

that rats learn from conspecifics which foods are preferable by experiencing the pairing of a distinctive (not necessarily novel) food odor with an odorous constituent of rat's breath (carbon disulfide). We have recently found this form of paired-associate learning is blocked by selective elimination of hippocampal neurons, indicating that the learning of paired associates does depend on the hippocampus itself in a situation in which the relevant stimulus relationships are set in a natural context. An important issue here may be that in natural paired-associate learning, as in the typical verbal paired-associate task used in humans, at least one of the elements of the association (the carbon disulfide in rats' breath) has long-standing familiarity and independent significance to the subject. Interestingly, human amnesics can be very successful in implicit learning of new associations when the cues are readily fusible according to natural perceptual arrangements (e.g., associations between color words and the colors in which they are printed).¹⁰ These findings are consistent with the view that, in both rats and humans, different types of representation may predominate depending on the format of stimulus presentation and familiarity with the stimuli, and hippo-

campal involvement may distinguish between these strategies.

FINAL THOUGHTS

Our views on cognitive and neural strategies for the representation of associations parallel aspects of a proposal offered by Gluck and Myers (this issue) in their neural network model of the hippocampal region. They suggest that the hippocampal region mediates both the fusion (what they call "compression") of stimulus representations that are predictively redundant and the differentiation of stimulus representations that are predictively distinct. Based on circuitry constraints of the parahippocampal region, they suggest an anatomical distinction corresponding to these two functions along the same lines as we propose here. The combination of Gluck and Myers's model and our findings on paired-associate learning offers some real hope for progress in understanding the binding problem in memory, suggesting different types of binding may be accomplished by separate brain structures within a single memory system. This account, focused on the interplay of components of the hippocampal sys-

tem and based on explorations of memory representation, suggests that a resolution to that long-standing issue may be emerging from endeavors in cognitive neuroscience.

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Representation and Association in Memory: A Neurocomputational View of Hippocampal Function

Mark A. Gluck and Catherine E. Myers

How do people represent their experiences in memory and then associate these stored representations with their representations of other related experiences? Recent experi-

mental findings suggest that the hippocampus and related structures in the medial temporal lobe play a fundamental role in determining how stimulus events are represented in

Mark A. Gluck is an Assistant Professor at the Center for Molecular and Behavioral Neuroscience at Rutgers University. **Catherine E. Myers** is also at the Rutgers neuroscience center, where she has a postdoctoral fellowship. Address correspondence to Mark Gluck, Center for Molecular and Behavioral Neuroscience, Rutgers University, Newark, NJ 07102; e-mail: gluck@pavlov.rutgers.edu or myers@pavlov.rutgers.edu.

the brain.^{1,2} Previous characterizations of animal learning have noted the importance of hippocampal-region function in diverse behaviors including spatial navigation, contextual processing, and attention.³ Meanwhile, studies of the neuropsychology of human learning have emphasized the importance of the hippocampal region for storing explicit declarative memories marked by their accessibility to conscious recollection.²

Rather than focusing on particular classes of tasks for which the hippocampus is required, we and other investigators have suggested that it is more accurate to say that the hippocampus plays a role in most if not all forms of memory. In some cases, the hippocampus may not be strictly needed, so that some tasks are still learnable after hippocampal lesion; other tasks that do require hippocampal involvement will be disrupted after the lesion. On this view, the critical issue is to identify what function the hippocampus could be computing such that lesioning the hippocampus results in the observed pattern of performance deficits.

One way to proceed is through the use of computational models. A model of intact hippocampal function is compared against a model of hippocampal lesion, across a range of tasks. This hippocampal-region model may have a high degree of anatomical and physiological realism, or it may be intended only to capture the basic functionality. If the model includes an accurate representation of hippocampal-region function, then removing this process from the model should alter the model's performance on the same tasks in which hippocampal-region damage compromises animal performance. In this article, we review some of our efforts to describe such a hippocampal function and implement it in a computational model that correctly describes cortico-hippocampal interaction in associative learning and memory.

A COMPUTATIONAL THEORY OF HIPPOCAMPAL FUNCTION

We have recently presented a computational theory of hippocam-

pal-region function in elementary associative learning.⁴ Central to this account is the idea of a stimulus representation, the internal pattern of activity evoked by a stimulus input. This representation is presumed to be distributed over many elements, which could be neurons in a brain or nodes in a connectionist network, which models psychological processes by means of a network of neuronlike elements. Within this conceptual framework, learning about stimuli is equivalent to associating the representations these stimuli activate with appropriate behavioral outputs. This conceptualization of representation and association can be illustrated by examining how a simple multilayer connectionist network learns a set of stimulus-output associations. In such a network, shown in Figure 1, inputs are mapped through weighted connections to a representation in an internal layer of nodes; associations are formed between these representations and an appropriate output.

Learning about one stimulus will generalize to other stimuli as a function of the similarity (or overlap) between their representations. If the representations of two stimuli are very similar, associations that have accrued to one stimulus will tend to generalize to the other. If the representations are very dissimilar, the associations to one stimulus will generalize only minimally to the other. Learning will be facilitated if the representations are biased by two constraints that can be characterized as follows: First, stimuli that are to be associated with maximally different outputs should have minimally overlapping representations, so as to lessen the generalization between them. We call this constraint representational *differentiation*. Second, stimuli that co-occur and are thus likely to have the same consequence should have similar representations so as to increase generalization between the stimuli. We call this a bias to *compress* the representations of

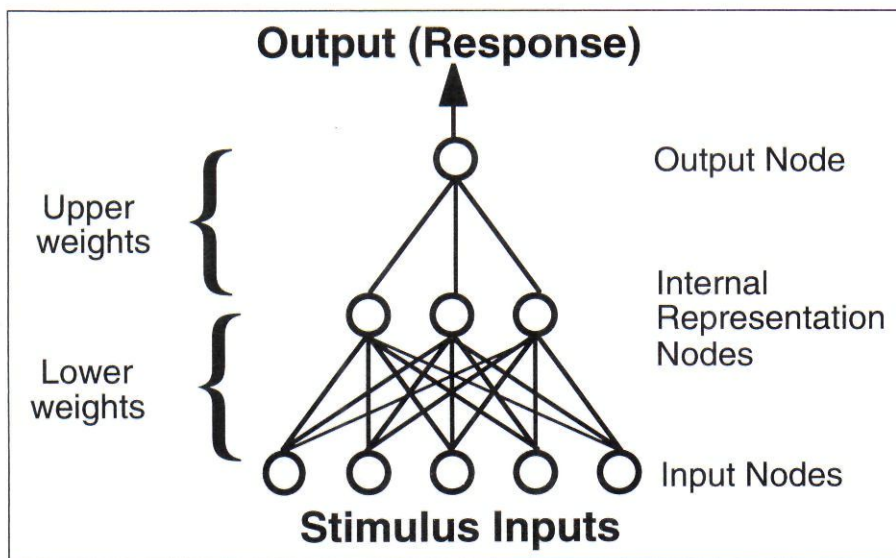


Fig. 1. A multilayer connectionist network containing three layers of nodes with weighted connections between layers. The input layer is activated by stimulus inputs; activations in the internal, or middle, layer are a function of weighted sums of the input activations. The internal-layer activations form a new representation of the input. The output-node activation is likewise a function of weighted sums of internal-node activations, and is the system response. All connection weights are plastic, and their adaptation represents learning in the system.

co-occurring, mutually redundant information.

The core of our proposal is that the hippocampal region has the ability to construct new stimulus representations that are biased by the constraints of differentiation and compression. Other regions in the cerebral and cerebellar cortices, which are presumed to be the sites of long-term memory, may not be able to form appropriately biased representations themselves; these other regions can, however, adopt the new representations formed in the hippocampal region.

This theory can be instantiated, and tested, with a simple connectionist network model of classical Pavlovian conditioning.⁴ In classical conditioning, a response-evoking stimulus (unconditioned stimulus, or US) is repeatedly paired with a previously neutral cue (conditioned stimulus, or CS); with sufficient pairing, the CS alone can come to evoke a preparatory response. The hippocampal network (on the right in Fig. 2a) learns to map from patterns of activation of the input nodes, repre-

senting the presence of CSs, to outputs that contain a reconstruction of each input plus a prediction of external feedback, such as US arrival. The internal layer of nodes in this network (the middle layer shown in the hippocampal-region network in Fig. 2a) is assumed to be narrower than the input layer that drives it; this narrowing forces the network to form an internal layer representation that compresses redundant information while differentiating information that predicts different external feedback (different USs). A training algorithm that accomplishes this compression and differentiation is error backpropagation (a well-known rule for updating associative weights in multilayered connectionist networks), although other training regimes are equally possible.^{4,5}

The cortical network on the left in Figure 2a represents a highly simplified model of some aspects of long-term memory in the cerebral and cerebellar cortices. This network takes the stimulus input and maps it through weighted connections to an internal layer and, from there, to an

output node. The activation of the output node is the system's behavioral response—a prediction of US arrival. This cortical network adapts its weighted connections by a simple incremental learning rule, such as the least-mean-squares (LMS) algorithm, which is a generalized form of Rescorla and Wagner's⁶ trial-level model of classical conditioning. This rule computes error according to the difference between the output-node activation and the actual external feedback it is to predict, and distributes this error among the top layer of weights (i.e., adjusts these weights) according to how strongly various internal-layer nodes are activated (i.e., nodes sending strong signals have their weights adjusted more than nodes sending weak signals). This rule does not directly allow training the lower layer of weights because the error is not defined for the internal-layer nodes; there is no way to determine what their activations should be per se. However, if the activations of the internal layer of the hippocampal-region network are taken to provide external feedback to the internal layer of the cortical network, the LMS rule can be used to adjust the lower layer of weights. To avoid requiring one-to-one connections between nodes in the hidden (i.e., internal) layers of the two networks, many-to-one connections are used. That is, a hidden node in the cortical network does not respond the same way as a particular hidden node in the hippocampal network, but instead learns to produce an output that is a linear recombination, or weighted sum, of the outputs of several hidden nodes in the hippocampal network. Over time, the cortical network will then come to adopt the new representations formed in the internal layer of the hippocampal-region network.

A hippocampal lesion is simulated in this model by disabling the hippocampal network (Fig. 2b). The cortical network remains intact but, in the absence of the hippocampal

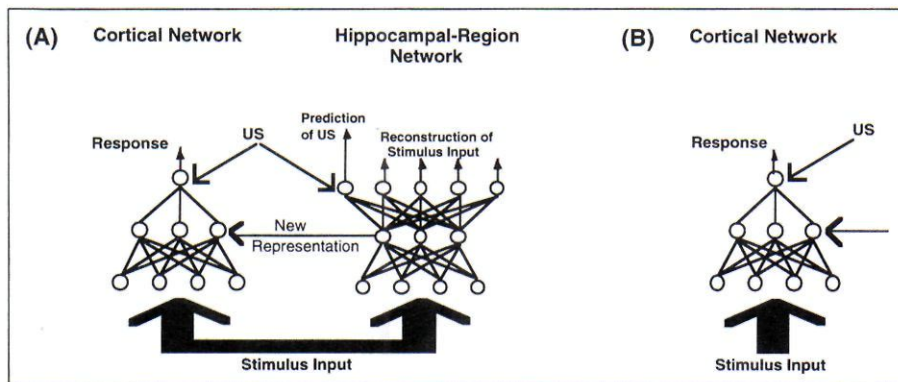


Fig. 2. A model of hippocampal-region function in classical conditioning, based on connectionist networks like that in Figure 1. (a) In the intact model, a hippocampal-region network learns to map from stimulus input through a narrow internal layer of nodes, to output nodes that reconstruct the input plus a prediction of the response-evoking unconditioned stimulus (US). In the process, the internal layer of this network forms new stimulus representations that are biased to compress redundant information and differentiate predictive information. A simpler cortical network, assumed to be the site of long-term storage, can learn to produce a behavioral response that anticipates and predicts the US, but cannot independently construct new representations in its internal layer. However, it can acquire internal representations from the hippocampal-region network. (b) In the lesioned model, the cortical network can still learn new behavioral responses based on whatever representations exist a priori in its internal layer, but can no longer acquire new representations in the absence of the hippocampal-region network. After Gluck and Myers.⁴

network, can no longer train its lower layer of weights. It can, however, still train its upper layer of weights to map from preexisting internal representations to new responses. Together, the intact model of Figure 2a and the lesioned model of Figure 2b have been shown to predict accurately a wide range of learning and generalization behaviors in intact and lesioned animals.^{4,7}

For example, normal intact animals show latent inhibition, in which preexposure to a cue retards later association of that cue with a response; broad hippocampal-region damage abolishes or attenuates the latent inhibition effect.⁸ The intact model shows this effect (Fig. 3a) because during the preexposure phase, the hippocampal network constructs a new representation that compresses, or deemphasizes, the unreinforced cue; this representation retards subsequent learning about that cue. The lesioned model does not show latent inhibition because the model cannot create new,

compressed representations during the preexposure phase.⁴

WHEN IS THE HIPPOCAMPUS INVOLVED?

This computational theory of hippocampal-region function can also help resolve some seemingly paradoxical experimental data. For example, it has long been known that hippocampal lesion can facilitate acquisition of some simple tasks, such as discrimination of successively presented odors.¹ This same facilitation is shown in our lesioned model: Whereas the intact model is required to form new representations for the stimuli to be discriminated, the lesioned model simply acquires new associations based on its existing representations. If the task is simple enough that these existing representations suffice, the lesioned model learns faster.⁴ Although the intact system may learn more slowly, it is learning more be-

cause it is forming new representations as well as new associations (Fig. 3b).

This example points out a critical difference between our account and some earlier characterizations of hippocampal function that focused on distinguishing tasks in which the hippocampus is involved from tasks in which it is not involved. In our model, the hippocampus is always active, forming new stimulus representations even during the most elementary associations. An implication is that whether intact or lesioned animals learn a task faster may not be the most informative measure. Rather, by employing a task that can be learned by both intact and lesioned animals, we can compare their performance on subsequent transfer tasks as a means of indirectly inferring the representations (or rules) by which the animals solved the initial training task. This account is compatible with a previous suggestion by Eichenbaum and colleagues that animals with hippocampal lesions are characterized by an inability to apply learned knowledge flexibly in a new situation.¹

Another seemingly paradoxical experimental result can be understood in a similar way: Whereas outright hippocampal lesion may facilitate discrimination, electrical or chemical disruption of the hippocampus disrupts learning severely.⁹ In our model, hippocampal disruption may be approximated by adding random noise to the hippocampal network. This noise in turn disrupts the representation signals sent from the hippocampal network to the cortical network, thereby causing the cortical network to develop internal representations that change continually and randomly. The cortical network tries to form associations based on these meaningless internal representations, and learning is slowed or impossible (Fig. 3b). These results indicate further that the hippocampal region is always involved in discrimination learning, even in the

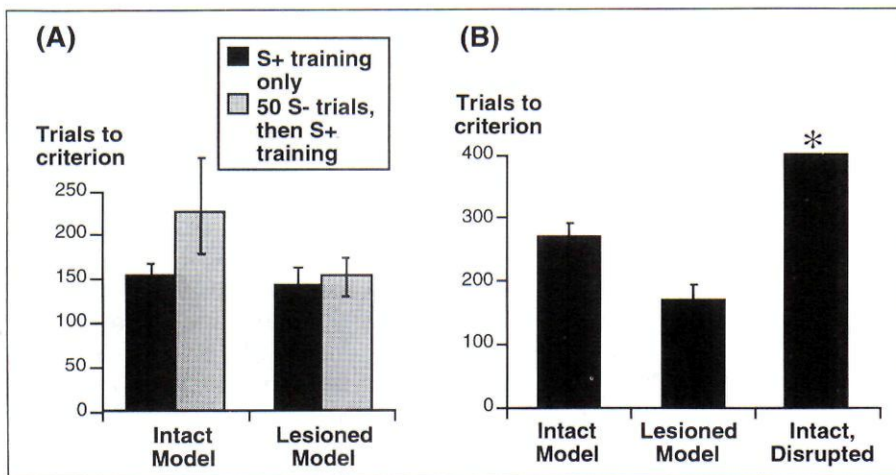


Fig. 3. Behavioral results in the intact and lesioned models. (a) In the intact model, learning (measured as number of trials to reach a criterion level of performance) of a response to a reinforced stimulus, S+, is slowed when preceded by 50 trials of unreinforced preexposure to the same stimulus (the S- trials). This latent inhibition phenomenon is not shown in the lesioned model. After Myers, Gluck, and Granger.⁵ (b) Learning to discriminate two cues is slightly faster in the lesioned model than in the intact model, in terms of number of trials needed to reach a performance criterion. Leaving the hippocampal-region network intact but disrupting its output results in a severe retardation of learning. The asterisk indicates that simulations were terminated if a criterion level of correct responding had not been reached by 400 trials. After Gluck and Myers.⁴

simplest tasks, such as stimulus discrimination and acquisition.

DISSOCIATING HIPPOCAMPAL AND ENTORRHINAL FUNCTION

Although it is important to understand the aggregate functional role of the hippocampal region, ultimately we would like to know how the proposed behavioral processes map onto more detailed anatomical structures. Figure 4 shows a schematic of some key information pathways in the hippocampal region. Of particular importance is the parahippocampal region, comprising ento-

rhinal, perirhinal, and parahippocampal cortices; these structures are the primary sites through which sensory information both enters and leaves the hippocampal formation.

In particular, we have focused on the entorhinal cortex. Drawing upon a previous computational model of olfactory cortex,¹⁰ which shares many physiological and anatomical characteristics with superficial entorhinal cortex, we have suggested that the entorhinal cortex implements similarity- and redundancy-based compression of stimulus representations.⁵ If the entorhinal cortex performs redundancy compression, the remaining postulated hippocampal-region function, predictive differentiation, would most likely be implemented elsewhere in

the hippocampal region, possibly in the hippocampal formation itself. The net result of both entorhinal and hippocampal processing would then be a new stimulus representation that is constrained by both compression and differentiation biases and that could be transferred to long-term storage sites in the cortex. Figure 5 illustrates this conjectured dissociation of parahippocampal and hippocampal function.

How can this hypothesis be tested? If there is division of function between the parahippocampal region and the hippocampal formation, then it is possible that a lesion selectively restricted to one area might leave processing in the other area relatively unaffected. New surgical techniques have recently made such selective lesions possible, and there is experimental evidence showing that at least some parahippocampal processing can survive lesions limited to the hippocampal formation.¹¹

According to our conjecture, such a selective hippocampal-formation lesion should interrupt predictive differentiation but not redundancy compression. Thus, a behavioral phenomenon assumed to depend on the latter process should survive the lesion. One such effect is latent inhibition, as described earlier; consistent with experimental data, the intact model of Figure 2a shows latent inhibition, but the fully lesioned model of Figure 2b does not. This effect has been explained in terms of redundancy compression, but not differentiation, in the intact model; therefore, latent inhibition should depend more on the entorhinal cortex than on the hippocampal formation. Consistent with this expectation, recent experimental evidence has shown that latent inhibition is not disrupted by selective hippocampal damage, although it is attenuated by broader hippocampal-region damage.⁸ Obviously, more experimental tests need to be performed, but this is at least initial

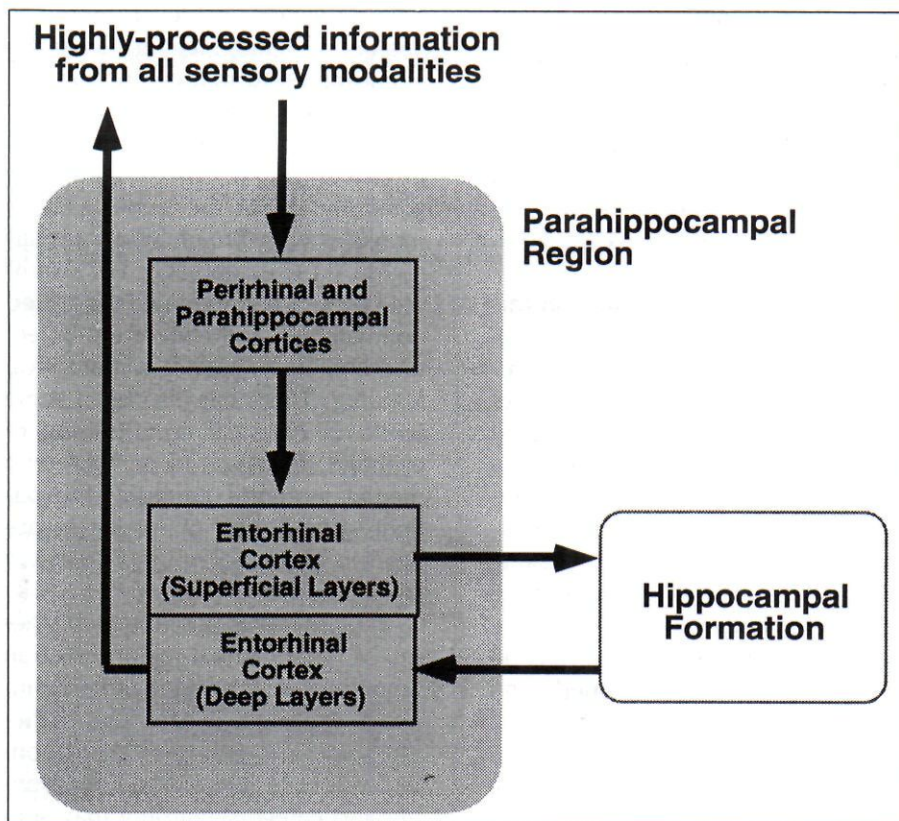


Fig. 4. Schematic and highly simplified representation of information pathways in the hippocampal region. Highly processed, multimodal sensory information travels from association and limbic cortex to the parahippocampal region, consisting of the parahippocampal, perirhinal, and entorhinal cortices. From there, information travels through the hippocampal formation (including dentate gyrus, hippocampus, and subiculum), before returning to entorhinal cortex and thence back to the same cortical areas from which it came originally.

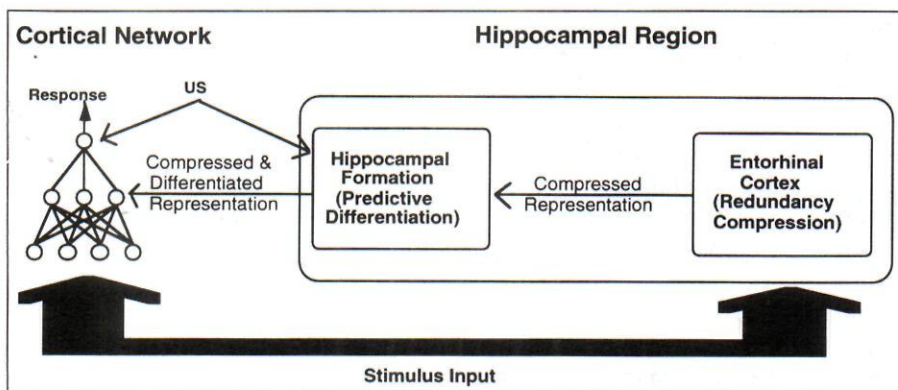


Fig. 5. A preliminary model of how hippocampal-region processing might be distributed among the anatomical structures within the region. The anatomy and physiology of entorhinal cortex are consistent with a redundancy compression function; the hippocampal formation may be left with the task of predictive differentiation. The model shown assumes that these two structures operate sequentially: First, the entorhinal cortex compresses stimulus inputs to remove redundancy, and then the hippocampal formation takes the result and differentiates information that is differentially predictive of the unconditioned stimulus (US). The final resulting representation is provided to the cortical network just as in the original model of Figure 2a.

support for the hypothesis schematized in Figure 5.

COMPARISON WITH OTHER THEORETICAL APPROACHES

Eichenbaum, Otto, and Cohen¹² have also addressed the selective contribution of parahippocampal structures (including entorhinal cortex) to hippocampal-region processing. They suggest that the parahippocampal region functions as an intermediate-term buffer, maintaining memories for the duration of a task. Although this intermediate-term memory differs from the clustering function we have proposed, the two functions are complementary. Events that should be treated as co-occurring may not co-initiate or co-terminate precisely; intermediate-term storage of recent events might allow the parahippocampal region to cluster stimuli that reliably occur nearly together in time. Conversely, the intermediate-term store most likely needs to perform some sort of clustering to reduce the information passing through it. Anatomical analyses suggest that the entorhi-

nal cortex contains many more inputs than outputs, supporting our suggestion that it compresses information into a more efficient signal.⁵ Thus, although these two hypotheses focus on different aspects of parahippocampal function, we feel these different interpretations are complementary and possibly interdependent.

In their companion article in this issue, Eichenbaum and Bunsey suggest that the parahippocampal region also mediates the "fusion" of co-occurring or nearly coincident stimuli; this process is functionally identical to the redundancy compression function we have proposed. It is interesting to note that whereas their fusion theory derives from behavioral observations comparing paired-associate learning in intact, hippocampal-lesioned, and parahippocampal-lesioned animals, our similar compression theory arises from an integration of both physiologically based and behaviorally based computational models of hippocampal-region function. We hope the convergence of these two widely different approaches to theory development is a sign that stimulus compression is a useful and

accurate description of parahippocampal-region function.

IMPLICATIONS FOR COGNITIVE NEUROSCIENCE OF MEMORY

Although most of the lesion data discussed here focus on animal learning, the end goal is to relate these data to a theory of hippocampal-region function in humans. One problem with comparing results in humans and animals is that hippocampal damage in humans usually results from stroke, Alzheimer's disease, or other brain insults that cause diffuse neuropathology. Structures within the hippocampal region may be only partially or unilaterally damaged, and damage almost always extends to other structures outside the region. However, there has been considerable recent success in equating performance in humans and monkeys with hippocampal-region damage.² Thus, humans with hippocampal-region dysfunction generally fail at the same tasks as monkeys with broad hippocampal-region (H++) damage: For example, they are impaired in delayed nonmatching and concurrent object discrimination, but not in motor skill learning. There has also been some work on classical conditioning of eyeblink responses in patients with medial temporal amnesia; the patients' acquisition of responses appears to be normal, much like that of H++ animals.¹³

Thus, it seems possible that a theory of hippocampal-region function in animals may be applicable to humans. We note, in fact, that earlier research on behavioral correspondences between classical conditioning and category learning may suggest possible avenues for integrating animal and human models of hippocampal-region function through the study of amnesic performance on probabilistic category-learning tasks.¹⁴

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The Articulated Thoughts in Simulated Situations Paradigm: A Think-Aloud Approach to Cognitive Assessment

Gerald C. Davison, Sandra G. Navarre, and Ralph S. Vogel

What's on your mind? What were you thinking when you encountered that situation you've been dreading? How did you arrive at that conclusion? These are but a few of the questions that cognitive and clinical

Gerald C. Davison is Professor of Psychology at the University of Southern California. **Sandra G. Navarre** and **Ralph S. Vogel** are graduate students there in the clinical psychology program. Address correspondence to Gerald C. Davison, Department of Psychology, University of Southern California, Los Angeles, CA 90089-1061; e-mail: gdaviso@almaak.usc.edu.

psychologists have put to subjects and patients for many years and in many different contexts. The answers to such queries inform psychologists' speculations about the role of thought in emotion and behavior.

In general, cognitive scientists have developed performance measures to infer cognitive processes. Experimental situations are created to make inferences about underlying cognitive processes. In contrast, clinical psychologists have tended to rely on clients' introspective and retrospective self-reports in interviews or on questionnaires.

The core assumption in cognitive-

behavioral theory, research, and practice is that affective and behavioral responses are mediated by thought processes, both conscious and unconscious.¹ Operating within this paradigm, researchers have focused in recent years on the development of cognitive assessment methods, but with little consensus in approach to the measurement of cognition. As a result, vastly different modes of cognitive assessment have proliferated, without adequate attention to validity issues. Indeed, measures of the same constructs often do not correlate highly.²

One mode of cognitive assessment, the think-aloud approach, is viewed as of particular value in accessing the products as well as the processes of cognition. Because think-aloud methods assess cognitions concurrently with their occurrence, they may be better suited to tapping actual thought content than other modes are. Standard think-aloud methods have subjects verbal-

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