

Contextual Control of Conditioned Responding in Rats With Dorsal Hippocampal Lesions

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The control exerted by contextual cues over classically conditioned responding was assessed for rats with electrolytic lesions of the dorsal hippocampus and sham-operated controls. In 3 experiments the rats received initial training with 2 reinforced cues, each presented in its own distinctive context, followed by a nonreinforced test in which the cues were presented in the other context. Both control and operated subjects showed context specificity, as evidenced by less vigorous responding to these cues than to cues presented on test in their original contexts. The groups did not differ in their ability to learn an explicit discrimination in which a given cue was reinforced in one context and nonreinforced in a different context (although the groups did differ on a simple autoshaping task). It is concluded that a special role for the hippocampus in the contextual control of conditioned responding still remains to be demonstrated.

A conditioned response (CR) established in a given training context will often fail to occur with full strength when the conditioned stimulus (CS) is presented in a different context (see Hall, 1991, for a review). This *context specificity* of conditioning can occur for a number of reasons. The CS may suffer generalization decrement as a consequence of the contextual change, or associative strength acquired by the cues that make up the training context may contribute to the magnitude of the CR shown to the CS (Lovibond, Preston, & Mackintosh, 1984). In some procedures, however, context specificity is seen when such factors are controlled for (e.g., Hall & Honey, 1989, 1990). This finding has been interpreted as showing that contextual cues can acquire conditional control over associations that are formed in their presence, that is, these cues become able to facilitate the retrieval of associative information so that, when the context is changed, retrieval is less efficient and performance less vigorous. Another possible interpretation, difficult to distinguish empirically from that just outlined, is that during acquisition, conditioning occurs to some configural cue composed of the nominal CS and features of the context. A change of context will degrade or eliminate this configural cue and the CR will be less likely to occur.

According to several theories of hippocampal function, context specificity should fail to occur in animals that have suffered hippocampal lesions. These theories agree in supposing that there is more than one system for learning and that the hippocampus is not involved in all of them. In particular, they

hold that the conditioning mechanism whereby a CS acquires associative strength will be largely intact after hippocampal damage but that the system responsible for the sensitivity to contextual change will be lost. Thus, Sutherland and Rudy (1989) distinguished between simple associative learning and configural associative learning and argued that the hippocampus provides the neural basis for the latter. Whereas intact subjects might form associations involving a configural cue composed of the CS and aspects of the context, lesioned subjects will be able to form only simple associations. Acquisition of the CR may be similar in the two groups, but the difference between them should be evident on a test of context specificity, with hippocampal subjects showing transfer to a new context when intact subjects do not.

Hirsh (1974, 1980) also allowed that simple associative learning can occur without hippocampal involvement but argued that the hippocampus is required for the form of learning whereby certain events can come to operate as conditional cues, capable of selecting a given item of stored information for retrieval. To the extent that the context specificity of conditioning shown by normal subjects reflects the operation of a process of contextual retrieval, it is predicted that the effect will be eliminated in subjects with hippocampal lesions. Myers and Gluck (1994) recently put forward a development of their computational account of hippocampal function that they say is entirely consistent with Hirsh's (1974) account and may be seen as constituting an instantiation of his ideas.

The notion that the hippocampus plays an important role in the processing of information about contextual stimuli is central to the account proposed by O'Keefe and Nadel (1978) with its suggestion that the hippocampus carries out a mapping function, constructing a representation of spatial relationships among features of the environmental context. This account had its origin in experiments on spatial learning but was subsequently extended (Nadel & Willner, 1980) to address the results of studies of the effects of hippocampal lesions on other learning paradigms. Nadel and Willner argued that the environmental context should not be treated as a cue (or set of cues);

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rather, the context is said to *contain* the events that occur in it, and the relationship between context and such cues is described as hierarchical. If knowledge of this hierarchical relation plays a role in producing context specificity in intact subjects, then it might again be expected that such context specificity would not be found in subjects with hippocampal lesions.

Experimental tests of these predictions have given mixed results. Winocur and Olds (1978) trained rats with electrolytic hippocampal lesions and control rats on a simultaneous discrimination between horizontal and vertical in one choice apparatus and then tested the subjects with these same stimuli presented in a quite different apparatus. Their results lend no support to the theories described earlier. Far from showing a lack of context specificity, hippocampally lesioned subjects showed a particular sensitivity to the context change: The performance of the control subjects was scarcely affected by the change of context, whereas animals with hippocampal lesions began to make a substantial number of errors. Quite contrary results come from an experiment by Penick and Solomon (1991) in which the subjects were rabbits with aspiration lesions of the hippocampus and the training procedure was conditioning of the nictitating membrane response to a tone CS. Acquisition of the CR proceeded identically in lesioned and control subjects; however, whereas control subjects showed a loss of responding when the tone was tested in a different context, lesioned subjects did not.

Given the extensive procedural differences between these two experiments, it is unwise to speculate as to the source of the discrepancy in the results. It may be noted, however, that in both studies the animals experienced the new context for the first time on the test. The animals' test performance might thus very well have been a product of the way in which the trained CR tended to interact with exploratory or other responses that might be evoked by the novel context. That hippocampal damage might influence the course or nature of this interaction is of interest in itself; but the possible occurrence of such an effect makes this experimental design inappropriate as a test of the theories described earlier. To avoid this problem, Good and Honey (1991; see also Honey & Good, 1993) used an experimental design that ensured that the training and test contexts were equated in all relevant respects. Their subjects (rats) were trained with two CSs, one (CS A) being reinforced in context X, the other (CS B) being reinforced in a discriminably different context (Y). Contexts X and Y were thus equally familiar, and the animals had received experience of conditioning procedures in both. The test procedure consisted of presenting each CS in the other context, that is, A in Y and B in X. Control subjects showed a reduction in the strength of the CR on this test, whereas subjects with electrolytic (Good & Honey, 1991) or neurotoxic (Honey & Good, 1993) lesions of the hippocampus did not.

To take further the analysis of the role of the hippocampus in the contextual control of conditioned responding, we decided to adopt the experimental design used by Good and Honey (1991). Our first step, in Experiment 1, was to attempt to replicate, using our own training procedures, their basic demonstration of the effects of electrolytic hippocampal lesions. To our surprise, we found no evidence that such lesions

eliminate context specificity; nor, in Experiment 2 did we find (in contrast to Good & Honey, 1991) that hippocampal lesions hinder the development of an explicit discrimination in which a given CS is reinforced in one context and nonreinforced in another. In an attempt to resolve these discrepancies, we made, in Experiments 3 and 4, a direct comparison of the effects of our training procedures and of those used by Good and Honey (1991). Finally, in Experiment 5, we attempted to confirm the observation made by Good and Honey (1991, Experiment 1) that rats with dorsal hippocampal lesions are retarded in the acquisition of autoshaped responding.

Experiment 1

The subjects in this experiment were two groups of rats: Group C, sham-operated controls, and Group H, animals with electrolytic lesions of the dorsal hippocampus. All received appetitive classical conditioning with two CSs, each CS being trained in its own distinctive context. The context dependency of the CRs established by this training was assessed by a test in which both CSs were presented in each context. This allowed a within-subject comparison, context dependency being demonstrated when animals show more vigorous responding to the CS presented in the same context as was used for training than to the CS presented in a different context from that in which it was trained.

Method

Subjects and surgery. The subjects were 16 male hooded Lister rats with a mean weight, at the start of the experiment, of 355 g (range = 325–375 g). Animals were assigned at random to one of two equal-sized groups: Group H and Group S. For surgery the rat was anesthetized with an intraperitoneal injection of Avertin (made up as 1.25 ml of Avertin concentrate added to 5 ml of absolute alcohol and 62.5 ml of physiological saline) at 10 ml/kg. (Avertin concentrate consists of 100 g of 2,2,2, tri-bromo-ethanol dissolved in 62 ml of tertiary amyl alcohol.) The animal was then placed in a stereotaxic frame, the scalp incised, a section of bone removed, and the dura parted. Bilateral dorsal hippocampal lesions were made by passing a 2.5 mA current from a constant current lesion maker for 25 s through a wire electrode insulated to within 0.5 mm of its tip. The electrode coordinates were 3 mm posterior to bregma, 2.5 mm lateral to the midline, and 3.5 mm ventral to the brain surface. For sham-operated subjects, the procedure was the same except that the electrode was lowered only to the level of the corpus callosum and no current was passed.

After surgery, the rats were allowed a 2-week recovery period during which they were left undisturbed with ad lib access to food. Thereafter they were housed in pairs and subjected to a schedule of food deprivation, being allowed access to food for only 1 hr each day.

Apparatus. Two sets of four Skinner boxes (supplied by Campden Instruments Ltd., Loughborough, UK) were used. Each box had three walls of aluminum, a transparent plastic door as the fourth wall, a grid floor, and a white translucent ceiling. A recessed food tray into which 45-mg pellets could be delivered was set into one of the walls adjacent to the door. The entrance to the tray was covered by a plastic flap, 6 cm high \times 5 cm wide, that was hinged at the top. Pushing the flap inward allowed access to the food tray and operated a microswitch. The flap automatically returned to its resting position when the animal removed its snout from the tray. Each closing of the microswitch was recorded as a single response. The box was housed in a sound-attenuating shell and was brightly lit by a 30-W striplight (rated for 240 V but operated

at 100 V) positioned above the translucent ceiling. A speaker fitted to the rear wall of the box allowed the presentation of auditory stimuli: white noise at an intensity of 80 dB (A) and a 20-Hz train of clicks at the same intensity. The background level in the absence of these stimulus was 65 dB.

The two sets of boxes differed in the following ways. One set was housed in a large experimental room. Each of these boxes was given a characteristic odor by the presence of a small amount of eucalyptus oil poured into the sawdust-filled tray below the grid floor before the start of training sessions on each day. For the other set of boxes the odor was provided by iso-amyl acetate. These boxes were housed in a smaller room in a different part of the laboratory. They were made visually distinctive by the presence of black and white checkered wallpaper on the wall that served as the door and the wall immediately opposite.

Procedure. There were two training sessions each day, one in each context. A given subject always experienced one type of box in its morning session and the other type in its afternoon session; thus, time of day was an additional feature by which the contexts could be distinguished. Previous experiments using this apparatus have successfully demonstrated context specificity of conditioning in intact rats (e.g., Hall & Honey, 1989, 1990). The context experienced in the morning is referred to as context X; that experienced in the afternoon (approximately 4 hr after the morning session) is referred to as context Y. Half the animals in each group experienced a given set of Skinner boxes as context X, and half as context Y. Sessions were 40 min in duration.

On the first 2 days of training, food pellets were delivered on a variable time 60-s schedule, and the subjects learned to retrieve them by pushing open the flap in front of the food tray. Over the next 6 days, the subjects received conditioning with both auditory stimuli, one in each context. For half the rats in each group, the noise was presented in context X and the click in Y; for the remaining rats, this arrangement was reversed. There were six trials per session, each consisting of a 10-s presentation of the stimulus followed immediately by the delivery of a food pellet. The interval between trials was 5.5 min. Operations of the magazine flap were recorded during CS presentations and, separately, during the 10-s stimulus-free period (the pre-CS period) that preceded each trial.

On the next 2 days, all subjects received test sessions organized in the same general way as the training sessions except that trials with both CSs (three trials of each type) occurred in each session. One trial type may thus be labeled S (for same), indicating that the CS was presented on test in the same context as that in which it had been trained during conditioning; on D (for different) trials the CS occurred for the first time in the other context. Half the animals in each group experienced the trial sequence SDDS in context X and the sequence DSSD in context Y; for the other subjects the reverse arrangement was used. No food pellets were presented during test sessions.

After completing this experiment and that described below as Experiment 2, the animals went on to take part in a study of flavor aversion learning (Purves, Bonardi, & Hall, 1995). The hippocampal animals were then killed for histological examination. They were deeply anesthetized with Pentobarbitone sodium and perfused intracardially with physiological saline followed by 10% formol saline. The brains were removed and stored in formol saline for 2 weeks before being embedded in paraffin wax and cut on a microtome in 10 μ m sections. Sections were retained at 150 μ m intervals throughout the lesioned area. They were mounted and stained with cresyl violet.

Results and Discussion

Histology. Figure 1 (reproduced from the report by Purves et al., 1995) presents coronal sections through the rat brain on

which are superimposed reconstructions of the lesion damage for all subjects in Group H. The striped area shows the maximum extent of the lesion and the stippled area the minimum extent. It is evident that all animals sustained dorsal hippocampal damage with minimal damage to underlying structures. A representative selection of brains from the sham-operated group was also sectioned; these exhibited no damage at all.

Behavior. Figure 2 shows the acquisition of conditioned responding by the two groups over the initial phase of reinforced training. The measure used on each trial was the number of operations of the magazine flap occurring during the CS, minus the number of such responses that occurred during the corresponding pre-CS period. These scores were pooled for each individual over all 12 trials (6 in each context) occurring on a given day and a response rate was calculated. The figure shows the acquisition of conditioned responding in both groups; Group H showed more rapid initial acquisition but a somewhat lower asymptotic rate. An analysis of variance (ANOVA) was conducted on the data summarized in the figure, the variables being group and day. There was no significant effect of group ($F < 1$), but a significant effect of day, $F(5, 70) = 15.18, p < .01$, and a significant Group \times Day interaction, $F(5, 70) = 5.14, p < .01$. Analysis of simple effects showed that the groups differed significantly on day 3, $F(1, 48) = 4.36, p < .05$; the difference fell just short of significance on days 5 and 6, $F(1, 48) = 4.06$ and $3.96, ps < .06$ in both cases. Baseline levels of responding were low for all subjects throughout training. Mean rates over all pre-CS periods were 0.98 responses per min (rpm) for Group H and 0.93 rpm for Group C, scores that did not differ significantly ($F < 1$).

The more rapid initial acquisition by Group H may be thought a surprising finding given that hippocampal lesions have usually been found to be without effect on simple classical conditioning, at least when the training parameters are optimal (e.g., Solomon, 1979). Studies using training procedures more directly parallel with the appetitive conditioning procedure used here have, however, produced mixed results. Honey and Good (1993) found no significant difference between hippocampals and controls (although the former group showed a numerically higher rate that persisted throughout training); Good and Honey (1991), by contrast, found hippocampals to respond less readily than controls particularly (as here) toward the end of training. We can conclude only that hippocampal lesions have no consistent effect on appetitive acquisition.

The results of the first test day are presented in Figure 3. (Very little responding occurred on the second test day—the tests were carried out in extinction—and the results of that day will not be considered further.) The scores given are group mean response rates (CS minus pre-CS) pooled over all trials of a given type (S or D) experienced on the test day. It is apparent that Group C tended to respond at a somewhat higher rate than Group H (reflecting the state of affairs at the end of acquisition) and that response rates on S trials were higher than on D trials—evidence for context specificity. There is no suggestion, however, that that difference between S and D trials was diminished in Group H. We conducted an ANOVA on the data shown in Figure 3, with group and stimulus type as variables. There was a significant effect of

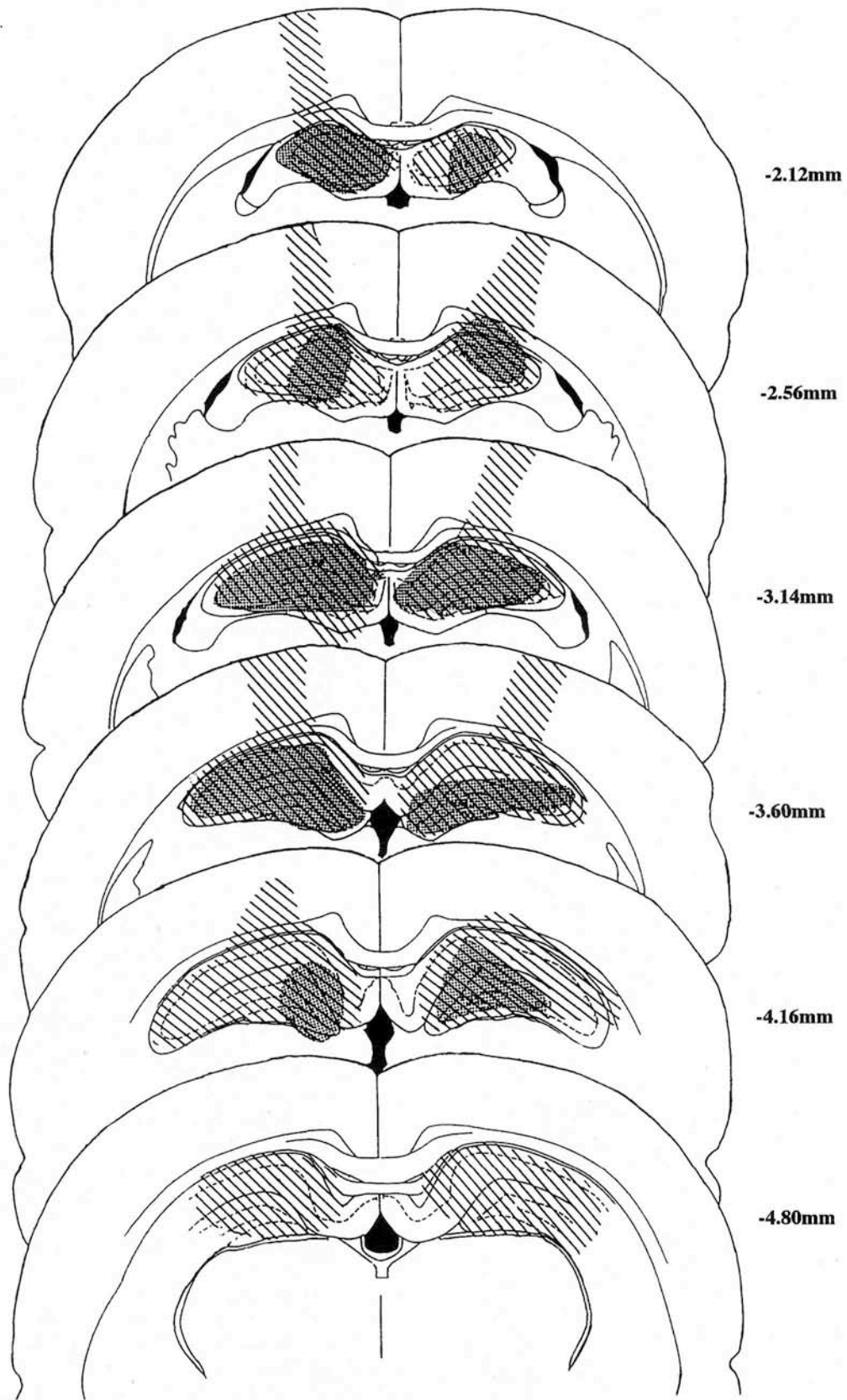


Figure 1. Experiments 1 and 2: Reconstructions of the maximum (striped) and minimum (stippled) extent of damage in the hippocampal lesioned group superimposed on coronal sections derived from the Paxinos and Watson (1982) atlas. Figures denote positions posterior to bregma.

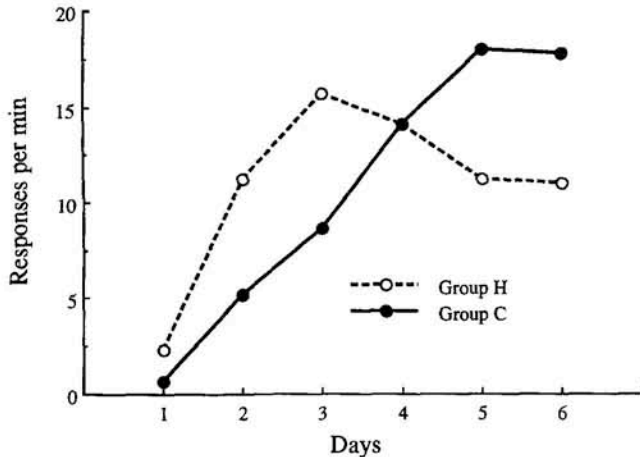


Figure 2. Experiment 1: Acquisition of conditioned responding in subjects with hippocampal lesions (Group H) and sham-operated controls (Group C). The score (response rate in the presence of the conditioned stimulus [CS] minus pre-CS rate) is the group mean pooled over both daily training sessions.

stimulus type, $F(1, 14) = 5.45, p < .05$, but no significant effect of group, $F(1, 14) = 2.96$, and no significant interaction ($F < 1$). There was little responding in the absence of the CSs, and the groups did not differ in this regard. The mean rate during pre-CS periods was 0.18 rpm for Group H and 0.13 for Group C ($F < 1$).

In experiments using procedures very similar to those used here, Good and Honey (1991) and Honey and Good (1993) produced results to suggest that, after hippocampal lesions, a CR acquired in one context will transfer well to another context. Our results, by contrast, show a sensitivity to context in hippocampal subjects that is as large as, if not even larger than, that seen in controls. Accordingly, our main concern in the rest of this article becomes that of attempting to account

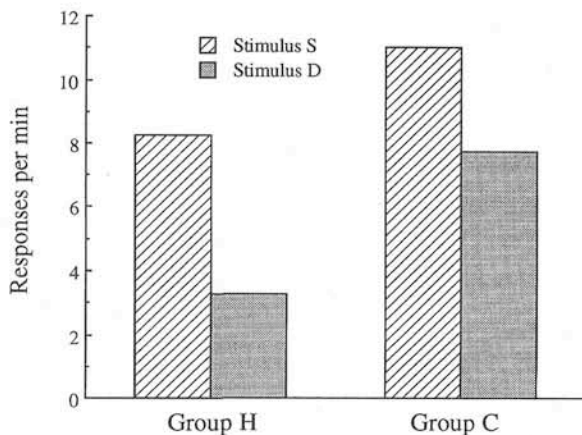


Figure 3. Experiment 1: Test day responding for subjects with hippocampal lesions (Group H) and control subjects (Group C). The score (response rate in the presence of the conditioned stimulus [CS] minus pre-CS rate) is the group mean pooled over all trials of a given type. Stimulus S: stimulus presented in the same context as used for conditioning; Stimulus D: stimulus presented in the different context.

for the discrepancy—a proper evaluation of the theories of hippocampal function described in the introduction will not be possible until the matter has been resolved. As a first step, we turned to another experimental design that was used by Good and Honey (1991) to assess contextual control over conditioned responding, the procedure they refer to as *switching*.

Experiment 2

In switching, subjects are trained explicitly on a contextual conditional discrimination in which the value of a cue varies according to the context in which it occurs. In the version studied here, and that used by Good and Honey (1991), two CSs were used, one reinforced in context X and nonreinforced in context Y, the other reinforced in Y and nonreinforced in X. Successful performance occurs when the subject comes to show a CR to the first CS only when it occurs in X and to the other CS only when it occurs in Y. Such performance has been taken to reflect a process of conditional learning whereby a given set of contextual cues acquires the ability to facilitate the retrieval of one association rather than another (e.g., Bouton & Swartzentruber, 1986; Good & Honey, 1991), although an interpretation in terms of the configural stimuli formed by different CS–context combinations is also possible (e.g., Preston, Dickinson, & Mackintosh, 1986). In either case, it would be of interest to determine, given the results of Experiment 1, whether hippocampal damage detracts from the ability of rats to learn the switching task.

Method

The subjects were the same rats that had served in Experiment 1. Training on this experiment was begun immediately after Experiment 1 had been completed. The apparatus, stimuli, and general training procedures were the same as in the previous experiment, with the following exceptions. In each of the two daily training sessions (one in each context), two trial types occurred. There were three reinforced presentations of the auditory cue that had been presented in that context during training in Experiment 1 (the S stimulus of the test phase of that experiment); there were also three nonreinforced presentations of the other stimulus (the D stimulus of Experiment 1). The sequence of trials was random apart from the constraint that no more than two trials of a given type could occur in succession. There were 12 days of training.

Results and Discussion

For each trial, we made a record of the magazine operations that occurred during the auditory cue minus any responding that occurred in the immediately preceding pre-CS period. Responses generated by both daily sessions for the two trial types were pooled, producing a single measure of responding to the two reinforced stimuli and a single measure for the two nonreinforced stimuli. Group means for these scores, reduced to 2-day blocks, are presented in Figure 4. The scores for cues occurring in the context in which they were reinforced are labeled S+; those for the cues when they occurred in the context in which they were nonreinforced are labeled S–.

With the reintroduction of reinforcement, response rates, which were low at the end of the test stage of the previous experiment, rapidly returned to their original higher levels.

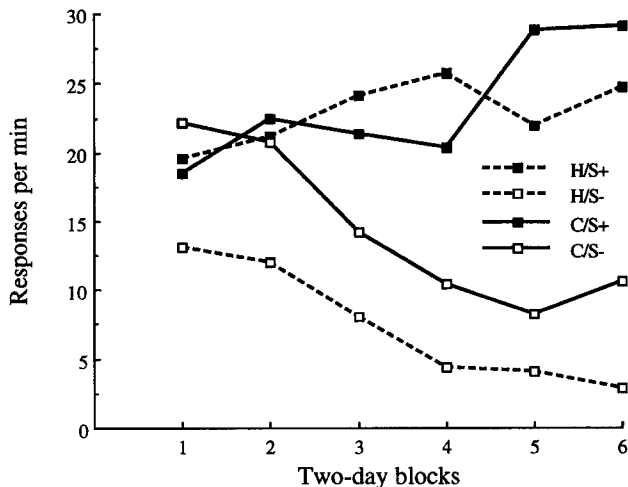


Figure 4. Experiment 2: Performance of subjects with hippocampal lesions (Group H) and sham-operated controls (Group C) on a switching task in which two stimuli were presented, each reinforced in one context and nonreinforced in another. The S+ score pools responding over all reinforced trials in a given training block; S- pools responding over nonreinforced trials.

Initially, the auditory cues evoked responding whichever context they were presented in (both cues had been reinforced during the training phase of Experiment 1), but thereafter the discrimination was acquired. Responding was maintained in the context in which reinforcement continued to occur, but with continued training, the ability of the cues to evoke responding was lost in the contexts in which they were nonreinforced. Subjects in Group H showed a lower rate to the S- cues than did subjects in Group C throughout the entire course of training, but apart from this there were no substantial between-group differences. There was certainly no sign that hippocampal subjects were unable to learn this discrimination.

An ANOVA conducted on the data summarized in Figure 4 confirmed this description of the results. There was no significant main effect of group, $F(1, 14) = 2.07$, or of block, $F(5, 70) = 1.84$, but the effect of stimulus type (S+ or S-) was reliable, $F(1, 14) = 140.55$, $p < .01$, as was the interaction between block and stimulus type, $F(5, 70) = 17.17$, $p < .01$. The interaction of group with block was not significant ($F < 1$), but there was a significant interaction between group and stimulus type, $F(1, 14) = 8.47$, $p < .05$, and a near-significant three-way interaction, $F(5, 70) = 2.19$, $p = .06$. The Group \times Stimulus interaction was explored by way of a simple-effects analysis that revealed no significant difference between the groups in their S+ scores ($F < 1$) but a significant difference in the S- scores, $F(1, 18) = 5.58$, $p < .05$. Response rates in the absence of the stimuli were low but were somewhat higher in Group C than in Group H. Mean scores over all pre-CS periods were 1.58 rpm for Group C and 0.85 rpm for Group H. The difference fell just short of significance, $F(1, 14) = 4.00$, $.05 < p < .10$.

In this experiment, as in Experiment 1, we have been able to find no evidence to suggest that hippocampal damage produces a deficiency in the ability of animals to learn about and

use contextual cues. The results presented in Figure 4 show clearly that hippocampal rats can perform perfectly well on a discrimination task in which the value of a given CS depends on the nature of the context in which that CS occurs. Indeed, in some respects their performance on this task was superior to that of control subjects in that they showed lower response rates to the S- stimuli while maintaining equivalent high levels of responding to the S+ stimuli. Although this result is consistent with the suggestion that Group H found it easier to learn the required discrimination, it should be acknowledged that another, less interesting, interpretation is possible. The pre-CS rates recorded during this experiment indicate that the general level of responsiveness might be somewhat lower in Group H than in Group C. If so, then a lower rate to the S- is to be expected. The absence of a difference between the groups in their responding to S+ can be readily explained as a "ceiling effect," making the high rates generated by reinforced training insensitive to a difference in general responsiveness that is apparent at lower rates.

This last observation may help provide an explanation for the apparent discrepancy between the results reported here and those from the similar study reported by Good and Honey (1991, Experiment 2), which have been taken to demonstrate a deficit in hippocampal animals in the acquisition of a conditional contextual discrimination. In this latter experiment, as in ours, the subjects received initial reinforced training with the cues subsequently to be used as S+ and S- in the switching task. But the training parameters used by Good and Honey (1991) differed substantially from those used in our experiment and were such (there were relatively few trials and the CSs were of long duration) as to generate only a low rate of response. Consequently, the difference between the lesioned and control subjects was evident only in performance to the S+; rates to S- started low in both groups and stayed low throughout discrimination training. Thus, these results too can be readily explained if we assume that, in these experiments, hippocampal animals tend to respond less vigorously than controls (an assumption well supported by the data for initial acquisition that Good and Honey, 1991, reported). Given the low starting rates, the difference would only be visible in S+ rates as they developed during training; a floor effect would obscure any difference in responding to S-.

The only safe conclusion to offer at this stage, therefore, is that experiments using the switching procedure have so far provided no compelling evidence that subjects with hippocampal lesions differ from controls in their ability to make use of contextual cues. This conclusion is in accord with the results of our Experiment 1 in which a simple test of the context specificity of conditioned responding yielded no difference between hippocampals and controls. What remains to be resolved, then, is the source of the discrepancy between Experiment 1 and the finding by Good and Honey (1991) that context specificity appears to be abolished in hippocampals. We take up this issue in Experiment 3.

Before turning to Experiment 3, a further point requires comment. Experiments 1 and 2 generated what may be regarded as null results in that no substantial between-groups differences were found. It might be argued, therefore, that the outcome of these experiments was a consequence of some

inadequacy in our surgical technique that produced a lesion that was, for some reason, ineffective. Examination of the anatomical results (see Figure 1) makes this an implausible suggestion, and there is also evidence to contradict it from behavioral work. After the experiments described above had been completed, the animals served as subjects in a study of latent inhibition using the flavor aversion learning paradigm. This experiment (Purves et al., 1995) revealed a substantial difference in performance between Group H and Group C, a result in accord with some previous work on latent inhibition using this procedure (Reilly, Harley, & Revusky, 1993).

Experiment 3

The discrepancy between the results of our Experiment 1 and those reported by Good and Honey (1991) must derive from some procedural difference between the two experiments. Both used electrolytic lesioning techniques, and the pattern of damage produced was closely similar in the two sets of subjects. As has already been noted, the experiments differed in the parameters used for conditioning: Good and Honey (1991) gave three trials per session with a CS duration of 30 s, whereas in Experiment 1 there were six 10-s trials per session. These differences produced different terminal response rates in the two experiments, but it is difficult to see why such a response rate difference should generate different patterns of context specificity on the test session. The events used as CSs differed between the experiments—clicker and light offset in the earlier study, clicker and noise in Experiment 1—but again it is not obvious that this difference should be critical.

Perhaps the most striking difference between the two experiments lay in the test procedure. Experiment 1 made use of a within-subject design in which both CSs were presented during the test session. Context specificity was thus evidenced when an animal responded more vigorously on S trials than on D trials. Good and Honey (1991) used a test procedure in which the subjects received just the D trials; a difference in test performance between the groups was taken to reflect a difference between them in sensitivity to the change of context.

The possibility that this difference in test procedures might be responsible for the discrepant results will be discussed further later. Our first step, however, was to attempt to reproduce as closely as we could the training and test procedures used by Good and Honey (1991) to confirm that we could replicate their finding.

Method

The subjects were an additional 16 rats, from the same stock and maintained in the same way as those used in Experiments 1 and 2. Eight rats (Group H) underwent surgery intended to produce lesions of the dorsal hippocampus; 8 rats (Group C) constituted a sham-operated control group. Procedures were identical to those described for Experiment 1.

After they had recovered from the surgery, the rats received appetitive conditioning in the contexts described for the previous experiments. The general procedure was the same as that described for Experiment 1 with the exception of the following modifications,

made in order to accord with the procedure used by Good and Honey (1991). The events used as CSs were the presentation of an 80-dB 20 Hz train of clicks and the offset of the houselight. The duration of the CS was 30 s, and there were three trials in each 40-min session, the first after 10 min and at 10-min intervals thereafter.

On the next 2 days, the subjects received test sessions. The procedure was the same as that used in training except that no food pellets were delivered, and each CS (dark or clicker) was presented not in its own context but in that used for acquisition for the other CS. Thus, all subjects received only D (different) test trials.

After completing a program of behavioral testing, the animals were killed for histological examination. The procedures used were the same as those described for Experiment 1.

Results and Discussion

Histology. Figure 5 presents reconstructions of the lesion damage for all subjects in Group H, with striped areas showing the maximum extent of the lesion and stippled areas the minimum extent. As before, all Group H animals sustained damage to the dorsal hippocampus.

Behavior. The acquisition of conditioned responding over the 8 days of initial conditioning is shown in Figure 6. The measure used is the rate of response in the CS minus the rate recorded for the pre-CS periods, averaged over all six trials (three with the clicker and three with light offset as the stimulus) on a given day. It is evident that for both groups the CS acquired the power to elicit responding and that the groups did not differ in this respect. An ANOVA conducted on the data summarized in Figure 6 revealed a significant effect of days, $F(7, 77) = 2.18$, but no difference between the groups and no significant interaction between the factors ($F < 1$ in both cases). But although the CR was acquired, the conditioning parameters used here were clearly much less effective than those used in Experiment 1 in generating a high rate of response. Indeed, 2 rats (1 in each group) failed to show any conditioned responding at all. They were excluded from further consideration, and the data shown in the figure are accordingly based on the scores of the 7 rats in each group that developed the CR. The rate of response in the absence of the CS remained low throughout training; the mean rates averaged over all pre-CS periods were 0.80 rpm for Group H and 0.86 rpm for Group C, scores that did not differ significantly ($F < 1$).

There was again little responding on the second test day, and Figure 7 shows the results of just the first test. (These results are thus directly comparable with those presented by Good and Honey, 1991, who conducted just a single test.) The response rates shown on the left of the figure are based on CS minus pre-CS scores pooled over all six trials on the first test day. Although the difference was small (the groups did not differ significantly on this measure, $F < 1$), it was immediately apparent that the pattern was the same as that reported by Good and Honey (1991); that is, Group H responded somewhat more than Group C and thus appeared to be less sensitive to the change of context.

Good and Honey (1991) presented their results not as simple response rates but as an elevation ratio of the following form: Rate in the presence of the CS divided by rate of responding in the remainder of the session. Accordingly, we

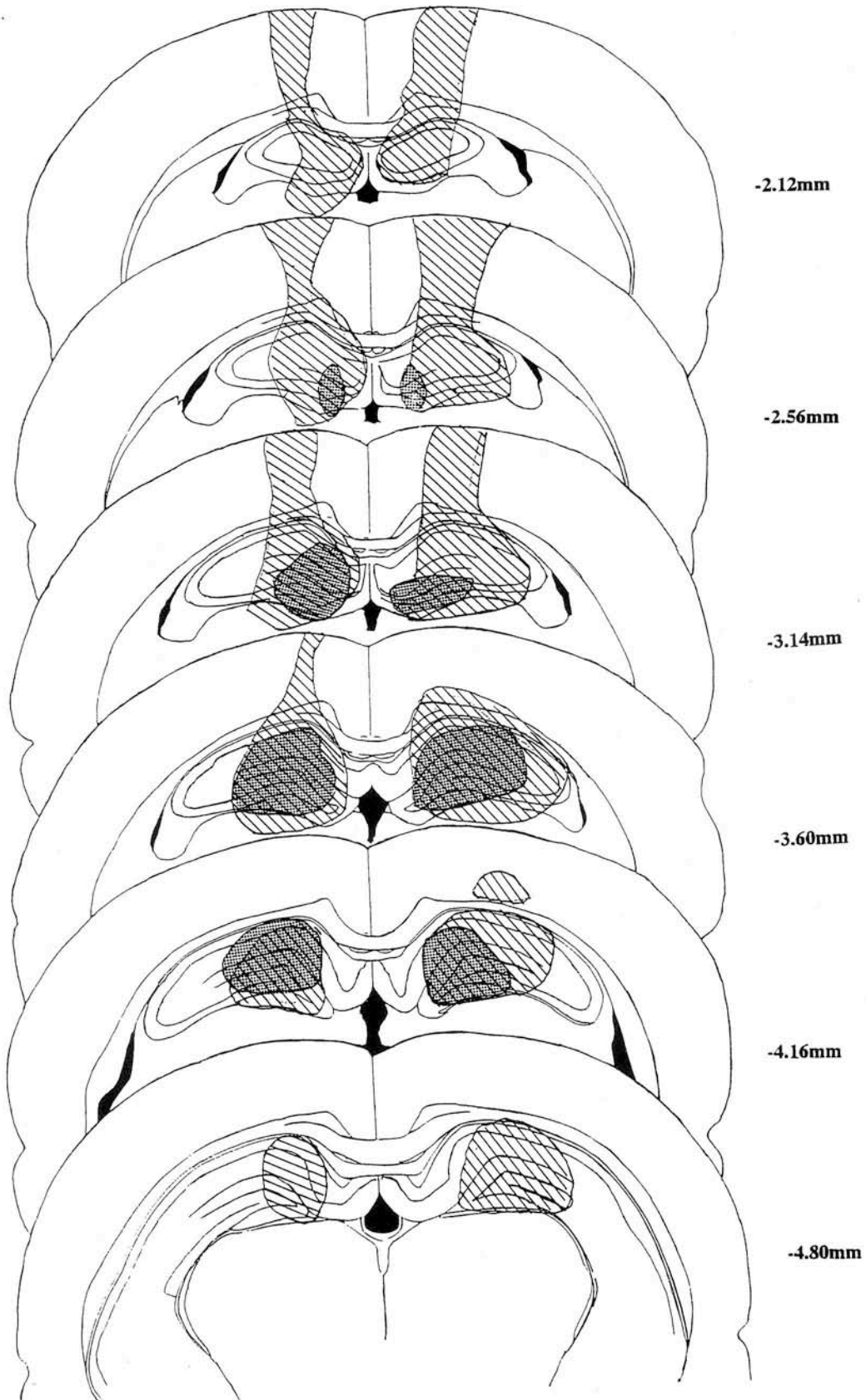


Figure 5. Experiments 3, 4, and 5: Reconstructions of the maximum (striped) and minimum (stippled) extent of damage in the hippocampal lesioned group superimposed on coronal sections derived from the Paxinos and Watson (1982) atlas. Figures denote positions posterior to bregma.

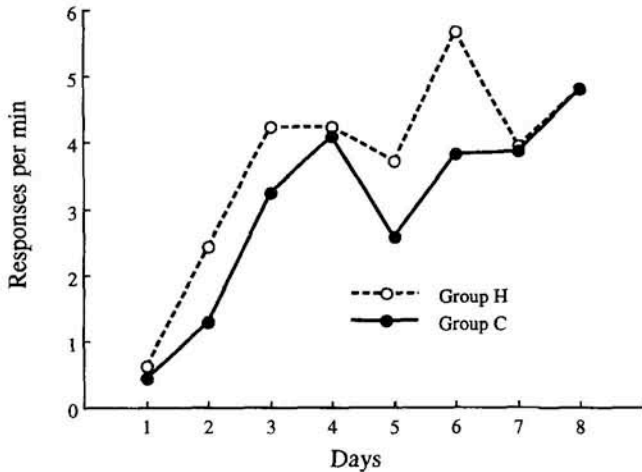


Figure 6. Experiment 3: Acquisition of conditioned responding in subjects with hippocampal lesions (Group H) and sham-operated controls (Group C). The score (response rate in the presence of the conditioned stimulus [CS] minus pre-CS rate) is the group mean pooled over both daily training sessions.

computed this ratio score for the performance of each subject on the first test day, and group mean ratios are shown on the right of Figure 7. Group H again showed more vigorous responding than Group C. Although again the difference is not statistically significant, $F(1, 12) = 2.24$, it may be noted that the numerical values presented in Figure 7 are closely similar to those presented by Good and Honey (1991), who did not report a direct statistical comparison of the test performance of the two groups. What they did compare was performance on the test with that shown on the last 2-day block of acquisition. They demonstrated that the ratio score tended to increase from acquisition to the test in the hippocampal subjects but to decline in the control subjects. The same was true for the

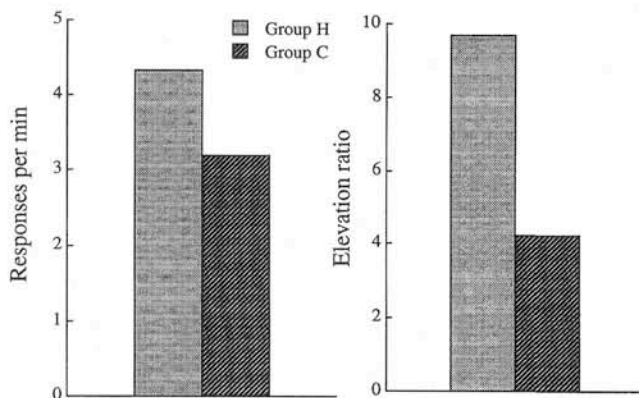


Figure 7. Experiment 3: Test day responding (group means) by subjects with hippocampal lesions (Group H) and controls (Group C) to conditioned stimuli (CSs) presented in a different context from that of original conditioning. The left-hand panel shows response rate in the presence of the CS (minus pre-CS rate). The right-hand panel shows a ratio score (rate in the presence of the CS divided by rate in the remainder of the session).

equivalent scores in the present experiment, the mean ratio score for Group H increasing from 5.31 to 9.67, the mean ratio score for Group C declining from 5.28 to 4.24. An ANOVA carried out on these scores with group and phase (acquisition or test) as variables yielded no statistically reliable effects; for the effect of group, $F(1, 12) = 2.26$; for phase, $F < 1$; and for the interaction, $F(1, 12) = 1.31$. It should be noted, however, that the equivalent analysis reported by Good and Honey (1991) also revealed no significant effects.

The results of this experiment match, almost exactly, those reported by Good and Honey (1991). Although in neither experiment were the effects statistically reliable, both found that, with a test procedure consisting solely of the presentation of nonreinforced D trials, hippocampal subjects maintained a somewhat higher level of responding than did control subjects. This indication of an apparent loss of sensitivity to context change in hippocampals contrasts with the results of the present Experiment 1. We have suggested that the critical difference between the experiments lies in the details of the test procedure used. This matter is taken up in the next experiment.

Experiment 4

Experiments 1 and 3 differed not only in the test arrangements they used but also in the nature and duration of the stimuli used as CSs and in the scheduling of stimulus presentations. To demonstrate that the source of the discrepancy in outcome lies in the details of the test procedure, we had to rule out any possible contribution from the other factors. In this experiment, therefore, the subjects experienced training parameters identical to those used in Experiment 3 but were given the within-subject test procedure in which all animals received both S and D trials in the test session. Would the pattern of context sensitivity observed in Experiment 3 (and by Good & Honey, 1991) be evident in these conditions?

Method

The subjects were the same 14 rats that completed Experiment 3. After completing the test phase of that experiment, they were returned to reinforced training; the conditioning parameters were the same as those used in the acquisition phase of Experiment 3. There were 4 days of such training. This was followed by a single test day organized in the same way as that described for Experiment 1 (i.e., consisting of a six-trial session in each context, three trials with the stimulus originally trained in that context and three with the stimulus trained in the other context). As the CS duration in this experiment was 30 s, the intertrial interval was reduced to 290 s, maintaining the session length at 40 min.

Results and Discussion

With the resumption of reinforced training, rates of responding rapidly returned to the levels achieved at the end of the acquisition phase of Experiment 3. The mean score (rate in the presence of the CS minus rate in the pre-CS periods) pooled over all trials on the last day of conditioning was 5.57 rpm for Group H and 4.81 rpm for Group C. These rates did not differ significantly ($F < 1$). The mean pre-CS rate on this day was rather higher (at 1.76 rpm) in Group H than in Group C (0.62

rpm), but the difference was not statistically significant, $F(1, 12) = 2.89$.

The results of the test day are shown in Figure 8. Rates of response are somewhat lower (as is to be expected given the training parameters used in this study), but the general pattern of results is the same as that seen in Experiment 1. There is evidence of sensitivity to the change of context in that rates are lower to stimulus D than to stimulus S, and the difference is present in both Group C and Group H. An ANOVA conducted on the data summarized in the figure showed that there was a significant effect of trial type, $F(1, 12) = 8.84, p < .01$, but no effect of group and no significant interaction between these two variables ($F_s < 1$). The baseline (pre-CS) response rates on the test day were low and similar in the two groups: 0.45 rpm for Group H and 0.31 rpm for Group C ($F < 1$).

To facilitate comparison with Experiment 3, we computed ratio scores for the two types of test trial exactly as in the previous experiment. For D trials, the mean ratio scores were 3.71 for Group H and 2.05 for Group C, a pattern like that observed in Experiment 3. But the higher rate of responding on D trials in Group H cannot be safely attributed to a reduced sensitivity to context change as, in the present experiment, exactly the same pattern was observed on S trials when ratios were computed; these scores were 8.45 for Group H and 6.99 for Group C. Indeed, the difference in the mean ratio scores for S and D trials was virtually identical in the two groups (4.74 for Group H and 4.94 for Group C).

In this experiment, rats with hippocampal lesions showed much the same degree of sensitivity to a change of context as was shown by control subjects. In Experiment 3, which used the same rats, stimuli, and general training procedures, hippocampal subjects showed an apparent lack of sensitivity to contextual change. The source of the discrepancy appears to lie in the use of a within-subjects comparison (both S and D trials presented on the test) in the present experiment as opposed to the test procedure of Experiment 3 in which

subjects received just D trials on the test. Looking at the ratio scores for the D trials shows a higher rate of responding in lesioned subjects in both experiments. But this appears to reflect not so much a reduced sensitivity to context as a tendency for Group H to respond generally more on these tests than does Group C—at least when ratio scores are used, the present experiment shows Group H to respond more than Group C on S trials as well as on D trials.

We conclude that measures of context specificity (such as that used in Experiment 3) that look only at responding on D trials should be treated with caution as they may be influenced by more general lesion-induced changes in responding. This reservation must be extended to a further report of the attenuation of contextual control in hippocampal rats offered by Honey and Good (1993, Experiment 1). This experiment investigated context specificity in rats with ibotenic acid lesions of the hippocampus using a test procedure almost identical to that used in the present Experiment 4. It showed that control subjects responded less on D trials than on S trials, whereas hippocampal subjects maintained responding on D trials at much the same level as was shown on S trials. The procedure used by Honey and Good differed, however, from that used in the present experiment in that in their experiment S trials were reinforced during the test phase. It is possible that the presence of reinforcement might act to reduce the sensitivity of the test, by obscuring a difference between hippocampal and control subjects in their response to the S stimulus. Without an accurate evaluation of the animals' tendency to respond on S trials, the difference seen on D trials cannot be interpreted with any confidence.

Experiment 5

Although in Experiment 3 the performance of our subjects closely paralleled that reported by Good and Honey (1991) for their Experiment 2, it remains the case that neither in this experiment nor in Experiment 4 were we able to obtain any statistically reliable difference between hippocampal and control subjects. This raises the possibility that the surgical procedures used for these rats failed, for some reason, to produce damage enough to influence performance on this, or indeed any other, supposedly hippocampally dependent task. The impact of the null results of Experiments 3 and 4 would be greatly heightened if it could be demonstrated that the lesioned rats used in these experiments show a clear deficit on some other behavioral task.

In the first experiment of their 1991 report, Good and Honey investigated the performance of their hippocampally lesioned subjects on an autoshaping task. The rats received classical conditioning trials in which the delivery of a food pellet was signaled by the insertion of a lever into the conditioning chamber for a period of 5 s. Although lever presses were not required, the rats developed the tendency to respond on the lever; this may be taken to be an instance of sign tracking (Hearst & Jenkins, 1974), in which animals come to approach and make contact with a signal for food. Hippocampal subjects acquired this response substantially less readily than did control subjects. In the present experiment, we attempted to obtain a similar deficit in our hippocampal subjects, using our

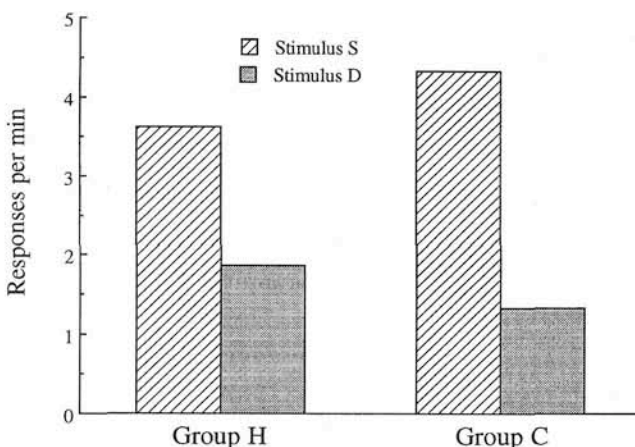


Figure 8. Experiment 4: Test day responding for subjects with hippocampal lesions (Group H) and control subjects (Group C). The score (response rate in the presence of the conditioned stimulus [CS] minus pre-CS rate) is the group mean pooled over all trials of a given type. Stimulus S: stimulus presented in the same context as used for conditioning; Stimulus D: stimulus presented in the different context.

own version of a sign-tracking task. In this, the signal was the onset of a small light located inside the food tray itself, presented immediately before food delivery. For the rat to make contact with this light, it would need to push its snout into the food tray aperture, and these responses could be readily recorded as movements of the flap that guarded the aperture.

Method

The subjects were the 16 rats that served in Experiment 3. The apparatus was the same as that used in the previous experiments, with the following modifications. No special cues were added to distinguish different contexts; the overhead striplight was not used, and dim background illumination was provided by a 3-W jewel light (rated for 24 V but operated at 15 V) located high on the rear wall of the chamber; a 3-W jewel light, operated at full intensity and located inside the food tray just above the aperture through which pellets were delivered, was used as the CS.

Training in this experiment commenced 1 week after the completion of Experiment 4. It began with a preliminary 40-min session of magazine training in which food pellets were delivered according to a variable time 60-s schedule. There followed 12 sessions of conditioning. Each was of 50 min duration and contained eight 10-s presentations of the tray light, each of which was followed by the delivery of a food pellet. The first trial occurred after 250 s, and the same period intervened between the offset of one trial and the start of the 10-s pre-CS period at the beginning of the next. Movements of the tray flap were recorded during the CS and separately during the pre-CS periods.

Results and Discussion

As a consequence, presumably, of the initial session of magazine training, the rats showed substantial background levels of responding on the first session of conditioning. Pooling all pre-CS periods on this session produced mean rates of 2.25 rpm for Group H and 4.04 rpm for Group C. This responding soon declined, so that by Session 3 the equivalent rates were 1.60 rpm for Group H and 1.31 rpm for Group C. These low levels were maintained throughout the rest of training. An ANOVA comparing pre-CS rates for the two groups over all 12 sessions of conditioning showed there to be a significant effect of session, $F(11, 154) = 2.85, p < .01$. There was, however, no significant difference between the groups, $F(1, 14) = 2.37$, and no significant interaction between the variables ($F < 1$).

The acquisition of conditioned responding was assessed by computing, for each session, each animal's rate of response in the presence of the CS minus its rate in the absence of the CS (i.e., during the pre-CS periods). Group mean scores are presented in Figure 9. This shows that for both groups the rate of response increased over the course of training, but that the level of responding shown by Group H was consistently less than that shown by Group C. An ANOVA with group and session as the variables showed that there was a significant effect of group, $F(1, 14) = 6.13, p < .05$, and a significant effect of sessions, $F(11, 154) = 12.54, p < .01$. The interaction between these variables was not significant ($F < 1$).

This result replicates that reported by Good and Honey (1991, Experiment 1) in that both show a marked deficit in

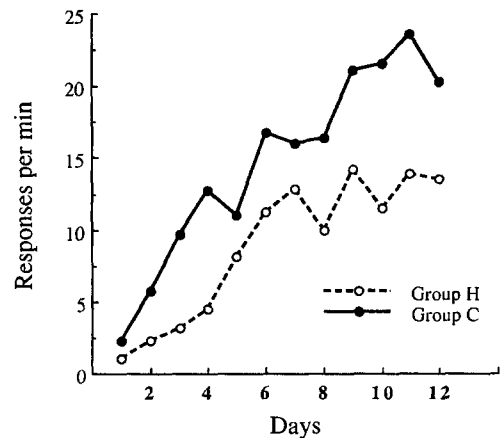


Figure 9. Experiment 5: Mean rate of response (rate in conditioned stimulus [CS] minus rate in pre-CS) for subjects with hippocampal lesions (Group H) and control subjects (Group C) trained with the presentation of a light in the food tray as a signal for food.

hippocampal subjects trained with an autoshaping or sign-tracking procedure. The source of this deficit is unclear. As Good and Honey themselves pointed out, it has commonly been found that hippocampal damage is without effect on the acquisition of a simple classical CR. Indeed, Experiment 3 of the present article shows, for the same subjects as were used in this experiment, that hippocampal subjects will acquire the CR of approaching the food tray on the presentation of a diffuse auditory or visual cue as readily as control subjects. This observation prompts the speculation that hippocampal rats behave as normal subjects when goal tracking is recorded (i.e., when the CR reflects only the tendency to approach the site of reinforcer delivery). The deficit will become apparent, however, when sign tracking is involved (i.e., when the CR includes behavior produced by a tendency to respond to the CS itself).

It will require further experimental work to assess the viability of this hypothesis, and we discuss it no further here. For our present purposes, the source of the effect obtained in this experiment can be neglected. The important outcome is that we have demonstrated a behavioral deficit in our rat subjects with hippocampal lesions and this implies that the null results obtained in previous experiments using these subjects are not to be explained in terms of an inadequacy in our lesioning technique. What is more, the task on which we have obtained a deficit in hippocampal subjects is formally equivalent to that used by Good and Honey (1991) in their Experiment 1, thus strengthening our confidence in the comparability of the two sets of experiments.

General Discussion

The experiments reported here were intended to determine how damage to the hippocampus influences the extent to which contextual cues gain control over the conditioned responding established to CSs reinforced in the presence of those cues. Contrary to previous reports, we found little evidence that the hippocampus plays an important role in contextual control. In Experiments 1 and 4, rats received

reinforced training with a different CS in each of two contexts before a test session in which both CSs were presented in each context. Responding was less when the CS was presented in the context in which initial training had not been given, but this was true both for hippocampal and for control animals. In Experiment 2, rats were trained on an explicit discrimination in which presentations of a given CS were reinforced when they occurred in one context and nonreinforced when they occurred in the other. Again, hippocampal subjects learned this task, in which adequate performance appears to imply conditional control by contextual cues, just as readily as did control subjects. Only in Experiment 3 was there any sign of a reduction in sensitivity to contextual cues in hippocampal subjects, and in this experiment the difference between operated and control subjects fell well short of statistical reliability.

In general, we may conclude that there is little in our results to support the contention that the processes responsible for context specificity in conditioned responding require an intact hippocampus. This is not to say that learning about contextual cues will always be normal. There have been many experiments designed to assess the vigor of the CR evoked by contextual cues themselves after animals have received presentations of a US (sometimes preceded by a discrete CS, sometimes not) in that context. The range of results has been wide. Some (e.g., Good & Honey, 1991, Experiment 3) found no effect of the lesion; others have revealed circumstances in which conditioning to the context will be attenuated (e.g., Phillips & LeDoux, 1992; Selden, Everitt, Jarrard, & Robbins, 1991) or even enhanced (Winocur, Rawlins, & Gray, 1987) by the lesion. It is clear that hippocampal lesions can modify the ability of the context to act as a CS. But our experiments were not concerned with responses directly conditioned to the context; rather, they were designed to investigate the role played by contextual cues in modulating the effectiveness of CSs occurring in their presence. In this process we find no evidence for hippocampal involvement.

Although in our experiments we have made use of cues provided by the experimental context, the issue of central interest does not require this. The procedures known as *occasion setting* and *negative patterning* constitute examples of the same basic effect—modulation of the responding governed by a given CS by the presence of some other event—using not contexts but a discrete cue as the modulating event. Studies of the effect of hippocampal damage on these tasks are thus directly relevant to our present concern. The pattern of results they show matches that emerging in experiments on contextual conditional control. Early experiments (e.g., Ross, Orr, Holland, & Berger, 1984; Rudy & Sutherland, 1989) appeared to demonstrate a selective disruption of occasion setting and of patterning discriminations by hippocampal damage. But later experiments with certain procedural refinements have failed to confirm the earlier findings (e.g., Davidson, McKernan, & Jarrard, 1993; Gallagher & Holland, 1992). The reasons for these discrepancies are not clear, but their existence is enough to justify the conclusion, prompted by the present experiments, that the claim that the hippocampus is specifically involved in the conditional control of simple associative learning should be treated with caution.

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