Bulletin of the Psychonomic Society
1977, Vol. 9 (6), 399-401

Dorsomedial amygdala damage: A time-after-surgery assessment of feeding

SHERWOOD O. COLE
Rutgers University, Camden, New Jersey 08102

The food consumption of Charles River male rats with dorsomedial amygdala lesions was compared with sham operates and normal controls on four tests spanning a 65-day postsurgery period. Following 24-h food deprivation (18- and 65-day postsurgery tests), lesioned animals ate significantly less than the other two groups ($p < .01$). Although 24-h food *and* water deprivation (35- and 50-day postsurgery tests) increased the absolute level of food consumption in all groups, the relative magnitude of the feeding deficit in lesioned animals remained approximately the same ($p < .01$). The results suggest that hypotheses which stress the "temporary deficit" nature of lesion effects do not adequately explain the changes in feeding following dorsomedial amygdala damage.

Changes in feeding behavior after amygdala damage are well documented, with destruction of dorsomedial regions decreasing food consumption (Cole, 1974; Fonberg, 1966) and destruction of more basolateral sites increasing food consumption (Fonberg, 1968). While the reduction in feeding by rats following dorsomedial amygdala damage appears to be food-deprivation dependent (Cole, 1974), the similar attenuation of the anorexigenic properties of amphetamine following recovery from dorsomedial amygdala lesions (Cole, 1973) and from lateral hypothalamic lesions (Carlisle, 1964) suggests that adrenergic processes underlie the regulation of feeding in both areas.

Evidence suggesting that amygdala damage also alters the subject's sensitivity or responsiveness to environmental stimuli (Nauta, 1963; Sclafani, Belluzzi, & Grossman, 1970; White, 1971) raises the question as to whether these changes in feeding are due to a temporary deficit in responding to stimulus properties of food or represent more stable changes in feeding. Also, Turner (1973) has indicated that medial amygdala lesions produce a rather extensive "sensorimotor syndrome," characterized by visual and olfactory unresponsiveness, absent or sluggish visual and proprioceptive placing, forelimb disuse, and inability to make localized and conditioned responses to somatosensory stimuli. Since recovery from the "sensorimotor syndrome" appears to take place within 10 to 22 days (Turner, 1973), any general effects of such a deficit on feeding behavior should be only temporary.

As a means of determining whether changes in feeding following dorsomedial amygdala damage are temporary (and therefore possibly due to transitory deficits) or more stable, the present study reports a preliminary assessment of lesion effects extending over a 65-day postsurgery period. While the first test was conducted toward the end of the proposed "sensorimotor syndrome" period, the other three tests extended well beyond the period where temporary deficits provide a viable explanation of findings.

METHOD

Subjects

Six amygdala lesion, six sham-operate, and six normal control male Charles River rats were subjects. At the time of surgery, the animals were 120-130 days of age, with body weights ranging from 350-450 g. They were housed individually in plastic cages in a temperature-controlled laboratory under a regular 12-h-light/12-h-dark schedule and had access to Purina Lab Chow and water in the home cage. While all animals were, on other occasions during the 65-day postsurgery period, also serving as drug-control subjects in a lesion-drug study, no subject received any drug injection during the entire period of assessment.

Surgical Procedure

In the lesioned group, bilateral electrolytic lesions were produced stereotaxically (2-mA anodal dc current for 20 sec) with monopolar stainless steel electrodes insulated except at the tip while animals were anesthetized with nembutal sodium (50 mg/kg). A rectal cathode completed the circuit. Flat-skull coordinates for the dorsomedial amygdala (Skinner, 1971) were −.5 mm posterior to bregma, ±4.0 mm lateral to midline, and −8.0 mm vertical depth. Surgical procedure for the sham-operated group was identical to the above, except that no current was passed after electrode placement.

Testing Procedure

During the 65-day postsurgery period, the lesioned animals were compared with the sham-operated and control animals on the following tests. On Test 1 (18 days postsurgery) and Test 4 (65 days postsurgery), subjects in all groups were 24-h food deprived and food and water consumption was measured. On Test 2 (35 days postsurgery) and Test 3 (50 days postsurgery), subjects in all groups were 24-h food *and* water deprived and food and water consumption was measured.

Each test session was conducted in the animal's home cage and was 1 h in length. The animals were weighed immediately prior to each test session, in order to assess changes in the body weights of the groups over the course of the 65-day period. To begin the test, a cup containing dry-ground Purina Lab Chow was introduced into the home cage. Food consumption was measured by weighing the cup at the beginning of the test, weighing it again at the end of the test, and taking the difference in weight (corrected for any spillage) as amount consumed. Water consumption during the test was measured by means of a graduated bottle attached to the front of the cage.

At 70-75 days postsurgery, the lesioned and sham-operated animals were sacrificed and perfused with 37% formaldehyde

Table 1
Mean Food Consumption (±SE), Mean Water Consumption (±SE), and Mean Body Weight of Amygdala Lesion (AL), Sham-Operate (SO), and Normal Control (NC) Subjects on Four Postsurgery Assessment Tests

Group	Condition	Days Postsurgery 18	35	50	65
NC	Food Consumption (g)	15.67 ± 0.99	24.33 ± 1.78	22.50 ± 2.40	17.83 ± 1.28
	Water Consumption (ml)	6.67 ± 3.07	18.33 ± 2.11	15.00 ± 1.29	8.83 ± 1.83
	Body Weight (g)	448.33	485.83	510.00	535.00
SO	Food Consumption (g)	17.50 ± 1.75	28.33 ± 1.73	27.00 ± 2.87	20.83 ± 2.30
	Water Consumption (ml)	6.67 ± 1.67	20.00 ± 1.29	15.83 ± 0.83	6.67 ± 1.67
	Body Weight (g)	455.50	500.00	526.83	555.50
AL	Food Consumption	9.83 ± 0.83*†	14.33 ± 2.17*†	12.17 ± 2.09*†	11.33 ± 1.40*†
	Water Consumption (ml)	5.00 ± 1.82	15.83 ± 2.39	12.50 ± 1.12††	6.67 ± 1.67
	Body Weight (g)	439.50	474.83	492.67	524.17

Note–On 18- and 65-day postsurgery tests, animals were 24-h food deprived; on 35- and 50-day postsurgery tests, animals were 24-h food and *water deprived.*
†Significantly different from SO at .01 level.
**Significantly different from NC at .01 level.*
††Significantly different from SO at .05 level.

solution. The brains were extracted for frozen sectioning (40 micra) and stained for purposes of histological verification.

RESULTS AND DISCUSSION

By the second day postsurgery, the average body weight of the lesioned group had dropped to approximately 91% of its presurgery level. Because of this drop in body weight, the lesioned animals' normal laboratory chow diet was supplemented with a damp-mash diet for several subsequent days. By the seventh or eighth day postsurgery, the lesioned animals had regained some of this loss and appeared to be eating satisfactorily; therefore, the supplemental diet was discontinued. Relative to the sham-operated and control animals, the body weight of the lesioned animals remained quite stable thereafter for the duration of the 65-day postsurgery assessment period. On none of the four assessment tests did the body weights of the three groups differ significantly (t test for independent groups).

The food consumption, water consumption, and body weights of the three groups of subjects on each of the four postsurgery tests are summarized in Table 1. The t-test analysis of food consumption indicated that the lesioned subjects ate significantly less than the controls ($p < .01$) and the sham-operated subjects ($p < .01$) following 24-h food deprivation (18- and 65-day postsurgery tests) and following 24-h food *and* water deprivation (35- and 50-day postsurgery tests). Food and water deprivation increased the absolute level of food consumption in all three groups, but did not significantly alter the relative magnitude of the feeding deficit observed in the lesioned group. While the food consumption of the sham-operated group was slightly higher than that of the control group on all four tests, this difference did not reach statistical significance on any of the tests.

Although the water consumption of the lesioned subjects was significantly less than that of the sham-operated subjects following 24-h food *and* water deprivation on the 50-day postsurgery test ($p < .05$, t-test comparison of groups), this difference appeared to be due to a rather selective reduction in the within-groups variance of these subjects on this particular test. This finding did not prove to be reliable, as no significant difference in the water consumption of the three groups was observed on the preceding test following 24-h food *and* water deprivation (35-day postsurgery test).

Histological examination of the lesioned brains indicated that all six lesioned animals sustained bilateral damage to dorsomedial amygdala structures, including areas in the region of the stria terminalis. A lesion representative of the site and extent of damage is reconstructed in Figure 1. Histological examination of the sham-operated brains indicated that the bilateral

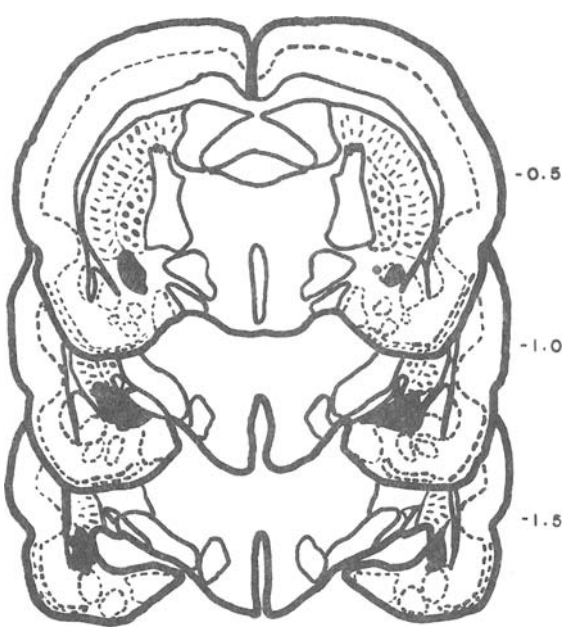

Figure 1. Reconstruction of dorsomedial amygdala damage representative of that found in the lesioned group. Reconstruction is adopted from Skinner (1971).

placements of the electrode tips were in the same general dorsomedial region of the amygdala as were the lesions.

The results of the present study suggest that the feeding deficit of rats with dorsomedial amygdala damage, while it may be food-deprivation dependent (Cole, 1974), is quite stable over a 65-day postsurgery period. Although specific features of the deprivation condition (24-h food deprivation only vs 24-h food *and* water deprivation) may change absolute levels of food consumption, the relative magnitude of the deficit in feeding by lesioned animals remains much the same.

Of primary importance in the findings of the present study is a comparison of the results on the 18-day postsurgery test with those on the 65-day postsurgery test, where identical food deprivation conditions were used. While the first of the tests came at the end of the proposed recovery period from the "sensorimotor syndrome" (Turner, 1973), the second of the tests came some 40-45 days after this period. Yet, the similarity in the feeding deficit of the lesioned animals on the two tests is quite apparent. The findings clearly suggest that temporary deficits (whether they be due to "sensorimotor syndrome" or to a more general alteration in subjects' responsiveness to stimulus properties of food) do not provide an adequate explanation of the food-deprivation dependent hypophagia observed in rats following dorsomedial amygdala damage. Rather, the findings suggest that such damage alters more basic central structures underlying the regulation of feeding. Dorsomedial amygdala sites may not, however, serve a primary function in the central regulation of feeding, but probably "modulated" the regulation of feeding by hypothalamic sites through fiber systems (stria terminalis) connecting the two sites (Grossman, 1964; Sclafani et al., 1970).

REFERENCES

CARLISLE, H. J. Differential effects of amphetamine on food and water intake in rats with lateral hypothalamic lesions. *Journal of Comparative and Physiological Psychology*, 1964, **58**, 47-54.

COLE, S. O. Changes in amphetamine anorexia following amygdala lesions in rats. *Proceedings of the 81st Annual Convention, APA*, 1973, 1043-1044.

COLE, S. O. Changes in the feeding behavior of rats after amygdala lesions. *Behavioral Biology*, 1974, **12**, 265-270.

FONBERG, E. Aphagia, produced by destruction of the dorsomedial amygdala in dogs. *Bulletin of the Polish Academy of Science* (Biology), 1966, **14**, 719-722.

FONBERG, E. The role of the amygdaloid nucleus in animal behavior. *Progress in Brain Research*, 1968, **22**, 273-281.

GROSSMAN, S. P. Behavioral effects of chemical stimulation of the ventral amygdala. *Journal of Comparative and Physiological Psychology*, 1964, **57**, 29-36.

NAUTA, W. J. H. Central nervous organization and the endocrine motor system. In A. V. Nalbandov (Ed.), *Advances in neuroendocrinology*. Urbana: University of Illinois Press, 1963.

SCLAFANI, A., BELLUZZI, J. D., & GROSSMAN, S. P. Effects of lesions in the hypothalamus and amygdala on feeding behavior in the rat. *Journal of Comparative and Physiological Psychology*, 1970, **72**, 394-403.

SKINNER, J. E. *Neuroscience: A laboratory manual*. Philadelphia: Saunders, 1971.

TURNER, B. H. Sensorimotor syndrome produced by lesions of the amygdala and lateral hypothalamus. *Journal of Comparative and Physiological Psychology*, 1973, **82**, 37-47.

WHITE, N. Perseveration by rats with amygdaloid lesions. *Journal of Comparative and Physiological Psychology*, 1971, **77**, 416-426.

(Received for publication December 30, 1976.)