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Reversible Inactivation of the Hippocampal Formation in Food-storing Black-capped Chickadees (*Poecile atricapillus*)

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ABSTRACT: The role of the hippocampal formation (HF) in memory processing was assessed in food-storing black-capped chickadees (*Poecile atricapilla*) by reversibly inactivating the HF during different memory tests. The memory tests required birds to remember a location based on spatial cues only, or based on a combination of both spatial and distinct visual cues. Inactivation of the HF impaired short-term spatial memory, but not visual-spatial memory. Inactivation of the HF impaired the retrieval of short-term (15 min) spatial memories, but not long-term (3-h) spatial memories. The pattern of deficits produced by inactivation of the HF in chickadees suggests a possible function of the hippocampal specialization of food-storing birds, as well as extends the notion of functional homology between the avian and mammalian HF. *Hippocampus 2003;13: 437–444.* © 2003 Wiley-Liss, Inc.

KEY WORDS: food storing; hippocampus; chickadee; spatial memory; reversible inactivation; lidocaine

INTRODUCTION

Food-storing birds, of which the most extensively studied are the parids (tits and chickadees) and corvids (jays and nutcrackers), create scattered food caches and use memory to retrieve their caches (Cowie et al., 1981; Shettleworth and Krebs, 1982; Balda and Kamil, 1989). Comparisons of the behavior of food-storing species and closely related non-storing species on memory tasks demonstrate that species that store food have a specialized memory for spatial locations (Brodbeck, 1994; Clayton and Krebs, 1994; Shettleworth, 1995; Hampton et al., 1998; Biegler et al., 2001). Likewise, the hippocampal formation (HF), known to be important for spatial memory in birds as well as mammals, is relatively larger in food-storing species than non-storing species (Krebs et al., 1989; Healy and Krebs, 1992, 1996; Hampton et al., 1995). Furthermore, with the onset of hoarding behavior in the fall, the HF in black-capped chickadees (*Poecile atricapilla*) increases in volume (Smulders et al., 1995). This seasonal change in volume is attributable to a net addition of cells in the HF (Smulders et al., 2000). An increase in cell number in the HF may result from increased survival of newly generated neurons in the fall (Barnea and Nottebohm, 1994, 1996).

The HF of food-storing birds therefore appears specialized for the spatial memory demands imposed by caching behavior. However, the phases or features of memory processing for which the HF is necessary remain unclear. Lesions of the HF in black-capped chickadees impair cache recovery (Sherry and Vaccarino, 1989) as well as memory for spatial locations (Hampton and Shettleworth, 1996). However, with this technique, it is not possible to determine whether the observed memory deficit is a result of a failure to acquire, store, or retrieve memory. In black-capped chickadees, immediate-early genes (IEG) are expressed in the HF while the bird stores as well as retrieves food caches (Smulders and DeVoogd, 2000). This finding suggests that the HF is active during both the acquisition and retrieval of memory for spatial locations. In the present experiments, we aim to specify in greater detail the phases or features of memory for which the HF in food-storing birds might be involved. To do this, we functionally inactivate the HF in a food-storing bird by infusing lidocaine during different memory tests. By temporarily impairing neural function, reversible inactivation provides a means of dissociating the different phases of memory processing for which a particular brain area might be involved. If the HF is involved in processing spatial information, we predict that inactivation of the HF will impair performance on a spatial memory task, but spare performance on a task employing both visual and spatial cues, as the use of spatial cues is not necessary to solve this task. If the HF has a time-limited role in the retrieval of memory, we predict that inactivation of the HF will impair retrieval of short-term but not long-term memories.

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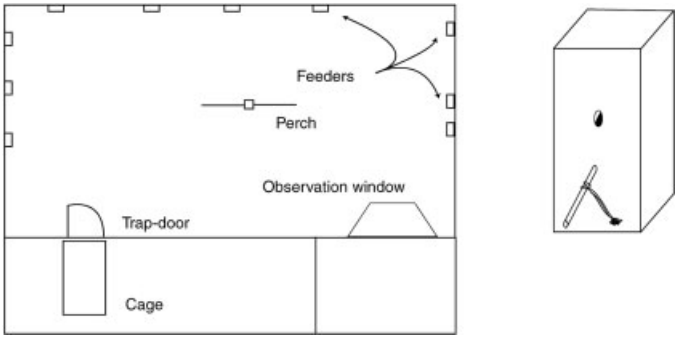

FIGURE 1. Schematic of the testing environment. Birds entered the testing arena from their cage through a trapdoor. Ten wooden feeders (see inset) were mounted on three walls in a pseudo-random distribution. A perch made of hardware cloth was positioned in the center of the room. Observations were made through a Plexiglas window.

MATERIALS AND METHODS

Subjects

Five black-capped chickadees were caught near Ithaca, New York, under state and federal permits. Three birds were caught in early July, and an additional two birds were caught in September. Experiments were carried out from July through the end of September. Subjects were fed mealworms and a mix of ground beef, carrot baby food, hardboiled eggs, wheat germ and turkey pellets. Water was provided ad libitum. Subjects were housed in 45.7 × 21.9 × 24.4-cm wire cages and kept on a 12/12-light/dark cycle.

Testing Environment

The experiment was carried out in a 4.5 × 4 × 2.5-m room (Fig. 1). The room contained a single wooden stand with a 92-cm × 142-cm vertical hardware cloth screen attached, which the birds used as a perch. The wooden stand was centered in the room equidistant from the walls. Three of the walls of the testing room were covered with hardware cloth stretching from the ceiling to 1 m above the floor, upon which feeders could be hung. The feeders consisted of 9-cm × 11.5 cm × 4-cm wooden blocks with a wooden dowel 2 cm from the base, and an 8-mm deep hole 3 cm above the dowel. If a feeder was baited, one-half of a mealworm was placed inside the hole. To prevent the bird from casually observing whether a feeder was baited, all feeders were fitted with a string attached to the dowel, the end of which was knotted and covered the hole. For the spatial memory task, all feeders were painted green; for the visual-spatial memory task, each feeder was painted in individual colors and patterns such that no two feeders appeared similar. The birds entered the room from their home cages through a trapdoor in the wall. The experimenter operated the trapdoor through a pulley system. Observation of the bird's behavior took place through a window of darkened Plexiglas.

Pre-training

Birds were pre-trained to enter the test room when the trap door was raised, to search for mealworms by pulling knots from feeders, and to leave the test room and return to the home cage when the test room lights were turned off. During pre-training five green feeders were present in random locations in the testing room and each contained one-half of a mealworm. When the birds removed worms from all five feeders, the feeders were re-baited, and the birds were again allowed to retrieve worms. This training continued until birds reliably visited all five feeders.

Surgery

At 1–3 days after pre-training concluded, the birds were anesthetized (5mg/kg Xylazine, and 87.5 mg/kg ketamine, injected intramuscularly). Birds were placed in a stereotactic apparatus and the coordinates of the site where the midline meets the cerebellum were determined. A rectangular section of skull spanning the midline was removed 2 mm rostral to this reference site. Two 4-mm-long stainless steel cannulae (26 gauge, Small Parts) were implanted within the rectangular window, through a slit in the dura mater, 0.6 mm on either side of the midline. The cannulae were secured to the skull with cyanoacrylate glue (ZAP/CA) and embedded in a dental cement platform (Perm Reline and Repair Resin). The scalp was closed around the platform and secured with cyanoacrylate glue. Birds were allowed 2 days to recover.

Memory Test

In the testing situation, 10 feeders were placed in new locations based on a pseudo-random assignment. The testing room was divided into five regions and, within each region, two feeders were assigned to random locations. We randomly baited 1 of the 10 feeders in the room and released the bird into the testing arena. Before testing the bird, we deprived it of food for 2 h. When the bird found the baited feeder, it was allowed to eat the mealworm, after which the lights in the testing room were turned off and the bird returned to its cage for 1 min. While the bird remained in its cage, we rebaited the feeder that previously contained the mealworm; we replaced any knots the bird had removed from this and other feeders while it had searched for the bait.

After remaining in its cage for 1 min, the bird reentered the arena and again searched for the bait. The same process was repeated five times, such that birds entered and searched the arena on five consecutive occasions with a 1-min interval between entries. The same feeder was baited for each of the five entries. Because the bird has to rely on information learned from previous entries into the arena, this can be considered a reference memory task. Birds were tested on two variants of the memory task (Fig. 2). In the spatial memory task, each of the 10 feeders were identically colored. Birds therefore had to rely on positional information to encode the location of the baited feeder. In the visual-spatial memory test, each of the 10 feeders differed in color and paint pattern. Birds therefore could use both positional as well as proximate visual cues associated with the feeder to encode the location of the baited feeder.

For all entries into the arena, the number of feeders the bird visited, including the baited feeder, was recorded by hand. Only unique visits were scored. Rechecks to feeders that had already been visited were not counted because once a bird had removed a knot from a feeder it was not possible to determine whether the

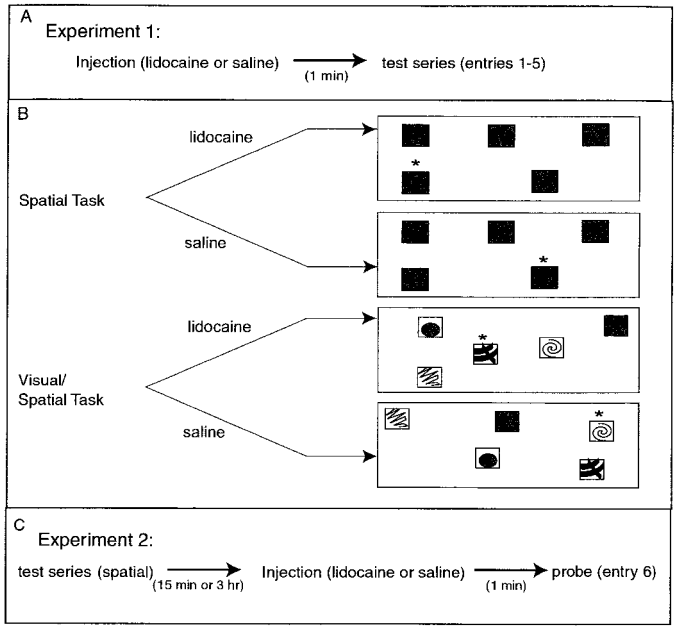

FIGURE 2. Schematic of the experimental design. A: In Experiment 1, infusions of lidocaine or saline preceded the memory test. B: The two memory tests in experiment one involve remembering either spatial or spatial and visual components. In the purely spatial memory test, each feeder was the same color. Each feeder remained in the same location for every test series, while the location of the baited feeder (marked with an asterisk) changed between the various test series. In the visual-spatial memory test, each feeder was a different color and paint pattern, and the locations and identity of the baited feeder changed between test series. A set of noninfusion spatial test series was interleaved with the infusion test series (see Materials and Methods). The same array of feeders was used for both the infusion and noninfusion spatial test series. C: In Experiment 2, the memory test was followed by a delay of 15 min or 3 h, after which birds received an infusion or lidocaine or saline, followed by a single probe entry into the testing arena.

bird was rechecking an already visited feeder or merely perching at the feeder. If a bird visited the baited feeder before any other feeders on the first entry, then the trial was discarded, since it is not possible for the bird to demonstrate any improvement across entries. For an unimpaired bird, the number of feeders it visits for each entry into the arena should decrease from the first entry to the fifth in the series, as the bird should learn the location of the baited feeder and use its memory to guide its subsequent searches.

Temporary Inactivation

Birds were removed from their home cages, restrained by hand, and a Hamilton syringe (32 gauge, 5 μl) attached to a stereotactic apparatus was inserted into each cannula. Approximately 0.1 μl of 2% Lidocaine (Radix) was infused gradually over the course of 1 min. The needle was left in the cannula for an additional minute. Birds were then returned to their home cages for 1 min, after which they were released into the arena. In the control condition, birds were infused in the same manner with 0.8% saline.

Experiment 1: Short-Term Spatial or Visual-Spatial Memory

In Experiment 1, birds performed in a spatial memory test, or a visual-spatial memory test as described above. At 1 min before beginning the memory tests, birds were infused with lidocaine or a saline control and then were exposed to the feeder array five times in rapid succession as described above. Subjects performed a total of four such test series in this experiment (spatial or visual-spatial after saline or lidocaine infusions). Each experimental test series was run on a separate day, at least 2 days apart from the other experimental test series. Birds were allowed 15 min to complete a test series. We randomly determined the order of tests (visual-spatial vs. spatial tests) and infusate (lidocaine vs. saline) for each bird. On days between the experimental (infusion) test series, we conducted a spatial test series with no infusions. These noninfusion test series were included to minimize any effects of the previous testing experience on the bird's test performance in subsequent experimental tests. The randomized order of tasks and infusions for each bird, as well as the noninfusion test series interleaved between infusion test series, make any effect of treatment order on task performance unlikely.

Experiment 2: Short-Term Versus Long-Term Spatial Memory Retrieval

In Experiment 2, we provided birds (the same birds from experiment 1) with 10 identically colored feeders in an arrangement not used previously. We baited one feeder, and birds performed a single test series of five consecutive entries into the arena, identical to the spatial memory test in experiment 1, thereby learning the location of the baited feeder. After the five entries, birds remained in their cages for a delay of either 15 min (short-term) or 3 h (long-term). After the delay, we inactivated the HF with infusions of lidocaine or we infused saline. At 1 min after receiving the infusion, birds reentered the arena. Before the bird's sixth entry into the arena, we replaced any knots the bird removed during its previous entry and rebaited the feeder.

Subjects performed a total of four experimental test series (two delay conditions × lidocaine or saline infusions). Each experimental test series was run on a separate day at least 2 days apart from others. We randomly determined the order of tests (short-term or long-term) and infusions (lidocaine vs. saline) for each bird. Between days on which we infused birds, we ran birds on a spatial test series with no infusions.

Histology

Two weeks after the end of the second experiment, the birds were infused with 0.1 μl of 2% fluorescein-labeled dextran, in the same manner as the lidocaine infusions. The birds were then anesthetized with ketamine/xylazine and were perfused transcardially with 0.1 M sodium phosphate-buffered saline (PBS), followed by 10% formalin. The brains were removed from the skulls, fixed in formalin for 1 h, and transferred to 30% sucrose/10% formalin for 24 h. Brains were then embedded in 10% gelatin/30% sucrose and cut at 40 μm on a freezing microtome. Every other section was examined on a microscope with fluorescent optics to determine the

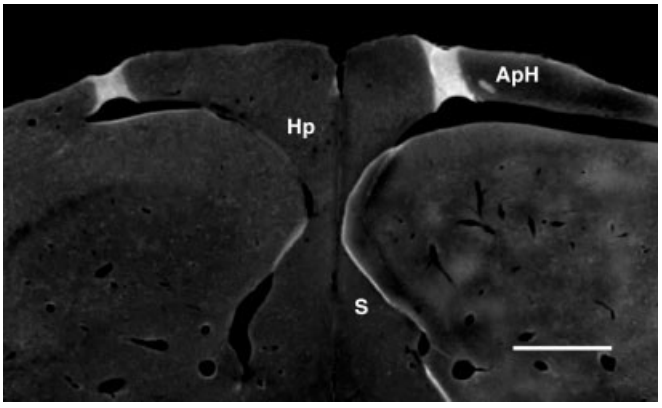

FIGURE 3. Distribution of fluorescein-labeled dextran infusions for one subject. Fluorescence appears in the right hippocampal formation (HF) and lateral to the HF in left hemisphere. With a few exceptions, fluorescence from the labeled dextran amines was confined to the hippocampal formation (which consists of the hippocampus proper and the area parahippocampalus). ApH, area parahippocampus; Hp, hippocampus; S, septum. Scale bar = 1 mm.

location and spread of fluids from the cannula infusions. Alternate sections were stained with cresyl violet and were coverslipped and examined with a light microscope.

Statistics

To compare a subject's performance for different test series within a treatment condition as well as across treatment conditions, we used a repeated-measures general linear model analysis of variance (ANOVA) and paired *t*-tests. The dependent variable for all experiments was the number of feeders the bird visited. When examining performance on the spatial and visual task, the fixed factors in the GLM equation were entries (1–5) and Drug (lidocaine or saline). When examining the retrieval of spatial memory, paired t-tests were performed for the different delay conditions comparing performance on the probe after lidocaine or saline infusions.

RESULTS

Histology

Fluorescence from the labeled dextran amines could be detected within the HF in every animal (Fig. 3). The positions of cannulae were identified in cresyl violet-stained sections; they revealed small amounts of tissue damage (Fig. 4). In two birds, fluorescence and corresponding cannulae positions were found lateral to the left HF.

Effects of Hippocampal Inactivation on Short-term Spatial and Visual Memory

A total of five birds were tested in the spatial task. In this task, feeders were the same color such that birds had to rely on spatial cues to remember the location of the baited feeder. There was an overall tendency for birds to improve their performance across entries ($F_{4,16} = 8.74$; $P < 0.001$), however no main effect of drug ($F_{1,4} = 2.21$; $P = 0.21$). There was a significant entry × drug interaction ($F_{4,16} = 9.56$; $P < 0.001$) (Fig. 5A), indicating that improvement on the spatial task occurred after saline infusions, but not after lidocaine infusions.

Five birds were tested in the visual-spatial task, in which each feeder was painted a unique pattern of colors. For birds in the visual-spatial task, there was an overall tendency to improve performance ($F_{4,16} = 11.46$, $P < 0.001$), but no main effect of drug was found ($F_{1,4} = 0.83$, $P = 0.41$). Unlike the spatial task, there was no entry × drug interaction ($F_{1,4} = 2.54$, $P = 0.08$). (Fig. 5B) Thus inactivation of the HF in food-storing birds did not impair the acquisition of a task that can be solved with visual patterns as well as spatial memory.

Effects of Hippocampal Inactivation During the Retrieval Process

Five birds received a single spatial test series of five consecutive entries, followed by a delay of either 15 min or 3 h. For subjects in both the 15-min delay condition and the 3-h delay condition,

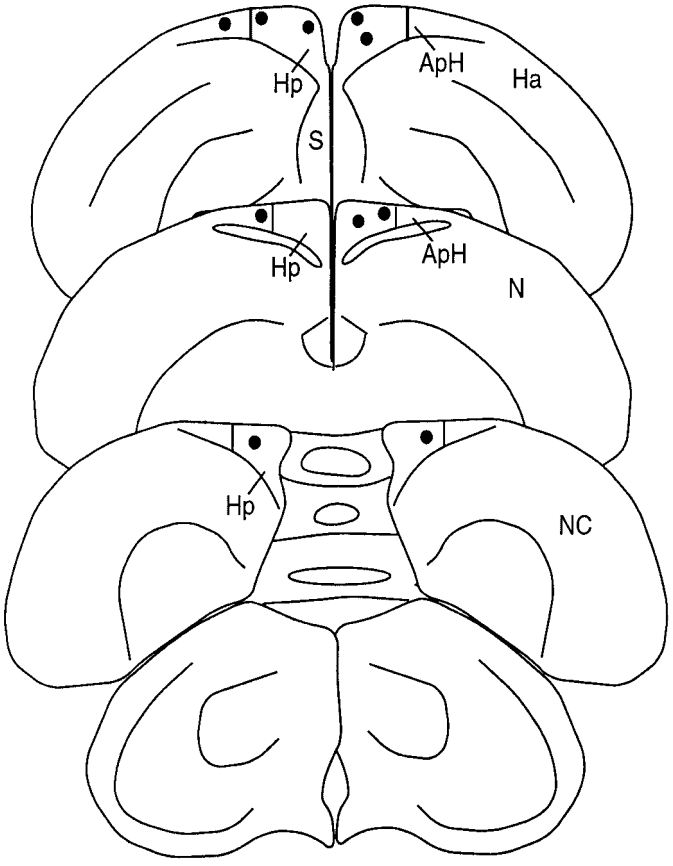

FIGURE 4. Position of cannulae. Black circles represent position of cannulae tips for different subjects. The position of cannulae was mainly confined to the hippocampal formation, which consists of the area parahippocampus (ApH) and hippocampus proper (Hp). Ha, hyperstriatum accessorium; N, neostriatum; NC, neostriatum caudale; S, septum. Sections correspond roughly with Karten and Hodos. 1967. templates A6.75, A5.75, and A4.00, rostral to caudal.

performance improved across the five initial entries preceding the drug infusion (15-min delay: $F_{4,16} = 8.88$; $P < 0.001$; 3-h delay: $F_{4,16} = 8.44$; $P < 0.001$), which demonstrates that birds, when performing in either condition, learned the location of the correct feeder before infusion. After the delay, birds received infusions of either saline or lidocaine and then reentered the arena. On this probe trial after the infusion, an effect of drug was found for the 15-min delay group ($t_4 = 3.674$, $P = 0.02$), but no effect of drug was found for the 3-h delay group ($t_4 = 0.59$, n.s.) (Fig. 6).

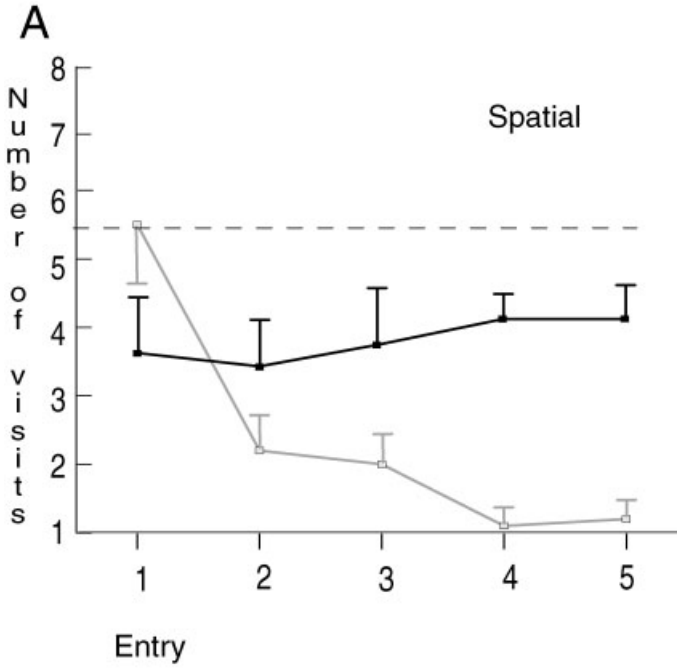

FIGURE 5. Performance on the spatial task (A) and the visual-spatial task (B). The number of feeders visited (±SEM) is plotted across five consecutive entries into the arena for subjects infused with saline (gray) or lidocaine (black). The hatched horizontal line indicates chance performance on the task. Lidocaine-treated birds do not improve their performance unless the task can be solved by visual as well as spatial cues.

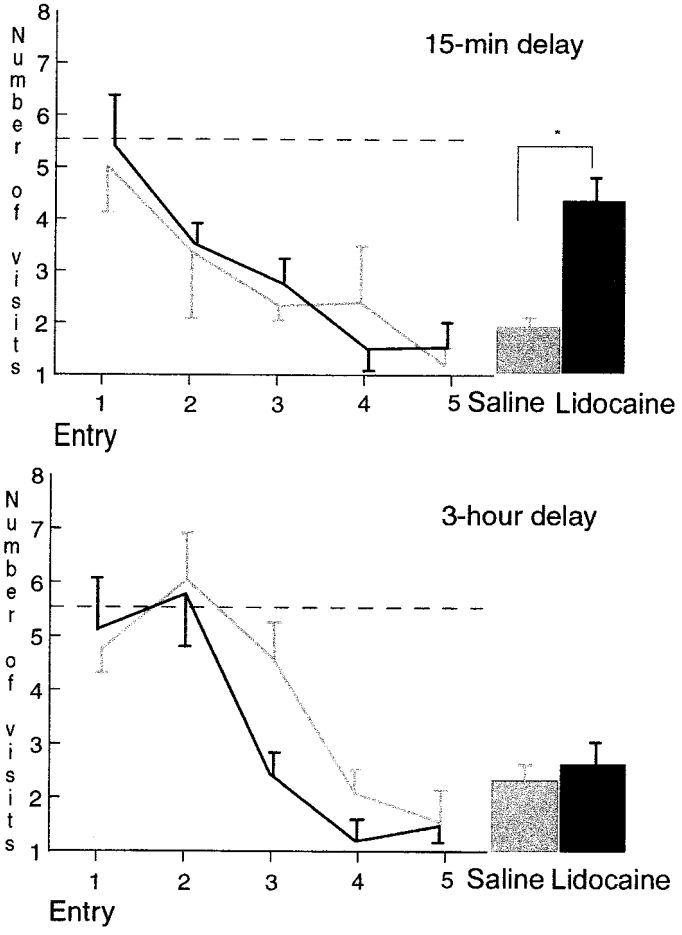

FIGURE 6. Retrieval of short-term and long-term spatial memories: The number of feeders visited (±SEM) is plotted for the two infusion conditions. For the five trials leading up to the infusion, both saline (gray) and lidocaine (black) show a decrease in the number of feeders visited in both the 15-min retrieval (A) and 3-h retrieval (B) conditions. The bar graph on the right represents the number of feeders visited during the probe when subjects were treated with lidocaine (black bar) or saline (gray bar). In the 15-min retrieval condition, a significant difference was found between lidocaine and saline-infused subjects on the number of feeders checked during the probe entry. In the 3-h retrieval condition, no difference was found between groups. The hatched horizontal line indicates chance performance on the task.

DISCUSSION

We have found that inactivation of the HF with lidocaine in food-storing black-capped chickadees impairs the acquisition and short-term retrieval of spatial memories. Inactivation of the HF does not affect performance on memory tasks containing a visual cue component, nor does it affect retrieval of long-term spatial memories. Furthermore, the impairment in lidocaine-infused

birds is not caused by any disruptive effects of the infusion procedure itself, but by the pharmacological action of the lidocaine. Lidocaine uniformly blocks Na^+ channels; therefore, both local circuit-based and fibers of passage through the HF might also be disrupted with our treatment. We therefore cannot distinguish our results between the effects of lidocaine on altered synaptic integration within the HF and altered communication with other brain regions. Drugs targeting synaptic transmission may provide a more precise means of assessing HF function.

We have shown that inactivation of the HF immediately before memory retrieval impairs retrieval of short-term (15-min) memories. However, HF inactivation does not impair the retrieval of long-term (180-min) memories. These results suggest that short-term retrieval of spatial memories is dependent on the HF and that over a longer duration, they become independent of the HF. That 180 min is a sufficient period for memory consolidation is somewhat surprising, as longer delays are typically necessary for memory consolidation to occur in mammals. The rapid consolidation of memories may be a feature unique to birds. Alternatively, after 180 min memories may still reside in the avian HF but over time become more robust and less susceptible to our technique of partial inactivation of the HF. In either case, some sort of consolidation of the memory trace occurs within the longer delay.

The spared performance of chickadees on the visual-spatial task after HF inactivation eliminates a number of alternative explanations for our results. Specifically, the observed deficit in the spatial task is not due to a loss of response inhibition (Hazeltine et al., 2000), which would be the case if birds could not inhibit the behavior of pulling knots when presented with feeders. Furthermore, the lack of impairment in the visual-spatial memory test argues against HF inactivation causing a generalized deficit in acquiring memory, or in motivation to perform accurately. The addition of visual cues prevented the lidocaine-induced memory impairment observed in the spatial memory task, which suggests that the observed deficit in learning is specific to spatial memory. However, since birds could still be using positional as well as visual information to encode the location of the baited feeder during the visual-spatial memory task, it is possible that the spared performance during the visual-spatial memory task represents spared spatial ability rather than the workings of a hippocampal-independent visual memory system.

Another interpretation of our results is that the HF is involved in processing information about the relationships between landmarks to allow navigation, without involvement in the process of storing or retrieving memories about these relationships (Bolhuis et al., 1994). The memory acquisition task does not rule out a general navigational deficit without a memory component, since the differently colored feeders could act as beacons, making integration of spatial landmarks unnecessary for this task. However, this concern is addressed by our memory retrieval task. The lack of impairment after 180 min rules out the idea that the HF is involved in a purely navigational process.

Our results show that in the spatial task, lidocaine-infused birds were performing below chance on their first entry into the arena. Lidocaine infusions could not make birds better at guessing where the bait was hidden, since they had no basis for knowing where the bait was located on their first entry into the arena. Given the small number of subjects (five), we believe this result arose by chance. More important though is that the lidocaine-infused birds show no improvement with each entry, whereas in every other condition birds are showing a decrease in the number of feeders checked with each entry, and rapidly out-perform the lidocaine-infused birds.

A comparison of the behaviors mediated by the HF in birds and mammals suggests that these structures are functionally homologous (Lee et al., 1998; Colombo and Broadbent, 2000). In both birds and mammals, the HF is necessary for tasks involving spatial memory (Morris et al., 1986; Squire, 1992; Bingman et al., 1995; McDonald and White, 1995; Bohbot et al., 1996; Colombo et al., 1997a; Fremouw et al., 1997; Strasser and Bingman, 1999). In both birds and mammals, tasks involving the processing of visual information appear to be independent of hippocampal function (Good and Macphail, 1994; McDonald and White, 1994, 1995; Packard and McGaugh, 1996; Colombo et al., 1997b). In mammals, the HF is differentially involved in different phases or features of memory. Acquisition of spatial memories (Poucet et al., 1991; Lassalle et al., 2000), as well as retrieval of short-term (working) memory (Poucet and Buhot, 1994; Moser and Moser, 1998; Riedel et al., 1999), is dependent on hippocampal function while long-term memory retrieval is not dependent on the HF (Zola-Morgan and Squire, 1990; Squire, 1992). In the present study, the pattern of deficits we find after reversible inactivation of the HF is similar to those found in similar studies in mammals and therefore further support the functional homology of the HF between birds and mammals.

Food-storing birds have an HF that is proportionately larger than in non-storing birds. In food-storing black-capped chickadees the HF shows seasonal plasticity with increased cell numbers and volume in the fall (Smulders et al., 1995, 2000). Given that the seasonal onset of neurogenesis appears to precede hoarding behavior (Smulders et al., 2000), neurogenesis in the HF likely functions to facilitate new learning as opposed to encoding the events that may have caused neurogenesis (Martin et al., 2000; Shors et al., 2001). A larger HF in the fall (when caches are hoarded), but not in winter (when they are retrieved), may therefore facilitate the learning or consolidation of memories associated with many cache locations, as opposed to their long-term storage or retrieval.

Our results suggest that the HF in food-storing birds does not support the retrieval of long-term spatial memories. The observation by Smulders and DeVoogd (2000) of elevated levels of IEG expression in the HF being associated with greater accuracy of cache retrieval by black-capped chickadees does not necessarily contradict our findings. As mentioned above, it is possible that the HF is still involved in the retrieval of long-term memories, but that the memory trace becomes more resistant to our technique of partial inactivation of the HF. Another possibility is that procedural differences between the present study and Smulders and DeVoogd (2000), who tested chickadees in a food-hoarding task, resulted in differential hippocampal involvement during memory retrieval. In a hoarding situation, animals create their own caches, and they can therefore learn the location of a cache after only one encounter. Furthermore, during retrieval of natural caches, animals should avoid revisiting locations from which they have previously harvested caches. In our task, we provided subjects with five separate episodes in which to learn a location, and they were

trained to revisit the same location. It is possible that the hoarding situation, which resembles a working-memory paradigm, and the task used here, which resembles a reference memory paradigm, placed different demands on the hippocampus during memory retrieval.

Our results suggest the HF in food-storing black-capped chickadees functions during short-term learning and retrieval of spatial memory and may have a role in the process of consolidation of short-term memories to long-term memories. Whether the process of memory consolidation in mammals and birds involves the same mechanisms is not known. However, the similarities between hippocampal function in birds and mammals suggest a functional homology between the two structures. With respect to food-storing birds, the enlarged HF, as well as the seasonal fluctuation in hippocampal volume, may act to facilitate the learning and/or consolidation of memories associated with cache locations, as opposed to their long-term storage or retrieval.

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