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Lesions of the Dorsal Hippocampus or Parietal Cortex Differentially Affect Spatial Information Processing

Jason L. Rogers
University of Utah and Medical University of South Carolina

Raymond P. Kesner
University of Utah

The present experiments used 2 versions of a modified Hebb–Williams maze to test the role of the dorsal hippocampus (dHip) and parietal cortex (PC) in processing allocentric and egocentric space during acquisition and retention. Bilateral lesions were made to either the dHip or PC before maze testing (acquisition) or after maze testing (retention). The results indicate that lesions of the dHip impair allocentric maze acquisition, whereas lesions of the PC impair egocentric maze acquisition. During retention, lesions of the PC produced a significant impairment on both maze versions, whereas lesions of the dHip produced short-lived, transient impairments on both maze versions. These results suggest that during acquisition, the hippocampus and PC process spatial information in parallel; however, long-term retention of spatial information requires the PC with the dHIP as necessary for retrieval and/or access but not necessarily storage.

Keywords: hippocampus, parietal cortex, allocentric space, egocentric space, Hebb–Williams maze

The purpose of the present experiments was to test several prominent theories of hippocampal and parietal cortex (PC) function. Although the functions served by the hippocampus and PC are varied, many researchers agree that the hippocampus and PC process spatial information in an allocentric and egocentric frame of reference, respectively. An *allocentric framework* refers to the spatial relationship between objects relative to the environment. This frame of reference appears to involve navigation through large-scale space, such as an open field or maze surrounded by extramaze cues. An *egocentric framework*, however, refers to the perception of space based on the position of the eye, head, hand, or body (Berthoz, 2000; Burgess, Jeffery, & O'Keefe, 1999). The integration of these two reference frames—allocentric and egocentric—creates the way in which an animal perceives space.

Data supporting allocentric and egocentric frames of reference stem from the classic work of Bisiatch and Luzzatti (1978). These authors asked PC-lesioned patients to recall the layout of the Piazza del Duomo in Milan, Italy. From their perspective, the patients neglected the left side of the piazza, recalling only the right side; however, when asked to view the piazza from the opposite end, they recalled the previously neglected side. In this illustration, an allocentric frame of reference refers to the layout of the entire piazza, whereas an egocentric frame of reference refers to the location of the observer. Put another way, using an allocentric frame of reference could refer to the use of exocentric (e.g., extramaze cues, buildings) cues located in the environment (e.g., maze, piazza); whereas using an egocentric frame of reference could refer to the use of ideothetic information (on the basis of inputs from the vestibular system and optic flow; see Berthoz, 2000). In the Piazza del Duomo experiment, an allocentric representation of the piazza was intact in the parietal damaged patients, whereas the viewpoint of the observer (egocentric representation) resulted in recall for only one side of the piazza. These data suggest that the PC represents egocentric information. Further, Save, Guazzelli, and Poucet (2001) reported that PC lesions disrupted ideothetic processing during path integration, whereas hippocampal lesions had a general deficit in the processing of space. Traditional maze experiments, such as the Morris water maze task, are sensitive to disruption of both the hippocampus and PC and are considered allocentric (see Kesner, 2000), suggesting that the hippocampus and PC process allocentric information. Similarly, Save and Poucet (2000b) found that lesions of the hippocampus disrupted learning during water maze testing with distal (room) cues, whereas lesions of the PC disrupted learning during water maze testing with proximal (object placed directly in the swimming pool) cues. Anatomical connectivity of these regions further support their role in their respective spatial framework. Mishkin and Ungerleider (1982) suggested that the PC is a part of the dorsal "where" stream of visual information is processed. PC also receives sensory information from motor and premotor cortices. Cells in the PC combine signals from these sensory modalities, such as vision, audition, and somatosensation. In addition, PC integrates signals from the vestibular system and signals indicating eye position and eye velocity (see Anderson, 1999), further implicating the PC in processing egocentric spatial information. Alternatively, the hippocampus, located within the temporal lobe, receives two major sources of information. First, subcortical inputs, such as the anterior thalamic nuclei, reach the hippocampus via the medial septum. In support of this pathway in spatial orientation, Moran and Dalrymple-Alford (2003) showed that lesions of the

Jason L. Rogers, Department of Psychology, University of Utah, and Department of Neurosciences, Medical University of South Carolina; Raymond P. Kesner, Department of Psychology, University of Utah.

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Correspondence concerning this article should be addressed to Raymond P. Kesner, Department of Psychology, University of Utah, 380 South 1530 East, Room 502, Salt Lake City, UT 84112. E-mail: rpkesner@behsci.utah.edu

anterior thalamic nuclei disrupt spatial memory in a 12-arm maze. Furthermore, early lesions of the hippocampus (i.e., Miller & Best, 1980) were made by transacting the medial septum. The second source of information to the hippocampus is via the entorhinal cortex perforant path. Recently, Parron, Poucet, and Save (2006) showed via a disconnection study that the entorhinal cortex and hippocampus interact during spatial information processing. Moser, Moser, and Andersen (1993) reported spatial impairments after dorsal, but not ventral, hippocampal lesions; therefore, in the present set of experiments, *hippocampus* refers to *dorsal hippocampus* (for a discussion of dorsal vs. ventral hippocampus, see Bannerman et al., 2004). "Place cells" in rodent hippocampus respond to an animal's location within its environment, such that each cell responds whenever the animal is in a particular place (O'Keefe, 1976; O'Keefe & Dostrovsky, 1971), further implicating the hippocampus in processing allocentric spatial information. Finally, computational models (e.g., Rolls & Treves, 1998) suggest that the hippocampus mediates spatial memory processes, such as pattern separation, working memory, pattern association, and pattern completion (see Kesner, Gilbert, & Wallenstein, 2000). The result of such processes is the creation of allocentric space. From these data, one may suggest that both the hippocampus and PC process spatial information; however, it appears that the hippocampus is biased toward an allocentric frame of reference, whereas the PC is biased toward an egocentric frame of reference.

Although the data clearly suggest the hippocampus and PC process allocentric and egocentric information, respectively, there is quite a discrepancy in the literature as to the acquisition of spatial information relative to long-term storage. For example, Burgess et al. (1999) have argued that the PC encodes spatial information, whereas the hippocampus serves to store spatial information for long-term use. These authors have argued two main points. The first point is that the PC encodes spatial information in an egocentric frame of reference for short-term use. The second point is that the hippocampus stores spatial information in an abstract allocentric frame of reference, irrespective of the viewer, for long-term usage. Such a viewpoint is heavily biased by the traditional cognitive map hypothesis of O'Keefe and Nadel (1978). A second perspective (Save & Poucet, 2000a) suggests that the PC encodes spatial information in an egocentric framework followed by transfer to the hippocampus for consolidation in an allocentric framework. Finally, spatial information is sent back to PC for subsequent long-term storage in an egocentric framework. Of interest to these researchers are the dynamics of the transfer and the coding of space from egocentric to allocentric to egocentric. A slightly different perspective (Kesner, 1998, 2000) suggests that the hippocampus and PC encode spatial information in parallel. Long-term storage of space is held in the PC, which serves to integrate both allocentric and egocentric frames of references. Finally, hippocampus is necessary for retrieval or access of spatial information but not necessarily storage; a view supported by additional models of hippocampal function, such as the multiple memory trace hypothesis (Nadel & Moscovitch, 1997). In sum, in one perspective, PC encodes spatial information, and hippocampus stores the information. In the second perspective, PC encodes spatial information, hippocampus consolidates the information, and PC stores it. In the third perspective, hippocampus and PC encode spatial information in parallel, and PC combines the information for storage. Thus, the following experiments were designed to test these two models of spatial memory. It was hypothesized that lesions of the hippocampus would impair acquisition of the allocentric maze, whereas lesions of the PC would impair acquisition of the egocentric maze. Furthermore, lesions of the PC, but not the hippocampus, would impair retention for both maze versions.

On the basis of the use of the two reference frames described above, it was assumed that normal animals use extramaze cues to guide navigation on the allocentric version of the maze. When those cues are removed (i.e., when given a dark probe trial), animals should make many maze errors because of the lack of cues. Animals can switch strategies—that is to say, navigate the dark maze with increased accuracy across subsequent dark trials. Further, on the egocentric version of the maze, in which the walls of the maze prohibit the use of extramaze cues, it is assumed that normal animals use self-motion (i.e., ideothetic information) to guide navigation; therefore, when given a dark probe trial, these animals should make many maze errors because of the inability to update ideothetic information with current maze position. The dark probe trial, therefore, is used to assess the strategy of the animal after maze testing. It is also assumed that during acquisition, the hippocampus represents allocentric place, on the basis of the use of extramaze cues; therefore, hippocampal-lesioned animals should not learn the allocentric version of the maze. On the egocentric version of the maze, however, hippocampal-lesioned animals should have no impairment. On the contrary, it was assumed that the PC represents egocentric place, on the basis of ideothetic information; therefore, during acquisition, PC-lesioned animals should not learn the egocentric version of the maze but should have no impairment on the allocentric version of the maze. Finally, it was assumed that the PC binds allocentric and egocentric place for long-term retention. Therefore, PC-lesioned animals should be impaired during retention of both maze versions, whereas hippocampal-lesioned animals should have little-to-no impairment. Although it was assumed that some animals would have a deficit acquiring and/or retaining the maze, it is possible that these animals can, over time, learn the maze paradigm. If it were the case that animals exhibited a deficit to learn the maze, it was assumed that it was by the use of a "backup" response strategy, mediated by the striatum (see Kesner & Rogers, 2004; Packard & McGaugh, 1996), on the basis of proprioception and the memory for body turns.

Experiment 1a: Maze Acquisition

Materials and Methods

Rats

Thirty-four male, Long–Evans rats (Simonsen Laboratories, Gilroy, CA) approximately 4 months of age at the start of the experiment, weighing ~350 g, served as subjects. The rats were housed individually in plastic tubs located in a colony with a 12-hr light–dark cycle. All rats had free access to water, with food restricted for the duration of testing to maintain each rat at approximately 85%–90% of its free-feeding weight. All testing was conducted during the light portion of the light–dark cycle.

Surgery

Surgical procedures were consistent across all experiments. Rats were randomly assigned to a surgery group. Rats were anesthetized and main-

tained with a combination of isoflurane (2%) and medical air and given atropine sulfate (0.54 mg/kg im) as a prophylactic. Each rat was placed in a stereotaxic apparatus (David Kopf Instruments, Tujunga, CA) with its head level. The scalp was incised and retracted to expose bregma and lambda in the same horizontal plane. Hippocampal lesions were made with ibotenic acid (6 mg/ml; Sigma, St. Louis, MO) infused with a microinfusion pump (Cole-Parmer, Vernon Hills, IL) and a 10-μL Hamilton syringe (Hamilton, Reno, NV) at a rate of 6 μL/hr and a volume of .20 μL per site into three sites within the dorsal hippocampus (2.8 mm posterior to bregma, 1.6 mm lateral to the midline, 3.0 mm ventral to dura; 3.3 mm posterior to bregma, 1.8 mm lateral to the midline, 2.8 mm ventral to dura; 4.1 mm posterior to bregma, 2.6 mm lateral to the midline, 2.8 mm ventral to dura). After infusion, the cannula remained in each site for 2 min to allow proper diffusion of the excitotoxin. Moser et al. (1993) reported spatial impairments after dorsal, but not ventral, hippocampal lesions; therefore, only dorsal lesions were made. Sham lesions were made with the same coordinates as the hippocampal group; however, saline was infused. PC lesions were made via aspiration. The intended lesions were 1 mm posterior to bregma and 4.5 mm posterior to bregma, 2 mm lateral to midline to approximately 1 mm above the rhinal sulcus in the medial-lateral plane, and 2 mm ventral to dura. Following surgery, the incision was sutured, and the rats were allowed to recover on a heating pad before returning to their home cage. In addition, rats received acetaminophen (children's Tylenol; 200 mg/100 ml of water) as an analgesic and mashed food for 3 days following surgery. In the acquisition experiment there were six groups, three groups per each maze version. On the allocentric version, one group received bilateral lesions of the hippocampus ($n = 6$). A second group received bilateral lesions of the PC ($n = 5$). A third group received a sham lesion and served as the control group ($n = 5$). On the egocentric version, one group received bilateral lesions of the hippocampus ($n = 6$). A second group received bilateral lesions of the PC ($n = 6$). A third group received a sham lesion and served as the control group ($n = 6$).

Histology

After all behavioral testing commenced, rats were anesthetized with a lethal dose of sodium pentobarbital (100 mg/mL ip) and perfused intracardially with 0.9% phosphate buffered saline (pH 6.0) for 2 min followed by 10% buffered formalin (pH 7) for another 5 min. The brains were then extracted and stored in 30% sucrose formalin for 3 days, frozen, and sliced coronally into 40-μm sections with a freezing-stage microtome. All lesioned brains were cut along the coronal plane; every third section was stained with cresyl violet for verification of the lesions.

Apparatus

Allocentric version. The maze used in these experiments was a modified Hebb–Williams maze. The base, which was painted gray, measured 72.6 × 72.6 cm and was made from 1.9-cm thick wood. The walls were 25 cm high and made of .60-cm Plexiglass. A 5-cm black strip was painted along the bottom. Holes were drilled every 15 cm on the floor, creating a grid-like pattern. Four start/goal boxes (13 cm wide, 25 cm long, 17.5 cm high, made of the same wood used to construct the maze) were placed at each corner of the maze. Unlike traditional Hebb–Williams mazes, this maze was made of 1.3-cm Plexiglass and measured 25 cm in height with a 7.5-cm strip, also painted black, placed on the bottom of the barriers. This spatial arrangement allowed the rat to use extra maze cues. Extra maze cues included two posters, a map, and a hanging doll. Given that this maze allows for the use of extra maze cues, it may be considered allocentric.

Egocentric version. The second maze used in these experiments was the same modified Hebb–Williams maze mentioned above; however, the walls were 50.8 cm high, made of .60-cm red Plexiglass. The apparatus was kept in a well-lit room with no windows or extramaze cues. This maze is considered egocentric because the walls were raised, made opaque, and there were few, if any, extra maze cues. An egocentric frame of reference requires inputs from the eyes and position of the head; therefore, the lights were kept on. In addition, the lack of extra maze cues and high, opaque walls did not allow for an allocentric frame of reference, further promoting the use of an egocentric frame of reference.

Training

All rats were handled 3 days postoperatively. This continued for 5 days or longer depending on the activity level of the rat. All rats were kept at 85%–90% of original body weight beginning 1 week prior to testing, and weights were maintained on a 23-hr food deprivation schedule for the duration of testing. The rats were pretrained in a shuttle box. This consisted of training rats to run back and forth in a runway attached to two start/goal boxes. The boxes and runway were lined with 10 Froot Loops (Kellogg's brand), and rats were allowed to roam freely for the first 2 days. On Day 3 the doors to the boxes were introduced, and rats were given Froot Loops at the end of each box. Training continued for another 3 days or until they achieved a short latency (1 s) to run across the runway from start box to goal box.

Testing

Duration of testing varied across days depending on the rats' latency to run; however, testing usually lasted 30–45 min per day. A 45-s intertrial interval was used to allow the experimenter time to clean the maze using a high-power cleaner (HDQ cleaner) so that olfactory cues could be eliminated. Rats received 10 trials per day for 6 consecutive days on Problem 4 (see Figure 1) as described by Rabinovitch and Rosvold (1951). The original maze session consisted of 12 maze configurations, or problems, ranging from easy to difficult. In these tasks, rats ran from one corner of a 4-in. (10.16-cm) high maze to the opposite side navigating through the maze configurations. The original maze contained only two boxes, one start and one goal. The maze used for this experiment had a box placed at each corner of the maze. Error zones in these experiments match the ones used by Rabinovitch and Rosvold (1951). In addition, an error was recorded when an rat entered one of the extra boxes. Three errors zones also were added for this experiment. These zones were recorded as errors only when a rat crossed them on its way back to the start box. Rats were tested until they reached a criterion of less than 10 errors per day across 10 trials; therefore, a rat with a deficit may require many more days of testing to achieve criterion. Nonetheless, after testing culminated, rats were given one, random dark trial to assess the strategy used to solve the maze (e.g., place, response). If the rat made many errors in the dark, it is suggested that the rat used a place strategy. If, however, the rat made few errors, it is suggested that the rat used a response strategy (see Olton, 1979, for review). Errors were recorded with an infrared video camera and scored exactly the same as the 6 previous days of testing.

Data Analysis

Acquisition was assessed by the number of errors made per day across all 6 days of testing. Using a Student-Newman-Keuls test, we analyzed data with a repeated measures analysis of variance (ANOVA) and post hoc analyses when necessary.

Results

Histology

Figure 2A shows a representative, albeit large, PC lesion. The aspiration lesions were complete and consistent across subjects. In most rats, the damage extended to the somatosensory cortex barrel field; however, retrosplenial and motor cortices remained intact.

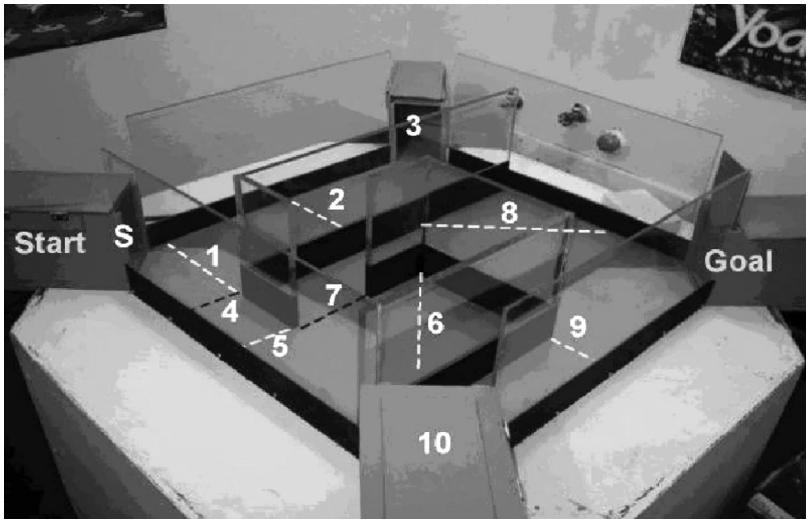

Figure 1. A modified Hebb–Williams maze. Rats were trained on Problem 4 as described by Rabinovitch and Rosvold (1951). Dashed lines indicate error zones.

Figure 2B shows a representative dorsal hippocampal lesion. The excitotoxic lesions tended to be complete and limited to the dorsal component of the hippocampus. There was no damage in control subjects.

Lesions of the Hippocampus but Not the PC Impair Allocentric Maze Acquisition

When lesions were made to the dorsal hippocampus, but not to the PC, an overall deficit in acquisition was observed when tested on the allocentric modified Hebb–Williams maze (see Figure 3). A repeated measures ANOVA was performed on the data with groups as the between-subjects variable and blocks of trials (days) as the within-subjects variable. There was a significant main effect for the groups, $F(2, 13) = 13.4$, $p < .05$, as well as days, $F(5, 65) = 14.7$, $p < .05$. There was no significant interaction between group and days, $F(10, 65) = 1.4$, $p > .05$. Post-hoc analysis (Student-Newman-Keuls test) performed on the group main effect revealed that during acquisition, the hippocampal-lesioned group made significantly more errors than did either the PC-lesioned group or control group ($p < .05$); however, the PC-lesioned and control groups were not significantly different from each other. Dark trial probe data indicate that the control and PC-lesioned groups made many errors, whereas the dorsal hippocampal-lesioned group made few errors, suggesting that the control and PC-lesioned groups used a place strategy, whereas the

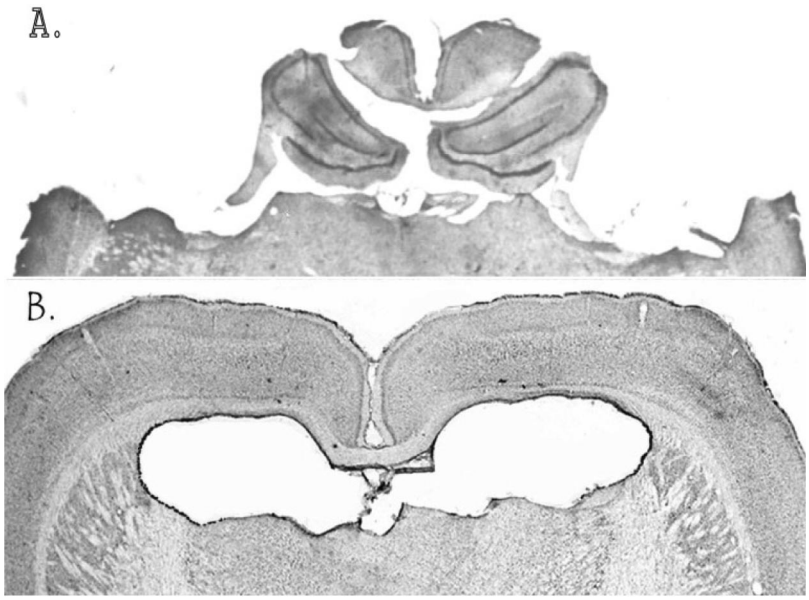

Figure 2. A large but representative parietal cortex lesion (A) and dorsal hippocampal lesion (B).

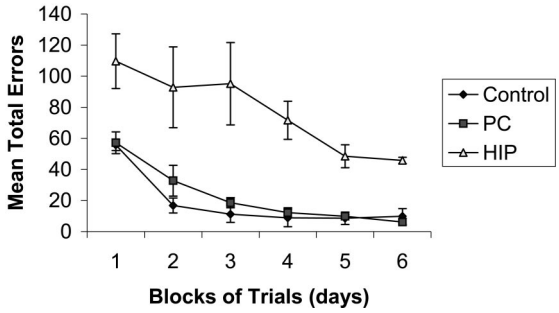

Figure 3. Acquisition of the allocentric version of the maze. Note that the hippocampal (HIP)-lesioned group made significantly more errors than the parietal cortex (PC)-lesioned group or the control group.

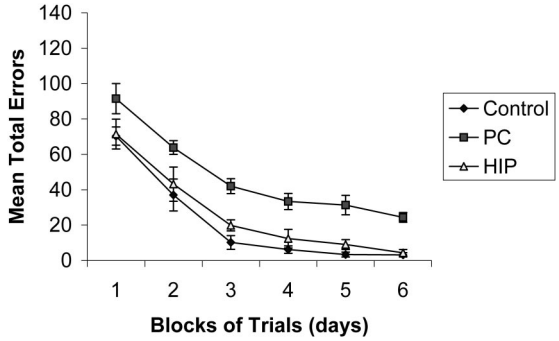

Figure 5. Acquisition of the egocentric version of the maze. Note that the parietal cortex (PC)-lesioned group made significantly more errors than the hippocampal (HIP)-lesioned group or the control group.

hippocampal-lesioned group used a response strategy to solve the maze (see Figure 4). A one-way ANOVA was performed on the dark probe trial data. There was a significant main effect, $F(2, 13) = 177.3$, $p < .05$. Post-hoc analysis (Student-Newman-Keuls test) performed on the dark probe trial data revealed that the hippocampal-lesioned group made significantly fewer errors than did either the PC-lesioned group or control group ($p < .05$). The PC-lesioned and control groups were not significantly different from each other.

Lesions of the PC but Not the Hippocampus Impair Egocentric Maze Acquisition

When lesions were made to the PC, but not to the hippocampus, an overall deficit in acquisition was observed when tested on the egocentric modified Hebb–Williams maze (see Figure 5). A repeated measures ANOVA was performed on the data, with groups as the between-subjects variable and blocks of trials (days) as the within-subjects variable. There was a significant main effect for groups, $F(2, 15) = 14.5$, $p < .05$, as well as days, $F(5, 75) = 112.0$, $p < .05$. There was no significant interaction between group and days, $F(10, 75) < 1.0$, $p > .05$. Post hoc analysis (Student-Newman-Keuls test) performed on the group main effect revealed that during acquisition, the PC-lesioned group made significantly more errors than did either the hippocampal-lesioned or control groups ($p < .05$); however, the hippocampal-lesioned group and control group were not significantly different from each other. Dark trial probe data indicate that the control and hippocampal-lesioned groups made many errors, whereas the PC-lesioned group made few errors, suggesting that the control and hippocampal-lesioned groups used a place strategy, whereas the PC-lesioned group used a response strategy to solve the maze (see Figure 6). A one-way ANOVA was performed on the dark probe trial data. There was a significant main effect, $F(2, 15) = 4.0$, $p < .05$. Post-hoc analysis (Student-Newman-Keuls test) performed on the dark probe trial data revealed that the PC-lesioned group made significantly fewer errors than did either the hippocampal-lesioned group or control group ($p < .05$); however, the hippocampal-lesioned and control groups were not significantly different from each other.

Experiment 1b: Maze Retention

Materials and Methods

Rats

Thirty-one male, Long–Evans rats (Simonsen Laboratories, Gilroy, CA) approximately 4 months of age at the start of the experiment, weighing ~350 g, served as subjects. The rats were housed individually in plastic

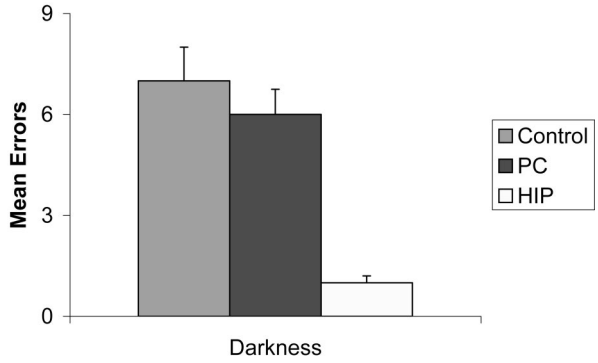

Figure 4. Dark probe trial data indicate that the hippocampal (HIP)-lesioned rats made significantly fewer errors than the parietal cortex (PC)-lesioned rats or the control rats, suggesting that these rats used a response strategy to solve the maze.

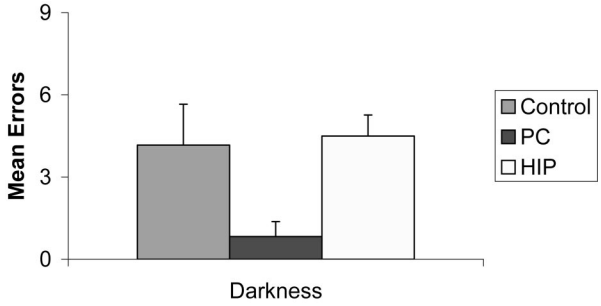

Figure 6. Dark probe trial data indicate that the parietal cortex (PC)-lesioned rats made significantly fewer errors than the hippocampal (HIP)-lesioned rats or the control rats, suggesting that these rats used a response strategy to solve the maze.

tubs located in a colony with a 12-hr light–dark cycle. All rats had free access to water, with food restricted for the duration of testing to maintain each rat at approximately 85%–90% of its free-feeding weight. All testing was conducted during the light portion of the light–dark cycle.

Surgery

Surgical procedures were identical to that described in Experiment 1a. In this experiment there were six groups, three groups per each maze version. On the allocentric version, one group received bilateral lesions of the hippocampus ($n = 5$). A second group received bilateral lesions of the PC ($n = 5$). A third group received sham lesion and served as the control group ($n = 5$). On the egocentric version, one group received bilateral lesions of the hippocampus ($n = 5$). A second group received bilateral lesions of the PC ($n = 5$). A third group received sham lesion and served as the control group ($n = 6$).

Histology

Histological procedures were identical to that described in Experiment 1a.

Apparatus

Apparati were identical to that described in Experiment 1a.

Training

All rats were kept at 85%–90% of original body weight beginning 1 week prior to testing, and weights were maintained on a 23-hr food deprivation schedule for the duration of testing. The rats were pretrained in a shuttle box. This consisted of training rats to run back and forth in a runway attached to two start/goal boxes. The boxes and runway were lined with 10 Froot Loops (Kellogg's brand), and rats were allowed to roam freely for the first 2 days. On Day 3 the doors to the boxes were introduced, and rats were given Froot Loops only at the end of each box. Training continued for another 3 days or until they achieved a short latency (1 s) to run across the runway from start box to goal box.

Testing

Duration of testing varied across days depending on the rats' latency to run; however, testing usually lasted 30–45 min per day. A 45-s intertrial interval was used to allow the experimenter time to clean the maze using a high-power cleaner (HDQ cleaner) so that olfactory cues could be eliminated. Rats were tested for 6 consecutive days on Problem 4 as described by Rabinovitch and Rosvold (1951). After testing, rats were randomly assigned to a surgery group (described above). All rats were handled 3 days postoperatively. This continued for 5 days or longer depending on the activity level of the rat. Following recovery, typically 5–7 days after initial test, all the rats were retested for 6 consecutive days for maze retention. After acquisition and retention testing ended, or after rats had reached criterion (less than 5 errors per day), a probe test was given. A dark probe test assessed the strategy used by the rats to solve the maze. This probe trial was identical to that described in Experiment 1a.

Data Analysis

Acquisition and retention were assessed by the number of errors made per day across all days of testing. Using a Student-Newman-Keuls test, we analyzed data with a repeated measures ANOVA and post hoc analyses when necessary.

Results

Histology

The PC and dorsal hippocampus lesions were comparable with the lesions illustrated and described for Experiment 1a.

Lesions of the Hippocampus and the PC Impair Allocentric Maze Retention

When lesions were made to the dorsal hippocampus or the PC, an overall deficit in retention was observed when tested on the allocentric version of the maze (see Figure 7). A repeated measures ANOVA was performed on the data, with groups as the between-subjects variable and blocks of trials (days) as the within-subjects variable. There was a significant main effect for the groups, $F(2, 12) = 23.9$, $p < .05$, as well as days, $F(5, 60) = 26.1$, $p < .05$. There was a significant interaction between group and days, $F(10, 60) = 4.7$, $p < .05$. Post hoc analysis (Student-Newman-Keuls test) performed on the interaction revealed that during retention, the PC-lesioned group made significantly more errors than the hippocampal-lesioned or control groups ($p < .05$) on Day 1; however, the hippocampal-lesioned group also made significantly more errors than the control group ($p < .05$) on Day 1. Furthermore, both the hippocampal and PC-lesioned groups made significantly more errors than the control group ($p < .05$) on Day 2; however, the hippocampal and PC-lesioned groups were not significantly different. In addition, the PC-lesioned group made significantly more errors than the control group ($p < .05$) on Day 3; however, the PC and hippocampal groups were not significantly different, nor were the hippocampal-lesioned and control groups. Finally, the PC-lesioned group made significantly more errors than the hippocampal-lesioned or control groups ($p < .05$) on Day 4; however, the hippocampal and control groups were not significantly different. None of the groups was significantly different on Days 5 and 6.

Dark trial probe data indicate that the control and hippocampal-lesioned groups made many errors, whereas the PC-lesioned group made few errors (see Figure 8). A one-way ANOVA was performed on the dark probe trial data. There was a significant main effect, $F(2, 12) = 9.3$, $p < .05$. Post hoc analysis (Student-Newman-Keuls test) performed on the dark probe trial data revealed that the PC-lesioned group made significantly fewer errors than did either the control or hippocampal-lesioned groups ($p < .05$); however, the hippocampal-lesioned and control groups were not significantly different from each other.

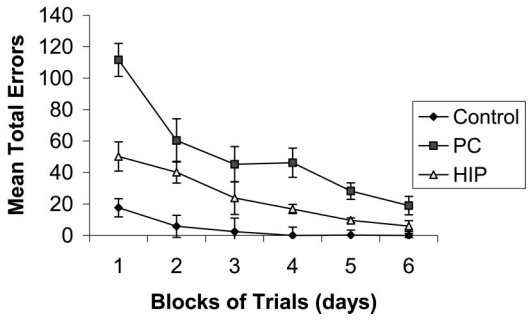

Figure 7. Retention of the allocentric version of the maze. Note that the parietal cortex (PC)-lesioned group made significantly more errors than the hippocampal (HIP)-lesioned group or the control group, whereas the HIP-lesioned group had a short-lived, transient impairment.

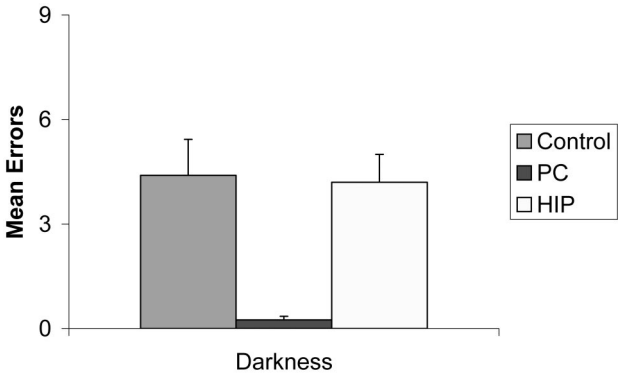

Figure 8. Dark probe trial data indicate that the parietal cortex (PC)-lesioned rats made significantly fewer errors than the hippocampal (HIP)-lesioned rats or the control rats, suggesting that these rats used a response strategy to solve the maze.

Lesions of the PC and Hippocampus Impair Egocentric Maze Retention

When lesions were made to the dorsal hippocampus or the PC, an overall deficit in retention was observed when tested on the egocentric version of the maze (see Figure 9). A repeated measures ANOVA was performed on the data, with groups as the between-subjects variable and blocks of trials (days) as the within-subjects variable. There was a significant main effect for groups, $F(2, 13) = 20.0$, $p < .05$, as well as days, $F(5, 65) = 31.5$, $p < .05$. There was a significant interaction between group and days, $F(10, 65) = 9.9$, $p < .05$. Post hoc analysis (Student-Newman-Keuls test) performed on the interaction revealed that during retention, the PC-lesioned group made significantly more errors than the hippocampal-lesioned or control groups ($p < .05$) on Days 1–5; however, the hippocampal-lesioned group made significantly more errors than the control group ($p < .05$) on Days 1 and 2. Furthermore, the PC-lesioned group made significantly more errors than the control group ($p < .05$) on Day 6; however, the hippocampal-lesioned group and controls were not significantly different after Day 3.

Dark trial probe data indicate that the hippocampal-lesioned and control groups made many errors, whereas the PC-lesioned group made few errors (see Figure 10). A one-way ANOVA was performed on the dark probe trial data. There was a significant main effect, $F(2, 13) = 4.6$, $p < .05$. Post hoc analysis (Student-Newman-Keuls test) performed on the dark probe trial data revealed that the PC-lesioned group made significantly fewer errors than did either the hippocampal-lesioned or control groups ($p < .05$); however, the hippocampal-lesioned and control groups were not significantly different from each other.

Discussion

Two regions of the brain heavily implicated in spatial information processing are the hippocampus and PC. It was hypothesized that lesions of the hippocampus would impair acquisition of the allocentric maze, whereas lesions of the PC would impair acquisition of the egocentric maze. Furthermore, lesions of the PC, but not the hippocampus, would impair retention for both maze versions. The present results indicate a double dissociation during acquisition between the hippocampus and PC for allocentric and egocentric spatial information, respectively. Furthermore, dark probe trial data indicate that during acquisition of the allocentric version of the maze, the hippocampal-lesioned group used a response strategy to solve the maze compared with a place strategy used by the PC-lesioned and control groups. Dark probe trial data also indicate that during acquisition of the egocentric version of the maze, the PC-lesioned group used a response strategy to solve the maze compared with the place strategy used by the hippocampal-lesioned and control groups. Thus, during acquisition, rats that could not learn the maze task—that is to say, made significantly more errors regardless of the maze version (i.e., allocentric, egocentric)—used a response strategy, whereas rats that learned the task, or made fewer errors each day, used a place strategy. Furthermore, the present results show that lesions of the PC significantly impair maze retention, regardless of the maze version. Lesions of the hippocampus result in a short-lived, transient impairment during maze retention, regardless of the maze version; however, there is recovery after a few days. Dark probe trial data indicate that the control and hippocampal-lesioned

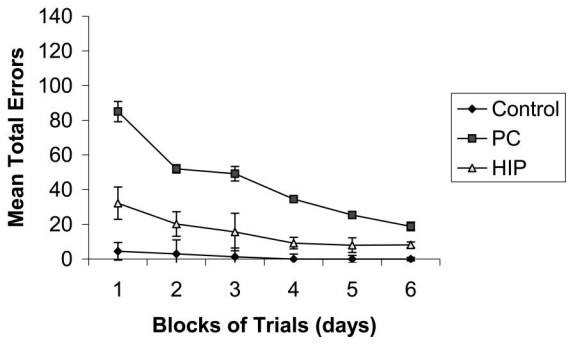

Figure 9. Retention of the egocentric version of the maze. Note that the parietal cortex (PC)-lesioned group made significantly more errors than the hippocampal (HIP)-lesioned group or the control group, whereas the HIP-lesioned group had a short-lived transient impairment.

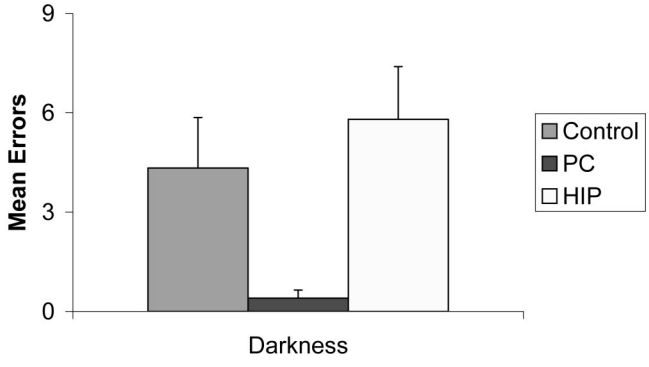

Figure 10. Dark probe trial data indicate that the parietal cortex (PC)-lesioned rats made significantly fewer errors than the hippocampal (HIP)-lesioned rats or the control rats, suggesting that these rats used a response strategy to solve the maze.

groups used a place strategy in both maze versions, whereas the PC-lesioned group used a response strategy in both maze versions.

Histological verification of the PC lesions (see Figure 2A) indicated damage to the somatosensory cortex barrel field. Jucker, Kametani, Bresnahan, and Ingram (1990) showed that vibrissae clipping had no effect on maze performance in rats with a PC lesion, although these rats had an increase in runtime. The present study did not measure runtime during maze testing; thus, barrel cortex damage was not likely to be a factor. It is possible, however, that damage to barrel cortex altered the rat's ability to feel its way during the dark probe trial; however, given that the PC-lesioned rats made few errors on the egocentric version, this is not likely. The fact that lesions of the hippocampus produced a mild impairment during maze retention for both maze versions was not altogether predicted; however, these data support findings by Gilbert and Kesner (2004), who found that lesions of the hippocampus initially impair the retention of object–place associations followed by recovery. The retention data in this study indicate similar results. One potential explanation is that the hippocampus is necessary for the consolidation of spatial information in a manner that supports the multiple memory trace hypothesis (Nadel & Moscovitch, 1997). Initially, the hippocampus is necessary for the consolidation and/or retrieval of spatial information; however, over time and experience, this information gets extracted out of the hippocampus into higher cortical areas, such as the entorhinal cortex. Along these lines, were the rats to receive 4 weeks between acquisition and retention, perhaps the hippocampus would no longer be necessary for maze retention.

In the context of the models presented above, the present findings are most consistent with the multiple attribute model. As such, the present results suggest that the hippocampus and PC process spatial information in parallel; however, the PC, not the hippocampus, plays a critical role in the retention of spatial information. These data further support Nadel and Moscovitch (1997), who suggest that the hippocampus serves to store spatial information initially followed by reassignment to more temporal lobe structures, such as the entorhinal cortex (see also Nadel, Samsonovitch, & Moscovitch, 2000). Such structures could serve to store spatial information for intermediate-term memory usage, thus allowing the PC to create a "grand" cognitive map, integrating allocentric and egocentric spatial information.

The findings from the dark probe trials suggest that rats exhibiting a deficit can solve the maze using a response strategy. Historically, transfer tests are used to elucidate a response strategy—that is to say, the relation between body turns, body position, and the rat's location, from a place strategy, on the basis of the cues in the environment independent of the rat (Olton, 1979). Although the present experiments concentrated on the place aspect of maze learning, data suggest that the striatum mediates a response strategy (Packard & McGaugh, 1996; for review, see Kesner & Rogers, 2004). Normal rats make many errors on the Hebb–Williams maze when tested in the dark, suggesting the use of a place strategy; however, they switch to a response strategy after a few dark trials. Thus, both strategies exist for the rat's use, although the place strategy is used before the response strategy. Along these lines, Packard (1999) demonstrated that on a cross-maze, rats preferred a place strategy initially, followed by a shift to a response strategy later in training. One could suggest, therefore, that a place strategy is preferred to a response strategy. The use of a response strategy by the PC-lesioned rats during retention of both maze versions could reflect the "binding" properties of the PC for long-term storage of spatial information. Kesner and Long (1998) have suggested that the PC stores long-term memory for spatial information in the form of a cognitive map. According to this view, the PC integrates (a) egocentric spatial information and (b) proximal and/or kinesthetic information with allocentric spatial information, which is cue-based information encoded by the hippocampus. This view is consistent with Treisman's feature integration theory (Treisman & Gelade, 1980), which suggests that intact parietal function is essential for binding object features with spatial information, such as a "master map of locations" (Robertson, Treisman, Friedman-Hill, & Grabowecky, 1997, p. 296) or a cognitive map. Integration of egocentric and allocentric spatial information occurring in the PC explains the deficits seen during retention of both maze versions with lesions of the PC. Furthermore, PC-lesioned rats used a response strategy during maze retention, whereas control and hippocampal-lesioned rats used a place strategy, further implicating the PC in the integration of egocentric and allocentric spatial information for long-term storage. The initial transient deficits observed with hippocampal lesions during maze retention, consistent with Gilbert and Kesner (2004), could reflect a retrieval, or access, process occurring in the hippocampus.

Furthermore, Takehara, Kawahara, and Kirino (2003) found that lesions of the hippocampus disrupted retention of eyeblink conditioning 1 day but not 4 weeks after training. Izquierdo et al. (1997) demonstrated that the glutamate antagonist, AP-5, or the gamma aminobutyric acid agonist, muscimol, produced retrograde amnesia when injected into the hippocampus immediately after (0 min) inhibitory avoidance training. These authors reported retrograde amnesia for PC injections after 180-min posttraining. The alpha-amino-3-hydroxy-5-methylisoxazole-4-propionic acid (CNQX) antagonist produced a retrieval deficit when injected into the hippocampus 1 or 31 days posttraining, whereas CNQX produced a retrieval deficit in the PC 1, 31, or 60 days posttraining. Finally, using three recognition memory tasks, Shannon and Buckner (2004) reported that retrieval success was reflected by activity (measured via functional magnetic resonance imagining) in the posterior PC. These data suggest that the hippocampus plays a role in the retrieval of spatial information; however, this spatial information is not stored in the hippocampus but perhaps the PC. Taken together, the multiple attribute model, the current literature cited above, and the data from the present experiments suggest that the hippocampus and PC operate in parallel during acquisition of spatial information; however, during retention, there could be interactions with the PC that are important for the binding of spatial information and with the hippocampus that are important for the initial access of spatial information.

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