.77
Adx-Ovx-Dexamethasone	36.00 ± 10.88	6.00 ± 3.38

TABLE 2

MEAN LORDOSIS QUOTIENTS (± SE) FOR ESTROGEN-PRIMED OVX AND ADX-OVX FEMALES GIVEN CORTICOSTERONE OR OIL VEHICLE IN EXPERIMENT 2

Treatment	Dose 2 mg/kg or vehicle for 5 days	Dose 4 mg/kg or vehicle for 9 days
Ovx-Vehicle	15.83 ± 6.79	21.67 ± 7.37
Ovx-Corticosterone	11.82 ± 6.44	30.91 ± 6.80
Adx-Ovx Vehicle	42.31 ± 9.48	45.46 ± 9.94
Adx-Ovx Corticosterone	20.77 ± 6.65	28.33 ± 7.67

intact ovx females) show lower levels of lordosis than those with the source of corticosteroids removed (adx-ovx females). These two findings, taken together, suggest a hypothesis that natural corticosteroids can suppress estrogen-induced lordosis. In the present experiment, both adx-ovx and ovx females were treated with corticosterone, the major adrenal steroid in this species, to examine this hypothesis.

METHOD

Initially, 48 females were prepared as described for the previous experiment, half adx-ovx and half ovx. Also according to the procedures of Experiment 1, all females were primed with estradiol benzoate before testing for lordosis. Half of the adx-ovx and half of the ovx females were given daily SC injections of 2 mg/kg corticosterone (Steraloids) dissolved 10 mg/ml in peanut oil, heated prior to injection to improve solubility. As in Experiment 1, injections were given for 5 days with the final injection occurring 1 hr prior to lordosis testing. The remaining adx-ovx and ovx females each received daily injections of the oil vehicle alone according to the same procedure. Subsequently, this procedure was repeated with a different set of females, similarly prepared for adx-ovx and ovx conditions and primed with estradiol benzoate. However, here the corticosterone dose was raised to 4 mg/kg dissolved 10 mg/ml in oil, and the injections were given for 9 successive days, with the last injection occurring 1 hr prior to testing. Lordosis testing in this experiment followed the procedures described for Experiment 1. Body weights of females were within a range of 210–235 g at the time of the first estrogen injection. There were 10–13 subjects in each treatment combination.

RESULTS AND DISCUSSION

Table 2 presents lordosis quotients from all tests. Under both dose regimens the lordosis quotients were highest in adx-ovx females tested with the oil vehicle. Both doses of corticosterone reduced the level of lordosis in adx-ovx females, but not to the level shown by ovx vehicle females. This reduction in lordosis in adx-ovx females with corticosterone treatment was similar under the two dose regimens. Corticosterone did not substantially alter the performance of ovx females. A 2×2×2 analysis of variance indicated that there was a significant interaction between the corticosterone vs oil factor and the adx-ovx vs ovx factor, $F(1,86)=4.64$, $p=0.032$. Subsequent Newman-Keuls comparisons ($p<0.05$) indicated that the adx-ovx oil vehicle scores exceeded those of all other treatment groups, which in turn did not differ from one another. There was also a significant main effect in the adx-ovx vs ovx factor, $F(1,86)=5.93$, $p=0.016$, whereas no other effects were significant.

These data suggest, then, that corticosterone treatment lowers the lordosis quotients of adx-ovx females, returning their performance to the level observed in adrenally intact females. It is possible that adrenalectomy facilitates lordosis because it removes adrenal steroids, which may have a small inhibiting effect on estrogen-induced lordosis.

EXPERIMENT 3

The results of Experiments 1 and 2 suggest that adrenal steroids can inhibit estrogen-induced lordosis, and that removal of the primary source of such steroids, the adrenal, facilitates lordosis. Adrenalectomy produces a chronic increase in endogenous ACTH levels [5,11] and, as indicated above, facilitates lordosis in estrogen-primed ovx females. Experiment 1 indicates that dexamethasone, a powerful inhibitor of ACTH release [17,19], suppresses lordosis and blocks a facilitation of lordosis by adrenalectomy. Experiment 2 demonstrates that corticosterone, which also inhibits ACTH release [11,17], produces a moderate suppression of lordosis in adx-ovx females. Thus, in the absence of the adrenal, high endogenous levels of ACTH are associated with high levels of lordosis behavior.

Feder and Ruf [10] reported that acute administration of ACTH facilitates lordosis in ovx, estrogen-primed females, an effect that they attributed to stimulated release of adrenal progesterone. However, because the present results suggest the possibility of extra-adrenal effects of ACTH on lordosis, there is a need for clarification of peripheral ACTH effects. Although Feder and Ruf also assayed progesterone levels, finding them elevated in ACTH treated females, this does not prove a causal link between adrenal progesterone levels and the facilitation of lordosis by ACTH. An hypothesis that ACTH facilitates lordosis through some extra-adrenal mechanism remains consistent with the available data. The present study was an attempt to replicate the study of Feder and Ruf, but expanded on their design by including a group of adx-ovx females as well as ovx females. If peripheral ACTH administration facilitates lordosis through adrenal action, an effect of this hormone should be absent in adx-ovx females. The present study also involved examination of lordosis at a 7 hr as well as a 2 hr interval following acute ACTH, and also after chronic administration of the hormone.

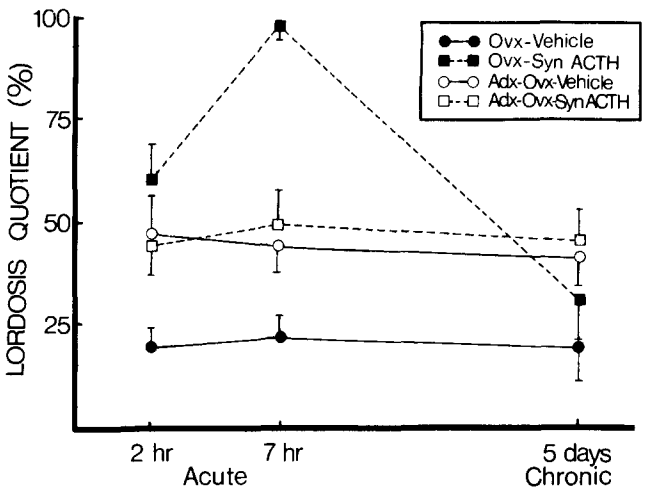

FIG. 1. Mean Lordosis Quotients (± SE) for estrogen primed ovx and adx-ovx females given synthetic ACTH (cosyntropin zinc hydroxide) or saline in Experiment 3. Lordosis was tested at 2 and 7 hr after an acute dose and after a chronic treatment of 5 successive daily doses. Time points are on a logarithmic scale.

METHOD

Females were obtained, prepared for adx-ovx and ovx conditions, primed with estradiol benzoate, and maintained according to the procedures of Experiment 1. About half of both the adx-ovx and ovx females were additionally given 0.5 mg/kg of the synthetic ACTH preparation, cosyntropin zinc hydroxide (Synacthen Depot, CIBA), SC 2 hr prior to testing. This dose was identical to the dose of synthetic ACTH used by Feder and Ruf [10]. The remaining females were given the equivalent amount of saline vehicle. Lordosis was scored as described for Experiment 1. Five hr after the first test (7 hr after ACTH or saline injection), lordosis behavior was measured again. Every 24 hr following the first ACTH injection, the ACTH or saline vehicle injection was repeated until the hormone had been given for 5 consecutive days. Two hr following the last injection, lordosis behavior was again tested. Animals were also each given another 10 μg estradiol benzoate 48 hr prior to this last test of lordosis. There were 12 adx-ovx and 12 ovx vehicle, and 11 adx-ovx and 11 ovx ACTH females. Body weights at time of the first estrogen injection ranged from 205–220 g.

RESULTS AND DISCUSSION

Figure 1 gives the results for all tests of lordosis in Experiment 3. Adx-ovx ACTH-treated and vehicle-treated females consistently showed higher levels of performance than did ovx vehicle females. Ovx ACTH females showed substantially higher levels of performance than did all other groups following acute administration of ACTH. At the 7-hr interval, all but two of the animals from the ovx ACTH condition showed lordosis quotients of 100%, with these two showing quotients of 90%. Chronic administration of the ACTH compound, however, did not appear to facilitate performance in ovx females. Individual analyses of variance were conducted at each time point. At the 2-hr acute dose there was a significant drug by surgery interaction, $F(1,42)=8.32$, $p=0.006$. Subsequent Newman-Keuls comparisons ($p<0.05$) indicated that the ovx ACTH group significantly exceeded the ovx vehicle group. At the 7-hr interval there was also a significant interaction, $F(1,42)=30.64$, $p<0.001$. Newman-Keuls comparisons indicated that the ovx ACTH group exceeded both the adx-ovx groups, which in turn exceeded the ovx vehicle group. However, with the chronic dose of ACTH, none of the differences reached significance.

These results are consistent with the finding of Feder and Ruf [10] that acute ACTH administration induces behavioral estrus in ovx, estrogen-primed females. The absence of a similar effect in adx-ovx females strengthens their conclusion that these effects relate to adrenal, rather than extra-adrenal, effects of ACTH. The present results also extend previous findings by indicating that the effect is stronger 7 hr after acute administration of the compound and by indicating an absence of this effect after chronic administration.

GENERAL DISCUSSION

This series of experiments confirms the finding that adrenalectomy facilitates lordosis [8] in estrogen-primed ovx rats and that ACTH can play a role in receptivity of adrenally-intact females through its effects on adrenal steroids [10]. The results also demonstrate that the artificial steroid, dexamethasone, may strongly suppress and the natural corticosteroid, corticosterone, may mildly suppress lordosis under chronic administration. These last effects are most pronounced in adrenalectomized females, which lack adrenal steroids and show high baseline levels of lordosis.

Adrenalectomy-facilitated lordosis would appear to be a fairly robust phenomenon occurring under fairly limited hormonal conditions. Whereas only one previous study [8] reports a strong effect, others [6,9] report a consistent trend in this direction, while elsewhere [16] it is reported that several adx-ovx females were eliminated from a pharmacological study because they showed high levels of lordosis with estrogen alone. The one study [21] reporting apparent deficits in estrogen-induced lordosis following adrenalectomy also indicates substantial regeneration of adrenocortical tissue upon histology and employed only a few subjects. Facilitation of lordosis by adrenalectomy in estrogen-primed ovx females may not be evident when progesterone is also given [23], perhaps because progesterone exerts powerful effects on lordosis that mask adrenalectomy effects. Another limited exception is that adx-ovx females may show deficits in lordosis relative to ovx females when the surgery is performed during proestrus and lordosis measured shortly thereafter [20], an effect that may be attributable to acute effects of adrenal steroids.

To the extent that dexamethasone specifically affects release of ACTH from the pituitary, Experiment 1 could indicate that adrenalectomy facilitates lordosis because it raises ACTH. Despite the fact that dexamethasone is often used to effect a functional adrenalectomy, its action was to decrease lordosis, rather than to increase it as does adrenalectomy. If dexamethasone had induced a functional adrenalectomy, it might have increased lordosis in ovx females to the levels found in adx-ovx females. However, dexamethasone blocked lordosis in adx-ovx females and had relatively little effect on ovx females. This may suggest that adrenalectomy facilitates lordosis not because it removes direct effects of adrenal steroids, but rather because it blocks elevation of ACTH by adrenalectomy.

There is, however, some evidence suggesting that effects of dexamethasone may not be specific to ACTH output. A study [17] of (^{3}H) estradiol retention in the uterus, vagina, and pituitary of the rat found that dexamethasone blocked such retention. If this action of dexamethasone extends to brain tissue, it would provide an alternative explanation for the results of Experiment 1, since decreased estradiol uptake could reduce induction of receptivity by this steroid. Similarly, dexamethasone might have effects on natural glucocorticoid receptors other than those regulating ACTH release. The chronic stimulation of such glucocorticoid receptors may through unknown mechanisms act to inhibit lordosis.

The action of corticosterone observed in Experiment 2 is similar to that of dexamethasone, albeit less powerful, suggesting that the synthetic steroid produces its effect by mimicking natural adrenal steroids. These results furthermore implicate adrenal steroids in differences between adx-ovx and ovx females. Chronic administration of corticosterone appears to reverse the effect of adrenalectomy on lordosis. This reversal may not be complete, unlike that produced by dexamethasone. This suggests that other adrenal steroids, such as progesterone or desoxycorticosterone, are also involved. Indeed, although an acute release of progesterone facilitates lordosis, chronic progesterone may actually suppress it [18]. Also, desoxycorticosterone may share the behavioral effects of progesterone [14], and has some affinity for estrogen target tissues [22] as does dexamethasone [17]. Thus, the full difference in lordosis quotients between adx-ovx and ovx females might be accounted for by additive effects of several adrenal steroids, whose chronic presence may produce a relative suppression of lordosis in ovx females.

The results of Experiment 3 indicate that acute peripheral administration of ACTH facilitates lordosis in ovx but not adx-ovx females. This strongly suggests that the adrenal mediates the effect of peripheral ACTH on lordosis, supporting the interpretation [10] that an acute, ACTH-induced elevation of adrenal steroids increases lordosis. Nevertheless, these data do not rule out the possibility that ACTH, through extra-adrenal action, mediates the effects of both adrenalectomy and chronic corticosteroid administration on lordosis. Recent evidence suggests that peripherally-administered ACTH may not reach the brain because of blood-brain barrier mechanisms [1,7]. However, endogenous ACTH may still act directly on the brain, perhaps through some sort of retrograde transport of the hormone from the pituitary to the ventricular system [1,4]. It remains possible that chronic elevation of ACTH mediates adrenalectomy-facilitated lordosis and that both dexamethasone and corticosterone suppress lordosis in adx-ovx females by eliminating this chronic elevation.

In Experiment 3, a chronic peripheral administration of ACTH, unlike the acute administration, did not facilitate lordosis in ovx females. Rather, ovx females chronically treated with ACTH showed quite low levels of lordosis. This occurred despite the fact that the last administration of ACTH was 2 hr prior to the chronic test, whereas a simple acute injection of ACTH 2 hr prior to testing produced a marked facilitation of lordosis. Chronic ACTH administration may cause chronic elevation of adrenal steroids, which, as discussed above, may suppress lordosis. Thus, the data of Experiment 3 further support the conclusion that the chronic presence of adrenal steroids inhibits lordosis, while an acute elevation of these steroids facilitates it.

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