other description available of their biology, in D. H. Janzen, *Nat. Hist.* **83**, 48 (1974)].
15. This is based on more than 400 pod collections from more than 600 trees between 1968 and 1975.
16. They also disperse from pasture trees that will have a pod crop the following year (there is no way the beetle can know this); the result is that the same absolute number of pods are killed in successive seed crops rather than an increasing number, as would be expected if the weevils could respond to this newly displayed behavior by the trees.
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19. This is based on information in G. N. Wolcott, *J. Agric. Univ. P.R.* **20**, 1 (1936) and on field collections of several thousand pods during February 1969. Furthermore, J. H. Frank writes from Jamaica that he has found no *Rhinochenus* there, although *H. courbaril* is common (13 May 1971). There are no specimens of *Rhinochenus* from any Caribbean island, except Trinidad, in either the British Museum of Natural History or the U.S. National Museum of Natural History.
20. The only predispersal seed predator of *H. courbaril* in Puerto Rico is the generalist scolytid, *Stephanoderes buscki* Hopkins (determined by S. Wood) which may kill some of the seeds if the pods lie intact on the ground for many months.
21. Most passable roads on the island were driven; all trees encountered were below an elevation of 400 m and in the moister, eastern half of the island. They were in pastures, roadsides, and forest.
22. Thus in the more northern Mexican populations of *H. courbaril*, where *Rhinochenus* has never been recorded, a high resin content in the pod walls is still expected since there are numerous other potential seed predators (parrots, squirrels, Hemiptera, and so forth) in these habitats.
23. R. Cates and D. Rhoades, Department of Zoology, University of Washington, Seattle, 12 January 1973.
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26. Supported by NSF grant GB-35032X and the teaching program of the Organization for Tropical Studies. Numerous persons aided in fieldwork, as will be documented in a longer report on the interactions of *Rhinochenus* and *Hymenaea* (*25*). Constructive criticism of the manuscript was offered by H. G. Baker, K. S. Bawa, G. W. Frankie, J. H. Langenheim, P. A. Opler, C. M. Pond, A. K. Sakai, C. C. Smith, R. L. Trivers, and D. R. Whitehead.

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Sexual Behavior: Extreme Reduction of Postejaculatory Refractory Period by Midbrain Lesions in Male Rats

Abstract. *The refractory period that characteristically follows ejaculation was abolished or significantly reduced by rostral midbrain lesions in male rats. The postejaculatory vocalization was also abolished or reduced, but other aspects of copulatory performance were unaffected. The results were attributed to disruption of biogenic amine pathways that pass from the ventral part of the rostral midbrain into the posterior hypothalamus.*

A period of sexual incapacity characteristically follows ejaculation in male mammals. In the rat this phase, the postejaculatory refractory period, is highly stable and only drastic experimental procedures are capable of greatly reducing or abolishing it. In a few instances electrical stimulation of preoptic and hypothalamic regions has had this effect (*1*), and, in one study, massive lesions at the midbrain-diencephalic junction reduced the refractory period to about 25 percent of normal (*2*).

Exogenous stimulation such as handling, electrical shock to the skin, or electrical stimulation of the brain normally does not reduce the refractory period to less than about 75 percent of normal (*3*). The resistance of the refractory period to abbreviation is due to a preeminent inhibitory state that prevails during the first three-fourths of the refractory period (*4*). This phase, designated as the absolute refractory period, is characterized by a high-amplitude, slow-wave electroencephalogram (EEG), the emission of 22-khz vocalizations (*5*), and a lack of spontaneous resumption of sexual activity (*6*).

The postejaculatory vocalization begins about 30 to 40 seconds after ejaculation, and shortly thereafter the animal shows a predominance of sleeplike EEG. The mechanism that underlies this behavior is not known; however, it is possible that the ejaculatory reflex triggers the postejaculatory refractory period and its concomitant vocalization by a neurohumoral link. Serotonergic, noradrenergic, and dopaminergic pathways run via the medial forebrain bundle from cell bodies in the midbrain to the hypothalamus (*7*). The biogenic amines have been implicated in the regulation of sleep and arousal (*8*), and are theoretically capable of regulating both the refractory period and endogenous sexual arousal (*9*).

Ejaculation occurs after a cerebral disinhibition of spinal reflexes (*10*). The refractory period could be triggered by events that underlie the disinhibition that allows ejaculation or in response to messages emanating in the spinal cord. In either case the mediation of the refractory period could involve transmission in specific neurohumoral pathways. We predicted that if a mechanism of this sort were involved, destruction of one or more of these pathways would result in a diminished refractory period and, possibly, altered sexual arousal.

We can now take a fresh look at earlier studies on the destruction of posterior hypothalamic and midbrain loci. Heimer and Larsson (*2*) reported dramatic reduction of the refractory period with massive lesions that might have destroyed the critical pathways. Subsequent attempts to achieve the same effect with smaller lesions in the mammillary region were unsuccessful (*11*). In similar experiments discrete lesions in the mammillary area of the brain of male rats resulted in an increased production of copulatory plugs (*12*); however, the effect was due to a reduction in the time to ejaculation and not to a decreased refractory period (*13*). Other studies have shown that posterior hypothalamic and rostral midbrain destruction is generally disruptive to male copulatory behavior (*14*). Thus the findings of Heimer and Larsson remained an enigma.

We report here that destruction of the focal point of the biogenic amine pathways in the anterior midbrain abolishes or severely curtails the postejaculatory refractory period and its concomitant postejaculatory vocalization and, in general, leaves other aspects of copulatory behavior unaltered.

Selection of male Wistar rats for the experiment was based on their ability to execute the complete pattern of male copulatory behavior on two baseline tests. These subjects received bilateral electrolytic lesions in either the area through which the biogenic amine pathways course [an area about the ventral portion of the medial lemniscus (VML)] or in other hypothalamic and midbrain loci (*15*). Postoperatively, animals were tested two or three times weekly for 1 to 4 weeks, depending on the time required for them to achieve a stable and repeatable level of sexual performance (*16*). Standard measures of male copulatory behavior were recorded through two ejaculatory series as follows: intromission frequency, the number of intromissions to ejaculation; ejaculation latency, the time from the first intromission until ejaculation (a rate measure of copulatory activity, the intercopulatory interval, is obtained by dividing ejaculation latency by intromission frequency); and postejaculatory interval, the time from ejaculation until the next intromission (the time from ejaculation to the next mount was also noted). The occurrence and timing of the postejaculatory vocalization was recorded in terms of vocalization latency and vocalization termination; respectively, these are the times from ejaculation until the beginning of the vocalization and its termination. The ultrasound was monitored with a capacitance microphone, the output of which was displayed on an oscilloscope screen (*17*).

Twenty-three of the animals regained good postoperative health and all exhibited complete mating behavior. The remaining six did not survive. The animals were divided into four main groups based on the location of their lesions. Those animals

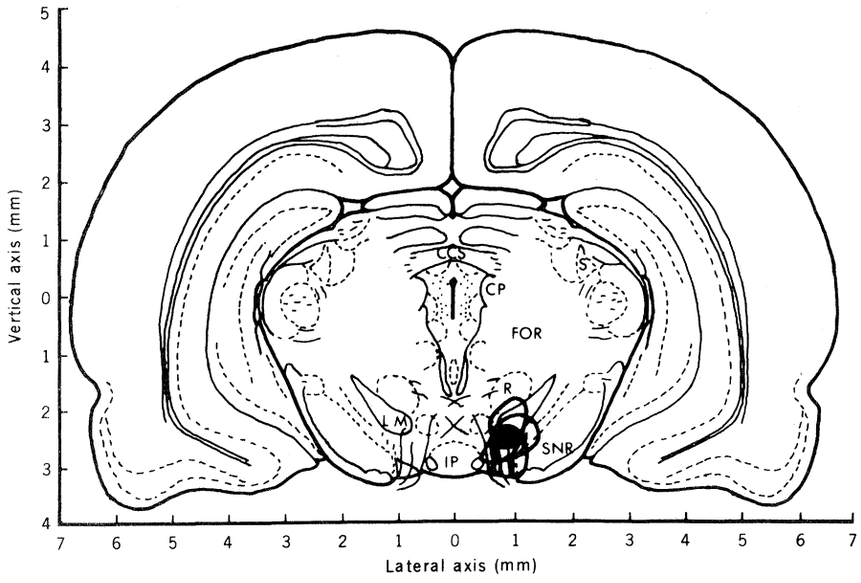

Fig. 1. A transverse section through the rat brain 1.9 mm anterior to the zero plane [from König and Klippel (*15*)]. The lesions of five of the six bilaterally lesioned rats of group A are shown, and the black area indicates where all the lesions overlap. The lesions were all at their largest between 1.5 and 2.4 mm anterior to the zero plane. *CP*, posterior commissure; *FOR*, reticular formation; *LM*, medial lemniscus; *SNR*, substantia nigra, reticular zone; and *R*, nucleus ruber.

with bilateral destruction of the area just ventral to the medial lemniscus in the rostral midbrain (see Fig. 1) showed an extreme reduction of the postejaculatory interval ($P < .001$) (see Table 1). Furthermore, the postejaculatory vocalization was abolished in four of seven of these rats and significantly reduced in the remaining three ($P < .01$). Other parameters of copulatory behavior were not significantly altered. Animals with lesions placed anteriorly or ventrally to the critical site or with lesions in the midbrain raphe area showed no substantial change in copulatory performance.

The performance of the VML-lesioned males reveals the extremes of the effect. Five of these males displayed refractory periods (from ejaculation to the next mount) of 40 to 90 seconds on one or more occasions (*18*). The minimum postejaculatory intervals for these animals were 65, 75, 90, 120, and 130 seconds, respectively. The refractory periods following the second ejaculations were also reduced below normal values. In one extraordinary test 17 ejaculations were recorded and the longest postejaculatory interval was 390 seconds. During the tenth interval the tail of the rat was pinched, and this resulted in a striking reduction of that interval (140 seconds) as compared with the preceding and following ones.

The lesions that eliminated or drastically reduced the postejaculatory refractory period were located just ventral to the medial lemniscus in the rostral midbrain behind the posterior hypothalamus (see Fig. 1) (*19*). This location corresponds precisely with that indicated by Ungerstedt (*7*) as an area through which biogenic amine pathways converge to course from the midbrain into the hypothalamus and also as a site of dopaminergic cell bodies. The obvious interpretation, therefore, is that the observed behavioral effect was due to destruction of these structures. The refractory period is essentially a sleeplike state (*4, 5*), and the midbrain systems apparently regulate sleep by transmission of biogenic amines to telencephalic loci (*8*).

One can speculate that the depletion or reduction of one or more of these amines is responsible for the observed effect. Serotonin, a likely candidate for regulator of the refractory period, seems not to be involved for two reasons. (i) *p*-Chlorophenylalanine,

Table 1. Copulatory performance of male rats before and after lesions in the rostral midbrain, posterior hypothalamus, and midbrain raphe area. Scores before the lesions (Pre) were based on the means of two tests for each subject; scores after the lesions (Post) were based on a mean of three tests per subject. All values are given as the mean ± standard error of the mean. Abbreviations: *N*, number of animals; IF, intromission frequency; ICI, intercopulatory interval; VL, vocalization latency; VT, vocalization termination; and PEI, postejaculatory interval.

Location of lesion	Time of test	Measures of copulatory behavior				
		Mean IF	Mean ICI (sec)	Mean VL (sec)	Mean VT (sec)	Mean PEI (sec)
			Group A			
Ventral medial lemniscus ($N = 7$)	Pre	11.1 ± 0.9	44.4 ± 7.7	33.1 ± 4.3	252.3 ± 30.9	302.8 ± 22.6
	Post	9.8 ± 1.8	39.6 ± 11.9	40.3 ± 6.7	118.0 ± 30.3*†	141.7 ± 12.9†
			Group B			
Ventral to area A ($N = 7$)	Pre	12.3 ± 0.5	37.3 ± 4.0	44.4 ± 7.4	249.1 ± 18.2	342.1 ± 22.3
	Post	11.7 ± 1.2	44.4 ± 6.1	52.7 ± 5.3	242.8 ± 23.2	368.3 ± 47.3
			Group C			
Posterior hypothalamus (1 to 2 mm anterior to A) ($N = 3$)	Pre	16.3 ± 2.9	45.3 ± 18.6	38.0 ± 4.6	253.0 ± 28.4	320.3 ± 34.0
	Post	10.3 ± 1.2	26.3 ± 2.8	52.7 ± 7.8	191.0 ± 31.0	262.7 ± 25.3
			Group D			
Midbrain raphe area ($N = 6$)	Pre	13.0 ± 1.5	53.7 ± 7.5	31.0 ± 2.6	302.0 ± 9.2	345.3 ± 11.0
	Post	10.8 ± 0.7	49.8 ± 6.5	43.7 ± 6.4	265.8 ± 18.3	322.3 ± 17.1

*$P < .01$. †These values were based on only three rats, since the others in this group did not vocalize. ‡$P < .001$; comparisons with preoperative scores by *t*-tests.

a serotonin depletor, has minimal effects on the postejaculatory interval (*20*) and no effect on the postejaculatory vocalization, especially in vigorous animals (*21*). (ii) Our lesions in the midbrain raphe system (an area rich in serotonergic cell bodies) did not affect this measure appreciably (*22*). Depletion of noradrenaline was correlated with deficiencies in sexual activity and unaltered postejaculatory intervals (*23*). In examining the biogenic amine pathways we found the most effective lesions were placed at the site of the dopaminergic cell bodies in the rostral midbrain. To suggest that dopamine normally inhibits male copulation is contrary to the findings of Malmnäs (*24*) who concluded, after altering catecholamine levels in the whole brain, that dopamine stimulates sexual behavior in male rats. However, several opposing neuronal systems may be involved in controlling sexual behavior, and dopamine may be a transmitter in more than one of these systems. Finally, since VML lesions probably also damage noradrenergic, serotonergic, and other pathways, the possibility of their involvement cannot be discounted (*25*).

The part of the refractory period during which vocalization appears, the absolute refractory period, is characterized by a predominance of rest behavior and sleeplike electroencephalographic activity (*4, 5*). Animals with reduced or abolished vocalization periods and reduced refractory periods showed little, if any, rest behavior; judging by overt behavior it is doubtful whether any sleeplike EEG would be evidenced in these animals. Since vocalization was absent or greatly reduced in these animals and they were behaviorally aroused, we assume that the absolute phase of the period is the one most affected and that what remains is mainly the labile, relative refractory period. The fact that a tail pinch was capable of reducing the refractory period of a late ejaculatory series to a minimal value supports our interpretation.

Regulation of the copulatory cycle is viewed as resulting from the interaction between facilitative and inhibitory processes (*4, 26*). The principal effect of the lesions described here was to abolish the profound inhibition that prevails during the refractory period. This same inhibitory process apparently does not regulate the temporal periodicity of the copulatory cycle, that is, the pacing of mount-bouts, since other parameters of sexual behavior were unaffected by the lesions.

In contrast, the massive lesions of the posterior hypothalamic-midbrain junction of Heimer and Larsson (*2*) not only reduced the refractory period but also caused a facilitation of copulatory rate and a reduction in intromission frequency. It is possible that their large lesions destroyed additional inhibitory structures or pathways, involved in the regulation of the copulatory cycle, which were not destroyed by the more circumscribed lesions that we used. A better understanding of the inhibitory mechanism (or mechanisms) that regulate male copulatory behavior will come from further analyses of this system.

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15. J. F. R. König and R. A. Klippel, *The Rat Brain: A Stereotaxic Atlas* (Williams & Wilkins, Baltimore, 1963). The critical coordinates for the VML area were 2 mm anterior to the zero plane, 1 mm lateral to the midline, and 8 mm down from the surface of the cortex. The coordinates for the midbrain raphe area were 0.3 mm anterior to the zero plane, on the midline, and 4.5 mm (dorsal raphe) and 6.5 mm (medial raphe) down from the surface of the cortex. The current used was 900 μa of cathodic current for 3 minutes on each side.
16. Lesioned animals sometimes showed transient deficits in copulatory behavior. Frequently their performance improved with time and then stabilized. Performance was considered stable and repeatable when (i) the animal did not continue to show obvious improvement from test to test and (ii) the variability among three or more consecutive tests was comparable to that in control tests in the same population, according to the judgment of the experimenter.
17. Ultrasounds were recorded with a condenser microphone constructed after the plans of J. J. G. McCue and A. Bertolini [*IEEE Trans. Sonics Ultrason.* **SU-11** (1964), p. 41]
J. D. Pye and M. Flinn [*Ultrasonics* **2**, 23 (1964)]. The microphone output was fed into a preamplifier built according to the pla
J. D. Pye [*Ultrasonics* **6**, 32 (1968)]. The resulting signal was displayed on a Tektronix oscilloscope.
18. In preoperative tests this measure did not differ significantly from the postejaculatory interval, and in no case was less than 220 seconds.
19. After long-term estrogen treatment female rats execute the complete male ejaculatory pattern. The refractory period of these females is extremely short, in some cases approaching those of the lesioned males in the present study, and they do not exhibit the postejaculatory vocalization. These females possess large pituitary tumors and substantial destruction of the basal posterior hypothalamus and midbrain [D. E. Emery and B. D. Sachs, *Science*, in press]. It is possible that the extraordinary postejaculatory behavior of these females was due to destruction of the same area of the brain as in our lesioned animals.
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† We are saddened by the death of Peter McDonald and his wife Wendy in an airline crash on 3 March 1974.

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Social Modification of Alcohol Consumption in Inbred Mice

Abstract. *The strain-specific "preference" for, or "aversion" to, an alcohol solution in a choice situation on the part of C57BL and DBA mice is believed to be under genetic control. But social rearing conditions are now shown to alter the voluntary consumption of alcohol, so that DBA weanling mice housed for 7 weeks with adult C57BL mice increase—and C57BL weanling mice housed with DBA adults decrease—their alcohol intake. Although substantial and highly significant changes in alcohol self-selection occur, strain-specific phenotypes are not reversed.*

Social facilitation, a specific type of interindividual interaction, has been defined (*1*) as ". . . any increment or decrement in an individual's behavior resulting from the presence of another organism." The exact nature or specific role of the companion in influencing behavior is, however, seldom asserted in general definitions.

In addition to man, the phenomenon has been observed in many species in a variety