

Olfactory Bulbectomy, Peripheral Anosmia, and Mouse Killing and Eating by Rats

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Seventeen rats which had not killed a mouse in 6 wk sustained bilateral olfactory bulbectomies. Fifteen of these started to kill after surgery, and nine consistently killed on the same day the mouse was introduced. Twelve nonkillers sustained surgical ablation of the olfactory receptors. None of these killed after surgery. Of eight spontaneous killers which consistently killed on the day a mouse was introduced, four sustained receptor ablations, and three, bulbectomies. One remained an unoperated control. All killed as quickly and as consistently as before. Most bulbectomized rats employed an "emotional," messy style of killing, and bulbectomy-facilitated killers ate less of their prey than did spontaneous killers. Deafferentation had no effect on killing or eating. This is interpreted as evidence for irritable aggression on the part of bulbectomized rats, and for a nonolfactory role of the olfactory bulbs.

There have been numerous reports that olfactory bulbectomy increases the percentage of rats which kill mice (Alberts and Friedman, 1972; Bernstein and Moyer, 1970; Cain and Paxinos, 1974; Eichelman, Thoa, Bugbee, and Ng, 1972; Karli, Vergnes, and Didiergeorges, 1969; Kumadaki, Hitomi and Kumada, 1967; Spector and Hull, 1972; Thorne, Aaron, and Latham, 1973; Ueki, Nurimoto, and Ogawa, 1972). Several of these authors reported that bulbectomy-facilitated killers adopted an emotional style of killing, readily distinguishable from the relatively clean, quick kills of naturally muricidal rats. However, Thorne *et al.* (1973) found increased killing without increased emotional behavior, and Bandler and Chi (1972) found that while bulbectomy facilitated killing in animals which previously were nonkillers, it somewhat decreased that of spontaneous killers. Nevertheless, because of the style of killing observed in many bulbectomized rats and the correlation with irritability (Bernstein and Moyer, 1970; Cain, 1974; Didiergeorges, Vergnes, and

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Karli, 1966), it has been suggested that bulbectomy-facilitated muricide should not be classified as predatory aggression but rather as an instance of irritable aggression. (See Moyer, 1968, for discussion of different types of aggression.) If this is the case, both the behavioral components of the kill and the consumption of the prey would be expected to be different from the natural predatory kill. However, it may be that once killing has been initiated out of irritability, the response of eating the prey would be sufficient reinforcement for the muricidal response and all irrelevant responses would be extinguished. In this case, the bulbectomized killer would become indistinguishable from the natural predatory killer. Furthermore, if a natural predatory killer is bulbectomized, one might expect him to become more emotional in his kills after surgery, followed perhaps by a return to the normal predatory response.

Since peripherally induced anosmia does not lead to increases in killing or irritability (Alberts and Friedman, 1972; Cain and Paxinos, 1974; Spector and Hull, 1972), it appears that anosmia per se is not the cause of the behavioral changes. However, Alberts and Friedman (1972) and Cain and Paxinos (1974) utilized zinc sulfate for induction of peripheral anosmia, and a question has arisen as to the side effects of this procedure. Sieck and Baumbach (1974) reported that even though their zinc sulfate-treated animals appeared generally normal, they exhibited weight loss and periods of lethargic inactivity, both of which were mimicked by systemic injections of zinc sulfate. Thus, failure to kill mice may have been correlated with these systemic effects. In the Spector and Hull (1972) experiment, peripheral deafferentation was achieved by surgical removal of the nasal mucosa, and did not facilitate mouse killing. However, since the number of animals in that experiment was small, further corroboration of behavioral effects of surgical deafferentation would lend additional support to the hypothesis that anosmia per se does not facilitate muricide.

In no previous study has a systematic observation been made over a number of weeks of the killing and eating responses of bulbectomized, peripherally deafferented, and control rats. The present study was undertaken to investigate these responses and to further elucidate the role of anosmia as separate from central brain damage in producing the behavioral effects. Both spontaneous killers and nonkillers were studied before and after bulbectomy or surgical deafferentation in order to discover any differences in latency, consistency, or method of attack, or pattern of eating the mouse after the kill.

METHOD

Subjects. Fifty-seven male Sprague-Dawley rats, from Chordata, Inc., were maintained in individual metal cages, 18 X 20 X 25 cm. Food and water were available ad lib. The mice were Swiss albino males.

Procedure. After the rats had lived in their cages for 2 days, a mouse was introduced into every cage. The mouse was left in the cage either until it was killed or until 3 wk had elapsed, at which time a new mouse was substituted and left for 3 more wk. If a rat did not kill once in the 6 wk, it was classified as a nonkiller. Bilateral olfactory bulbectomies were performed on 21 nonkiller rats and on 3 spontaneous killers which consistently killed on the same day the mouse was introduced. Eleven nonkillers and four consistent spontaneous killers were subjected to surgical removal of olfactory mucosa and afferents. Twelve nonkillers and five spontaneous killers were maintained as unoperated controls.

Sodium pentobarbital (35 mg/kg) and atropine sulfate (.30 mg) were administered prior to surgery. Olfactory bulbs were aspirated in the usual manner. Peripheral deafferentation was accomplished by aspirating all tissue in the nasal cavity rostral to and beneath the cribriform plate. In addition, the aspirating needle was passed systematically over the cribriform plate to remove any remaining axons. Gel Foam was used to obtain hemostasis and Sulfathiazole was sprinkled on the wound to minimize infection. After the skin was sutured, any blood present in the animal's trachea and external nares was aspirated.

After a 7-day recovery period a new mouse was placed into each rat's cage and allowed to remain either until it was killed or 3 wk had passed, at which time it was replaced with a new mouse. This procedure continued until the end of the study (12 wk after recovery) or until the rat would consistently kill a mouse the same day it was introduced. When a rat had established a pattern of relatively short latency attacks, several kills were observed and the latency before attack, ferocity of attack, number of bite marks on the mouse, and amount of the mouse eaten were recorded immediately, $\frac{1}{2}$ hr, 2.5 hr, and 24 hr after the kill. Mice were removed and not returned to the cage, so that scores for the different observation times were for different mice. Photographs of the dead mice were taken at these times (except for the 24-hr observation) and later shown to two naive observers, who were asked to judge number of bloody patches on the fur, and amount of the mouse eaten. A single-blind procedure was used with these observers. The averages of the two scores for each mouse were compared by means of a one-way analysis of variance.

Forty-four (81%) of the original animals were nonkillers. Of these, 21 received bilateral bulbectomies and 11 were peripherally deafferented. One peripherally deafferented and four bulbectomized animals died during or shortly after surgery. Twelve nonkillers were kept as unoperated controls and did not kill any mice during the entire experiment (19 wk). Thirteen (19%) of the original animals killed at least one mouse, and eight (12%) of these became consistent killers, launching their attack either immediately or within a few hours after introduction of each mouse. Of the eight consistent killers,

four were peripherally deafferented, three were bulbectomized, and one was kept as an unoperated control.

Near the termination of the experiment all bulbectomized and deafferented animals were tested for their ability to smell. Ten unoperated rats served as controls. A beaker containing 100% acetic acid was placed under a wire mesh floor at one end of a 6-ft runway. Fumes were removed by a ceiling exhaust fan which blew the air to the outside of the building. Each animal received two 5-min tests and was placed into the middle of the runway facing one direction for the first test and the opposite direction for the second. Length of time in the half of the runway nearest the acetic acid was recorded.

At the termination of the experiment the gross extent of surgery was verified by visual inspection.

RESULTS

As seen in Table 1, no peripherally deafferented nonkiller became a killer during the 12 wk after recovery. However, 15 (88%) of the bulbectomized animals killed at least one mouse during that time, and 9 of these became consistent killers with relatively short latency attacks upon each mouse presented. The difference between number of bulbectomized rats compared with peripherally deafferented rats which became killers after treatment was statistically significant ($\chi^2 = 18.54$, $df = 1$, $P < .001$).

All spontaneous killers killed as consistently after surgery as before. All of the peripherally deafferented killers continued to attack in the quick, well

TABLE 1

Number of Animals Killing Mice

Pretreatment mouse killing	Treatment	Animals killing at least one mouse after treatment	Animals killing consistently after treatment
44 Originally nonkillers	17 Bulbectomized	15 (88%)	9 (60%)
	11 Deafferented	0 (0%)	0 (0%)
	12 No operation	0 (0%)	0 (0%)
	5 Died		
13 Spontaneous killers	3 Bulbectomized	3 (100%)	3 (100%)
	4 Deafferented	4 (100%)	4 (100%)
	1 No operation	1 (100%)	1 (100%)
	5 Inconsistent killers—no operation	5 (100%)	0 (0%)

directed manner displayed by all the spontaneous killers before surgery. The average latency to attack was 4 min, 40 sec, with two animals consistently attacking within 30 sec and the other two delaying their attack for up to 30 min. No matter what the latency was, however, once the attack was launched it was always swift and well directed.

Of the three bulbectomized spontaneous killers, two began killing in a very bloody manner. The average latency to attack was about 40 sec with these animals, and their attack was generally less well directed and seemed to be a frenzied, thrashing type of attack. Since the third animal usually waited for several hours before attacking, his kills were not observed; however, his prey sometimes had many bite marks and sometimes only a few.

Of the 15 animals which killed only after bulbectomy, 9 became consistent killers, attacking each mouse the same day it was introduced. Five of these nine (55%) killed in a bloody manner with a "frenzied" or agitated type of attack. On several occasions as much as 15 min elapsed between onset of the attack and the death of the mouse, with the rat renewing his attack periodically. Also on several occasions the rat was observed to continue his attacks for as much as 5 min after the mouse was apparently dead. Three of the nine consistent bulbectomy-facilitated killers killed in an efficient manner with an attack similar to that of the spontaneous killers. One exhibited a long latency to attack, so that his kills were not observed; however, his prey usually had numerous bite marks. These nine animals fell into two categories with respect to average latency to attack. Five rats usually attacked within 1 min, with an average latency of 20 sec; the other four often waited more than an hour, with a mean latency of about 2.5 hr. There appeared to be no correlation between latency to attack and ferocity of the kill.

Naive observers' scores for amount of blood on the fur of dead mice were found to be significantly different ($F = 3.82$, $df = 3/34$, $P < .05$), when mice killed by the four types of killers were compared. Mice killed by bulbectomy-facilitated killers had an average of nine patches of blood on their fur; those killed by bulbectomized spontaneous killers had an average of six patches; those killed by deafferented and unoperated spontaneous killers each had an average of four patches.

Animals which killed only after bulbectomy ate an average of 53.1% of the bodies of their prey within 24 hr after the kill, compared with 78.8% eaten by deafferented natural killers and 78.2% consumed by bulbectomized natural killers. Since the percentages eaten by both groups of anosmic natural killers were so similar, their scores were combined and compared with those of the bulbectomy-facilitated killers. The difference was found to be significant at the .05 level ($t = 2.13$, $df = 44$). Combined scores for amount of the prey eaten after ½ and 2.5 hr showed an even greater difference ($F = 9.604$, $df = 3/25$, $P < .01$). Bulbectomy-facilitated killers ate an average of only 4% of each mouse, with most of these animals consuming none of their prey.

However, bulbectomized, deafferented, and unoperated spontaneous killers ate an average of 21, 27, and 24% of each mouse. Most often the head and neck of the mice were eaten, but occasionally the viscera were consumed in addition to or instead of the head.

Smell tests for anosmic animals were significantly different from those of unoperated animals ($t = 12.93$, $df = 17$, $P < .001$). Unoperated animals rather consistently avoided the half of the runway near the acetic acid, spending an average of 53 sec out of 5 min there. Anosmic animals were visibly less bothered by the acid, spending an average of 2 min, 4 sec in the half of the runway nearest the acetic acid.

Visual inspection of extent of bulb removal revealed no obvious differences among bulbectomized animals. All sustained complete ablations of the olfactory bulbs, including the accessory olfactory bulbs, comparable to Sieck and Baumbach's (1974) Deep Bulbectomy group. Most animals sustained some damage to the lateral olfactory tract and anterior olfactory nucleus. As in Cain's (1974) experiment, there appeared to be no correlation between extent of surgical damage and postoperative killing.

DISCUSSION

As in a previous study (Spector and Hull, 1972) surgical removal of olfactory mucosa did not facilitate mouse killing in any rat which did not originally kill mice. Bulbectomy, however, did facilitate the muricidal response in 88% of the previous nonkillers. Approximately half of all the bulbectomized killers, including both bulbectomy-facilitated and bulbectomized spontaneous killers, consistently employed a lengthy, agitated manner of killing. A few killed in a quick, clean manner; the rest killed either inconsistently or after such long latencies that their attacks were not observed. All of the deafferented and unoperated spontaneous killers displayed the quick clean kill. Since surgical deafferentation did not interfere with spontaneous muricide, the failure of deafferented nonkillers to kill mice cannot be attributed to any disability wrought by the surgery. Furthermore, bulbectomy-facilitated killers ate much less of their prey than did all types of spontaneous killers. Many mice killed by bulbectomized animals were found the following day with bite marks on their bodies, but with none of the carcass eaten.

The bulbectomized spontaneous killers provide an interesting case. After surgery they usually killed in the messy fashion typical of bulbectomy-facilitated killers; however, they ate the same percentage of their prey as did deafferented and unoperated killers. It would appear that they had had sufficient experience eating prey before surgery to convince them of the palatability of the food. It is possible that our bulbectomy-facilitated killers

would have eventually developed consummatory responses similar to those of natural killers. However, we have demonstrated that for many bulbectomy-facilitated killers, at least the initial attacks (over a period of several months) are not motivated primarily by predatory goals.

Paul (1972) suggests that even among normal rats, mouse killing should not be considered strictly a predatory response, insofar as it does not always obey the laws governing eating behavior. However, hunger, unlike other drives, had potentiated killing by her rats, and killing was usually followed by eating of the prey. Thus, while muricide probably should not be considered simply a response preparatory to eating, it is nevertheless closely associated with eating in the normal rat. Since a few of our bulbectomy-facilitated killers ate as much or almost as much of their prey as did spontaneous killers, we cannot rule out predation as a factor in killing facilitated by bulbectomy. However, since several bulbectomy-facilitated killers never ate any part of a mouse, and others ate either sporadically or smaller fractions of the mice, and since they often killed in a more bloody, "emotional" fashion, we suggest that muricide is even less closely tied to predation in these animals than in normal rats.

The present study supports the following hypotheses: 1. Anosmia per se does not facilitate mouse killing; muricide facilitated by bulbectomy is related to destruction of central nervous tissue presumably having a tonic inhibitory influence on the rest of the limbic system. 2. The relative weight of irritable, as compared with predatory, components of muricide is greater in bulbectomy-facilitated killers than in spontaneous killers. 3. The previously observed differences between central and peripheral anosmia in mouse killing experiments was not due merely to systemic poisoning by zinc sulfate.

REFERENCES

- Alberts, J. R., and Friedman, M. I. (1972). Olfactory bulb removal but not anosmia increases emotionality and mouse killing. *Nature (London)* 238, 454-455.
- Bandler, R. J., Jr., and Chi, C. C. (1972). Effects of olfactory bulb removal on aggression: A reevaluation. *Physiol Behav.* 8, 207-211.
- Bernstein, H., and Moyer, K. E. (1969). Aggressive behavior in the rat: Effects of isolation and olfactory bulb lesions. *Brain Res.* 20, 75-84.
- Cain, D. P., (1974). Olfactory bulbectomy: Neural structures involved in irritability and aggression in the male rat. *J. Comp. Physiol. Psychol.* 86, 213-220.
- Cain, D. P., and Paxinos, G. (1974). Olfactory bulbectomy and mucosa damage: Effects on copulation, irritability, and interspecific aggression in male rats. *J. Comp. Physiol. Psychol.* 86, 202-212.
- Didiergeorges, F., Vergnes, M., and Karli, P. (1966). Privation des afférences olfactives et agressive interspecificque du rat. *C. R. Sci. Soc. Biol.* 160, 866-868.
- Eichelman, B., Thoa, N. B., Bugbee, N. M., and Ng, K. Y. (1972). Brain amine and adrenal enzyme levels in aggressive, bulbectomized rats. *Physiol. Behav.* 9, 483-485.
- Karli, P., Vergnes, M., and Didiergeorges, F. (1969). Rat-mouse interspecific aggressive behavior and its manipulation by brain ablation and by brain stimulation. In S. Garattini and E. B. Sigg (Eds.), "Aggressive Behavior." New York: Wiley.

- Kumadaki, N., Hitomi, M., and Kumada, S. (1967). Effect of psychotherapeutic drugs on hyperemotionality of rats in which the olfactory bulb was removed. *Jap. J. Pharmacol.* 17, 659-667.
- Moyer, K. E. (1968). Kinds of aggression and their physiological basis. *Comm. Behav. Biol.* 2, 65-87.
- Paul, L. (1972). Predatory attack by rats: Its relationship to feeding and type of prey. *J. Comp. Physiol. Psychol.* 78, 69-76.
- Sieck, M. H., and Baumbach, H. D. (1974). Differential effects of peripheral and central anosmia producing techniques on spontaneous behavior patterns. *Physiol Behav.* 13, 407-425.
- Spector, S. A., and Hull, E. M. (1972). Anosmia and mouse killing by rats: A nonolfactory role for the olfactory bulbs. *J. Comp. Physiol. Psychol.* 80, 354-356.
- Thorne, B. M., Aaron, M., and Latham, E. E. (1973). Effects of olfactory bulb ablation upon emotionality and muricidal behavior in four rat strains. *J. Comp. Physiol. Psychol.* 84, 339-344.
- Ueki, S., Nurimoto, S., and Ogawa, N. (1972). Characteristics in emotional behavior of the rat with bilateral olfactory bulb ablations. *Folia Psych. Neurol. Jap.* 26, 227-237.