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Role of Catecholamines in the Courtship Behavior of Male Ring Doves

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BARCLAY, S. R. AND M.-F. CHENG. *Role of catecholamines in the courtship behavior of male ring doves.* PHARMACOL BIOCHEM BEHAV **41**(4) 739–747, 1992. – The role of catecholamines in the expression of male courtship behavior in ring doves was examined using central administration of pharmacological agents. Males treated with 6-hydroxydopamine or U-14,624, which depleted norepinephrine (NE) levels in the preoptic-hypothalamic area, showed increased levels of bow-coo and nest-coo displays. Conversely, males treated with tyramine or desipramine, which elevated NE levels in the preoptic-hypothalamic area, showed decreased levels of bow-coo and nest-coo displays. Drug-induced changes in dopamine levels were not consistent with any changes in behavior. This suggests that in the male ring dove NE in the preoptic-hypothalamic area is important in the expression of courtship displays.

Catecholamines Male sexual behavior Vocalizations Noradrenergic system

CATECHOLAMINES (CA's) have been shown to modulate male sexual behavior in rats. Pharmacological agents that enhance dopaminergic activity, such as L-DOPA or apomorphine, facilitated male sexual behavior by a reduction in the ejaculatory threshold (10,28,46). On the other hand, pharmacological agents that enhance the noradrenergic (NA) system, such as clonidine, an agonist, or nialamide, a monoamine oxidase inhibitor, or the reuptake blocker, imipramine, appeared to impair the expression of male sexual behavior (17,21). Electrolytic lesions of the dorsal noradrenergic bundle reduced the postejaculatory period and increased the frequency of mounting and intromission (18). Depletion of CA's by electrolytic or chemical lesions impaired sexual behavior in rats (14,15,38,49). This suggests that CA's play an important role in the mediation of male sexual behavior in rats. Similar pharmacological and lesion studies have not been performed in an avian species.

The present study examined whether CA's also mediate male sexual behavior in an avian species, ring doves. During the breeding cycle, many species of birds perform a series of complex behaviors, courtship that incorporates both vocal and visual displays (16). In most avian species, these vocal and visual displays have been shown to be androgen dependent by the following criteria. Castration abolished courtship behaviors, such as bow-cooing and nest-cooing in the ring dove (29,33), crowing and mounting in the quail (9), and reduced other behaviors, such as singing in the zebra finch (2). Testosterone (T) administered systemically (1,2,30,33) or centrally (29) reinstated these behaviors. Furthermore, it appeared that both aromatization and 5α-reduction in the brain were involved in the effect of T in reinstating these behaviors (1, 23,26,30). Consistent with these findings was a strong correlation found between changes in the level of androgens and behavioral changes during the breeding cycle (24,25,51).

In the present study, we determined if changes in synthesis, reuptake, or release of CA's would induce changes in male sexual behavior by administering a variety of drugs known to have specific actions on various aspects of the catecholaminergic system. Pharmacological alterations in brain monoamine content, in the preoptic-hypothalamic region in particular, were verified by high-performance liquid chromatography with electrochemical detection (HPLC-EC). We focused on the preoptic-hypothalamic (HYP) region primarily because this region has been implicated as an important region mediating male courtship behavior in the ring dove. Direct androgen implant in this region reinstates male courtship behavior in castrated doves (7,29). Furthermore, the activity of aromatase in this region appeared to be critically involved in changes in male courtship behavior (23,50). We did not directly examine the interaction of CA's and androgen in this study; this question will be addressed in future studies.

METHOD

Subjects

Subjects were 175 male and 120 female ring doves (*Streptopelia risoria*) from the laboratory colony at the Institute of Animal Behavior. All birds had experienced at least one com-

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plete breeding cycle and had spent a minimum of 3 weeks in isolation cages prior to the beginning of the experimental procedure. Birds were maintained on a 14 L:10 D cycle. Food, water, and grit were available ad lib. All observations were done between 0800–1300 h in breeding cages (70 × 65 × 35 cm) in which males had been allowed to habituate overnight. Stimulus females used in these experiments were receptive when paired with a male under these conditions.

Behavioral Observations

A female was placed in the breeding cage and the opaque partition separating the pair was removed. Pairs were observed for 15 min and courtship behaviors were scored once if they occurred in a 30-s interval regardless of how many times they might occur in that interval. Eight behaviors were scored: aggressive courtship: bow-coo, hop-charge cackle, peck; nest-oriented courtship; nest-coo and wing-flip; preen; crouch; and mount. Bow-coo and nest-coo displays consist of coos delivered while assuming specific postural displays. These displays are repeated many times during the initial courtship period. The bow-coo is performed only by the male and directed toward the female, while the nest-coo is performed by both the male and female in the area of the nest site. The nest-coo and bow-coo sound similar to the human ear; however, they are acoustically distinct (27,35). Hop-charge cackle is performed almost exclusively by the male and directed at the female. The other behaviors are self-explanatory and have previously been described (42). Male doves typically initiate courtship with hop-charge cackle and bow-coo displays followed by nest-coo displays; as courtship progresses and the frequency of the male's nest-coo displays increase, the female begins to nest-coo and several days later lays a clutch of two eggs.

Cannulations

A chronic cannula was stereotaxically implanted in the third ventricle. All cannulations were performed under Chloropent anesthesia (Fort Dodge Laboratories, Inc., Fort Dodge, IA: 0.4 ml, IM) according to the method of Crane and Glick (20) with a few minor modifications. Stainless steel 28-ga hypodermic tubing was cut into 15-mm lengths and the tip beveled to a 45° angle. The bird's beak was clamped to a holder equipped with a 45° adapter slide that permitted the head to be tilted 45° in the sagittal plane. The site of the implant (3.7 mm anterior on the midline suture) was determined from interaural zero. Each cannula was lowered 8 mm till the tip rested above or pierced the roof of the third ventricle. The cannula was then secured with dental cement and a dummy inner cannula (30-ga) lowered into the outer cannula. Birds were allowed a minimum of 4 days to recover after surgery before drugs were infused. After testing, the cannula site was checked by infusion of India ink; if the position of the chronic cannula did not allow the drug to infuse into the third ventricle, data obtained from behavioral observations of that subject were not included in the statistical analysis.

Drug Administration

The following procedure was used to administer drugs ICV. Microline tubing was filled with the appropriate drug or control vehicle (isotonic saline unless otherwise stated) and attached to an inner cannula (30-ga). All drugs were placed into solution immediately prior to infusion. The tubing was then attached to a 50-μl Hamilton syringe in a microinfusion pump (Harvard Bioscience, Inc.). This arrangement delivered the drug at a steady rate of 1 μl/40 s. At the time of infusion, the dummy inner cannula was removed from the bird and the new inner cannula lowered 1 mm past the outer cannula into the lumen of the third ventricle. After 3 μl was infused, the inner cannula was left in place for 3 min to prevent the drug from being drawn back into the outer cannula. The male was then placed back into the observation cage. Behavioral observations were 15 min after drugs were administered. 6-Hydroxydopamine hydrobromide (6-OHDA) was administered twice with a 2-day interval between the first and second infusion to ensure maximal destruction of catecholamine neurons (53);

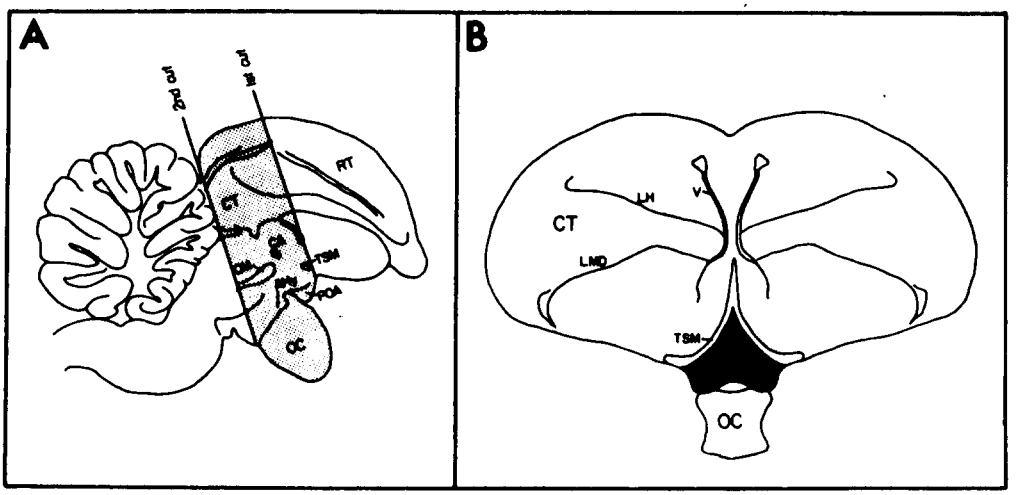

FIG. 1. (A) Schematic diagram of the ring dove brain detailing the two coronal cuts made to isolate the brain slice containing HYP and CT. (B) Details of the brain slice with the dissection of HYP indicated by shaded area. CT, caudal telencephalon; HYP, preoptic-hypothalamic region; LH, hyperstriatal lamina; LM_D, dorsal medullary lamina; OC, optic chiasm; RT, rostral telencephalon; TSM, septomesencephalic tract; V, ventricle; CA, anterior commisure; OM, occipitalmesencephalic tract; AHy, anterior hypothalamus; POA, preoptic area; LMD, dorsal medial lamina.

the observations began the morning after the second infusion.

Drugs

Drugs were infused intraventricularly in a 3-μl volume. 6-OHDA (25.0, 50.0 μg, Sigma Chemical Co.), a neurotoxin, was dissolved in bidistilled water and the vehicle was pH-matched bidistilled water. A sympathomimetic amine, tyramine (TYR; 1.0, 10.0, 100.0 μg, Sigma Chemical Co.), a dopamine-β-hydroxylase inhibitor, U-14,624 (0.04, 0.4, 4.0 μg, Aldrich Chemical Co.), and a reuptake blocker, desmethylimipramine hydrochloride (DMI; 0.2, 2.0, 20.0, 200.0 μg, a gift from CIBA-GEIGY), were dissolved in isotonic saline. Dosages refer to the salt.

Prazosin hydrochloride, administered systemically (2.0 μg, IM, a gift from Pfizer Co.), is relatively insoluble in saline so the drug was dissolved in bidistilled water that was gently heated.

Brain Dissections

The effects of TYR, U-14,624 and DMI on brain monamine content were determined in a second group of males administered the drugs ICV 15 min before being sacrificed. The effects of 6-OHDA on brain monoamine content were determined in the same group of birds that had been tested for behavioral effects; they were sacrificed 24 h after behavioral observations were completed. These experimental paradigms reflect different pharmacological actions of the drugs involved. Whereas the other drugs have a much shorter period of action and the effects may disappear after 24 h, the effects of 6-OHDA persist. All subjects were sacrificed during the same period of time (between 1300–1500 h) to minimize any confounding effects of circadian rhythms in brain monoamine content. Rapid decapitation was used to sacrifice the subjects.

Four samples were dissected from the whole brain and analyzed for monoamine content. The preoptic-hypothalamic region was chosen for its association with sexual behavior and gonadotropin secretion. The other areas were chosen to determine the extent of diffusion of the drug from the third ventricle area and to serve as points of comparison for changes in the preoptic-hypothalamic region. The brain was removed from the skull and placed on its dorsal surface on a chilled glass plate and quickly dissected. The initial cut was made on the ventral surface of the brain just anterior to the septornesencephalic tract. The second cut was made caudal to the first cut at the posterior margin of the optic chiasm (see Fig. 1). This created a brain slice containing the preoptic-hypothalamic (HYP) and caudal telencephalon (CT) regions. The HYP was dissected from this brain slice by following the lateral lines of the septomesencephalic tract and trimming away the optic tract. The remaining tissue (CT) in this brain slice, dorsal to the HYP, is mainly neostriatum, with hippocampus, septum, and archistriatum. The rostral telencephalic (RT) sample

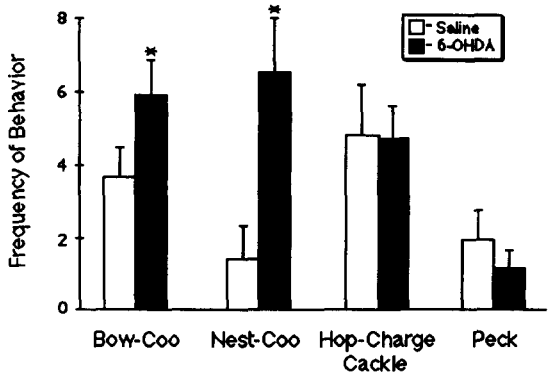

FIG. 2a. Effects of 6-OHDA (25 μg) administered ICV on courtship behavior in male ring doves (n = 13). A significant enhancement of bow-coo and nest-coo behavior (*$p < 0.01$) occcurred in comparison to VEC-treated controls.

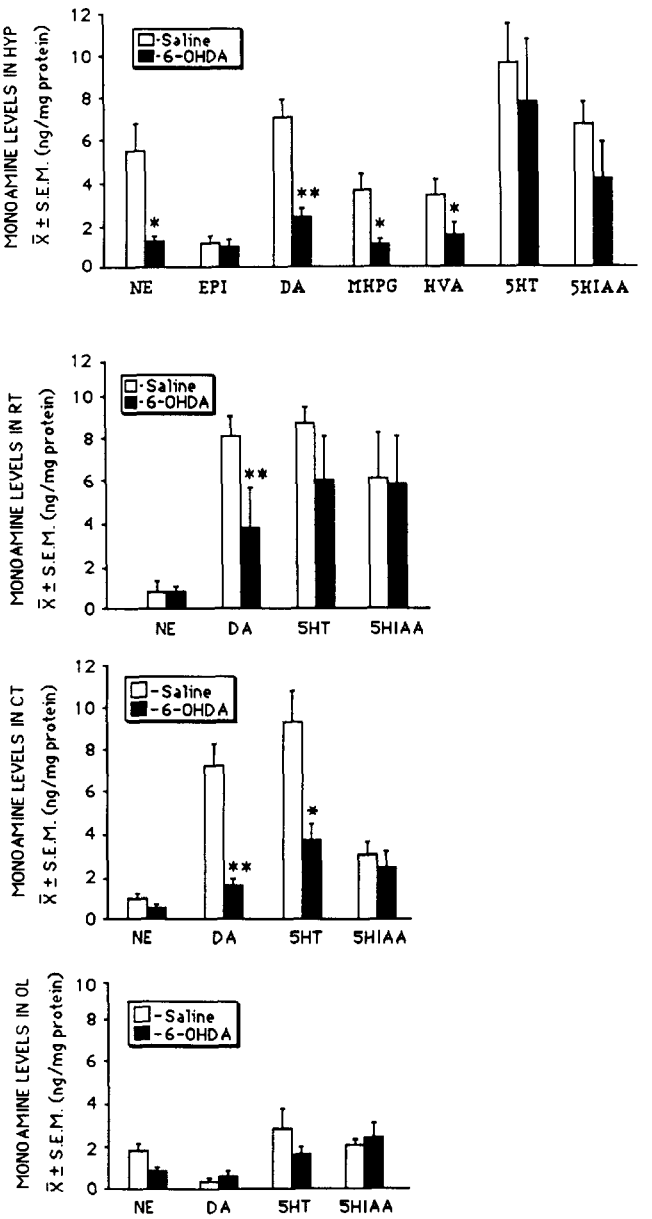

FIG. 2b. Effects of 6-OHDA (25 μg) administered ICV on brain monoamine content. NE levels were significantly depleted (*$p < 0.01$) in the HYP, while DA levels were significantly depleted (**$p < 0.05$) in the HYP, RT, and CT. In the CT, 5-HT levels were also significantly depleted (*$p < 0.01$). The levels of primary amines and significantly depleted metabolites are shown for each area (n = 6).

was the entire section rostral to the first cut, consisting of the paraolfactory lobe, hyperstriatal areas, ectostriatum, and anterior neostriatum. The optic lobe (OL) sample contained both lobes caudal to the HYP. All tissue was quickly frozen on dry ice and remained frozen at −80°C until further preparation.

Monoamine Assay

The frozen tissue was homogenized in a sodium acetate buffer (pH 5.0) containing α-methyldopamine (1.7×10^{-7}M, a gift of Merck, Sharp and Dohme) as an internal standard. The sample was then centrifuged at 27,000 *g* for 15 min and the supernatant removed and stored at −80°C until analysis. The pellet was resuspended and protein content was determined according to the method of Bradford (11). On the day of analysis, each sample was thawed, 10 μl of an ascorbate oxidase solution (1 mg/10 ml bidistilled water) added, and the sample recentrifuged at 27,000 *g* for 10 min. Samples were stable at 4°C for up to 10 h. The addition of ascorbate oxidase to the sample decreased the level of ascorbic acid, which elutes in the solvent front and interferes with resolution of the NE peak (39).

The monoamines, NE, DA, and 5-HT, and their primary metabolites were measured by HPLC-EC according to the methodology of Renner and Luine (48) with a few modifications. The sample (50 μl) was injected into a Beckman chromatographic system with a 5-μm Ultrasphere column (25 cm × 4.6 mm, length × diameter) and electrochemically analyzed using an LC-4A/19 potentiostat (Bioanalytical Systems) with a glassy carbon electrode. Flow rate was maintained at 0.75 ml/min with a back pressure of 1600 psi. The electrode potential was set at +0.65 V with respect to an Ag/AgCl reference electrode.

Levels of monoamines and their metabolites in the brain sections were calculated by multiplying the area of each peak by the appropriate response factor (a measure of the efficiency of detection of any given compound in comparison to the internal standard) and correcting for preparation volume and percentage recovery of the internal standard. The minimal detectable level of each amine was 100 pg (2 : 1, signal : noise ratio) and the assay was linear for the range of concentrations present in the samples. Levels were expressed as ng amine/mg protein.

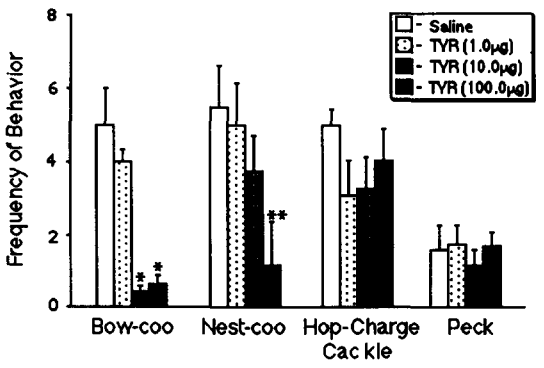

FIG. 3a. Effects of TYR administered ICV on male courtship behavior ($n = 7$). A significant decrease in bow-coo (*$p < 0.01$) and nest-coo (**$p < 0.05$) behavior occurred when 100 μg was infused in comparison to VEC-treated controls. A significant decrease in only bow-coo (*$p < 0.01$) behavior occurred when 10 μg was infused.

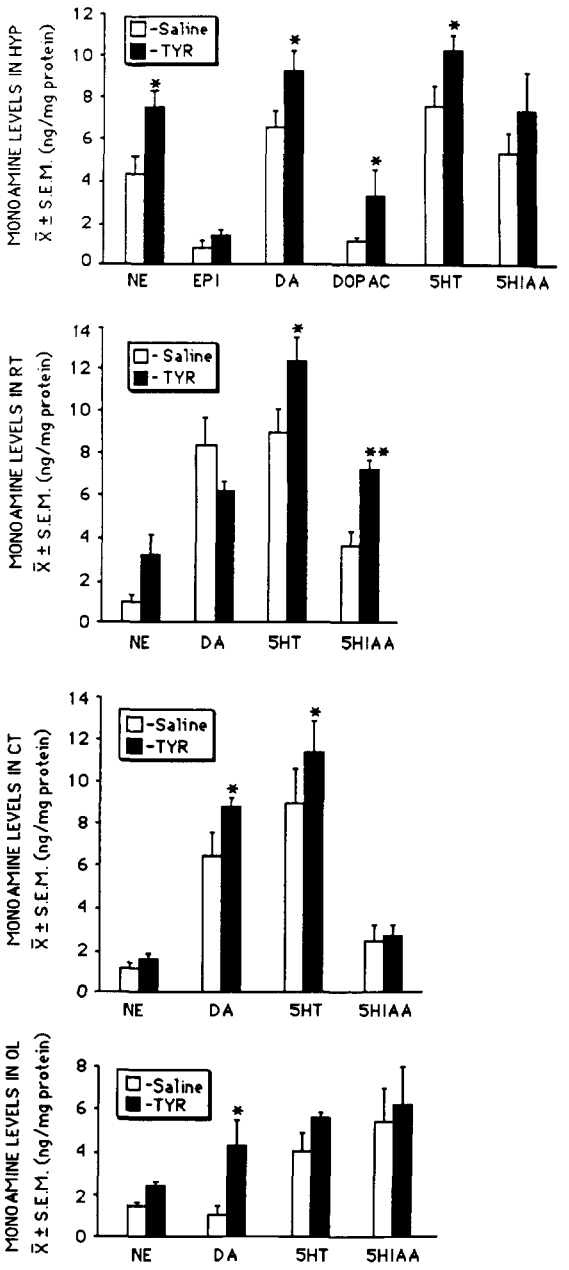

FIG. 3b. Effects of TYR (100 μg) administered ICV on brain monoamine content. In the HYP, levels of the primary amines significantly increased (*$p < 0.01$). DA levels were also significantly increased in the RT and OL (*$p < 0.01$). 5-HT levels were significantly increased (*$p < 0.01$) in the CT and RT, where 5-HIAA levels (**$p < 0.05$) were significantly greater than control levels. The levels of primary amines and significantly affected metabolites are shown for each area ($n = 6$).

Statistics

The statistical significance of the effect of drug treatment on the frequency of male courtship behavior and brain monoamine content was determined by one-way analysis of variance (ANOVA) followed by the Newman-Keuls multiple range test (13). Separate analyses were done for each monoamine and each of the scored behaviors.

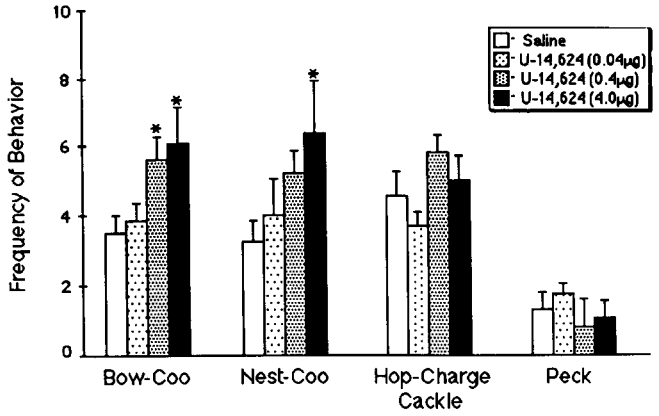

FIG. 4a. Effects of U-14,624 administered ICV on male courtship behavior (n = 8). A significant increase in bow-coo and nest-coo behavior ($*p < 0.01$) occurred when 4.0 μg was infused in comparison to VEH-treated controls. A significant increase in only bow-coo ($*p < 0.01$) behavior occurred when 0.4 μg was infused.

RESULTS

Experiment 1

Effects of 6-OHDA on male courtship behavior. The low (25 μg per infusion) dose of 6-OHDA administered ICV caused a significant enhancement of bow-coo ($F = 17.35$, $p < 0.01$) and nest-coo ($F = 11.37$, $p < 0.01$) behavior (see Fig. 2a, n = 13) as compared to saline-treated controls. There were no significant changes in the frequency of hop-charge cackle, peck, or preen. No mounting behavior occurred in either group.

Administering (25 μg per infusion, n = 6) 6-OHDA resulted in significant depletion of NE (75%; $F = 13.7$, $p < 0.01$) and DA levels (67%; $F = 4.1$, $p < 0.05$) in the HYP region as compared to the saline-treated controls. Significant depletion of DA was also recorded in the RT (51%; $F = 4.1$, $p < 0.05$) and the CT (77%; $F = 4.1$, $p < 0.05$) samples, but NE content was not affected by the drug treatment. At this dosage, 6-OHDA also significantly lowered 5-HT content of CT (69%; $F = 8.92$, $p < 0.01$). Optic lobe samples showed no drug treatment effects on the levels of all three amines (see Fig. 2b).

When a higher dose (50 μg per infusion) of 6-OHDA was administered ICV (n = 8), a substantial number (50%) of subjects exhibited severe motor disturbances, and others in the group showed mild disturbances such as feather-ruffling and head-cocking. Symptoms developed gradually overnight and persisted till the day of testing. Due to the severity of the motor disturbances in such a large number of experimental subjects, the behavioral data from these subjects were not included in statistical analysis. At the lower dose (25 μg per infusion) of 6-OHDA, no subjects exhibited motor disturbances and only 4 of 13 subjects showed minor symptoms, such as feather-fluffing.

Experiment 2

Effects of tyramine on male courtship behavior. A high dose of TYR, a sympathomimetic amine (100 μg, n = 7), administered ICV caused a significant decrease in the frequency of bow-coo ($F = 8.27$, $p < 0.01$) and nest-coo ($F = 3.58$, $p < 0.05$) behavior as compared to VEH-treated controls (see Fig. 3a). At an intermediate dose (10 μg, n = 7) only bow-coo behavior was suppressed, while levels of other behaviors (nest-coo, peck, and hop-charge cackle) were unaffected. At the lowest dose (1.0 μg, n = 7), none was affected.

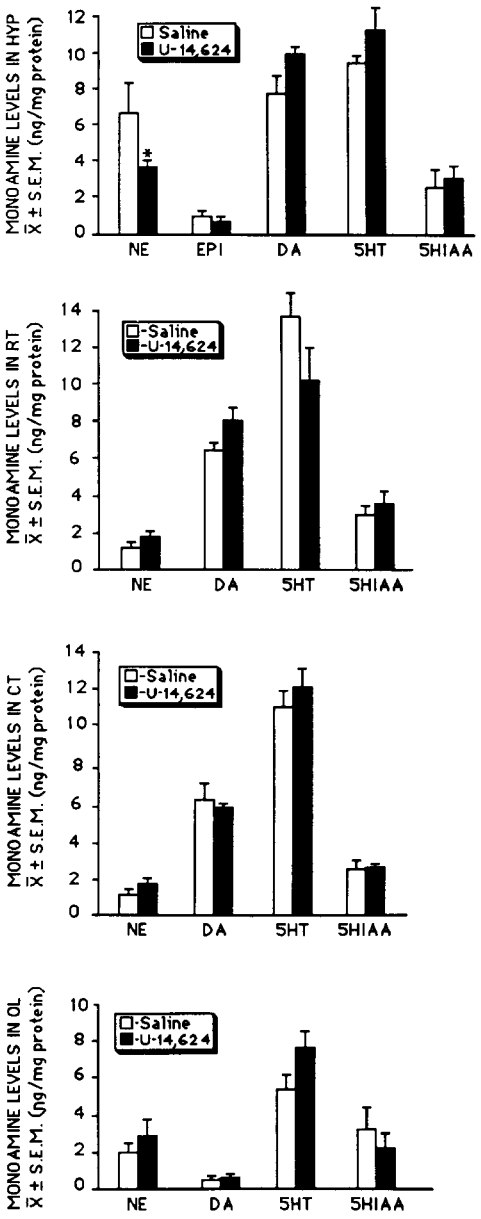

FIG. 4b. Effects of U-14,624 (4.0 μg) administered ICV on brain monoamine content (n = 6). In the HYP, levels of NE were significantly decreased ($*p < 0.01$); no other amines were affected. The levels of the primary amines and any other significantly affected metabolites are shown for each brain area (n = 6).

TYR (at 100 μg) had potent effects on the levels of all three monoamines in the HYP sample (see Fig. 3b, n = 6). Levels of the primary amines, as well as some of their respective metabolites, increased significantly (NE: 48%, $F = 19.04$, $p < 0.01$; DA: 43%, $F = 6.15$, $p < 0.01$; 5-HT: 35%, $F = 10.94$, $p < 0.01$). In other regions, the effect of the drug was not as simple. In the CT sample, there was a 36% increase in

the content of DA ($F = 6.15$, $p < 0.01$). The level of DA was also significantly increased (270%, $F = 6.15$, $p < 0.01$) in the OL sample. In the RT sample, 5-HT and its metabolite 5-HIAA were significantly increased (5-HT: 38%, $F = 10.94$, $p < 0.01$; 5-HIAA: 95%, $F = 3.12$, $p < 0.05$).

Experiment 3

Effects of U-14.624 on male courtship behavior. ICV administration of the dopamine-β-hydroxylase inhibitor, U-14,624 (4.0 μg, $n = 8$), enhanced the expression of two courtship behaviors, bow-coo ($F = 5.34$, $p < 0.01$) and nest-coo ($F = 3.89$, $p < 0.01$). An intermediate dose (0.4 μg, $n = 8$) significantly increased the frequency of bow-coo but not nest-coo. All other courtship behaviors (nest-coo, peck, hop-charge cackle, and mount) were not affected by both doses of U-14,624; they occurred at levels comparable to the VEH-treated controls during the 15-min observation. The lowest dose (0.04 μg, $n = 8$) had no effects (see Fig. 4a).

Administration of U-14,624 (4.0 μg, $n = 6$, ICV) significantly depleted NE levels in the HYP region (see Fig. 4b, $F = 3.92$, $p < 0.05$). There were no other measurable changes in monoamine levels.

Experiment 4

Effects of desipramine on male courtship behavior. The highest dose of DMI (200.0 μg, $n = 6$) administered ICV, significantly suppressed bow-coo ($F = 8.73$, $p < 0.01$), nest-coo ($F = 5.65$, $p < 0.01$), and hop-charge cackle ($F = 4.02$, $p < 0.01$) compared to VEH-treated controls (see Fig. 5a). At intermediate doses (20.0 μg, $n = 6$, and 2.0 μg, $n = 6$), DMI-treated males exhibited a lower incidence of bow-coo and nest-coo behavior, while the frequency of other courtship behaviors was not significantly altered from that of control birds. At the lowest dose (0.2 μg, $n = 6$), there was no effect on behavior.

Administration of DMI (20 μg, $n = 6$, ICV) increased NE levels in HYP (61%, $F = 19.41$, $p < 0.01$). There was also a decrease in DA levels in CT (47.1%, $F = 3.28$, $p < 0.05$) and in 5-HT levels in RT (48%, $F = 3.40$, $p < 0.05$; see Fig. 5b).

If the DMI-induced changes in courtship behavior are mediated through an increased availability of NE in the HYP acting on α-adrenoceptors, then pretreatment with prazosin (2.0 μg, iM), an α-adrenoceptor antagonist, might block these behavioral effects. As expected, pretreatment with prazosin 1 h before ICV infusion of DMI (2.0 μg) prevented the behavioral effects of DMI treatment. This group did not exhibit the suppression of bow-coo ($F = 8.29$, $p < 0.01$) or nest-coo ($F = 7.34$, $p < 0.01$) behavior seen in the VEH-pretreated DMI group ($n = 8$).

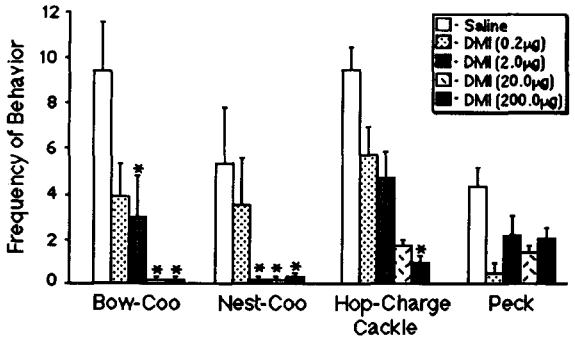

FIG. 5a. Effects of DMI administered ICV on male courtship behavior ($n = 6$). A significant decrease in bow-coo (*$p < 0.01$) and nest-coo (*$p < 0.01$) behavior occurred when 2.0, 20.0, and 200. μg DMI was infused in comparison to VEH-treated controls. Hop-charge cackle was also suppressed when 0.2 μg was infused.

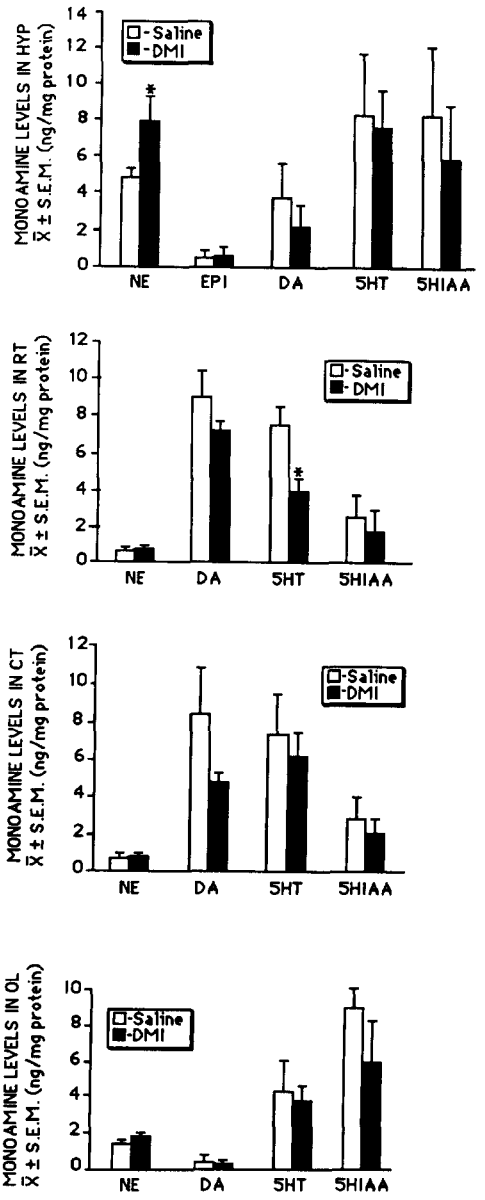

FIG. 5b. Effect of DMI (20 μg) administered ICV on brain monoamine content. In the HYP, levels of NE significantly increased (*$p < 0.01$); no other amines were affected. In the RT, levels of 5-HT significantly decreased ($p < 0.01$). The levels of primary amines and significantly affected metabolites are shown for each brain area ($n = 6$).

DISCUSSION

These experiments clearly show that in the male ring dove pharmacologically induced changes in the catecholaminergic system alter the expression of two vocal courtship displays.

Pharmacological treatments that enhance the activity of CA neurons, either by stimulating the release of CA's or by blocking reuptake, inhibit the expression of bow-coo and nest-coo behavior. The expression of these behaviors is enhanced by pharmacologic treatments that suppress the activity of CA neurons. Since the drug-induced changes in the expression of courtship behavior specifically involve the two vocalization displays, bow-coo and nest-coo, and both of these behaviors are androgen-dependent, it can be suggested that the observed behavioral changes are the indirect effect of CA regulation of hormonal secretion. These experiments did not distinguish whether these pharmacological effects are hormonally dependent or independent; however, in another study we specifically addressed this question (6).

In mammals, infusion of 6-OHDA ICV results in a behavioral syndrome characterized by aphagia, adipsia, and debilitating sensorimotor disturbances such as catalepsy, akinesia, and impaired orientation to stimuli (3,34,37), but such impairments did not occur when at least 5–10% of the DA terminals remained (56). These drug-induced behavioral effects in mammals were also observed in the high-dose (50 μg per infusion) group of ring doves. The effects of 6-OHDA on monoamine levels in the ring dove were consistent with the effects found in rats (12), that is, widespread depletion of NE and DA contents.

At a lower dose, the 6-OHDA-treated bird showed highly coordinated patterns of courtship behavior, bow-coo, and nest-coo; both displays were in fact enhanced by this drug treatment. This result in the ring dove appears to contradict the cataleptic state induced by this drug in rats. However, a closer examination of the rat literature reveals some striking similarities between our 6-OHDA-treated male doves and similarly treated male rats. It has been reported that male rats will pursue and copulate successfully if the female actively solicites (15). The females used in the present study were extremely receptive.

Since 6-OHDA destroyed most if not all NE and DA terminals, results based on this drug do not distinguish between the two systems. Another way to examine the role of CA's in male courtship behavior is to utilize pharmacological agents that specifically affect the activity of either DA or NE neurons. When a dopamine-β-hydroxylase inhibitor, U-14,624, was administered to mammals, NE content in the hypothalamic region was significantly depleted (31,45). This inhibition of noradrenergic transmission blocked the facilitation of lordosis by estrogen and progesterone in the female guinea pig (44) and impaired arousal levels in the male rat without affecting other parameters of copulatory behavior (38). In the ring dove, administration of U-14,624 also depleted the NE content in the preoptic-hypothalamic region and had behavioral effects consistent with those of 6-OHDA in the male, that is, an increase in the expression of courtship vocalization displays. No other measurable changes in brain monoamine content were found. This is somewhat inconsistent with the effects of U-14,624 in rats, where an increase in the synthesis of 5-HT was shown (32). However, the most plausible explanation for the lack of change in 5-HT synthesis in the ring dove is that the behaviorally effective dose in this study is significantly lower than the dosages typically used in mammals.

To further determine the specific role that the noradrenergic system may play in male sexual behavior, we examined the effects of enhancement of noradrenergic neurotransmission by different pharmacological action. TYR has both indirect and direct sympathomimetic actions on peripheral tissues innervated by noradrenergic nerves; it enhances noradrenergic action by releasing NE from the nerve terminal that stimulates adrenoceptors or it acts directly on α_1-adrenoceptors (43,52, 54). ICV administration of TYR in the ring dove increased preoptic-hypothalamic content of NE, DA, and 5-HT. These males when paired with very receptive females showed low levels of bow-coo and nest-coo behavior in a dose-dependent manner.

Desipramine enhances noradrenergic activity by still another mechanism. DMI, a secondary derivative of tricyclic antidepressants, has been shown to be a potent inhibitor of NE reuptake while having only minimal effect on DA or 5-HT reuptake (19). It has been suggested that acute administration of DMI does not change the response of cells activated by NE but appears to prolong NE-induced activations (40,41). Administration of this drug in rats resulted in highly significant increases in noradrenergic metabolites in the cerebral cortex (47) and plasma concentrations of NE (55). When administered to the ring dove, it produced a fairly specific increase in the level of NE in the HYP. This drug treatment significantly suppressed the expression of male courtship behavior in the ring dove.

Our studies suggest that expression of male courtship behavior in ring doves requires the suppression of noradrenergic activity in the preoptic-hypothalamic region. Since the first report on some of our findings in 1984 (5), there have been a number of studies in a different avian species that confirm our basic findings (4,22).

Beach proposed two separate and independent neural systems for sexual arousal and copulatory behavior (8). The present study was not designed to distinguish between the two. However, we would like to propose that these pharmacologically induced changes in male courtship levels result from changes in arousal states that are mediated by the noradrenergic system in the bird. This idea is consistent with the findings based on extensive pharmacological and electrophysiological studies in pigeons that convincingly establish the role of CA's, specifically hypothalamic NE, in the induction of both behavioral and electrocortical sleep (36).

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