Dissociations in the effect of delay on object recognition: Evidence for an associative model of recognition memory

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ABSTRACT

Rats were administered three versions of an object recognition task: In the spontaneous object recognition task (SOR) animals discriminated between a familiar object and a novel object; in the temporal order task they discriminated between two familiar objects, one of which had been presented more recently than the other; and, in the object-in-place task, they discriminated among four previously presented objects, two of which were presented in the same location as in preexposure and two in different but familiar locations. In each task animals were tested at two delays (5 minutes and 2 hours)—between sample and test phases in the SOR and object-in-place tasks, and between the two sample phases in the temporal order task. Performance in the SOR was poorer with the longer delay, whereas in the temporal order task performance improved with delay. There was no effect of delay on object-in-place performance. In addition the performance of animals with neurotoxic lesions of the dorsal hippocampus was selectively impaired in the object-in-place task at the longer delay only. These findings are interpreted within the framework of Wagner's (1981) model of memory.

Recognition memory is procedurally defined as the ability to judge a previously experienced item as familiar (Mackintosh, 1987; Mandler, 1980), and is said to be a fundamental component of declarative memory. Much work has thus been devoted to isolating the neural substrates of recognition, and tests of recognition memory are routinely used in evaluation of the cognitive effects of genetic, pharmacological and brain manipulations—yet relatively little attention has been devoted to analysing the cognitive mechanisms that underlie this process. In animals recognition memory is typically examined using the spontaneous object recognition task (SOR; Ennaceur & Delacour, 1988). In this task the animal (usually a rodent) is presented with two identical objects and then, after a short delay, one of the original objects is replaced by a novel item. Animals tend to show enhanced exploration of the novel object, which is taken to indicate recognition of the object that was preexposed. There are several variants of this task that have been used to tap the different aspects of recognition memory. In the temporal order task (Mitchell & Laiacona, 1998) animals are sequentially presented with two pairs of identical items, and at test given a choice between one item from each pair. Animals preferentially explore the item that was experienced less recently. In contrast, in the object-in-place task (Dix & Aggleton, 1999) several items are presented in an array, and at test some of these items change location, moving either to previously unoccupied, and hence novel, locations, or to familiar locations previously occupied by other objects (achieved by swapping the position of two or more items). In both versions animals preferentially explore the items in the "wrong" locations. (In the present article we will concentrate on the case in which the familiar items are moved to a familiar location, to rule out the more mundane possibility that the animals are responding to the novelty of the location *per se*, rather than the detection of the item in the "wrong" location.)

Although all three of these tasks are widely used, there is controversy over how performance on each is to be explained. For example, there is a family of theories that proposes recognition memory involves two independent processes, familiarity and recollection—illustrated by the distinction between knowing *that* an item has been encountered before and, for example, knowing something *about* that item (Yonelinas, 2002). It has also been argued that these processes depend on different anatomical areas. For example, Aggleton and Brown (1999, 2006) have presented a body of evidence suggesting that the contribution of different brain areas to the different types of recognition memory can be dissociated, and that the different types of task have differential dependence on familiarity and recollection. They proposed that familiarity depends on a circuit involving the perirhinal cortex, while recollection depends on connections between the hippocampus and structures such as the anterior thalamus. Others have argued that all types of recognition rely on one underlying memory process, and that apparent differences stem from differences in the strength of the memory trace (Squire, Wixted & Clark, 2007). These two theoretical positions thus differ as to whether or not performance in the SOR, object-in-place, and temporal order tasks depends on the same underlying mechanisms.

A more recent suggestion, proposed by Sanderson and Bannerman (2011; see also Honey & Good, 2000; Whitt, Haselgrove & Robinson, 2012; Whitt & Robinson, 2013), is that all three types of recognition memory can be understood in terms of a model of memory proposed by Wagner and colleagues (e.g., Brandon, Vogel & Wagner, 2003; Wagner, 1981). This account assumes that a stimulus may be conceptualised as a set of constituent elements that can exist in several different memory states. When a stimulus is first presented, some its constituent elements go from an inactive state (I) to a primary (A1) state of activation, from which they decay rapidly to a secondary (A2) state of activation, and thence more slowly to their initial inactive state. Importantly the sequence of transition between these states is fixed,

such that elements can never go directly from the A2 state into the A1 state without first decaying into the inactive state. A stimulus must be active to elicit a response, and a stimulus element in A1 elicits a stronger response than one in A2. This model was originally formulated to explain associative learning, and incorporates the additional principle that if a stimulus that has previously been associated with the target stimulus is presented, elements of the target will directly enter the secondary, A2 activation state—a process termed *retrieval-generated priming*.

This model can explain performance in the simple SOR task if it is assumed that the unconditioned response to a novel object is exploration. When an object is familiarised in the sample phase, its elements initially enter the A1 state, but then rapidly decay into the A2 state (a process termed *self-generated priming*). Because each stimulus only has a finite number of elements, if the test is conducted while some of the preexposed stimulus elements are still in A2, these elements cannot be activated into A1 when the stimulus is next presented. Subsequent presentation of the familiar stimulus will therefore result in less A1 activity than if the stimulus is entirely novel—resulting in more exploration of the novel item. This reasoning depends critically on the assumption that some of the preexposed stimulus elements are still in A2 at test. Thus if the next presentation of this stimulus is delayed sufficiently, all of its constituent elements will have returned to the initial inactive state and be just as susceptible to A1 activation as those of the novel object.

Performance in the temporal order task relies on a very similar process, but here both stimuli are presented in the sample phases, so the comparison at test is between two familiar stimuli whose elements have decayed into A2 to differing extents. As elements of the *more* recently presented item will have had less opportunity to decay from A2 into the inactive state than those of the less recent item, the more recent object will command less A1 activity, and hence less responding at test—exactly what is observed. This task, like the SOR task, is

also predicted to be delay dependent, but here the critical delay is between the two sample phases: The longer the delay between presentation of the two stimuli, the greater the difference in A2 activity between the to-be-discriminated items, and the greater the difference in performance (although as in the SOR tasks, if the interval between presentation of the second object and the test is sufficiently long, then both items' elements will have decayed to their inactive states by the time of the test).

In contrast to these two, highly delay-dependent tasks, object-in-place performance is explained through a slightly different mechanism. Here it is assumed that during initial exposure all objects become associated with their surroundings, so that the environmental cues accompanying them in preexposure become capable of activating their elements directly into the A2 state, via the retrieval-generated priming mechanism. Objects that are moved from their preexposure location at test will be less susceptible to this priming process than those presented in the original location—with the result that the shifted items will command more A1 activity, and thus more exploration. (The same process may occur in the SOR task, which would further reduce preference for the familiar object). Because object-in-place performance arises from an association between the objects and the context which persists over time, performance in this task is predicted to be *independent* of the delay between the sample and test phases.

Some evidence has been presented to support this interpretation of object recognition performance in terms of Wagner's model. For example, Whitt et al. (2012) demonstrated that preference between two equally familiar objects could be influenced by presenting a stimulus that had been *associated* with one of the objects just before test. According to the model, this event produces retrieval-generated priming of the associated target object's elements into the A2 state—resulting in preferential exploration of the alternative object; this is what the authors observed (see also Sanderson & Bannerman, 2011; Whitt & Robinson, 2013).

Thus the aim of the present experiment was to provide a different test of the predictions of Wagner's model, by examining the differing effects of delay on these three types of recognition task. We are not aware of any studies that have systematically compared the effect of delay on these three different tasks under directly comparable conditions. Animals were given the SOR, object-in-place, and temporal order tasks, and the delays between sample and test phases (in SOR and object-in-place tasks) and between the two sample phases (in temporal order task) were manipulated. We anticipated that the effect of increasing delay would vary among these tasks. For example, in the SOR, at short delays the elements of the sample item will be largely in A2 at test, meaning they will be unavailable for recruitment into A1. Thus the novel item will command more activity than the preexposed, and performance will be good. As the sample-test delay increases, however, the sample item's elements will increasingly return to the inactive state, so that when it is re–subsequently presented more of its elements will be available to enter A1—like those of the novel comparison item. Thus with longer delays the preexposed item becomes more like the novel item, and it becomes more difficult to discriminate between the two objects at test.

In contrast, the same analysis predicts that increasing the sample-sample delay should produce *more* differential behaviour in the temporal order task. This is because differential exploration of the less recent item here depends on the two items commanding differing levels of A2 activity at test, and the level of A2 depends on the time since item presentation. A long sample-sample interval will ensure that the first item's elements will have had more time to decay from A2, and thus be more available for A1 activation at test than those of the second item—fostering differential exploration of the two items at test. In contrast a short sample-sample interval will result in the two items eliciting similar A1 activity in the test stage, resulting in less discrimination between them. (Note that manipulating the interval between the *second* sample and the test is predicted to have relatively little effect on task

performance, as the *difference* in the ability of the two items to elicit A2 activity is not affected by this factor[1]).

Manipulating delay is, put simply, predicted to influence performance on the SOR and temporal order tasks because both depend on the delay-dependent self-generated priming process. In contrast, manipulating sample-test delay is predicted, by comparison, to have no effect on object-in-place performance. This is because differential exploration of the displaced items depends on the ability of the contextual cues associated with the target items to put their elements into the A2 state through the retrieval-generated priming process. But the strength of the learned associations underlying this effect is relatively independent of delay: Although ability to *retrieve* an association can be attenuated by environmental factors such as a change of context, performance can typically be restored by testing in the training context.

An additional aim of this study was to examine the effects of dorsal hippocampus (DHPC) damage in these tasks. There is considerable evidence that the hippocampus, and more specifically its dorsal portion, is involved in some types of recognition memory. Nonetheless, it is difficult to draw conclusions about which underlying processes are impaired by DHPC damage, as there is inconsistency in the results that have been reported. The majority of studies have failed to demonstrate a deficit in the SOR task, either after complete hippocampal lesions (Barker & Warburton, 2011; Good, Barnes, Staal, McGregor & Honey, 2007; Mumby, Gaskin, Glenn, Schramek & Lehmann, 2002), or lesions confined to the DHPC (Lee, Hunsaker & Kesner, 2005; Save, Poucet, Foreman & Buhot, 1992). Nonetheless, deficits have been reported after complete hippocampal damage if the delay between training and test is extended (Clark, Zola & Squire, 2000; although see Forwood, Winters & Bussey, 2005; Mumby, Tremblay, Lecluse & Lehmann, 2005 for failure to observe any such effect of delay using slight variants on the standard task), and after DHPC

lesions if they are sufficiently large (Broadbent, Squire & Clark, 2004). There is more consistent evidence that hippocampal damage can affect performance in the temporal order task: Deficits have been reported after both complete hippocampal (Barker & Warburton, 2011; DeVito & Eichenbaum, 2010; but see Good et al., 2007) and DHPC (specifically CA1) lesions (Hoge & Kesner, 2007; Hunsaker, Fieldsted, Rosenberg & Kesner, 2008; Hunsaker & Kesner, 2008). Finally, there are many reports that hippocampal damage can impair object-in-place performance. Lesions of the entire structure produce impairments both in the version of the task in which the target object is moved to a novel location (Barker & Warburton, 2011; Mumby et al., 2002), and in the version of the task in which it is shifted to a different, but familiar, location (Barker & Warburton, 2011; DeVito & Eichenbaum, 2010; Good et al., 2007; Liu & Bilkey, 2001; but see Langston, Stevenson, Wilson, Saunders & Wood, 2010; Langston & Wood, 2010). When lesions are confined to the DHPC, however, while deficits have been found in the novel location version of the task (Lee et al., 2005; Save et al., 1992), we are only aware of one report of a deficit when the object is shifted to a familiar location (Goodrich-Hunsaker, Hunsaker & Kesner, 2008—again specifically after CA1 damage)—and not all authors have managed to replicate this finding (Lee et al., 2005). Thus, after the type of DHPC lesions used in the present study, performance in the temporal order task has been the most consistently affected (Hoge & Kesner, 2007; Hunsaker et al., 2008; Hunsaker & Kesner, 2008). In contrast, performance in the SOR was affected only when lesions were sufficiently large (Broadbent et al., 2004), and the effect is only rarely reported in the object-in-place task (Goodrich-Hunsaker et al., 2008).

In view of this inconsistency in the reported findings, we chose to adopt the novel approach of exploring whether damage to the DHPC influences the effect of *delay* in any of these tasks. As noted above, few studies have looked *specifically* at the effects of delay on recognition performance after hippocampal damage, and these have used only the SOR task;

moreover none have examined these effects in animals with lesions confined to the DHPC. Moreover, there are independent reasons for thinking that hippocampal damage might interact with the effect of delay in these tasks. The DHPC is often considered to be crucial for maintaining stimulus representations across time (McEchron & Disterhoft, 1999). For example, DHPC lesions in rabbits and rats often impair, respectively, acquisition of eyeblink and fear trace conditioning, in which the conditioned stimulus (CS) terminates before the onset of the unconditioned stimulus (US). Conversely, DHPC lesions have no effect on conditioning when the CS and US are temporally contiguous (Fendt, Fanselow & Koch, 2005; Solomon, Vander Schaaf, Thompson & Weisz, 1986; but see Tam & Bonardi, 2012). If the DHPC has a general role in maintaining across time the stimulus traces required for recognition performance in these tasks, then might influence the effects of delay on these tasks.

METHODS

Animals

Forty-eight Lister Hooded male rats (Harlan, Bicester, UK) were used, and they weighed about 300 g at the start of surgery. Half were randomly assigned to the sham-lesioned group, and the remaining half to the DHPC-lesioned group. Animals of the same group were caged in pairs in a colony with a light-dark cycle of 12 hr, with the light phases started at 07:00.

Surgery

Animals were anaesthetised with isofluorane at the start of surgery. The scalp was incised along the midline and the facial muscles were retracted. Portions of cranial bone above the DHPC were removed with an electric drill. In the DHPC-lesioned group, bilateral lesions

were achieved by injecting ibotenic acid into 14 sites: anterior-posterior (AP) −2.4 mm, medial-lateral (ML) ±1.0 mm, dorsal-ventral (DV) −3.0 mm; AP −3.1 mm, ML ±1.4 mm, DV −2.1 mm; AP −3.1 mm, ML ±1.4 mm, DV −3.0 mm; AP −3.1 mm, ML ±3.0 mm, DV −2.7 mm; AP −3.9 mm, ML ±2.2 mm, DV −1.8 mm; AP −3.9 mm, ML ±2.2 mm, DV −3.0 mm; and AP −3.9 mm, ML ±3.5 mm, DV −2.7 mm. The AP and ML coordinates were relative to bregma, whereas the DV coordinates were relative to the brain surface. The volume of ibotenic acid injected at sites AP −2.4 mm, ML ±1.0 mm, DV −3.0 mm was 0.05 µl; the volume injected at all other sites was 0.1 µl. The concentration of the injected ibotenic acid solution was 63 mM, which was made from dissolving 5 mg of ibotenic acid solids (Sigma-Aldrich, Dorset, UK) into 0.5 ml of 0.1 M phosphate-buffered saline (pH 7.4). Injections were administered by an infusion pump (KD Scientific, Holliston, Massachusetts) at rates of 0.03 µl/min using a 2-µl syringe (Hamilton, Bonaduz, Switzerland) with a 25-gauge, bevel-tip needle. After each injection, the needle was left in situ for 1 min before it was withdrawn and moved to the next site. In the sham-lesioned group, the needle was lowered into the same sites but no ibotenic acid was injected. The scalp was sutured after all sites had been visited. Animals were injected subcutaneously with 1 ml/kg of Rimadyl (Pfizer, Surrey, UK) as analgesic and 0.5 ml of warmed saline to prevent dehydration; all recovered from surgery within two weeks.

Apparatus and Stimuli

Four rectangular open fields (length × width × height: 50 cm × 40 cm × 40 cm), located in a brightly lit room, were used simultaneously. The walls and floors of the open fields were white translucent plastic. Each field was equipped with two spotlights, 20 cm apart from each other and 85 cm above the floor of the apparatus, which were switched on at the start of each phase and off when the phase ended. A web camera was installed between the spotlights; an animal's trajectory was tracked and recorded by the Any-Maze software (version 4.5;

Stoelting, Wood Dale, Illinois). Objects were made of various materials (e.g., plastic, glass, stainless steel, and ceramic) and of different sizes and shapes; different sets of objects were used for each type of task, and at each delay. There were 16 types of object in total and 4–6 copies of each type of object. Hereafter, the different copies of an object, for example, A, are indicated by A, A′, A″, etc.

Procedure

The procedures employed here were adapted from Good et al. (2007). Initially animals received two open-field habituation sessions. During each session, the subject was placed in the apparatus facing the midpoint of the long wall and was allowed to explore the apparatus for 10 min. Half of the subjects in the sham- and DHPC-lesioned groups faced the upper long wall and the remaining half the bottom long wall; the starting position remained unchanged across all sessions. The apparatus was cleaned with a cloth moistened with diluted alcohol after each animal was tested.

After two habituation sessions, animals received two SOR trials, two object-in-place trials, and two temporal order trials. Recognition trials were given in two blocks of three trials in the following order—Block 1: SOR, temporal order, and object-in-place trials; Block 2: object-in-place, temporal order, and SOR trials; this order of trial presentation was the same for every animal. For half of the animals in each group, 5-min delays were used in Block 1 and 2-hr delays were used in Block 2; this assignment was reversed for the remaining animals. Each animal was tested over a course of six days (1 trial per day).

SOR trials. Each SOR trial consisted of a sample phase, a delay period, and a test phase; the delay between the sample and test phases was either 5 min or 2 hr. During the sample phase, the subject was allowed to explore two copies of a novel object, A and A′, for 5 min. The two copies were placed at the centres of two notional, 10-cm radius circles located

along the horizontal midline of the apparatus; the centres of the circular zones were 30 cm apart. After the required delay, the subject was returned to the apparatus, which now contained an entirely novel object, B, and a new copy of the familiar object, A″, and was tested for a duration of 3 min (Figure 1, *top* panel). During both the sample and test phases, the Any-Maze software tracked the animal's head, and the amount of time its head was within the left and right circular zones was recorded. On each trial, object identities and the location of the novel object at test were counterbalanced within and between groups. More specifically, for half of the animals in each lesion group, B was the novel object at test; for the remaining animals, A was novel at test. In addition, for half of the animals in each of these subgroups, the novel object at test was placed in the left zone, whereas for the remaining animals it was in the right zone.

Object-in-place trials. The object-in-place trials were similar to the SOR trials, but with the following exceptions. First, during the sample phase there were four novel objects, A, B, C, and D, each located at the corners of the apparatus. Each of the four zones was a quadrant of a notional 15-cm radius circle at the corner of the apparatus. During the test phase, new copies of the four familiar objects, A′, B′, C′, and D′, were presented and the locations of two objects that were previously diagonally opposite to each other were switched (Figure 1, *middle* panel). On each object-in-place trial, object identities and the corners at which the switch occurred were counterbalanced within and between groups. For example, for half of the animals in each group, A and D were selected as the objects remained in the same locations at test, and B and C were swapped; this assignment was reversed for the remaining animals. In addition, for half of the animals in each of these subgroups, the objects that remained in the same locations were in the top-right and bottom-left corners, and the objects that were swapped were in the top-left and bottom-right corners; this assignment was reversed for the remaining animals.

Temporal order trials. The temporal order trials were similar to the SOR trials, but there were two sample phases, and the choice at test was between a less recent object and a more recent object. In the first sample phase, two copies of a novel object, A and A′, were presented; in the second sample phase, two copies of a different object, B and B′, were presented; at test, new copies of the two familiar objects, A″ and B″, were presented (Figure 1, *bottom* panel). Intact animals tend to explore A″ more than B″ (Mitchell & Laiacona, 1998). The delay between the two sample phases was either 5 min or 2 hr, but the delay between the second sample and test phases was always 5 min. On each temporal order trial, the identities of the objects that were presented in the first and second sample phases, and whether they were placed in the left or right zone at test, were counterbalanced as on the SOR trials. Thus, for half of the animals in each group, A was presented in the first sample phase and B in the second, and this assignment was reversed for the remaining animals. In addition, for half of the animals in each of these subgroups, at test the object from the first sample phase was placed on the left and that from the second sample phase on the right; this assignment was reversed for the remaining animals.

Histology

After the experiment, animals were sacrificed with an overdose of pentobarbitone and perfused intracardially with formal saline. Their brains were stored in formal saline at room temperature for two days, subsequently in 20% sucrose solution at a temperature of 4 °C for two days. Brains were then cut with a cryostat at a temperature of −19 °C; coronal sections were 40 μm in thickness, and every fifth section was collected. The recovered sections were stained with cresyl violet solution and were dried at room temperature. For each animal, the AP coordinates of the recovered coronal sections were identified using the Paxinos and Watson (2005) atlas. For each identified section, the intact hippocampus in each hemisphere

was outlined using ImageJ (version 1.40; National Institutes of Health, Bethesda, Maryland); the hippocampal areas in both hemispheres were estimated (in pixels), and the overall hippocampal area was calculated. Subsequently, the mean overall hippocampal area of the sham-lesioned group was calculated, and the extent of hippocampal damage of each animal in the DHPC-lesioned group was expressed as a percentage of the mean of the sham-lesioned group.

Data Treatment and Analyses

Recognition performance on each trial was expressed as a recognition ratio of the form $(x_1-x_2)/(x_1+x_2)$, where x_1 was the total time spent in contact with the novel objects on the SOR trials, the displaced objects on the object-in-place trials, or the less recent objects on the temporal order trials; and x_2 was the total time spent in contact with the familiar objects on the SOR trials, the non-displaced objects on the object-in-place trials, or the more recent objects on the temporal order trials. An animal indifferent to the objects would score zero; preference for the novel/displaced/less recent object yields a score greater than zero, and a preference for the familiar/non-displaced/more recent object a score less than zero. In addition, in order to examine if DHPC lesions exaggerated the effect of delay on recognition performance, for each animal a *delay ratio* was determined for each task, to capture the relative difference in performance at 5-min (r_1) and 2-hr delays (r_2): $(r_1-r_2)/(|r_1+r_2|)$. The higher the delay ratio is above zero, the greater is the detrimental effect of delay on performance; scores below zero mean increasing the delay improves performance. Data were analysed using split-plot analysis of variance (ANOVA); partial η^2 and its 95% confidence interval (CI) were given for significant main effects and interactions in the ANOVA; significant interactions were explored with simple main effects analysis using pooled error term.

RESULTS

Histology

The twenty-four animals that received ibotenic-acid injections sustained bilateral damage to the anterior dorsal pole of the dentate gyrus, CA3 and CA1 subregions. Hippocampal damage tended to start at AP bregma −1.80 mm (Plate 48 in Paxinos & Watson, 2005) and extend to AP −4.68 mm (Plate 72). The mean hippocampal damage was about 38% of total hippocampal volume among these twenty-four animals (ranged from 15–45%); no dorsal subicular damage was detected. No hippocampal or subicular damage was detected in the sham-lesioned group. Example photomicrographs from representative sham- and DHPC-lesioned animals are shown in Figure 2.

Recognition Performance

The recognition ratios, denoting group mean recognition performance, are shown in Figure 3. For both SOR and temporal order version of the task there appeared to be a clear effect of delay: Delay appeared to worsen performance in the SOR task but improve performance in the temporal order task. There also appeared to be a slight effect of lesion in the SOR task but little in the temporal order task. In the object-in-place task, in contrast, although there seemed to be little effect of delay in the sham-lesioned group, there was an apparent reduction in accuracy in the DHPC-lesioned group. This interpretation was in part confirmed by a Lesion (Sham or DHPC) × Task (SOR, Object-in-place, or Temporal Order) × Delay (5 min or 2 hr) split-plot ANOVA. This revealed a main effect of Task, $F(2,92)=13.56$, $p<.0005$, partial $\eta^2=.228$, CI=[.085, .354], but critically also a Task × Delay interaction, Greenhouse-Geisser corrected $F(1.74,92)=10.49$, $p<.0005$, partial $\eta^2=.186$, CI=[.044, .292]. However, neither the main effect of Delay or any other interaction involving this factor was significant, Fs<1; the effect of Lesion was not significant, $F(1,46)=2.57$ $p=.12$. Simple main effects analysis

revealed that, in the SOR task recognition performance was better when the delay was 5 min than when it was 2 hr, $F(1,138)=5.60$, $p<.025$, while in the temporal order task performance was better when the delay was 2 hr than when it was 5 min, $F(1,138)=10.79$, $p<.005$; there was no effect of delay in the object-in-place task, $F(1,138)=3.12$, $p=.08$. Thus, in accord with our predictions based on Wagner's (1981) model, increasing the sample-test delay impaired SOR performance but had no effect on object-in-place recognition performance; performance in the temporal order task was, in contrast, enhanced by an increase in delay.

Although it appeared numerically that DHPC lesions produced a selective effect of delay in the object-in-place task, the critical three-way interaction among Lesion, Task and Delay was not statistically significant ($F<1$). However, it is possible that the evident difference in absolute levels of performance in the three types of task might mask any interactive effect, and that a measure of the effect of delay that normalised for absolute performance might prove more sensitive. Thus for each animal a delay ratio was calculated; the higher this score is above zero, the greater the detrimental effect of delay on performance. The resultant scores are shown in Figure 4. It is clear that for the SOR task the ratios were above zero for both groups, indicating that recognition deteriorated over the delay; the opposite was true in the temporal order task, where for both groups performance was *better* after 2 hr than after 5 min. However, for the object-in-place task it appeared that, while the sham-lesioned animals were numerically better at the longer delay, the opposite was true for the DHPC-lesioned subjects. These conclusions were supported by the results of a Lesion × Task ANOVA, which revealed a significant Lesion × Task interaction, $F(2,92)=4.69$, $p<.025$, partial $\eta^2=.093$, CI=[.005, .202]; the main effects of Lesion and Task were not significant, $F(1,46)=2.71$, $p=.11$ and $F<1$, respectively. Simple main effects analysis revealed a significant effect of Lesion in the object-in-place task, $F(1,138)=12.57$, $p<.001$, but not in the SOR and temporal order tasks, Fs<1. This suggests that, while the effect of delay did not

differ between the two groups in the SOR and temporal order tasks, the 2-hr delay produced a significantly greater reduction in performance in the DHPC-lesioned group.

General Object Exploration

Two Lesion × Task × Delay ANOVAs were conducted on the amount of time spent exploring objects during the sample phases and the test phases (Table 1; for the temporal order task this referred to the mean for sample phases 1 and 2); there were no main effects of Lesion and no interactive effects involving this factor, Fs<1.

DISCUSSION

In the SOR task, increasing the delay from 5 min to 2 hr led to a decrement in recognition performance; no such effect was observed in the object-in-place task, while in the temporal order task increasing the delay between the sample phases improved performance. These results support an account of recognition performance in terms of Wagner's (1981) model (see also Whitt et al., 2012; Whitt & Robinson, 2013). According to this theory, self-generated priming is the primary mechanism responsible for performance in the SOR and temporal order tasks, while retrieval-generated priming underlies object-in-place performance. In terms of the distinction between familiarity and recollection outlined above, there is an apparent correspondence: Self-generated priming produces memory for the item itself without giving any associated information, and is thus related to the notion of familiarity. In contrast retrieval-generated priming could be regarded as being analogous to recollection, in that it involves associations between the target object and other items. In this sense our interpretation of recognition performance could be seen as consistent with, rather than in opposition to, the traditional two-process account of recognition memory adopted by

authors such as Aggleton and Brown (2006) as a framework for their theory of which neural structures mediate the various components of the recognition memory process.

But despite the fact that these two perspectives are not in opposition to each other, interpreting recognition effects within the framework of Wagner's model provides an additional level of specification and detail that was absent from the more traditional two-process view. It thus not only yields new insights into recognition memory, but also generates novel predictions—such as that concerning the differential effects of delay on the two processes that was tested in the present study. Another interesting issue relates to the independence of the two memory processes. It is often assumed that familiarity and recollection are independent, and in traditional models there has been little elaboration of how they might be interrelated (Yonelinas, 2002). In contrast, the relationship between self- and retrieval-generated priming is rather better specified within the framework of Wagner (1981). Retrieval-generated priming, as explained previously, depends on an association between, for example, surrounding cues and the target object. However, in order for an excitatory association to be established between two stimuli both their elements must be in the A1 state. Thus in certain situations there can effectively be competition between the self- and retrieval-generated priming processes. If treatment of the to-be-associated events favours self-generated priming, their elements will be predominantly in A2, precluding formation of the associations required for the retrieval-generated priming process. That such competition exists has been demonstrated in different species (Davis, 1970; Pedreira, Romano, Tomsic, Lozada & Maldonado, 1998; Sanderson & Bannerman, 2011; Tomsic, Berón de Astrada, Sztarker & Maldonado, 2009). In short, applying Wagner's more general model of learning to recognition memory not only provides an explanation that is consistent with prior approaches, but adds a new level of specification which yields a number of novel and testable predictions.

However, there are some problems with drawing a parallel between self-generated priming and familiarity as understood by these dual-process models. First, although Wagner's model can only make relative predictions about the time course of decay between the various activation states, it pushes credibility to draw an exact parallel between familiarity, which can last days, and self-generated priming, which would most naturally be assumed to have dissipated completely over a 24-hr period (cf. Sanderson & Bannerman, 2011). That familiarity-based judgements can be made over longer intervals suggests they cannot be explained solely in terms of this self-generated priming process. The model can, nonetheless, offer explanations of performance on "familiarity" tasks like the SOR task over these longer intervals. First, as was alluded to earlier, some retrieval-generated priming may occur in these tasks, so that the presentation of the arena itself may also prime the target object into its A2 state. In other words SOR does not depend solely on familiarity (which could explain the inconsistent effects of lesions on this task: Barker & Warburton, 2011; Broadbent et al., 2004; Clark et al., 2000; Good et al., 2007; Mumby et al., 2002). Other possibilities must also be considered. Experiments on habituation have a strong parallel to the recognition paradigms used here, in the sense that a stimulus is presented, and then the unconditioned response to that stimulus is compared to the case in which the stimulus is novel. Less unconditioned responding is seen to the familiar stimulus, even after long delays, which Wagner's original theory explained in terms of a retrieval-generated priming effect. However, if this were the case, then changing the context at test should remove the cues required to prime the target object, and eliminate the habituation effect. But although this is sometimes the case (Honey, Good & Manser, 1998; Jordan, Strasser & McHale, 2000), the fact that habituation can remain intact in a new and discriminably different context (Hall & Channell, 1985) challenges Wagner’s account of habituation. In this case the persistent habituation effect can still be explained in terms of retrieval-generated priming, but among the elements

that compose the target stimulus (cf. McLaren & Mackintosh, 2000). In summary, although at face value the parallel between self- and retrieval-generated priming on the one hand and familiarity and recollection on the other seems attractive, in fact it might be less direct than it first appears; and that for familiarity in particular, recognition over longer delays is more likely to be due to a retrieval-generated priming process.

Dorsal hippocampal lesion had no effect on performance on either the SOR or the temporal order tasks. The failure to find a deficit in the SOR task is consistent with the majority of previous studies; the only report of an impairment after lesions confined to the DHPC was in animals in whom damage encompassed 75–100% of total hippocampal volume (Broadbent et al., 2004)—significantly more than the 15–45% damage present in our subjects. Moreover, although deficits have been reported in the temporal order task with DHPC lesions (Hoge & Kesner, 2007; Hunsaker et al., 2008; Hunsaker & Kesner, 2008), these studies employed a more complex task in which subjects were exposed to three pairs of identical objects with a short, 3-min delay between presentations, and then given a choice between the items from the first and third pairs. As our findings suggest that short sample-sample delays maximise difficulty in this task, this in combination with the need to discriminate among more objects makes it likely that their tasks were considerably more difficult than ours, which could explain the apparent discrepancy between the two sets of findings.

In contrast, using the delay ratio measure, there was a significant deficit in the object-in-place task after the longer delay. A previous study examining the effect of selective DHPC lesions has failed to find a deficit in this version of the task (Lee et al., 2005). However, this study used a relatively short delay between the sample and test phases (3 min), which could explain the apparent discrepancy with our findings. We also failed to observe a lesion deficit with an equivalently short, 5-min delay between the sample and test phases, and the effect of

DHPC lesions was only evident when the delay was 2 hr. However, Goodrich-Hunsaker et al. (2008) reported a deficit after CA1 lesions in a variant of the task with only a 10-min delay between sample and test phases. It is possible, although perhaps unlikely, that ten minutes is long enough to observe a deficit whereas 5 minutes is not. An alternative possibility is that it is attributable to the fact that their task had two test phases, unlike that used here (or that used by Lee et al., 2005). More specifically, after two phases of exposure to a 4-item array, animals received a test in which two of the four items exchanged places. This test also constituted the first of two additional sample phases, this time with the test array, after which a second test was administered in which the remaining two items exchanged places. Data were pooled across the two test sessions. Thus it is at least conceivable that this task is more difficult than the one used here, and thus more sensitive to lesion effects; but this explanation is necessarily speculative.

It is possible to attempt an interpretation of this pattern of results in terms of Wagner's model. According to this view the three tasks depend on two different underlying mechanisms—self- and retrieval-generated priming—either of which could be affected by DHPC lesions. Assuming that performance on our temporal order and SOR tasks relied on self-generated priming, and the object-in-place task on retrieval-generated priming, then the fact that DHPC lesions produced a selective deficit in the object-in-place task is consistent with the proposal that DHPC lesions are selectively involved in the retrieval-generated priming process.

The question remains as to why lesions accentuated the effect of delay on this retrieval-generated priming process. If lesion simply impaired retrieval-generated priming, then one might expect deficits at both short and long delays. One possibility is that the effect of lesion on this priming process is more evident in more difficult instantiations of the task—such that any manipulation that made the task generally more difficult might have an

especially detrimental effect on DHPC-lesioned subjects' performance. For example, as noted above, successful associative retrieval is fostered when the test is conducted in a similar context to that in which training occurred, and it is now well established that one important component of the functional context is time—the longer after preexposure the test occurs, the greater the difference in any temporal cues that were present during preexposure, and so the more the context is deemed to have changed (Bouton, 1990). It is possible that the resultant, albeit modest, increase in task difficulty at the longer delay pushed the already challenged lesioned animals over a threshold, and only then resulted in a measurable deficit in performance. Another possibility is that, rather than relying solely on environmental cues as we have implicitly assumed, animals were also using egocentric cues—which allow an animal to distinguish between different locations with respect to its fixed starting position in the arena without using any surrounding contextual cues (Simons & Wang, 1998; Wang & Simons, 1999)—to solve the task. There is evidence that egocentric spatial representations decay rapidly across time (Chen, Byrne & Crawford, 2011), which could mean they would be available to the DHPC-lesioned subjects at the 5-min, but not 2-hr, tests, giving rising to the apparent intact object-in-place performance at the short delays. A further and more mundane possibility that must be acknowledged is simply that the object-in-place is inherently more difficult than the SOR and temporal order versions, because it requires memory for four items rather than two. It might be that if the SOR and temporal order tasks were sufficiently difficult, then a lesion effect at the longer delays might also be revealed in these tasks. There is nothing in the present data that allows us to definitively reject this possibility.

The idea that retrieval-generated priming is selectively affected by DHPC lesions has, however, parallels in the literature. In their original model, Aggleton and Brown (1999) proposed on the basis of lesion evidence that the hippocampus was involved in circuits responsible for recollection rather than familiarity. Moreover, more recently Sanderson et al.

(2011) reported that deletion of the AMPA receptor's GluA1 subunit has an apparently complementary effect—selective impairment of self-generated priming, but intact retrieval-generated priming. As this receptor type is thought to be an important mediator of synaptic plasticity in the hippocampus, this suggests that some aspects of hippocampal function may be usefully considered within this theoretical framework (cf. Honey & Good, 2000).

We have presented an interpretation of recognition performance in terms of Wagner's (1981) model of memory. Consistent with the predictions of this model, increasing sample-test delay worsened performance in the SOR task, increasing sample-sample delay enhanced performance in the temporal order task, and delay had no effect on object-in-place performance. In addition animals with DHPC damage showed a selective effect of delay in the object-in-place task, which is consistent with the possibility that retrieval-generated priming, which has been likened to the recollection process, is impaired in these animals. We suggest that this model might provide useful insights not only into the processes underlying recognition memory (cf. Whitt et al., 2012; Whitt & Robinson, 2013), but also into the effects of hippocampal damage on performance in various types of recognition task (cf. Sanderson & Bannerman, 2011).

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FOOTNOTE

[1]This prediction is influenced by the form of the decay from A1 to A2; if decay is linearly related to elapsed time, then the second sample-test delay would be completely without effect on performance. However if, as Wagner predicts, it is exponential, then the increasing this delay will have a slightly advantageous effect on performance—but this will be much smaller than the effect of manipulating the sample-sample delay. Moreover, with sufficiently long sample-test delay both items' representations will return to the inactive state, such that no differential behaviour should be observed.

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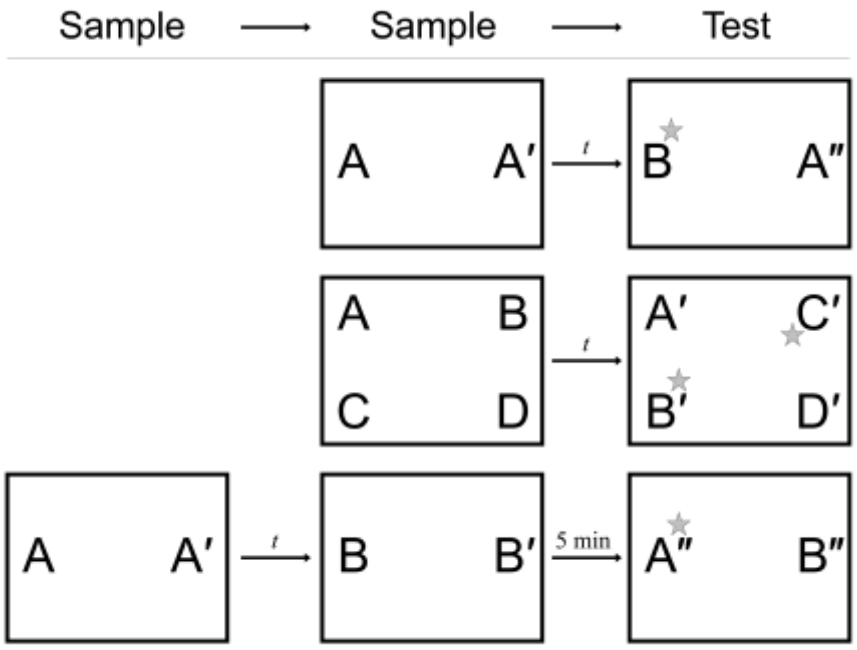

Figure 1. Schematic showing the SOR (*top*), object-in-place (*middle*), and temporal order trials (*bottom*). A, B, C, and D represent different types of object; different copies of the same object, for example, A, are represented by A, A′, and A″. *t* represents the variable delay interval, which was either 5 min or 2 hr. (Note that the delay between the second sample and test on the temporal order trial was always 5 min.) Asterisks indicate hippocampus-intact subjects' exploratory preference at test (Dix & Aggleton, 1999; Good et al., 2007; Mitchell & Laiacona, 1998).

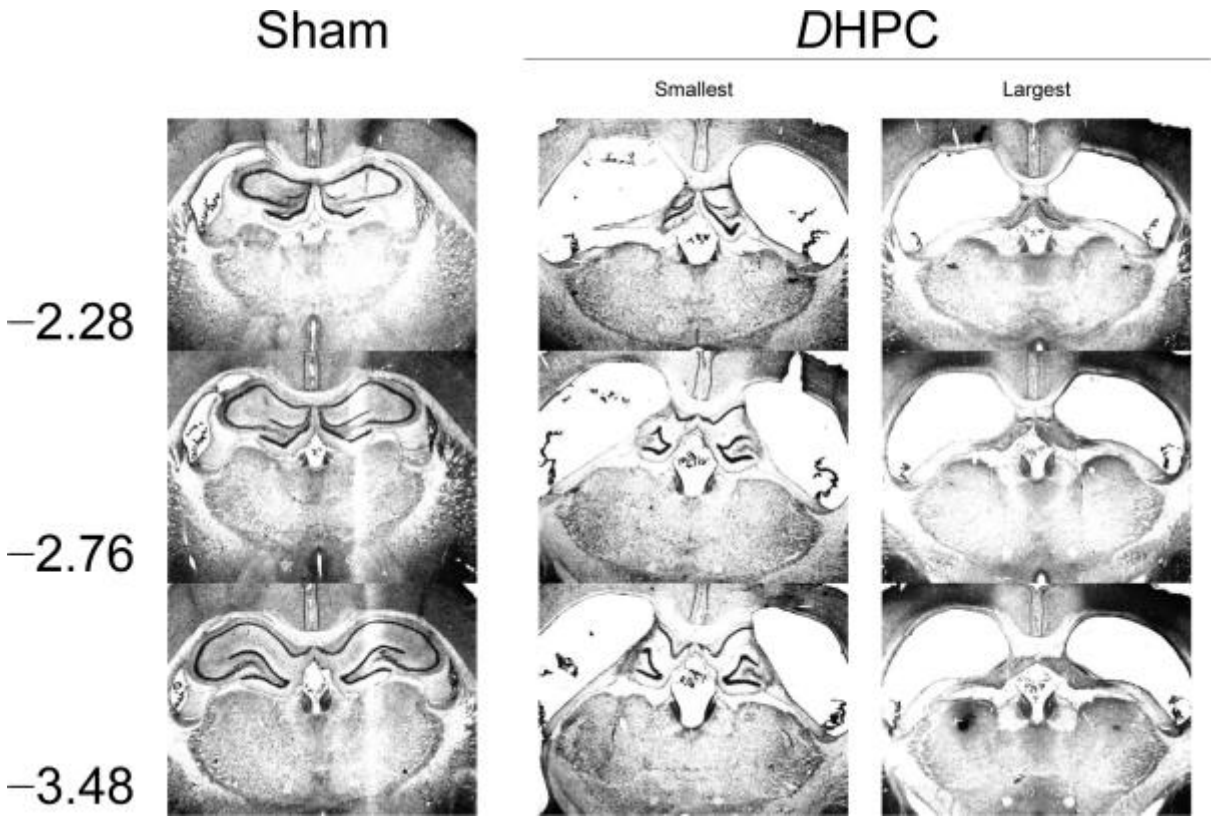

Figure 2. Example photomicrographs of coronal sections from a representative sham-lesioned animal (*left*), and two animals from the DHPC-lesioned group that sustained the smallest and largest extent of damage (15% and 45%, respectively; *right*). The top, middle, and bottom rows show, respectively, coronal sections about 2.28 mm, 2.76 mm, and 3.48 mm posterior to bregma, which correspond to Plates 52, 56, and 62 in the Paxinos and Watson (2005) atlas.

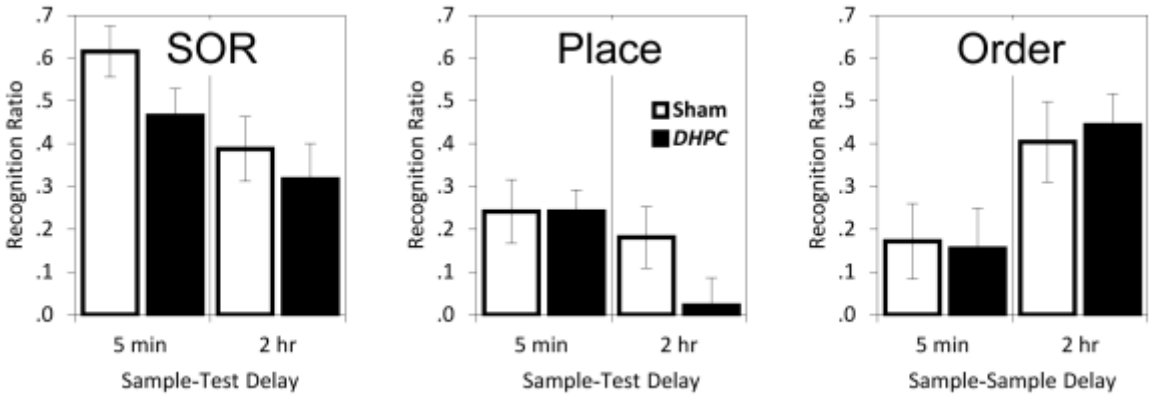

Figure 3. Recognition ratios for the SOR (*left*), object-in-place (*middle*), and temporal order tasks (*right*) after 5-min and 2-hr delays; the higher the ratio is above zero, the better the performance. The delay period was defined as the interval between the sample and test phases in the SOR and object-in-place tasks, and the interval between the two sample phases in the temporal order task; the interval between the second sample and test phases in the temporal order task was always 5 min. Error bars indicate standard errors of means.

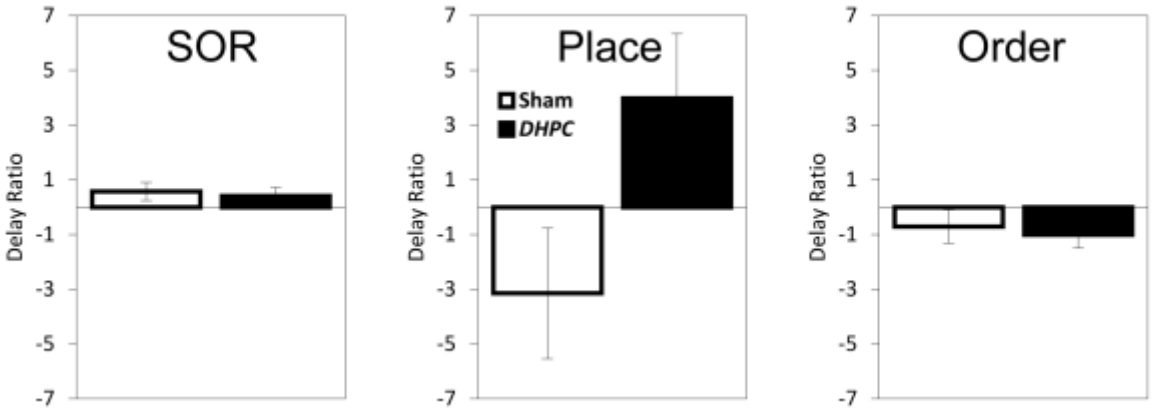

Figure 4. Delay ratios, reflecting relative differences in recognition performance between 5-min and 2-hr delays in the SOR (*left*), object-in-place (*middle*), and temporal order tasks (*right*); the higher the score is above zero, the greater the detrimental effect of the delay on recognition performance; scores below zero indicate performance is enhanced by increased delay. Dorsal hippocampal lesions exacerbated the effect of delay in the object-in-place task, but not in the SOR and temporal order tasks. Error bars indicate standard errors of means.