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Short Communication

Reversible inactivation of the lateral dorsal thalamus disrupts hippocampal place representation and impairs spatial learning

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Abstract

Place-specific discharge of hippocampal cells was monitored while rats performed daily 15 trials of a spatial memory task. During the intertrial interval between trials 5 and 6, the lateral dorsal nucleus of the thalamus (LDN) was reversibly inactivated. Choice accuracy on the maze became impaired, and many hippocampal place fields became disrupted. These data support the proposition that the LDN passes onto hippocampus important (spatial) information that is used for accurate maze navigation.

Key words: Lateral dorsal thalamus; Hippocampus; Place cell; Spatial learning

Several lines of evidence support the hypothesis that the hippocampus importantly contributes to normal spatial learning. For example, lesion of part or all of hippocampus produces impaired performance on spatial navigation tasks [14,21,27]. Also, a prominent behavioral correlate of hippocampal cell discharge is the location of an animal in its environment [6,15,16,18,20,23]. Such cells are referred to as place cells, and the portion of the environment that elicits elevated discharge is referred to as the cell's place field. The fact that hippocampal cells code location presumably reflects the nature of spatial information processing accomplished by hippocampus. Although the computational power of specific hippocampal subregions is under intense study in many laboratories [7,17,24], the precise nature of the association represented by a place field remains unknown.

It has been argued that one factor which likely contributes to the uncertainty regarding the precise significance of hippocampal place fields is the relative lack of knowledge about the nature of the afferent information [12]. Therefore, given the known visual sensitivity of hippocampal place cells [4,15,19], the nature of visual input to hippocampus by one of two visual pathways, the tectocortical system, was evaluated [12]. It was found that neurons in the lateral dorsal nucleus of the thalamus (LDN), which provide extensive afferent input to the subicular complex of the hippocampal formation [26], selectively discharge when animals align their heads with particular directions in space. Such directional firing was shown not to be solely dependent on intramaze cues, geomagnetic information, or specific motor acts. Rather, the specificity of the directional firing was experience-dependent, and therefore postulated to reflect associations between visual and nonvisual (perhaps vestibular) sensory information. Location-specific firing such as that observed in hippocampus was not observed in the LDN. The authors hypothesized that LDN directional information may provide hippocampal structures with preprocessed visual directional input that becomes associated with other neocortical afferents to result in the directional place fields observed in hippocampus proper. Taube and colleagues [25] reported that neurons in the postsubicular region of the hippocampal formation code *both* head direction and location information. The postsubiculum may thus be an important integrator of LDN and neocortical spatial information.

If the LDN provides hippocampus with spatial information that is necessary for normal place representation, removal of LDN input should disrupt hippocampal place fields. Also, since the integrity of at least some subpopulations of hippocampal place cells is essential for normal performance on a spatial maze task

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[9], LDN deafferentation of hippocampus should also produce a spatial learning impairment. The present study tested these two hypotheses by evaluating concurrently the effects of LDN inactivation on place representation in hippocampus and on hippocampal-dependent spatial learning.

Upon arrival in the laboratory, male Fischer-344 rats (9-month-old retired breeders) were given free access to food and water for 2–4 wk before maze training, at which time animals were maintained at about 80% of their ad libitum body weights. Lights were on in the colony room from 07.00 to 19.00. Behavioral testing occurred between 12.00 and 18.00. A semi-automated 8 arm radial maze [9] was used to evaluate spatial learning. The maze consisted of 8 alleys, or arms (58.0 cm by 5.5 cm), which radiated symmetrically about a round central platform (19.5 cm dia) that was elevated 65 cm above the floor. The center of each arm was hinged perpendicular to the longitudinal axis such that the experimenter could raise, or lower, the proximal portion of each arm to present particular sequences of choices to the rat. Access to chocolate milk food reward, located at the distal ends of the arms, was controlled by a remote switch. The open maze was situated in a room (270 cm by 435 cm) that contained several items that could serve as visual cues for the animals, such as tables, chairs and miscellaneous laboratory equipment. The maze room was illuminated by a single 25 W incandescent lamp located in the southeast corner of the room.

The maze training procedure was similar to those described in previous reports [9–12]. Briefly, a rat was placed on the central platform of the maze, with simultaneous access to all 8 arms. The optimal strategy for solving the maze was to enter each arm once per trial. Selection of previously entered arms constituted errors. A trial ended when the rat entered all 8 arms. When rats performed 1 trial within 15 min, the rats began a 'partial forced-choice' maze training procedure. The latter procedure involved first presenting sequentially to the rat 4 randomly selected arms (sample phase). The specific arms presented during the sample phase varied from trial to trial. After consumption of food reward on the fourth arm, all 8 maze arms were presented simultaneously (memory phase), and the rats were required to select those arms not previously chosen during the sample phase. When rats were able to complete 1 partial forced-choice trial within 15 min for 3 consecutive days, they were then required to perform 10 partial forced-choice trials per day (2 min intertrial interval). When rats completed 10 such trials within 1 h for 7 consecutive days, they were given free access for food and water for 2–3 days before surgical implantation of recording electrodes and injection cannulae.

The rats were food deprived for 24 h prior to surgery. Animals were initially anesthetized with 35 mg/kg sodium pentobarbital (50 mg/ml Nembutal), followed by supplements of 0.05 ml as needed. The recording electrodes were placed just dorsal to hippocampus, and the injection guide cannula placed just dorsal to the lateral dorsal nucleus of the thalamus, or LDN ([22]: hippocampus—AP −4.0 mm, L ±2.4 mm, DV −1.8 mm; LDN—AP −2.3 mm, L ±1.7 mm, DV −4.0 mm). The guide cannula consisted of a 25 ga. stainless steel tube that was part of a cannula assembly and was identical in design to one described previously [9]. The reference electrode (114 μm Teflon-coated stainless steel wire) was placed in corpus callosum (AP −1.5 mm, L ±1.5 mm, DV −2.0 mm), and a ground lead was soldered to a jeweler's screw that was attached to the skull. Connecting pins from the unit recording and reference electrodes, and ground screw were inserted into a connecting socket that was permanently attached to the rat's head with dental acrylic. Following surgery, 0.1 ml Bicillin (300,000 U/ml) was injected into each hindleg to protect against infection. Upon recovery from surgery, the rats were trained to perform 15 trials per day within about an hour.

Details concerning the construction of recording electrodes and microdrives can be found in previous reports [5,8,9]. Briefly, the recording electrodes consisted of two lacquer-coated tungsten wires (20 μm dia; California FineWire) that were twisted together. The 'stereotrode' allows the simultaneous and independent recording of cellular activity on two chanenels (described below). The tip of each stereotrode was gold-plated to give final impedances of 50–150 kΩ (tested at 1 kHz). The stereotrode was threaded through a 30 ga. stainless steel cannula that was attached to a moveable microdrive [5]. Each microdrive contained two stereotrodes that could be simultaneously lowered into the brain in about 20 μm increments.

Single units were isolated by comparing between electrode wires the characteristics of the analog signals. Incoming signals were amplified 3,000–10,000 times, then filtered (at half amplitude) between 600–800 Hz (high pass) and 6 kHz (low pass). Signals were then passed through a window discriminator such that only those whose amplitude surpassed a predetermined threshold were accepted (sampling rate of 32 kHz). The ratio of spike amplitudes of signals from the two stereotrode wires, as well as the difference in latency to the first peak from the beginning of the sampling period (1 ms), were recorded. A template matching algorithm was also used to further isolate spikes that corresponded to a single cell. Data were acquired and subsequently analyzed with a BrainWave Neuroscience Workstation.

Hippocampal single units were initially identified on-line as single-spike or complex-spike neurons. Single spike cells tended to display interspike intervals of greater than 6.0 μs, and discharged spikes of relative

short duration (200–300 μs). Spike duration was defined as the time between the maximum and minimum voltage points of a sampled analog trace. Complex-spike cells, on the other hand, frequently showed high frequency bursts (interspike intervals of 2.5–4.0 ms) with progressively smaller amplitude action potentials within a burst. Also, complex-spike cells tend to discharge spikes of relatively long durations (300–400 μs). The complex-spike cells likely correspond to the pyramidal cell population of hippocampus since (unlike many single-spike cells) complex-spike cells are found in or near the pyramidal layers [1,2]. Given that past studies have shown that place fields of complex-spike cells are more robust and more common than spatial fields of single-spike cells [3,6,9], and since the purpose of this study was to examine LDN inactivation effects on hippocampal place-related firing, the inactivation tests were conducted only for complex-spike cells.

In addition to differentiating between the electrophysiological properties of recorded cells, the particular hippocampal subfield in which the cell belonged was identified by depth of the electrode from the brain surface. Verification of the subfield identification was subsequently confirmed by histological analysis. Each day, the recording stereotrodes were lowered until stable, isolated units were encountered, or up to a maximum movement of 200 μm per day. If units were encountered, the animal was prepared for the reversible inactivation procedure described below. If no clear units were found, the animal was still required to perform 15 trials on the maze before returning to the colony room. During all postsurgical recording sessions, the rat was connected to a recording headstage comprised of 5 unity gain FET preamplifiers and an infrared light-emitting diode. An automatic tracking system (Dragon Tracker Inc.) monitored the *XY* coordinate position of the diode by sampling its location at a rate of 20 Hz. The tracker information was transmitted to the BrainWave station, which also logged the time of each position event.

The guide cannula assembly contained a guide tube and stylet. LDN activity was reversibly inactivated using 2% tetracaine solution that was prepared by dissolving it in sterile 0.9% NaCl. 0.5 μl of fluid was pressure ejected using a microinjection procedure described previously [9].

About 15 min after stable, well-isolated hippocampal complex-spike units were encountered, animals began maze trials. The single unit–behavioral correlates observed during trials 4–5 served as baseline data. The LDN was inactivated bilaterally during the intertrial interval between trials 5 and 6. The behavioral correlates observed during the baseline trials were compared to those observed during trials 6 and 7 (tetracaine trials), and during trials 14–15 (recovery trials). Based on past studies (e.g. see discussion in [9]), it was assumed that the inactivating effects of tetracaine lasted about 15–20 min under these test conditions.

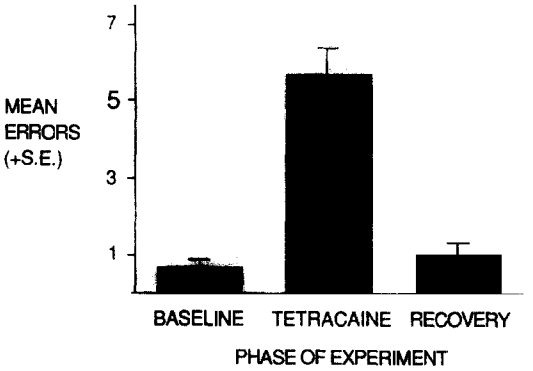


Fig. 1. The effects of LDN inactivation on choice accuracy as rats perform a spatial memory task on a radial arm maze. During the trials before tetracaine was injected into the LDN (BASELINE), rats made on average less than one error per trial. Immediately following LDN inactivation (TETRACAINE), rats made significantly more errors. At the end of the recording session (RECOVERY), the error rate was no longer significantly different from that observed during the baseline period.

At the time of testing for LDN inactivation effects, rats were performing at asymptote levels. Thus, as shown in Fig. 1, the mean (±S.E.) number of errors made during the baseline period (6 rats; 11 recording sessions) was 0.68 ± 0.21. Following injection of tetracaine into the LDN, the number of errors increased to 5.41 ± 0.88. Unlike hippocampal-lesioned rats, LDN inactivated rats did not show increased reactivity to stimulation, nor did they show perseverative behaviors. By the end of the recording session, the error score dropped to 1.00 ± 0.36. A repeated measures analysis of variance revealed a significant repeated measures effect, $F_{2,10} = 31.84$, $P < 0.001$. Post-hoc Scheffé analyses ($\alpha = 0.05$) showed that the number of errors made during the tetracaine period was significantly greater than those made during either the baseline or recovery periods ($P < 0.05$). Thus, LDN inactivation produced a spatial learning impairment. A behavioral impairment of similar magnitude has been described following reversible inactivation of the medial septal afferent system to the hippocampal formation [9,11].

Of the 24 complex-spike cells tested, 21 were CA1 cells and 3 were hilar units. A spatial specificity score was calculated for each cell. This calculation involved determining the mean firing rate as the rat moved inward or outward on each of the 8 maze arms. Of these 16 rates, the highest rate was divided by the average of the remaining 15 rates to arrive at a spatial specificity score. Thus, such a score takes into account not only place specificity, but also directional specificity. Both of these considerations are important since past studies [5,6,9] have shown that most hippocampal place fields have a directional as well as location bias

when rats perform a radial maze task. It should be noted however, that a small subpopulation of hippocampal place cells show clear bidirectional discharge, i.e. cells show elevated firing as the rat moves both inward and outward on a maze arm that contains a place field. In such cases, the place specificity computation will underestimate true location specificity. Nevertheless, our place specificity measure has proven to be sufficiently sensitive to experimental manipulation that important features of place cell function have been disclosed. If a specificity score was greater than 2.0, the cell was classified as a place cell and its response to LDN inactivation evaluated. There were no clear differences in the response properties of CA1 and hilar cells. Therefore, these data were combined for the group summary provided below.

Coincident with the behavioral impairment, a variety of cell responses to LDN inactivation were observed. Fig. 2 illustrates these responses for location-specific hippocampal cells that are typically described in the literature. That is, these cells preferentially discharged as animals traversed a particular subregion of a maze arm. Dots represent locations on the maze that were sampled by the rat. The radius of the circles is proportional to the local firing rate. Vectors radiating from the center of the circles indicate the direction of diode movement at the time the cell fired. The top portion of Fig. 2 shows a specific place field that was observed during the baseline period (left), then after the LDN was injected with tetracaine (right). It can be seen that after LDN inactivation, this cell showed elevated firing as the animal traversed many locations on the maze. In contrast, many other hippocampal cells responded to LDN inactivation by reducing spontaneous firing (middle portion of Fig. 2). In both examples (Fig. 2A,B), spatial specificity declined dramatically. The third example (bottom portion of Fig. 2) illustrates a unit that responded to LDN injection of tetracaine by reducing it's firing rate and, unlike in the previous examples, showing increased spatial specificity.

The left portion of Fig. 3 illustrates the baseline response of a hippocampal cell that increased firing when the animal moved along the northwest direction on the maze (i.e. inbound on the southeast maze arm and outbound on the northwest maze arm). This cell also increased discharge when the rat moved west, although this change in firing was observed when the rat traversed only the east arm of the maze. Thus, unlike most hippocampal place cells described in the literature, this unit exhibited *both* location and heading directional specificity. The center panel shows that after LDN injection of tetracaine, the unit essentially ceased discharge. By the end of the recording session, the spatial specificity of discharge was clearly returning. The northwest and west directional preferences

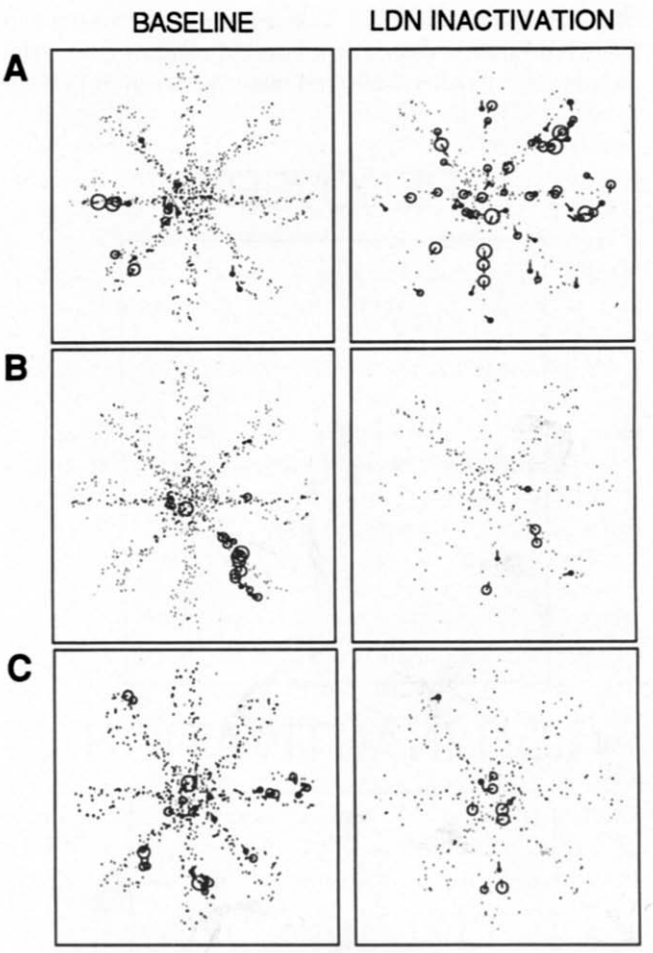
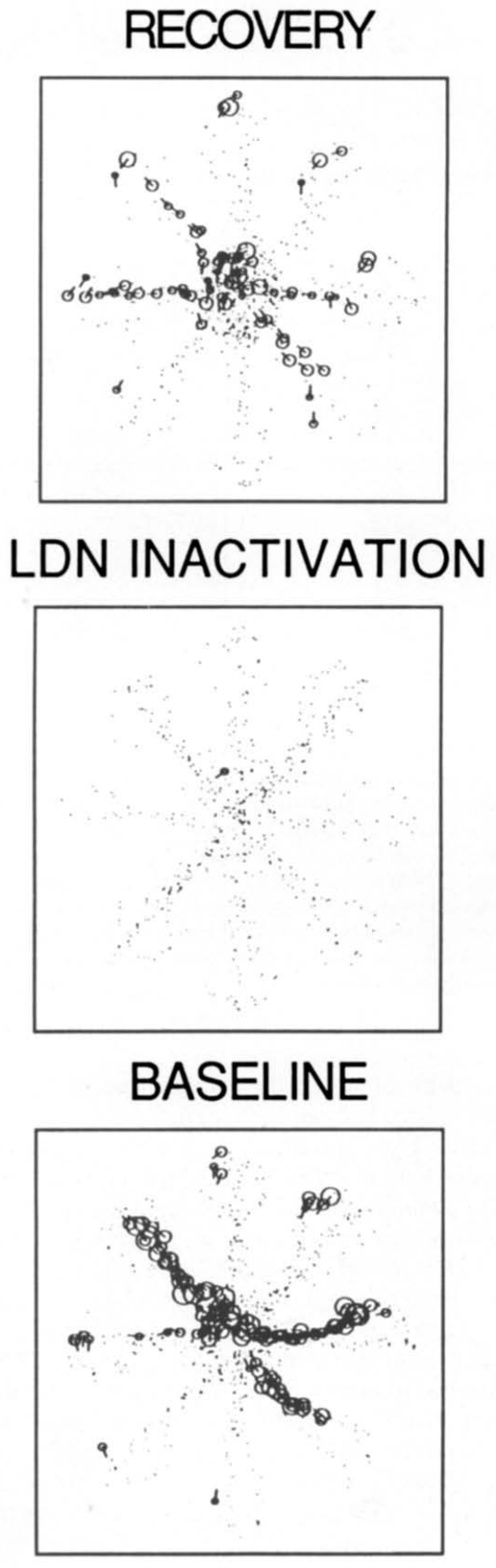


Fig. 2. Examples of the spatial distribution of firing by hippocampal place cells before (BASELINE) and soon after (LDN INACTIVATION) tetracaine was injected into the LDN region. A variety of responses were observed. A: one type of response is shown by this hippocampal neuron which increased firing, and reduced location specificity, following LDN inactivation. B: as illustrated by this cell, other neurons showed both reduced firing rates and place specificity. C: still other neurons showed reduced firing rates accompanied by an increase in place-specific firing.

were evident, although the location-specificity of the west preference had not reappeared. Nevertheless, it is clear that LDN inactivation resulted in dramatic changes in spatial coding by this hippocampal cell.

Fig. 4 summarizes the proportional change in place specificity scores and mean firing rate for all complex-spike units recorded. Specificity scores greater than 1.0 reflect cells that increased specificity after LDN inactivation, while values less than 1.0 indicate cells that responded with reduced specificity. About 41.7% of cells showed reduced place specificity of more than 25%, and 41.7% of cells showed increased place specificity of greater than 25%. Thus, the mean ($\pm$S.E.) change in specificity was 32% $\pm$ 29%. In contrast, most cells (i.e. 66.7%) showed reduced firing rates by more than 25%, while only 20.8% of cells showed elevated

firing by more than 25%. Consequently, in contrast to place specificity scores, the over firing rate for the population of cells recorded was reduced from 1.99 (±0.55) Hz to 0.82 (±0.32) Hz following LDN inactivation.



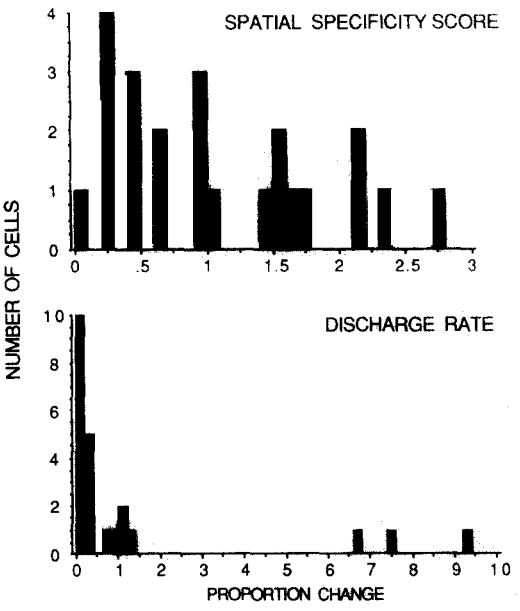


Fig. 4. The proportion change in place specificity and mean firing rate for all complex-spike cells tested. Proportion scores for place specificity or discharge rate that were greater than 1.0 indicate increased specificity or firing rates following LDN inactivation, while scores less than 1.0 reflect reduced specificity or firing rates. It can be seen that roughly equal proportions of cells showed increased or decreased place specificity after LDN inactivation. In contrast, the average firing rate of a majority of hippocampal cells was reduced following LDN inactivation.

It could be argued that the hippocampal unit and behavioral effects were observed because tetracaine spread dorsally to the hippocampus. If this were the case, it would be expected that all hippocampal units should reduce or stop firing after injection. On the contrary, a variety of responses were observed, even within the same recording and injection session. For example, some cells showed increased firing, while others showed reduced firing rates, or no change in firing. Therefore, it is unlikely that the spread of tetracaine to hippocampus can adequately explain our findings.

Previously, we postulated that the LDN is part of a neural system that subserves experience-dependent spatial navigation [12]. Specifically, it was proposed that LDN directional information becomes integrated

Fig. 3. On occasion, we recorded hippocampal neurons that showed head direction correlates similar to those described for the postsubiculum [25] and the LDN [12]. In addition to the directional correlate, during the baseline period, this particular neuron showed a location preference on the east arm of the maze. Following LDN inactivation, this cell ceased firing altogether. By the end of the recording session, much of the original place and direction correlates were restored.

with detailed visual information provided by the geniculostriate system. Such integration may occur in the subicular complex and/or the entorhinal cortex, both of which receive LDN and neocortical afferents. The integrated spatial information of (e.g.) the entorhinal cortex may then enter into further computations that associate it with nonspatial data such that place representation in Ammon's horn and the dentate regions of hippocampus proper is achieved. In this way, hippocampal spatial representations become more dynamical and less sensory dependent than those observed in afferent structures (as shown in [13]). The present finding that LDN inactivation disrupted both place fields of hippocampal neurons as well as what is generally considered to be hippocampal-dependent behavior strengthens the argument for a behaviorally important functional connection between the LDN and the hippocampus.

The fact that all hippocampal place cells were not disrupted in a similar manner by LDN inactivation suggests that hippocampal spatial representations do not reflect the mere addition of bits of spatial information provided by its complex afferent systems. If this were the case, one might have expected that LDN inactivation would eliminate the directional component of the place fields while leaving location specificity intact. Instead, a rather drastic reconfiguration of the overall spatial firing patterns of hippocampal place cells was observed. Thus, we suggest that the hippocampal formation qualitatively transforms individual elements of afferent information as they become associated with other types of afferent information. The resultant hippocampal spatial representation would therefore reflect a highly integrated form of spatially-relevant data. As a result, prediction of the specific influence of each input on the character of a given hippocampal representation (or association) of space may not be possible until one identifies all of the constituent afferent elements, as well as the nature of the associative computations performed by hippocampus.

Although identification of the nature of the association of afferent information within hippocampus awaits further research, the above hypothesis regarding the highly integrated nature of associative representations in hippocampus predicts that removal of even a single afferent input should disrupt the overall integrity of hippocampal spatial firing. This result was observed in the present study: elimination of the LDN afferent input (that presumably provides visual directional information) to hippocampus produced changes in multiple aspects of the place fields being monitored.

One implication of the interpretation that location-specific firing by hippocampal neurons represent highly integrated sensory associations is that the directional correlate of hippocampal place fields may not necessarily reflect to the animal the same information as directional firing by cells that are afferent to the hippocampus. That is, while the LDN may code directional heading information, the directional component of hippocampal place fields may *not* necessarily refer to heading direction within a place field. Rather, such hippocampal directional firing may reflect other factors such as the significance of movement along certain trajectories through particular locations.

The results of the present experiment are congruent with the postulate that the LDN provides important spatial information to the hippocampal formation. The finding that reversible inactivation of the LDN produced spatial learning impairments on a maze, as well as disruption of place representation by hippocampal neurons, supports the hypothesis that the LDN plays an important mnemonic function in the neural system that mediates accurate spatial navigation.

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