AN INVESTIGATION
OF THE ROLE OF THE AMYGDALA
IN TASTE AVERSION LEARNING

by

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ABSTRACT

Contemporary theories of amygdaloid function postulate that the amygdala is involved in the association of events with aversive consequnces or in the inhibition of responses. Experiment 1 investigated the possibility that the amygdala is necessary for the learning of a conditioned taste aversion, a task requiring the association of taste with gastric distress and subsequent response inhibition. The performance of a group of intact rats was compared with that of two groups of rats with basolateral or corticomedial amygdaloid lesions. Since the groups of rats with amygdaloid lesions were impaired in the task, it was suggested that the amygdala is involved in the successful acquisition of a conditioned taste aversion.

In Experiment 2, an attempt was made to provide some information about the nature of amygdaloid involvement in taste aversion learning. Groups of rats trained before receiving amygdaloid lesions or trained after lesioning were found to be equally impaired in the task. It was suggested that the deficit produced by amygdaloid lesions is not simply an impairment in the learning of a taste aversion, but that it may be an impairment of performance. These findings are discussed from the view that the amygdala is necessary for the establishment of context and for relating events to an established context.

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The amygdala is known to be involved in the acquisition and performance of avoidance tasks which require the withholding of a response. A controversy concerning the exact nature of amygdaloid involvement exists, however. It is unclear whether the amygdala is involved with the learning (i.e., the association of events) or with the performance of such tasks. The present investigation attempts to clarify this issue by examining the nature of amygdaloid involvement in the acquisition of a conditioned taste aversion. The following discussion will reveal why such an investigation might yield information about some aspects of amygdaloid functioning.

Functional Anatomy of the Amygdala

The amygdala, a temporally-located subcortical structure, forms an integral part of the limbic system. Although the amygdala is generally divided into seven nuclear groups (medial basal, lateral basal, cortical, intercalated, central, medial, and lateral) most investigators follow the terminology of Johnston (1923) and recognize two groups of amygdaloid nuclei: basolateral and corticomedial. Due to the complexity of the amygdala, the differentiation between various nuclear groups is somewhat subjective (cf. Hall, 1972).

Progressing up the phylogenetic scale, the corticomedial zone becomes reduced in size while the basolateral area develops. These changes are accompanied by a rotation of the amygdala so that in man

the medial nucleus is located dorsally and the lateral nucleus assumes a ventral position (Gloor, 1960).

One of the main afferent connections to the amygdala is from the olfactory bulbs via the lateral olfactory tract to the corticomedial nuclei (Gloor, 1960). While this path is direct, a more indirect connection via the pyriform cortex exists also (Lammers, 1972). Although the olfactory connections appear to be primary, the amygdala receives indirect input from all sensory modalities (Gloor, 1960).

There is general recognition of two efferent systems from the amygdala--the dorsal system of the stria terminalis and the ventral amygdalofugal pathway--and it has long been noted that both these pathways are not exclusively efferent but contain amygdalopetal fibres as well (Cowan, Raisman, & Powell, 1965; Lammers, 1972). The stria terminalis has its origin in the corticomedial and basal nuclei and, skirting the internal capsule, distributes fibres to the septal area, olfactory tubercle, medial preoptic hypothalamic junction area, ventromedial hypothalamic nucleus, and the ventral premammillary area (Cowan et al., 1965; Heimer & Nauta, 1969; Lammers, 1972; de Olmos, 1972). The afferent fibres in the above system arise in the preoptic area, the anterior hypothalamic area, and the bed nucleus of the stria terminalis and terminate in the corticomedial and basolateral amygdaloid nuclei as well as in the periamygdaloid cortex (Lammers, 1972; see Figure 1).

Although there are difficulties in differentiating between

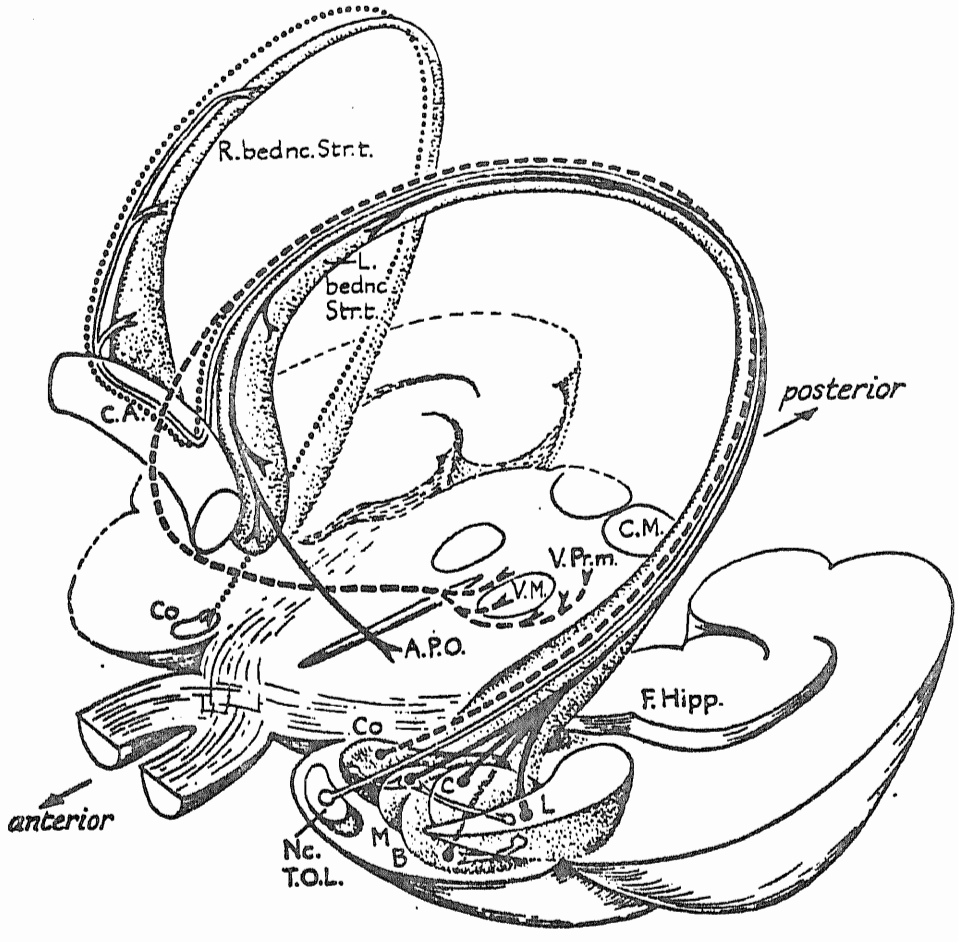

Fig. 1. Dorsal amygdalofugal pathway (From Lammers, 1972).

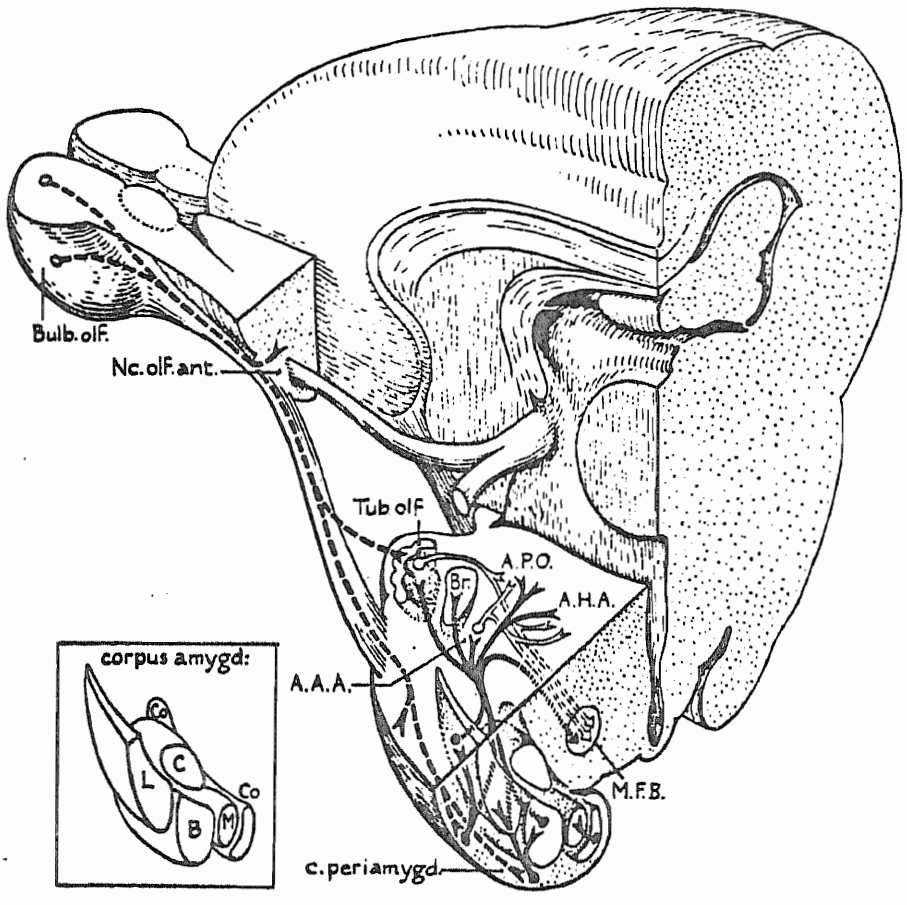

Fig. 2. Ventral amygdalofugal pathway (From Lammers, 1972).

those fibres originating in the pyriform cortex and those which originate in the basolateral amygdaloid area and merely pass through the pyriform cortex (Cowan et al., 1965), it appears that the ventral amygdalofugal pathway originates in the corticomedial and possibly in the basolateral amygdala and terminates primarily in the olfactory tubercle, diagonal band of Broca, lateral preoptic area, anterior hypothalamic area and lateral hypothalamic area (de Olmos, 1972; Lammers, 1972). The amygdalopetal portion of the ventral pathway originates in the preoptic area and in the rostral half of the hypothalamus and terminates in all the amygdaloid nuclei with the exception of the central nucleus (Cowan et al., 1965; see Figure 2).

A general note should be made concerning the efferent connections of the amygdala. While the ventral amygdalofugal pathway is larger and terminates in lateral areas of the brain, the dorsal pathway is more direct and terminates more medially (Cowan et al., 1965; Lammers, 1972).

Functions of the Amygdala

The amygdala is believed to be involved with a multitude of behaviours including emotionality, food and water regulation, avoidance learning, and sexual behaviour (cf. Eleftheriou, 1972; Gloor, 1960, 1972; Goddard, 1964b, 1972). Of interest here is the role of the amygdala in food and water intake and in avoidance learning. A discussion of these topics follows.

The amygdala and food and water regulation. In the early studies of the effects of temporal limbic lesions, Klüver and Bucy (Bucy & Klüver, 1940, 1955; Klüver & Bucy, 1938) reported subtle changes in the eating habits of the monkeys studied. These alterations usually involved new dietary choices--meat, for example. Later studies have reported both decreases in food intake following amygdaloid lesions (Brady, Schreiner, Geller, & Kling, 1954; Fonberg & Sychowa, 1968; Kling & Schwartz, 1961) as well as amygdaloid hyperphagia (Fonberg, 1968; Fuller, Rosvold, & Pribram, 1957; Green, Clemente, & DeGroot, 1957; Schwartzbaum, 1961). Amygdalectomy does not result in increased food motivation (Masserman, Levitt, McAvoy, Kling, & Pechtel, 1958) nor does it produce the voracity characteristic of animals with ventromedial hypothalamic lesions. Rather, in some cases amygdaloid hyperphagia seems to be characterized by constant nibbling which may be a component of the increased "orality" often seen in amygdalectomized animals (Klüver & Bucy, 1938). In any case, ablation studies would indicate that the amygdala appears to be involved in food intake regulation.

The results of stimulation studies generally are consistent with the findings of research using amygdaloid lesions. It has been shown that electrical stimulation of the basolateral amygdala results in the inhibition of food intake (Fonberg, 1963; Fonberg & Delgado, 1961; Lewińska, 1968; Robinson & Mishkin, 1968) while facilitation appears to result from corticomedial amygdaloid stimulation (Lewińska,

1968; Grossman & Grossman, 1963).

Because of the correspondence between the effects of hypothalamic and amygdaloid ablation and stimulation, it has been suggested (Kaada, 1972) that the medial and basolateral areas of the amygdala may duplicate the lateral hypothalamic "feeding centre" and the ventromedial hypothalamic "satiety centre". White and Fisher (1969) have shown that suppression of food intake resulting from amygdaloid electrical stimulation does not occur following lesions in the stria terminalis or in the ventromedial hypothalamus. Besides providing behavioural confirmation for anatomical data indicating that the stria terminalis is an important amygdalo-hypothalamic connection, the above findings led the authors to suggest that amygdaloid inhibition of the lateral hypothalamus is effected by impulses which are carried by the stria terminalis and ventromedial hypothalamus. Thus, the amygdala and hypothalamus may be components of a system of subcortical structures governing food regulation.

Since the effects of amygdaloid lesions are not as severe as those resulting from hypothalamic lesions, though they are of like kind, it has been suggested (Grossman, 1964) that the amygdala has a modulatory relationship with the hypothalamus. The results of chemical stimulation studies with the hypothalamus and amygdala lend support to the above notion. Grossman (1962) showed that adrenergic and cholinergic stimulation of the hypothalamus in sated animals would elicit food and water intake, respectively, and that adrenergic

and cholinergic blocking agents would produce the opposite effects. Adrenergic and cholinergic stimulation of the amygdala increased food and water intake, respectively, but the stimulation was effective only if the animals were deprived; in sated subjects, stimulation was ineffective (Grossman, 1964). These findings suggest that the amygdala may modulate the activity of the hypothalamus, but that its influence is ineffective in initiating hypothalamic activity. The results of the above studies also suggest that food and water intake may be governed by a cholinergic-adrenergic system common to the amygdala and hypothalamus (cf. Singer & Montgomery, 1969). Furthermore, it is evident that anatomical locus is not an exclusive determinant of functional specificity but that neuropharmacological "coding" is also important (Grossman, 1964).

The amygdala and avoidance learning. It is well established that animals with basolateral amygdaloid lesions are impaired in the performance of passive avoidance tasks (Goddard, 1964b; Kemble & Tapp, 1968; Pellegrino, 1968). Corticomedial amygdaloid lesions seem to result in a less severe passive avoidance deficit (Kemble & Tapp, 1968; Pellegrino, 1968). The above results and others showing amygdaloid impairment in reversal tasks (White, 1971), in conditioned emotional response (CER) tasks (Thompson & Schwartzbaum, 1964), and in unsignalled differential reinforcement of low rates (DRL) tasks (Pellegrino & Clapp, 1971) have led some authors to suggest that the amygdala should be intact if an animal is to withhold a response (cf. Goddard,

1964b), particularly one based on an internal cue (Pellegrino & Clapp, 1971).

Amygdaloid stimulation applied after the unconditioned stimulus (UCS) in CER tasks or after passive avoidance training has been shown to impair subsequent performance of these tasks (Goddard, 1964a; Kesner & Doty, 1968). These findings have led the authors to conclude that "near normal function in the amygdala is necessary for fixation of an aversive experience" (Kesner & Doty, 1968, p. 58) or that "one of the major functions of the amygdala is the consolidation of the association of a neutral stimulus with an aversive stimulus " (Goddard, 1964a, p. 30).

The above evidence suggests that the amygdala is involved with the "fear-motivated inhibition of responses" (Goddard, 1969, p. 17) and with the association of events and aversive consequences.

Theories of Amygdaloid Function

A number of general theories of amygdaloid function have been offered (Barrett, 1969; Gloor, 1972; Schwartzbaum, 1960a). Although these theories have a great deal in common, Gloor's (1972) position is the most comprehensive and for this reason it will be considered in some detail.

It is well established that the amygdala is involved with the motivational and affective life of animals (cf. Gloor, 1960; Goddard, 1964b); however, as previously noted in the discussion of the anatomy

of the amygdala and of its relation with other structures, the neural mechanisms subserving affect and motivation appear to be duplicated in the hypothalamus and amygdala. An explanation of the evolutionary significance of this occurrence requires consideration of the role of the amygdala in the life of lower organisms.

The primary afferent connection of the amygdala in lower organisms is with the vomeronasal organ (cf. Gloor, 1960), the phylogenetically older counterpart of the olfactory apparatus. In lower mammalian forms olfaction becomes the dominant sense in that it is involved with food-getting, maternal behaviour, reproduction, social behaviour, and avoidance behaviour (Pfaffmann, 1972; Ralls, 1971). In social behaviour, for instance, olfaction is important for the recognition of and differentiation between the dispositions of individuals (Ralls, 1971). This function obviously requires the formation of an olfactory memory and some mechanism for relating present events to the pattern of past olfactory experience. In this sense olfaction provides lower mammals with some flexibility in a system of behaviour patterns which often appears pre-programmed and inflexible. The place of the amygdala, then, is conceptualized by Gloor as a link between sensory input supplied by the olfactory apparatus and appropriate behaviour effected by the hypothalamus. Thus, the amygdala is "considered as a part of a system which processes olfactory signals, classifies them in the light of past experience, and thus takes part in the programming of motivated responses whose

effector mechanisms are integrated at the hypothalamic level" (Gloor, 1972, p. 436).

In primates and in man, visual and auditory information has greater importance than olfactory information and, accordingly, the temporal neocortex and amygdala in these higher mammals are known to be important in the receipt of such information (Gloor, 1972). Since hallucinations preceeding epileptic seizures and those produced by stimulation of the temporal lobe often are detailed, emotionally charged, and contain accurate images of an individual's experience (Penfield & Perot, 1963), Gloor suggests that in primates and man the temporal lobe "may be the neural substrate for the representation of the visual and auditory world" (Gloor, 1972, p. 438).

Taste Aversion Learning

Taste factors in the learned and innate control of the intake of specific nutrients have long been recognized (Harris, Clay, Hargreaves, & Ward, 1933; Richter, 1943; Scott & Quint, 1946a, 1946b). The classical work in this area established that an appetite for a nutrient can develop because eating an appropriately supplemented diet makes deficient animals "feel better" and that such learning occurs through the modality of taste. Furthermore, in the study of bait-shyness, Garcia and Ervin (1968) observed that rats which became ill, but survived, after eating a poisoned food formed an aversion to the taste of that food, but not to the container, location, or appearance

of the food. Such learned taste preferences and aversions are of obvious adaptive significance in an animal's regulation of its food intake.

In laboratory settings, conditioned taste aversions are formed by pairing a distinctive flavour with illness. An animal which has experienced both the taste and the illness will avoid consuming a substance with that taste when it is presented again. The generality of the phenomenon is indicated in that a number of conditioned stimuli (CSs) including saccharin and sucrose solutions, laboratory chow, salt solution, alcohol, chocolate milk, and Welch's grape juice have been used successfully. The illness (UCR) has been induced by a number of different agents such as X-irradiation and injections of lithium chloride, apomorphine, cyclophosamide, hypertonic saline, or insulin (see Revusky & Garcia, 1970, for a comprehensive review of this literature). A conditioned taste aversion is a very strong type of learning as indicated by its formation following one pairing of taste and illness with CS-UCS intervals as long as 7 to 12 hours (Revusky, 1968; Smith & Roll, 1967).

The mediation of a conditioned taste aversion does not seem to occur peripherally. Since the CS-UCS interval can be extremely long it has been suggested that a component of the CS lingers as an aftertaste or as a high concentration of the CS in the blood or that the CS reappears through regurgitation and is then associated with the illness. All these suggestions can be refuted. First, rats

cannot vomit (Garcia & Ervin, 1968) so the possibility of a regurgitated CS may be doubted. Using a 0.05% HCl solution as a CS and a 1 hour CS-UCS interval, Garcia, Green, and McGowan (1969) demonstrated taste aversions in rats. Since testing the rats' tongues with litmus paper 2 minutes after they stopped drinking revealed normal oral acidity, it is unlikely that an aftertaste bridged the 1 hour interval. Rozin (1969), using 2% and 10% solutions of casein hydrolysate, only one of which was followed 30 minutes later by an injection of apomorphine, has shown that rats can learn an aversion to a particular concentration of a substance. The difference in aftertaste between the two solutions would be negligible after 30 minutes. It has also been demonstrated that taste aversions for a particular temperature of water can be created (Nachman, 1970). It seems unlikely that an aftertaste could be operating for temperature cues. Rats are capable of learning a taste aversion to a specific solution even when other solutions are consumed between the drinking of the first solution and the illness (Kalat & Rozin, 1970; Revusky & Bedarf, 1967). This finding also suggests that aftertastes are not important in taste aversion learning. All the above evidence indicates that taste aversions are not peripherally mediated and that they do not occur as an artifact of the laboratory procedure.

The phenomenon of taste aversion learning has been used recently in arguments against an assumption of general process learning theory. While traditional views of learning have stated that all CSs

and UCSs are equally associable (cf. Estes, 1959, p. 399; Pavlov, 1927, p. 17), contemporary theorists have indicated that depending on the evolutionary history of the animal CSs and UCSs may be more or less associable (cf. Breland & Breland, 1961; Lockard, 1971; Rozin & Kalat, 1971; Seligman, 1970; Shettleworth, 1971). The finding that rats readily associate tastes with illness but have difficulty associating tastes with foot shock or exteroceptive stimuli with nausea (Garcia & Koelling, 1966) suggests that rats are "prepared" to make the former association but are "contraprepared" to form the latter ones (Seligman, 1970).

Rationale for the Present Investigation

It is proposed that the possible involvement of the amygdala in taste aversion learning should be investigated. Presented below are the four lines of reasoning from which this proposal develops.

It has been noted that although other cues are available, aversions as a result of poisoning are formed to taste cues. It would appear that the process by which the taste rather than other properties of the food becomes associated with aversiveness requires a distinction to be made between these cues in terms of their relevance in the taste aversion situation. Since it has been suggested that a function of the amygdala may be the formation of such distinctions, the amygdala may be involved in the acquisition of a taste aversion.

The ability to form taste aversions has obvious survival value.

A component of food intake regulation may follow a taste aversion--taste preference mode (Revusky & Garcia, 1970; Rozin & Kalat, 1971). Since the amygdala has been shown to be involved in food intake, it may also be involved in taste aversion learning.

Possible functions of the amygdala are the formation of associations between neutral and aversive stimuli and the inhibition of responses. Since the successful acquisition of a taste aversion requires the formation of such an association as a result of training and the subsequent inhibition of drinking in the test setting, the amygdala may be involved in the acquisition of a taste aversion. Since the basolateral amygdala appears to be more critical than the corticomedial amygdala in passive avoidance tasks, the basolateral amygdala might be expected to play a greater role than the corticomedial amygdala in the formation of a taste aversion.

If the amygdala is involved in taste aversion learning, amygdaloid lesions should disrupt the performance of the task. It has been shown that lesions in the lateral hypothalamus (Roth, Schwartz, & Teitelbaum, 1973), in the olfactory bulbs (Dinc & Smith, 1966), or in the ventromedial hypothalamus (Gold & Proulx, 1972) impair taste aversion learning. In the discussion of amygdaloid anatomy it was noted that the amygdala has connections with the olfactory bulbs and with both the ventromedial and lateral hypothalami via the stria terminalis and ventral amygdalofugal pathway. It may be that the olfactory bulbs, hypothalamus, and amygdala are components of a system

the integrity of which is necessary for the acquisition of a taste aversion. In this case, amygdaloid lesions would be expected to impair taste aversion learning.

In the present study the effect of corticomedial and basolateral amygdaloid lesions on taste aversion learning was examined.

EXPERIMENT 1

Method

Subjects

Thirty-one experimentally-naive male hooded rats (Quebec Breeding Farm and Laboratory, LaPrairie, Quebec) served as subjects. They weighed 258 - 281 gm at the beginning of behavioural testing.

Procedure

The animals were anaesthetized with an intraperitoneal (i.p.) injection of sodium pentobarbital (Diabutal, 60 mg/kg). A stainless steel electrode insulated except for 0.5 mm at the tip was stereotaxically located and electrolytic lesions were produced by passing 2 ma anodal dc current through the electrode for 15 seconds. Eleven animals received bilateral basolateral amygdaloid lesions (1.0 mm posterior to bregma, $\pm$ 5.0 mm lateral to the midline, and 7.0 mm ventral to the dura; incisor bar 5.0 mm above the intra-aural line [Pellegrino, 1968]) and 10 sustained bilateral corticomedial amygdaloid lesions (1.0 mm posterior to bregma, $\pm$ 3.5 mm lateral to the midline and 8.0 mm ventral to the dura; incisor bar 5.0 mm above the intra-aural line [Pellegrino, 1968]). Immediately after surgery, the animals were given an i.p. injection of 10 mg pentylenetetrazol (Metrazol) and an intramuscular injection of 0.1 ml streptomycin-penicillin suspension (Crystamycin).

All animals were housed in individual cages in which all behavioural testing took place. Throughout the experiment food (Purina Rat Chow) was available ad lib. The animals were weighed every second day. Six to eight days after surgery all animals including 10 unoperated control animals were placed on a restricted drinking schedule of 20 minutes per day which continued to the end of the experiment. All drinking sessions were run at the same time each day. The water bottles were weighed before and after being placed on the cage fronts and the difference in weight was taken as the measure of ingested fluid to the nearest 0.5 ml.

The training and testing schedule follows a procedure frequently used for taste aversion learning (cf. Garcia & Ervin, 1968). The animals were habituated to the drinking schedule for 7 days. On Day 8 and on every third day up to Day 17 sodium saccharin solution (0.1% w/w) was presented instead of water. On the first two saccharin days (Days 8 and 11) the animals were given a 10 ml/kg i.p. injection of 0.4 M lithium chloride 30 minutes after the end of the drinking period. These two sessions constituted the taste aversion training trials. The last three saccharin presentations (Days 11, 14, and 17) were used to assess the effect of taste aversion training.

Upon completion of testing, the lesioned animals were sacrificed with an overdose of sodium pentobarbital and were perfused intracardially with saline and 10% formalin. The brains were removed and preserved in 10% formalin. Sections at 40μ were made with a

freezing microtome (International Equipment Company, model CTD-1) and every fifth section was mounted on a glass slide. Using a projection microscope (Bausch and Lomb, model 42-63-65) the size and placement of lesions was determined by reference to the Pellegrino and Cushman (1967) rat brain atlas. Lesion reconstructions were done by a "blind" assistant.

Results

Behavioural Observations

The effects of lithium chloride injections were consistent across all groups. The symptoms of lithium chloride-induced nausea--partially closed eyes, drooping ears, lethargy--occurred reliably in all groups, regardless of whether they were intact or lesioned.

Histological Observations

For the purpose of the analysis of results, membership in the basolateral amygdalectomized (BLA) group required maximal basolateral amygdaloid damage and minimal corticomedial amygdaloid damage. Similarly, membership in the corticomedial amygdalectomized (CMA) group required maximal destruction of the corticomedial amygdaloid nuclei with minimal damage to the basolateral amygdaloid nuclei. Due to the nature of the lesions sustained, satisfaction of the above criteria meant that the eight animals which constitute the BLA group for the analysis of the results sustained more than 40% bilateral damage to the basolateral amygdalae and less than 5% damage to the corticomedial

amygdalae. Similarly, the six animals which make up the CMA group sustained more than 40% bilateral corticomedial amygdaloid damage and less than 5% damage to the basolateral nuclei. Reconstructions of two typical lesions as well as maximum and minimum extent of the lesions for both groups are presented in Appendix A.

Body Weight

Because animals in the control group tended to weigh more than animals in the lesioned groups (mean body weight: control, 251 gm; BLA, 227 gm; CMA, 225 gm; $p < .01$), a correction for these body weight differences was necessitated and intake is presented as the volume of fluid ingested per 100 gm of body weight (Figure 3 and in Appendix B). All statistical analyses were preformed on these corrected intake values. Tables for all analyses of variance are presented in Appendix C.

Taste Aversion

Acquisition of a taste aversion is indicated by a reduction in fluid intake when saccharin solution is presented on Days 11, 14, and 17. Retention was assessed by comparing the amount of saccharin solution ingested on each test day to the mean volume of water ingested on the two preceeding days. Simple effects analyses (Bruning & Kintz, 1968, pp. 120-122) were used to compare the volume of saccharin solution consumed on each test day (Days 11, 14, and 17) with the mean volume of water ingested on the appropriate two preceeding days. These tests indicated that the lesioned groups, as

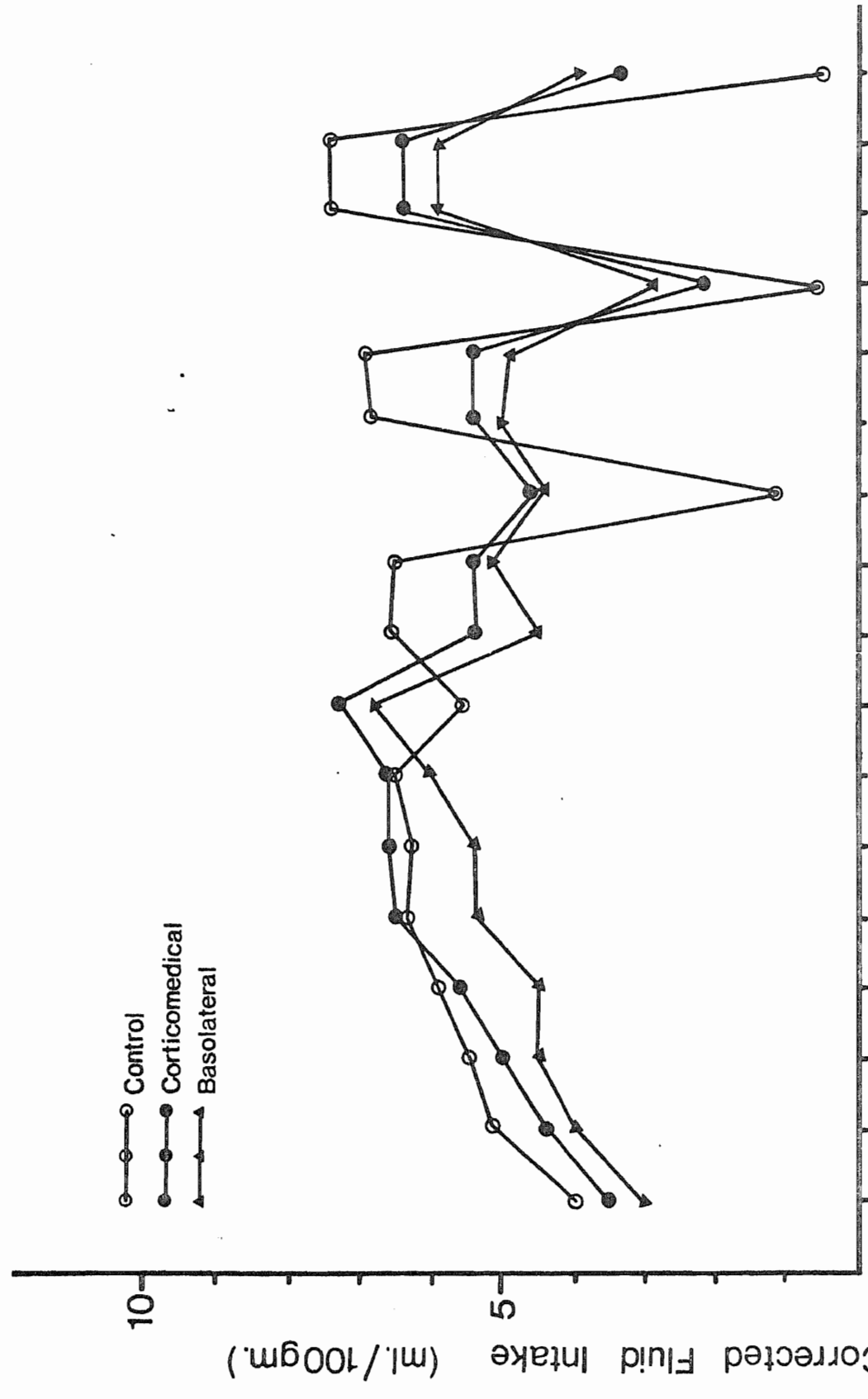

Fig. 3. Fluid intake per 100 gm of body weight (Experiment 1). Sodium saccharin solution (0.1% w/w) was presented on Days 8, 11, 14, and 17 and was followed 30 minutes later by an i.p. injection of lithium chloride (0.4 M, 10 ml/kg) on Days 8 and 11.

compared to the control group, were deficient in their retention of the taste aversion task (Days X Groups interactions: p's $< .001$).

The t tests comparing the volume of saccharin solution consumed on test Days 11, 14, and 17 to the mean volume of water ingested on the appropriate two preceeding days revealed that while the control group showed a significant aversion to saccharin on all three test days (Day 11: $t = 11.52$, $df = 9$, $p < .001$; Day 14: $t = 16.92$, $df = 9$, $p < .001$; Day 17: $t = 20.31$, $df = 9$, $p < .001$), the BLA and CMA group; showed a significant aversion only on the last two test days (BLA - Day 11: $t = 1.39$, $df = 7$, $p > .20$; Day 14: $t = 3.74$, $df = 7$, $p < .01$; Day 17: $t = 4.63$, $df = 7$, $p < .01$; CMA - Day 11: $t = 1.29$, $df = 5$, $p > .20$; Day 14: $t = 4.29$, $df = 5$. $p < .01$; Day 17: $t = 4.48$, $df = 5$, $p < .01$).

These results indicate that the taste aversion formed by the animals with basolateral or corticomedial amygdaloid lesions was weaker than that of the controls. Whereas the control group acquired a strong aversion in one trial, two training trials were necessary before the amygdalectomized animals showed a significant aversion.

Since the lesioned groups differed significantly from the unoperated control group on all measures of taste aversion learning but did not differ significantly from each other, the possibility that the impairment was due to a general disturbance produced by the lesioning procedure rather than to specific basolateral or corticomedial amygdaloid damage was considered. For this reason the data from the

animals rejected because of insufficient amygdaloid damage were also examined. The four animals (two from each of the BLA and CMA conditions) which sustained the least damage to the amygdaloid nuclei but which had lesions comparable in size to those of the animals in the BLA and CMA groups did not differ significantly from the unoperated controls on any measure of taste aversion learning (all $F's < 1$) but did differ from the BLA and CMA groups on all measures of taste aversion learning (all $F's \geq 13.70$, $df = 1/24$, all $p's < 0.005$). Therefore, the impairment shown by the BLA and CMA groups appears to be due to damage to the basolateral and corticomedial amygdalae rather than to the lesioning procedure.

Extinction

Since the third saccharin presentation (day 14) was not followed by a lithium chloride injection, the fourth saccharin session (Day 17) was an extinction trial. Analyses of variance and simple effects analyses between the quantities of saccharin solution consumed on the last two test days (Days 14 and 17), see Figure 3) revealed that the rate of extinction of the lesioned groups was more rapid than that of the control group (Days X Groups interactions: $p's < .001$). This result is consistent with the finding that the amygdalectomized groups showed a less marked aversion.

Response to the Initial Presentation of Saccharin

The initial presentation of saccharin solution (Day 8, see Figure 3) resulted in a decrease in fluid intake from the previous

day's level for the control group while the lesioned groups did not show this decrease, but rather increased their fluid intake (Days X Groups interaction: $p < .025$). Simple effects analyses revealed that while the BLA and CMA groups performed similarly the control group differed significantly from the BLA and CMA groups ($p < .01$, $p < .05$ respectively). This difference between the control group and the amygdalectomized groups may indicate a failure of the amygdalectomized groups to show avoidance of the novel solution (neophobia).

Water Intake

Before training, the BLA, CMA, and control groups did not differ significantly in water intake (mean water intake on Days 6 and 7: $p > .10$). Between the first training day and the following day the lesioned groups, unlike the control group, showed a reduction in fluid intake (Days X Groups interaction: $p < .005$; see Figure 3). Simple effects tests revealed that this difference is due to the differences between the control group and the lesioned groups (p's $< .025$) rather than to differences between the lesioned groups ($p > .10$).

Comparison of the mean water intake of the groups on the pairs of days between the saccharin days revealed that on the days before the first and last test days (Days 9 and 10 and Days 15 and 16) the BLA and CMA groups drank significantly less water than did the control group (p's $< .01$; see Figure 3). On the days between the first and second test days (Days 12 and 13) the water intake of the groups did not differ significantly ($.10 > p > .05$).

These differences in water intake might be account for in part by the fact that the control animals tended to drink less on the saccharin days than did the lesioned animals (see Figure 3).

Discussion

The results of the above experiment indicate that basolateral or corticomedial amygdaloid lesions impair the acquisition of a conditioned taste aversion. The finding that basolateral amygdalectomized animals are impaired in the performance of this task is consistent with other studies showing basolateral amygdaloid impairment in other tasks requiring response inhibition (e.g., Kemble & Tapp, 1968; Pellegrino, 1968; Thompson & Schwartzbaum, 1964). In studies comparing the effects of basolateral and corticomedial amygdaloid lesions in passive avoidance tasks involving electric shock it has been shown that corticomedial amygdaloid lesions tend to produce a smaller impairment than do basolateral amygdaloid lesions (Kemble & Tapp, 1968; Pellegrino, 1968). The present finding that basolateral and corticomedial amygdaloid animals tend to be equally impaired in taste aversion learning raises the possibility of a greater involvement of the corticomedial amygdala in this task than in other passive avoidance tasks. Alternately, cytoarchitectural and chemoarchitectural studies of the amygdala (cf. Hall, 1972) have suggested that the amygdala is a very heterogeneous structure and that the division of the amygdala into corticomedial and basolateral areas may be somewhat artificial. From this perspective,

variability in the behavioural effects of amygdaloid lesions reported by others might result from minor variations in lesion size and placement between studies.

EXPERIMENT 2

Although Experiment 1 indicated that amygdaloid lesions tend to disrupt the acquisition of a conditioned taste aversion, the nature of amygdaloid involvement in this task was not determined. Since it is already well established that basolateral amygdaloid lesions impair the performance of tasks requiring response inhibition (Kemble & Tapp, 1968; Pellegrino, 1968), it was decided to continue the investigation of amygdaloid involvement in taste aversion learning using corticomedial amygdaloid lesions.

Impaired taste aversion learning after amygdaloid lesions is consistent with the contemporary views of amygdaloid lesion effects. The disruption may be due to a failure to associate taste with illness (a deficit in the ability to learn) or it may be due to a failure of response inhibition (a performance deficit). These possibilities can be separated in the following manner. To ensure unimpaired learning and to assess the effect of amygdaloid lesions on performance alone a group of animals can be trained while intact and lesioned prior to testing. If amygdaloid lesions disrupt the learning but not the performance of the conditioned taste aversion, those animals trained prior to lesioning would be unimpaired in the performance of the task. However, if amygdaloid lesions disrupt the performance of a taste aversion, such a group of animals would tend to be impaired in the performance of the task. Similarly, if amygdaloid lesions disrupt the

learning of the task, a group of animals lesioned prior to training as well as a group lesioned after training would tend to be impaired in performance. Therefore, the role of the amygdala in taste aversion learning was examined by comparing the effects of amygdaloid lesions sustained before or after taste aversion training.

Method

Subjects

Thirty experimentally naive male hooded rats obtained from the Quebec Breeding Farm and Laboratory, La Prairie, Quebec, served as subjects. They weighed 164 - 212 gm at the beginning of the experiment.

Procedure

The surgical and histological procedures were the same as in Experiment 1. Twenty animals sustained corticomedial amygdaloid lesions and ten animals received sham lesion operations. The sham lesion operating procedure was identical to the lesioning procedure except that no current was passed through the electrode.

All testing took place in the individual cages in which the animals were housed. The animals were provided with food (Purina Rat Chow) ad lib. throughout the experiment and were weighed daily. The restricted drinking schedule and taste aversion training procedure were identical to those used in the previous experiment.

The animals were randomly assigned to four groups. Of the two experimental groups, each consisting of ten animals, one group was lesioned prior to training (the lesion-train group) and the other

was trained before being lesioned (the train-lesion group). For each of these experimental groups a group consisting of five animals was sham lesioned at the same time as its corresponding experimental group was lesioned.

As illustrated in Table 1, the experiment was composed of four stages. The first stage consisted of 9 days of restricted drinking. The eighth day of the period was the taste aversion training day for the train-lesion group and its control group. (Throughout this first stage, the lesion-train group and its control group had access to water on the restricted schedule to equate the effects of the restricted drinking schedule across groups. These latter two groups did not receive taste aversion training in this stage of the experiment).

The second stage of the experiment (Days 10 - 19) consisted of 10 days free access to food and water. On the fourth day of this period (Day 13) the animals received either bilateral corticomedial amygdaloid lesions or sham lesions depending on whether they belonged to an experimental or control group. The remaining 6 days of this period were provided to allow recovery from surgery and, in the case of the lesioned animals, to reduce any proactive effects of lesioning. The lesion-test interval was held constant in this experiment to equate the effects of recovery of function between the experimental groups.

For the train-lesion group and its control group, the third

TABLE 1

Design of Experiment 2

Days	Group			
	Train-lesion	Train-lesion Control	Lesion-train	Lesion-train Control
(Restricted drinking)				
1				
8	train	train		
9				
(Food and water ad lib.)				
10				
13	lesion	sham	lesion	sham
19				
(Restricted drinking)				
20				
27			train	train
29				
(Restricted drinking)				
30	test	test	test	test
33	test	test	test	test
36	test	test	test	test

Note: Unless otherwise specified, during the restricted drinking periods, all animals were allowed 20 minutes daily access to water. During the training and testing sessions, 0.1% w/w sodium saccharin solution replaced water and on the training days, the saccharin drinking period was followed 30 minutes later by a 10 ml/kg i.p. injection of 0.4 M lithium chloride.

stage (Days 20 - 29) consisted of the presentation of water daily for 20 minutes. For the lesion-train group and its control group, this stage consisted of 20 minutes daily access to water for the first 7 days (Days 20 - 26), the presentation of saccharin and taste aversion training on Day 27, followed by 2 days of restricted access to water.

The final (testing) phase of the experiment (Days 30 - 36) consisted of 7 days on the restricted drinking schedule for all groups. Saccharin solution was presented on Days 30, 33, and 36, and water was available on the intervening days.

Results

Behavioural Observations

As had occurred in Experiment 1, the symptoms of lithium chloride-induced nausea were consistent across all groups. No differences in the effects of the poison could be ascertained between lesion and control groups.

Histological Observations

Of the ten animals receiving lesions after training, seven sustained more than 40% bilateral corticomedial amygdaloid damage and less than 5% damage to the basolateral amygdalae. These seven animals make up the train-lesion group. Of the ten animals receiving lesions prior to training, one died in surgery and three failed to meet the above criteria for lesion size and placement. The remaining six animals comprise the lesion-train group. Reconstruction of two

typical lesions and maximum and minimum extent of the lesions are presented in Appendix D.

Body Weight

Unlike Experiment 1, there were no significant differences in body weight between groups ($p > .20$) in this experiment so a correction for body weight differences was not necessary. (Since the animals of Experiment 1 and Experiment 2 differ neither in body weight (t = 1.66, df = 45, $p > .10$) nor in fluid intake per 100 gm of body weight (t = 1.50, df = 45, $p > .10$) any differences in results between experiments may not be attributed to body weight factors or to factors concerning the relative quantities of fluid ingested by animals in either experiment.) The data are presented uncorrected for body weight differences (Figure 4 and in Appendix E) and all statistical analyses were performed on the uncorrected fluid intake values. Analysis of variance tables are presented in Appendix F.

Taste Aversion

To measure the acquisition and retention of the taste aversion, the difference between the volume of saccharin solution consumed on each test day and the mean volume of water consumed on the appropriate 2 preceeding days was considered. These differences were compared using unweighted means analyses (Winer, 1971, pp. 445 - 449). On the first test day (Day 30), the lesioned animals, regardless of their time of training, tended to be significantly impaired in the performance of the taste aversion ($p < .01$). The unweighted means

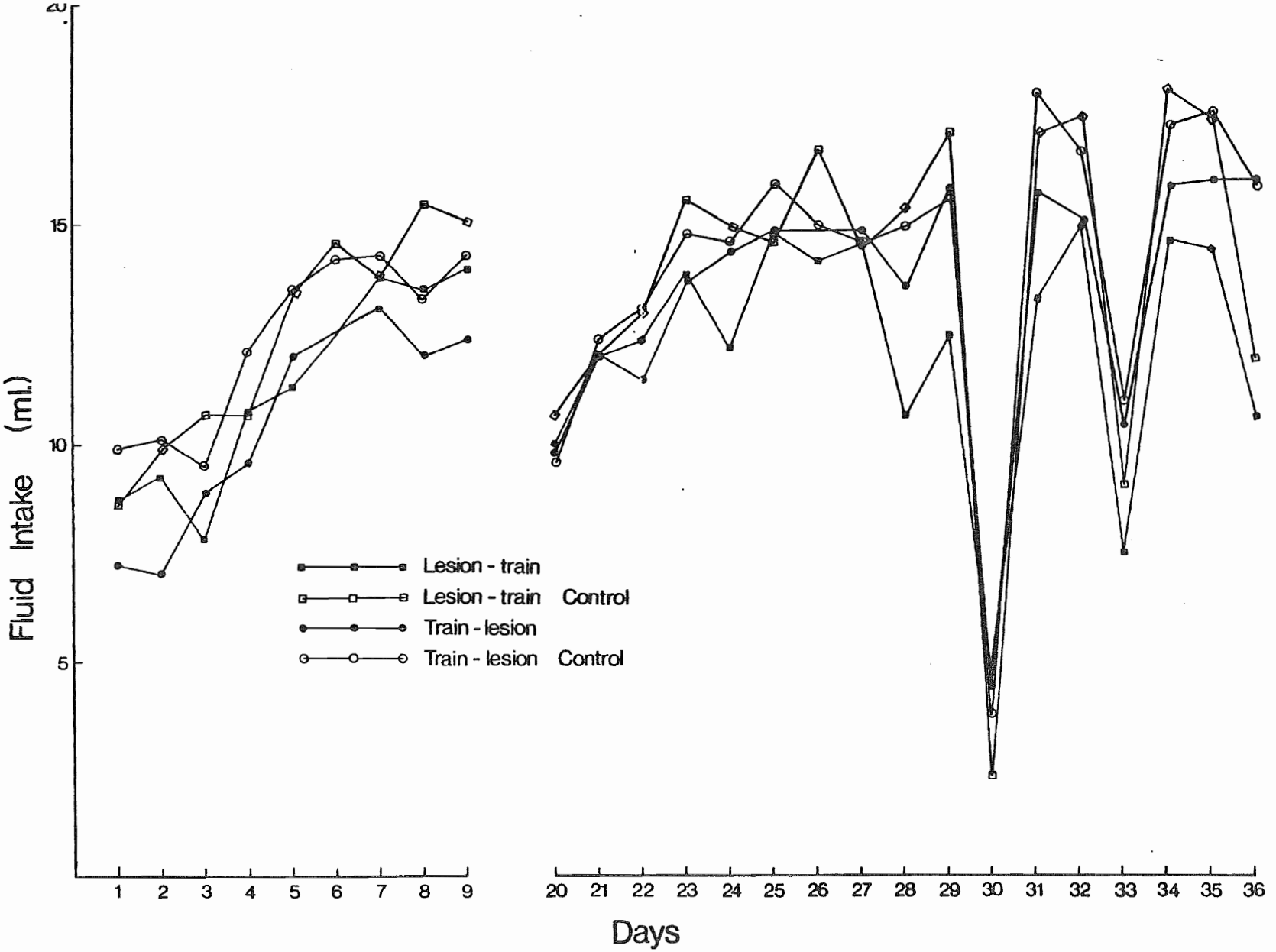

Fig. 4. Fluid intake (Experiment 2). The train-lesion group and train-lesion control group were trained on Day 8 and the lesion-train and lesion-train control group were trained on Day 27. To test acquisition of the taste aversion saccharin solution was presented to all groups on Days 30, 33 and 36.

analysis for the second test day (Day 33) yielded no significant differences between lesion and time of training conditions (F's < 1). On the third test day (Day 36), the animals which had been trained most recently (i.e., on Day 27 rather than on Day 8), whether or not they had been lesioned, tended to perform the taste aversion better than those which had been trained earlier ($p < .05$). In no case did the animals trained before being lesioned perform differently from those trained after lesioning (p's $> .05$).

Extinction

Since the three final saccharin presentations (on Days 30, 33, and 36) were not followed by injections of lithium chloride, the rates of extinction of the taste aversion may be analyzed by comparing the amounts of saccharin solution consumed on the last 2 test days. There is an obvious increase in saccharin consumption for all groups between the last 2 test days ($p < .001$, see Figure 4), but there is no significant difference in the rates of extinction between groups (Days X Groups interaction: $p > .20$).

Response to the Initial Presentation of Saccharin

As in Experiment 1, the amygdalectomized animals tended to increase their fluid intake when the saccharin solution was first presented while the unlesioned animals showed some avoidance of the novel situation. In the present experiment, however, comparison of the volume of saccharin solution consumed on the training day with the mean volume of water ingested on the previous day for the lesion-

train group versus the control groups and train-lesion group combined (all of which were unlesioned at the time of training) indicated that the effect is not statistically significant (Days X Groups interaction: $p > .20$).

Water Intake

The lesion-train group showed a reduction in fluid intake between the training day and the following day in contrast with the groups unlesioned at this time (Days X Groups interaction: $p < .025$). Consideration of the means of Days 25 and 26, Days 28 and 29, Days 31 and 32, and Days 34 and 35 revealed that only after training was there a significant difference in water intake between groups (Days 28 and 29 mean: $p < .001$; all other p's $> .10$).

These findings would suggest that the reduction in water intake by the amygdalectomized animals is related to their experience with taste aversion training rather than with saccharin solution alone.

Discussion

Since the group trained before lesioning showed the same impairment in taste aversion learning as did the group trained after lesioning, it may be stated that the effect of corticomedial amygdaloid lesions is not simply an impairment in learning. It is possible that corticomedial amygdaloid lesions produce a deficit in the ability to inhibit responses.

GENERAL DISCUSSION

Amygdaloid involvement in taste aversion learning will be discussed from three perspectives. The amygdala may be involved in this task because of its postulated involvement in response inhibition, in the association of neutral and noxious events, or in the establishment of context.

Impaired acquisition of a taste aversion may be viewed as resulting from one or more of the following: (a) the inability to distinguish between the test solution (e.g., saccharin) and water, (b) the inability to sense illness, (c) the inability to associate taste with illness, or (d) the inability to refrain from drinking in the test situation. Since amygdalectomized animals appear to be impaired in performing the taste aversion task, they would be expected to have at least one of the above inabilities.

Amygdalectomized monkeys have been shown to retain the ability to discriminate between saccharin and water (Weiskrantz, 1960) and amygdalectomized rats have been shown to be able to discriminate between sucrose and water (Rolls & Rolls, 1973a). The very fact that the amygdalectomized rats increased their fluid intake when saccharin was first presented indicates that they retained the ability to discriminate between saccharin and water.

Careful observation of the subjects in the present studies revealed that the symptoms of illness after the administration of

lithium chloride are consistent between amygdalectomized and control rats. Gastric upset is difficult to determine from external observation, but in the absence of a way to meter internal distress sensitively, this method must suffice.

The deficit resulting from amygdaloid lesions may be considered to be (a) an inability to form associations between events and aversive consequences (Goddard, 1964a) or (b) an inability to inhibit responding (Pellegrino, 1968). In Experiment 1, the relative inability of amygdalectomized rats to acquire a taste aversion may be seen as a failure to form the association between the taste of saccharin and gastric distress and, consequently, continued saccharin drinking. Alternately, the continued consumption of saccharin may be viewed as perseverative drinking in spite of the formation of an association between taste and illness. Since either hypothesis can explain the impaired performance of amygdalectomized animals, the results of Experiment 1 show simply that amygdaloid lesions tend to produce an impairment in taste aversion learning but they do not clarify the nature of the effect of amygdaloid lesions in this task or in others requiring response inhibition.

The results of Experiment 2 shed some light on the issue of corticomedial amygdaloid involvement in the learning and performance of a taste aversion. If the only effect of amygdaloid lesions were an impairment in the ability to associate taste and illness, the group trained after lesioning (i.e., the lesion-train group) would be

impaired in the task while the group trained prior to lesioning (i.e., the train-lesion group) would not be impaired. If amygdaloid lesions disrupted only the performance of a taste aversion, both of the above groups would be impaired by virtue of the lesion's presence during the retention tests. If both the learning and performance of the task were dependent on an intact amygdala, both of the above groups would be impaired. Thus, because chronic lesions were used, the experiment did not allow for differentiation between amygdaloid involvement in performance and in both learning and performance. Since both lesion-train and train-lesion groups were impaired in taste aversion learning, the results suggest that the corticomedial amygdala is involved in the performance of the task or that it is involved in learning and performance. Thus, the results indicate that the corticomedial amygdala is not involved simply in the association of taste with illness.

In both experiments reported here, the unlesioned animals tended to reduce their fluid intake when they first encountered saccharin. The amygdalectomized animals behaved differently, however, increasing their fluid intake when saccharin was first presented. Although neophobia in laboratory rats is weak in comparison with that of wild rats (Barnett, 1963, p. 29), laboratory rats do exhibit a substantial hesitancy to partake of novel foods (cf. Hankins, Garcia, & Rusiniak, 1973). Thus, the reduction in fluid intake by the unlesioned animals on the first saccharin day may be attributed to neophobia. The increase in fluid intake by the amygdalectomized

rats may be viewed as an absence of neophobia combined with a positive response to the sweet taste of saccharin.

The loss of neophobia through amygdalectomy may be due to the loss of a fear response to a novel taste or to a failure to refrain from drinking even if fear is present. Since the results showing involvement of the amygdala in taste aversion learning indicate that the effect of amygdaloid lesions is more than simply a disturbance in the ability to associate fear with a flavour, the results showing a loss of neophobia would indicate that this deficit is not simply a failure to associate fear with a novel flavour. The absence of neophobia may be interpreted as a simple failure to inhibit drinking or as a failure of inhibition together with a failure to associate fear with a novel flavour.

Since the absence of neophobia in amygdalectomized animals may be interpreted equally plausibly by either of these hypotheses, the finding that amygdalectomized animals are deficient in neophobia does not cast any further light on the nature of amygdaloid functioning.

The reduced water intake by amygdalectomized animals on the day after training is not explanable by either of the major recognized theories of amygdaloid function. If amygdaloid lesions result in a loss of response inhibition (Pellegrino, 1968) one would certainly not expect a reduction in fluid intake since such a reduction requires the inhibition of drinking. The suggestion that amygdalectomized

animals are characterized by a difficulty in forming associations between events and aversive consequences (Goddard, 1964a) does not predict an alteration in the level of water intake. It has been proposed that amygdalectomized animals have difficulty responding to internal cues (Pellegrino & Clapp, 1971). On the basis of the above hypothesis, an impairment in the acquisition of a taste aversion might be expected, nausea being an internal cue, but the intake of water should not be affected. Since amygdaloid lesions have been shown not to affect water intake (Rolls & Rolls, 1973a) and since the results of the present studies indicate that the posttraining reduction of water intake appears to be a function of taste aversion training, the present reduction in fluid intake does not appear to be a result of amygdaloid involvement in the regulation of water intake.

Because the reduced water intake of the amygdalectomized animals on the day following training is not explanable by the theories usually used to explain the behaviour of amygdalectomized rats, a different view of the role of the amygdala must be brought to bear. Gloor's (1972) conception of the amygdala as part of a system responsible for making decisions for the programming of motivated responses on the basis of past experience and present sensory input suggests that amygdaloid lesions would result in impairment in making such decisions. This point of view is not entirely novel. It has been proposed that "the difficulty experienced by amygdalectomized [animals] occurs in making the present relevant to past experience

(Barrett, 1969, p. 11)" and that "the impairment may...be related to processes which are necessary if the organism is to generalize appropriately among stimuli and utilize its experience when confronted with 'new' events (Schwartzbaum, 1960a, p. 394)". This accounts for amygdaloid impairment in tasks such as those involving internally ordered sequences (Barrett, 1969) and changes in reinforcing conditions (Schwartzbaum, 1960b), and transposition of a visual discrimination (Schwartzbaum & Pribram, 1960). Deficits in such tasks are not readily explanable by the postulation of failures in the association of events with aversive consequences or in response inhibition. From the perspective of the above theory, the reduced water intake by amygdalectomized rats on the day following training results from a relative inability of these animals to differentiate between those events which have and do not have aversive consequences. As has already been established, amygdalectomized animals have no difficulty differentiating between stimulus events (e.g., Weiskrantz, 1960; Rolls & Rolls, 1973a); the difficulty occurs in establishing the significance of events or in placing events in the appropriate context. Thus, the initial reduction in water intake may be thought of as a result of confusion on the part of the amygdalectomized animals as to which stimulus event, saccharin or water, results in illness.

In Experiment 1, the reduced water intake of amygdalectomized animals was prolonged while in Experiment 2, the water intake readily returned to its previous level. The difference between the results

of the two experiments may be seen as a function of the greater number of training trials given in Experiment 1. If amygdalectomized animals have difficulty establishing the context of events on the basis of their consequences, in Experiment 1, where there are repeated training trials the context remains unclear: only sometimes does nausea occur, and the water intake remains depressed. In Experiment 2, in which there is only one training trial, the context becomes more straightforward as the experiment progresses: illness occurs only once; the confusion disappears and the water intake returns to normal. Thus, although differences in water intake between groups may be related to the differences between control and lesion groups in quantities of saccharin solution ingested on the test days, it would appear that a difference between groups in interpretation of taste aversion training also plays a significant role.

The findings of amygdaloid impairment in neophobia and taste aversion learning may also be interpreted according to the view that amygdalectomized animals have difficulty forming an appropriate context and responding within it. The inability to acquire a taste aversion may indicate that amygdalectomized rats have difficutly establishing the context in which the taste of saccharin becomes an aversive cue. This deficit appears to be more than a failure to associate taste with gastric distress, however. The impairment in the performance of a taste aversion which was formed when the animal was intact suggests that the amygdalectomized animals are impaired in

responding within an appropriate context; that is, they are impaired in utilizing their experience. Since normal rats tend to avoid novel stimuli (Barnett, 1963, p. 28), the relative failure of amygdalectomized rats to exhibit neophobia may be regarded as a failure to respond within the context of a normal tendency to avoid novel stimuli.

Therefore, the present results are consistent with the theory that the amygdala may be involved in the relation of present events to a context based on past experience and that it may also be involved in the creation of that context. Because the suggestion that the amygdala is necessary for the association of events with aversive consequences or that the amygdala is necessary for response inhibition can account for only some of the results of the present experiments, while the argument that the amygdala is involved in the creation of context can account for all the results, the latter suggestion is deemed more adequate.

A Note and Some Possibilities for the Future

Since the above experiments were completed, the reports of two investigations of amygdaloid involvement in taste aversion learning have been published (cf. McGowan, Hankins, & Garcia, 1972; Rolls & Rolls, 1973a) and one paper has been presented on the same topic (cf. Ashe & Nashman, 1973). Because of experimental design differences between the present investigation and the published ones,

the absence of the neophobic response and the posttraining water intake reduction by the amygdalectomized animals are not evident in the published reports. (Neophobia impairment following amygdalectomy is reported by Rolls & Rolls, 1973b). Amygdaloid lesion disruption of taste aversion learning is consistent among all the studies.

Rolls and Rolls' (1973a) interpretation of the finding that basolateral amygdaloid lesions impair taste aversion learning is that the basolateral amygdala is involved with the inhibition of food intake on the basis of previous experience. The authors also suggest that amygdaloid modulation of food and water intake is mediated by neural pathways connecting the amygdala with the hypothalamus. McGowan, Hankins, & Garcia (1972) interpret the finding that amygdalectomized rats are unable to learn a taste aversion to indicate that amygdaloid lesions result in an inability to inhibit responses based on internal cues.

The analyses of Ashe and Nachman (1973) are more similar to the present analyses than are the published ones and, consequently, Ashe and Nachman's interpretation of their findings is closer to the interpretation of the results reported here. As in the present investigation, Ashe and Nachman report that amygdalectomized rats are deficient in neophobia and are impaired in the ability to acquire a taste aversion whether the animals are lesioned before training or before testing. They also report that amygdalectomized rats show a significant depression in water intake on the day following training

indicating "that there is some learning, although there is some confusion as to which is the appropriate fluid to avoid (Ashe & Nachman, 1973)". The above authors further report that although taste discrimination by amygdalectomized rats is normal, these animals do not respond to desoxycorticosterone--induced sodium appetite. This finding is attributable neither to a deficit in the ability to associate events with aversive consequences nor to an impairment in response inhibition. The authors conclude that "the amygdalectomized rat's inability to learn taste aversions is due to a deficit in recognizing the significance of taste stimuli (Ashe & Nachman, 1973)", a conclusion very similar to that reached on the basis of the data reported here. In contrast with the results of Experiment 1 above, Ashe and Nachman report that in their preliminary investigations more laterally placed amygdaloid lesions produced greater deficits in taste aversion learning; that is, basolateral amygdaloid lesions produced a greater deficit than did corticomedial amygdaloid lesions. Ashe and Nachman do not report their parameters but it is possible that a shorter CS - UCS interval or a higher dosage of lithium chloride obscured the difference in performance of the BLA and CMA groups in Experiment 1 reported here.

The conditioned taste aversion paradigm has demonstrated its utility in other areas. Because the exceptionally long CS - UCS interval allows various experimental treatments to be interposed,

the taste aversion paradigm has aided in investigations of memory consolidation and in determining the nature of such treatments as electroconvulsive shock (Ahlers & Best, 1972; Kral, 1970, 1971a, 1971b, 1972; Kral & Beggerly, 1973; Riege, 1969). Information about the role of specific brain structures in learning and general integrative information concerning the nature of brain function can be provided through research investigating the effects of stimulation of brain structures during the CS - UCS interval.

As noted in the General Discussion, the interpretation of the results of the present investigation is not unequivocal due to a difficulty which arises from the use of chronic lesions. An impairment in performance alone is indistinguishable from an impairment in both learning and performance. This problem in studying amygdaloid lesion effects on tasks involving response inhibition can be surmounted by employing a reversible lesion approach similar to that used by Albert and Mah (1973) to investigate the effects of septal lesions on the learning and performance of a passive avoidance task. The results of the studies reported here have implications, however, for the interpretation of such an investigation involving the use of reversible amygdaloid lesions. It is suggested that if there were a choice between interpreting the results of the proposed study from the point of view of amygdaloid impairment in associating events and inhibiting responses and from the point of view concerning amygdaloid involvement in the

establishment of context and in the ability to respond appropriately, that the latter viewpoint be taken.

Because ablation studies have indicated that the olfactory bulbs, amygdala, ventromedial hypothalamus, lateral hypothalamus, and perhaps the hippocampus (cf. Best & Orr, 1973; McGowan, Hankins, & Garcia, 1972; Miller, Elkins, & Peacock, 1971) are involved in taste aversion learning and because anatomical and physiological data indicate that important connections between these structures exist, it is proposed that the above structures are components of a subcortical system involved with the acquisition and performance of a conditioned taste aversion. The functioning of such a system and the relationships between the components of the system can be determined through a systematic investigation involving the use of cuts of suspected connections between structures (cf. Albert, 1969; Ellison, 1967; Halasz & Pupp, 1967; Sclafani & Grossman, 1969), chronic lesions, and reversible lesions. The involvement of structures can be determined by examining the effects of chronic lesions on the acquisition and performance of a conditioned taste aversion as well as on related measures (e.g., posttraining water intake in the present investigation) in the course of the experiment. Whether or not these structures are components of a system can be ascertained by determining whether or not severence of the connections between structures (without damage to the structures themselves) results in a deficit in the acquisition and performance of the taste aversion.

Information regarding the time at which a particular structure is involved can be gained by investigation of the effects of a reversible lesion applied during the CS, during or after the UCS, at various times between CS and UCS, or during the CS in the test setting. Because at various points in the learning process of the subjects only certain events have occurred, knowledge of the time at which a structure is involved in acquisition yields information concerning the nature of its involvement. Coincident with or following an investigation such as that proposed above, an investigation of the roles of the above structures in related learning tasks (e.g., one-trial passive avoidance, active avoidance, CER learning, and DRL learning) should occur to provide an integrated theory of the relation of a neural system to behaviour.

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APPENDICES

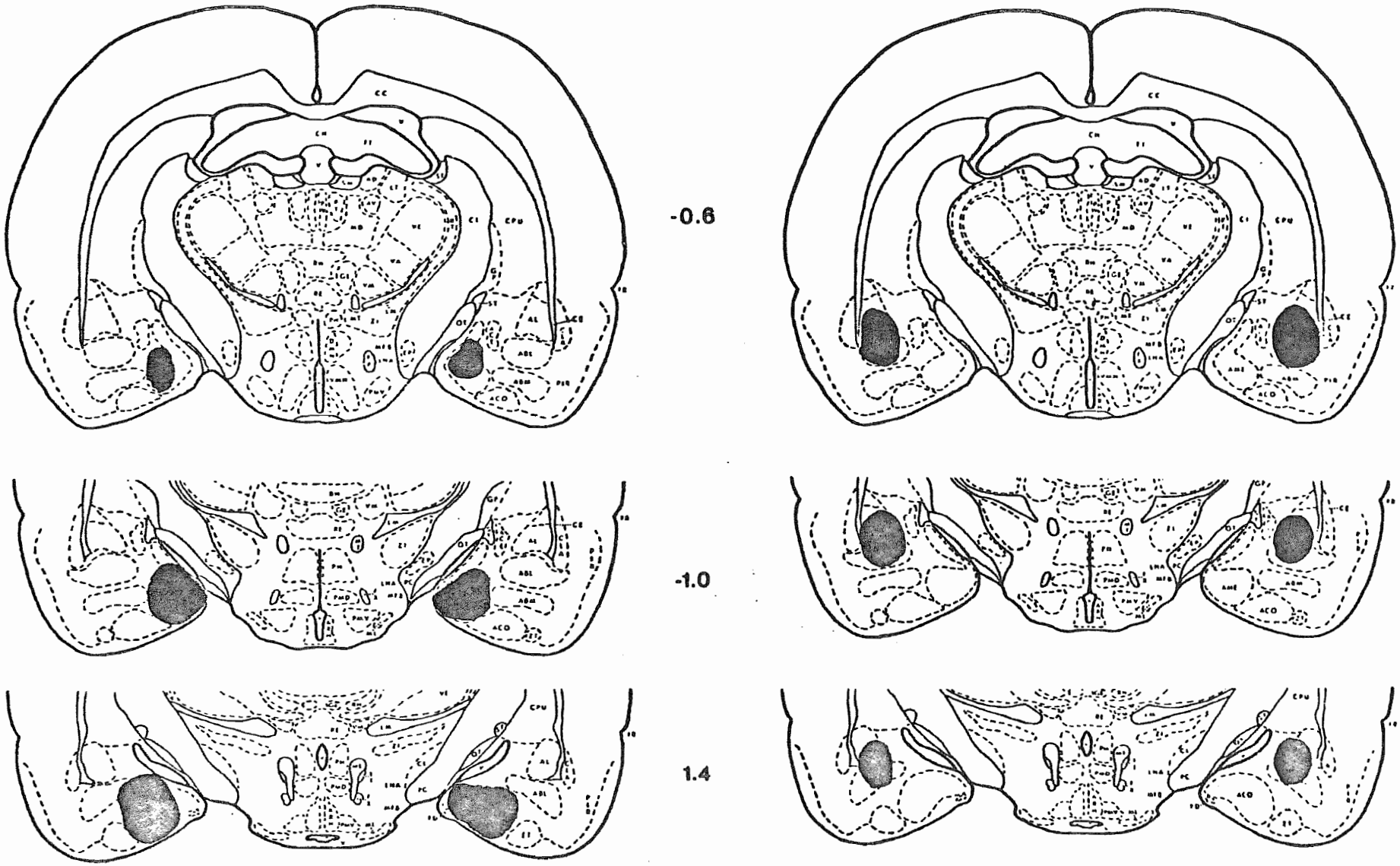

Fig. 1. Reconstructions of typical corticomedial (left) and basolateral (right) amygdaloid lesions drawn on sections from the Pellegrino and Cushman (1967) rat brain atlas. (Numbers refer to the anterior-posterior co-ordinates of the sections relative to bregma in the atlas.)

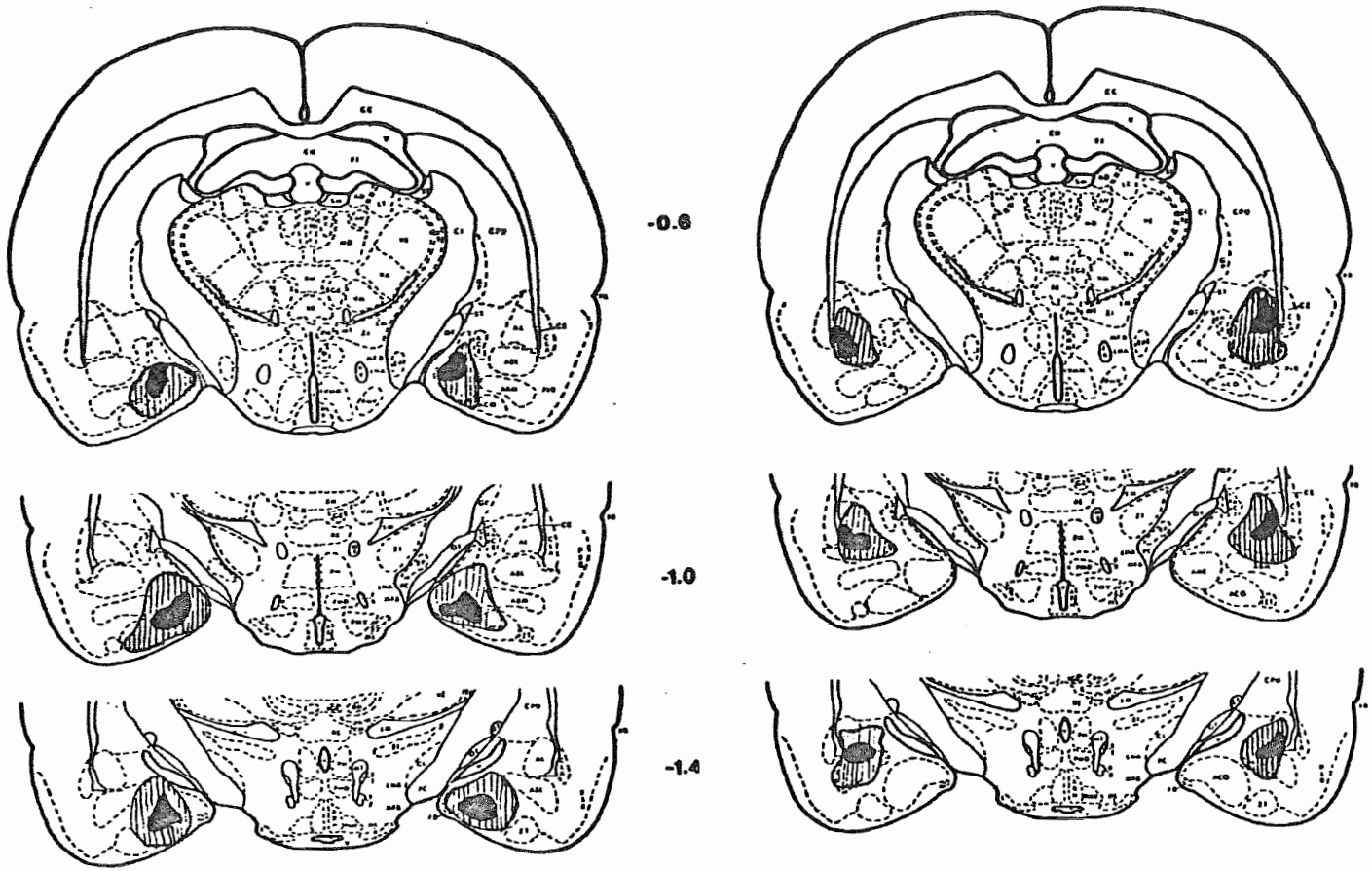

Fig. 2. Reconstructions of maximum (striped) and minimum (black) extent of corticomedial (left) and basolateral (right) amygdaloid lesions drawn on sections from the Pellegrino and Cushman (1967) rat brain atlas. (Numbers refer to the anterior-posterior co-ordinates of the sections relative to bregma in the atlas.)

APPENDIX B

TABLE 1

Mean Daily Fluid Intake (ml/100 gm) of Groups in Experiment 1

Days	Group		
	Basolateral	Corticomedial	Control
1	3.0	3.5	3.9
2	3.9	4.3	5.2
3	4.5	5.0	5.4
4	4.4	5.6	5.8
5	5.3	6.5	6.3
6	5.3	6.7	6.2
7	6.0	6.7	6.6
8	6.8	7.3	5.6
9	4.5	5.3	6.7
10	5.2	5.4	6.6
11	4.4	4.6	1.1
12	5.1	5.4	6.8
13	4.9	5.5	7.0
14	2.8	2.2	0.4
15	5.9	6.5	7.5
16	6.0	6.6	7.6
17	3.8	3.3	0.4

APPENDIX C

Analysis of Variance Tables of Experiment 1

The analysis of variance tables are presented here in the order in which the analyses are reported in Results of Experiment 1.

Error (a) denotes the pooled variance between subjects within groups, and Error (b) denotes the pooled variance between subjects within groups by days.

TABLE 1

Body Weight Comparison

Source	df	MS	F
Between groups	2	1645.77	6.09*
Within groups	21	269.98	

*$p < .01$

TABLE 2

Taste Aversion Analysis:
Comparison of Saccharin Solution Consumption on Day 11 with Mean Water Intake on Days 9 and 10 for All Groups

Source	df	MS	F
Groups	2	5.21	3.45*
Error (a)	21	1.51	
Days	1	86.14	88.80**
Days X Groups	2	34.63	35.70**
Error (b)	21	0.97	

*$p > .05$
**$p < .001$

TABLE 3

Taste Aversion Analysis:
Comparison of Saccharin Solution Consumption on Day 11 with Mean Water Intake on Days 9 and 10 (Simple Effects Analyses)

Source	df	MS	F
Days X Groups (BLA and control)	1	54.88	56.58*
Days X Groups (CMA and control)	1	42.06	43.36*
Days X Groups (BLA and CMA)	1	0.09	<1
Error (b)	21	0.97	

*$p < .001$

TABLE 4

Taste Aversion Analysis:
Comparison of Saccharin Solution Consumption on Day 14 with Mean Water Intake on Days 12 and 13 for All Groups

Source	df	MS	F
Groups	2	0.25	<1
Error (a)	21	1.44	
Days	1	217.18	182.50*
Days X Groups	2	22.24	18.69*
Error (b)	21	1.19	

*$p < .001$

TABLE 5

Taste Aversion Analysis:
Comparison of Saccharin Solution Consumption on Day 14 with Mean Water Intake of Days 12 and 13 (Simple Effects Analyses)

Source	df	MS	F
Days X Groups (BLA and control)	1	39.99	33.61*
Days X Groups (CMA and control)	1	18.44	15.50*
Days X Groups (BLA and CMA)	1	2.01	1.69**
Error (b)	21	1.19	

*$p < .001$
**$p > .20$

TABLE 6

Taste Aversion Analysis:
Comparison of Saccharin Solution Consumption on Day 17 with Mean Water Intake on Days 15 and 16 for All Groups

Source	df	MS	F
Groups	2	5.25	2.45*
Error (a)	21	2.14	
Days	1	248.43	264.29**
Days X Groups	2	30.97	32.95**
Error (b)	21	0.94	

*$p > .05$
**$p < .001$

TABLE 7

Taste Aversion Analysis:
Comparison of Saccharin Solution Consumption on Day 17 with Mean Water Intake on Days 15 and 16 (Simple Effects Analyses)

Source	df	MS	F
Days X Groups (BLA and control)	1	55.01	58.52*
Days X Groups (CMA and control)	1	29.52	31.40*
Days X Groups (BLA and CMA)	1	1.74	1.85**
Error (b)	21	0.94	

*$p < .001$
**$p > .10$

TABLE 8

Extinction Analysis:
Comparison of Saccharin Solution Intake on Days 14 and 17 for All Groups

Source	df	MS	F
Groups	2	42.26	10.59*
Error (a)	21	3.99	
Days	1	3.91	39.10**
Days X Groups	2	1.85	18.50**
Error (b)	21	0.10	

*$p < .01$
**$p < .001$

TABLE 9

Extinction Analysis:
Comparison of Saccharin Solution Intake on Days 14 and 17 (Simple Effects Analyses)

Source	df	MS	F
Days X Groups (BLA and control)	1	2.36	23.60*
Days X Groups (CMA and control)	1	2.84	28.40*
Days X Groups (BLA and CMA)	1	0.06	<1
Error (b)	21	0.10	

*$p < .001$

TABLE 10

Neophobia Analysis:
Comparison of Saccharin Solution Intake on Day 8
with Water Intake on Day 7 for All Groups

Source	df	MS	F
Groups	2	3.00	1.67*
Error (a)	21	2.57	
Days	1	0.03	<1
Days X Groups	2	4.25	5.31**
Error (b)	21	0.80	

*p > .20
**p< .025

TABLE 11

Neophobia Analysis:
Comparison of Saccharin Solution Intake on Day 8 with
Water Intake on Day 7 (Simple Effects Analyses)

Source	df	MS	F
Days X Groups (BLA and control)	1	7.20	9.00*
Days X Groups (CMA and control)	1	4.55	5.69**
Days X Groups (BLA and CMA)	1	0.11	<1
Error (b)	21	0.80	

*p < .01
**p < .05

TABLE 12

Water Intake Comparison:
Mean of Days 6 and 7 for All Groups

Source	df	MS	F
Between groups	2	2.15	1.78*
Within groups	21	1.21	

*p > .10

TABLE 13

Water Reduction Analysis:
Comparison of Day 8 Saccharin Solution Intake
with Day 9 Water Intake for All Groups

Source	df	MS	F
Groups	2	1.57	<1
Error (a)	21	2.24	
Days	1	7.36	3.40*
Days X Groups	2	17.92	8.27**
Error (b)	21	2.17	

*p > .05
**p < .005

TABLE 14

Water Reduction Analysis:
Comparison of Day 8 Saccharin Solution Intake with
Day 9 Water Intake (Simple Effects Analyses)

Source	df	MS	F
Days X Groups (BLA and control)	1	28.97	13.37*
Days X Groups (CMA and control)	1	17.16	7.92**
Days X Groups (BLA and CMA)	1	5.58	2.57***
Error (b)	21	2.17	

*p < .005
**p < .025
***p > .10

TABLE 15

Water Intake Comparison:
Mean of Days 9 and 10 for All Groups

Source	df	MS	F
Between groups	2	6.92	6.29*
Within groups	21	1.10	

*p < .01

TABLE 16

Water Intake Comparison:
Mean of Days 12 and 13 for All Groups

Source	df	MS	F
Between groups	2	3.91	3.18*
Within groups	21	1.23	

*$p > .05$

TABLE 17

Water Intake Comparison:
Mean of Days 15 and 16 for All Groups

Source	df	MS	F
Between groups	2	5.73	6.66*
Within groups	21	0.86	

*$p < .01$

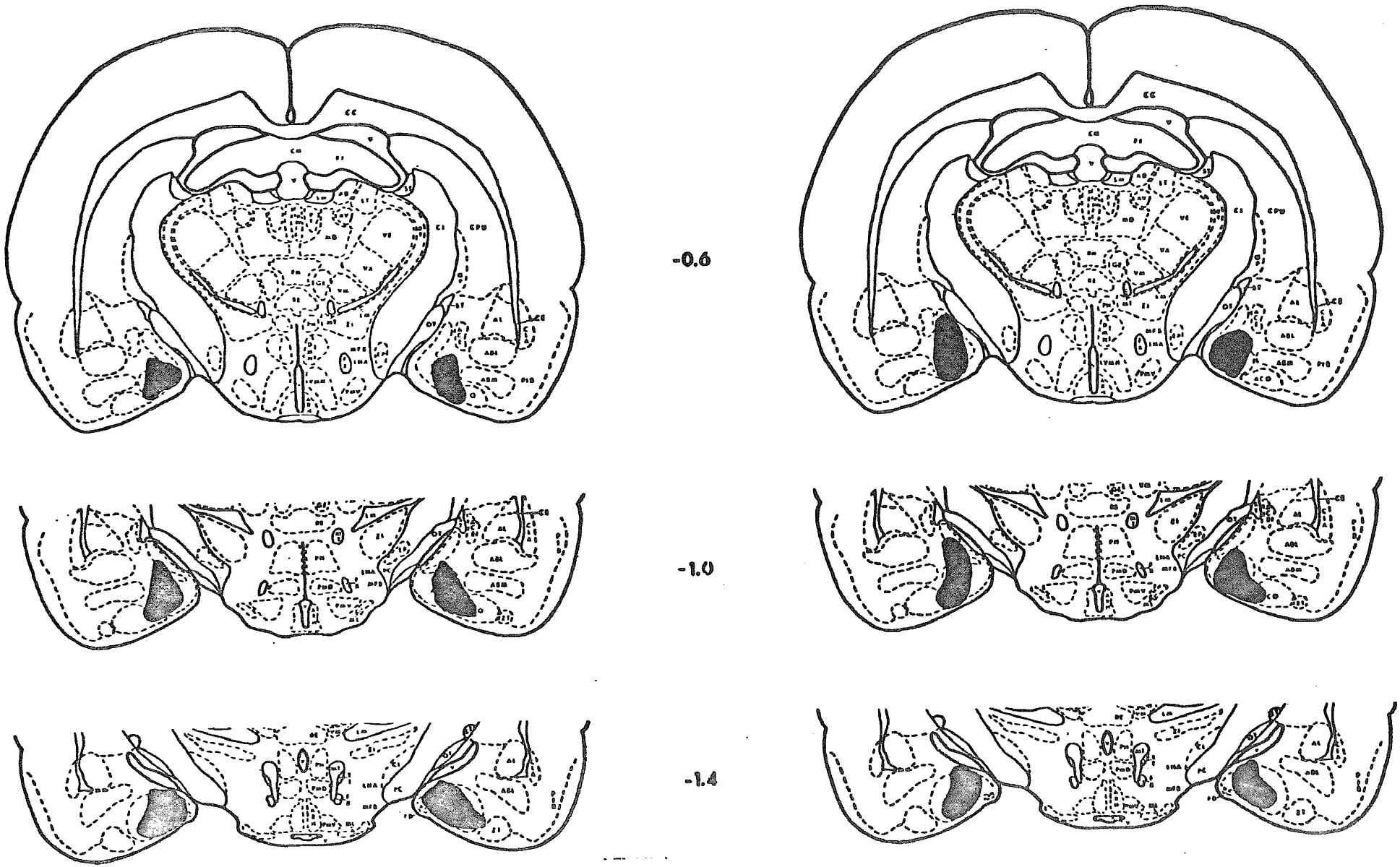

Fig. 1. Reconstructions of typical corticomedial amygdaloid lesions for the train-lesion (left) and lesion-train (right) groups drawn on sections from the Pellegrino and Cushman (1967) rat brain atlas. (Numbers refer to the anterior-posterior co-ordinates of the sections relative to bregma in the atlas.)

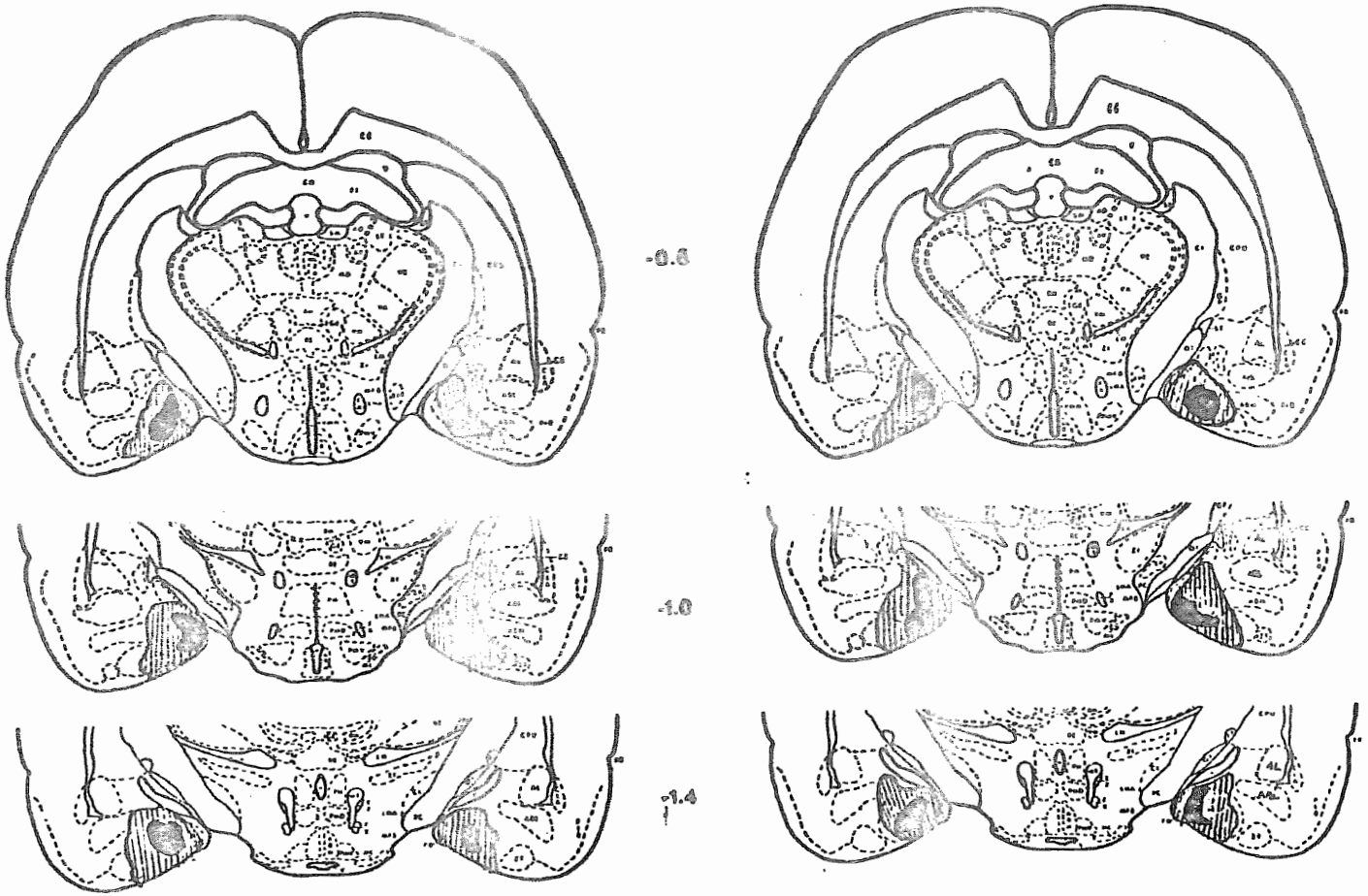

Fig. 2. Reconstructions of maximum (striped) and minimum (black) extent of lesions for the train-lesion (left) and lesion-train (right) groups drawn on sections from the Pellegrino and Cushman (1967) rat brain atlas. (Numbers refer to the anterior-posterior co-ordinates of the sections relative to bregma in the atlas.)

APPENDIX E

TABLE 1

Mean Daily Fluid Intake (ml) of Groups in Experiment 2

Days	Group			
	Train-lesion	Train-lesion Control	Lesion-train	Lesion-train Control
1	7.2	9.9	8.7	8.6
2	7.0	10.1	9.3	9.9
3	8.9	9.5	7.8	10.7
4	9.6	12.1	10.8	10.7
5	12.0	13.5	11.3	13.3
6	12.6	14.2	12.5	14.6
7	13.1	14.3	13.8	13.8
8	12.0	13.3	13.5	15.5
9	12.4	14.3	14.0	15.1
20	9.8	9.6	10.0	10.7
21	12.1	12.4	12.0	12.1
22	11.5	13.1	12.4	13.0
23	13.7	14.8	13.9	16.6
24	14.4	14.6	12.2	15.0
25	14.9	16.0	14.8	14.6
26	14.9	15.0	14.2	16.8
27	14.9	14.6	14.6	14.5
28	13.6	15.0	10.7	15.4
29	15.9	15.7	12.5	17.2
30	4.5	3.8	4.6	2.3
31	15.8	18.1	13.4	17.2
32	15.2	16.8	15.1	17.6
33	10.5	11.0	7.5	9.1
34	16.0	17.4	14.7	18.2
35	16.1	17.7	14.5	17.6
36	16.1	15.0	10.7	12.0

APPENDIX F

Analysis of Variance Tables of Experiment 2

The following analysis of variance tables are presented in the order in which they are reported in Results of Experiment 2.

Error (a) denotes the pooled variance between subjects within groups, and Error (b) denotes the pooled variance between subjects within groups by days.

TABLE 1

Body Weight Comparison

Source	df	MS	F
Between groups	3	498.59	1.206*
Within groups	19	413.27	

*$p > .20$

TABLE 2

Taste Aversion Analysis:
Unweighted Means Analysis of Difference between Mean Water Intake of Days 28 and 29 and Day 30 Saccharin Solution Intake

Source	df	MS	F
Lesion	1	98.05	8.33*
Time of Training	1	1.19	< 1
Lesion X Time of Training	1	47.75	4.06**
Within cell	19	11.77	

*$p < .01$
**$p > .05$

TABLE 3

Taste Aversion Analysis:
Unweighted Means Analysis of Difference between Mean Water Intake of Days 31 and 32 and Day 33 Saccharin Solution Intake

Source	df	MS	F
Lesion	1	12.69	< 1
Time of Training	1	18.27	< 1
Lesion X Time of Training	1	0.06	< 1
Within cell	19	20.60	

TABLE 4

Taste Aversion Analysis:
Unweighted Means Analysis of Difference between Mean Water Intake of Days 34 and 35 and Day 36 Saccharin Solution Intake

Source	df	MS	F
Lesion	1	30.74	2.19*
Time of Training	1	77.39	5.51**
Lesion X Time of Training	1	0.7142	<1
Within cell	19	14.04	

*p > .10
**p < .05

TABLE 5

Rate of Extinction Analysis:
Comparison of Saccharin Solution Intake on Days 33 and 36 for All Groups

Source	df	MS	F
Groups	3	49.32	1.92*
Error (a)	19	25.68	
Days	1	188.02	44.45**
Days X Groups	3	4.84	1.14***
Error (b)	19	4.23	

*p > .10
**p < .001
***p > .20

TABLE 6

Neophobia Analysis:
Comparison of Saccharin Solution Intake on Training Day
with Water Intake on Previous Day for
Lesion-train Group vs. Control Groups and Train-lesion Group Pooled

Source	df	MS	F
Groups	1	2.84	<1
Error (a)	21	16.17	
Days	1	11.50	2.53*
Days X Groups	1	6.15	1.35**
Error (b)	21	4.54	

*p > .10
**p > .20

TABLE 7

Water Reduction Analysis:
Comparison of Saccharin Solution Intake on Training Day
with Water Intake on Following Day for
Lesion-train Group vs. Control Groups and Train-lesion Group Pooled

Source	df	MS	F
Groups	1	6.34	<1
Error (a)	21	10.39	
Days	1	2.88	<1
Days X Groups	1	47.38	7.18*
Error (b)	21	8.60	

*p < .025

TABLE 8

Water Intake Comparison:
Mean of Days 25 and 26 for All Groups

Source	df	MS	F
Between groups	3	1.72	<1
Within groups	19	3.21	

TABLE 9

Water Intake Comparison:
Mean of Days 28 and 29 for All Groups

Source	df	MS	F
Between groups	3	23.74	9.44*
Within groups	19	2.51	

*$p < .001$

TABLE 10

Water Intake Comparison:
Mean of Days 31 and 32 for All Groups

Source	df	MS	F
Between groups	3	13.48	2.11*
Within groups	19	6.39	

*p > .10

TABLE 11

Water Intake Comparison:
Mean of Days 34 and 35 for All Groups

Source	df	MS	F
Between groups	3	14.53	2.20*
Within groups	19	6.59	

*p > .10