5 Memory in PTSD: A Neurocognitive Approach

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Abstract

Memory changes associated with post-traumatic stress disorder (PTSD) are pervasive. How the traumatic event is encoded and retrieved is central to the experience of the disorder; however, more general abnormalities in autobiographical recollection and in new learning are also commonly observed. These memory abnormalities are likely due at least in part to the impact of the traumatic event and the neurobiological alterations associated with PTSD, but memory factors that exist prior to trauma also play a role in the development and maintenance of PTSD. Thus, we consider memory as both a symptom and a risk factor for PTSD. In the domain of autobiographical memory, we examine recollection of personally experienced general and trauma-related events. In the domain of new learning, we characterize everyday memory for emotionally neutral information and consider distinct changes in PTSD in the encoding and retrieval of information that has strong emotional valence and trauma relevance. We highlight specifically the contribution of neuropsychological and cognitive neuroscience studies, with the goal of elucidating the neurocognitive mechanisms underlying memory changes associated with PTSD.

Key Words: Amygdala, autobiographical memory, frontal lobes, hippocampus, new learning, trauma memory.

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INTRODUCTION

Over the last few years, the centrality of memory to a comprehensive understanding of post-traumatic stress disorder (PTSD) has become abundantly clear. First, memory alterations are a core feature of PTSD. PTSD reexperiencing symptoms include unwanted, distressing, and seemingly poorly controlled recollections of a personally experienced traumatic event. Further, an inability to recall important aspects of the trauma event is considered a diagnostic symptom of PTSD *(1)*, albeit one that appears to occur in only a smaller subset of trauma victims *(2)*. More generally, impairments in declarative memory are commonly observed in trauma survivors with PTSD, and these encompass disturbances in the retrieval of autobiographical memories as well as inefficiencies in the encoding and retrieval of new information. Consequently, many theories of PTSD involve memory as a central component, providing explanations for different facets of the memory impairment in terms of psychosocial, cognitive, or neurobiological factors.

Second, and no less important, memory factors play a critical role in the development and maintenance of PTSD. Individual differences in memory and processing style prior to trauma exposure influence susceptibility to PTSD, and how victims process, encode, and subsequently relive traumatic events has a direct impact on the development and maintenance of PTSD symptoms. In the most radical expression of this view, Rubin, Berntsen, and Johansen *(2)* suggested that a pathogenic memory rather than a pathogenic event is the critical causal event in PTSD.

In this chapter, we consider memory both as a symptom and as a risk factor for PTSD, focusing separately on autobiographical memory (i.e., the retrieval of personally experienced past events) and on new learning (i.e., the encoding and retrieval of current information). In the domain of autobiographical memory, we review recent work on personal memories generally as well as on trauma memory specifically. In the domain of new learning, we consider everyday memory for emotionally neutral information and then examine whether there are distinct alterations in PTSD in the encoding and retrieval of information that has strong emotional valence. We focus specifically on the cognitive and neural mechanisms underlying memory changes associated with PTSD, and in so doing, highlight the contribution of neuropsychological and cognitive neuroscience approaches to the study of memory in PTSD.

AUTOBIOGRAPHICAL MEMORY

As is true for all memory, the recollection of personal past events reflects a construction of the past in light of current goals and attitudes rather than the retrieval of an actual record of past events. This is particularly important to keep in mind when examining autobiographical memory in PTSD because, as noted, the formation and recall of autobiographical memories, especially those involving the trauma event, are thought to be central to the experience of PTSD. Paradoxically, alterations in memory for the trauma event take the form both of intrusive recollections

and of inability to recall aspects of the trauma. Adding further to the complexity, autobiographical abnormalities associated with PTSD are not limited to recall of trauma-specific memories but extend to events unrelated to the trauma, suggesting a larger, overarching autobiographical memory impairment.

In the following sections, we focus on general abnormalities in autobiographical memory in individuals with PTSD and on the cognitive processes underlying them. We next examine PTSD-associated abnormalities in memory for the trauma specifically. A comprehensive discussion of the formation and recall of trauma memories is beyond the scope of the chapter (for reviews, *see* Refs. *2–6)*. Instead, we summarize here the central themes that are directly relevant to understanding memory in PTSD from a neurocognitive perspective.

Overgeneral Nature of Autobiographical Memory

One of the most common methods of assessing autobiographical memory is by means of a cued recall task known as the Autobiographical Memory Test (AMT) *(7)*. The AMT requires the respondent to generate specific autobiographical episodes in response to cue words that vary in their emotional valence (e.g., positive, negative, neutral). These narratives are then rated for their specificity. *Specific recall* refers to a description of unique events that occurred at a specific time and place. In contrast, *overgeneral memory recall* is vague and refers to entire classes of events rather than to discrete events.

Among studies using the AMT paradigm in trauma-exposed samples, a fairly consistent pattern has emerged. Trauma survivors with acute stress disorder *(8,9)* or a PTSD diagnosis *(10–12)* tend to produce fewer specific and more overgeneral autobiographical memories relative to trauma survivors without psychiatric diagnoses. Although most studies have used word cues, a similar pattern was reported in a study that used concrete, imageable pictorial cues *(13)*, suggesting that overgeneral autobiographical memory is not simply a consequence of the abstract processing elicited by verbal cues but also occurs in response to stimuli that more closely approximate sensory cues encountered in the environment.

Although overgeneral recall has been observed when memories are generated in response to both negative and positive cues, individuals with PTSD appear to have particular difficulty recalling positive memories with specificity *(10,14)*. In one study, priming with a trauma-related video led to more general recall of events *(11)*. In another study, the difficulty in retrieving specific memories among war veterans with PTSD was particularly pronounced in those individuals wearing military regalia *(10)*; these individuals also retrieved a disproportionate number of war-related memories, a finding that was interpreted as reflecting the centrality of their war experience to their sense of self. One explanation of these findings is that impoverished recall of positive memories reflects a problem among PTSD-diagnosed trauma survivors in accessing the emotionally positive aspects of their histories and identities *(10,11,14)*. In keeping with this formulation, when asked to produce self-defining memories, PTSD-diagnosed trauma survivors, as compared to non-PTSD-diagnosed trauma survivors and

nonexposed controls, reported more self-defining memories that were trauma related and of negative valence *(15)*. Similarly suggesting a link between self-identity and autobiographical recall, trauma survivors with PTSD were more likely to provide trauma-focused memories in response to positive cue words when their perception of actual self fell short of their perception of ideal self than when actual and ideal perceptions were more closely aligned *(16)*.

Mechanisms of Overgeneral Memory in PTSD

Attempts to understand the mechanisms responsible for overgeneral memory draw heavily on a cognitive model of autobiographical memory proposed by Conway and Pleydell-Pearce *(17,18)*. According to this model, autobiographical memory is part of a larger self-memory system that maintains an integrated representation of one's sense of self as well as a record of ongoing experiences as they contribute to one's goals and sense of self. Autobiographical memory depends on the retrieval of information from an autobiographical knowledge base by an executive system, called the *working self*. The autobiographical memory base is organized hierarchically, with representations of life time periods at the highest level, general event descriptions at an intermediate level, and unique, event-specific representations that contain sensory-perceptual information at the lowest level. According to this model, retrieval of an autobiographical memory in response to a verbal cue requires top-down search processes to access specific event representations as well as executive control processes to evaluate whether the retrieved memory fits the search criteria. Overgeneral memory occurs because the search during top-down retrieval is aborted too early, at the level of general event descriptions.

Building on this model, Williams et al. *(19)* proposed three mechanisms that may contribute to truncation of the retrieval search in emotional disorders: (1) capture and rumination, (2) functional avoidance, and (3) executive deficits. The capture/rumination component involves difficulty disengaging from the level of categorical retrieval when the retrieval cue activates abstract, conceptual self-representations. Such capture may occur in individuals in whom negative self-representations are already highly activated and elaborated. The retrieval cue primes other negative categories, thereby facilitating rumination and perpetuating a negative-feedback loop by which activation is maintained at an abstract conceptual level rather than propagating to a more specific level. The role of self-focused abstract thinking in decreased memory specificity has been studied in detail in depressed patients *(20,21)*. Although it has not been tested directly in PTSD, it has been speculated that PTSD-related ruminative tendencies may operate in a similar manner *(22)*.

The second mechanism of truncated memory search is based on the "affect regulation" hypothesis *(23)*, which proposes that trauma-exposed individuals fail to retrieve specific trauma memories as a means of avoiding the distress associated with remembering details of the trauma. The impact of avoidance, however, is not limited to the trauma memories but instead extends to the whole

domain of autobiographical memory. Generalization to non-trauma-related autobiographical memories purportedly occurs because the avoidance truncates effortful, hierarchical search of the entire autobiographical knowledge base at the level of general event descriptors.

Several studies provided support for this hypothesis. Schonfeld, Ehlers, Bollinghaus, and Reif *(12)* examined the conditions under which overgeneral memory was most likely to occur in assault-related PTSD by manipulating the instructions to either suppress or not suppress assault memories during the AMT task. PTSD-diagnosed survivors retrieved fewer, and more general, memories when asked to suppress assault memories than when instructed not to suppress assault memories. The authors interpreted these results in relation to cognitive avoidance associated with PTSD; specifically, attempts to suppress negative trauma-related intrusions that are highly specific may have led to difficulty with recall of specific non-trauma-related life events. Also consistent with the affect regulation model, Wessel, Merckelbach, and Dekkers *(24)* found that overgeneral memory production among patients previously exposed to war atrocities was associated with more frequent intrusions and greater avoidance of trauma reminders.

Interestingly, although linked mechanistically to avoidance, reduced memory specificity may not effectively regulate affect. Golden, Dalgleish, and Mackintosh *(25)* compared the memories of bereaved individuals with complicated grief to those without complicated grief; they used an autobiographical memory test and two biographical tests, one cueing memories from the deceased's life and one cueing memories from a living significant other's life. They found that participants with complicated grief, as compared to those without complicated grief, showed reduced specificity in response to negative cue words when retrieving both autobiographical memories and memories about living others. In contrast, when retrieving memories about deceased others in response to negative cues, those with complicated grief retrieved more specific memories than those without complicated grief. Although this study did not examine trauma and PTSD specifically, the finding that distress-related memories seem to be resistant to affect regulation may also apply to trauma memories in individuals with PTSD.

The third mechanism of truncated memory search is a reduction in executive resources, thought to be associated with prefrontal dysfunction *(19)*. This represents the most well-developed neurocognitive hypothesis of overgeneral autobiographical memory in PTSD. In the autobiographical memory model of Conway and Pleydell-Pearce *(17,18)*, executive resources, including working memory and inhibitory functions, are critical at several stages in the process of memory retrieval. For instance, working memory is critical for holding a retrieval template in mind both during generation of retrieval cues and in a final search and comparison stage. Cognitive inhibition is important during the search process as a mechanism to sort through relevant and irrelevant autobiographical memories. The reduced executive capacity hypothesis is compelling

in relation to PTSD given (1) the documented association between PTSD and working memory and inhibition deficits *(26–28)*, (2) the observable difficulties individuals with PTSD have in inhibiting unwanted trauma-related memories (i.e., reexperiencing symptoms), and (3) neuroimaging (e.g., *29,30–34)* and electrophysiological (e.g., *35,36)* findings suggestive of attenuated prefrontal functioning in PTSD. However, the strongest empirical support that executive resources are mechanistically related to autobiographical memory comes from non-PTSD psychiatric samples *(19)*.

Only a few studies have directly examined the relationship between cognitive resources more broadly and overgeneral memory in trauma-exposed samples. For example, Schonfeld et al. *(12)* found that in assault survivors with PTSD, verbal intelligence was negatively associated with overgeneral memory production, whereas working memory measures (digit span forward and backward) were not significantly associated with AMT performance. De Decker, Hermans, Raes, and Eelen *(37)* observed nonsignificant, albeit moderate, associations between immediate and delayed recall of standardized narratives and autobiographical specificity in trauma-exposed adolescents, but the specificity of autobiographical memories was only very weakly related to performance on a working memory task. Wessel et al. *(24)* did not find any significant relationships between anterograde memory performance on tasks of immediate recall, total memory capacity, and strategic retrieval from semantic memory. Thus, although a neurocognitive basis of overgeneral retrieval in PTSD is compelling theoretically, the few available studies have not revealed strong evidence for such an association.

Overgeneral recall characterizes autobiographical memory not only in PTSD but also in clinically significant depressive disorders (e.g., major depressive disorder) and in subclinical presentations of dysphoric mood *(19,38)*. Because of the dysphoric characteristics of some PTSD symptoms (e.g., diminished interest in normal activities) and the high comorbidity of depression with PTSD *(5,39,40)*, it could be hypothesized that the overgeneral memory observed in PTSD is due primarily to associated depression symptoms. However, when depressive symptoms are controlled statistically, overgeneral memory remains associated with symptoms of PTSD *(13)* and acute stress disorder *(9)*, suggesting that depression is not the sole cause of overgeneral memory in trauma survivors with PTSD. In contrast, overgeneral autobiographical recall is rarely observed in non-PTSD anxiety disorders unless the anxiety is accompanied by comorbid depression *(41,42)*. In fact, although trauma history has been linked to overgeneral memory *(43–45)*, the vast majority of studies examining the relationship of trauma exposure to overgeneral memory recall suggest that either PTSD or depressed mood must be present for overgeneral memory to occur in trauma survivors *(19)*.

Alterations in Autobiographical Memory: Predisposition or Consequence?

To help determine the causal direction of the relationship between overgeneral autobiographical recall and PTSD, several studies have used longitudinal methodology. In general, findings indicate that preexposure tendencies for overgeneral mem-

ory are associated with increased risk of PTSD. For example, Bryant, Sutherland, and Guthrie *(14)* found that impaired retrieval of specific memories in response to positive cues prior to trauma exposure among trainee firefighters predicted posttraumatic stress symptom severity after trauma exposure, a finding consistent with the cross-sectional literature. Harvey and Bryant *(46)* likewise found that overgeneral retrieval of autobiographical memories shortly after motor vehicle accident exposure predicted subsequent PTSD. In contrast, Kangas, Henry, and Bryant *(9)* found that early deficits in recalling specific memories among cancer patients with acute stress disorder did not predict subsequent PTSD 6 months later, raising the possibility that trauma characteristics may influence the relationship between overgeneral memory and PTSD. In the only treatment study that assessed autobiographical memory in participants exposed to assault or motor vehicle accident before and after cognitive-behavioral therapy, it was found that improvement in PTSD symptoms was related to improved retrieval of specific memories and decreased overgeneral recall in response to positive cues *(47)*.

Although the mechanism by which overgeneral memory contributes to the development or maintenance of PTSD is unknown, it is of note that several studies have demonstrated a link between overgeneral memory on the one hand and decreased ability to imagine future events *(48)* and solve social problems *(49,50)* on the other. Such a link is intriguing as recent cognitive neuroscience findings suggest that the ability to retrieve past events has a direct impact on the ability to coherently simulate future events because future thought requires the flexible recombination of details from the past *(51)*.

Trauma Memory

One of the most debated conceptual issues surrounding the study of memory in PTSD concerns the uniqueness of trauma memories in comparison to other autobiographical memories. Central in this discussion is the question of whether trauma memories differ solely in quantity (e.g., frequency and intensity of occurrence) or also in quality (i.e., underlying memory processes or representations).

One consideration concerns the accuracy of trauma memories in comparison to other memories. The literature on flashbulb memories *(52)* has been invoked as potentially relevant to this question as it is often assumed that events that are extremely surprising, infrequent, and relevant to the individual are remembered in a more durable and fixed form. However, the available evidence suggests that trauma memories, like other autobiographical memories with high emotional intensity *(53)*, are prone to errors and distortions. A recent review of studies that assessed on two separate occasions memories of assault or of wartime exposure indicated that inconsistencies in report of the index event over time are common *(54)*. Of note, in several studies, there was a slight tendency for changes in report, including amplification of the memory, to be associated with severity of PTSD symptoms *(55–58)*.

With regard to qualitative characteristics of trauma memories, we noted that there is some evidence to suggest that the overgeneral recall of autobiographical events may not extend to trauma-related memories (*see also 9,25*). One

interpretation of these findings is that trauma-related memories, because of their vividness and potency, do not require a hierarchical search of the autobiographical memory base but rather are accessed directly through activation of event-specific information *(17)*.

A number of studies have used self-reported memory ratings to evaluate the characteristics of trauma memories. These studies indicate that, in comparison to trauma-exposed control subjects, trauma survivors with PTSD have more vivid recollection, experience their trauma memories as richer in feelings and sensory details, and find the memories to be of greater personal significance *(59–61)*. One study that directly compared traumatic and positive memories found that these differences were specific to traumatic memories *(59)*. However, another study limited to individuals with PTSD found no differences in trauma and nontrauma memories with regard to their sensory qualities or stability *(62)*. Findings from these studies are somewhat difficult to interpret, however, because ratings may be subject to mood-related biases due to their subjective and retrospective nature and because studies differ in terms of the specific memory attributes rated.

Arguments for the uniqueness of trauma memories center on the issue of "memory fragmentation." *Memory fragmentation* refers to the lack of coherence within a memory and reflects a failure to integrate different aspects of a memory into an internally consistent whole. Such fragmentation is thought to result from disorganized initial encoding of the traumatic event, which leads to inconsistent consolidation and poorly regulated retrieval. Van der Kolk *(63,64)*, for example, proposed that the lack of narrative coherence of trauma memories is a reflection of emotionally induced dissociative states at the time of trauma, which result in routing of the memories through distinct neurochemical pathways. According to this model, trauma memories are preserved in an implicit memory system as vivid sensory and perceptual experiences but are not accessible as explicit verbal narratives. Ehlers and Clark *(65)* likewise purported that some trauma memories remain inaccessible because they are poorly integrated during encoding but believed that the initial encoding is dependent on the amount of "conceptual" (i.e., relating to the meaning of the event and its integration into a larger autobiographical context) versus "data-driven" (i.e., sensory) processing.

Brewin *(66,67)* proposed a dual-representation model according to which trauma memories are based on two separate representations: (1) a hippocampally mediated narrative representation that supports verbally accessible memories that are integrated with the rest of the autobiographical memory base and can be retrieved either automatically or strategically, and (2) an image-based representation mediated by the amygdala that does not interact with the autobiographical memory base and can only be retrieved automatically by trauma cues (i.e., is situationally accessible). According to this model, the two systems may be differentially impacted by neurohormonal responses to stress, leading to enhanced encoding of situationally accessible trauma memories and reduced encoding of verbally accessible trauma memories. Further, it is suggested that as part of the

process of healthy adaptation to trauma, such imbalance is corrected through a process of reencoding image-based memories into verbally accessible memories; in PTSD, by contrast, such reencoding does not take place, with the result that verbal memories cannot inhibit maladaptive amygdala responses *(68)*.

Most memory fragmentation theories of trauma memory assert that some combination of heightened arousal, emotional distress, and dissociation at the time of the event lead to disorganized encoding of trauma memories. Consistent with the view that extreme distress during trauma affects the manner in which an event is encoded, studies of emotional memory encoding in nonclinical samples suggest that intensely negative and arousing memories lead to enhanced memory for the information central to the event but impoverished memory of peripheral details (for a review, *see 69)*, a phenomenon referred to as *tunnel memory (70)*. However, tunnel memory is a common source of memory distortion for emotionally significant events and can equally be explained with reference to general principles of autobiographical memory *(71)*. Further, as noted by Rubin et al. *(2)*, the phenomenon of tunnel memory is inconsistent with one of the symptoms of PTSD *(1)* (criterion C3: difficulty remembering an important aspect of the event), although as noted, this symptom may occur relatively infrequently. More important, perhaps, the phenomenon of tunnel memory fails to explain the enhanced clarity of peripheral trauma details thought to be associated with intense reliving phenomena *(72)*.

The empirical basis for memory fragmentation models has largely been based on narrative recall paradigms and metamemory studies. In narrative recall paradigms, participants are asked to describe the trauma event, and the narratives are then coded for their cohesiveness and semantic structure. In metamemory studies, participants are asked to make a subjective appraisal of the coherence of their own memory. Some studies have found that traumatic memories are more fragmented in individuals with acute stress disorder (e.g., *46)* or PTSD *(73)* than in trauma-exposed control groups, but other studies have failed to find group differences *(59,60)*. In a recent comprehensive review of this literature, Zoellner and Bittenger *(6)* concluded that the lack of clarity can be attributed in large part to methodological limitations of the current literature, including the frequent failure to control for PTSD diagnosis and trauma characteristics and the failure to consider non-PTSD psychopathology.

Of special note in the literature on trauma memory is the subgroup of studies that have attempted to link other symptoms to autobiographical memories of the trauma. These studies are particularly interesting because they address a seeming paradox in PTSD: the prominence of unwanted intrusions of the trauma memory as reexperiencing symptoms in the context of difficulties with deliberate retrieval of certain aspects of the trauma event. This has led some theorists to maintain that those memories manifested in the form of reexperiencing symptoms, in particular, may differ qualitatively from other trauma and nontrauma autobiographical memories *(17,66,74)*. Brewin *(75)*, for example, has suggested that flashbacks are clearer than other trauma memories, albeit more

fragmented and less easy to retrieve in a well-regulated manner. Supporting this notion, Hellawell and Brewin *(72,76)* found that "flashback" reexperiencing episodes, as compared to "normal" trauma memories, were associated with more autonomic and motor behaviors and greater sensory detail; they were experienced more often in the present tense and were characterized more often by primary emotions such as horror, fear, and helplessness. In examining nonflashback intrusions, Rubin, Feldman, and Beckham *(61)*, however, found no evidence that trauma memories differed from nontrauma memories in coherence, although reliving phenomena increased as memories were more related to the trauma. Indeed, recurrent involuntary trauma memories appear to share many of the same characteristics (e.g., proportion of positive to negative memories, associated emotional intensity, dominance for recent events) as ordinary autobiographical memories *(77)*. However, even different symptoms within the reexperiencing class (e.g., recurrent thoughts vs. nightmares) are poorly correlated *(77)* and therefore may differ in their associated features.

NEW LEARNING

Although alterations in autobiographical memory in PTSD are well documented, clinical complaints of memory problems in individuals with PTSD typically concern the ability to remember day-to-day information and events experienced after the trauma. In this section, we characterize the nature of these difficulties; consider how they can best be understood in terms of underlying cognitive and neural mechanisms; and evaluate how they are causally related to the development of PTSD. We also examine the impact of emotional valence on new learning and consider factors that may be responsible for the enhanced learning of trauma-related as compared to trauma-neutral information in PTSD.

Characterizing the New Learning Impairments

The results of neuropsychological studies examining the presence and nature of difficulties in new learning remain unclear. On the one hand, there are now a considerable number of studies that have documented impairments in new learning and memory in PTSD *(78,79)*. Several of these observed that memory impairments tend to be selective to the verbal domain *(80)*, but others have documented impairments in visual memory as well *(28,81–83)*. Different conclusions also have been drawn with regard to the nature of the impairment, with studies variably highlighting problems with the initial registration of information *(28,84,85)*, with both immediate and delayed memory *(86–88)*, and with sensitivity to interference *(28,89,90)*.

On the other hand, a number of studies have failed to find evidence for memory impairment specific to PTSD *(91–94)*. These studies raise the question of whether impaired memory, when observed, may be due to comorbid conditions such as substance abuse or depression *(95)*. Examining specifically the contribution of comorbid alcohol use, Samuelson and colleagues *(84)* found that alcohol history was associated with visual memory impairments, but that PTSD was

associated with problems in verbal memory, even controlling for alcohol use. Similarly, although depression can lead to memory impairment in its own right, PTSD-associated memory impairments cannot fully be accounted for in terms of depression *(96,97)*. Other potentially important variables in explaining discrepant findings concern the nature of the control group to which PTSD patients are compared (individuals with trauma exposure without PTSD or individuals who did not experience trauma), the inclusion of individuals with head injury in the PTSD group, the chronicity of PTSD, and the nature of the trauma itself.

A meta-analysis by Brewin, Kleiner, Vasterling, and Field *(78)* helps to clarify the role of several of these variables. These authors concluded that there is a robust and selective association between PTSD and verbal memory problems that exists in both civilian and military samples and that cannot be explained by concurrent head injury. Although the magnitude of memory impairment is greater when the comparison group consists of individuals without trauma exposure, a significant impairment remains when a trauma-matched control group is used, which is on the order of one-third of a standard deviation. Perhaps less clear at this point is the role of time since trauma. In a majority of studies, participants have been combat veterans who are more likely to have a chronic, and possibly intractable, form of PTSD. To the extent that continued stress exposure may lead to a progressive course of the disorder, cognitive deficits might be more obvious over time; indeed, in at least one study, memory impairment was absent on most measures of memory in recent adult trauma survivors with post-traumatic stress symptoms *(98)*. However, memory impairments have been documented in children and adolescents with PTSD following a recent trauma *(99)*, as well as in veterans with PTSD tested within 5 years of return from the Gulf War *(28)*. These findings suggest that memory problems are not limited to individuals with chronic PTSD. Aside from chronicity, age per se may also need to be taken into account as there is some evidence for greater memory decline associated with PTSD in elderly than in younger individuals *(100)* and a steeper longitudinal decline in elderly individuals with PTSD than without PTSD *(101)*. Both of these findings suggest an interaction between PTSD-related memory loss and aging, the exact nature of which remains to be further elucidated.

Neural and Cognitive Bases of New Learning Impairments

Successful memory depends on both the medial temporal lobes (MTLs) and the frontal lobes, and thus a natural question arises regarding relative contributions of compromised MTL versus frontal lobe functioning to PTSD-associated memory impairments. The MTL system is the core memory system that binds together different aspects of an episode into a single memory trace and allows reactivation of that trace in the context of an appropriate cue. The frontal lobes, by contrast, are critical for strategic aspects of encoding and retrieval. They organize the input to the MTL and allow its output to be monitored and used deliberately, in the function of task goals and requirements.

Against the backdrop of diminished attention and executive function in PTSD *(26–28)*, several aspects of memory performance have been attributed to

impaired frontal function. Specifically, it has been suggested that a disruption of strategic encoding processes, leading to impoverished encoding, may underlie enhanced susceptibility to interference *(79)*. A similar sensitivity to interference is often seen in patients with structural lesions to the frontal lobes *(102,103)*. Further, since strategic encoding processes are more critical for encoding of unrelated compared to related word lists, the fact that memory impairment is seen more consistently on the Rey Auditory Verbal Learning test (which is composed of unrelated words) than on the California Verbal Learning Test (which is composed of categorized words) is consistent with this interpretation *(79)*. It should be noted that such frontal encoding deficits are not simply a consequence of poor attention as memory impairments persist even when attention is controlled for *(84,87)*.

Frontal functions are also critical for monitoring the appropriateness of a retrieved memory. Memory errors such as intrusions in free recall and false alarms in recognition, which are thought to reflect impoverished monitoring, provide further evidence for a frontal contribution to the memory impairment seen in PTSD. Examining the relationship between monitoring errors and aspects of psychopathology, we found that intrusions and false alarms were positively correlated with reexperiencing symptoms and negatively correlated with avoidance and emotional numbing symptoms *(28)*. One explanation of these findings is in terms of a failure to inhibit task-irrelevant processes, reflecting a faulty gating mechanism for the controlled processing of task-relevant information *(28)*. Another explanation is that hyperarousal and associated frontal system disruption may interfere with the controlled aspects of memory. The relationship between symptom severity and memory performance, however, is likely multidetermined as another study found that memory performance correlated not with current symptoms but rather with reported worst-episode symptoms *(104)*.

Interest in the role of MTL impairment in the day-to-day memory problems seen in PTSD stems from the established effects of severe stress on hippocampal structure and function in animals, which are thought to be mediated at least in part through elevated levels of glucocorticoids *(105,106)*. Analogously, it has been suggested that in individuals with PTSD, excessive release of cortisol during the acute stress or throughout the course of illness might lead to hippocampal abnormalities *(107)*, although it is now apparent that stress also has a direct impact on frontal lobe functioning *(108)*. The well-established link between stress and the hippocampus, coupled with reports starting in the mid-1990's of reduced hippocampal volume in individuals with PTSD *(80,109–111)* initially appeared to provide a ready explanation for the memory deficits seen in PTSD.

Subsequent studies, however, have questioned whether there is a direct link between MTL dysfunction and the observed memory deficits. First, although a majority of studies have documented hippocampal volume reductions in PTSD (for review, *see 112*,*113)*, there are a number of exceptions to this pattern, both in patients with recent-onset *(98,114,115)* and those with chronic PTSD *(116,117)*. Second, despite initial evidence of a direct relationship between hippocampal

volume and memory performance *(80)*, a number of more recent studies have failed to find such a correlation *(89,118,119)*. One caveat, however, is that most of these studies have used clinical neuropsychological tests that rely to a varying extent on both MTL and frontal functions rather than tests that are selectively sensitive to MTL function. For example, several studies have examined memory retention over a delay, but findings concerning the rate of forgetting in PTSD have been inconsistent. Further, although an abnormally fast rate of forgetting is often seen in patients with MTL lesions, it is also seen in association with severe memory loss that results from some frontal lesions *(120)* and thus does not in itself help to specify the neural basis of the impairment.

Of note, in a recent study that specifically targeted the role of the hippocampus in configural processing, the ability to perform a configural task was significantly correlated with hippocampal volume *(119)*. It has been suggested that the ability of the hippocampus to process and encode the configural relationships among multiple elements is critical for the contextual regulation of emotional responses *(121)*. It also forms the hallmark of episodic memory.

In light of the mounting evidence for both frontal and MTL abnormalities in PTSD (see Chapter 15, this volume), it seems increasingly unlikely that PTSD-related memory impairments are exclusively frontal or MTL based, but rather dysfunction in each of these regions may contribute in distinct ways to the observed memory impairment. To elucidate which aspects of memory or memory processes are linked to MTL or frontal dysfunction in PTSD, memory probes that are uniquely sensitive to the contribution of each these regions will be necessary. Perhaps more important, given the intricate link among these regions, functional imaging studies that examine not only distinct patterns of activation in each of these regions but also the functional connectivity among these regions will be critical.

New Learning Impairments: Predisposition or Consequence?

The findings reviewed leave unanswered the question of whether PTSD causes memory impairment, whether poorer memory is a risk factor for PTSD, or whether the observed association between memory and PTSD is bidirectional. Until recently, the notion that PTSD leads to memory impairment was largely based on evidence from animal studies documenting that stress leads to neurobiological changes in the hippocampus that have a direct impact on learning and memory *(122–124)*. In the last few years, several prospective studies in humans have documented similar adverse effects on memory of acute stress associated with military exercises *(125)* or special operations *(126)*. In the first prospective study of Army soldiers deployed to Iraq, significant pre- to post-deployment declines were observed in both verbal and visuospatial memory *(127)*. Of note, these studies all documented memory decline immediately following stress exposure, but whether such changes are transient or chronic, and how they relate to PTSD, is currently unknown. In the Iraq deployment study, memory decline was more pronounced in individuals who had symptoms of PTSD, but PTSD in itself did not fully account for the deployment-associated

memory loss *(127)*. Thus, the cognitive effects of stress and PTSD may be partially dissociable.

Initial recognition of the possibility that lower cognitive function might serve as a risk factor for PTSD came from studies demonstrating that lower intelligence is associated with more severe PTSD symptoms *(128,129)*, and importantly, that the development of PTSD was not associated with a deterioration in IQ *(129)*. Specifically with respect to memory, Bustamante, Mellman, Davide, and Fins *(130)* found that less-proficient verbal memory shortly after trauma exposure was related to subsequent development of PTSD. Studies comparing the performance of twin pairs, in which one twin is a combat veteran and the other twin is not, provide further compelling evidence for the notion that memory and hippocampal functioning may be predictive of the development of PTSD following trauma *(96,119,131)*. Comparing the performance of pairs in which the combat-exposed twin developed PTSD (PTSD pairs) to that of pairs in which the combat-exposed twin did not develop PTSD (non-PTSD pairs), it was found that both the combat-exposed twin and the nonexposed sibling from the PTSD pairs performed similarly on tests of verbal memory and performed more poorly than the combat-exposed twin and nonexposed sibling of non-PTSD pairs *(96)*. Both siblings of the PTSD pairs also had reduced hippocampal volumes in comparison to the siblings of the non-PTSD pairs, and the hippocampal volumes of the combat-exposed twin and nonexposed sibling of the PTSD pair were equally predictive of the severity of PTSD of the combat-exposed twin *(131)*. Finally, the configural processing deficit mentioned earlier, which appears to be a sensitive measure of hippocampal functioning, was present in both the combat-exposed twin and nonexposed sibling of the PTSD pairs, suggesting that these specifically hippocampal impairments may predispose individuals to PTSD.

The evidence reviewed suggests that the link between memory impairment and PTSD is likely bidirectional. In particular, we suggest that premorbid differences in cognitive ability, including memory, may act as a preexisting risk factor for PTSD, but that independent of risk for PTSD, stress itself also affects cognitive functioning. Preliminary evidence for this bidirectional relationship is emerging *(132)*, but further studies examining the combined effects of risk factors for PTSD and actual stress exposure are needed to uncover the exact bases of the memory impairments associated with PTSD.

New Learning of Emotional Information

Against the backdrop of a general impairment in new learning, a question of considerable interest is whether there are differences in the way individuals with and without PTSD process and learn emotional, and especially trauma-related, information in comparison to nonemotional information. To address this question, a number of studies have compared memory for neutral and trauma-related words in the context of list-learning tasks. Several studies have done so following conditions of incidental learning, when participants processed the information without

a subsequent memory task in mind. Individuals with PTSD recalled more trauma-related words than did individuals without PTSD, whereas the groups did not differ in their recall of neutral words *(133–135)*. When memory was assessed by means of recognition rather than recall, groups did not differ in their memory for trauma-related words, but one study found that PTSD patients had a more liberal response bias for trauma-related information regardless of whether that information had in fact been presented *(136)*. These findings suggest a processing bias that may favor both attending and responding to trauma-related information and that may have an impact on memory, especially under conditions that require self-initiated retrieval.

It is unlikely, however, that the trauma-related memory advantage seen in PTSD can be accounted for completely in terms of a processing bias. In one of the recall studies mentioned *(133)*, an independent probe indicated that trauma-related words did not receive preferential attention, and yet they were better recalled. Moreover, in intentional memory studies, in which attention is focused directly on each stimulus that is to be memorized, individuals with PTSD also show relatively better memory for trauma-related information. In two studies, recall of neutral words was impaired in individuals with PTSD, whereas recall of trauma-related words was not *(129,137)*. In a third study, recall was generally impaired, but the PTSD group showed an advantage in memory for trauma-related information that was not present in the group without PTSD *(138)*.

On a cognitive level, this "memory bias" may be due to the fact that trauma-related information is encoded more richly and, to the extent that it evokes memories of the traumatic event, is assimilated within an already existing emotional memory network, thus making it easier to retrieve later. Emotion enhances the subjective characteristics associated with memory, such as its vividness and recollective quality *(139,140)*, and interestingly, preliminary evidence suggests that in individuals with PTSD memory for trauma-related information may be associated with enhanced feelings of recollection *(141)*. On a neurobiological level, emotional arousal activates b-adrenergic receptors in the amygdala, and amygdala activation in turn modulates hippocampally mediated consolidation *(142)*. Functional imaging studies have demonstrated that individuals with PTSD show greater enhancement in amygdala activation than individuals without PTSD in association with the recall or imagination of traumatic events *(143,144)*. It is possible that increased amygdala responsivity plays a similar role in the enhanced encoding of trauma-related information.

Laboratory studies of emotional memory have also tried to shed light on the autobiographical memory abnormalities described in the beginning of this chapter. As discussed, some have suggested that the repeated intrusion of highly detailed traumatic memories triggered by situations that reinstate some characteristic of the traumatic event reflects the operation of involuntary, implicit memory processes that automatically activate information related to the traumatic event. If this view is correct, one might expect implicit memory for emotional information to be enhanced in individuals with PTSD. The evidence does not strongly support this view: Some studies have found enhanced implicit memory for threat- *(145,146)* or trauma-related *(147)* information in PTSD, but several other studies have not

(134,138,148). It is important to keep in mind, however, that the presentation of threat-related words in the laboratory in no manner mimics the richness of information—physiological, emotional, and cognitive—that may act as cues to activate intrusive memories in the real world.

CONCLUSION

For the sake of conceptual organization, we have discussed separately the status of autobiographical memory and new learning in PTSD, but of course, in the development and maintenance of PTSD, these two forms of memory are intrinsically linked. Less-proficient new learning prior to trauma will necessarily have an impact on the encoding of the trauma event and, consequently, its subsequent retrieval. For instance, a configural deficit as postulated by Gilbertson et al. *(119)* could lead to a failure to encode relevant contextual information during the traumatic event, with the result that later retrieval of the trauma memory will be more fragmented or lacking in important details. Conversely, intrusive memories (or attempts to suppress their occurrence) expend limited attentional and executive resources. As a result, the elaboration and organization of new incoming information may suffer, and retrieved information may not be adequately monitored. Longitudinal studies that evaluate new learning as well as autobiographical memory prior to and after trauma, or prior to and after treatment, will be critical to fully elucidate these interactions.

Just as there is a dynamic interplay between the encoding of new information and the revival of established memories, it is important to emphasize that memories themselves are not fixed but rather are continually susceptible to change. Upon retrieval of a memory, the memory trace may be updated with new information obtained during the retrieval situation, or new memories may be formed that become interlinked with the already existing one. Further, ample evidence now suggests that by virtue of retrieval, some forms of memory can be returned to a labile state in which the memory trace is modifiable and subject to reconsolidation *(149,150)*.

The malleability of memories has important consequences for PTSD. On the one hand, ongoing modification of memories can potentially provide a mechanism for the maintenance or exacerbation of PTSD symptoms. Reactivation of intrusive memories, accompanied by emotional and physiological changes experienced during the trauma, can lead to the incorporation of new contextual elements that are part of the current retrieval situation but not the original trauma event. As such, intrusive memories may strengthen memory of the traumatic event, not only through reactivation, but also by virtue of the fact that additional contextual elements may now in themselves act as cues to activate the traumatic memory.

On the other hand, the dynamic nature of memory may also offer the potential for altering traumatic memories in adaptive ways. Several successful cognitive-behavioral interventions for PTSD center on the trauma memory. The impetus for exposure interventions, for example, relies strongly on emotional processing

theory, which suggests that PTSD emerges and is maintained via a fear network in memory that leads to avoidance behaviors and emotional numbing. According to this theory, fear is reduced through activation of the fear structure and the introduction of new information inconsistent with the maladaptive components within the fear network. Thus, exposure interventions require not only activation of the trauma memory but also its modification *(151,152)*. Likewise, recent evidence indicates that it may be equally important to alter the meaning of the traumatic event, suggesting that access to cognitions associated with the trauma memories may be as essential to positive outcomes, if not more so, than reexposure per se *(153)*. To the degree that access to trauma memories can be improved, as shown by Sutherland and Bryant *(47)*, it could be reasoned that incorporation of new information in memory in ways that reduce fear, sadness, anger, and other trauma-related emotions could be facilitated.

Pharmacological interventions may also provide a mechanism by which traumatic memories can be altered. Preliminary evidence suggests that administration of a b-adrenergic blocker in association with retrieval of a traumatic memory may reduce physiological responding to imagery of that traumatic event a week later *(154)*, but much further work is needed to explore the boundary conditions and mechanisms of this effect.

Finally, in considering the clinical significance of memory to PTSD, it is critical to keep in mind that memory does not work in isolation but operates in the service of a person's current goals and concerns, serving an important functional role in maintaining a coherent sense of self. In Conway and Pleydell-Pearce's *(17,18)* model, the encoding and retrieval of autobiographical memories reflect a constant interplay between the working self and the autobiographical memory knowledge base. Thus, the need to maintain a coherent sense of self will bias which memory contents are retrieved as well as the particular features or aspects of a memory that are emphasized; the dominance of particular memories will in turn influence one's sense of self. Illustrating the former, the amplification of trauma memories over time in PTSD may reflect at least in part an attempt to make sense of developing psychopathology and the accompanying change in the sense of self *(56,58,155–157)*. Consistent with the latter, a positive relation has been found between the extent to which trauma-focused memories become a central reference point for organizing other memories and the presence and severity of symptoms of PTSD *(158)*. The intimate relationship between memory and self has important implications for treatment. Changing the memory bias, by virtue of altering or better integrating trauma memories or by introducing nontraumatic memories in situations that are perceived to be threatening, may dampen the impact of a traumatic experience on one's sense of self. Likewise, changes in the sense of self and its associated goals may have an impact on the accessibility of memories, favoring those that are consistent with a more positive self-appraisal.

In closing, the study of memory in PTSD is a rich and fruitful area of research. At a theoretical level, a number of debates remain—the nature of trauma memory

and the underlying neuroanatomical basis of memory abnormalities in PTSD, to name but two. Future work will undoubtedly lead to a better understanding of the basic mechanisms underlying memory alterations in PTSD; it likely also will have important ramifications for cognitive and neural theories that aim to elucidate the complex interactions between emotion and memory in normal functioning. At a clinical level, recognition of the centrality of memory in PTSD now opens the way to the development and refinement of treatment options, whether behavioral or pharmaceutical, that are informed by our understanding of the nature of memory processes and representations. This convergence of basic and applied interests and the potential for the study of each to inform the other provide a particularly exciting juncture in the study of memory in PTSD.

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