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Maps, Routes, and the Hippocampus: A Neural Network Approach

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ABSTRACT

This study describes hippocampal participation in maze navigation in terms of a real-time, biologically plausible neural network. The system is composed of (1) a goal-seeking mechanism, (2) a cognitive map system, and (3) a route system. The goal-seeking mechanism displays exploratory behavior until either the goal is found or a sufficiently strong prediction of the goal is generated. The cognitive map is a topological map that stores associations between places and views of accessible places, and between places and reward. The route system establishes associations between cues and reward. Both systems compete with each other to establish associations with the reward, with the cognitive system generally overshadowing the route system. In agreement with previous models, it is assumed that the hippocampus modulates the storage of cognitive maps in cortical areas and mediates the competition between cognitive maps and route systems. After hippocampal lesions, animals navigate through mazes making use of the route system. Computer simulations show that the network effectively describes latent learning, detour behavior, and place learning in normal and hippocampal- and cortical-lesioned animals.

Key words: spatial learning, cognitive maps, maze learning, hippocampus, cortex

In 1978, O'Keefe and Nadel introduced the appealing view that the hippocampus functioned as a cognitive map. The view primarily found support from data showing that hippocampal lesions impair some types of spatial learning and that neuronal activity in the hippocampus is correlated with locations in space. According to O'Keefe and Nadel, spatial navigation can be performed using either a cognitive map system or a route system. A cognitive map is defined as a "set of connected places which are systematically related to each other by a group of spatial transformation rules . . . [that] preserve the Euclidian relationship between angles and dimensions." Routes are defined as lists of Tolmanian stimulus-response-stimulus (S-R-S) instructions, which define the specific responses to perform in the presence of particular cues. Whereas spatial navigation in normal animals is carried out with the help of the cognitive map system, hippocampal-lesioned animals navigate through space making use of the route system.

The present paper presents a neural network that describes cognitive map and route systems. Like O'Keefe and Nadel, we define cognitive maps as sets of connected places. However, our maps encode only the adjacency, not the distance or direction, between places. Also in contrast to O'Keefe and Nadel, we define a route as a set of associations of cues with the reward (regarded as analogous to an unconditioned stimulus, US). Whereas O'Keefe and Nadel suggested that the cognitive map is stored in the hippocampus, we assume that the hippocampus is necessary to store cognitive maps in cortical areas. Finally, in agreement with O'Keefe and Nadel, we propose that hippocampal-lesioned animals navigate through mazes making use of the route system. Computer simulations of the network analyze the effect of hippocampal lesions (HL) and cortical lesions (CL) on latent learning, detour learning, and water maze learning.

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THE NEURAL NETWORK

Recently, Schmajuk and Thieme (1992) illustrated how a neural network can mechanistically implement Tolman's (1932) concept of purposive, goal-seeking behavior. The present study incorporates the cognitive map architecture suggested by Schmajuk and Thieme. However, whereas Schmajuk and Thieme's (1992) system consists only of a goal-seeking mechanism and a cognitive map, the system described in the present paper also includes a route system.

Purposive behavior

In order to establish a connection between cognition and behavior, the goal-seeking system implements a "stimulus-approach" view of behavior control by which animals approach appetitive stimuli and avoid aversive stimuli. When

an appetitive stimulus is encountered, it activates approach responses and inhibits search responses. Search behavior can be inhibited either by the reward itself, or when reward is predicted by the cognitive map or route system (the internal representations of the world). As the animal explores the environment under the control of the goal-seeking system, it builds a cognitive map and a route representation of the external world. Both the cognitive map and the route system are updated whenever a mismatch between predicted and actual inputs is detected.

The cognitive mapping system

Figure 1 shows a neural network capable of cognitive mapping and route learning. The neural network is a real-time mechanism that describes behavior as a moment-to-moment phenomenon. Nodes in the network represent neural populations, rather than individual neurons. Appendix I presents a formal description of the network as a set of differential equations that depict changes in the values of neural activities and connections as a function of time.

A cognitive map represents the connectivity between discriminable regions of space, referred to as places. Therefore, a cognitive map can be built only when different spatial regions have been discriminated. For simplicity, we assume that specific locations in space can be discriminated by learning the visual angles to distal extramaze cues (e.g., pictures on room walls, objects in the room) as perceived from that location (Zipser, 1985, 1986; Schmajuk, 1990). Place discrimination may be accomplished by a neural network with nodes that become maximally active at given spatial locations and display a decremental generalization at other locations (see Schmajuk, 1990; Schmajuk and Blair, 1993). At any given time, the node that is the most active represents the place where the animal is located (place i), whereas all other active nodes represent views of neighboring places (view j). Notice that view j does *not* represent a "local view", that is, the visual inputs while the animal is at a particular location and looking in a given direction. The current implementation assumes that animals only perceive views of places directly connected to and accessible from the place where they are located.

The network shown in Figure 1 incorporates Kohonen's (1977) heteroassociative matrix with two types of inputs: places and views. Place i is assumed to give rise to a short-term memory trace, x_i (equation 1 in Appendix I). Place trace x_i may become associated to the views of other places to form long-term associations V_{ij} between place i and view j. The strength of modifiable synapses in the network, indicated by open triangles in Figure 1, represents V_{ij} associations. When the animal is at place i and perceives view j, V_{ij} increases. When the animal is at place i and cannot perceive view j, V_{ij} decreases. Each time the animal enters place i, V_{ij} associations generate real-time predictions, B_j, of views j to be seen from place i. That is, as long as the animal stays in place i, place i activates neurons y_j proportionally to V_{ij}, and this activity represents the prediction that view j is available from place i (equation 3 in Appendix I). V_{ij} is readjusted to reflect the maze configuration whenever there is a mismatch between the actual and the predicted view, B_j (equation 4 in Appendix I). The cognitive map built by our network is a topological map, that is, it represents only the connectivity, but not distance or direction, between places. V_{ij} associations are the learned representations of the topological relations in the external world.

In addition to place–view associations, Place–US associations are also formed (see equation 8 in Appendix I). The output of neuron y_{US} represents the prediction of the US by the cognitive map.

The route system

We assume that in addition to place–view associations, V_{ij}, animals form associations between intramaze cues found in place i and the US. A cue in place i is assumed to give rise to a trace, c_i (equation 5 in Appendix I). We assume that the trace of a cue, c_i, decays at a slower rate than the trace of a place, x_i. Cue trace c_i may become associated with the US to form long-term associations D_i (see equation 7 in Appendix I). The output of neuron z_{US} represents the route prediction of the US.

Interactions between the cognitive map system and the route system

Table 1 summarizes the properties of the cognitive map and the route systems. Similar to the model of O'Keefe and Nadel (1978), the cognitive map is built upon exploration, learns and extinguishes rapidly, and is insensitive to intertrial interval; the route system is trained through reward or punishment, learns and extinguishes incrementally, and is sensitive to intertrial interval. These assumptions are also analogous to Hirsh's (1974) suggestion that off-line (cognitive) associations change at a faster rate than on-line (route) associations.

Places are assumed to sustain relatively short temporal traces, whereas cues are believed to maintain relatively long temporal traces. Consequently, places may become associated only with views of neighboring places but cues may acquire direct associations with spatially (and thus temporally) distant USs. Given that place–view associations change at a fast rate, the cognitive system shows great flexibility. Con-

Fig. 1. Cognitive mapping and route systems. Cognitive map system: The cognitive mapping system associates place representations with view and US representations. The first derivatives of the outputs of neurons y_j representing views are fed back into the neurons x_j representing place j corresponding to view j. The first derivatives of the outputs of neurons y_j are also conveyed to neurons p_j. V_{ij}, place–view associative weights; d_j, neurons computing first derivatives. Route system: The route system associates cues with US representations. D_i, cue–US associative weights; d_j, neurons computing first derivatives. Decision system: G_i, neurons integrating the pulses generated by the brief examination of alternative next places; $p_{i,US}$, working memory associative weights of fast-time predictions of the US. Arrows, fixed excitatory connections, open triangles, variable excitatory connections.

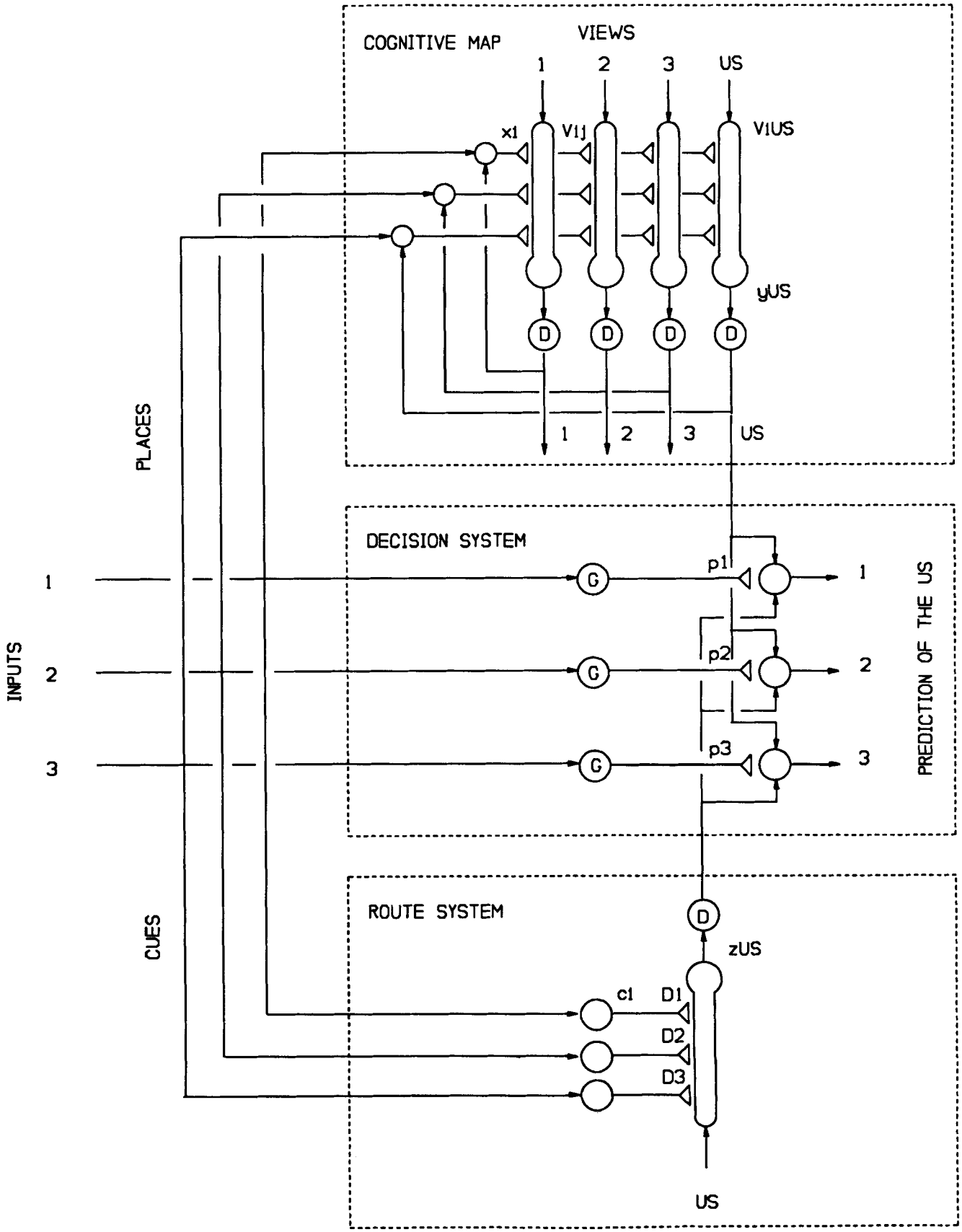

COGNITIVE MAP
VIEWS
1
2
3
US
x1
Vij
ViUS
yUS
D
1
2
3
US
PLACES
DECISION SYSTEM
INPUTS
1
2
3
G
p1
p2
p3
PREDICTION OF THE US
ROUTE SYSTEM
zUS
CUES
c1
D1
D2
D3
US

Table 1. Properties of the Cognitive Map and Route Systems

Cognitive Maps	Routes
Fast decay in STM traces	Slow decay in STM traces
Short-range associations	With delta rule (normal animals), short-range associations; without delta rule (hippocampal-lesioned animals), long-range associations
Associations between places	Cue–US associations
Fast change in place–view associations (flexibility)	No cue–cue associations
Fast change in place–US associations	Slow change in cue–US associations
Associations can be combined in a cognitive map	Associations cannot be combined
Place–US associations compete with cue–US associations	Cue–US associations compete with place–US associations
Stored in cortical areas	Stored in subcortical areas

STM, short-term memory; US, unconditioned stimulus.

versely, cue–US associations change at a slow rate, a property that confers great stability to the route system.

It is also assumed that the predictions of the US generated by the cognitive map and the route systems are combined into the real-time aggregate prediction of the US, B_{US} (equation 6 in Appendix I), which is used to control the growth of both cognitive V_{iUS} associations and route D_i associations. Once place discrimination is completed, cognitive V_{iUS} associations are assumed to grow at a faster rate than route D_i associations and to have a stronger influence on the aggregate prediction B_{US}. In consequence, V_{iUS} associations in the cognitive system limit the formation of D_i associations in the route system, a phenomenon known as overshadowing in classical conditioning. Therefore, after the cognitive mapping system has accrued strong associations, the route system will accrue only weak associations. This assumption is supported by Diez-Chamizo, Sterio, and Mackintosh's (1985) and Whishaw, Mittleman, Bunch, and Dunnett's (1987) results demonstrating that extramaze landmarks (used to discriminate places) overshadow intramaze cues, but not vice versa. Notice that O'Keefe and Nadel did not specify the interaction between cognitive and route systems in normal animals.

Inferential maze navigation with a cognitive map and a route system

Inferential navigation allows the system to find the shortest route to a goal from any location in the maze. Through inferential navigation, animals can detour to novel routes when old ones have been blocked, or navigate toward the goal from different starting points. During inferential navigation, the system generates fast-time predictions of the goal, that is, predictions that proceed at a faster pace than the real movement through the maze. In contrast, real-time predictions, B_j and B_{US}, occur in close temporal proximity to the animal's behavior.

Like Tolman (1932), we assume that before making a decision the animal briefly enters each alternative next place h linked to place i (vicarious trial and error, or VTE behavior). Each brief inspection results in a fast, short, and relatively weak activation pulse of the place input and its corresponding trace, x_h. Place trace x_h activates cells y_j in proportion to their V_{hj} connections. Therefore, view cells, y_j, are activated by fast-changing signals proportional to $x_h V_{hj}$, which are recurrently reinjected into x_j through cells d_j. Subsequently, x_j activates y_k, y_k activates x_k, and so forth, spreading the activation over the network. Because activation spreads decrementally (inversely proportional to the magnitude of a reinjection constant), the output of cell y_{US} reflects the known distance (measured as the number of intermediate places) of the alternative next place h to the US. As the animal examines all the alternative next places h linked to place i, short and relatively weak activation pulses of the cue inputs and their corresponding traces, c_h, are also generated. Cue traces c_h activate cells z_{US} in proportion to their D_h connections. Because D_h is inversely proportional to the spatial (and therefore, temporal) distance between place h and the goal where the US is located, the output of cell z_h roughly reflects the distance of the alternative next place h to the goal where the US is encountered. As the animal examines all the alternative next places, fast-time predictions of the US (the derivatives of the output of cognitive map cells, y_{US}, and route cells, z_{US}) are stored into a different working memory $p_{h,US}$ for each alternative next place h. Each of these working memories, $p_{h,US}$, is proportional to the prediction of the US by the alternative next place h and cue h, and they decay at a relatively fast rate (equation 9 in Appendix I). Competition among the working memories, $p_{h,US}$, allows the animal to decide which of the alternative next places (and corresponding cues) is the best predictor of the US. We assume that working memories $p_{h,US}$ decay back to zero by the time the animal arrives at the next choice point.

Under the "stimulus-approach" view, the alternative next place that best leads to the goal becomes a subgoal and is subsequently approached. If the magnitude of the aggregate prediction of the US by all alternative next places and cues is smaller than a certain minimum value, animals engage in a random exploratory (search) behavior. If the magnitudes of two predictions are identical, animals choose at random between the two stimuli generating the predictions. For simplicity, we assume that each alternative next place is examined for the same amount of time. Maze navigation proceeds until the US is found or the animal is removed from the maze.

REGIONAL MAPPING OF THE MODEL

Figure 2 suggests how nodes and connections in the network map onto different brain regions. This mapping is presented at the "regional level" (see Squire et al., 1989). Based on the brain-mapped network, this section also explains the effects of lesioning different areas of the brain.

Hippocampus

Aggregate predictions

Following Schmajuk and DiCarlo (1992), we assume that during spatial learning, the hippocampus computes real-time aggregate predictions of views and the US. Experimental data

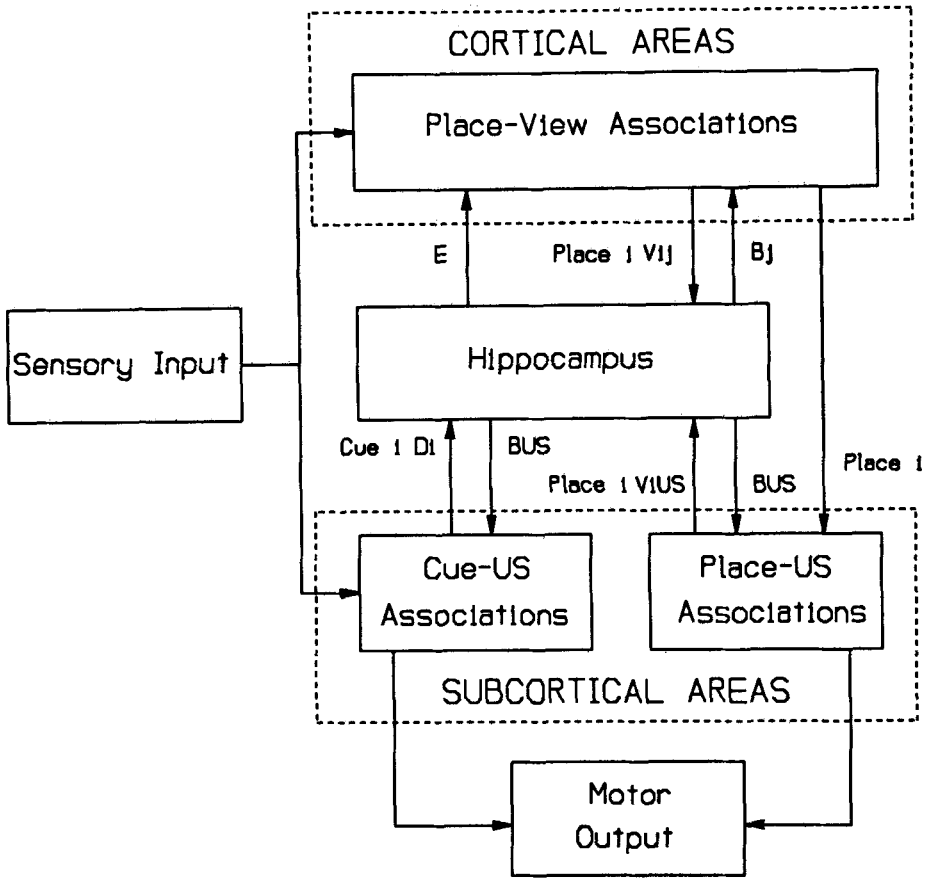


Fig. 2. Mapping of the network onto a schematic diagram of cortical, hippocampal, and subcortical interconnections. V_{ij}, place–view associative weights; V_{iUS}, place–US associative weights; D_i, cue_i–US associative weights; B_{US}, aggregate prediction of the US; B_j, aggregate prediction of view j; E, activating signal controlling cortical learning.

suggest that aggregate predictions are coded by the activity of hippocampal pyramidal cells. Hippocampal pyramidal cells become maximally active at different spatial locations (see Muller et al., 1987), and place cell firing seems to predict the animal's future location by about 150 ms (Muller and Kubie, 1986). In the network, nodes representing the aggregate prediction of view i, $B_i = \Sigma_j\ place_j\ V_{ji}$, show place-related activity because cells computing B_i are active when the animal is located at any $place_j$ associated with $view_i$ (i.e., V_{ji} larger than zero). Since B_i increases as the animal approaches $place_j$ associated with $view_i$, the model correctly shows that pyramidal activity increases at specific places and predicts possible future locations. Aggregate prediction B_i is used to determine the output error signal ($View_i - B_i$), that controls the formation of $place_j$–$view_i$ associations in cortical areas.

In addition to their place-related activity, hippocampal pyramidal cell firing might also represent the real-time prediction of biologically significant cues or places (Breese et al., 1989). This feature is captured by the model's assumption that hippocampal place activity represents the aggregate prediction of the US, $B_{US} = \Sigma_i\ cue_i\ D_i + \Sigma_j\ place_j\ V_{jUS}$, generated by both the cognitive and the route systems. Since B_{US} becomes large as the animal approaches a location or cue where the US has been presented before, the model correctly predicts that hippocampal pyramidal activity will increase under these circumstances. Aggregate prediction B_{US} is used to determine the output error signal, ($US - B_{US}$), that controls the formation of place and cue associations with the US in subcortical areas.

Place–View associations

Schmajuk (1989) suggested that CS–CS associations are stored in neocortex under hippocampal modulation, a notion that is compatible with data showing that sensory preconditioning[1] is impaired either by neocortical (Thompson and Kramer, 1965) or hippocampal (Port and Patterson, 1984) lesions. In line with this assumption, we conjecture that place–view associations (considered similar to CS–CS associations) are stored in cortical regions and not in the hippocampus (see Fig. 2). The hippocampus modulates the updating of the cognitive map in neocortex whenever a mismatch between predicted and actual inputs is detected (see Schmajuk and DiCarlo, 1992).

[1] Sensory preconditioning consists of a first phase in which two conditioned stimuli, CS(A) and CS(B), are paired together in the absence of the US. In a second phase, CS(A) is paired with the US. Finally, when CS(B) is presented alone it generates a weak but distinct conditioned response (CR).

Hippocampal lesions

Hippocampal lesions refer to the complete bilateral ablation of the dentate gyrus, CA1, CA2, and CA3 regions and the subicular complex. They impair both the acquisition and retention of cognitive mapping. Acquisition is impaired because HL animals cannot store cortical place–view associations. Retention is impaired because, in the absence of the competition mediated by the aggregate prediction, HL animals acquire associations between distal cues and the US (see Schmajuk, 1990) and these associations produce a generalization that impedes the precise localization of the rewarded place. Therefore, the model suggests that HL impair the retention of maze learning not as a result of a loss of the cognitive map, which is stored in cortical areas, but as a consequence of a failure to discriminate the location of the reward.

Hippocampal lesion animals are still capable of spatial navigation by storing cue–US associations, D_{iUS}, in subcortical areas. Thus, HL animals are able to reach the goal using their route system, a view supported by Winocur's (1982) data showing that HL animals impaired in place learning are still able to perform adequately when distinctive cues are introduced in a radial maze. A formal description of the effects of HL is presented in Appendix II.

Association cortex

Place–View associations

We conjecture that place–view associations are stored in cortical regions. Figure 2 shows that hippocampal output proportional to the aggregate prediction of view j, B_j, regulates the competition among place–view associations in cortical areas. Cortical projections to the hippocampus provide information about place–view associations, $place_i$ V_{ij}, used to compute B_j.

Cortical lesions

Cortical lesions refer to the complete bilateral ablation of the entorhinal, parietal, or cingulate cortices. Acquisition and retention of cognitive mapping are impaired because CL animals do not store cortical place–view associations. However, CL animals are still capable of spatial learning by storing cue–US associations, D_{iUS}, in subcortical areas. Because the hippocampus is still available for the computation of the aggregate prediction of the US, B_{US}, cue–US associations do compete, and consequently, only cues close to the goal accrue a strong association with the US. Thus, CL animals are capable of maze navigation by making use of their route system. A formal description of CL effects is presented in Appendix II.

Subcortical areas

Place–US and cue–US associations

Following Schmajuk and DiCarlo (1992), we assume that place–US and cue–US associations are stored in subcortical areas. Figure 2 shows that copies of $place_i$ V_{iUS} and cue_i D_i are relayed from subcortical areas to the hippocampus, where the aggregate prediction of the US, $B_{US} = \Sigma_i\, cue_i\, D_{iUS} + \Sigma_j\, place_j\, V_{jUS}$, is computed. The hippocampus inhibits the US input to subcortical regions, proportionally to B_{US}. As B_{US} increases during acquisition, further increments in subcortical $place_j$–US and cue_i–US are prevented.

Some experimental data support the assumption that place–US and cue–US associations are stored in subcortical areas. For instance, Whishaw et al. (1987) reported that caudate-putamen lesions partially impair acquisition and retention of both cue and place learning tasks in a water maze. Recently, Packard and McGaugh (1992) communicated that lesions limited to the caudate impaired the acquisition of cue but not of place learning. In contrast to Whishaw et al. (1987) and Packard and McGaugh (1992), Mitchell and Hall (1988) found that rats with caudate-putamen lesions were impaired in a spatial task when required to make the correct position response, but not when required to approach the correct cue.

COMPUTER SIMULATIONS

We compared experimental data with computer simulations of the effect of HL and CL in latent learning, detour tasks, and place learning in the water maze, obtained with the network described in Figure 1 and the brain mapping depicted in Figure 2. All simulations were carried out with identical parameter values. Parameter values used in the simulations are presented in Appendix III.

Latent learning

Experimental data

In latent learning, animals are exposed to a maze without being rewarded at the goal box. When a reward is later presented, animals demonstrate knowledge of the spatial arrangement of the maze, which remains "latent" until reward is introduced (Blodget, 1929).

Hippocampal lesions The effect of HL on latent learning has been reported in several studies. With relatively large HL, Kimble and Greene (1968) found that HL rats are impaired in latent learning of a Lashley III maze. When lesions were mostly confined to the dorsal hippocampus, Kimble and BreMiller (1981) and Kimble, et al. (1982) found that although HL rats show clear deficits in learning a Hebb-Williams (1946) maze, preexposed HL animals show a smaller number of errors than their nonpreexposed counterparts on the first training trial. However, the interpretation of this latter result as latent learning may be debatable for several reasons. First, when performances on the second trial are compared, both preexposed and nonpreexposed HL groups show a similar number of errors. Second, neither preexposed nor nonpreexposed HL groups show significant improvement over training trials. Third, as suggested by Means (1969), even when preexposed HL animals require fewer trials to reach criteria than the nonexposed, the effect might reflect a general habituation to the maze rather than actual maze learning. In agreement with Kimble and Green (1968), Herrmann et al. (1978) reported that fornically damaged animals were unable to solve Maier's (1929) three-table spatial problem, a paradigm similar to latent learning.

Cortical lesions Herrmann et al. (1985) reported that animals with frontal lesions are impaired in Maier's three-table problem.

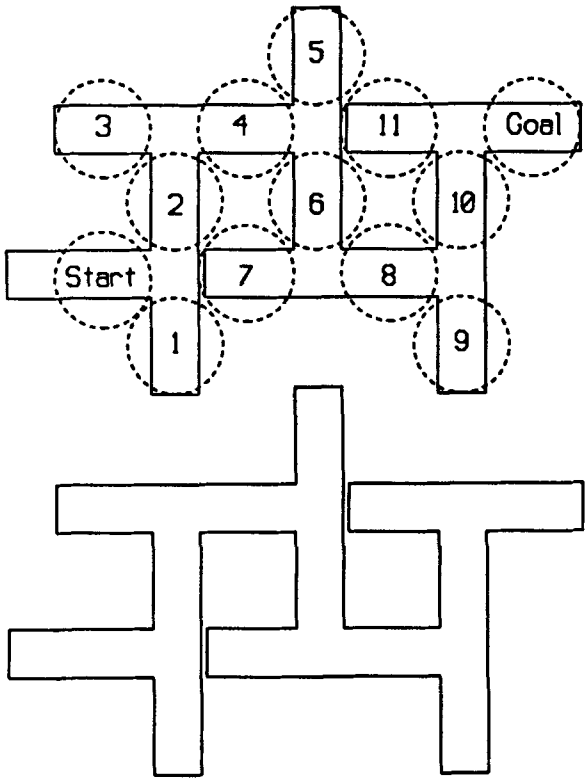


Fig. 3. Latent learning. Diagram of a multiple-T maze and its representation in the computer model. Dashed circles represent different places discriminated by normal animals.

Simulation results

Figure 3 portrays a multiple-T maze and its representation in the computer model. Dashed circles represent places, discriminated by normal animals and used to build a cognitive map. We assume that HL and CL animals build a route based on distinctive intramaze cues encountered at those places.

Normal case The upper panel in Figure 4 shows the number of moves to reach the goal as a function of trials for two different normal groups. Group A is rewarded at the goalbox on the first trial and group B on the sixth trial. Group A stores the organization of the maze in the cognitive map. Because cue–US and goal place–US associations compete against each other to predict the US, and because place–US associations grow at a fast rate, they overshadow all cue–US associations. Normal animals can approach the goal by using their cognitive maps to generate fast-time predictions of the US.

During latency, group B constructs a cognitive map of place–view associations. When no goal place–US associations are available, the map is not utilized. When goal place–US associations are established, group B is able to determine the correct path, therefore showing faster improvement in performance than unexposed animals in group A (latent learning). These results are in accordance with Blodget's (1929) latent learning data.

Hippocampal lesion case The middle panel in Figure 4 shows the number of moves to reach the goal and cue–US associations as a function of trials for two different HL groups. Group A is rewarded at the goalbox on the first trial and group B on the sixth trial. In the HL case, the cognitive map is not available and group A accomplishes maze navigation with the assistance of the route system. In addition, because cue–US associations do not compete against each other through the aggregate prediction, cues become associated to the US in inverse proportion to their distance to the US and independently of the associations accrued by other cues. Therefore, cues located at places close to the US will not overshadow cues far from the US. Since cues located far from the goal can still become associated with the US, HL animals are able to approach the US from remote locations in the maze.

During latency, because HL animals form only cue–US associations, group B cannot construct a cognitive map. When goal cue–US associations are established, group B is able to determine the correct path, showing improvement in performance identical to unexpected animals in group A (absence of latent learning). These results are in accordance with Kimble and Greene (1968) and Hermann et al. (1978) but not with Kimble and BreMiller (1981) and Kimble et al. (1982).

Cortical lesion case The lower panel in Figure 4 shows the number of moves to reach the goal and cue–US associations as a function of trials for two different CL groups. Group A is rewarded at the goalbox on the first trial and group B on the sixth trial. Group A accomplishes maze navigation with the assistance of the route system. Because cue–US associations compete against each other through the aggregate prediction, cues become associated to the US in inverse proportion to their distance to the US and depending on the associations accrued by other cues. Therefore, cues located at places close to the US will overshadow those cues far from the US. Since cues located far from the goal cannot become associated with the US, CL animals are unable to approach the US from remote locations in the maze.

During latency, because CL animals form only cue–US associations, group B cannot construct a cognitive map. When goal cue–US associations are established, group B is able to determine the correct path, showing a small improvement in performance identical to unexposed animals in group A (absence of latent learning).

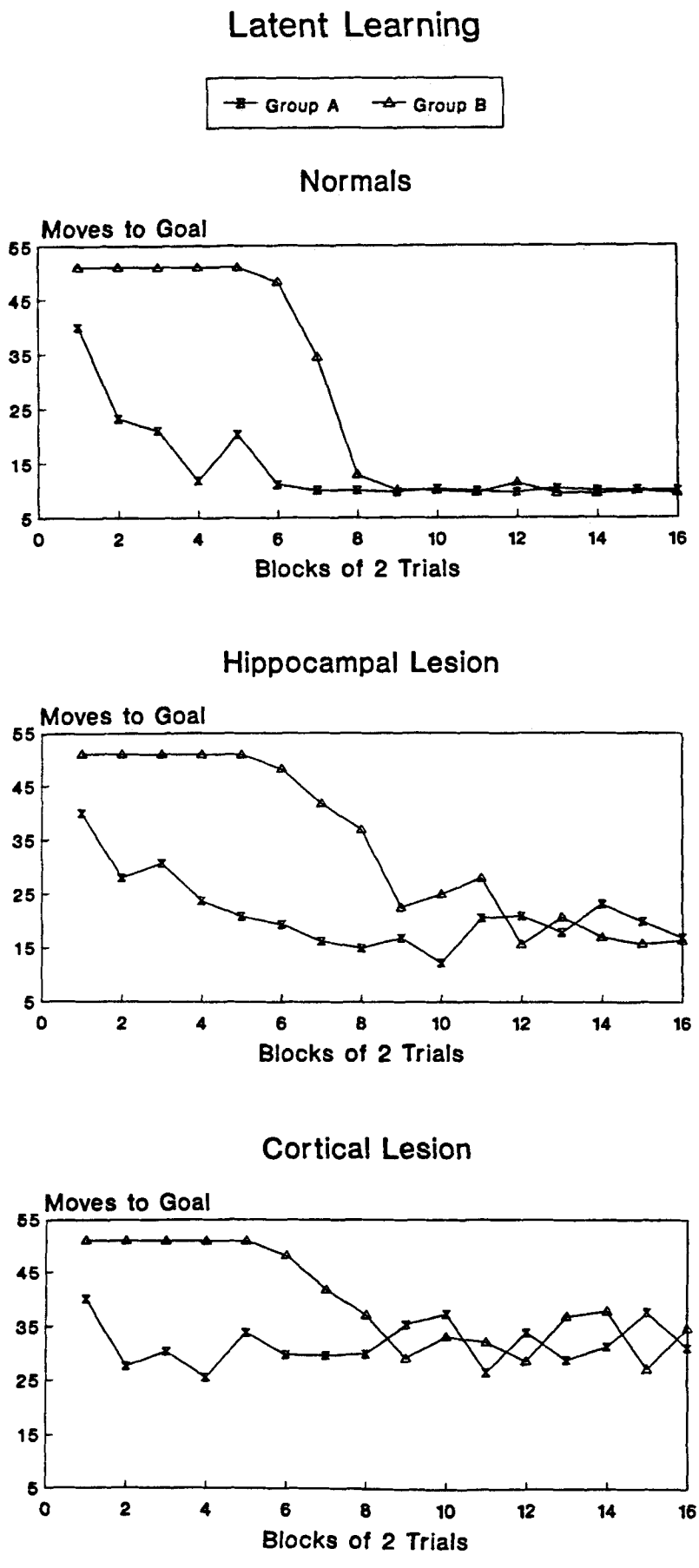
Detour problem

Experimental data

Thompson et al. (1984) studied the effects of different lesions in detour problem-solving behavior. The apparatus consisted of a startbox and a choice chamber separated from the goalbox by a partition. During preliminary training, animals reach the goal through a door in the partition. After several training trials, the door in the partition is blocked and the goal can be reached only through a new pathway. Thompson et al. (1984) reported that normal animals showed fast detour behavior, whereas frontal, parietal, and HL animals showed increasing degrees of impairment in the task.

Simulation results

Figure 5 portrays the maze used by Thompson et al. (1984) in the detour problem and its representation in the computer model. As before, dashed circles represent places discrimi-


Latent Learning
Group A
Group B
Normals
Moves to Goal
55
45
35
25
15
5
0
2
4
6
8
10
12
14
16
Blocks of 2 Trials
Hippocampal Lesion
Moves to Goal
55
45
35
25
15
5
0
2
4
6
8
10
12
14
16
Blocks of 2 Trials
Cortical Lesion
Moves to Goal
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Blocks of 2 Trials

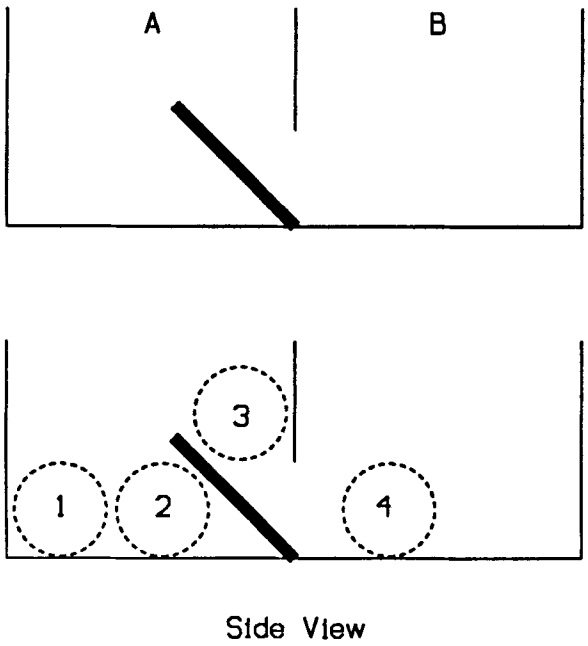

Fig. 5. Detour problem. Diagram of the apparatus employed by Thompson et al. (1984) to study a detour problem. Dashed circles represent different places discriminated by normal animals. Path A is formed by places 1, 2, and 4. Path B is formed by places 1, 3, and 4.

nated by normal animals and used to build a cognitive map. We assume that HL and CL animals build a route based on distinctive cues encountered at those places.

Path A is formed by places 1, 2, and 4. Path B is formed by places 1, 3, and 4. The animal starts each trial from $place_1$, and the US is located at $place_4$. During the first 54 trials of the simulation, the block is not present and the animal takes path A to the goal. Notice that $place_3$ is defined as the region above the barrier, so that prior to introduction of the block, $place_3$ does not exist. On trial 55, the barrier is introduced so that the animal must detour to path B. Figure 6 shows the number of moves to the goal, place–view associations V(2,4), and cue–US association D(2), as a function of trials after the block is introduced.

Normal case In the normal case, animals navigate through the maze with the assistance of the cognitive map. During the first phase of training (first 54 trials), the association V(2,4) between $place_2$ and $view_4$ increases. On trial 55 path A is blocked, so that from the starting location at $place_1$, the animal can choose to enter either $place_2$ or $place_3$. Since V(2,4) is large at the start of trial 55, $place_2$ generates a fast-time prediction of the US. Therefore, $place_2$ is entered by the animal and the barrier is encountered. When the animal discovers that it can no longer reach $place_4$ from $place_2$, the association V(2,4) rapidly decays to zero (upper panel of Fig. 6). Once V(2,4) has decayed, the animal begins exploring and quickly discovers $place_3$ on path B. This causes the association V(3,4) between $place_3$ and $view_4$ to increase, so that on subsequent trials, the animal will choose to enter $place_3$ from $place_1$ instead of entering $place_2$. This simulation result is in agreement with the results of Thompson et al. (1984), showing that normal rats quickly detour when path A is blocked. This fast detour, termed *response flexibility* by Thompson et al. (1984), is the consequence of fast changing associations in the cognitive map system.

It is important to notice that in the normal case, cue–US associations are overshadowed by place–US associations and therefore cue_2 (located at $place_2$) does not accrue an association D(2) with the US. As explained, the animal detours to the new pathway only after the fast-changing V(2,4) association is extinguished. Had cue_2 been associated to the US, the slow extinction of the cue_2–US association would have retarded the detour.

Hippocampal lesion case In the HL case, the cognitive system is disabled, and the animal navigates with the help of the route system. As in the normal case, the HL animal takes path A for 54 trials. However, place–view association V(2,4) does not increase. Since cue–US associations are no longer overshadowed by place–US associations, cue_2 (located at $place_2$) is able to accrue an association D(2) with the US. On trial 55 path A is blocked, so that from the starting location at $place_1$, the animal can choose to enter either $place_2$ or $place_3$. At the start of trial 55, D(2) is large and therefore the animal chooses to approach cue_2. When $place_4$ cannot be reached from $place_2$, the association D(2) begins to decay slowly (upper panel of Fig. 6). The animal returns to $place_1$, but since association D(2) decays very little during the visit to $place_2$, the animal continues to reenter $place_2$ from $place_1$. The lower panel of Figure 6 shows that during the first three trials following introduction of the block, the animal fails to find the goal in the maximum number of allowed moves (26). Finally, on the fourth trial, D(2) has decayed enough that the animal discovers path B. This simulation result is in agreement with Thompson et al. (1984), who show that HL rats take longer than normal rats to detour through the new path. Simulations also reflect the observation of Thompson et al. (1984) that HL rats tend to persevere in nonrewarded strategies.

Cortical lesion case As in the HL case, CL animals navigate with the assistance of the route system. Cue–US associations are not overshadowed by place–US associations, and therefore cue_2 (located at $place_2$) is able to accrue an association D(2) with the US. However, cues are still forced to compete with each other to gain associations with the US, because the intact hippocampus is still able to compute aggregate predictions based on all cues. Therefore, as shown in the upper panel of Figure 6, D(2) does not become as large in the CL case as it does in the HL case. On trial 55 path A is blocked, so that from the starting location at $place_1$ the animal can choose to enter either $place_2$ or $place_3$. At the start of trial 55, D(2) is large enough to predict the US, and therefore the

Fig. 4. Latent learning. Simulated moves to reach the goal as a function of the number of blocks of trials. Animals in group A are rewarded at the goalbox on the first trial, animals in group B are rewarded at the goalbox on the sixth trial. Upper panel: Normal case. Middle panel: Hippocampal lesion case. Lower panel: Cortical lesion case.

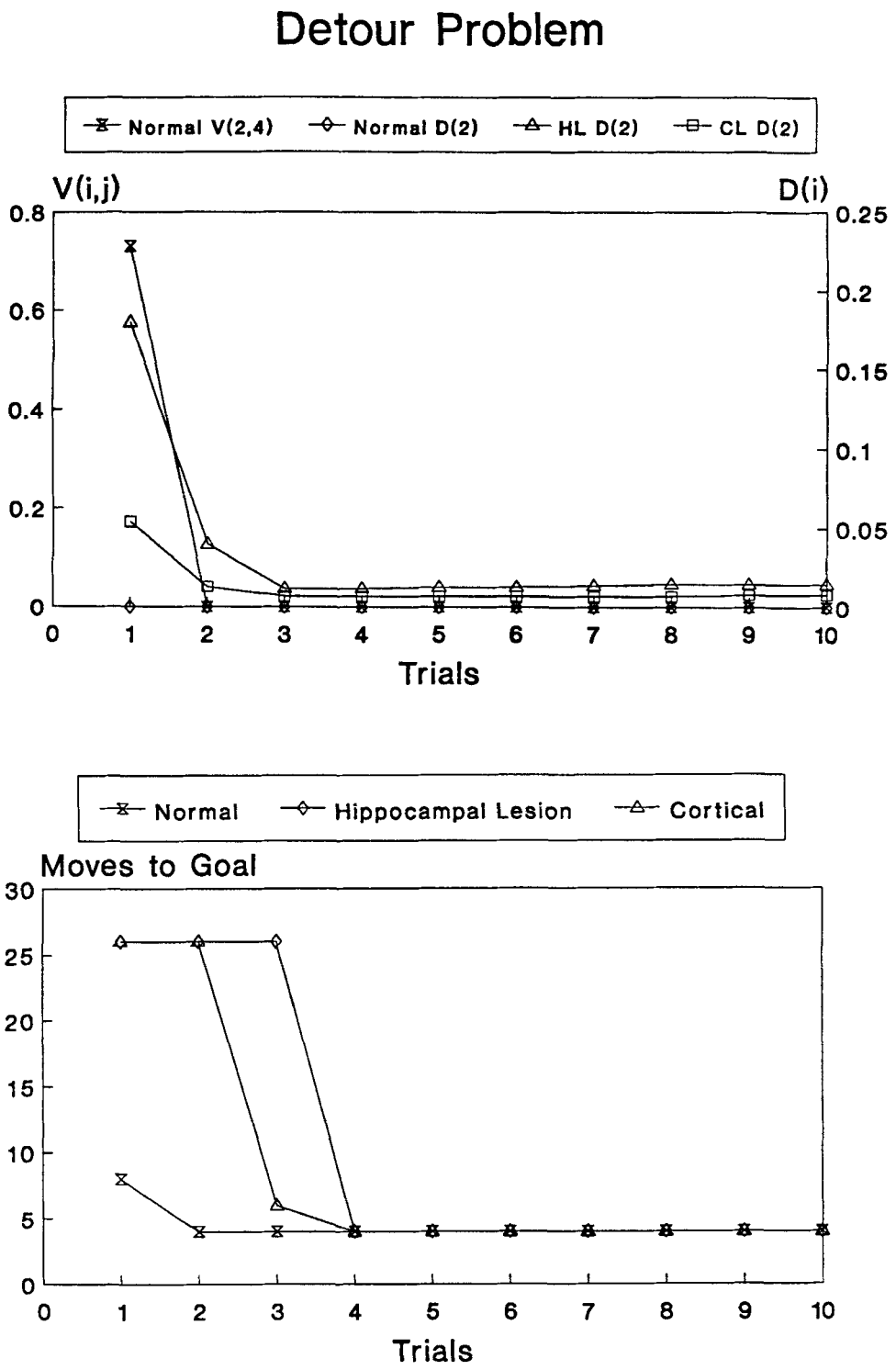

Fig. 6. Detour problem. Upper panel: Place–View and cue–US associative weights as a function of trials. Lower panel: Simulated moves to reach the goal as a function of trials after the block is introduced.

animal chooses to approach cue_2. When the animal encounters the block, the association D(2) begins to decay (upper panel of Fig. 6). The animal returns to $place_1$, but since association D(2) decays slowly, the animal continues to reenter $place_2$ from $place_1$. However, since D(2) does not become as large as it does in the HL case, it takes less time to extinguish, and therefore the animal detours faster. This simulation result is in agreement with Thompson et al. (1984) who show that CL rats take longer than normal rats, but shorter than HL rats, to detour to the new path.

Place and cue learning

Experimental data

During place learning, intramaze cues are minimized by making the maze as homogeneous as possible, and animals must approach a rewarded spatial location by making use of extramaze cues. Conversely, during cue learning, animals must approach an intramaze cue in order to be rewarded.

Whishaw (1991) studied the effect of different training procedures on the performance of normal animals in a task in which rats had to search for a hidden escape platform in a tank filled with opaque water (Morris tank). They found that one very brief exposure to a platform location (goal exposure) is effective in improving performance. Performance is further improved following a single swimming trial (goal approach), and platform placement combined with a swimming trial result in best execution of the task.

Hippocampal lesion Morris et al. (1982) and Sutherland et al. (1982) reported that HL caused a profound impairment in the acquisition of place learning in the Morris tank. In addition, Morris et al. (1990) reported that separate ibotenate hippocampal and subicular lesions initially impair acquisition of place learning, but that with overtraining animals eventually learn the task. Combined ibotenate hippocampal and subicular lesions, however, result in permanent impairment. DiMattia and Kesner (1988) reported that electrolytical HL impair not only acquisition, but also retention of a previously acquired Morris tank place task. Importantly, Sutherland and Arnold (1987) found that retention of place learning is affected by HL only when lesions are performed less than 12 weeks after training.

In contrast to place learning, Morris et al. (1982) reported that HL did not affect acquisition of a cue learning task in which rats had to approach a visible platform to escape from the water.

Cortical lesions Whishaw and Kolb (1984) reported that decortication abolishes place learning, but not cue learning, in rats. Similarly, Schenck and Morris (1985) found that lesions of the entorhinal cortex impair acquisition and retention of place learning. DiMattia and Kesner (1988) found that lesions of the parietal cortex impair Morris tank performance even more severely than HL. Medial frontal cortex lesions disrupt acquisition, but not retention, of place learning in a water maze (Sutherland et al., 1982; Kolb et al., 1983). Cingulate cortex lesions impair place learning in the Morris tank (Sutherland et al., 1988).

Simulation results

Figure 7 depicts a Morris tank and its representation in the computer simulations. Again, dashed circles represent places discriminated by normal animals and used to build the cognitive map employed to navigate toward the submerged platform. Because the platform allows the animal to stay away from the water, it represents a positive reinforcer.

The upper panel of Figure 8 shows the simulated performance of normal animals in a water maze with and without goal preexposure. In the normal case, simulations assumed that goal place–US associations are formed either during goal preexposure or when nonpreexposed animals reach the platform on the first trial. The connectivities between different places in the tank are stored as V_{ij} associations in the cognitive map. Normal animals can approach the platform by using their cognitive maps to generate fast-time predictions of the US. For example, from any of the outlying places the animal can enter either the goal place or an adjacent peripheral place. When the goal place has become associated with the US, the fast-time prediction of the US will be greater when the animal examines the goal place than when it inspects any other place. Therefore, the animal will decide to enter the goal place. Because all peripheral places are adjacent to the goal place, the animal can reach the goal from any peripheral starting place. In agreement with experimental data, preexposed animals performed as well as nonpreexposed animals after one trial.

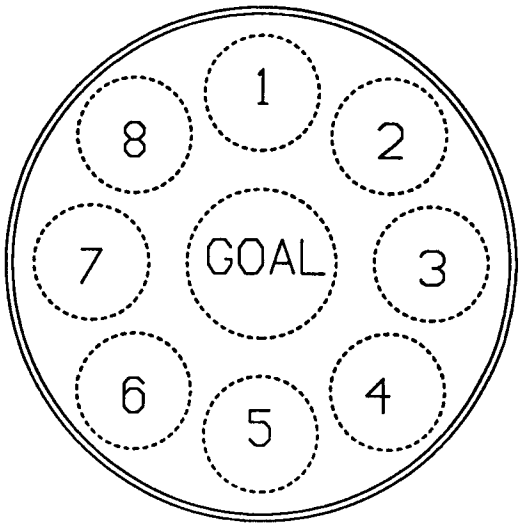

Fig. 7. Water maze. Representation of the maze employed by Morris et al. (1982). Dashed circles represent different places discriminated by normal animals.

The upper panel of Figure 8 also shows simulated performance in a water maze for HL and CL cases. In HL and CL cases, because places cannot be discriminated, animals cannot learn about the hidden location of the platform. In addition, because distinctive cues are absent from the water, HL and CL animals are unable to build a route based on cue–US associations. Therefore, lesioned animals reach the goal by chance. Simulated results are in general agreement with DiMattia and Kesner (1988), Morris et al. (1982), and Sutherland et al. (1982), showing that HL and CL animals take longer than normal animals to reach the goal. However, the present version of the model does not account for the fact that HL animals show some improvement over trials, seemingly by limiting their random search to a circumscribed region of the environment. In agreement with DiMattia and Kesner (1988), the model also predicts that HL impair the retention of water maze learning by allowing distal cues defining the location of the platform to acquire direct associations with the US and thereby impairing place discrimination.

The lower panel of Figure 8 shows that normal, HL, and CL cases show similar performance when the platform is visible. A visible platform becomes an intramaze cue that can be

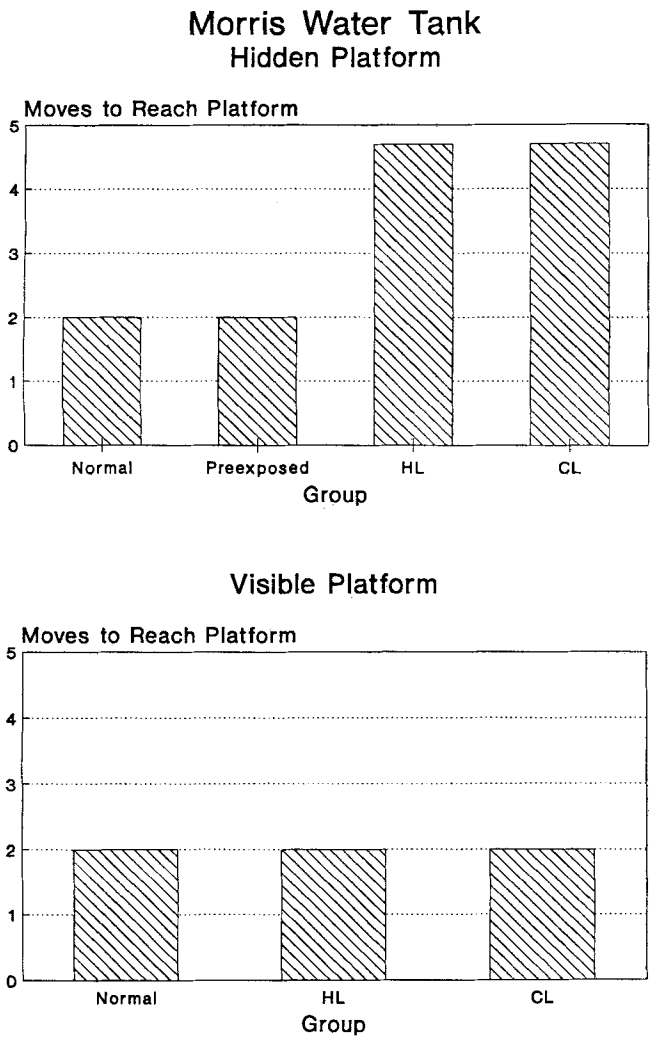

Fig. 8. Water maze. Simulated moves to reach a hidden and a visible platform. Preexposed, normal animals preexposed to the platform; HL, hippocampal-lesioned animals; CL, cortical-lesioned animals.

approached by the animals. In the normal case, goal place–US associations overshadow cue–US associations, and the animal still navigates to the platform using its cognitive map. Although HL and CL animals cannot use their cognitive map, they can still navigate toward the visible platform based on the cue–US associations stored in the route system. Because the goal cue (platform) becomes associated with the US, both HL and CL animals are able to approach the platform. Simulated results are in agreement with data showing that HL and CL animals are not impaired at reaching a visible goal.

DISCUSSION

This study presents a real-time neural network that describes how animals might use cognitive maps and routes to navigate in space. Like O'Keefe and Nadel (1978), we define cognitive maps as sets of connected places. However, our maps encode only the adjacency (place–view associations) not the distance or direction, between places. Also in contrast to O'Keefe and Nadel, we define routes as sets of cue–US associations.

Table 2. Comparison of Experimental and Simulated Results

	Hippocampal Lesion		Cortical Lesion	
	Data	Model	Data	Model
Latent Learning				
Acquisition	0, −	−	?	−
Detour Learning	− −	− −	−	−
Place Learning				
Acquisition	−	−	− −	−
Retention	0, −	−	− −	−
Cue Learning				
Acquisition	+	+	0	0

+, facilitation; −, deficit; 0, no effect; ?, no available data.

In agreement with O'Keefe and Nadel's (1978) view, in the absence of the cognitive map, the route system might still guide maze navigation. However, whereas O'Keefe and Nadel (1978) assume that the cognitive map is stored in the hippocampus, we assume that the hippocampus modulates the formation of cognitive maps in cortical areas. Therefore, removal of the cognitive map might result from HL according to O'Keefe and Nadel's (1978) model or from CL according to our model. Thus, CL simulations in this study can also be interpreted as simulations of the "hippocampal lesion" case according to O'Keefe and Nadel's theory. It is important to notice, however, that because our mapping assumes that HL not only impair cognitive mapping but also the computation of aggregate predictions, our model explains why detour behavior is more impaired by HL than by CL and why cue learning is facilitated by HL but not by CL.

The network describes latent learning, detour behavior, and place learning in simulated normal, HL, and CL animals. Table 2 compares experimental and simulated results. The model correctly describes the effect of HL and CL on the acquisition and retention of place learning, cue learning, and detour learning. For latent learning, the model correctly describes the effect of HL and generates novel predictions regarding the effect of CL.

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APPENDIX I: FORMAL DESCRIPTION OF THE MODEL

Place–View associations

Changes in the representation of place i, x_i, are given by

$$d\,x_i/dt = -k1\,x_i + k2\,(1 - x_i)\,T_i, \quad (1)$$

where k1 is a decay constant and k2 is a rise constant.

T_i is given by

$$T_i = place_i + k3\,f[d(B_i)/dt], \quad (2)$$

where $place_i$ is 1 if the animal is at place i, 0.1 if the animal glances into place i while at another position, and 0 otherwise. k3 is the reinjection constant. $f[d(B_i)/dt]$ is the fast-time prediction of $place_i$. $f[d(B_i)/dt] = d(B_i)/dt$ if $d(B_i)/dt > 0$, and $f[d(B_i)/dt] = 0$ otherwise. Therefore, the representation of place i, x_i, is active when the animal is at place i or when place i is predicted by the network.

The real-time prediction of view j, B_j is given by

$$B_j = \Sigma_j\,V_{ij}\,x_i, \quad (3)$$

where V_{ij} is the association between place i and view j.

Changes in association V_{ij} between place i and view j are given by

$$dV_{ij}/dt = k4\ x_i\ (view_j - B_j)(1 - V_{ij}), \quad (4)$$

where $k4 = k4'$ when $view_j > B_j$, $k4 = k4''$ when $view_j < B_j$, and $k4 = 0$ when $i = j$. Because $d(B_j)/dt$ is a very short (one time unit) pulse generated when the animal glimpses into different places, x_i is essentially proportional to $place_i$ (see equation 2), and therefore V_{ij} reflects the association between $place_i$ and $view_j$.

Place–US and cue–US associations

Changes in the representation of cue i in place i, c_i, are given by

$$d\ c_i/dt = -k5\ c_i + k6\ (1 - c_i)\ cue_i, \quad (5)$$

where k5 is a decay constant and k6 is a rise constant. Cue_i is 1 if the animal is at place i and 0 otherwise. Cue_i is 0.1 and lasts one time unit if the animal glances into place i while at another position and 0 otherwise.

The real-time prediction of the US, B_{US} is given by

$$B_{US} = k6\ \Sigma_j\ D_j\ c_j + k8\ \Sigma_j\ V_{jUS}\ x_j, \quad (6)$$

where $V_{j,US}$ is the association between place j and the US and D_j is the association between cue j and the US. k6 and k8 are coefficients that weight the predictions of the US made upon the cognitive map and the predictions made directly by the cues.

Changes in association D_i between cue i and the US are given by

$$dD_i/dt = k9\ c_i\ (\lambda_{US} - B_{US}), \quad (7)$$

where λ_{US} represents the US intensity, $k9 = k9'$ when $\lambda_{US} > B_{US}$, and $k9 = k9''$ when $\lambda_{US} < B_{US}$. Because c_i is a short pulse generated when the animal glimpses into $place_i$, c_i is essentially proportional to cue_i (see equation 5), and therefore D_i basically reflects the association between cue_i and the US.

Changes in association V_{iUS} between place i and the US are given by

$$dV_{iUS}/dt = k10\ x_i\ (\lambda_{US} - B_{US}), \quad (8)$$

where $k10 = k10'$ when $\lambda_{US} > B_{US}$ and $k10 = k10''$ when $\lambda_{US} < B_{US}$. Because $d(B_j)/dt$ is a short pulse generated when the animal glimpses into different places, x_i is essentially proportional to $place_i$ (see equation 2), and therefore V_{ij} reflects the association between $place_i$ and the US.

Decision making

Before making a decision, the animal briefly enters (for one time unit) all the alternative accessible places h linked to place i and chooses to approach the place with the highest predictive value of the US. This is accomplished by associating a short-term memory of the pulse of the next place or cue, generated by neurons g_h in Figure 1, with $d(\Sigma_j D_j c_j)/dt$ and $d(\Sigma_j V_{jUS} x_j)/dt$. The association of the output of neurons g_h with $d(\Sigma_j D_j c_j)/dt$ and $d(\Sigma_j V_{jUS} x_j)/dt$ represents a working memory, $p_{h,US}$, of the fast-time predictions of the US. Changes in $p_{h,US}$ are given by

$$d(p_{h,US})/dt = -k11 p_{h,US} + g_h(k12\ d(\Sigma_j D_j c_j)/dt + d(\Sigma_j V_{jUS} x_j)/dt). \quad (9)$$

According to equation 5, $p_{h,US}$ increases when alternative h and the fast-time predictions of the US ($k12\ d(\Sigma_j D_j c_j)/dt + d(\Sigma_j V_{jUS} x_j)/dt$) are active together and decreases otherwise. If the largest $p_{h,US}$ is less than a threshold value (k13), the next place is randomly visited. If two $p_{h,US}$ have identical values, the next place is randomly decided between those two places.

APPENDIX II

Effects of hippocampal lesions

Changes in association V_{ij} between place i and view j are given by $dV_{ij}/dt = 0$. Changes in association D_i between cue i and the US are given by

$$dD_i/dt = k9 c_i(\lambda_{US} - D_i c_i). \quad (11)$$

where $k9 = k9'$ when $\lambda_{US} > D_i c_i$ and $k9 = k9''$ when $\lambda_{US} < D_i c_i$.

Effects of cortical lesions

Changes in association V_{ij} between place i and view j are given by $dV_{ij}/dt = 0$. Changes in association D_i between cue i and the US are given by

$$dD_i/dt = k9 c_i(\lambda_{US} - B_{US}). \quad (12)$$

where $k9 = k9'$ when $\lambda_{US} > B_{US}$ and $k9 = k9''$ when $\lambda_{US} < B_{US}$.

APPENDIX III: PARAMETER VALUES

Computer simulations generate values of the relevant variables only at discrete time units. In our simulations we assumed that one time step is equivalent to 1 ms. Parameters used in all simulations were k1 = .99, k2 = .25, k3 = .25, $k4' = 10^{-3}$, k4″ = 15, k5 = $5\ 10^{-3}$, k6 = .25, k7 = 1, k8 = 2, $k9' = 2.5\ 10^{-4}$, $k9'' = 8\ 10^{-4}$, $k10' = 10^{-2}$, k10″ = .128, $k11 = 10^{-4}$, $k12 = 4\ 10^{-6}$, and $k13 = 10^{-8}$. A complete description of the simulation procedures is given in Schmajuk and Thieme (1992).