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Role of Hypothalamic Alpha and Beta Adrenergic Receptors in the Control of Lordotic Behavior in the Ovariectomized-Estrogen Primed Rat[1]

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FOREMAN, M. M. AND R. L. MOSS. *Role of hypothalamic alpha and beta adrenergic receptors in the control of lordotic behavior in the ovariectomized-estrogen primed rat.* PHARMAC. BIOCHEM. BEHAV. 9(2) 235–241, 1978.—The role of hypothalamic α- and β-adrenergic receptors in the control of lordotic behavior was studied by infusing α and β receptor stimulants and blockers into either the medial preoptic area (MPOA), arcuate-ventromedial area (ARC-VM) or lateral hypothalamic area (LHA) in ovariectomized (OVX), estrone primed rats. In the first experiment, OVX rats were primed with low doses of estrone (100–250 μg) in order to maintain low preinfusion receptivity (mean preinfusion L/M=0.169). The infusion of α receptor blockers, phentolamine and phenoxybenzamine; β receptor stimulants, isoproterenol; dopamine, norepinephrine; or epinephrine into either the MPOA or ARC-VM produced increases in lordotic behavior in OVX rats primed with low doses of estrone. Using the same protocol, MPOA or ARC-VM infusions of methoxamine, an α receptor stimulant, or propranolol, a β receptor blocker, produced decreases in the lordotic behavior. Infusions of any of these agents into the LHA had no effect upon sexual receptivity. In order to corroborate the pharmacological effects observed with low preinfusion receptivity, a second experiment was conducted in which high preinfusion receptivity (mean preinfusion L/M=0.898) was maintained by priming the OVX rats with higher doses of estrone. Using this second protocol, methoxamine and propranolol infusions into the MPOA or ARC-VM depressed lordotic response. However, further enhancement by α receptor blockade or β receptor stimulation from the initially near maximal lordotic response could not be obtained. A third experiment was designed to evaluate the effects of adrenergic receptor activity on the stimulatory effects of luteinizing hormone-releasing hormone (LRH) upon lordotic behavior. This protocol allowed comparisons among lordotic responses to MPOA and ARC-VM infusions of vehicle, LRH, LRH with methoxamine and LRH with propranolol. Infusions of LRH into the MPOA or ARC-VM significantly enhanced lordotic behavior, whereas the addition of either methoxamine or propranolol to LRH infusates abolished this response. Alternative mechanisms were proposed for the possible roles of α- and β-adrenergic receptors in the hypothalamic control of estrogen-induced and LRH-facilitated lordotic behavior.

Lordosis Ovariectomy Estrogen α- and β-adrenergic receptors

PREVIOUS investigations have proposed prominent roles for adrenergic neurons in the hypothalamic control of gonadotropin release and sexual behavior [1, 6, 19]. In support of these proposals, neurohistological studies have demonstrated synaptic terminals for norepinephrine (NE) and epinephrine (E) containing neurons within the preoptic area (POA) and medial basal hypothalamus (MBH) [2, 9, 11, 33]. This midline band of neural tissue stretching from the POA to MBH has been shown through lesion, stimulation and steroid implantation studies to have a regulatory role in gonadotropin release and lordotic behavior [3, 12, 13, 14, 18, 23, 25, 27, 32]. Indeed, autoradiographic studies have found estrogen and progestin concentrating neurons within this midline continuum [10,26].

Although it is generally accepted that catecholamines can influence neuronal activity within this POA-MBH regulatory continuum, the relative importance of these influences upon neuroendocrine and behavioral changes has yet to be determined. The stimulation of adrenergic receptors with third ventricular infusion of NE and E have been shown to be either facilitatory or ineffective in eliciting gonadotropin release [15, 29, 34]. Recent behavioral studies, in contrast to

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the studies measuring gonadotropin secretion, have proposed primarily inhibitory effects of NE and E. These latter studies have described elevations in sexual behavior following the inhibition of tyrosine hydroxylase and dopamine β-hydroxylase and following blockade of α- and β-adrenergic receptors [4, 6, 35, 36]. However, stimulatory effects of NE have also been proposed based upon elevations in sexual receptivity following amphetamine administration in pimozide blocked rats [6].

The purpose of the present study was to evaluate the roles of α- and β-adrenergic receptors in the hypothalamic mediation of estrogen-induced and luteinizing hormone releasing hormone (LRH)-facilitated mating behavior.

GENERAL METHOD

One hundred-one female Wistar albino rats (250–275 g) and 50 Sprague-Dawley male albino rats (350–450 g) (Simonsen Laboratories, Gilroy, California) were used in these studies. All rats were housed in a temperature controlled room with a modified day-night schedule of 14 hr of light and 10 hr of dark (dark cycle beginning at 2 p.m.). All animals were given Purina Rat Chow and water *ad lib*. Behavioral testings were performed between 3 and 7 p.m.

All female rats were ovariectomized and implanted with a unilateral stainless steel cannula [8] in either the medial preoptic area (MPOA, N=44), arcuate-ventromedial area (ARC-VM, N=44) or lateral hypothalamic area (LHA, N=13). A minimum of 1 week postoperative recovery was allowed for both ovariectomy and cannulation. The DeGroot coordinates for the cannulation sites were as follows: MPOA: AP 7.8 mm, H 1.0 mm, depth 8.0 mm; ARC-VM: AP 5.9 mm, H 0.5 mm, depth 9.0 mm; LHA: AP 7.0 mm, H 2.5 mm, depth 7.0 mm (AP=anterior-posterior, H=horizontal and depth=depth from skull surface; see Fig. 1) [5].

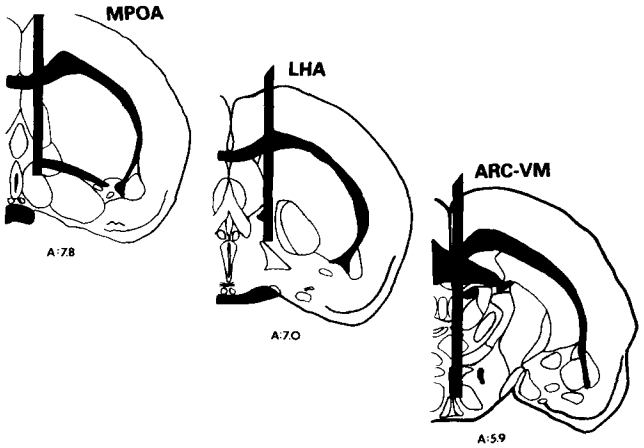

FIG. 1. Frontal section representation of cannulation sites. The following abbreviations were used: MPOA, medial preoptic area; ARC-VM, arcuate-ventro medial area; LHA, lateral hypothalamic area. See test for procedural details of DeGroot coordinates.

In order to ensure that the cannulation procedures did not disrupt subsequent mating behavior, each OVX-cannulated rat was tested for sexual receptivity following priming with estrone (250 μg Theelin in oil, 48 hr prior to mating) and progesterone (2.5 mg Lipolutin, 6 hr prior to mating). Only animals which achieved an arbitrary minimum lordosis to mount (L/M) ratio of .85 for 2 successive weekly estrogen-progestin trials were used for pharmacological testing.

All infusions consisted of a constant 0.5 μl volume delivered over 60 sec under light ether anesthesia. The infusion apparatus consisted of a Stoelting microinjection apparatus (Model SA-1138) and a 5 μl Hamilton syringe connected with a 30 ga stainless steel injection needle via a 23 ga polyethylene tubing.

Alterations in lordotic behavior were evaluated by comparing the changes in L/M from preinfusion to postinfusion mating (ΔL/M) for both vehicle (0.9% NaCl, 5.0% glucose, 0.1% ascorbic acid, pH 3.16 ± 0.3) and experimental solution. All ΔL/M values were normalized using arcsine transformation and were tested for significance using Newman-Keuls test.

Following behavioral testing, cannulated animals were sacrificed and perfused with 0.9% NaCl and neutral buffered Formalin. The brains were removed and placed in neutral buffered Formalin solutions. Paraffin sections were cut (15 μ) and stained with cresyl violet. Cannulae positions were verified by microscopic examination.

EXPERIMENT 1

Experiment 1 investigated the effects of norepinephrine, epinephrine, methoxamine, phentolamine, phenoxybenzamine, isoproterenol and propranolol infusions into the MPOA, ARC-VM and LHA upon lordotic behavior in OVX rats primed with low doses of estrone.

Method

All animals tested for drug induced enhancements of sexual behavior were injected with estrone 48 hr prior to mating. The dose of estrone was adjusted for each animal to achieve an arbitrary preinfusion receptivity value of less than 0.333 L/M. The dosage of estrone ranged from 100–250 μg. Following preinfusion mating, each animal was infused with either the saline-glucose-ascorbic acid vehicle, 200 ng NE (Sigma), 200 ng E (Sigma), 200 or 800 ng methoxamine (Burroughs Wellcome), 200 or 800 ng phentolamine (CIBA), 200 or 800 ng phenoxybenzamine (Smith, Kline and French), 200 or 800 ng isoproterenol (McNeil Laboratories) or 200 or 800 ng propranolol (Sigma). The postinfusion mating session was begun 1.75 hr after infusion (2.5 hr after preinfusion mating). The time for maximal postinfusion mating response was estimated through the use of time-response trials with alternating 15 min mating-rest intervals [7,8] and test solutions of DA (200 ng), NE (200 ng), E (200 ng) or saline-glucose-ascorbate vehicle (Fig. 2). Both pre- and postinfusion matings were performed using 15 min of 15 mount protocol described previously [8].

Results

Significant elevations in lordotic behavior were observed following MPOA or ARC-VM infusions of 200 ng NE (both vehicle comparisons: $p<0.001$) or 200 ng E (MPOA: $p<0.005$ and ARC-VM: $p<0.001$) (see Table 1). Infusions of 200 or 800 ng of α receptor blockers, phentolamine and phenoxybenzamine, into either the MPOA or ARC-VM significantly elevated sexual receptivity (all comparisons with vehicle: $p<0.001$). Although the mean ΔL/M values for phentolamine and phenoxybenzamine infusions suggested increases in sexual receptivity associated with increasing

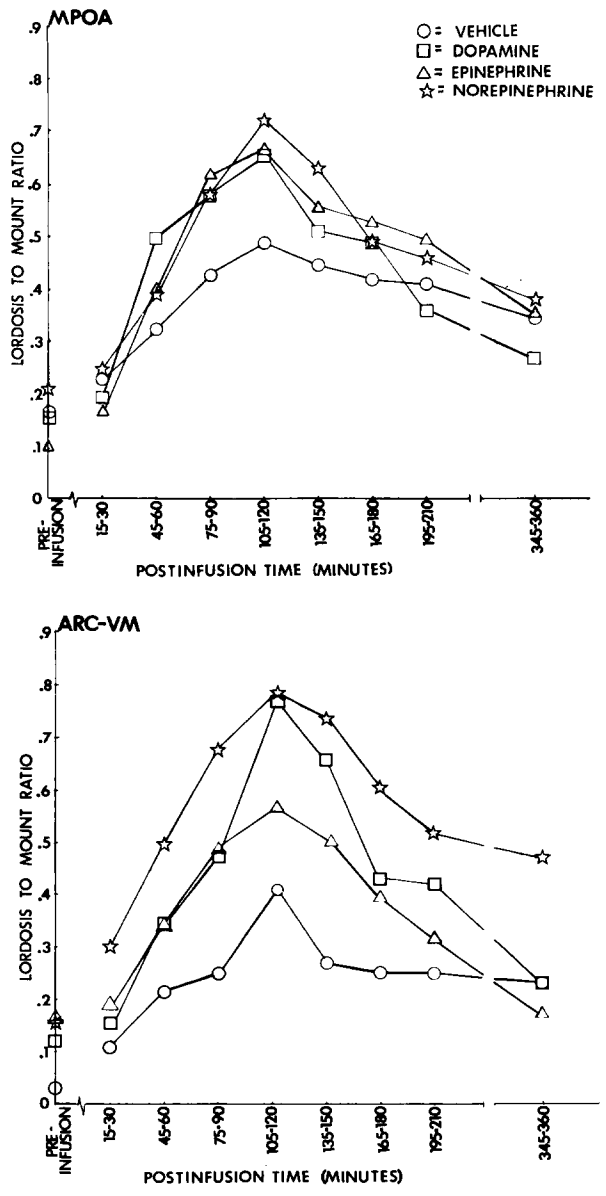

FIG. 2. (Top) Time course graph of lordotic response to dopamine (200 ng), norepinephrine (200 ng), epinephrine (200 ng) or vehicle into the medial preoptic area (MPOA). (Bottom) Time course graph of lordotic response to dopamine (200 ng), norepinephrine (200 ng), epinephrine (200 ng) or vehicle into the arcuate-ventromedial area (ARC-VM). In both graphs, circles, squares, triangles and stars represent vehicle, dopamine, epinephrine and norepinephrine, respectively.

concentrations, no significant changes could be found. Infusions of methoxamine, an α receptor stimulant, into either the MPOA or ARC-VM significantly depressed mating behavior with 800 ng doses (both vehicle comparisons: $p<0.05$), however, no significant ΔL/M alterations were observed with 200 ng doses in either area.

Significant enhancements in sexual behavior (ΔL/M) were observed following MPOA or ARC-VM infusions of 200 or 800 ng of a β stimulant, isoproterenol (minimum level of significance: $p<0.005$). Significant elevations in sexual receptivity were also associated with increasing dosage of isoproterenol in both areas (200 vs 800 ng, MPOA: $p<0.001$, ARC-VM: $p<0.05$). Although, the mean ΔL/M values for propranolol infusions into the MPOA or ARC-VM were observed to be consistently lower than vehicle, statistically significant depressions were found only with infusions into the ARC-VM ($p<0.05$).

None of the adrenergic drugs significantly altered lordotic behavior when infused into the LHA.

EXPERIMENT 2

Experiment 2 studied the effects of α- and β-adrenergic drugs upon lordotic behavior in OVX rats primed with high doses of estrone.

Method

Although the arbitrary preinfusion maximum used in Experiment 1 increased the reliability of this protocol to measure enhancements in sexual behavior, this restriction decreased the sensitivity to detect inhibitory drug effects; therefore, a second protocol using high preinfusion receptivity was developed to corroborate the findings of Experiment 1. The high preinfusion receptivity was induced by injecting each OVX-cannulated rat with 350 μg estrone (Theelin in oil) 48 hr prior to mating. In preliminary experiments, the lowest lordotic response observed with this level of estrone pretreatment was 0.67 L/M. This value was used as an arbitrary preinfusion receptivity minimum. The MPOA or ARC-VM cannulated animals which achieved this arbitrary minimum, were infused with either methoxamine (1000 ng), phenoxybenzamine (1000 ng), isoproterenol (1000 ng), propranolol (1000 ng) or saline-glucose-ascorbate vehicle. The postinfusion mating sessions were begun 1.75 hr after infusion as in Experiment 1.

Results

Infusions of 1000 ng methoxamine or propranolol in both MPOA or ARC-VM significantly lowered sexual receptivity when compared to vehicle control (all comparisons: $p<0.001$, Fig. 3). The mean ΔL/M values were significantly lower ($p<0.005$) for propranolol infusions compared to methoxamine infusions into the MPOA and slightly lower for the same comparison in the ARC-VM. No significant elevations in sexual behavior were observed following infusions of phenoxybenzamine or isoproterenol in either area with near maximal preinfusion receptivity used in this protocol.

EXPERIMENT 3

Experiment 3 studied the effects of simultaneous infusions of inhibitory monoaminergic drugs (methoxamine and propranolol) with LRH upon LRH-facilitated lordotic behavior in OVX rats primed with low doses of estrone.

Method

In order to test the possibility of adrenergic mediation of LRH facilitation of sexual behavior, comparisons were made among the behavioral responses to infusions containing LRH (50 ng); LRH (50 ng combined with methoxamine (1000 ng); LRH (50 ng) combined with propranolol (1000 ng) and saline-glucose-ascorbate vehicle into the MPOA or ARC-VM. All OVX cannulated animals were primed with low

TABLE 1

CHANGES [Δ ± SE (N)] IN LORDOSIS-TO-MOUNT RATIO FOLLOWING INTRAHYPOTHALAMIC INFUSIONS

Drug	Cannula Site ARC-VM	MPOA	LHA
Vehicle	0.227 ± 0.029(16)	0.177 ± 0.030(15)	0.176 ± 0.027(13)
Norepinephrine 200ng	0.555 ± 0.510(14)‡	0.173 ± 0.023(10)	
Epinephrine 200ng	0.521 ± 0.039(14)‡	0.414 ± 0.065(11)†	0.175 ± 0.033(10)
Adrenergic α Stimulant			
Methoxamine			
200ng	0.145 ± 0.025(13)	0.153 ± 0.041(12)	N.T.
800ng	0.068 ± 0.050(12)*	0.090 ± 0.032(13)*	0.187 ± 0.029(10)
Adrenergic α Blocker			
Phentolamine			
200ng	0.488 ± 0.042(11)‡	0.490 ± 0.033(12)‡	N.T.
800ng	0.590 ± 0.046(12)‡	0.631 ± 0.046(12)‡	0.179 ± 0.036(10)
Phenoxybenzamine			
200ng	0.533 ± 0.057(12)‡	0.412 ± 0.049(11)‡	N.T.
800ng	0.567 ± 0.088(9) †	0.572 ± 0.048(13)‡	0.180 ± 0.036(10)
Adrenergic β Stimulant			
Isoproterenol			
200mg	0.466 ± 0.036(12)‡	0.426 ± 0.044(11)‡	N.T.
800ng	0.673 ± 0.051(14)‡	0.647 ± 0.045(13)‡	0.140 ± 0.037(13)
Adrenergic B *Blocker*			
Propranolol			
200ng	0.107 ± 0.033(13)*	0.130 ± 0.047(11)	N.T.
800ng	0.108 ± 0.049(11)*	0.130 ± 0.067(14)	0.157 ± 0.020(14)

N=Number of Animals; SE=Standard Error of mean; N.T.=Not Tested.
*$p<0.05$
†$p<0.005$
‡$p<0.001$

doses of estrone (100–250 μg Theelin in oil) 48 hr prior to mating. The specific estrone dose for each animal was titrated to maintain an arbitrary preinfusion maximum of 0.250 L/M.

Results

Lordotic behavior was significantly enhanced following infusions of LRH into the MPOA or ARC-VM (both LRH vs vehicle comparisons: $p<0.001$, Fig. 4). The addition of methoxamine or propranolol to the LRH infusates abolished the effectiveness of LRH in enhancing sexual behavior when infused into either area. The lordotic behavior observed with infusions containing methoxamine or propranolol with LRH were not significantly different from vehicle response. In this experiment, as in the two previous experiments, no alterations in locomotor or other consumatory behaviors were observed following drug infusions.

DISCUSSION

The results of Experiment 1 indicate that both NE and E have stimulatory effects upon lordotic behavior when infused into the MPOA and ARC-VM. The stimulatory effects of isoproterenol (Experiment 1) and the inhibitory effects of propranolol (Experiment 2) infusions upon lordotic behavior indicate that the actions of NE and E upon sexual behavior may be mediated through postsynaptic β-adrenergic receptors. These findings are in conflict with previous reports of behavioral enhancement following hypothalamic implantation of a β receptor blocker, LB-46 [35]. It is difficult to make dose-response comparisons between these studies, since the actual dose of crystalline LB-46 delivered could not be determined. However, the facilitatory effects of LB-46 observed by Ward and co-workers may also be due to repetitive mating alone, since comparisons were not made between control and experimental animals at each mating interval. As observed in the time trial matings (see Method) and Experiments 1 and 3, repeated coital stimulation has a facilitatory effect upon lordotic response.

The inhibitory effects of α receptor blockade (Experiment 1) are in agreement with previous reports of enhanced sexual behavior following systemic administration of α receptor blockers [4,6]. However, in view of the stimulatory effects observed with hypothalamic infusion of all the catecholamines, these results indicate that α-adrenergic receptors may have a more subordinate role in hypothalamic control of sexual behavior. There are at least two possible

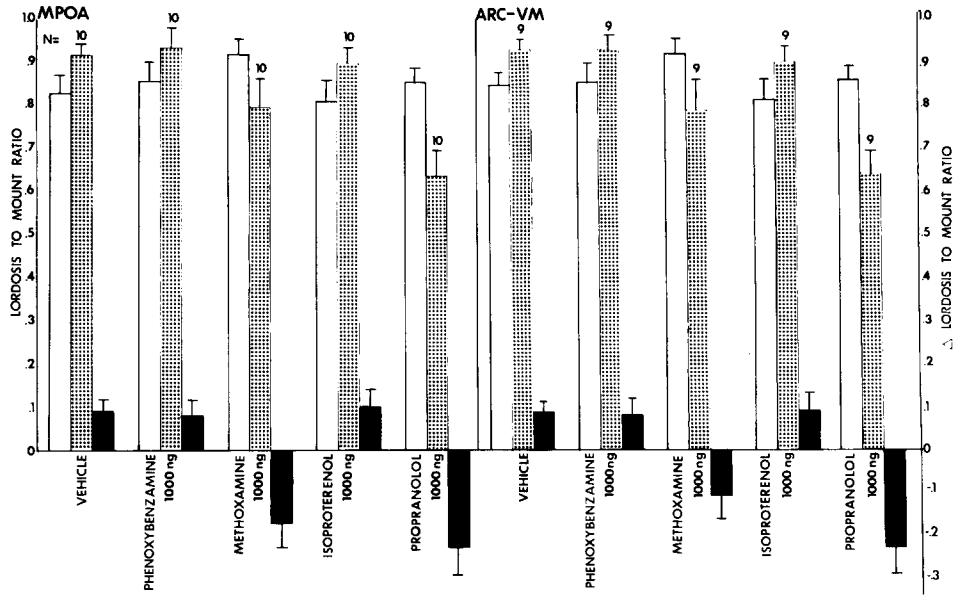

FIG. 3. Histogram representation of lordotic responses to MPOA and ARC-VM infusions of vehicle; phenoxybenzamine (1000 ng); methoxamine (1000 ng); isoproteranol; and propranolol (1000 ng); in OVX rats primed with high doses of estrone. The mean preinfusion L/M values are represented by open bars, mean postinfusion L/M values by stippled bars, mean ΔL/M values by closed bars and standard errors by brackets.

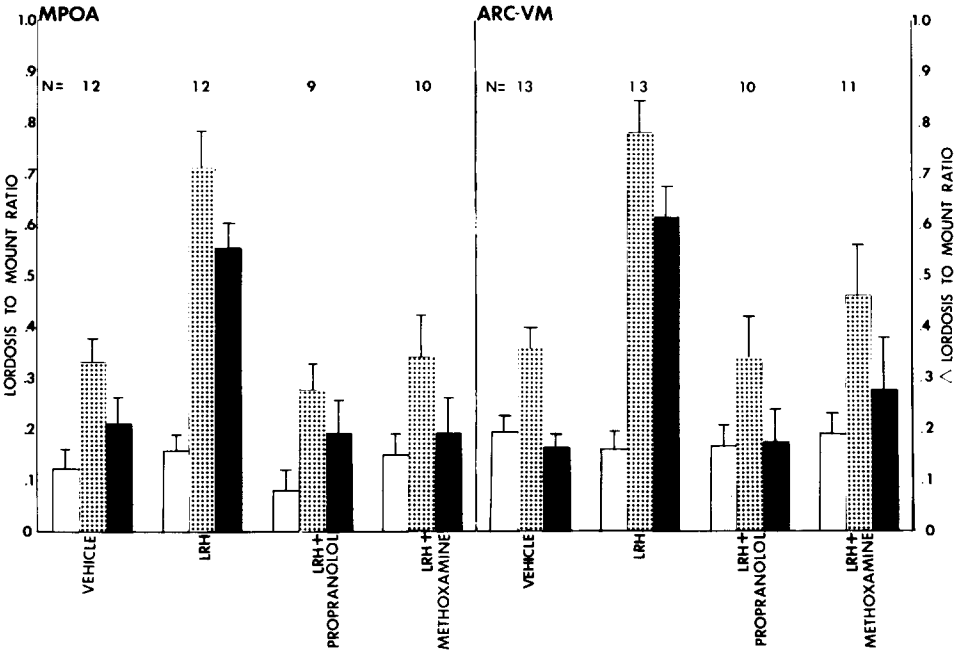

FIG. 4. Histogram representation of lordotic response to MPOA and ARC-VM infusions of vehicle; LRH (50 ng); LRH (50 ng) with propranolol (1000 ng) and LRH (50 ng) with methoxamine (1000 ng) in OVX rats primed with low doses estrone. The mean preinfusion L/M values are represented by open bars, mean postinfusion L/M values by stippled bars, ΔL/M values by closed bars and standard errors by brackets.

mechanisms by which a masking of an inhibitory receptor may occur.

First, the synaptic frequency of α receptors may be lower than β receptors and/or the distribution of these receptors may be more distal to the axon hillock. In either theoretical situation, the expression of α-adrenergic activity would require the suppression of β-adrenergic activity, since both situations would lessen the relative influence of α receptors with respect to β receptors in influencing propagated neuronal activity [28]. An alternative hypothesis to this frequency-distribution hypothesis is simply that α-adrenergic receptors may be located upon presynaptic membranes and have inhibitory effects upon NE and/or E release. This type of heterogeneous pre- and postsynaptic receptors has been proposed previously for other adrenergic systems [16,17].

The role of α-adrenergic receptors may be even more complex when one considers the possible indirect effects upon other neuronal types within the hypothalamus, such as LRH-containing neurons. Since α-adrenergic receptors have been proposed to have a stimulatory role upon LRH discharge [30,31] and LRH has been found to have a stimulatory effect upon lordosis behavior [7, 8, 20, 21, 22, 24, 26], facilitatory effects of α-adrenergic stimulation may also be possible through the amplified discharge of LRH. Indeed, the failure of Ward and co-workers [35] to observe enhancements in lordotic behavior following hypothalamic implantation of phentolamine, an α receptor blocker, may be indicative of these secondary effects upon other neuronal systems.

The blockade of LRH-induced enhancement of sexual behavior with the addition of either methoxamine or propranolol to the infusates suggests that the actions of LRH upon lordotic behavior may be mediated through NE or E neurons. According to this hypothesis, the action of LRH in stimulating the release of NE or E could be counteracted by presynaptic inhibition (α receptor stimulation) or through the blockade of postsynaptic receptors (β receptor blockade). Although these results can not preclude the possibility that LRH and catecholamines act independently upon the hypothalamic neurons mediating the lordotic reflex, the proposed interactions between LRH and catecholaminergic neurons may represent a means by which behavioral responses may be rapidly adjusted with gonadotropin release.

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