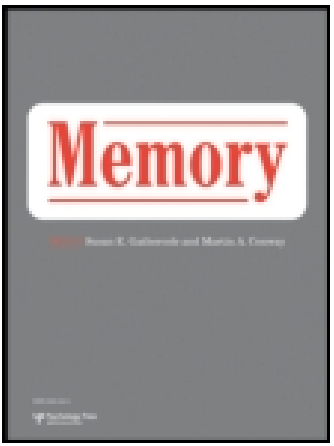

Memory
Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/pmem20

Implicit Memory: What Must Theories of Amnesia Explain?

Tim Curran Daniel L. Schacter

Published online: 15 Oct 2010.

To cite this article: Tim Curran Daniel L. Schacter (1997) Implicit Memory: What Must Theories of Amnesia Explain?, Memory, 5:1-2, 37-48, DOI: 10.1080/741941153

To link to this article: http://dx.doi.org/10.1080/741941153

MEMORY, 1997, 5 (1/2), 37–47

Implicit Memory: What Must Theories of Amnesia Explain?

Tim Curran

Case Western Reserve University, Cleveland, USA

Daniel L. Schacter

Harvard University, USA

In their target article on explaining functional deficits in amnesia, Mayes and Downes (this issue) discuss the relevance of implicit memory. Our commentary considers a number of implicit memory phenomena that may be especially pertinent to understanding the functional deficits of amnesia. Recent evidence suggests that amnesic patients do not benefit normally from an exact perceptual match of stimuli between study and text. We propose that this impairment may reflect one manifestation of a more general deficit in associative binding of information across different brain subsystems. This idea helps to clarify the distinction between implicit and explicit memory, and suggests that studies of implicit memory can help to elucidate the functional deficits in amnesia.

INTRODUCTION

Mayes and Downes (this issue) make several points about the relevance of implicit memory to understanding the nature of functional deficits in amnesia. We believe that one issue that they raise—the distinction between implicit memory for "old" and "new" information—is particularly important theoretically. Mayes and Downes point out that amnesic patients have consistently shown normal implicit memory for old (i.e. previously familiar) stimuli that are represented in memory prior to an experiment. However, Mayes and Downes note that amnesic patients may not show normal implicit memory for novel information that does not have a pre-existing representation in memory.

In this commentary, we consider recent evidence that bears on the question of whether amnesic patients can exhibit implicit memory for novel information by

Requests for reprints should be sent to Daniel L. Schacter, Department of Psychology, Harvard University, 33 Kirkland St., Cambridge, MA 02138, USA.

Preparation of this commentary was supported by grant R01 NH45398-01 A3 from the National Institute of Mental Health to D.L. Schacter and by NINDS program project grant NS26985 to Boston University.

focusing on studies of repetition priming. We note several experiments in which amnesics have shown priming deficits and attempt to characterise the processes responsible for those deficits. Some have gone so far as to argue that priming is generally impaired in amnesic patients and that the medial temporal lobe/diencephalic system that is damaged in amnesia plays a critical role in both implicit and explicit memory (Ostergaard & Jernigan, 1993). By contrast, we will argue that the medial temporal lobe/diencephalic system may play a limited role in priming that can help to clarify, rather than undermine, the distinction between implicit and explicit memory. Furthermore, we think that the characterisation of priming impairments can help to elucidate the nature of functional deficits in amnesic patients.

PRIMING OF FAMILIAR AND NOVEL INFORMATION

Mayes and Downes (this issue) conclude that priming of previously familiar stimuli is spared in amnesia, whereas priming of novel information may be impaired. As noted by Mayes and Downes, evidence for impaired priming of novel information has been best documented in experiments testing memory for novel associations between pre-experimentally unrelated words (e.g. Schacter & Graf, 1986; Shimamura & Squire, 1989; for review, see Bowers & Schacter, 1993). However, comparing priming of novel associations with single-word priming confounds two factors: novelty and association. That is, amnesics may show impaired priming of novel associations because they are unable to acquire any novel information or because they are unable to form the requisite inter-word associations. Other evidence suggests that amnesics can show implicit memory for novel information when there is no associative requirements, as with stimuli such as pseudowords (Haist, Musen, & Squire, 1992; Musen & Squire, 1991), novel objects (Schacter, Cooper, Tharan, & Rubens, 1991; Schacter, Cooper, & Treadwell, 1993), and novel visual patterns (Gabrieli, Milberg, Keane, & Corkin, 1990; Musen & Squire, 1992). Of course, it can be argued that priming of these novel stimuli takes place at a sub-stimulus level that taps pre-experimentally familiar features (e.g. letters composing pseudowords or geons composing novel objects; Bowers & Schacter, 1993). Nevertheless, it seems clear that the best documented impairments of priming for novel stimuli all include an associative component. Therefore, priming deficits in amnesic patients seem more likely to be related to associative factors rather than to novelty *per se*.

This associative priming deficit may reflect amnesics' inability to form what Mayes and Downes refer to as ''complex associations''. A number of researchers have emphasised that a major function of the medial temporal lobe/diencephalic system is related to the formation of complex associations between multiple stimuli (see the chapters in Schacter & Tulving, 1994). Here

we discuss some specific ideas about what might constitute ''complex associations'', but first we will mention two recently documented priming impairments in amnesic patients that may also reflect an associative deficit in amnesia—even though the paradigms that were used are not typically thought of as involving associative factors. These experiments suggest that amnesics do not benefit normally from an exact perceptual match of target materials between study and test.

In one experiment, study–test typography was manipulated in a word fragment completion paradigm (Kinoshita & Wayland, 1993). Control subjects, but not Korsakoff amnesics, showed significantly greater priming when typography was the same at study and test than when it was different. It is possible that Kinoshita and Wayland's control subjects showed a typography effect because they made use of intentional retrieval processes that are not available to Korsakoff patients. This possibility cannot be ruled out, because Kinoshita and Wayland did not test explicit memory.

A similar result has recently been obtained in a study of auditory priming that examined whether amnesic patients exhibit voice-specific priming—more priming when speaker's voice is the same at study and test than when it is different. Schacter, Church, and Bolton (1995) examined auditory priming with a low-pass filter identification test, which had previously yielded evidence of voice-specific priming in college students under conditions in which intentional retrieval can be ruled out as the source of the effect (Church & Schacter, 1994). In Schacter et al.'s experiment, amnesic patients of mixed etiologies and control subjects heard words spoken in one of six different voices. At test, the words were degraded by low-pass filtering. Half of the words were presented in the same voice as during study, and half were repaired with a different voice from the study list. Schacter et al. found that control subjects, but not amnesic patients, showed more priming in the same-voice condition than in the repaired-voice condition (Table 1A). These observations, like Kinoshita and Wayland's failure to observe font-specific visual priming in Korsakoff patients, raise the possibility that some perceptual specificity effects in priming depend on brain systems that normally subserve explicit memory. Although modality specificity has been previously observed in amnesics (Carlesimo, 1994; Graf, Shimamura, & Squire, 1985), the apparent lack of within-modality specificity may reflect a medial temporal lobe/diencephalic contribution to font- and voice-specific effects under the experimental conditions used by Kinoshita and Wayland and by Schacter et al.

A more recent study provides suggestive evidence that amnesic patients can exhibit voice-specific priming under different experimental conditions than those employed by Schacter et al. In two experiments by Schacter and Church (1995), words were spoken by one of two voices during the study task. On a subsequent low-pass filter identification test, studied words were spoken either by the same voice used at study or by an entirely novel, unfamiliar voice. Thus,

TABLE 1
Proportion Correct in Auditory Filter Identification Experiments

A: Schacter, Church, & Bolton (1995)	*Studied Words*		
	Same Voice	*Repaired Voice*	*Nonstudied Words*
Amnesic Patients	0.35	0.43	0.30
	(0.05)	(0.13)	
Control Subjects	0.56	0.40	0.36
	(0.20)	(0.04)	
B: Schacter & Church (1995), Experiment 1	*Studied Words*		
	Same Voice	*Unfamiliar Voice*	*Nonstudied Words*
Amnesic Patients	0.49	0.44	0.34
	(0.16)	(0.11)	
Control Subjects	0.61	0.54	0.44
	(0.17)	(0.10)	
C: Schacter & Church (1995), Experiment 2	*Studied Words*		
	Same Voice	*Unfamiliar Voice*	*Nonstudied Words*
Amnesic Patients	0.76	0.69	0.57
	(0.19)	(0.12)	
Control Subjects	0.73	0.65	0.51
	(0.22)	(0.15)	

Values in parentheses are priming scores obtained by subtracting the nonstudied identification rate from each studied condition.

rather than repairing familiar voices from study to test, as was done in the Schacter et al. experiment, all words were tested in either the same voice or in a new voice. In this new-voice design, control subjects and a mixed-etiology group of amnesic patients showed very similar patterns of priming (Table 1B & C). Although the voice effect was nonsignificant in Experiment 1 and marginally significant in Experiment 2, the pattern and magnitude of priming effects in amnesics and controls were nearly identical (difference between Experiments 1 and 2 are reconsidered later in this paper).

To understand the difference between the repaired-voice design and the new-voice design, we have found it useful to reconceptualise such within-modality perceptual specificity effects in terms of associative binding between perceptual cues and abstract word forms. That is, the particular perceptual features of the word during the study episode (e.g. font, voice) are linked together with either an orthographic word form or a phonological word form; perceptual specificity effects on a subsequent implicit test reflect this binding. Thus, amnesics may be able to show perceptual specificity effects when studied words are unambigu-

ously associated with familiar perceptual formats (Schacter & Church, 1995). When words and voices are repaired from study to test, amnesics do not appear to benefit from the same-voice compared to the repaired-voice conditions (Schacter et al., 1995). Therefore, when familiar voices cue both studied and nonstudied words, amnesics may lack the necessary ability to bind voices with specific studied words.

Viewed from this associative binding perspective, the repaired-voice experiment is much like experiments investigating priming of novel inter-word associations. In the novel association paradigm, subjects who exhibit an associative priming effect benefit from specific associations between a cue word and a target word. In the repaired-voice paradigm, subjects who exhibit voice-specific priming benefit from specific associations between a voice and a target word. Experiments testing novel-association priming are more analogous to the repaired- than the new-voice design, because cues and targets are repaired between study and test in associative priming experiments. In the new-voice design, the associative binding requirements may be less complex or absent. Unfortunately, nothing akin to the new-voice design has been used to study novel word associations in amnesics. Further evidence from experiments using designs analogous to the new-voice design in auditory priming are needed before we can offer confident conclusions concerning the conditions under which amnesic patients do and do not exhibit specificity effects in priming. Moreover, even with normal subjects, perceptual specificity studies have yielded notoriously inconsistent results (for review, see Roediger & McDermott, 1993).

MECHANISMS OF PRIMING IN AMNESIA

Although existing data must be interpreted cautiously, we believe that some theoretical speculation is warranted and may help to stimulate future investigation concerning the mechanisms involved in priming of novel and familiar information in amnesic patients. For instance, Ratcliff and McKoon (1988) have presented a compound-cue theory of priming that might usefully explain the difference between the new- and repaired-voice designs that we considered in the preceding section. Assume that priming is a function of the match between information in memory and the available retrieval cues. In a hypothetical repaired-voice design, the subject studies two words in two different voices (v1-A, v2-B), and then degraded versions of the words (a, b) are tested in either the same voice (v1-a) or the different voice (v2-a). If priming is a linear function of the match between individual words and individual voices—that is, cues are used additively—v2-a and v1-a would be equally primed. In both cases, the test stimulus consists of a primed word and a familiar voice, so the overall amount of additive activation is identical in the two scenarios. Consider, however, an alternative scenario in which voices and words are interactively (or nonlinearly) combined—during study, test, or both. Now, the

combination of v1-a would give much stronger priming than v2-a, and a voice-specific priming effect would be observed. Thus, amnesics may lack the normal ability to use cues interactively (see also Humphreys, Bain, & Pike, 1989). In a new-voice design (study: v1-A, v1-B; test: v1-a vs. v2-a) additive cue use would give stronger priming of v1-a than v2-a, so amnesics may show some benefit.

As previously noted, associative priming deficits are generally consistent with the notion that amnesics are unable to encode, store, or retrieve complex associations. From a cognitive neuroscience perspective, this deficit may reflect an inability to bind or integrate information from different information-processing systems. For example, various priming effects appear to depend on perceptual brain mechanisms that process specific types of information (Schacter, 1990a, 1994; Tulving & Schacter, 1990). In the visual domain, priming might be subserved by both a right-hemisphere subsystem that processes low-level visual attributes and by a left-hemisphere subsystem that processes visual word forms at a more abstract level (Marsolek, Kosslyn, & Squire, 1992). Visual-specificity effects might reflect the interactive activation of these mechanisms. In auditory word priming, an abstract auditory word form subsystem interacts with an acoustic subsystem that handles prosodic information (e.g. fundamental frequency and other spectral information about a voice, Church & Schacter, 1994). If these mechanisms additively contribute to priming, their combined influence may only be observable under restricted situations (e.g. new-voice design). However, the interactive combination of voice and abstract word form information can produce voice-specificity effects in normal subjects.

Our proposal is much like other recent theories of the medial temporal lobe contribution to explicit memory (Cohen & Eichenbaum, 1993; Cohen, Poldrack, & Eichenbaum, this issue; Johnson & Chalfonte, 1994; McClelland, McNaughton, & O'Reilly, 1995; Moscovitch, 1994; O'Reilly & McClelland, 1994; Squire, 1994). According to such theories, the medial temporal lobe is critically involved with binding or integrating information that may be stored in separate cortical modules. Extensive integration is required for the construction of explicit memory episodes. Priming may be attributable to the activity of a subset of the cortical mechanisms that collectively contribute to explicit memory. Under conditions in which these subsystems singly or additively influence performance, amnesics show normal implicit memory. When information from these subsystems is interactively combined by normal subjects, amnesics are impaired. The extent of this interaction may vary continuously—from the limited interaction of perceptual mechanisms that produces perceptual specificity effects to the massive interaction of multiple brain mechanisms from which explicit memory emerges.

Our view may appear to be contradicted by the well documented finding of normal priming in amnesic patients when the perceptual format of study and test items remains constant in all conditions. If amnesics do not show normal

perceptual specificity effects, it may seem reasonable to expect that control subjects would always show an advantage when study–test format is constant. However, it is possible that most priming experiments are more analogous to the new-voice than the repaired-voice design discussed earlier. Perceptual specificity effects may normally depend on some variation or recombination of perceptual formats. With no such manipulations, the bulk of the evidence suggests that amnesic patients show normal priming.

IS PRIMING GENERALLY IMPAIRED IN AMNESIA?

In contrast to our suggestion that only a specific kind of priming is impaired in amnesic patients, Ostergaard and Jernigan (1993; Ostergaard, 1994) have recently argued that priming is generally impaired in amnesia. They note a number of published reports in which priming in amnesic patients is below that of control subjects. In other cases, it is argued that the experiments often lack the statistical power to detect differences between amnesics and controls if such differences were truly to exist. Ostergaard and Jernigan conclude that implicit and explicit memory are mediated by the same brain mechanisms, especially the medial temporal lobe. By their view, the distinction between implicit and explicit memory need not reflect the contribution of distinct brain systems.

Ostergaard and Jernigan suggest that previously published reports of normal priming in amnesic patients may be an artifact of priming scores being inflated by baseline information-processing impairments. This suggestion has been addressed by testing perceptual identification performance across a wide range of conditions in a mixed group of patients with amnesia (Hamann, Squire, & Schacter, 1995). Non-Korsakoff patients showed baseline performance that was consistently similar to control subjects. Korsakoff patients showed normal baseline performance except when words were extremely small. A final experiment compared the groups on priming at four different baseline levels by manipulating the exposure duration of the words. At each level of exposure duration, Korsakoff and non-Korsakoff amnesics showed both baselines and priming that were not significantly different from controls. Hamann et al. also reviewed a number of other published reports of normal baseline performance and normal priming by amnesic subjects.

Schacter and Church (1995, see Table 1B & C) have reported analogous data in two experiments that used the new-voice design discussed earlier. In Experiment 1, target words were spoken by either a male or female speaker during the study task, and test words were spoken by the same speaker or a new speaker. In this experiment, Korsakoff amnesics showed substantially impaired baseline performance compared to control subjects, together with a trend for greater overall priming. Non-Korsakoff amnesics showed slightly lower baselines than did control subjects and slightly lower levels of priming. Internal analyses suggested that the low levels of baseline performance were attributable

to the fact that some patients had difficulty discriminating words spoken by the female voice. In Experiment 2, which used only male voices (one at study and two at test), both Korsakoff and non-Korsakoff amnesic patients showed entirely normal baseline levels of performance together with normal priming. In view of these results and those of Hamann et al., we agree with Ostergaard and Jernigan that baseline performance must be considered when interpreting priming results in amnesics, and that baseline differences can pose interpretive problems (however, we disagree with their claim that lower baselines inevitably inflate priming scores, for reasons discussed at length by Chapman, Chapman, Curran, & Miller, 1994; see also, Schacter & Church, 1995). Nevertheless, such baseline differences are not always present and normal priming has been observed in the absence of baseline differences. Therefore, it seems clear to us that the medial temporal lobe/diencephalic system is not ubiquitously or inevitably involved in priming, as Ostergaard and Jernigan have contended.

CONCLUDING COMMENTS

We have argued that priming may be impaired in amnesic patients under circumstances where normal subjects bind information across multiple brain mechanisms. Mayes and Downes suggest that the comparison of implicit and explicit tasks allows for the separation of storage deficits and retrieval deficits. We agree with some, but not all, aspects of their logic. We think that they are correct that the same cortical storage mechanisms are likely to be involved in both implicit and explicit memory. For example, voice-specificity effects in priming probably depend on much of the same stored voice information that makes a contribution to explicit voice recognition. We also agree that the application of this logic depends on equating the ''informational features'' that are tapped by the implicit and explicit tasks (see Schacter, 1990b). However, if amnesics lack the ability to bind information across multiple brain systems, Mayes and Downes' approach of comparing implicit and explicit task performance may be misleading when the informational features tested require such binding. For instance, using a repaired-voice design, one might have concluded that amnesics do not store any voice information, but the evidence for a specificity effect in the new-voice design suggests a binding deficit rather than a storage deficit. More generally, we do not take the view that whenever amnesic patients exhibit an impairment on an implicit test, a storage deficit can be automatically assumed. If performance on the particular implicit task is enhanced by access to associations between qualitatively different kinds of information (e.g. word and voice), then amnesics may have stored individual information attributes but failed to bind them together into an integrated representation.

Mayes and Downes imply that the distinction between implicit and explicit memory may reduce to a distinction between different retrieval operations that

act on common memory representations. We certainly agree that retrieval differences are key to the implicit/explicit distinction. Indeed, the original formulations of the distinction (e.g. Graf & Schacter, 1985; Schacter, 1987) emphasised that it is concerned with different ways in which the effects of past experience can be expressed. Subsequent discussions noted the need to distinguish between two important dimensions of the implicit/explicit distinction: retrieval intentionality (unintentional retrieval vs. intentional retrieval) and subjective recollective experience (aware vs. unaware; see Schacter, Bowers, & Booker, 1989; for related discussion, see Richardson-Klavehn & Bjork, 1988). Implicit memory reflects primarily the bottom-up, nonconscious effects of prior experience on single brain subsystems, and may also involve interactions between a limited number of brain subsystems. Explicit memory reflects the top-down, simultaneous retrieval of information from multiple information-processing brain mechanisms. This massive integration of information (e.g. perceptual, semantic, temporal, spatial, etc.) may be necessary to support conscious recollection of previous experiences. Thus, when priming or similar phenomena are driven largely by individual brain subsystems, retrieval is involuntary and there is no conscious experience of remembering. Limited interactions that we have hypothesised are involved in certain kinds of priming effects (e.g. perceptual and word form features) may be sufficient for influencing behaviour involuntarily, but may not be sufficient for coherent conscious recollection. Alternatively, these kinds of priming effects may constitute an example of what has been called ''involuntary conscious memory'' (cf Richardson-Klavehn, Gardiner, & Java, 1994; Richardson-Klavehn, Lee, Joubran, & Bjork, 1994; Schacter, 1987, 1994). We think that it is reasonable to hypothesise that as more and more brain systems contribute to a particular retrieval effect, the expressed knowledge becomes more ''explicit'' and less ''implicit''.

To summarise, we agree with many of Mayes and Downes' arguments concerning implicit memory, but we have tried to point out and elucidate a number of phenomena and issues that are especially relevant to amnesia. Implicit memory phenomena such as priming are normally spared in amnesia, but deficits may become apparent under conditions in which normal subjects are able to integrate different types of information that are associated with a target stimulus. These ideas are somewhat speculative, still evolving, and based primarily on the results of a few experiments that need replication and extension. Nevertheless, we think that there are still many lessons to be learned by studying implicit memory in amnesic patients, and hope that we have identified a few that merit serious attention in future research.

REFERENCES

Bowers, J., & Schacter, D.L. (1993). Priming of novel information in amnesic patients: Issues and data. In P. Graf & M. Masson (Eds.), *Implicit Memory* (pp.303–326). Hillsdale, NJ: Lawrence Erlbaum Associates Inc.

Carlesimo, G.A. (1994). Perceptual and conceptual priming in amnesic and alcoholic patients. *Neuropsychologia*, *32*, 903–921.

Chapman, L.J., Chapman, J.P., Curran, T., & Miller, M.B. (1994). Do children and the elderly show heightened semantic priming? How to answer the question. *Developmental Review*, *14*, 159–185.

Church, B.A., & Schacter, D.L. (1994). Perceptual specificity of auditory priming: Implicit memory for voice intonation and fundamental frequency. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *20*, 521–533.

Cohen, N.J., & Eichenbaum, H. (1993). *Memory, amnesia, and the hippocampal system.* Cambridge, MA: MIT Press.

Cohen, N.J., Poldrack, R.A., & Eichenbaum, H. (this issue). Memory for items and memory for relations in the procedural/declarative memory framework. *Memory*, *5*(1/2).

Gabrieli, J.D.E., Milberg, W., Keane, M.M., & Corkin, S. (1990). Intact priming of patterns despite impaired memory. *Neuropsychologia*, *28* (414–427).

Graf, P., & Schacter, D.L. (1985). Implicit and explicit memory for new associations in normal and amnesic subjects. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *11*, 501–518.

Graf, P., Shimamura, A.P., & Squire, L.R. (1985). Priming across the modalities and priming across category levels: Extending the domain of preserved function in amnesia. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *11*, 386–396.

Haist, F., Musen, G., & Squire, L.R. (1992). Intact priming of words and nonwords in amnesia. *Psychobiology*, *19*, 275–285.

Hamann, S.B., Squire, L.R., & Schacter, D.L. (1995). Perceptual thresholds and priming in amnesia. *Neuropsychology*, *9*, 3–15.

Humphreys, M.S., Bain, J.D., & Pike, R. (1989). Difference ways to cue a coherent memory system: A theory for episodic, semantic, and procedural tasks. *Psychological Review*, *96*, 208–233.

Johnson, M.K., & Chalfonte, B.L. (1994). Binding complex memories: The role of reactivation and the hippocampus. In D.L. Schacter & E. Tulving (Eds.), *Memory Systems 1994* (pp.311–350). Cambridge, MA: MIT Press.

Kinoshita, S., & Wayland, S.V. (1993). Effects of surface features on word-fragment completion in amnesic subjects. *American Journal of Psychology*, *106*, 67–80.

Marsolek, C.J., Kosslyn, S.M., & Squire, L.R. (1992). Form-specific visual priming in the right cerebral hemisphere. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *18*, 492–508.

Mayes, A.R., & Downes, J.J. (this issue). What do theories of the functional deficit(s) underlying amnesia have to explain? *Memory*, *5*(1/2).

McClelland, J.L., McNaughton, B.L., & O'Reilly, R.C. (1995). Why there are complimentary learning systems in the hippocampus and neocortex: Insights from the successes and failures of connectionist models of learning and memory. *Psychological Review*, *102*, 419–457.

Moscovitch, M. (1994). Memory and working with memory: Evaluation of a component process model and comparisons with other models. In D.L. Schacter & E. Tulving (Eds.), *Memory Systems 1994* (pp.269–310). Cambridge, MA: MIT Press.

Musen, G., & Squire, L.R. (1991). Normal acquisition of novel verbal information in amnesia. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *17*, 1095–1104.

Musen, G., & Squire, L.R. (1992). Nonverbal priming in amnesia. *Memory & Cognition*, *20*, 441–448.

O'Reilly, R.C., & McClelland, J.L. (1994). Hippocampal conjunctive encoding, storage, and recall: Avoiding a tradeoff. *Hippocampus*, *4*, 661–682.

Ostergaard, A.L. (1994). Dissociations between word priming effects in normal subjects and patients with memory disorders: Multiple memory systems or retrieval. *Quarterly Journal of Experimental Psychology*, *47*A, 331–364.

Ostergaard, A.L., & Jernigan, T.L. (1993). Are word priming and explicit memory mediated by different brain structures? In P. Graf & M.E.J. Masson (Eds.), *Implicit memory* (pp.327–349). Hillsdale, NJ: Lawrence Erlbaum Associates Inc.

Ratcliff, R., & McKoon, G. (1988). A retrieval theory of priming in memory. *Psychological Review*, *95*, 385–408.

Richardson-Klavehn, A., & Bjork, R.A. (1988). Measures of memory. *Annual Review of Psychology*, *39*, 475–543.

Richardson-Klavehn, A., Gardiner, J.M., & Java, R.I. (1994). Involuntary conscious memory and the method of opposition. *Memory*, *2*, 1–29.

Richardson-Klavehn, A., Lee, M.G. Joubran, R., & Bjork, R. (1994). Intention and awareness in perceptual identification priming. *Memory & Cognition*, *22*, 293–312.

Roediger, H.L., & McDermott, K.B. (1993). Implicit memory in normal human subjects. In H. Spinnler & F. Boller (Eds.), *Handbook of neuropsychology* (pp.63–131). Amsterdam: Elsevier.

Schacter, D.L. (1987). Implicit memory: History and current status. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *13*, 501–518.

Schacter, D.L. (1990a). Perceptual representation systems and implicit memory: Toward a resolution of the multiple memory system debate. In A. Diamond (Ed.), *The development and neural bases of higher cognitive functions* (pp.543–571). New York: Annals of the New York Academy of Sciences.

Schacter, D.L. (1990b). Toward a cognitive neuropsychology of awareness: Implicit knowledge and anosagnosia. *Journal of Clinical and Experimental Neuropsychology*, *12*, 155–178.

Schacter, D.L. (1994). Priming and multiple memory systems: Perceptual mechanisms of implicit memory. In D.L. Schacter & E. Tulving (Eds.), *Memory systems 1994* (pp.233–268). Cambridge, MA: MIT Press.

Schacter, D.L., Bowers, J., & Booker, J. (1989). Intention, awareness, and implicit memory: The retrieval intentionality criterion. In S. Lewandowsky, J.C. Dunn, & K. Kirsner (Eds.), *Implicit memory: Theoretical issues* (pp.47–65). Hillsdale, NJ: Lawrence Erlbaum Associates Inc.

Schacter, D.L., & Church, B.A. (1995). Implicit memory in amnesic patients: When is auditory priming spared? *Journal of the International Neuropsychological Society*, *1*, 434–442.

Schacter, D.L., Church, B., & Bolton, E. (1995). Implicit memory in amnesic patients: Impairment of voice-specific priming. *Psychological Science*, *6*, 20–25.

Schacter, D.L., Cooper, L.A., Tharan, M., & Rubens, A. (1991). Preserved priming of novel objects in patients with memory disorders. *Journal of Cognitive Neuroscience*, *3*, 117–130.

Schacter, D.L., Cooper, L.A., & Treadwell, J. (1993). Preserved priming of novel objects across size transformation in amnesic patients. *Psychological Science*, *4*, 331–335.

Schacter, D.L., & Graf, P. (1986). Preserved learning in amnesic patients: Perspectives from research on direct priming. *Journal of Clinical and Experimental Neuropsychology*, *8*, 727–743.

Schacter, D.L., & Tulving, E. (Eds.). (1994). *Memory systems 1994*. Cambridge, MA: MIT Press.

Shimamura, A.P., & Squire, L.R. (1989). Impaired priming of new associations in amnesia. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *15*, 721–728.

Squire, L.R. (1994). Declarative and nondeclarative memory: Multiple brain systems supporting learning and memory. In D.L. Schacter & E. Tulving (Eds.), *Memory systems 1994* (pp.203–231). Cambridge, MA: MIT Press.

Tulving, E., & Schacter, D.L. (1990). Priming and human memory systems. *Science*, *247*, 301–305.