The Recovered Memory Debate

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July 1, 1997

Introduction

Understanding memory, *the search for the engram*, is the holy grail of psychology and neuroscience. Researchers are continually searching for answers to different aspects of how memory works. Yet, in 1997 when someone mentions the 'memory debate' it is generally understood that this refers to the controversy involving the recovery of repressed memories of childhood abuse, and the possibility that such memories are illusory. The reasons for this controversy being a 'memory debate' lies in the fact that otherwise objective scientists have become adversarial in their treatment of the issue. One camp, 'the skeptics' represented by the False Memory Syndrome Foundation (FMSF), has been vehemently arguing that recovered memories are generally unreliable and that therapists have convinced unwitting patients that childhood abuse occurred when it had not. The other camp, 'true believers' formed as a reaction to FMSF in order to protect people who claimed to have recovered memories; but, they seem to be willing to believe in the authenticity of any claim of recovered memory without any scientific evidence (Loftus, 1994b). Unfortunately, much of the argument has been political rhetoric and lacking in the hard data that is required for a scientific debate. The media has also contributed to the fray: accepting without question assumptions about recovered memories. For example, one reporter, Lawrence Wright, wrote in a *New Yorker* article that "thousands of ...people have been accused on the basis of recovered memories...and certainly many are false"(May 24, 1993 p.76). When personally asked how many cases Wright had actually documented, he admitted, "One" (Pope, 1996). Since, the debate deals with very sensitive and important issues, scientists investigating memory must be careful not to appear as trying to feed the fire of one side or the other. Even in our definition of the 'memory debate' we must be careful with using words such as 'repressed', and 'illusory'. These terms will be explicitly defined as they are discussed later in the paper.

While many issues surrounding the 'memory debate' remain unresolved, psychologists and neuroscientists have answered many questions, including the fundamental question "Do false memories exist?" Through clinical and laboratory studies investigating suggestibility, accuracy of regular and emotional memories, and accuracy of source memories (the knowledge of where,

when, how a memory was acquired), it has been shown that memories are not always accurate and sometimes memories are disturbingly inaccurate. Moreover, young children and some adults are especially suggestible. As well, confidence in accuracy of memory is not necessary correlated with accuracy (Schacter, 1996a). On the other side of the debate, can people recover repressed memories? Several clinical cases indicate that people *can* recover previously repressed memories of childhood abuse (Martinez-Taboas, 1996).

To explain the neural substrates for the repression and later recovery of memories, neuroscientists have been investigating implicit (unconscious, emotional) vs. explicit (conscious, declarative) memory, as well as memory modulatory systems. A memory modulation system has been defined as a system that : does not depend on the type of memory (e.g. Spatial memory, procedural memory, motor memory, etc.); is not required for learning; and the locus of the enhancement/impairment should be stored separately from the memory modulation site (Packard et al, 1994). The amygdala has been identified as the site of modulation of memory due to emotion and stress (Gallagher et al, 1996). Furthermore, epinephrine, norepinephrine, and cortisol (a glucocorticoid), have been implicated in being responsible for many of the effects related to stress: enhanced memory for acute emotional events, as well as the neurotoxic effects of chronic stress (Cahill et al, 1996). These physiological investigations are a step towards a physiological explanation for repressed and recovered memories.

One goal of scientists should be to try and find a way to determine the accuracy of memories to prevent families from being destroyed. While that goal may be far off, there are steps that can be taken: to try and unravel the neural mechanisms at work and to try and minimize the damage to individuals and society. This paper is an effort to investigate the different aspects of the debate and possibly uncover some of the ways that the problems surrounding the memory debate can be alleviated.

False Memories

The False Memory Syndrome Foundation (FMSF) was founded in 1992 by psychologists and parents who were concerned that there was an epidemic of false accusations of sexual abuse in America. Illusory, or false, memories can be memories of an event that never took place or a distorted memory of an event that did take place. The claim of the FMSF is that accusations of sexual abuse stem from memories implanted by negligent clinicians. While the FMSF's reputation has been tarnished by comments of some of its members (For example, Dr. Richard Underwager was quoted in a Dutch journal that sex with children is a 'responsible

choice for the individual'. He resigned from the FMSF after that comment), their concerns have some validity.

The case of Diana Halbrooks is especially interesting because she recovered memories of abuse in therapy and then later decided that those memories were false; she is a retractor. It is also of interest to note that Diana's memories were first recovered using hypnosis. Then, she joined a support group of women who were exploring traumatic memories. Diana recalls "The memories that others were sharing ...were getting more and more bizarre : satanic ritual abuse, babies being sacrificed ....horrible tortures." Eventually, these images found there way into Diana's memories. The therapist diagnosed almost all the members of the group as multiple personalities. Diana's case is not isolated. A recent survey of twenty retractors shows that 95% of the subjects recovered memories in suggestive therapy. 90% reported that hypnosis was used (Schacter, 1996a).

A patient, Ann, who had emotional issues with her parents because of their (the parents) separation, began therapy. There Ann discovered that she harbored multiple personalities and recovered memories of satanic ritual abuse at the hands of her parents. There was no evidence for this abuse; Ann had perfect attendance in school and no teacher had ever seen bruises or injuries that would have resulted from the reported abuse. The therapist stated, "I don't care if it's true, what's important to me is the patient's truth ...We all live in a delusionary world, just more or less delusionary" (Schacter, 1996). The attitude that therapists are not detectives searching for 'objective truths', rather they are there to support their patients is shared by many in the profession (Martinez-Taboas, 1996). But it is questionable whether therapists should validate accusations of abuse when there is no supporting evidence. "Not only does rape hurt everyone, but false memories of rape hurt everyone too" (Loftus et al, 1994a. pp. 1180).

In addition, it has been established that suggestive questioning can have devastating effects on the accuracy of children's memory (Ceci et al, 1981; Ceci, 1995; Schacter, 1996). In one experiment children were asked questions about everyday events that had occurred but also asked questions about events that hadn't occurred. For example, an interviewer would once a week, for ten weeks, ask a child to visualized the time "she caught her finger in a mousetrap." Finally, the interviewer would probe the child's memories. "Did you ever get you finger caught in a mousetrap?" Over half the children produced memories for a made-up incident (Ceci, 1995). Moreover, the children developed detailed narratives to surround the incident. More disturbing, perhaps, is that experts who witnessed videotapes of children's interviews could not distinguish confabulations from accurate memories. This evidence demonstrated the importance

of taking precautions to avoid suggestive questioning in cases of sexual accusations from children.

Other experiments have shown that as children age, they are less suggestive (Bremner et al, 1996). So what is it about young children that make them so suggestible? It seems that their 'false memories' are formed because of poor source memory. In Ceci's experiment the children were told to visualize the incident for 10 weeks. Thus, they had in their heads a narrative of the event: an imaginary, but complex and detailed one. The problem was that they didn't remember that it was a story told to them and not an actual event. This kind of source amnesia is seen in patients with frontal lobe damage. One patient, Gene, had some damage to the medial temporal lobe and extensive damage to frontal lobes. Dr. Schacter would tell him bits of trivia ("Richard Nixon loves oatmeal") Then a minute later would ask Gene, "What does Nixon like to eat for breakfast?". Surprisingly, Gene could sometimes give the right answer, at a higher than chance level. However, when asked how he knew it he would say that it was a lucky guess, or he'd read in the paper. He never said, "You just told me a minute ago" (Schacter, 1996a, pp. 118-119). The link between Gene and children's source amnesia, is that children's implicit memory systems are in place very early, possibly at birth, while their explicit memory systems -including source memory- which depend on the hippocampus and frontal cortex, are not mature until much later in life (Hovstead et al, 1996).

There are theories that some memories of emotional events are indelible (LeDoux, 1994; Bremner et al, 1996). For example, many people claim they know exactly where they were, what they were doing, etc. when they heard of President Kennedy's assassination. These memories are coined "Flashbulb memories". To test the validity of these memories, several studies have been conducted. Subjects were given a questionnaire one week, and then two years after, an historic event. e.g. the explosion of the Challenger space shuttle. These studies found that "flashbulb memories" are special in that people have a high confidence in them, but unfortunately these memories are subject to distortions nonetheless (for review see Schacter, 1996a. pp. 195-201). This data, along with the Ceci's experiments, and cases like Ann's and Diana's demonstrate that memories are not always accurate, from slight distortions to complete fabrications, and can be the result of source amnesia or suggestion.

Dissociative Amnesia / Recovered Memories

While false memories can occur, there is also evidence that recovery of previously forgotten memories does occur, as well. Retrograde amnesia followed by sudden recovery is well

documented (Lucchelli et al, 1995; Schacter et al, 1982; Martinez-Taboas, 1996). The cases studied by Lucchelli and his colleagues are amnesiacs, whose functional impairments were caused by head trauma and stroke. By studying these organic amnesias and the following recoveries, much can be learned about retrograde amnesia and retrieval mechanisms. However, more applicable to this paper are the cases documented by Martinez-Taboas.

In his paper, he describes two cases of dissociative amnesia where there was proof of childhood sexual abuse. As well, both patients were previously diagnosed with dissociated identity disorder (DID). In the case of Madeline , an new alter, named André, appeared during therapy, who admitted to raping the patient as a child. Madeline knew André, a cousin, and he admitted to the abuse when confronted. In the second case, a patient, Evelyn was in therapy because of DID. As part of the initial interview the therapist asked if she had ever been abused. Evelyn stated that while her family life was bad, no abuse had ever occurred. Months later a new child alter appeared (outside of therapy) who was frightened and claimed that her father was touching her. This abuse was corroborated by the patient's sister who had previously told the therapists that her father had raped her and her sister. These cases are clear evidence that dissociation and later recovery of memory can occur. It is important to note that in both these cases there was a history of psychopathology, specifically DID. Thus, evidence of the phenomena of recovery of repressed memories is not in question; nonetheless, evidence of the phenomena with no previous history of dissociative disorders is still unavailable. And in the opinion of many of the experts in this field, it may never become available (Bremner et al, 1996; Schacter et al, 1996a; Hovstead et al, 1996).

Psychologists have thought deeply about the different mechanisms that would allow a survivor of a traumatic event to forget the incident. The first possibility is that the process is the same as the process by which one forgets a normal event. While this seems unlikely (as we usually remember very important and novel experiences), it is conceivable that the victim of one incidence of abuse may forget it (likewise a victim of chronic abuse may forget a specific incident of abuse) if they never talk about it or rehearse the memory verbally. Since abuse is a taboo subject, this situation is possible. The second possibility is that there is a special mechanism for forgetting traumatic events.

These kinds of forgetting were documented in a study by Williams et al. (1994). Williams reported interviews with 129 women, who 17 years earlier had been admitted to the hospital as victims of sexual abuse ages 2-12. 38% of the women did not remember that specific incident. Moreover, 12% of the women had no memory of any childhood abuse. How is that the 12%

forgot? It is possible that some of the 16 (12% of 129) women were withholding information, but Williams' study is one of many to document forgetting of traumatic incidents (for review see Hovstead et al, 1996). In this case, regular forgetting does not seem powerful enough. The common term to describe this forgetting is repression. There are two different definitions of repression. Repression can be defined as the process of directed forgetting, where the survivor consciously avoids to think about, talk about, or otherwise rehearse an unpleasant experience. This is conscious repression. On the other hand, unconscious repression, dissociation, may occur: where the mind fails to include the traumatic experience as part of the autobiographical narrative of the person. The first kind of repression seems unlikely to be able to cause someone to forget a period of chronic abuse, while dissociation has been implicated strongly through studies from all field of cognitive neuroscience (Bremner et al, 1996; Hovstead et al, 1996).

Modulation of multi-modal memory

It has been known for decades that more than one kind of memory exists in the human brain. Patient HM, demonstrated that without any conscious explicit memory for a task (due to extensive medial temporal lesions), he could improve, showing that there was a kind of procedural memory, separate from explicit memory (Corkin, 1984). Tranel et al (1995) revealed a very relevant separation of memory systems, when they demonstrated that patients with a bilateral ventromedial frontal lesion had explicit but no emotional (judged through galvanic skin response) knowledge of familiar faces, whereas subjects with occipitotemporal lesions had no explicit memory for familiar faces, but did have emotional memory for the faces. Therefore, dissociation due to trauma, may be dissociation between the explicit and implicit memory systems which are to some extent independent. While this is still speculation, a lot of research has been performed to try and elucidate the mechanisms of the implicit , emotional memory system and to determine the effects of stress molecules: glucocorticoids, epinephrine, norepinephrine etc. on memory.

Researchers have established that emotional experiences have a modulatory effect on memory. Subjects who watch an emotional narrative vs. controls watching a neutral story , show enhanced memory for central details but inhibited memory for peripheral details. So, not only is there separation of implicit and explicit memory, but emotional, stressful, situations can have a modulatory effect on explicit memory. This effect was shown to be dependent on β- adrenergic systems (Cahill et al, 1994). In another experiment, lesions of the amygdala were shown to reduce dopamine release into the frontal cortex, which is crucial for the body's reaction to stress.

(Davis et al, 1994). The amygdala was firmly established as a site of memory modulation in a study by Packard et al. (1994). In this two part experiment, rats were trained on two tasks, one a spatial task, shown to require hippocampus, and one cued task , shown to require the caudate nucleus. One group of rats on each task were giving post-training injections of amphetamine, previously shown to enhance memory, into the hippocampus, caudate, or amygdala. Only caudate injected rats did not have enhanced for the spatial tasks compared to controls, and only hippocampus injected rats did not show enhanced performance on the cued tasks. Injections into the amygdala enhanced performance on both tasks. In the second part, Packard et al established that the amygdala was not the locus of change, by injecting lidocaine into the amygdala before testing. The results demonstrated that the amygdala was not the locus of enhancement and was not required for the performance of the task, satisfying the criteria for a memory modulatory system. Every study reviewed here so far has linked stress to enhanced memory, however, several studies, mainly on war veterans and survivors of childhood sexual abuse, have outlined the detrimental effects of stress on memory.

In a recent study Southwick et al established a correlation between post-traumatic stress disorder (PTSD) and consistency of memory (Southwick et al, 1997). Veterans of Desert Storm filled out a questionnaire one month and two years after combat. The questions were of the form, ”Did you have this experience in the war?” e.g. “Did you witness a death?” Over 61% of the respondents changed more than 2 (out of 19) answers and veterans with PTSD had a tendency to change no answers to yes answers. In a study like this many factors could influence memory : media, family, etc. Nonetheless, the trend of PTSD subjects to ‘recall’ more stressful experiences two years later, could be due to an attempt to justify their current conditions. In an earlier study Bremner et al (1995) established a possible physiological correlate for this result while demonstrating the neurotoxic effect of stress. Veterans of Vietnam with PTSD had on average 8% smaller right hippocampus when controlled for by age, education, height, weight, alcohol abuse and comparison structures. There was no significant difference between PTSD and Non-PTSD veterans in left hippocampus, caudate or temporal lobe volumes. This finding was corroborated by a study that found reduced hippocampal volumes (5%) in survivors of childhood abuse compared to controls (reviewed in Schacter et al, 1996a). Again, without pre-morbid measurements, it is hard to say that stress resulted in damage to the hippocampus. However, in studies with monkeys, increased cortisol has resulted in damage to CA2 and CA3. Newcomer et al (1994) demonstrated that glucocorticoid injections had a delayed detrimental effect on verbal memory. In addition, glucose, epinephrine, norepinephrine, and ACTH all have

inverted-U functions on memory: too little or too much of these compounds can have detrimental effects on memory (reviewed in Bremner et al, 1996; Cahill et al, 1996). Based on these studies, Terr established a theory of trauma, where Type I trauma was acute and resulted in enhanced memory for the event, whereas, Type II trauma was chronic and could result in dissociation, and forgetting. However, there is no evidence that survivors of chronic abuse remember their abuse less than survivors of one incident. (Schacter, 1996a)

Although, it's been documented that chronic stress can have detrimental effects on memory and possibly a neurotoxic effect on the brain, possibly through increasing neurons vulnerability to amino acid neurotransmitters, such as glutamate (Bremner et al, 1995), the above studies do not explain how dissociation, resulting in selective retrograde amnesia, could occur. One study by Sternberg et al (1981) demonstrated that both α- and β-adrenergic antagonists attenuate retrograde amnesia that was produced by sub-seizure electrical stimulation of the amygdala. This study suggests a model where stimulation to the amygdala causes dissociation of implicit and explicit memory through adrenergic systems.

There are two paths from the outside world to the amygdala. There is a fast, sensory-thalamic-amygdala path and a slower sensory- thalamic-cortical-amygdala path (LeDoux, 1994). These two streams are illustrated by the following example. Pretend you are walking in a dense forest at twilight. You hear a rustling of leaves to your left. You turn and see a large shadowy object, possibly a bear? Your sympathetic nervous system activates, raising your blood pressure, heart rate, etc. This is the fast thalamic-amygdala path. Then, as your cortex processes the visual information and you realize it's only a large rock, you calm down. But, in a situation of extreme stress, such as sexual abuse, it's possible that the fast connection to the amygdala stimulates it so much that it inhibits the slower inputs from the cortex to the amygdala and possibly to other sub-cortical structures (LeDoux, 1992). This would result in the kind of dissociation seen in patients, where memories are fragmented and very sensory as opposed to our normal memories that are placed in a narrative context. "*The more a person experiences extreme terror during an event, the more likely the imagery of that event will be inscribed in the same sensory modalities of memory as the perceptions"* (Horowitz et al, 1992)

State-dependent retrieval will also play a role in traumatic memories. During trauma a victim is in an extreme psychological state, one that is unlikely to recur in normal everyday life and this adds to the problems with retrieval. This dissociation means that patients must 'relive' experience to recall initial one. For example, some women only remember childhood abuse during a rape incident, where both the physical experience and the mind-state of the original

incident are reproduced. Moreover, a process coined "kindling" may take place, where repeated stimulation of a memory trace can cause long-term sensitization to the point where very general input can cause activation of the trace. In the case of a child who suffers chronic abuse, just seeing the room where the abuse occurs, or the perpetrator of the abuse, could evoke the memory trace, activating stress molecules, like norepinephrine and endogenous opiods that may trigger the dissociative state. (Hovstead et al, 1996)

Much research needs to be done in order to truly understand the mechanisms through which traumatic memories are encoded, stored and retrieved. Nonetheless, there are several courses which this research should take. One course would be to examine the inhibitory effect of amygdala stimulation on cortical inputs to sub-cortical structures. Another path is to discover the quality and neural correlates of illusory memories (Schacter et al, 1996b; Schacter et al, 1996c; Schacter , 1996b). Although, it is a preliminary finding , imaging studies have indicated that the key to differentiating between illusory and veridical memories is that veridical memories have a sensory component. For example, the subject remembers hearing, or seeing the word, whereas, in an illusory memory there is semantic association but no sensory memory (Schacter et al, 1996d).

Conclusion

This review has covered information from different fields of psychology: social, clinical, and physiological. The unifying force is that all the data presented can shed some light upon the recovered memory debate. Let us construct a fictional character, Mary, a child whose father at age 10 begins to sexually abuse her. During the abuse encoding of the memory takes place. Normally this involves the hippocampus and explicit memory systems. However, because of the extreme terror of the situation, the fast thalamic-amygdala -path stimulates the amygdala and triggers the release of massive amounts of stress-related molecules, especially norepinephrine and endogenous opiods. These neurotransmitters act to inhibit the projections from the cortex to the sub-cortical structures resulting in memories of the abuse that are fragmentary and sensory, and not available as explicit memories. The abuse continues; Mary experiences extreme terror in the presence of her father, and the dissociation is exacerbated by kindling, sensitizing the implicit memory of the abuse associated with her father, that the dissociation occurs whenever she is alone with her father. At age 14 Mary's father leaves his wife and children. Mary has a lot of trouble at school, because of anti-social behavior and intermittent flashbacks: terrifying sensory experiences that have no context. As well, during sleep, when the frontal cortex is less

active, the implicit memories sometimes surface, resulting in nightmares. As a young adult, 19, with symptoms characteristic of DID, she begins therapy. She does not remember any abuse, although she is aware of her poor family life as a child. The therapist creates a trusting relationship, where Mary feels that she can consciously and unconsciously lower her defenses. She discusses the flashbacks and nightmares. Her therapists has her draw images from the fragmentary memories, and uses other techniques. This therapy helps Mary to integrate the fragmentary implicit memories into her autobiographical context, and eventually retrieval takes place; Mary recovers memories of her abuse. Nonetheless, four years of chronic abuse and subsequent dissociation have had a detrimental effect, resulting in neuroses and below average scores on verbal memory tests, because of the neurotoxic effects of the chronic stress.

On the other hand there is Jane, who seeks therapy at 32 because of insomnia, eating disorders and compulsive behavior. Her therapist, who recovered memories of his own childhood abuse, believes that these symptoms indicate a history of abuse. When normal therapy fails to result in Jane's retrieval of those memories, her therapist uses hypnosis, trance writing, and other suggestive techniques, asking Jane to visualize the abuse taking place. During a hypnotic trance, Jane "sees" the abuse taking place. In this situation Jane is like the children in Ceci's experiment: the hypnosis weakens her source memory. Jane then accuses her parents of abuse, which they deny. Her extended family and friends urge Jane to see another therapist. Reluctantly she does, and after a few weeks is diagnosed and treated with medication for obsessive compulsive disorder. Jane retracts her accusations, and reunites with her family.

These two fictitious stories were presented for two reasons. The first being to integrate the research findings into a narrative to see how the results apply to someone's life. The second is to try and illustrate how much cognitive neuroscience has contributed to the recovered memory debate and where it can, and must, contribute further. As previously mentioned, Schacter and his colleagues continue to contribute through research into the differences between recall of actual events and recall of illusory events. This effort is drawing on behavioral data, human lesion data, and medical imaging techniques (Schacter, 1996b). Other scientists are working to uncover the secrets of the amygdala and emotional memory (Gallagher et al, 1996, LeDoux 1996). Perhaps studying how the memory modulatory systems works in normal stress conditions, will lead to solutions to the problem of dissociation. Cognitive scientists are applying parallel distributed processing theory to create models of memory which can account for dissociation and later recovery (Luchelli et al, 1995) and models that account for the unusual aspects of PTSD (Brewin et al , 1996). From the clinical aspect, efforts must be made to establish a set of guidelines when

dealing with patients whose symptoms may indicate a history of repressed childhood abuse (Pope, 1996). Moreover, all parties involved in the recovered memories debate must remember that political and emotional rhetoric only exacerbate an already sensitive situation, and that scientific facts must be the basis of any relevant argument.

References

Bremner JD, Krystal JH, Charney DS, Southwick SM (1996) Neural Mechanisms in Dissociative Amnesia for Childhood Abuse : Relevance to the Current Controversy Surrounding the "False Memory Syndrome". *Am J Psychiatry Festschrift Supplement* 153 : 7 : 71-82

Bremner JD, Randall P, Scott TM et al (1995) MRI-based Measurement of Hippocampal Volume in Patients With Combat-Related Stress Disorder. *Am J Psychiatry* 152:973-981

Brewin CR, Dagleish T, Joseph S (1996) A Dual Representation Theory of Posttraumatic Stress Disorder. *Psychological Review* Vol 103 No 4 : 670-686

Cahill L and McGaugh JL (1996) Modulation of memory storage. *Current Opinion in Neurobiology* 6: 237-242

Cahill L, Prins B, Weber M, McGaugh JL (1994) B-adrenergic activation and memory for emotional events. *Nature* Vol 371:702-704

Ceci SJ, Caves RD, Howe MJA (1981) Children's long term memory of information that is incongrous with their prior knowledge. *BR J Psychol*. 72 : 443-450

Ceci SJ (1995) False Beliefs: Some developemental and Clinical considerations. , Memory distortion: How minds, brains, and societies reconstruct the past. (pp. 91-128) Cambridge: Harvard University Press.

Corkin S (1984) Lasting consequesnces of bilateral medial temporal lobectomy: Clinical course and experimental findings in H.M. *Seminars in Neurology*. 4 :249-259

Davis M, Hitchcock JM, Bowers MB, Berridge CW, Melia KR, Roth RH (1994) Stress-induced activation of prefrontal cortex dopamine turnover:blockade by lesions of the amygdala. *Brain Research* 664 : 207-210

Gallagher M, Chiba A (1996) The amygdala and emotion. *Current Opinion in Neurobiology* 6:221-227

Horowitz MJ, Reidbord SP (1992) Memory , Emotion, and response to trauma. In S.A. Christianson (Ed.) The handbook of emotion and memory (pp. 307-340) Killsdale, NJ

Hovstead W, Kristiansen C (1996) Mind meets Body: On the Nature of Recovered Memories of Trauma. *A Feminist Clinician's Guide to the Memory Debate* Vol 19 No 1. pp 31-45.

LeDoux JE (1994) Emotion, memory and the brain. *Scientific American* 270(6) :50-57

LeDoux JE (1992) Emotion as memory: Anatomical systems underlying indelible neural traces. In S.A. Christianson (Ed.) The handbook of emotion and memory. (pp 269-288) Killskale, NJ

LeDoux, J. E. (1996). The emotional brain. New York: Simon & Schuster.

Loftus E, Garry M, Feldman J (1994a) Forgetting Sexual Trauma: What does it mean when 38% forget? *Journal of Consulting and Clinical Psychology*. 62: 1177-1181

Lofus E & Ketcham K (1994b) The Myth of the Repressed Memory. New York, St. Martin's Press.

Lucchelli F, Muggia S, Spinnler H (1995) The 'Petites Madeleines' phenomenon in two amnesiac patients Sudden recovery of forgotten memories. *Brain* 118:167-183

Martinez-Taboas A (1996) Repressed Memories: Some Clinical Data Contributng toward Its Elucidation. *American Journal of Psychotherapy*. V. 50 No. 2 :213-230

Newcomer JW, Craft S, Hershey T, Askins K, Bardgett ME(1994) Glucocorticoid-induced Impairment in Declarative Memory Performance in Adult Humans. *J. Neurosci* 14(4):2047-2053

Packard MG, Cahill L, McGaugh JL (1994) Amygdala modulation of hippocampal-dependent and caudate nucleus-dependent memory processes. *Proc Natl Acad Sci USA* 91:8477-8481

Pope K (1996) Scientific Research, Recovered Memory , and Context: Seven Surprising Findings. *A Feminist Clinician's Guide to the Memory Debate* Vol 19 No 1. pp123-140

Schacter D, Koustaal W, Norman K. (1996a) Can Cognitive Neuroscience illuminate the nature of traumatic childhood memories?. *Current Opinion in Neurobiology*. 6: 207-214

Schacter DL, Verfaellie M, Pradere D (1996b) The Neuropsychology of Memory illusions: False Recall and Recognition in Amnesiac Patients. *J Memory and Language* 35 :319-334

Schacter DL, Alpert NM, Savage CR, Rauch SL, Albert MS (1996c) Conscious recollection and the human hippocampal formation: Evidnece from positron emission tomography. *Proc Natl Acad Sci USA* 93 : 321-325

Schacter DL, Reiman E, Curran T, Yun LS, Bandy D, McDDermott KB, Roediger HL (1996d) Neuroanatomical Correlates of Veridical and Illusory Recognition Memory: Evidence from Positron Emission Tomography. *Neuron* 17: 267-274

Schacter DL, Wang PL, Tulving E, Freedman M. (1982) Functional retrograde amnesia: a quantitative case study. *Neuropsychologia* 20:223-32

Schacter, DL (1996a). Searching for Memory; Basic Books, New York

Schacter DL (1996b) Illusory Memories : A cognitive neuroscience analysis. *Proc Natl Acad Sci*. 93 : 13527-13533

Southwick SM, Morgan CA, Nicolaou AL, Charney DS (1997) Consistency of Memory for Combat-Related Traumatic Events in Veterns of Operation Desert Storm. *American Journal of Psychiatry* 154: 173-177

Sternberg D, Gold P (1981) Retrograde Amnesia produced by electrical simulstion of the amygdala:attenuatuon with adrenergic antagonists. *Brain Research* 211:59-65

Tranel D, Damasio H, Damasio AR (1995) Double Dissociation between Overt and Covert Face Recognition. *Journal of Cognitive Neuroscience* 7 (4) : 425-432

Williams, LM (1994) Recall of childhood trauma:a prospective study of women's memories of child sexual abuse. *J Consult Clin Psychol*. 62 : 1167-1176.