Journal of Comparative and Physiological Psychology
1972, Vol. 80, No. 2, 354-356

ANOSMIA AND MOUSE KILLING BY RATS:
A NONOLFACTORY ROLE FOR THE OLFACTORY BULBS

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Pure anosmia, produced by deafferentation, does not facilitate mouse killing by rats. Anosmia produced by olfactory bulbectomy does facilitate an irritable form of mouse killing. Thus, it is not anosmia which is responsible for bulbectomy-facilitated muricide, but some nonolfactory function of the olfactory bulbs.

A very common means of producing anosmia in experimental animals is to remove the olfactory bulbs. Often there seems to be an unspoken assumption that anosmia is the only effect of bulbectomy. Recently, however, a number of behavioral effects of bulbectomy have been demonstrated that may not be due entirely to loss of olfactory information. These studies have shown that 50%–100% of nonkilling rats will kill mice after bulbectomy (Bernstein & Moyer, 1970; Karli, 1956; Karli & Vergnes, 1963; Karli, Vergnes, & Didiergeorges, 1969; Kumadaki, Hitomi, & Kumada, 1967; Malick, 1970), and that pain-induced defensive reactions in mice (Ropartz, 1968) and ability of both rats and pigeons to solve visual discrimination problems (Phillips, 1970; Wenzel & Salzman, 1968) are affected by ablation of the olfactory bulbs. The olfactory bulbs are known to have direct or indirect connections with several parts of the limbic system, and may have other functions than mere sensory analysis of olfactory information.

In light of possible multifunctional involvement of the olfactory bulbs, the present study compared two kinds of "anosmia" for their effects on mouse killing: that induced by removal of nasal mucosa and afferents (bulbs intact) and that induced by removal of olfactory bulbs. Our hypothesis was that if both techniques facilitate killing in previously nonkilling rats, then anosmia per se is implicated; whereas if the muricidal response was obtained only with bulbectomy and not with deafferentation, it is not loss of olfaction but disruption of some limbic function which facilitates the killing.

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METHOD

Subjects

Twenty male rats of the Holtzman strain were used. From 10 to 14 days before the start of testing each rat was housed individually in a metal cage, 18 × 20 × 25 cm. They were maintained on an ad-lib food and water schedule throughout the experiment. The mice were Swiss albino males, 20–30 gm. each.

Testing began by placing one mouse into each of the 20 cages and leaving it there for 1 wk. After approximately 30 min. of the first day of testing, two rats killed the mice placed in their cages. These were the only rats to exhibit the muricide response during the 7 days of testing, and these were removed from the group. Since the muricide response has been found to be completely constant during normal conditions, the other 18 rats were categorized as nonkillers. Ten of these nonkillers were subjected to bilateral deafferentation and 5 received bilateral olfactory bulbectomies, in an attempt to replicate Karli's (1956) findings. The remaining three nonkillers served as unoperated controls for the possibility that mere removal and replacement of a mouse in a rat's cage might alter the rat's behavior toward the mouse. The testing procedure was repeated 3 days after surgery for experimental animals, and control animals were retested concurrently. If a rat killed a mouse at any time during the 7 days, it was classified as a killer.

Surgical Procedure

All surgery was performed under sodium pentobarbital (Diabutal) anesthesia (35 mg/kg ip), with atropine sulfate (.30 mg.) to control mucus secretions. The method of deafferentation was adapted from Estable-Puig and de Estable (1969), using bilateral rather than unilateral ablation. Briefly, the frontal bone in a small area close to the frontal suture was removed, and the right and

left nasal cavities were opened. The mucoperiosteum was removed from the posterior portion of the cavities, as were all other structures on the way to the nasal septum and lamina cribrosa. The under surface of the cribriform plate was scraped, thereby transecting the fila olfactoria. The cavity was filled with gel foam and sulfathiazole, and the skin was sutured over it. Each animal was aspirated postoperatively to remove blood from the trachea, and was given an ip injection of Metrazol (2 mg.). Olfactory bulbectomy was accomplished by removing a 2-mm.^{2} portion of skull, about 9 mm. anterior to bregma by means of a dental drill. The exposed bulbs were removed by aspiration. The cavity was filled with gel foam and sulfathiazole and closed as before. These animals were also aspirated and injected with Metrazol.

Behavioral and Histological Verification

All rats were tested for ability to perceive a noxious olfactory stimulus (Thiessen, Lindzey, & Nyby, 1970). All control animals showed very definite signs of trying to avoid an ether-soaked cotton swab placed in front of them. No experimental animal showed any sign of avoidance or of being disturbed by the odor. At the end of the experiment all experimental animals were perfused intracardially with saline and Formalin and examined for surgical damage. Deafferented animals showed almost total destruction of the mucosa and fila olfactoria, and no damage to the bulbs. Bulbectomized animals showed complete destruction of the olfactory bulbs, with little extraolfactory damage.

Results and Discussion

The results can be seen in Table 1. None of the deafferented rats or the unoperated controls showed any signs of aggression toward the mouse in their cage. Rat and mouse usually lived in separate corners of the cage and showed little interest in each other. However, four of the five bulbectomized rats killed mice. Statistical differences between deafferented and bulbectomized animals were highly significant ($p = .009$, Fisher's exact probability test). Two rats killed approximately 2 hr. after the mice were placed in their cages. One rat killed during the first night; and the fourth killer displayed the muricide response 3 days after the mouse had been placed in its cage. The fifth rat showed no signs of aggression toward the mouse in its cage at any time during the experiment.

The kills made by the bulbectomized rats were not the efficient, stereotyped kind displayed by natural killers. Bite marks covered the bodies of all dead mice. Only one of the four mice killed was partially eaten. Bernstein and Moyer (1970) argue that the mouse killing produced by bulbectomy is more closely related to irritable aggression than to predatory aggression. The former is more emotional and less organized than the latter. In bulbectomized animals the quick predatory bite into the spinal cord is replaced by emotional, disorganized biting all over the body of the mouse, as first described by Karli (1956).

TABLE 1
Muricidal Responses

Group	No. tested	No. of killers	% killers
Bulbectomized	5	4	80
Deafferented	10	0	0
Control	3	0	0

The deafferented rats in the present study were of noticeably different temperament than the bulbectomized ones. Before surgery all animals could be handled comfortably only with a glove. After the operation deafferented animals could be handled easily without a glove. On the other hand, the bulbectomized rats always showed exaggerated startle responses and often showed attach responses when they were handled postoperatively.

Reported discrepancies in the percentage of bulbectomy-induced killing may well be attributed to differences in the criterion used. No bulbectomized rat in the present study would have been classified as a killer on the basis of previously used criteria consisting of a kill within 2 (Smith, King, & Hoebel, 1970), 5 (Malick, 1970), 30 (Bernstein & Moyer, 1970), or 60 (Myer, 1964) min. after introduction of the mouse, for several consecutive trials. We have used, instead, Karli's (1956) technique of allowing rat and mouse to live together continuously until a kill occurs, in this case, up to 1 wk. There is general agreement that once a rat kills one mouse, it is likely to develop sooner or later into a killer as defined by virtually any criterion (Karli, 1956; Myer, 1971). Therefore, it would appear that many bulbectomized rats previously classi-

fied as nonkillers according to a rather strict criterion would have become killers if given sufficient opportunity.

In summary, olfaction seems to be directly involved in mating behavior and intermale, territorial, and fear-induced aggression. However, removal of the bulbs also releases irritable aggression from an inhibitory control. The pathway and termination points of this inhibitory pathway are unknown, but the ventromedial hypothalamus, amygdala, and cingulum are likely to be involved (Moyer, 1968; Vergnes & Karli, 1965). Since the presence of the bulbs in the deafferented animals in the present study was sufficient to exert the normal inhibition on irritable aggression, and since anosmia did not diminish inhibitory control, the olfactory bulbs are shown to have a nonolfactory, limbic function, and should not be cast in a purely sensory role.

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(Received October 20, 1971)