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Role of Noradrenaline and Serotonin in the Basolateral Region of the Amygdala in Food Preferences and Learned Taste Aversions in the Rat

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BORSINI, F. AND E. T. ROLLS. *Role of noradrenaline and serotonin in the basolateral region of the amygdala in food preferences and learned taste aversions in the rat.* PHYSIOL BEHAV 33(1) 37–43, 1984.—First, it was confirmed that bilateral lesions in the basolateral region of the amygdala (ABL) of the rat increased the time spent eating novel as compared to familiar food in a food preference test, and that the lesions impaired learned taste aversion to a sucrose solution which had been paired with lithium chloride. Then the roles of noradrenaline and serotonin in the amygdala in these aspects of food intake were investigated. In Experiment 2, it was shown that injections of 10 and 20 nmoles of noradrenaline (NA) into the ABL increased the time spent eating familiar food in the food preference test. Higher doses of NA (50 and 100 nmoles) increased the total time spent eating without changing the preference of the rats for familiar or novel food, and produced behavioral side effects. Serotonin (5HT) injected into the ABL in doses of 10, 50 and 100 nmoles did not modify the pattern of choice of the foods. In Experiment 3, it was shown that depletion of NA in the ABL with 10 μg 6-hydroxydopamine did not alter the level of feeding of novel and familiar foods, but did impair taste aversion Depletion of 5HT in the ABL with 10 μg 5,7 dihydroxytryptamine did not alter food preferences or impair the taste aversion learning. The depletions of NA and 5HT were confirmed biochemically. These results provide further evidence for a role of the amygdala in preferences for novel as compared to familiar foods and in learning that the ingestion of a food is associated with sickness, and suggest that noradrenaline but not serotonin in the amygdala is involved in these types of control of food intake.

Amygdala Feeding Noradrenaline Serotonin Neophagia Neophobia Novelty Food preference Learned aversion

THERE is evidence that noradrenaline (NA) and serotonin (5HT) play a role in the eating of novel foods (neophagia). For example the consumption of novel food has been shown to be reduced by fenfluramine or 5MeODMT [20], 5HT mimetic drugs, and enhanced by methysergide or propranolol [21], antagonists respectively of 5HT and beta-adrenergic receptors. There is also evidence that the amygdala, an area containing 5HT and NA neurons [3,18], is involved in the neophagic response of rats to food [2,17]. Thus, electrolytic lesions of the basolateral region of the amygdala (ABL) lead rats to spend more time eating novel as compared to familiar food [17].

Given these findings, we investigated whether NA or 5HT in the amygdala are involved in these effects of previous experience on food intake. To investigate this, we determined whether injection of NA or 5HT into the ABL or the selective destruction of NA or 5HT neurons in the ABL by 6-hydroxydopamine (6OHDA) [7] or 5,7-dihydroxytryptamine (5,7DHT) [1], modified the choice of food in a food preference test.

As the basolateral amygdala has also been shown to be involved in another situation in which previous experience affects food intake, learned taste aversion [13,16], we also investigated whether NA or 5HT depletion in the amygdala modified learned taste aversion to a solution paired with sickness.

EXPERIMENT 1

The aims of this experiment were to confirm that lesions of the basolateral amygdala in the rat do lead to increased eating of novel foods and to a deficit in learned aversion as previously reported [16,17], and to determine the validity of the test paradigms and the coordinates of the amygdala in which it was appropriate to inject or deplete NA and 5HT in this strain of rats.

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METHOD

Male CD-COBS rats weighing 250–270 g initially were used throughout these experiments. The animals were housed singly at constant room temperature (21±1 degree C) and relative humidity (60%) with a 12 hr light-dark cycle (lights on at 7 a.m.).

For the food preference test, the procedure used by Rolls and Rolls [17] was followed. The rats were deprived overnight and tested in the morning in a test cage containing familiar food (chow pellets) and novel foods (greens, sultanas, potatoes, cookies and carrots). During the 10 min test, the time spent eating each type of food was recorded.

For the taste-aversion test, the rats were trained to a daily 10-min single bottle drinking schedule with tap water for 7 days. On the eighth day the tap water was replaced with 15% (w/v) sucrose and the rats were injected intraperitoneally with 20 ml/kg of 0.15 M LiCl after the drinking session to produce sickness [13]. On day 9 the rats were again given a single-bottle drinking test with 15% sucrose for 10 min, and the difference in the weight of the bottle before and after the test was recorded as a measure of learned aversion. This particular procedure has been validated, and discussed, in earlier work ([13], page 630).

Bilateral electrolytic lesions were made in the basolateral part of the amygdala shown previously to reduce neophobia and taste aversion learning [16,17], using ethyl ether anesthesia and coordinates from the König and Klippel atlas [10] as follows: A=3.75; L=±3.7; H=−2.9 mm. The lesions were made with stainless-steel electrodes with a 0.3 mm diameter and the length of exposed tip, using an anodal current of 3 mA for 15 sec. Control animals were operated on similarly, but no lesion was made. Ten days after surgery the rats were tested in the food-preference test, and 20 days after surgery in the learned aversion test. Only animals with bilateral lesions confirmed by histology to be within the ABL are included in the results described below. There were nine such rats, and 15 sham-operated controls.

RESULTS

Examples of the bilateral lesions made in the 9 rats are shown in Fig. 1. The lesions were confined to the region between A=3.1 and A=4.13 mm according to the König and Klippel atlas [10]. No difference was found in body weight or daily food and water intake, measured on the 8th day after surgery, between ABL-lesioned and sham operated rats.

In the food-preference test, the electrolytically lesioned rats spent significantly more time eating novel foods (sham: 123±21 sec±SE; lesioned: 198±24 sec; $p<0.05$ by Student's t-test for independent means), and somewhat less time eating familiar chow (sham: 80.3±19.6; lesioned: 49.6±20.4 sec; ns). The interaction in a 2×2 (between-within) analysis of variance (SPLIT-PLOT, [9]) was significant at the 0.05 level, $F(1,16)=7.3$. (The degrees of freedom reflect a comparison between the 9 lesioned rats, and because this was a SPLIT-PLOT analysis, of the same number of sham operated rats.) The total eating times for the two groups of rats were not significantly different (sham: 203±14; lesioned 248±16 sec).

In the taste aversion test, electrolytically lesioned rats drank more sucrose solution than sham-operated animals (sham: 20.4±1.2 g sucrose±SE; sham + LiCl: 0.6±0.2; lesioned: 13.7±1; lesioned + LiCl: 3.7±1.4; $p<0.001$ for the interaction term in a 2×2 (between-between) analysis of variance (ANOVA), $F(1,20)=26.5$). (It may be noted that this effect was not due to altered acceptability of or exposure to the sucrose in the lesioned group, in that on the conditioning day, day 8, the sucrose intake of the lesioned group was 14.1±1.5 g±SE, and of the control group was 14.8±1.0 g.)

DISCUSSION

These results confirm the findings of Rolls and Rolls [16,17] that lesions of the basolateral amygdala in the rat result in decreased neophobia for foods (i.e., increased neophagia), and in impaired learned taste aversion. The results also established the coordinates of the amygdala in which injections or depletions of NA or 5HT were appropriate in the following experiments, and established the baseline level of performance in these tests in these rats.

EXPERIMENT 2

The aim of this experiment was to determine the effects of injections of NA and 5HT into the basolateral region of the amygdala on neophagia. The injections were made into the region of the amygdala shown in Experiment 1 to be involved in neophagia and learned aversion.

METHOD

The behavioral tests were the same as those used in Experiment 1.

Bilateral guide cannulae were constructed from 0.65 mm diameter stainless-steel tubing, fixed in Plexiglas holders, and implanted stereotaxically under ethyl ether anesthesia to end 2 mm above the ABL, using the coordinates established as appropriate in Experiment 1. Stainless-steel stylets, 0.3 mm in diameter and as long as the guide, kept the guides patent until the animals were given intracerebral injections 10 days later. The rats were accustomed to handling, and on the day of the test the stylets were withdrawn and replaced by bilateral injection units (0.3 mm diameter stainless-steel tubing) terminating 2 mm below the tip of the guides.

L-noradrenaline hydrochloride and serotonin creatinine sulphate (both from Fluka, Buchs, Switzerland), calculated as free bases (NA 100 nmoles=16.9 μg; 5HT 100 nmoles =17.7 μg) were delivered in volumes of 1 μl (for 5 to 50 nmoles) or 2 μl (for 100 nmoles) of 0.02% ascorbic acid (w/v) through an Agla micrometer syringe coupled to the injection units. NA or 5HT or vehicle was injected at a rate of 1 μl/min just before the food preference test. The locations of the cannulae were determined histologically after the experiments. Only data from rats in which the cannulae were located bilaterally in the ABL are included in the results described below.

RESULTS

The results of a first series of injections are shown in Table 1. It is shown that bilateral injections of 10 nmoles of NA produced a tendency to eat more of the familiar food and less of the novel foods than the vehicle injected rats, although this effect was not quite significant, $p=0.08$, $F(1,8)=3.8$ for the interaction term in a 2×2 (SPLIT-PLOT). The larger injections of NA (50 and 100 nmoles) increased the total feeding time without changing the pattern of choice, and produced side effects on behavior such as crawling and reduced grooming (numbers of groomings±SE: vehicle: 1.6±0.4; NA 50 nmoles: 0.3±0.2; NA 100 nmoles: 0.0). The dose of 100 nmoles of NA reduced the latency to eat (vehicle: 172±28 sec; NA 100 nmoles: 39±14 sec; $p<0.05$).

In view of the finding that the dose of 10 nmoles of NA

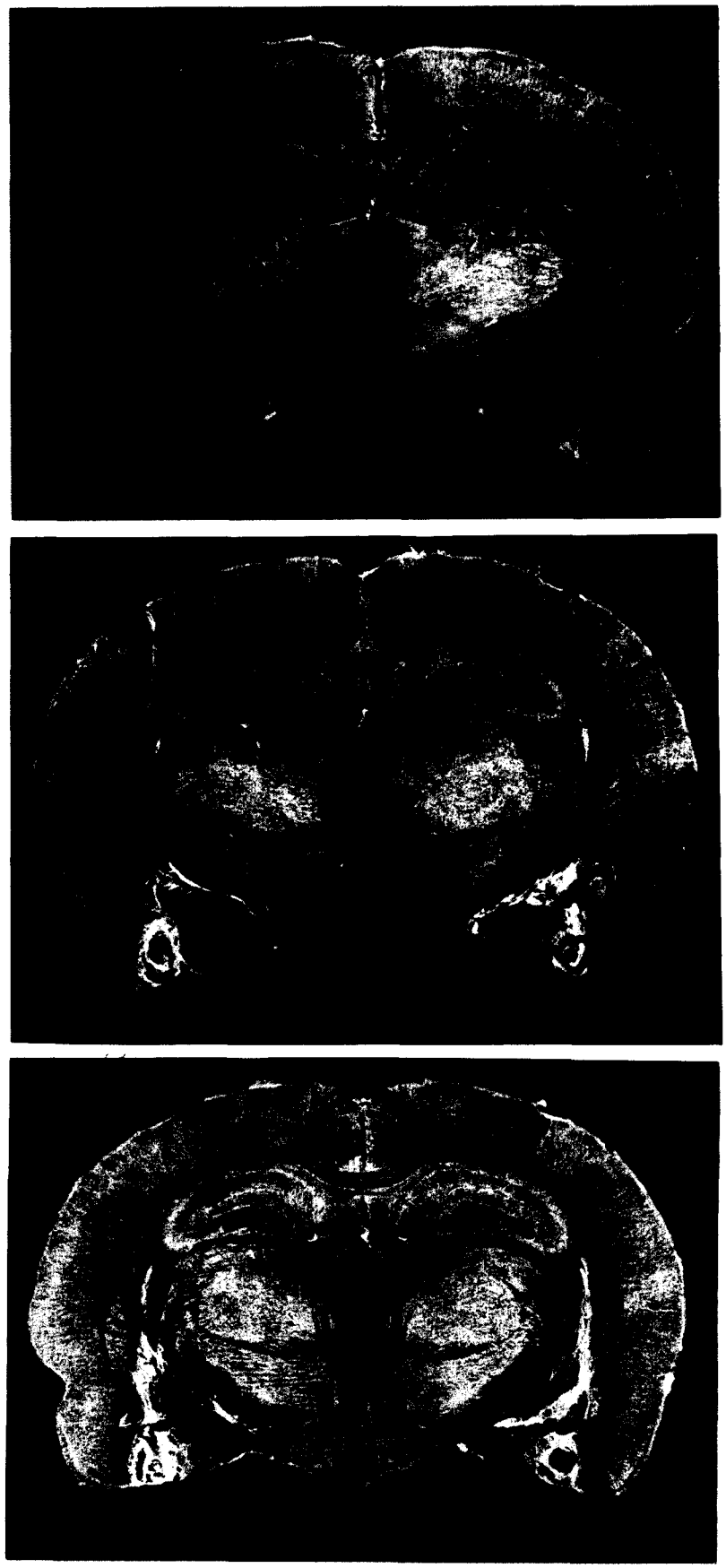

FIG. 1. An example of the location and extension of the bilateral electrolytic lesions in the basolateral region of the amygdala of one rat. The coronal sections are taken at different anterior coordinates to illustrate the anterior, middle, and posterior parts of the lesions.

TABLE 1

EFFECT OF NORADRENALINE (NA) AND SEROTONIN (5HT) INJECTED INTO THE BASOLATERAL AMYGDALA ON TIME SPENT EATING FAMILIAR OR NOVEL FOOD

Group	Dose (nmoles)	Total Time (sec)	Time (sec) Spent Eating: Familiar Food	Time (sec) Spent Eating: Novel Food	% of Time Spent Eating Familiar Food
Vehicle (5)		202 ± 11	119 ± 36	83 ± 28	59
NA (6)	10	225 ± 18	180 ± 16	45 ± 21	80
NA (6)	50	345 ± 33*	211 ± 62	134 ± 59	61
NA (6)	100	458 ± 32*	225 ± 88	233 ± 89	49
Vehicle (7)		251 ± 28	113 ± 23	138 ± 22	45
5HT (6)	10	370 ± 50†	209 ± 77	161 ± 75	56
5HT (5)	50	277 ± 19	174 ± 45	103 ± 33	63
5HT (4)	100	330 ± 36	205 ± 56	125 ± 39	62

Values are mean ± S.E. The number of rats used is reported in brackets.
NA and 5HT were injected just before the 10-min food preference test.
*$p<0.01$; †$p<0.05$ vs. respective vehicle-group (two-tailed Dunnett's test).

TABLE 2

EFFECT OF NORADRENALINE (NA) INJECTED INTO THE BASOLATERAL AMYGDALA ON TIME SPENT EATING FAMILIAR OR NOVEL FOOD

Group	Dose (nmoles)	Total Time (sec)	Time (sec) Spent Eating: Familiar Food	Time (sec) Spent Eating: Novel Food	% of Time Spent Eating Familiar Food
Vehicle (6)		228 ± 19	143 ± 13	86 ± 19	62
NA (4)	5	256 ± 33	174 ± 15	82 ± 23	59
NA (5)	10	347 ± 42†	291 ± 31*	56 ± 25	84
NA (4)	20	385 ± 30*	316 ± 35*	68 ± 51	82

Values are mean ± S.E. The number of rats used is reported in brackets.
NA was injected just before the 10-min food preference test.
*$p<0.01$; †$p<0.05$ vs. respective vehicle-group (two-tailed Dunnett's test).

tended to alter the rats' choice in the food preference test, the effects of NA were further investigated in a second series of injections of 5, 10 and 20 nmoles in a new group of rats. It is shown in Table 2 that 10 and 20 nmoles of NA significantly increased the amount of time spent eating the familiar food, and produced a nonsignificant decrease in the amount of time spent eating novel foods. (The 2×2 interaction SPLIT-PLOT was significant for the 10 nmole dose, $F(1,8)=12.8$, $p<0.01$, and for the 20 nmole dose, $F(1,8)=7.0$, $p<0.5$.) The total eating times were also increased by these injections. The dose of 5 nmoles of NA did not produce significant effects.

The injections of 5HT did not alter the selection of familiar as compared to novel foods (see Table 1). The lowest dose of 5HT led rats to spend more time eating than vehicle injected rats (see Table 1). The highest dose of 5HT produced side effects on motor behavior and flabbiness.

DISCUSSION

These findings are consistent with the hypothesis that NA can act in the amygdala to increase the acceptability of familiar foods relative to new foods (see significant interactions above). The effective doses were 10 and 20 nmoles of NA, which altered food preferences away from novel foods towards familiar food as well as having some effect on total eating time. The lower dose of 5 nmoles was ineffective, and the higher doses of 50 and 100 nmoles increased total eating time without altering food preference, and also had side effects on behavior.

EXPERIMENT 3

The aims of this experiment were to investigate the effects of depletion of endogenous NA and 5HT in the amygdala on food preferences and on taste aversion learning.

METHOD

NA depletion was produced by injections of 6OHDA and 5HT depletion by injections of 5,7-dihydroxytryptamine (5,7DHT) into the basolateral region of the amygdala. Ten μg of 6OHDA hydrochloride (Aldrich, Beerse, Belgium) or of 5,7DHT creatinine sulphate (Serva, Heidelberg, Germany),

TABLE 3

EFFECT OF 6-HYDROXYDOPAMINE (6OHDA) AND 5,7-DIHYDROXYTRYPTAMINE (5,7DHT) INJECTED INTO THE BASOLATERAL AMYGDALA ON TIME SPENT EATING FAMILIAR OR NOVEL FOOD

Group	Dose (μg)	Total Time (sec)	Time (sec) Spent Eating: Familiar Food	Time (sec) Spent Eating: Novel Food	% of Time Spent Eating Familiar Food
Vehicle (7)		157 ± 33	75 ± 33	82 ± 32	48
6OHDA (8)	10	108 ± 26	49 ± 25	60 ± 19	45
Vehicle (8)		127 ± 18	85 ± 18	42 ± 8	67
5,7DHT (8)	10	106 ± 21	68 ± 18	38 ± 11	64

Values are mean ± S.E. The number of rats used is reported in brackets.
Neurotoxins were injected 20 days before the food preference test.

both calculated as free bases, were dissolved in 2 μl of 0.1% (w/v) ascorbic acid and infused in 2 min by micropump using the coordinates for the ABL established in Experiment 1. The injection cannula was left in place one minute longer to allow for diffusion. Since it has been reported that 5,7DHT can be taken up into catecholaminergic endings [1], rats were pretreated intraperitoneally with 15 mg/kg nomifensine, a catecholaminergic uptake blocker [19], 30 min before receiving this neurotoxin. Thirty min before the 6OHDA injection, rats were treated intraperitoneally with 50 mg/kg pargyline, since this procedure is known to increase the neurotoxic effect of 6OHDA [7]. The behavioral tests were carried out 20 days after the neurotoxin injections, using the method described under Experiment 1.

The day after the behavioral experiment, the ABL of the rats was removed by the punching technique using the method described by Palkovits [14], and the content of NA and 5HT was assayed according to the methods described respectively by Keller *et al.* [8] and Ponzio and Jonsson [15].

RESULTS

As shown in Table 3, neither 6OHDA nor 5,7DHT modified food selection or the time spent eating. 6OHDA-treated rats showed an increased latency to eat (vehicle: 116±21 sec±SE; 6ODHA: 263±40 sec; $p<0.05$ by Student's t-test). No difference was found in body weight, or daily food and water intake of 6OHDA or 5,7DHT treated rats compared with vehicle injected rats 8 and 18 days after surgery.

The results of the taste aversion learning test are shown in Table 4. The 6OHDA-treated rats drank more sucrose in the test of learned aversion than the control rats (2.6±0.3 g vs. 0.6±0.3 g; $p<0.01$). A 2×2 analysis of variance indicated a significant interaction ($p<0.05$; $F(1,29)=4.9$) between 6ODHA injection and the LiCl treatment. Thus the 6ODHA produced an impairment of the learned aversion. (It may be noted that this effect was not due to altered acceptability of or exposure to the sucrose in the 6OHDA group, in that on the conditioning day, day 8, the sucrose intake of the 6OHDA group was 12.6±1.3 g±SE, and of the vehicle group was 12.3±1.0 g). The 5,7DHT did not produce and increase in the amount of sucrose consumed in the learned aversion, LiCl treated, group. (The significant interaction term, $F(1,12)=6$, $p<0.05$, was due to the increase in the drinking of sucrose evident in the 5,7DHT treated rats given only a saline injection on the previous day, as shown by post hoc Tukey analysis.)

TABLE 4

EFFECT OF 6-HYDROXYDOPAMINE (6OHDA) AND 5,7-DIHYDROXYTRYPTAMINE (5,7DHT) INJECTED INTO THE BASOLATERAL AMYGDALA IN THE TASTE AVERSION TEST

Group	Dose (μg)	10 min Sucrose Intake (g) By Rats Given The Previous Day: Saline	10 min Sucrose Intake (g) By Rats Given The Previous Day: or LiCl
Vehicle (15)		16.6 ± 1	0.6 ± 0.3*
6OHDA (18)	10	17.2 ± 1.1	2.6 ± 0.3*†
Vehicle (8)		16.6 ± 1	0.8 ± 0.2*
5,7DHT (8)	10	19.9 ± 1‡	0.6 ± 0.2*

Value are mean ± S.E. The number of rats used is reported in brackets.
Neurotoxins were injected 20 days before the taste-aversion test.
ANOVA (2×2): $p<0.05$ for both experimental groups.
Tukey's tests: *$p<0.01$ vs. respective saline group; †$p<0.01$, ‡$p<0.05$ vs. respective vehicle group.

Biochemical assay of the ABL showed that NA levels fell markedly in 6OHDA treated rats (vehicle: 5.25±0.49 ng/mg protein; 6OHDA: not detectable), as did 5HT in 5,7DHT treated animals (vehicle: 11±3 ng/mg protein; 5,7DHT: not detectable). (The minimal detectable levels were 1 ng/mg protein for NA and DA and 1.1 ng/mg protein for 5HT.) No dopamine was detected in the ABL of the vehicle-treated rats.

DISCUSSION

The injection of NA into the basolateral region of the amygdala in doses of 10 and 20 nmoles led rats to eat relatively more familiar food and relatively less novel food in the food preference test. This compares with an increase of eating of the novel foods compared to the familiar food produced by lesions of the ABL, as shown by Rolls and Rolls [17] and confirmed here. These findings provide further evidence for the view that a role of the amygdala in feeding is in

modulating food preferences by limiting the selection of and ingestion of new foods. The results also provide evidence that NA in the amygdala is involved in this, with high levels of NA increasing the proportion of familiar as compared to novel food ingested in the food preference test. Thus the rat appears to be biassed towards familiar food away from novel food as a result of the NA injection. Decreased learning of which foods are familiar may also be produced by NA in the amygdala, for injections of NA into the amygdala 30 min after the ingestion of a novel food decrease the amount of it ingested at a later retest when normal animals treated it as more familiar and ingest more of it [6].

The finding that depletion of NA in the ABL left the proportion of familiar as compared to novel food eaten low is consistent with the evidence obtained above that an elevation of NA in the ABL increases the relative acceptability of familiar as compared to novel food.

It has been reported that rats spent more time eating familiar food after 22 hr of food deprivation than after 3 hr, while the time spent eating novel food remained relatively unchanged [4]. However, it is unlikely that the present results are due to a simple change in hunger, for lesions of the amygdala and injections of NA into the amygdala both tended to increase total eating times, yet had the opposite effects on the proportions of novel as compared to familiar foods consumed. Further evidence that the manipulations of the amygdala did not have their main effect on the basic controls of feeding or hunger is that body weight or daily food intake was not influenced by amygdala lesions in this or the previous study [17], or by depletion of NA in the amygdala.

NA in the amygdala also appears to be involved in learned taste aversions, as shown by the finding that after the destruction of NA-neurons in the amygdala by 6OHDA, there was an attenuation of learned aversion to a solution previously paired with sickness. Animals trained in the one bottle paradigm used here must develop a strong inhibition if they are to demonstrate the aversion [9], and NA may be one of the transmitters involved in the inhibition of behavior in this situation.

It appears from the present experiments that 5HT in the amygdala does not play a role in either the effects of novelty on food preferences, or in learned taste aversion. It was found that injections of 5HT did not alter reactions in the food preference test. It was also found that the destruction of 5HT neurons in the ABL, as confirmed by biochemical assay, modified neither the pattern of choice of foods nor taste aversion learning.

The doses of 6OHDA and 5,7DHT were similar to those used by other investigators [5,11], and produced severe depletions of NA and 5HT respectively. It is likely that the depletion of NA was mainly within the amygdala, for the effect produced by the 6OHDA, an impairment of learned aversion, is a type of deficit associated with disruption of amygdaloid function, whereas if the 6OHDA had produced widespread catecholamine depletion, then aphagia and adipsia would have been expected. It is likely that the 6OHDA would also have depleted dopamine in the amygdala [11], and further investigations to determine whether this contributed to the impairment of learned aversion are suggested. These investigations could include pretreatment with desmethylimipramine to produce selective depletion of dopamine by 6OHDA. It is also likely that the 5,7DHT did not affect catecholamines in the amygdala, for no learned aversion deficit was produced by the 5,7DHT (see also [5]).

In conclusion, the results described here implicate NA in the basolateral region of the amygdala, but not 5HT, in two situations in which learning affects food selection. In the first situation, ingestion of a novel food is normally partly inhibited, and NA in the amygdala appears to enhance this inhibition. In the second situation, ingestion of a food associated by learning with sickness is normally inhibited, and NA in the amygdala appears to be necessary for this to occur normally.

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