Failure to form a learned taste aversion in rats with amygdaloid lesions

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Rats sustaining bilateral amygdaloid lesions failed to develop aversion to 0.1% saccharin solution after it had been paired with a 0.12 M LiCl injection. These data confirm an important role for the amygdaloid complex in the formation of learned taste aversion and further substantiate the role of this structure in a wide variety of aversively motivated behaviors.

Recently, Rozin & Kalat (1971) have argued that data from both diet selection and taste aversion learning experiments make necessary the postulation of new learning principles which govern the feeding system. Since the amygdaloid complex is widely considered to be involved in the control of feeding (e.g., Goddard, 1964) as well as a variety of aversively motivated behaviors (e.g., Horvarth, 1963; Pellegrino, 1968; Ursin, 1965), it might be expected that amygdaloid damage would interfere with the formation of a learned taste aversion. Some data (McGowan, Hankins, & Garcia, 1972; Rolls & Rolls, 1973) have recently appeared which seem to support this expectation. McGowan et al (1972) found that amygdaloid damage interfered with a LiCl-induced aversion to saccharin solution. However, these authors fail to report pretest levels of saccharin consumption. In view of a later report (Rolls & Rolls, 1973), which reports increased comsumption of 25% sucrose solution following amygdaloid damage, the possibility that increased reactivity to saccharin solution in amygdaloid Ss offsets the aversive effects of LiCl cannot be ruled out. In a second report, appearing after completion of this experiment, Rolls & Rolls (1973) observed that amygdaloid Ss generally failed to form an aversion to NaCl after LiCl consumption. Since the amount of LiCl consumed by both amygdaloid and control Ss varied widely, and since some amygdaloid Ss did form an aversion to NaCl, their results are difficult to interpret.

The present experiment reports on amygdaloid lesion-induced failure to form a learned aversion to saccharin solution which avoids these difficulties.

METHOD

Subjects

The Ss were 15 male albino rats (Holtzman Co.), 225-250 days old at the beginning of the experiment. These Ss had previously served in studies of exploratory behavior, wood gnawing, and passive avoidance but had had no previous experience with the solutions to be tested in this experiment.

Surgery and Histology

Surgical techniques are described in detail elsewhere (Kemble & Beckman, 1970). Briefly, bilateral anodal electrolytic lesions (2.0 mA/20 sec) were sterotactically placed in the amygdaloid complex of seven Ss through stainless steel insect pins. Two Ss received control operations and the remaining Ss received scalp incisions only.

At the conclusion of testing, experimental brains were examined histologically. The amygdaloid lesions were similar in size and placement to those reported previously (Kemble & Beckman, 1970).

Apparatus and Procedures

All testing conducted in cages (Wahmann, LC-28) adapted to accept two 100-ml drinking tubes housed in a continuously illuminated animal room. All solutions were freshly mixed in deionized water on the day of use.

Initially, all Ss were deprived of food and water for 23 h and given 5 h access to a 0.1% saccharin solution (1.0 g/1,000 ml) and tap water (pretreatment). Amygdaloid and control Ss were then designated to receive LiCl (control, N = 4; amygdaloid, N = 4) or isotonic saline (control, N = 4; amygdaloid, N = 3) injections during the aversion trial. After 24 h ad lib access to food and water, all Ss were deprived of food and water for 23 h and then permitted to drink 0.1% saccharin solution (only) for 10 min. After 10 min access to saccharin solution, Ss received a 6-ml intraperitoneal injection of 0.12 M LiCl or isotonic saline; they were then returned to their cages and given ad lib access to food and water for 48 h. Finally, Ss were food and water deprived for 23 h and given 5 h access to 0.1% saccharin solution and tap water as described above (posttreatment).

RESULTS AND DISCUSSION

The results of this experiment are summarized in Table 1. It can be seen that, with one exception, all Ss showed a strong preference for saccharin solution before treatment. After treatment with LiCl, control Ss typically rejected saccharin while all amygdaloid Ss

Table 1
Percent Saccharin Solution in Total Fluid Intake During Preference Testing Before (Pretreatment) and After (Posttreatment) Injection of 0.12 m LiCl or Isotonic Saline

		Percent Saccharin Solution in Total Intake	
Group	S Number	Pre-treatment	Post-treatment
Control (LiCl Treated)	3	91.8	11.8
	8	92.9	90.6
	16	92.1	22.2
	17	97.5	23.1
Amygdaloid (LiCl Treated)	15	91.8	64.0
	18	100.0	96.0
	21	98.5	95.5
	23	95.7	96.2
Control (Saline Treated)	1	46.7	81.8
	13	96.8	95.7
	14	90.5	84.3
	20	87.8	89.3
Amygdaloid (Saline Treated)	19	88.6	94.4
	22	97.2	98.8
	24	96.6	94.7

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drank substantial amounts of this solution ($U = 1$, $df = 4/4$, $p < .03$). Both control and amygdaloid Ss continued to show a strong preference for saccharin solution after treatment with saline solution. Alternatively, if reactivity is indexed by *amount* of saccharin solution consumed (instead of a percent measure), closely parallel results are obtained. Although amygdaloid Ss drank slightly more saccharin solution ($\bar{X} = 56.0$ ml) than control Ss ($\bar{X} = 40.6$ ml) during the pretreatment test, these differences did not approach statistical significance ($p > .10$). After treatment with LiCl, amygdaloid Ss drank significantly ($U = 1$, $df = 4/4$, $p < .03$) more saccharin solution ($\bar{X}$=39.2 ml) than did control Ss ($\bar{X} = 9.0$ ml). Finally, the posttreatment saccharin consumption of saline-treated amygdaloid ($\bar{X} = 43.7$ ml) and control Ss ($\bar{X} = 35.8$ ml) did not differ reliably.

The present data are consistent with that of McGowan et al (1972) and Rolls & Rolls (1973) in suggesting an important role for the amygdaloid complex in the formation of learned taste aversions. Moreover, the pretreatment data indicate that this deficit does not reflect hyperreactivity to the test solutions. Neither would a general disorder of fluid intake or ingestive mechanisms seem to account for the amygdaloid deficit. Rolls & Rolls (1973) report normal responsivity in hypertonic saline- or ispoproterenol-induced drinking in amygdaloid Ss, and we have observed quite normal adaptation to food and water deprivation as well as cellulose-adulterated food in our laboratory (Kemble, unpublished observations). Thus, taste aversion may be added to the impressive list of aversively motivated behaviors disrupted by amygdaloid insult.

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