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ROLE OF STRIATAL DOPAMINE IN THE LORDOSIS BEHAVIOUR

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Abstract: Effects of intrastriatal injections of haloperidol (Dopamine antagonist) and D-amphetamine (Dopamine agonist) on lordosis behaviour were studied in ovariectomized female albino rats, after priming with subcutaneous injections of estrogen and progesterone.

The lordosis quotient (LQ) significantly increased after haloperidol, and decreased following D-amphetamine treatment. However, the inhibitory effect of D-amphetamine was transient and could be reversed by haloperidol in the same animal when given one hour after the D-amphetamine injection.

The ovarian hormones probably act centrally to supress the DA system in the striatum thereby enhancing the tonic and dorsal immobility responses associated with lordosis.

Key words : haloperidol D-amphetamine lordosis quotient

INTRODUCTION

Involvement of the neural mechanisms of the limbic system in both the facilitatory and inhibitory control of sexual receptivity is established (1-5). The striatum on the other hand is attributed mainly with the planning and programming of the voluntary movements (6) and little is known of its role in the sexual behaviour. Intrastriatal application of 6-hydroxydopamine (6-OHDA) did not cause a reliable change in the lordosis behaviour (7) whereas a dramatic increase in the intensity and frequency of lordotic response was observed when 6-OHDA was injected intraventricularly (8). Earlier reports from this laboratory revealed that, discrete bilateral electrolytic lesions in the ventral anterior striatum decreased the lordotic response in female rats (9) and potentiation of sexual activity occurred following similar liasons in the anterodorsal caudate in male rats (10). A number of neurotransmitters are located in this area (11-13). The electrolytic lesions could have interfered with any one or more of these to produce the observed effects, one such transmitter could have been dopamine. The present study was therefore designed to study whether this was infact involved in the modulation of lordotic response in female rats, through its action in the ventral anterior striatum.

METHODS

Forty four female albino rats 90 to 100 days old and weighing 150 to 200 gms were selected for this study and ovariectomized. They were housed individually in polypropelene cages with free access to food (pellets) and water. They were maintained on reverse 14/10 hours light/dark cycle and the testing was done during the dark period.

Bilateral intracerebral cannulae (made from 22 gauge hypodermic needles cut to size) were implanted and fixed with dental cement under ether anesthesia. The implantation sites were reached by using the

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stereotaxic coordinates of A 8 mm; L 1·5 mm Depth from the skull 5 mm, to reach the ventral anterior part of the caudate (14). All the animals were given four days rest for recovery.

The sexual receptivity was determined by the lordosis quotient (LQ) (15) after suitable priming with estradiol benzoate (EB) and progesterone (P).

After recovery from surgery (ovariectomy and intracerebral implantation of the cannulae), the rats were randomly sorted into three groups.

Group I (10 rats) were primed by subcutaneous injections of estradiol benzoate (2×8 µg/kg) to support only low levels of lordosis response (8). LQ was determined before and after intracerebral instillation of 20 µg haloperidol a DA receptor blocker (8) dissolved in absolute alcohol. Similar procedure was followed in 7 sham animals except that equal volume of absolute alcohol (solvent) was injected.

Group II (10 rats) were primed maximally by two subcutanesous injections of 100 µg/kg estadiol benzoate at 48 and 24 hr and a single injection of 500 µg/animal of progesterone at 5 h prior to testing (8). These rats were treated with intracerebral injections of 20 µg D-amphetamine a DA agonist (8) dissolved in distilled water. The seven sham rats after similar priming were treated with equal volume of distilled water (solvent) instead of D-amphetamine. The LQ was determined pre and post intracerebral instillation in both the experimental and sham animals.

Group III (rats) were primed maximally as in Group II tested for LQ and then given intracerebral injection of 20 µg D-amphetamine and LQ determined. One hour later 20 µg haloperidol was injected in the same animals and LQ determined once again.

In all the three groups, each animal was tested pre and post intracerebral injections on two occasions at week's interval after which electrolytic lesions were made at the site of injection. The animals were then sacrificed and the brain sites studied histologically (9).

The data on LQ was analysed using the student's "t" test. In Group I and II, the comparison was of LQ before and after the intracaudatal injections in experimental animals and sham treated animals separately. In Group III the significance was derived between the LQ determined after D-amphetamine injection and after haloperidol injection given one hour later in the same animals.

RESULTS AND DISCUSSION

The results as seen in Table I reveal that the lordosis quotient increased after the intracaudatal

TABLE I : Mean±SD Lordosis quotient determined before and after intrastriatal injections of haloperidol or/and D-amphetamine in ovariectomized female rats primed with suitable doses of estrogen and progesterone.

Group	Intrastriatal injection	LQ values (mean of 2 tests) Preinjection (A)	Post injection (B)	Difference of LQ (A and B)
I	Experimental (n = 10)	21.78	77.85	56.07*
	(Haloperidol)	±4.64	±7.77	
	Sham (n = 7)	20.60	24.00	3.40 (NS)
	(abs. alcohol)	±8.73	±6.88	
II	Experimental (n = 10)	96.46	50.71	45.75*
	(D-amphetamine)	±6.33	±9.50	
	Sham (n = 7)	97.00	96.15	0.85 (NS)
	(Dist. water)	±4.21	±6.51	
III	Experimental (n = 10)	92.85		Diff. of LQ (A and Bi)
	i) D-amphetamine	±6.11	i) 51.12	41.73*
	ii) Haloperidol		±7.55	Diff. of LQ (Bi and ii)
	1 hr after (i)		ii) 85.71	34.59*
			±9.30	

The LQ difference between Experimental and Sham animals intragroup (I & II) in preinjection trials NS-level of significance
*P<0.01; NS = not significant.

injection of haloperidol (DA receptor blocker) and decreased following D-amphetamine (DA agonist) injection as compared to the pre-injection values in the same animals in each group (I and II) and both these effects were statistically significant. In Group III, the inhibitory effect of D-amphetamine on LQ reversed following haloperidol injection given one hour after, to the same animals. The difference in LQ before and after intracaudatal instillation of same volume of absolute alcohol (sham animals in Group I) and distilled water (Sham animals in Group II) was not significant thus eliminating the effect of the procedure itself and/or of the solvent used for the dilution of the chemicals.

Lordosis behaviour is a sequence of sensorimotor reflexes, and from the results it appears that the dopamine (DA) in the striatum modulated by the ovarian hormones, has a role to play in this lordotic response in female rats. Earlier workers in the field have suggested a similar role for the DA systems in the forebrain, Smith et al (16) reported that the hormonal condition during estrus as well as the estrogen progesterone treatment in ovariectomized female rats significantly potentiated three immobility responses viz. lordosis, tonic immobility, and the dorsal immobility response and suggested that the ovarian hormones act through a common mechanism shared by all three responses. The DA Systems of the forebrain involved in the broader aspects of the sensorimotor function could be a component of such a mechanism. Selective destruction of DA Systems with 6OHDA (8), or blockade with dopamine antagonists (17), facilitated lordosis behaviour and DA receptor agonists blocked lordosis (18). These results concur with our present work. Further the suppresion of the DA Systems result in increased immobility responses and evidence is accummulating that acute exposure to estrogen suppresses behaviours mediated by DA systems (19, 20). That dopamine in the ventral anterior striatum is probably the neurotransmitter involved is supported by our study wherein haloperidol a potent and specific DA receptor blocker greatly facilitated the lordosis behaviour in ovariectomized female rats given low doses of estrogen.

Thus the increase in both the tonic and dorsal immobility and lordosis comprising the total behaviour, noted during estrus and in primed ovariectomized rats is probably due to the suppression of the DA systems by the ovarian hormones thereby decreasing the striatal dopamine activity and enhancing the immobility responses associated with lordosis.

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