Acquired Equivalence and Distinctiveness of Cues: II. Neural Manipulations and Their Implications

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Neural manipulations were used to examine the mechanisms that underlie the acquired equivalence and distinctiveness of cues in rats. Control rats and those with excitotoxic lesions of either the hippocampus (HPC) or entorhinal cortex (EC) acquired the following conditional discrimination: In Contexts A and B, Stimulus X → food and Stimulus Y → no food, and in Contexts C and D, Y → food and X → no food. Rats then received many food pellets in A but not in C. After this treatment, control rats showed more magazine activity in B than in D—an acquired equivalence–distinctiveness effect. This effect was also evident in HPC rats but not in EC rats. These results indicate that changes in stimulus distinctiveness are dissociable from the process of conditional learning.

Pavlov (1927, p. 113) observed that following conditioning with one stimulus, other stimuli “spontaneously” acquired the ability to elicit conditioned responding. The standard account for this observation, for stimulus generalization, is that the conditioned stimulus activates a set of representational elements (see Estes, 1950; Wagner, 1981), subsets of which are also activated by other stimuli (see Mackintosh, 1974, pp. 486–487; Rescorla, 1976). According to this account, stimulus generalization is simply a function of the degree to which stimuli activate shared or common elements. More recently, however, the suggestion has been made anew that this account of stimulus generalization is incomplete. Gluck and Myers (1993) have proposed that two representational biases modulate stimulus generalization. The first, redundancy compression, “has the effect of combining or clustering correlated (and hence redundant) stimulus features” and tends to increase generalization between two stimuli that have reliably (a) predicted the same event or (b) co-occurred (see p. 493). The second, complementary bias, is termed predictive differentiation and increases “the resources allocated to represent stimulus features that have predictive value” and tends to reduce the extent of generalization between two stimuli that have previously either reliably (a) predicted different events or (b) occurred separately (see pp. 492–493). These hypothetical biases share more than a passing resemblance to ideas expressed elsewhere. For example, the suggestion that the associative history of stimuli might influence generalization between them was foreshadowed by both Miller and Dollard (1941, pp. 62–65; see also Hull, 1939; Miller, 1948), who proposed that stimuli might become less distinct as a result of predicting the same event or eliciting the same response, and by James (1890, p. 511; see also Lawrence, 1949), who argued that the memories of stimuli might become more distinct as a consequence of coming to activate different associates. Support for these proposals can be derived from demonstrations of the acquired equivalence and distinctiveness of cues (see, e.g., Honey & Hall, 1989a, 1991; see also Honey, 1990; for a review, see Hall, 1991). Moreover, the suggestion that stimulus co-occurrence and separation influence subsequent generalization between stimuli receives support from demonstrations of sensory preconditioning (Brogden, 1939; see also Bateson & Chantrey, 1972; Honey, Bateson, & Horn, 1994; Rescorla & Cunningham, 1978) and perceptual learning (Gibson & Walk, 1956; Honey & Hall, 1989b; see also McLaren, Kaye, & Mackintosh, 1989).

Gluck and Myers (1993) made two additional suggestions that are of general interest. The first was that the biases for stimulus compression and predictive differentiation could be implemented within a connectionist or neural network in which a layer of hidden units mediates between sensory input units and behavioral output units. Within such networks, the representational biases identified previously influence which sensory units become linked to which hidden units: Stimuli that predict the same outcome or co-occur become linked to the same hidden unit (or units), and stimuli that predict different outcomes or those that do not co-occur become linked to different hidden units (see also Goldstone, 1998; Honey, 2000; Maki, 1993; Maki & Abunawass, 1991). This form of account has received direct support from a recent series of studies from our laboratory (Honey & Ward-Robinson, 2001; Honey & Watt, 1998, 1999; Ward-Robinson & Honey, 2000), culminating in those results reported in the preceding article by Honey and Ward-Robinson (2002; see also Delamater, 1998; Delamater & Joseph, 2000; Goldstone, 1998). The second suggestion made by Gluck and Myers (1993) concerned the neural mechanisms that mediate the processes that underlie redundancy compression and predictive differentiation: Gluck and Myers suggested that the hippocampal region (i.e., the hippocampus proper: dentate gyrus and CA1–3 fields, and adjacent cortical areas; e.g., the entorhinal cortex and subiculum) mediates these processes. To the best of our...
knowledge, this second suggestion has been the subject of little
direct experimental scrutiny, and the evidence that is available is
inconclusive. Thus, Ward-Robinson et al. (2001), using a proce-
dure developed by Rescorla and Cunningham (1978), showed that
excitotoxic lesions of the rat hippocampus have no effect on
sensory preconditioning (see also Honey & Good, 2000; Honey,
Watt, & Good, 1998; but see Port, Beggs, & Patterson, 1987; Port
& Patterson, 1984). It is possible, however, that either (a) sensory
preconditioning does not solely reflect the operation of the sorts of
representational processes on which Gluck and Myers’s (1993)
account is based, or (b) different structures within the hippocampal
region (e.g., the entorhinal cortex) might contribute to different
aspects of redundancy compression and predictive differentiation
(see Myers, Gluck, & Granger, 1995).

With the preceding considerations borne in mind, we have
conducted two experiments with rats in which we examined the
influence of excitotoxic lesions of the rat hippocampus (Experi-
ment 1) and entorhinal cortex (Experiment 2) on the acquired
equivalence and distinctiveness of cues effect reported by Ward-
Robinson and Honey (2000). This effect, as we have noted, pro-
vides support for the kind of connectionist analysis offered by
Gluck and Myers (1993) for the influence of experience with
stimuli on the extent of generalization between them (for further
discussion, see Honey & Ward-Robinson, 2002). As we shall see,
the results of these experiments have implications for both the
neural and conceptual mechanisms that underlie learning in gen-
eral and experience-based changes in stimulus distinctiveness in
particular.

Experiments

The experimental design used in Experiments 1 and 2 is iden-
tical to that described by Ward-Robinson and Honey (2000, Ex-
periment 2) and is summarized in Table 1. In the first stage, all rats
received a contextual conditional discrimination involving four
contexts (e.g., A = checked, B = warm, C = dotted, and D =
cool) and two auditory stimuli (e.g., X = tone and Y = clicks).
When rats were placed in either A or B, presentations of X were
followed by food and those of Y were not, and when they were
placed in C or D, presentations of Y were followed by food and
those of X were not. In the second stage, the revaluation stage, rats
were placed in Context A, where they received a large quantity of
food, and in Context C, where they instead received no food.
Ward-Robinson and Honey reported that this revaluation pro-
cedure resulted in a greater tendency for rats to approach the site of

food delivery in Context B than in Context D, and we anticipated
that our control rats would show this effect. The question of
interest was whether this effect would be apparent in rats that had
either received excitotoxic lesions of the hippocampus (Experi-
ment 1) or entorhinal cortex (Experiment 2) before training began.

Method

Subjects and Surgery

Sixteen naive adult male hooded Lister rats (supplied by Harlan Olac,
England) served in Experiments 1 and 2. There were 8 sham-operated
control rats (mean ad lib weight: 379 g; range: 340–430 g) in each
experiment. To simplify presentation of the results, to increase statistical
power, and to provide a common baseline against which to assess the
effects of our lesions, we combined the scores from these two sets of
control rats to form a single group, the control group. As will be shown,
separate analyses revealed that the patterns of statistical effects (during the
stages in which these animals received identical treatment) remained the
same when the results were not combined in this way. The 8 remaining rats in
Experiment 1 (mean ad lib weight: 364 g; range: 340–390 g) received
bilateral excitotoxic lesions of the hippocampus (HPC group), and those in
Experiment 2 (mean ad lib weight: 361 g; range: 330–400 g) received
bilateral excitotoxic lesions of the entorhinal cortex (EC group).
The surgical procedures were based on those used by Coutureau, Galani,
Gosselin, Majchrzak, and D. Scala (1999). Rats were first anaesthetised
using a halothane–oxygen mix and were then placed in a Kopf stereotactic
frame (Kopf Instruments, Tujunga, CA). The bone above the region to be
lesioned was removed, and ibotenic acid (Biosearch Technologies, Inc.,
San Rafael, CA; dissolved in phosphate buffered saline [pH 7.4] to provide
a solution with a concentration of 63 mM) was injected into the brain
through a glass pipette glued onto the end of a 5–μl Hamilton syringe held
with a Kopf microinjector (Model 5000). The stereotactic coordinates and
the amounts of ibotenic acid injected at each site are shown in Table 2.
Injections were made manually at a rate of 0.10 μl/min, and the pipette was
left in place for 1 min after the injection to allow diffusion of the solution
into the tissue. The rats in the control group were given a similar surgical
procedure, but the dura was simply perforated using a standard needle, and
no injection was given. A minimum period of 14 days recovery was
allowed before the rats were reduced to 80% of their ad lib weights and
behavioral testing began. The colony room used to house the rats was
illuminated between the hours of 8 a.m. and 8 p.m. Training and testing
started at approximately 9 a.m. The rats were housed in pairs with water
freely available to them in their home cages.

Apparatus

Four operant chambers (24.5 cm wide × 23 cm deep × 21 cm high;
supplied by Campden Instruments Ltd., Loughborough, England), housed
in sound-attenuating boxes and arranged in a 2 × 2 array, were used. These
chambers were housed in a brightly lit experimental room, and the doors to
the sound-attenuating boxes were left open throughout the experiment.
Each chamber had three aluminum walls, an aluminum ceiling, and a
Perspex door and received local illumination from a single 3-W lightbulb
located in the center of the ceiling. The right-hand pair of chambers
(Contexts A and C) had standard floors constructed from stainless steel
rods. The walls and ceilings of these chambers were lined with transparent
Perspex. White wallpaper with black dots was fixed both behind these
linings and on the outside of the door in the top chamber, and black and
white check wallpaper was fixed behind these linings and on the outside of
the door in the bottom chamber (for further details, see Honey & Watt,
1999). The left-hand pair of boxes (Contexts B and D) had aluminum floors
that could either be made warm (35 °C) or cool (10 °C) by inserting heated
or cooled Thermos picnic blocks in a bracket below the floor (for further

| Table 1 |
|---|---|---|
| Conditional discrimination | Revaluation | Test |
| AX → food & AY → no food | A → no food | B |
| BX → food & BY → no food | C → food | D |
| CX → no food & CY → food | DX → no food & DY → food |

Note. A, B, C, and D denote contexts (A and C: checked or dotted; B and
D: warm or cool); X and Y denote auditory stimuli (tone or clicker); food
and no food indicate trials on which the outcomes of the trials were food
and no food.
Details, see Ward-Robinson & Honey, 2000). The auditory stimuli (X and Y) were a 2-kHz tone and a 10-Hz train of clicks. These stimuli were presented at an intensity of approximately 75 dB (A) from a speaker located above the ceiling of the chamber. Food pellets (45 mg; P. J. Noyes, Lancaster, NH) delivered into a recessed food well (6 cm high and 5 cm wide) in the left-hand wall of each chamber, could be retrieved by moving a transparent Perspex flap. A hinge attached this flap to the top of the well, and a 2-mm movement of the bottom edge of the flap was recorded automatically as a single food-well entry or response.

**Conditional Discrimination Training**

On the first day, rats received two training sessions in which they were trained to retrieve food pellets from a food well in undecorated operant chambers with standard grid floors. On the first session of training, the flaps in front of the food wells were fixed in a raised position allowing the rats ready access to the food pellets; on the second session of training, these flaps were lowered, and rats had to move the flaps to gain access to the food pellets. During both sessions, 20 food pellets were delivered on a variable time (VT) 60-s schedule. On each of the next 16 days of training, rats received one session in each of the four contexts, A and C (check and dots) and B and D (warm and cool). The transfer of rats from one context to another within a day took on average 1 min. When rats were placed in Contexts A and B, presentations of X were followed by two food pellets and presentations of Y were nonevoked, whereas when rats were placed in Contexts C and D, presentations of X were nonevoked and presentations of Y were followed by two food pellets. The interval between the termination of the reinforced stimuli and the delivery of food pellets was 1 s (see Ward-Robinson & Honey, 2000). The auditory stimuli were both thermal contexts (warm or cool) that served as B and D were fully counterbalanced. Similarly, the identities of the auditory stimuli (tone or clicker) that served as X or Y were fully counterbalanced. On 8 days (Days 1, 4, 5, 8, 9, 12, 13, and 16) the top chamber was warm and the bottom chamber was cool, and on the remaining days this arrangement was reversed. The order in which rats received the four context sessions (A, B, C, and D) changed from one day to the next. In each of the four 4-day blocks of training, each context was presented in the four possible positions within a day (first, second, third, and fourth), and placement in any one of the contexts (e.g., A) was equally likely to be immediately followed by or preceded by placement in any of the other three contexts (B, C, or D).

**Revaluation of A and C and Test Trials With B and D**

Prior to the appetitive revaluation procedure, we established that rats’ behavior in the presence of B and D did not differ (see following Results section). We did this by recording the rates of magazine entries during the 10-s periods that immediately preceded presentations of X and Y during the final day of conditional training. On the 2 days of revaluation training, rats continued to receive training sessions in the visual contexts, A and C. Each session was approximately 20 min. On each day, when rats were placed in Context A, there were 20 pellets in the food hopper, and they received a further 20 pellets that were delivered on a VT 60-s schedule. No food pellets were delivered in Context C during the revaluation procedure. For half of the rats, the order in which rats received A and C was A, C on Day 1 and C. A on Day 2, and for the remaining rats, these arrangements were reversed. The interval between the sessions within a day was approximately 20 min. At the end of each of the sessions in which rats were placed in Context A, inspection of the food well revealed that the rats had consumed all of the food pellets. On the next day, rats received test trials on which they were placed in Contexts B and D. On this day, the top chamber was warm and the bottom chamber was cool. For half of the rats, the order in which rats received the two contexts was B, D, and for the remaining rats it was D, B. The interval between the tests was approximately 60 min. Each test trial started with a 20-s acclimatization period (see Ward-Robinson & Honey, 2000) followed by four consecutive 40-s periods in which we recorded the rate of food-well entries.

**Behavioral Measures**

To assess acquisition of the conditional discrimination, we calculated a discrimination ratio (responses per minute, rpm, during reinforced stimulus presentations divided by the combined rate of responding during reinforced and nonevoked stimuli) for each 4-day block of conditional training. A score of 0.50 indicates that responding is equivalent during the reinforced and nonevoked stimuli, whereas scores of 1.0 and 0 indicate that responding is restricted to presentations of the reinforced stimulus and nonevoked stimulus, respectively. To assess performance during the critical test trials, we used the rates of responding during Contexts B and D to calculate discrimination ratios of the form rate of responding during B divided by the combined rates of responding during B and D; a score above 0.50 indicates that responding was greater during B than during D.

**Reference Memory in the Water Maze**

After the test described previously, rats in Experiment 1 were returned to ad lib food for approximately 2 weeks and then received training in a spatial, reference memory task in a water maze using an apparatus and a training protocol identical to those described in Good and Honey (1997). This spatial memory task should allow us to assess whether the hippocampal lesions were behaviorally effective in a conventional test of hippocampal function. Briefly, on each of the 6 days of training, rats received four trials with an intertrial interval of 30 s. On each trial, the rats were released from a randomly selected point around the perimeter of the pool and were allowed to swim until they located a hidden platform or until 2 min had elapsed at which point the rat was placed on the platform for 30 s. For half of the rats in each group, the platform was placed in the northwest quadrant of the maze, and for the remainder it was placed in the southeast quadrant.

### Table 2

<table>
<thead>
<tr>
<th>Lesion</th>
<th>AP</th>
<th>ML</th>
<th>DV</th>
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<td>±5.0</td>
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<td></td>
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<td>±4.2</td>
<td>−3.9</td>
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<td></td>
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<td>±4.5</td>
<td>−6.5</td>
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<td>−4.7</td>
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<td>−3.0</td>
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<tr>
<td></td>
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<td>±1.0</td>
<td>−3.0</td>
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<tr>
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</tr>
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<td></td>
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<td>±4.7</td>
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<td></td>
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**Note.** Coordinates, in millimeters from bregma, for lesions in hippocampus (HPC) and entorhinal cortex (EC) groups. AP = anteroposterior; ML = mediolateral; DV = dorsoventral. Amounts of ibotenic acid (μl) injected at each site are as follows: * 0.075, † 0.08, ‡ 0.09, § 0.10, || 0.05.
Histological Analysis

After behavioral testing, rats were overdosed with sodium pentobarbital (Euthatal) and then transcardially perfused, first with 0.9% saline and then with 10.0% formal-saline. The brains were then removed, postfixed for 2 hr and subsequently transferred to phosphate-buffered (0.1 M) 30.0% sucrose solution in which they remained for 36 hr. Forty-micrometer-thick horizontal sections of the brains were made using a freezing microtome at room temperature for 24 hr before being stained with cresyl violet. Microscopic inspection was used to determine the location and extent of the lesions using the atlas of Paxinos and Watson (1998).

Results

Histological Analysis

Figure 1 represents a summary of the extent of cell loss in the rats with acceptable hippocampal lesions (left-hand panel) and entorhinal lesions (right-hand panel). Two rats, 1 in the HPC group (Experiment 1) and 1 in the EC group (Experiment 2), were excluded for the following reasons. The rat in the HPC group had substantial sparing of cells throughout the dorsal and ventral extent of the hippocampus, and the rat in the EC group died 2 weeks after surgery. Of the remaining rats in group HPC, all had sustained substantial cell loss within the dorsal hippocampus (extending from –3.6 mm to –5.6 mm from bregma) and cell loss that was more variable in the ventral aspect of the hippocampus (extending from –6.6 mm to –7.6 mm from bregma). Indeed, there was substantial sparing of cells in the ventral CA3 region and ventral aspects of the dentate gyrus in all rats. However, there was no detectable damage to cells in the subicular complex or to the entorhinal cortex at any of their dorsal or ventral levels. Of the remaining rats in the EC group, all lesions were restricted to the entorhinal cortex and were small. Typically, the lesion extended from –4.6 to –7.6 mm ventral to bregma and was confined to the medial part of the entorhinal cortex in all rats. There was no detectable damage to the hippocampus or subiculum. There was no overlap between the areas of damage in the HPC and EC groups.

Behavioral Analysis

Conditional discrimination training. Figure 2 presents the mean discrimination ratios for four blocks of training (that each comprised 4 days) for rats in the control group (left-hand panel; pooled across Experiments 1 and 2) and for those in the HPC (center panel) and EC (right-hand panel) groups. It is clear that over the course of conditional training, rats in all groups acquired the conditional discriminations involving visual contexts and thermal contexts and that the scores for the visual discrimination were somewhat higher than the scores for the thermal discrimination (see also Ward-Robinson & Honey, 2000). An analysis of variance (ANOVA) conducted on the scores from rats in the control and HPC groups (Experiment 1) revealed no effect of group ($F < 1$); main effects of block, $F(3, 63) = 21.40, p < .01$, and discrimination (visual vs. thermal), $F(1, 21) = 9.81, p < .01$; but no interactions between the factors, largest $F(3, 63) = 1.19, p > .1$. An ANOVA conducted on the scores from the control and EC groups (Experiment 2) revealed no effect of group ($F < 1$); main effects of block, $F(3, 63) = 24.54, p < .01$, and discrimination (visual vs. thermal), $F(1, 21) = 11.04, p < .01$; but no interactions between the factors, largest $F(3, 63) = 1.75, p > .22$. This pattern of results was consistent with that reported by Good, DeHoz, and Morris (1998).

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This pattern of results was also evident when the same analysis was conducted using only the 15 rats from Experiment 1: There was an effect of block and discrimination, minimum $F(1, 13) = 6.28, p < .05$, but no other effects or interactions, largest $F(1, 13) = 1.78, p > .20$. By the final block of training, the mean rate of responding during the reinforced stimuli was greater than

On Day 7, the hidden platform was removed and rats were placed in the pool for 1 min. The percentage of time rats spent in each quadrant of the maze was recorded.

Figure 1. Reconstructions of the hippocampal lesions (Experiment 1; left-hand panel) and entorhinal cortex lesions (Experiment 2; right-hand panel) on a series of horizontal sections at various distances in millimeters posterior to bregma (increasing in 1-mm steps from –3.6 mm for the top section to –7.6 mm for the bottom section). The maximum and minimum extents of the lesions are shown in black and oblique lines, respectively.
that during nonreinforced stimuli in the rats with EC lesions (visual: 14.57 rpm and 8.81 rpm; thermal: 13.09 rpm and 8.61 rpm). An ANOVA revealed that there was an effect of stimulus, $F(1, 21) = 97.19, p < .01$, no effects of group or discrimination, and no interactions between these factors, largest $F(1, 21) = 1.07, p > .61$. On the final day of training, the level of responding in the test contexts, B and D, in control rats (B: 4.28 rpm and D: 4.30 rpm) were similar to those in the HPC group (B: 3.56 and D: 3.22) and the EC group (B: 7.72 rpm and D: 5.92 rpm). The fact that the mean levels of responding were higher in the EC group reflected the behavior of a single rat. When this rat was excluded, the mean for Context B was 3.60 rpm and the mean for Context D was 3.63 rpm. ANOVAs comparing rats in the control group with those in the HPC or EC group revealed no effects of context or group and no interaction between these factors, largest $F(1, 21) = 1.27, p > .27$.

**Test trials with B and D.** The mean discrimination ratios for the tests with B and D (pooled across the four successive 40-s periods) were as follows: 0.59 (control), 0.61 (HPC), and 0.47 (EC). ANOVAs revealed that the control rats’ scores did not differ from those with hippocampal lesions ($F < 1$) but differed significantly from rats with EC lesions, $F(1, 21) = 4.33, p < .05$; these analyses yield the same pattern of effects when conducted with the 15 rats from Experiment 1, $F(1, 13) = 2.26, p > .15$, and Experiment 2, $F(1, 13) = 8.28, p < .05$. In fact, the effect of interest was most marked in the first 40-s period of the test (after acclimatization; see upper panel of Figure 3), and one-sample t tests revealed that both the control and HPC group scores differed from .50, $t(15) = 2.21, p < .05$, and $t(6) = 2.62, p < .05$, respectively, but that the scores from the EC group did not differ from .50, $t(6) = .07, p > .94$. Separate ANOVAs conducted on (a) the absolute rates of responding during D trials used to calculate the overall mean discrimination ratios ($M$: control = 5.31 rpm, HPC = 6.42 rpm, and EC = 6.63 rpm) and (b) those used to calculate the ratios for the first 40-s period of the test ($M$: control = 2.56 rpm, HPC = 2.57 rpm, EC = 3.28 rpm) showed that the control group’s scores did not differ from those of the HPC or EC groups ($F$s < 1).

**Reference memory in the water maze.** After 6 days of training, in which the rats from Experiment 1 were required to locate a hidden platform in a water maze, they received a 1-min test in which the platform was removed and the rats’ tendency to swim in the training quadrant was recorded: Control rats spent a greater percentage of time swimming in the training quadrant (46.26%) than rats with hippocampal lesions (29.00%), $F(1, 13) = 8.87, p < .01$. 

Figure 2. Mean discrimination ratios ($\pm$SEM) for the contextual conditional discrimination involving visual contexts (A and C) and thermal contexts (B and D) in control rats (left-hand panel), those with lesions of the hippocampus (HPC; center panel), and those with lesions of the entorhinal cortex (EC; right-hand panel).

Figure 3. Top panel: Mean discrimination ratios (rate of responding in Context B divided by the combined rate of responding in B and D; $\pm$SEM) for the initial periods of the critical test trials in the two thermal contexts, B and D, in control rats and those with lesions of either the hippocampus (HPC) or entorhinal cortex (EC; see text for details). Bottom panel: Correlations between the discrimination ratios for the thermal and visual contextual conditional discriminations during the final block of training in the control, HPC, and EC groups.
Discussion and Further Results

Control rats readily acquired a conditional discrimination in which they were placed in Contexts A and B. Presentations of X were followed by food and those of Y were not, whereas when they were placed in Contexts C and D, presentations of Y were paired with food and those of X were not. Lesions to the entorhinal cortex, like those of the hippocampus, had no effect on the readiness with which this discrimination was acquired. After the acquisition of this discrimination, rats received a large quantity of food in Context A but not in Context C. This revaluation procedure resulted in both control rats and those with lesions of the hippocampus showing higher levels of conditioned responding in Context B than in Context D. This acquired equivalence—distinctiveness effect was not evident in rats with selective lesions of the entorhinal cortex. One possible account for this latter observation is that rats with lesions of the entorhinal cortex failed to learn that food pellets were delivered in Context A but not in Context C. This suggestion seems rather implausible given the fact that the same rats acquired a complex conditional discrimination involving Contexts A and C. Nevertheless, to assess whether the revaluation procedure had been effective, we gave all rats from Experiment 2 a further test in which A and C were presented on the day following the test with B and D. This test was conducted in the same manner as that involving B and D. Although there was no significant difference in the levels of responding in A and C during the first 80 s of the test (control means: A = 4.40 rpm and C = 5.54 rpm; EC means: A = 5.46 rpm and 4.71 rpm), there was a difference during the second 80 s of the test (control means: A = 6.37 rpm and C = 3.75 rpm; EC means: A = 7.71 rpm and 3.85 rpm). ANOVAs with group and context as factors revealed no effect of either factor and no interaction between these factors during the first 80 s (Fs < 1), whereas there was an effect of context, F(1, 13) = 5.49, p < .05, and no effect of group and no interaction between these factors during the second 80 s (Fs < 1). Hence the failure of rats in the EC group to exhibit an acquired equivalence—distinctiveness does not reflect the fact that they did not learn the simple contextual discrimination between Contexts A and C and must instead lay elsewhere.

Additional evidence provides powerful converging support for the contention that it is the processes that underlie the acquired equivalence and distinctiveness of cues that were disrupted in the EC group. This evidence involves the behavior of the rats during conditional discrimination training. Briefly, if it is supposed that it is the processes of acquired equivalence and distinctiveness that are disrupted in the EC group, then this should be apparent as a difference in the way in which the visual and thermal conditional discriminations are solved. For example, if the visual and thermal contexts (e.g., A and B) are becoming equivalent during conditional discrimination training, then this would provide one basis for predicting that the discrimination ratios for the visual and thermal discriminations should be highly correlated. Thus performance in these two discriminations should be correlated in rats for whom this process of equivalence is in operation (i.e., rats in the control and HPC groups) but not in those for whom it is not (i.e., rats in the EC group). The lower panel of Figure 3 shows the Pearson product moment correlations between the visual and thermal discrimination ratios on the final block of conditional discrimination training: on this block, like on the other blocks of training, all three groups were performing at a similar level (see Figure 2). Nevertheless, the correlation between the visual and thermal discrimination was significant in the control (p < .01) and HPC (p < .01) groups but not in the EC group (p > .23).

The intriguing general implication of the previous results is that, contrary to first appearances (see Figure 2), the conditional discriminations are being acquired in different ways in the control and HPC groups than in the EC group. The issue of why these different ways of solving a conditional discrimination should result in relatively similar levels of performance is neither unexpected nor is it especially theoretically perplexing. For example, if the way in which the control and HPC groups are solving the conditional discrimination involves a greater modification to representations of the stimuli than the way in which the EC group is solving the discrimination (see following General Discussion section), then this might counteract the benefit that would otherwise accrue from acquired equivalence and distinctiveness effects in the control and HPC groups but not in the EC group.

General Discussion

Gluck and Myers (1993) suggested that two representational biases, redundancy compression and predictive differentiation, modulate the extent of stimulus generalization between stimuli: redundancy compression increasing generalization between stimuli that reliably predict the same event or co-occur and predictive differentiation reducing generalization between stimuli that reliably predict different events or occur separately. The existence of such representational biases, together with their suggested implementation in three-layer connectionist architecture, receives support from the results of a recent series of studies, including those reported by Ward-Robinson and Honey (2000, Experiment 2) and Honey and Ward-Robinson (2002). These studies demonstrate an acquired equivalence and distinctiveness of cues effect that is beyond the scope of standard associative accounts and instead provides support for a connectionist approach of the kind recommended by Gluck and Myers (1993: for an extended discussion, see Honey & Ward-Robinson, 2002). Gluck and Myers also suggested that the hippocampal region mediates the two representational biases that they described. Experiments 1 and 2 are a direct attempt to investigate these ideas using the procedures developed by Ward-Robinson and Honey (2000, Experiment 2).

The results of Experiment 1 show that excitotoxic lesions of the hippocampus—lesions that are sufficient to disrupt spatial learning in the water maze—do not influence the acquired equivalence and distinctiveness of cues effect, and those of Experiment 2 indicate that excitotoxic lesions of the entorhinal cortex abolish this effect. Although these results are broadly consistent with Gluck and Myers’s (1993) original account, they are inconsistent with a development of this account in which aspects of stimulus compression and predictive differentiation are partitioned between the hippocampal formation (dentate gyrus, CA1-3 fields, and subiculum) and the entorhinal cortex (see Myers et al., 1995). Myers et al. (1995) suggested that the hippocampal formation mediates both
predictive differentiation (reducing generalization between stimuli that have either predicted different events or occurred separately) and one aspect of redundancy compression (increasing generalization between stimuli that have predicted the same event). In contrast, they supposed that the entorhinal cortex mediated the second aspect of redundancy compression, increased generalization based on stimulus co-occurrence. The results of Experiment 1 and 2 are clearly inconsistent with the views expressed by Myers et al. (1995)—it is lesions to the entorhinal cortex, not the hippocampal formation, that influence the acquired equivalence and distinctiveness of cues. We shall now consider in turn two aspects of the results of Experiment 2 that are of more general interest: The first is the dissociation between conditional learning (spared in rats with lesions of the entorhinal cortex) and the acquired equivalence and distinctiveness of cues effect (abolished in the same rats), and the second is the possible theoretical loci at which these lesions are having their effect.

One general way to cast the dissociation described previously is to suggest that rats with lesions of the entorhinal cortex were sensitive to the contingencies between the various individual compounds and the outcomes with which they were paired (i.e., AX → food, BX → food, CX → no food, DX → no food, AX → no food, BY → no food, CY → food, DY → food) but did not additionally represent the fact that the components (e.g., A and B) of some similar compounds (e.g., AX and BX) were followed by the same outcome (e.g., food). The capacity for conditional learning can be explained by conventional unique-cue (Brandon, Vogel, & Wagner, 2000; Rescorla, 1972; Saavedra, 1975; Wagner & Rescorla, 1972) and configural (e.g., Pearce, 1994) accounts in which individual representations of each stimulus compound (e.g., AX and BX) are linked to the outcome with which they are paired (food). However, these accounts of conditional learning do not provide any basis on which the components (e.g., A and B) of two similar compounds (e.g., AX and BX) should come to address the same representations as a consequence of being paired with the same outcome; that is, they provide no explanation for recent demonstrations of the acquired equivalence and distinctiveness of cues (Honey & Ward-Robinson, 2001, 2002; Honey & Watt, 1998, 1999; Ward-Robinson & Honey, 2000). As we have argued, demonstrations of this type require a more complex analysis (see Honey & Ward-Robinson, 2002) within which the propagation of activity between (a) sensory units (a, b, x, y, c, and d), activated by the pattern of sensory input on a trial (e.g., AX, BX, and so on) and (b) output units, activated by the outcome of a trial (e.g., food or no food), is mediated by a set of hidden units (e.g., p and q; see Honey & Ward-Robinson, 2002, Figure 1). This analysis is a reasonable starting point for an appreciation of the dissociation between conditional learning and changes in stimulus distinctiveness that was observed in our rats with lesions of the entorhinal cortex.

According to such connectionist analyses, initially the links from a given sensory unit to each hidden unit are often assumed to be (relatively) weak and to have random starting weights or strengths. One interesting consequence of this assumption is that the pattern of sensory units (e.g., ax) activated by a stimulus (AX) will be more likely to activate one hidden unit than another. Another consequence is that, even from the outset of training, compounds that are similar to one another (e.g., AX and BX) will tend to activate the same hidden units, and dissimilar compounds (e.g., AX and DY) will be more likely to activate different hidden units. Thus, even without the capacity to modify the links between the sensory and hidden units, the initial pattern of links between these two types of units could provide a basis for conditional discrimination learning, and this might also provide a basis for the ability of rats with lesions of the entorhinal cortex to acquire the conditional discrimination summarized in Table 1 (Schmajuk & Blair, 1993; see also Pearce, 1994; Rudy & Sutherland, 1995). However, these links could not provide a basis for the acquired equivalence and distinctiveness of cues effect that was evident in our control rats and in those with hippocampal lesions (Experiments 1 and 2; see also Honey & Ward-Robinson, 2001, 2002; Honey & Watt, 1998, 1999; Ward-Robinson & Honey, 2000). To explain these results, one needs to suppose that when similar patterns of sensory units are activated (e.g., ax and bx) and are followed by the same outcome (e.g., food), their components (e.g., a and b) become increasingly likely to be linked to the same hidden unit (e.g., p), and when similar patterns (e.g., ax and dx) are followed by different outcomes (food and no food, respectively), their components (e.g., a and d) become increasingly likely to be linked to different units (p and q, respectively). Honey and Ward-Robinson (2002) have summarized how these changes might be brought about, and it is sufficient to note here that changes in the sensory-to-hidden layer links involve a number of elements (e.g., the links, the characteristics of the hidden units, and the possible [error-correcting] feedback processes from the output units to the hidden units). Lesions to the entorhinal cortex might disrupt any one of these elements of the process of tuning the sensory-to-hidden layer links and thereby abolish the acquired equivalence and distinctiveness of cues. However, the following observation is worth highlighting: We now have some evidence that indicates that feedback processes, operating between the output units and the hidden units, play a critical role in shaping the pattern of sensory-to-hidden layer links that underlie the acquired equivalence and distinctiveness of cues (Honey & Ward-Robinson, 2002, Experiment 2; see also Honey & Watt, 1998, Experiment 2). One distinct possibility then is that lesions of the entorhinal cortex disrupt the modification of sensory-to-hidden layer links by disrupting feedback from the output units to the hidden layer units (see also Schmajuk & Blair, 1993). In any event, and in more general terms, the analysis presented in this paragraph eschews the need for parallel or interactive systems (cf. Gluck & Myers, 1993) responsible for conditional learning, on the one hand, and changes in stimulus distinctiveness, on the other hand.

Before concluding this discussion, it is worth considering other evidence that is consistent with the more general suggestion that lesions of the entorhinal cortex disrupt experience-based changes in stimulus distinctiveness—evidence that was recently reported by Oswald et al. (2001). In the first stage of this study, control rats and those with excitotoxic lesions of the entorhinal cortex received a complex conditional discrimination in which each trial involved components from three dimensions (auditory, visual, and tactile). Stimuli from two of the dimensions were relevant to the solution of the discrimination, and those from the remaining dimension were irrelevant to the solution of the discrimination. For example, presentations of a tone in a chamber with checked walls were followed by food, and those of a clicker were followed by no food, irrespective of whether the chamber’s floor was bumpy or smooth (i.e., tone–checked–bumpy → food, tone–checked–smooth → food,
clicker–checked–bumpy → no food, clicker–checked–smooth → no food); similarly, presentations of a tone in a chamber with dotted walls were followed by no food, and those of a clicker were followed by food irrespective of whether the chamber’s floor was bumpy or smooth (i.e., tone–dotted–bumpy → no food, tone–dotted–smooth → no food, clicker–dotted–bumpy → food, clicker–dotted–smooth → food). That is, the auditory and visual stimuli were relevant, and the tactile stimuli were irrelevant. Control rats and those with lesions of the entorhinal cortex learned this conditional discrimination equally readily (see also Experiment 2).

In the second stage of the study, the rats received another conditional discrimination involving novel stimuli from the same three dimensions. For half of the rats in each group, the stimuli from the same dimensions (e.g., auditory and visual) remained relevant, and the irrelevant dimension (e.g., tactile) remained irrelevant; that is, these rats received an intradimensional shift (IDS). For the remaining rats, stimuli from the irrelevant dimension (e.g., auditory), whereas stimuli from the second formerly relevant dimension (e.g., visual) became irrelevant; that is, these rats received an extradimensional shift (EDS).

The control rats acquired the discrimination involving an IDS more readily than that involving an EDS; however, rats with lesions of the entorhinal cortex acquired the IDS and EDS with equal readiness.

The pattern of results described by Oswald et al. (2001), like those from Experiment 2, indicate that conditional learning proceeds in the absence of the entorhinal cortex but that the acquired changes in stimulus distinctiveness (Lawrence, 1949) that underlie the IDS–EDS effect do not. Oswald et al. (2001, pp. 846–848) presented an account for their results based on the connectionist approach to the acquired equivalence and distinctiveness of cues that was outlined previously and is described in more detail in our companion article (Honey & Ward-Robinson, 2002). Briefly, Oswald et al. (2001) suggested that lesions of entorhinal cortex (a) disrupt the ability to tune or modify the links between the sensory units activated by stimuli and a layer of hidden units to reflect the relevance of those stimuli to the conditional discrimination, but (b) leave a residual ability to acquire the conditional discriminations based on the kind of “snapshot” configural learning process that is captured by, for example, Pearce’s (1994) model. In this case, separate snapshots would represent each of the three-element compounds (e.g., tone–checked–bumpy → food, tone–checked–smooth → food). As we have already noted, models of the kind developed in Pearce (1994) provide no analysis for acquired equivalence–distinctiveness effects (see Honey & Ward-Robinson, 2002); but as we have also already indicated, the dissociation between conditional learning and changes in stimulus distinctiveness need not mean that there are separate or parallel interactive systems that underlie these two processes: It is possible to develop a single connectionist system that can provide an analysis for the effects of our behavioral and neural manipulations.

To conclude, the results of this study begin to reveal the neural mechanisms involved in the representational biases that Gluck and Myers (1993) referred to as stimulus compression and predictive differentiation. There is now an increasing body of evidence that is consistent with the operation of these biases, and Honey and Ward-Robinson (2002) have provided direct support for a connectionist analysis that implements these biases (see also Gluck & Myers, 1993; Goldstone, 1998; Honey, 2000; Maki, 1993; Maki & Abunawass, 1991). The results of Experiments 1 and 2 suggest that these biases continue to operate following selective lesions of the hippocampus proper but are disrupted following lesions of the entorhinal cortex. More generally, our results support the suggestion that acquired changes in stimulus distinctiveness, whether assessed using an IDS–EDS procedure or an acquired equivalence–distinctiveness procedure, are mediated by shared neural and conceptual mechanisms that can be dissociated from those that underlie conditional learning. Any adequate model of learning, and particularly any model concerned with how experience with stimuli modifies the distinctiveness of their representations, will need to address both the results presented here and those described in our companion article.

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