Differential Effects of Amygdaloid Lesions on Conditioned Taste Aversion Learning by Rats

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AGGLETON, J. P., M. PETRIDES AND S. D. IVERSEN. Differential effects of amygdaloid lesions on conditioned taste aversion learning by rats. PHYSIOL. BEHAV. 27(3) 397-400, 1981.—Rats with electrolytic lesions placed in either the basolateral or corticomedial divisions of the amygdala acquired a conditioned taste aversion to sucrose. Comparisons with a surgical control group indicated that damage to the corticomedial amygdala did not alter the animals' performance, while damage in the basolateral nuclei resulted in a small but significant attenuation of the aversion. Furthermore, these amygdaloid lesions did not alter the acceptability of two quinine hydrochloride solutions (0.01% and 0.001%). The daily drinking behavior of the rats with basolateral amygdaloid lesions appeared consistent with the hypothesis that this lesion affected the animals' appreciation of the novelty of the sucrose solution, and hence attenuated the subsequent aversion.

Corticomedial amygdala Basolateral amygdala Brain lesion Conditioned taste aversion Quinine

METHOD

Subjects
The subjects were 32 naive, male, Lister hooded rats whose weights ranged from 240-350 g. They were housed in similar, individual cages with ad lib laboratory chow (Dixons Diet 41B).

Surgery
Surgery was performed under Equithesin anesthesia (3.75 mg/kg). All lesions were made by passing a 1.8 mA DC current for 10 seconds through the uninsulated tip (00-gauge) of a stainless steel electrode (00-gauge). The stereotaxic coordinates, taken from Bregma, were as follows: BLA group, anterior -0.8 mm, lateral 5.2 mm and horizontal -3.0 mm; CMA group, anterior -0.8 mm, lateral 3.6 mm and horizontal -3.8 mm. In the control rats the electrode was lowered only as far as the amygdala, but without entering it and without passing any current. Six rats served as controls while basolateral and corticomedial amygdaloid lesions were produced in 13 rats respectively.

Procedure
For the duration of the experiment each rat was allowed

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10 minutes drinking time, while in a test box, and deprived of water for the remainder of the day. The test boxes had three metal walls, painted black, a glass front and a wire grid floor. The floor measured 30×30 cm and the box was 38 cm high. Each box was equipped with one burette.

For the first five days the rats were offered water. This water was replaced with a 15% sucrose solution on day 6 and immediately after this session the rats received an intraperitoneal injection of 0.15 M lithium chloride (20 ml/kg) to induce sickness. On days 7–10 the animals received water in the test box and by day 10 they had returned to the level of water consumption seen prior to the lithium sickness (day 5). On day 11 the rats were retested with the 15% sucrose solution.

Two hours after this sucrose retest the rats were returned to the test boxes and given water for 10 minutes. On the following day (day 12) the animals were offered a single bottle containing a 0.01% solution of quinine hydrochloride. This was followed two hours later by 10 minutes access to water. On the final test day (day 13) the rats were offered a 0.001% solution of quinine hydrochloride.

RESULTS

Histological Findings

At the completion of the experiment the animals were sacrificed and the brains fixed in 10% sucrose-Formalin, sectioned and mounted on slides. The lesions were reconstructed from enlarged projected images of the sections. The corticomedial lesions of both groups were comparable in size, while the basolateral lesions largely filling the region depicted in Fig. 1. There was, however, greater variability in the placement of the basolateral lesions, hence the zone in which these lesions fell is larger than that of the CMA group (Fig. 1). There was variable damage to the piriform cortex in the BLA group, and in two rats there was slight damage to the ventral most part of the caudate-putamen. Two rats in the CMA group sustained some damage to the hippocampus at the posterior extremity of the amygdala.

Behavioral Findings

Table 1 presents the mean intake of each group for days 5–11. To evaluate the acquired aversion to sucrose a suppression ratio was calculated:

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\frac{\text{sucrose intake first exposure (day 6)} \quad - \quad \text{sucrose intake retest (day 11)}}{\text{sucrose intake first exposure (day 6)}}
\]

A complete suppression of sucrose would produce a ratio of 1.0. The suppression ratios of the three groups were found to differ (Kruskal-Wallis, H=16.30, p<0.001; Fig. 2), and the BLA rats did not suppress their intake as strongly as either the controls (Mann-Whitney, U=1.5, p<0.001) or the CMA rats (U=19.5, p<0.001). The CMA group did not differ from the controls (U=30.0, p>0.05).

All animals reduced their water intake on day 7, the day following the lithium chloride injection. The ratios of the water intakes on day 7 to those of the previous day that water was offered (day 5) were calculated and compared. This analysis indicated that the groups differed (H=6.09, p<0.05) and that the BLA rats drank relatively more sucrose than the controls (U=15, p<0.025).

The experimental groups did not differ in the consumption of either 0.01% quinine solution (H=0.44) or 0.001% quinine solution (H=1.99, Fig. 3). There was however evidence that the groups drank differing amounts of water in the intervening session (H=15.05, p<0.002). The BLA group drank significantly less water on day 12 than either the controls (U=4.5, p<0.002) or the CMA group (U=19.5, p<0.002).

DISCUSSION

The results of this study provide a dissociation between the effects of damage to the basolateral and corticomedial amygdaloid nuclei. Lesions of the basolateral nuclei produced a mild, but significant, attenuation of a learnt taste aversion, while damage to the corticomedial region had no discernible effect. The effects of basolateral amygdaloid damage were qualitatively consistent with previous reports [9,11], while corticomedial amygdaloid lesions had not previously been tested formally on this paradigm. The normal reactivity of the rats with amygdaloid lesions to unpleasant tastes, such as quinine, in this and other experiments [5, 6, 9], shows that this interference is not due to a failure to suppress the drinking response.

Although rats with lesions in the basolateral amygdala were impaired on the conditioned taste aversion task the degree of this impairment appeared slight when compared to previous studies [6, 9, 11]. A possible explanation of this
discrepancy lies in the extent of the amygdaloid damage. The basolateral lesions of previous studies appear to have typically encroached upon the central nucleus, which is the primary amygdaloid recipient of gustatory inputs [10]. It is therefore possible that additional damage to the central nucleus, or its afferent fibers, might potentiate the disruptive effect of basolateral amygdaloid lesions.

The abnormal drinking patterns of the BLA animals, in this and a previous experiment [9], support the hypothesis that the conditioned taste aversion impairment reflects a failure to respond appropriately to the novelty of the sucrose solution, hence attenuating the subsequent aversion. Thus the rats with basolateral amygdaloid damage showed an excessive intake of sucrose on the first day it was offered, which contrasted with an abnormal decrease in water consumption the day after lithium poisoning. A similar lack of neophobia for novel fluids has been seen after amygdalec- tomy in other experiments [5,6] while the excessive decrease in water consumption the day after lithium poisoning has been interpreted as an overgeneralized aversion [9]. It is possible that the low level of water intake shown by the BLA rats on day 12, between the two quinine hydrochloride tests, reflects a similar overgeneralized aversion.

There is additional evidence that basolateral amygdaloid lesions will reduce neophobia in rats. Such animals will eat novel foods in a novel environment more readily than controls [1,12,14]. Furthermore, just as medial amygdaloid lesions do not interfere with a learnt taste aversion, similar medial lesions leave other neophobic responses intact [1,13]. Thus there is some consistent evidence that lesions of the basolateral amygdala are selectively disruptive in situations where the animal is confronted by novelty, while corticomedial lesions have little or no effect.
REFERENCES