BRIEF COMMUNICATION

Prefrontal Lesions Alter Eating and Hoarding Behavior in Rats

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KOLB, B. Prefrontal lesions alter eating and hoarding behavior in rats. PHYSIOL. BEHAV. 12(3) 507-511, 1974. -- Aspiration lesions of orbital frontal cortex in the rat produced adipsia and aphagia persisting from 4-7 days. After recovery, food hoarding behavior was normal. Medial frontal cortical lesions did not affect food or water intake but produced a permanent deficit in food hoarding behavior. These results are taken as evidence that, like the rhesus monkey, the prefrontal cortex of the rat is dissociable into discrete subfields.

RECENT studies have implicated several subcortical structures including the hippocampus [13], septum [13] and anterior thalamus [12] in the regulation of hoarding behavior in rats. Less is known about cortical involvement although Stamm [11] reported a reduction in hoarding following medial cortex lesions. Some of Stamm's lesions appear to have encroached on the medial frontal projection field [4] of thalamic nucleus medialis dorsalis (MD) (that is, prefrontal cortex as defined by Rose and Woolsey [9]) but it is very difficult to determine from his description whether these animals showed more or less change in hoarding than those animals with lesions restricted to the more posterior medial cortex. The following study was designed to examine the possibility that damage to the prefrontal cortex of the rat might influence hoarding behavior as well as to test whether the medial and orbital aspects of the prefrontal cortex might have different functions in this respect.

METHOD

Animals

The animals were 27 male Wistar albino rats aged roughly 130 days at the time of surgery and were divided into 3 equal sized groups (control group, medial frontal lesion group, and orbital frontal lesion group).

Surgery

Discrete lesions were made to the medial frontal (Leonard's [4] dorsal cortex) or orbital frontal (Leonard's [4] sulcal cortex) projection fields of MD by using surgical procedures outlined elsewhere [8]. The controls had the skin incised and were then sutured closed.

Apparatus

The apparatus was similar to the one designed by Hunt [2] and employed by Wishart, Brohman and Mogenson [10]. It consisted of a small living area (18 cm x 18 cm x 25 cm high) for each animal separated by a removable door from an alleyway 80 cm long x 25 cm wide x 30 cm high. Thirty Purina rat pellets were piled at the far end of the alley and were approximately 2.5 cm long and weighed about 6 g each. There were six hoarding boxes and rats from each group were assigned to each of the boxes to avoid lesion group x box confronting.

Procedure

All testing was carried out postoperatively and consisted of 2 phases. In Phase 1, each animal was allowed access to the alleyway for 30 min per day for 21 consecutive days. On Days 1-7 and 15-21, the animals were maintained

1This research was supported, in part, by Grant MH 04726 from the National Institute of Mental Health, U. S. Public Health Service, to J. M. Warren.
under ad lib food and water conditions and were allowed to keep any hoarded pellets, but on Days 8–14, they were maintained under 22 h food deprivation and ad lib water conditions. This was done by allowing the animals to eat pellets for the 2 h immediately following the test session and then all pellets were removed from the living quarters. Phase 2 began 4 months after Phase 1 was completed during which time all the animals had been maintained at roughly 80% body weight and were tested on various learning problems [8]. The animals were retested for hoarding behavior after this extended deprivation period as hoarding has long been known to be at least partly dependent on previous deprivation history [1, 5, 6]. The procedure in Phase 2 was the same as in Phase 1 except that the animals were tested 4 days ad lib, 3 days deprived and then 2 days ad lib. The measures recorded in both phases were: (1) the time taken to reach the pellets each day; (2) the time taken to hoard the first pellet; (3) the number of pellets hoarded; and (4) the time required to hoard all 30 pellets if the animal did so.

At the completion of testing the rats were deeply anesthetized with sodium pentobarbital and perfused with normal saline followed by 10% Formalin. The brains were photographed, embedded in celloidin and sections cut at 30 μ and stained with cresyl violet.

RESULTS

Anatomy

Cross sections through the region of lesion for the medial frontal and orbital frontal groups are shown in Fig. 1. Both lesions produced retrograde changes in MD as illustrated in Fig. 2. The locus of these changes fits well with Leonard's [4] conclusions that the medial portion of MD projects to the orbital frontal cortex while the lateral portion of MD projects to the medial frontal cortex since retrograde changes following orbital or medial frontal lesions were restricted to the medial and lateral portions of MD respectively. In addition, orbital frontal lesions frequently produced degeneration in the ventral portion of the ventral lateral thalamic nucleus while medial frontal lesions frequently produced degeneration in the anterior medial thalamic nucleus.

Postoperative Food Intake

All rats in the control group and in the group with medial frontal cortex lesions resumed eating and drinking within 24 h postoperatively. This differed dramatically from the behavior of rats with orbital frontal lesions who failed to eat or drink at all for periods ranging from 4–7

FIG. 1. The diagrams on the left show the area damaged in the largest, smallest and median medial frontal lesion while the diagrams on the right show the area damaged in the largest, smallest and median orbital frontal lesion. Each animal is represented by lines of one orientation so that the densest portion denotes the most frequently damaged region in each group.
FIG. 2. Photomicrographs (45X) showing the difference between the appearance of cells in the medial portion of thalamic nucleus medialis dorsalis (MD) in an animal with an orbital frontal lesion (A) and a control animal (B). Medial frontal lesions produced similar changes in the lateral portion of MD. See text for further explanation.
days postoperatively. This condition was transient, however, as the animals did eventually begin to eat and drink. Because of severe weight losses during this time, these animals were maintained on a special diet made from a mixture of milk, eggs and ground up Purina rat chow until they had regained their preoperative weights. Three of the rats with orbital frontal lesions eventually died, thus leaving group sizes of 9 controls, 9 medial frontals and 6 orbital fronts. Testing commenced following a postoperative recovery period of 10–12 weeks at which time all animals were healthy and were eating and drinking normally.

**Behavioral Results**

Table 1 shows that the rats with medial frontal lesions hoarded fewer pellets than both the controls and the rats with orbital frontal lesions throughout the experiment. However, the exact nature of the medial frontal deficit was unclear. It did not appear to be solely due to a motor deficit since the animals did hoard some pellets and when they did so, showed no obvious motor impairment. Furthermore, these animals were also responsive to the stress of food deprivation in that they hoarded more pellets during the deprived condition than during the ad lib conditions and were capable of hoarding a median of 15.4 pellets (deprived, Phase 1), which was more than either of the other groups hoarded under ad lib conditions (Phase 1). The time taken to hoard the first pellet was longer for the animals with medial frontal lesions (Median = 22 min 40 sec) than the controls (Median = 6 min 20 sec) or the animals with orbital frontal lesions (Median = 1 min 45 sec) during the ad lib stage of Phase 1. These differences were significant (Two-tailed Mann Whitney U = 17; p<0.05; U = 10; p<0.05) but there were no other significant differences in time to hoard among any of the groups at any other stage of the study.

**DISCUSSION**

The medial and orbital aspects of the rat prefrontal cortex [4] proved to be functionally distinct and they were doubly dissociated on the basis of postoperative food intake and hoarding. Orbital frontal lesions produced adipsia and aphagia lasting for 4–7 days postoperatively, but failed to significantly affect hoarding while medial frontal lesions did not affect postoperative food intake but significantly reduced hoarding behavior.

There is considerable anatomical [7] and behavioral [10] evidence that the orbital and lateral aspects of the rhesus monkey prefrontal cortex are functionally distinct. Until very recently it has been unclear whether the prefrontal cortex of other mammalian orders is arranged in a similar way. Recent research on dogs [2] suggests that the prefrontal cortex of carnivores is functionally dissociable into discrete subfields, and the data reported on rats in this project suggests that the prefrontal cortex of rodents is dissociable as well.

**REFERENCES**


**TABLE 1**

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<tr>
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<th>Phase I</th>
<th>Phase II</th>
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<tbody>
<tr>
<td></td>
<td>Ad Lib</td>
<td>Deprived</td>
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<tr>
<td>Controls (N = 9)</td>
<td>6.1</td>
<td>29.0</td>
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<tr>
<td>Orbitals (N = 6)</td>
<td>11.3</td>
<td>27.5</td>
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<td>Medials (N = 9)</td>
<td>0.9*†</td>
<td>15.4*†</td>
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*Significantly different from controls (Two-tailed Mann-Whitney, p<0.05)
†Significantly different from orbitals (Two-tailed Mann-Whitney, p<0.05)

