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Olfactory bulb removal and taste aversion learning in mice

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Bulbectomized and unoperated mice were given 1 h of exposure to a novel food (almond) before receiving an intraperitoneal injection of lithium chloride solution. Their consumption of almond was recorded for the subsequent 10 days. Mice of both groups acquired a marked aversion to almond, and the unoperated mice continued to eat little throughout the experiment. The bulbectomized mice resumed eating more rapidly and, by the third day after the induction of illness, their consumption of almond did not differ from that of a control group which had not been lithium treated. The integrity of the olfactory system appears necessary for the maintenance, though not the establishment, of a taste aversion.

A large body of recent research has shown that the integrity of the olfactory system is important for normal behavioral functioning in rodents. Bulbectomy has resulted in developmental abnormalities (McClelland & Cowley, 1972), impaired sexual behavior (Cain & Paxinos, 1974), heightened reactivity in social situations (Richardson & Scudder, 1970), altered levels of aggression (Rowe & Edwards, 1971), and changes in learning ability (Marks, Remley, Seago, & Hastings, 1971). There are, however, sufficient species and paradigm differences to support only the most general explanatory hypotheses; the bulbectomized animal's abnormal behavior stems from a reduced amount of sensory input and limbic system damage which in some way impairs utilization of available information. (For a systematic review of bulbectomy studies, see Wenzel, 1974.)

One obvious area in which the effects of bulbectomy might be studied is that of the learned flavor aversion. This involves the association of the flavor (smell) of a (preferably) novel food with gastrointestinal distress caused by poisoning, and results in the animal's avoidance of the food. Studies concerning the effects of brain lesions on learned aversions suggest that this learning is subcortically mediated (Divac, Gade, & Wikmark, 1975), and various limbic structures have been implicated: the hippocampus (Best & Orr, 1973), the hypothalamus (Gold & Proulx, 1972), and the amygdala (Nachman & Ashe, 1974).

The present study investigates the effects of bilateral bulbectomy on the establishment and maintenance of a learned aversion in the mouse.

METHOD

The subjects were 24 male mice of the outbred DUB strain supplied by Flow Laboratories, Dublin, Virginia. At the time of testing, they were 60-80 days old. The mice were housed in individual cages throughout the experiment, and were allowed unlimited food and water.

The novel food was a shelled and husked almond, almond being selected for palatability and convenience. Illness was induced by intraperitoneal injection of lithium chloride solution (a standard dose of .9 ml .12 m).

The subjects were randomly assigned to one of three experimental groups. Group A (N = 8) was bulbectomized (see Cooper, 1974, for surgical procedure) 7 days before exposure to the novel food and illness induction. At this time, an almond was placed in each animal's cage for 1 h and, after the remaining almond had been removed, the lithium chloride was administered. Group B (N = 8) differed from Group A only in that the animals were not bulbectomized, and Group C (N = 8) was exposed to almond without subsequent lithium treatment.

The experimental groups (N = 8 in each case) consisted of mice who ate part of their almond during the initial exposure. A few nonpreferrers were excluded from the experiment at this point. The effects of the lithium chloride treatment did not appear severe; the mice showed some ruffling of the fur, reduced activity, and slight diarrhea for about 1 h after injection. Recovery seemed complete after 2 h.

On each of the 10 following days, each mouse was given a whole almond for a 3-h period, and the amount eaten was recorded.

At the end of this period, Group A mice were killed, decapitated, and their brains were fixed in formalin and examined macroscopically. Two mice were found only to have received small lesions to the bulbs; the data from these were discarded. The remaining six were found to have received complete bilateral bulb ablation.

RESULTS

The data were analyzed using the Kruskal-Wallis one-way analysis of variance and Mann-Whitney U tests. Mice of Groups A and B showed a marked aversion to almond the day following illness and, in the case of Group B mice, this aversion persisted over 10 days. The Group A mice, however, increased their daily consumption until, by Day 3, it reached a level similar to that of Group C animals, whose consumption differed little from day to day. Table 1 shows daily consumption for each group, and this is also shown graphically in Figure 1.

Two Group A mice did not increase their consumption of almond during the experiment; these were the animals subsequently found only to have been partially bulbectomized.

Table 1
Mean Daily Consumption of Almond (Grams)

Day	Group A†	B††	C††	U A/B	U A/C	U B/C
1	.03	.02	.79	36	8*	10**
2	.19	.05	.51	13.5	10*	6.5***
3	.39	.16	.68	10*	11.5	7***
4	.57	.16	.67	7*	19.5	8***
5	.51	.12	.57	10*	24	8***
6	.69	.17	.62	5.5*	19	2***
7	.75	.24	.75	7*	21	7.5***
8	.61	.26	.72	10.5*	16	9.5**
9	.63	.28	.73	8*	29	9.5**
10	.54	.20	.74	7.5*	14	3.5***

†N = 6 ††N = 8 *p ≤ .05 **p ≤ .01 ***p ≤ .005

DISCUSSION

The results of this experiment show that mice are able to learn a taste aversion to almond following a single injection of lithium chloride solution and that this aversion is long lasting. Bulbectomized mice are unimpaired in their ability to learn the aversion, but they resume eating the food within a few days.

The question arises as to whether this effect is explainable by the reduced olfactory acuity of the bulbectomized animals, or the impairment of a more "central" mechanism, or both. The ability of the bulbectomized mice to learn the aversion as readily as normals points to the unimportance of olfactory cues in the establishment of the aversion and suggests that the nature of the putative "central" impairment is not such that it interferes with the registering of the novel taste and its association with sickness.

Why is the aversion of short duration in the bulbectomized mice? It is possible that cues remaining in the cage from the time of the original illness remain able to inhibit the normal mice from eating; but if such cues are important, one would predict that the bulbectomized mice would not acquire the aversion.

Although the defect may, in general, best be viewed as a failure to retain information brought about by an impairment of affective behavior, certain basic variables remain to be investigated. These relate to: (1) food habits. Do bulbectomized mice differ from normals in their food preferences and consumption levels? (2) Activity patterns. Does bulbectomy alter diurnal activity patterns, thereby increasing daytime eating? (3) Is the initial lithium-induced illness equally severe in normal and bulbectomized animals? Studies designed to investigate these variables are currently under way.

Fig. 1 ALMOND CONSUMPTION

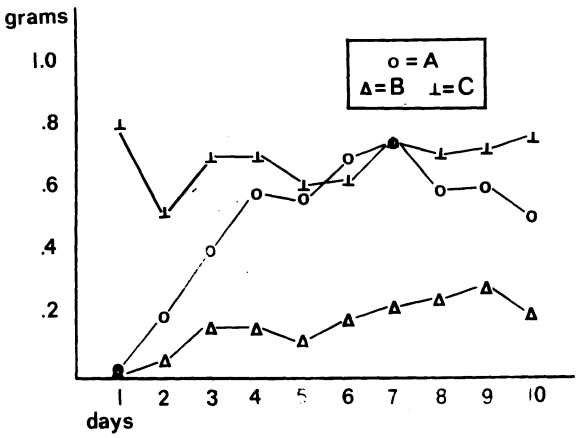

Figure 1. Mean almond consumption (grams/day).

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