(*12*), whole brains were immersed in a saturated solution of potassium permanganate in deionized water (*p*H 7.0). Fixation was allowed to proceed for 1 hour at 4°C. Large arteries from the base of the brain and smaller branches over the convexity of the hemispheres were dissected, dehydrated, and embedded in Epon 812 or Araldite 502 resin. Thick sections were prepared for phase-contrast microscopy, and ultrathin sections were examined in a Philips 200 electron microscope.

Axons are embedded in Schwann cell cytoplasm which is absent on the side adjacent to the smooth muscle cell (Fig. 1). There are no intervening collagen bundles or fibroblast processes, and only the coalesced axonal and smooth muscle cell basement membrane separates the plasmalemmas of the axon and smooth muscle cell. Some axons contain increased numbers of mitochondria as well as concentrations of synaptic vesicles. Axons with at least two different populations of vesicles occur. Certain axons characteristically possess many small granular vesicles (about 500 Å) and occasional agranular vesicles of varying sizes (including those of 300 to 500 Å in diameter). In others, small (500 Å) granular vesicles are absent. Occasional large (1000 Å) granular vesicles occur in both types of axons.

Neuromuscular contacts were present in the adventitia of both the large vessels at the base of the brain and smaller arteries over the convexities of the hemispheres. Axons with these characteristics were seen as close as 780 Å from the smooth muscle cell or as far away as 3000 Å. The more distant neuromuscular contacts were separated from smooth muscle cells by collagen or fibroblast processes.

The significance of these findings depends upon the certainty of the identification of noradrenalin within the smaller granular vesicles (*13*). Investigations combining histochemical, electron microscopic, or autoradiographic techniques would seem to establish beyond reasonable doubt that noradrenaline is present in the small (500 Å) granular vesicles (*14*). Thus, granular vesicles can be identified as sympathetic or noradrenergic. One might also speculate that axons lacking granulated vesicles of this size but containing concentrations of granular vesicles, shown in other locations to contain acetycholine (*15*), are cholinergic or parasympathetic.

A single muscle cell may receive neural influences from more than one axon (*16*). Furthermore, in arteries of other organs, at least, neuromuscular contacts do not represent nerve endings or nerve terminals but specialized areas along the course of the axon (*16*). Thus, these contacts are of the *en passage* variety. Because there are no convincing micrographs of nerves or neuromuscular contacts within the media, it is apparent that the vast majority of smooth muscle cells in the arterial media do not receive direct innervation. The distance across which a neurotransmitter substance might diffuse and cause excitation of smooth muscle cells is not known. It would appear that the morphological substrate for effective nervous control of cerebral blood flow exists on intracranial arteries, as on arterial vessels in other organs (*10*, *16*, *17*).

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Olfactory Bulb Removal Eliminates Mating Behavior in the Male Golden Hamster

Abstract. *Mating behavior in sexually naive and sexually experienced male golden hamsters was totally eliminated by removing their olfactory bulbs. In contrast, the mating behavior in blinded, unilaterally bulbectomized, and sham-operated controls was essentially normal. Testosterone injections restored normal sexual behavior in castrated controls but had no effect on the bilaterally bulbectomized animals.*

It has long been believed that no one sensory modality is essential to a mammal's sexual behavior. This was established for the rat and rabbit, and it was particularly emphasized that olfactory information was not required for normal sexual behavior in these animals (*1*). While this idea is often expressed as a general truth for the sexual behavior of all mammals, the importance of olfaction for sexual behavior has been tested adequately in only a few species (*2*). We now offer evidence that, in at least one mammal, the ability to smell is a prerequisite for mating.

When the receptive female golden hamster (*Mesocricetus auratus*) is sexually stimulated, an abundant, highly odorous substance, which has a stimulating or releasing effect on the sexual behavior of the male hamster, exudes from her genital region. The male hamster sniffs and licks this secretion before and intermittently throughout mating (Fig. 1). If we place a male or female hamster into the cage of a male hamster, the intruder will be treated quite differently depending on its sex. But if we place some of the female genital secretion on the fur of a male intruder, he will be treated like a female, and the other hamster will attempt to mate with him (*3*). Discrimination of sexual identity and arousal to

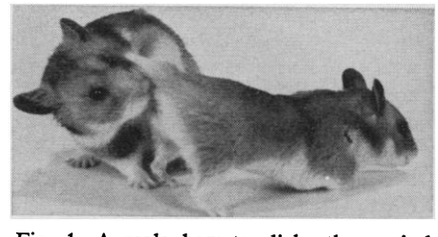

Fig. 1. A male hamster licks the genital secretion of a receptive female hamster prior to mating. This activity, along with other aspects of mating behavior, was eliminated after removal of olfactory bulbs.

sexual activity therefore seem to depend to a large extent on the presence of a particular olfactory stimulus. In this regard the genital secretion of the female hamster acts like a mammalian pheromone.

After we noted the importance of odors in the sexual behavior of hamsters, we wanted to determine whether the sense of smell might be an absolute prerequisite for mating in this species. To test this hypothesis we removed olfactory bulbs from five sexually naive and from five sexually experienced male hamsters (*4*). After the operation sexual behavior was absent in all of these animals. In 9 to 12 exposures to a receptive female, occurring 2 to 15 weeks after surgery, none of them showed the slightest sexual interest in the female. Nevertheless, the bulbectomized hamsters were active, and at perfusion their body weights were normal for hamsters of their age.

Removal of the olfactory bulbs not only eliminates the ability to smell, but also causes a general trauma and substantial damage to brain tissue (*5*). To test if these factors alone could be responsible for the loss of sexual behavior we sham-operated four animals, blinded four animals, and removed the olfactory bulb unilaterally from four others, all males. All of these hamsters mated normally after the operation (*6*).

To determine whether the sexual deficit was hormonal in origin we studied the sexual behavior of four castrated hamsters. By the fourth test of mating ability after the operation, the castrates ceased intromissions but continued mounting the female and investigating her genital area almost as much as before the operation. This degree of sexual activity was, of course, much higher than the total loss observed after bulbectomy. Exogenous testosterone injections restored the castrates to their full sexual capacity but had no effect on the sexual inactivity of bulbectomized animals (*7*). A summary of all these experiments is given in Table 1.

Table 1. Summary of results from all experiments on sexual behavior. Column headed "N" indicates number of animals in each group. Mating experience (M.E.) indicates whether or not animals were sexually experienced before treatment. Tests indicate number of 10-minute tests of mating behavior given to each animal in that group.

Treatment	N	M.E.	Tests (No.)	Sexual behavior
Sham-operated	4	No	6	Normal
Olfactory bulbs removed	5	No	9	None
Castrated	4	Yes	4	Intromissions declined to almost zero Mounts and genital licks remained high
Castrated; given testosterone (*7*)	4	Yes	1	Normal (completely restored)
Olfactory bulbs removed	5	Yes	12	None
Olfactory bulbs removed; given testosterone (*7*)	5	Yes	1	None (no change)
Eye enucleation	4	Yes	1	Normal
Unilateral olfactory bulb removal	4	Yes	3	Normal

To gain additional understanding of the behavior of bulbectomized hamsters, we systematically observed four normal and five bulbectomized hamsters in other situations where we thought olfaction might be important. A normal hamster usually scent-marks his home cage as his territory (*3*). By comparing the behavior of hamsters in their home cages with their behavior in the vacated cages of other hamsters, we easily determined that normal hamsters can distinguish their own cage-territory from that of others. Hamsters also have typical patterns of social behavior. When two males meet there is usually a period of upright greeting and a thorough mutual investigation, including sniffing of the flank glands, ears, and genital area. Then may come an attack and a fight, after which the loser assumes the submissive posture and shows signs of fear, such as squealing or defecation. Some hamsters exhibit fear responses upon initial contact with another hamster, without fight or provocation. All of these territorial and social behaviors were absent or markedly altered after bulbectomy. When placed in another hamster's cage, bulbectomized hamsters behaved as if they were in their own. They did not attack other hamsters and rarely greeted or investigated them. When bulbectomized hamsters were attacked they usually ignored the attacker and tried to pull away. If unable to avoid a fight, they defended themselves but showed little counteraggression. They rarely exhibited lasting signs of fear or submission even after being vigorously attacked. Instead of social behavior, the bulbectomized hamster showed the activities of a normal hamster when alone in its home cage—exploring, eating, grooming, arranging food hoard or nest, or sleeping.

In general the behavior of the bulbectomized male hamster was apathetic and unemotional toward both male and female hamsters. The loss of mating behavior must therefore be viewed as one aspect of a more general syndrome. The simplest explanation is that the bulbectomized hamster has been denied a crucial source of information or stimulation that he requires to respond normally to members of his own species. If this is true, then peripherally produced anosmia should have a behavioral effect similar to that of bulbectomy. It should also be possible to elicit some of the absent behaviors by electrical or chemical stimulation of the brain since, according to this hypothesis, the peripheral trigger for the behaviors—not the central mechanism—has been destroyed.

Until the present study it has been consistently reported that no one sensory modality is essential for mammalian mating behavior, but the results presented here indicate tha tthe male hamster that cannot smell will not mate. It thus appears that the hypothesis of "multisensory dependence" of mammalian sexual behavior may have been a premature generalization based on data from an insufficient number of species.

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2. L. Heimer and K. Larsson [*Physiol. Behav.* **2**, 207 (1967)] and G. Bermant and L. Taylor [*ibid.* **4**, 13 (1969)] have shown that the sexually experienced rat is not entirely unaffected by olfactory bulb removal. They found that after surgery there was a greatly increased latency to first ejaculation, even though latencies to first mount and intromission and the frequencies of ejaculation, mounts, and intromissions were not changed. This deficit is not only much smaller than that observed in the hamster, it is also basically different. An isolated change, like delayed latency to first ejaculation, is difficult to attribute to loss of the male's ability to smell the female, whereas the total elimination of sexual behavior that we observed in the bulbectomized male hamster is most satisfactorily explained on this basis.
3. These observations were reported by F. Dieterlen [*Z. Tierpsychol.* **16**, 47 (1959)] and have been repeated several times in our laboratory.
4. To excise the olfactory bulbs, a dental air drill was used to remove the overlying skull, and the olfactory bulbs were removed by suction. For the sham operations the bone, but not the bulb tissue, was removed. Pentobarbital sodium (Nembutal, 100 mg/kg) was used as anesthetic. The hamsters were housed individually in plastic cages with cedar shavings for bedding. Females were made receptive by subcutaneous injection with 0.10 ml of Progynon

benzoate (Schering; estradiol benzoate in oil, 1.0 mg/ml) 48 hours before testing and 0.05 ml of Proluton (Schering; progesterone in oil 25.0 mg/ml) 3 hours before testing. When the females were proved to be receptive with a stud male they were placed in the home cage of the male to be tested. Tests of mating behavior lasted 10 minutes, and during this time the latency, duration, and number of occurrences of relevant behaviors were marked on a continuous event recorder. Tests were at least 5 days apart, and no male was tested with the same female twice in succession. Animals that were sexually experienced had mated at least four times prior to the operation and were known to be sexually vigorous.

5. The brains of all operated animals were prepared for histological study. The animals were perfused transcardially with 0.9 percent saline followed by 10 percent formalin. The brains were placed in 10 percent formalin for at least 1 week and then in a 30 percent sucrose solution, made with 10 percent formalin, for at least 5 days. The brains were then embedded in albumin-gelatin and were sectioned on a freezing microtome at 25-μm thickness. Serial sections were stained with cresylecht violet for cell bodies and by modified Nauta techniques for degenerating axons and normal fibe → [R. P. Fink and L. Heimer, *Brain Res.* 4, 369 (1967); G. Schneider, *Science* **163**, 895 (1969)]. In the smallest lesion, pars rostralis of the anterior olfactory nuclear complex was destroyed and pars externa, pars lateralis, and pars dorsalis were damaged. Some apparently normal bulb tissue remained, but we do not know if this was functional. In the largest lesion, in addition to the above, pars externa and pars ventralis of the anterior olfactory nuclear complex were also destroyed and pars medialis was damaged as was the olfactory tubercle; no bulb tissue remained. Since the total absence of sexual behavior was equivalent in all ten animals, there was no correlation between severity of the deficit and size of the lesion.
6. In addition to our own negative results from unilateral bulb removal and bilateral eye enucleation, B. N. Bunnell, J. Friel, C. K. Flesher [*J. Comp. Physiol. Psychol.* **61**, 492 (1966)] also showed that the sexual behavior of hamsters is not especially sensitive to brain lesions. They found that dorsally placed lesions involving loss of 15 to 25 percent of the total cortex of the male hamster had only slight effect on his sexual behavior and that there was no correlation between the size of the lesion and the change in sexual behavior after the lesion.
7. Daily subcutaneous injections of 5 mg of testosterone propionate in oil were given to both bulbectomized and castrated animals for 7 days.
8. Supported by NSF predoctoral fellowship to M.R.M.; PHS grant NB-06542 to W. J. H. Nauta; NASA grant NGR 22-009-308 to H. L. Teuber.

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"Psychogenic" Death: A Reappraisal

Abstract. *Social, economic, and medical variables correlated with "psychogenic" death rates across about 30 countries. However, McClelland's psychological motives of achievement, affiliation, and power, determined for each country by content analysis of children's stories, did not. Status integration correlated positively with homicide and negatively with deaths from suicide and ulcers. Low life expectancy, wealth, economic growth, wine consumption, and zinc (cadmium) consumption correlated with deaths from homicide, suicide, ulcers, cirrhosis, and hypertension, respectively.*

Rudin (*1*) related five "psychogenic" death rates in various nations with each other and with the McClelland (*2*) motive scores derived from children's literature. The motives for achievement (success at task performance), for affiliation (interaction with others), and for power (control over others) were obtained by McClelland through a standardized scoring procedure (*2*). Rudin made rank-order intercorrelations of death rates in 17 countries around 1950 and found two clusters: (i) homicide, suicide, and cirrhosis of the liver ("aggressiveness cluster"), and (ii) ulcers and hypertension ("inhibition cluster"). He proposed that high power motivation in 1925 led to high rates of aggressive deaths in 1950, while high achievement motivation led to high inhibitive death rates.

In support of the two hypotheses, Rudin found that power motivation predicted deaths from suicide (Spearman rank-difference correlation, $\rho = .52$), and achievement motivation to predict rates of death from ulcers ($\rho = .57$) and from hypertension ($\rho = .52$). Other correlations between 1925 motives and 1950 death rates were not significant.

Rudin's (*1*) findings were marred by inadequacies in methodology and theory. In examining the Rudin hypotheses, we tested empirically an alternate hypothesis—namely, that social, economic, and medical variables, most of which have been studied previously, account for the differences in death rates cross-nationally. We show that the Rudin death clusters are inappropriate and that the psychological motives are unrelated to death rates. Also, we find that social, economic, and medical variables explain much of the differences in death rates.

Use of all data on death rates which were available from United Nations publications (*3*) for the 41 countries studied by McClelland (*2*) allowed us to expand the examination to 31 to 34 countries for 1950 and 1965. The number of countries sampled is critical. Failure to consider all available data in cross-national research can lead to results unrepresentative of broader world samples (*4*). It is also important that over a period of time the relations among death rates be stable. If one death rate correlates with another in 1950 but not in 1965, it indicates that part of the cluster is unstable.

Intercorrelations (product-moment) of the death rates across 31 to 34 countries in 1950 and in 1965 disclosed only one significant stable relation, that between suicide and ulcers ($r = .42$ and .51). This lack of consistent correlation (except for suicide with ulcers) contradicted Rudin's (*1*) finding of separate aggressiveness and inhibition clusters, because the factors making up those clusters do not correlate significantly at both points in time. The stable cluster (suicide with ulcers) that was obtained suggested an intropunitive response, or the directing inward of aggressive impulses (*5*).

Neither McClelland (*2*) nor Rudin (*1*) was certain whether the 1925 and 1950 psychological motives represent more the values of adults at the time the children's books were current, or the later values of the adults who were exposed to the books as children. Rudin assumed the latter, and tested his hypotheses with a lag model. Our analysis allowed for both possibilities and empirically tested the concurrent and the lag model with all combinations of both 1925 and 1950 motives and 1950 and 1965 death rates. Our evaluation included 22 to 24 of the 25 countries for which McClelland reported 1925 motives, and 31 to 34 of the 41 McClelland countries of 1950. The correlation coefficients (product-moment) between national motives in 1925 and 1950 and national death rates in 1950 and 1965 are shown in Table 1.

If the Rudin hypotheses were to be supported by this expanded analysis, 1925 power motivation would correlate positively and significantly with death rates in 1950 from homicide, suicide, and cirrhosis; achievement motivation would correlate positively and significantly with death rates in 1950 from ulcers and hypertension. Of the five death rates in 1950, only suicide was correlated with a motive as hypothesized. If Rudin's 25-year lag between motives and deaths could be extended to a 15- to 40-year lag, then correlations supporting the Rudin hypotheses would be expected from 1950 motives with 1965 deaths, and from 1925 motives with 1965 deaths. Of ten such relations which might support Rudin, only the positive correlation of 1950 power motive with 1965 homicide did so.

Further, if the model viewing the