12 Trauma and Memory Revisited

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Abstract

The idea that trauma causes amnesia by virtue of psychopathological processes such as repression and dissociation has been a fixture of clinical folklore (and popular culture) for more than a century. Repression, suppression, and dissociation are distinguished in conceptual terms, as are "functional" and "psychogenic" amnesia. Modern versions of the trauma-memory argument, involving such concepts as "memory suppression" and the distinction between "hot" and "cool" memory systems, are critically examined. In view of the paucity of evidence that trauma causes amnesia in the first place, laboratory analogs of traumatic amnesia appear to be models in search of a phenomenon in the real world, and theories of traumatic amnesia to be explanations in search of facts.

The idea that psychological trauma can render its victims amnesic for the traumatic event has been a fixture of psychotherapy from its beginnings in the late nineteenth century. It was firmly enshrined in popular culture in the mid-twentieth century in films like *Random Harvest* (1942), *Spellbound* (1945), and *The Snakepit* (1948). Despite the rise of biological psychiatry, and a progressive shift from psychogenic to somatogenic theories of mental illness, the trauma-memory argument – and its companion, recovered-memory therapy – were revived as post-traumatic stress disorder was established as the "new hysteria" of our time – often with a neuroscientific cover story designed to make it more appealing to both professional and nonprofessional consumers of the contemporary psychotherapeutic literature.

The trauma-memory argument proceeds as follows (Kihlstrom, 1996, 1997, 1998; Shobe & Kihlstrom, 1997):

- Traumatic levels of stress sometimes lead victims to invoke mental defenses, such as repression and dissociation, which result in a "psychogenic" or "functional" amnesia for the stressful event itself.
- This amnesia affects explicit memory for the trauma, but spares implicit memory, so that representations of the event persist in symptoms such as "body memories."

- The presence of such symptoms as "body memories" is a sign that a traumatic event occurred, and that a representation of the traumatic event has been encoded, and is available in memory.
- This traumatic memory, ordinarily denied to consciousness, may be recovered, either spontaneously or by means of such techniques as guided imagery, hypnosis, and barbiturate sedation.
- In the absence of independent corroboration, the accuracy of the recovered memory may be inferred from its explanatory value in the context of the person's symptoms – or, simply, from the fact that the person gets better after the memory has been recovered.
- Exhumation of the traumatic memory is essential for coping with the trauma itself.

A SHORT HISTORY OF REPRESSION AND DISSOCIATION

In one form or another, the trauma-memory argument and recovered-memory therapy have been features of clinical lore since the late nineteenth century, when Pierre Janet and Sigmund Freud announced their respective doctrines of dissociation and repression. In contemporary discourse about trauma, the terms *repression* and *dissociation* are sometimes used interchangeably; or, in other cases, the term *dissociation* seems to be invoked in order to escape the taint of Freudian psychoanalysis.

Historically, of course, the doctrine of dissociation came first (Ellenberger, 1970; Kihlstrom, Tataryn, & Hoyt, 1993). Pierre Janet, trained as both a neurologist and a psychologist, described the elementary structures of the mind as *psychological automatisms*, each representing a complex act, finely tuned to the situation (both external and intrapsychic), preceded by an idea, and accompanied by an emotion (Janet, 1889). Under normal circumstances, a person's entire repertoire of psychological automatisms is bound together into a single unified stream of consciousness. However, under certain circumstances, one or more of these automatisms can be split off from the rest, and function outside phenomenal awareness, voluntary control, or both. In Janet's case of Irene, for example, ideas related to the death of her mother – the sight of her face, the sound of her voice, and the movements of carrying her body – were broken off from the rest of the stream of consciousness. Janet's term for this situation was *désagrégation*, translated into English as *dissociation*. One circumstance promoting dissociation was psychological stress, and the dissociations associated with stress were held to be responsible for the major symptoms of hysteria, one of two major categories of neurosis in Janet's system (Janet, 1907). Another circumstance was hypnosis,

leading to the historical association of hypnosis with hysteria (Kihlstrom, 1979; Kihlstrom & McGlynn, 1991).

Enter Sigmund Freud. As is well known, Freud, who also trained as a neurologist, spent a kind of postdoctoral year (1885–6) with Charcot at the Salpetriere clinic in Paris, where he was introduced to the wonders of hysteria and hypnosis, and also began a kind of professional rivalry with Janet. Freud actually made his early reputation with the study of aphasia (Freud, 1891/1953) – and he coined the term *agnosia*. But he took his newly acquired interest in hysteria back to Vienna, and within a couple of years began to develop an alternative account of the syndrome in terms of repression (*Verdrängt*; Breuer & Freud, 1893–5/1953; Freud, 1915/1957) – a concept which Freud had borrowed from Herbart's analysis of unconscious percepts (Herbart, 1816/1881). For Freud, early sexual experiences, which were repressed, combined later with events at the time of puberty to generate symptoms that appear in adulthood. Although Freud's theory of hysteria shifted from a focus on actual sexual trauma to sexual fantasies, repression remained at the heart of the story (Macmillan, 1991/1997). By 1914, Freud had identified repression as "the foundation-stone on which the whole structure of psychoanalysis rests" (Freud, 1914/1957, p. 16). By means of repression, and its supplementary defenses, people deny themselves conscious awareness of primitive sexual and aggressive impulses that conflict with the demands of reality and the strictures of society – "Monsters from the Id," in the lovely phrase of *Forbidden Planet* (1956), my favorite science-fiction movie of all time.

Within just a few years, the psychoanalytic juggernaut had swept Janet and dissociation into the dustbin of history. Janet's views were more recently revived by the late Ernest R. Hilgard, who proposed a "neo-dissociation" theory of multiple controls in human thought and action, to describe hypnosis and related phenomena (Hilgard, 1977; see also Kihlstrom, 1992a). Publication of *Sybil* (Schreiber, 1973) had already renewed popular interest in multiple personality disorder, which had been rather dormant since *The Three Faces of Eve* (Thigpen & Cleckley, 1957; film, 1957) won an Academy Award for Joanne Woodward (in a classic case of "What goes around, comes around" Woodward later played a psychiatrist to Sally Field in the film version of *Sybil*, 1976). There followed a virtual epidemic of multiple personality disorder in the 1980s and 1990s (Boor, 1982) – including an amusing contest, of sorts, between Eve and Sybil to determine who had the most personalities. In 1980, the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-III*) reclassified psychogenic amnesia, psychogenic fugue, and multiple personality disorder (MPD) as "dissociative" disorders (for reviews, see Kihlstrom, 1994; Kihlstrom, 2001, 2005; Kihlstrom, Tataryn, & Hoyt, 1993). In 1984 the International Society for the Study of Multiple Personality and Dissociation held its first annual conference, and the first scholarly monographs on MPD appeared in 1986 (Bliss, 1986; Ross, 1986).

With a growing awareness of the problems of returning Vietnam War veterans, as well as the victims of sexual assault, the *DSM-III* also saw the emergence of a new diagnostic category of *post-traumatic stress disorder* (PTSD). PTSD quickly became a popular diagnosis, as clinicians began noticing its characteristic symptoms – especially anxiety and depression – even in patients who did not appear to have been exposed to the usual sorts of traumas. Increasing societal interest in very real problems of incest and childhood sexual abuse, including the beginnings of the sexual-abuse scandal in the Roman Catholic Church, ignited a revival of Freud's early theory that hysteria was caused by repressed memories of infantile seduction (Masson, 1984). In 1981, a clinical study of father–daughter incest had made no mention of repression, dissociation, or amnesia (Herman, 1981); in 1987, the same author published a study in which more than 25 percent of patients in a therapy group for incest survivors were amnesic for their incest (see also Harvey & Herman, 1994; Herman & Schatzow, 1987).

The idea that trauma could be repressed, and that lifting the repression was critical to full recovery, spread like wildfire through American culture. Popular books like *The Courage to Heal* (Bass & Davis, 1988), probably the best-selling self-help book of all time, fanned the flames:

> If you are unable to remember any specific instances . . . but still have a feeling that something abusive happened to you, it probably did. (p. 21)
>
> Many survivors suppress all memories of what happened to them as children . . . Coming to believe that the abuse really happened, and that it really hurt you, is a vital part of the healing process. (p. 58)
>
> If you don't remember your abuse, you are not alone. Many women don't have memories, and some never get any memories. This doesn't mean they weren't abused. (p. 81)

Bass and Davis also published a list of 74 different symptoms ostensibly associated with sexual abuse, so that readers could determine for themselves whether, in fact, they might ever have been abused. A similar list even appeared in a professional book published by the American Psychological Association (Walker, 1994).

And so, after 100 years, we come full circle: Janet and Freud were vindicated, PTSD was the new neurosis, or at least the new hysteria, and dissociation was the new repression. While the traditional diagnosis of PTSD referred to intrusive memories, the boundaries of the disorder were expanded to include amnesia as well. And because trauma caused dissociation, the dissociative disorders could be reconstrued as forms of PTSD. In 2000, the journal *Dissociation* was renamed the *Journal of Trauma and Dissociation*, as if the two were closely related. And in the run up to the publication of *DSM-V*, there is a serious move afoot to reclassify the dissociative disorders as forms of PTSD.

THE CURRENT SCENE

The trauma-memory argument is still with us. As recently as 2004, a highly regarded science writer published a book detailing how unconscious traumatic memories exert toxic effects on mind and body, and how secret traumas can be unlocked and overcome by "power therapies" such as Eye Movement Desensitization and Reprocessing, Thought Field Therapy, and the like (Scarf, 2004). As this paper was being written, the *New York Times* (02/08/05) reported that Paul Shanley, a former Catholic priest, had been convicted in Boston of a single count of child sexual abuse based on uncorroborated recovered memories from 20 years before. Shortly thereafter, the *Times* (02/24/05) carried a story about Martha Beck, a sociologist and psychotherapist who writes a self-help column for Oprah Winfrey's *O* magazine, who has written a memoir detailing recovered memories of sexual abuse allegedly perpetrated by her father, Hugh Nibley, a prominent Mormon scholar – claims strongly disputed by Beck's seven siblings (he died at age 94 the day the article was published).

Repression and dissociation compared and contrasted

In the contemporary literature, the terms *repression* and *dissociation* tend to be used interchangeably to refer to a lack of conscious awareness of trauma, conflict, and anxiety (Singer, 1990). In fact, Janet believed that repression was merely a special form of dissociation. But Freud held that dissociation was utterly trivial, and repression was a separate process with its own ontological status. In fact, the two concepts do seem to be different. As Hilgard (1977) noted, dissociation entails a vertical division of consciousness, while in repression the division is horizontal. For Freud (at least the early Freud), available memories are located in the System *Cs* (Conscious) and the System *Pcs* (Preconscious), while repressed memories are buried in the System *Ucs* (Unconscious) underneath a barrier of repression. For Janet, dissociations occur among memories that are normally available to consciousness. For Freud, repressed memories have special emotional and motivational properties, closely bound either to trauma (in his early theorizing) or with primitive sexual and aggressive impulses (in his later work). For Janet, any kind of memory at all can be subject to dissociation.

For Freud, repression is motivated by considerations of defense – the whole point of repression is to prevent us from becoming aware of threats and impulses that would cause us great anxiety. But in Janet's theory, dissociation *just happens* as a result of some weakness, or excessive strain, in the stream of consciousness – much the way a chain, when stretched too tightly, will break at

its weakest link. Further, Janet appears to believe that one could gain access to dissociated ideas directly, by techniques such as hypnosis that bridge the dissociative gap. By contrast, Freud seems to argue that repressed mental contents can be known only indirectly, by inference: hence Freud's abandonment of hypnosis and subsequent emphasis on the interpretation of dreams, and of symptoms as symbolic expressions of underlying conflict. In this respect, at least, modern recovered-memory therapy – while certainly inspired by Freud's ideas about repression – is closer to Janet's ideas about dissociation.

Implicit memory of trauma

One point on which both theories agree is that unconscious memories of trauma manifest themselves, unconsciously, in the form of symptoms, dreams, and the like. For Janet, these are the *stigmata* of hysteria (Janet, 1907). For Freud, "Hysterics suffer mainly from reminiscences" (Breuer & Freud, 1893–5/1953, p. 7), and their symptoms are evidence of "the return of the repressed" (Freud, 1896/1962, p. 169). Blume (1990) echoed Freud directly when she wrote:

> Hysterical symptoms . . . represent unremembered trauma or unacknowledged feelings. Because there is a physical distraction, the survivor is at once protected and blocked . . . Her body remembers, but her mind does not. (p. 93)

Similarly, van der Kolk (1994) asserted that unconscious memories of trauma are expressed as somatic symptoms. In his phrase, "the body keeps the score" (p. 253).

Fredrickson (1992) offered a catalog of the various ways in which repressed memories can return to haunt us:

Imagistic memory: incomplete or exaggerated pictures of the abuse scene.
Feeling memory: inexplicable emotions pertaining to the event.
Acting-out memory: including both verbal and bodily acts, as well as actions that occur in dreams.

In modern memory research, we would discuss repression and dissociation in terms of memories of traumatic events that have been encoded, and remain available in storage, but are inaccessible to retrieval. Evidently, the failure of access covers only explicit expressions of traumatic memory; implicit memories of trauma continue to influence the victim's ongoing experience, thought, and action – in the absence of conscious awareness, and independent of conscious control. Van der Kolk (1994) specifically invoked the concept of implicit memory when discussing the aftermath of trauma and abuse:

> Research into the nature of traumatic memories indicates that trauma interferes with declarative memory (i.e., conscious recall of experience) but does not inhibit implicit, or nondeclarative, memory, the memory system that controls conditioned emotional responses, skills and habits, and sensorimotor sensations related to experience. (p. 258)

Although the concept of unconscious memory is very old, predating Freud (Butler, 1880/1910), the formal distinction between explicit and implicit memory began to emerge only a century later, in the mid-1980s (Schacter, 1987). Like Hilgard's neo-dissociation theory of divided consciousness (Hilgard, 1977), this new work inadvertently gave intellectual aid and comfort to the recovered memory movement – at last, science had proven that unconscious memories can exist! But there is a big difference between inferring the existence of unconscious memories in the laboratory and doing so in the clinic.

In the laboratory, implicit memories are commonly expressed as priming effects – in which, for example, prior study of the word *ashcan* leads subjects, and even amnesic patients, to complete the word-stem *ash-* with the word *ashcan* rather than the far more common *ashtray*. When subjects who have studied *ashcan* respond with *ashcan* more often then subjects who have not, we can plausibly say that the stem-completion is an implicit expression of memory for the prior study episode. This is because we can tie the subject's task performance to some event in his or her past. A similar logic underlies the inference of unconscious memories from savings or interference effects. But clinicians typically lack the means to independently corroborate what happened to their patients – to confirm the inference that their patients' behaviors are, in fact, implicit memories of past experiences. Without this independent corroboration, the inference that some symptom actually reflects an unconscious traumatic memory is just an opinion. To insist on the correctness of the inference, in the absence of any positive evidence, verges on solipsism.

Consider, for example, a patient, known as Jane Doe, who could not identify herself or give any helpful information about her identity (Lyon, 1985). During an informal test of her ability to recognize and use common objects, the psychologist Lionel Lyon noticed that she dialed the same telephone number repeatedly. When he called the number himself, the person who answered proved to be the patient's mother. Now Lyon's clinical insight was brilliant, but it might have been wrong. Lyon tested his hypothesis, and found that he was right. But suppose that the person on the other end of the line had said that she did not know the patient: Lyon would have had no justification for saying "Yes, you do so too!" But that is the risk trauma therapists take when they persuade their patients, on the basis of symptoms that *might* be implicit memories, that they were in fact traumatized. The general lack of independent corroboration is the Achilles' heel in the trauma-memory argument.

Repression and suppression

In experimental psychology, of course, repression has had a vexed existence almost from the beginning. In contrast to psychodynamically oriented clinicians, researchers who have looked for repression in the laboratory have rarely found it, and even the few ostensibly positive findings are beset by a host of methodological problems (Holmes, 1974, 1990; Mackinnon & Dukes, 1964; Zeller, 1950). In the aftermath of seven decades of negative reviews, it was perhaps no oversight that the *Handbook of Emotion and Memory* did not even include a chapter on repression, and only six entries in the index (Christianson, 1992).

There is a further question about the relationship between repression (or dissociation, for that matter) and suppression or denial. It is one thing to deny that something happened, or to deliberately avoid thinking about something that did happen, and another thing entirely to be unaware of something that happened – or, for that matter, to be unaware that one is avoiding thinking about something that happened. Erdelyi has vigorously argued that Freud used the terms *repression* and *suppression* interchangeably throughout his career, and referred to conscious and unconscious forms of repression. In Erdelyi's view, the restriction of repression to unconscious defense was a later revisionist ploy by his daughter Anna (Erdelyi, 1990; Erdelyi & Goldberg, 1979). Erdelyi's philological legwork is on point, to be sure, but – as both Sigmund and Anna Freud surely understood – the concept of repression, and the technique of psychoanalysis, only make sense if repression is deployed *un*consciously. Rapaport (1942), who should know, seems to identify repression with unconscious defense; and if Wegner (1989; Wegner et al., 1987) is to be believed, unconscious thought suppression is the only kind of thought suppression that can work anyway.

The distinction between unconscious repression and conscious suppression is important because directed forgetting, a variant on conscious thought suppression, has recently been proposed as a model of Freudian repression. In such experiments, subjects study a list of items, and then receive instructions to forget some of them, followed by memory tests. The general finding of such experiments is that instructions to forget do seem to work under some circumstances (Anderson & Green, 2001). This much has long been known (Bjork, 1972, 1978; Kihlstrom, 1983), but Anderson and his colleagues, as well as some other researchers, have repeatedly referred to their experimental results as relevant to Freud's views about the repression of trauma (Anderson & Levy, 2002; Conway, 2001; Freyd et al., 2005; Levy & Anderson, 2002). However, there are a large number of differences between Freudian repression and directed forgetting (Kihlstrom, 2002; see also Schacter, 2001). In the first place, the

effect produced on explicit memory was nothing like a full-fledged amnesia: even after 16 suppression trials, the subjects still recalled more than 70 percent of the critical material. Presumably, none of them forgot that they had studied a list of words, much less that they had just been in an experiment. Moreover, the experiment provided no evidence of a persisting influence of unconscious (implicit) memory, or of the reversal of the "amnesia" and recovery of the lost memories – points that are critical to the classical concept of repression. Most important, however, even the small effects observed were the product of *conscious* thought suppression – and conscious thought suppression cannot be a model of repression if repression must be unconscious. Nevertheless, this work has been endorsed as a model of repression by some of the same professionals who criticized the associative memory illusion as a model of false memory (Freyd & Gleaves, 1996; Freyd et al., 2005).

Sincere attempts to study repression in the laboratory have often been rejected as irrelevant by psychoanalysts themselves. Partly, this is due to a methodological problem identified early on by Sears (1936) and Rapaport (1942): repression is not about forgetting the merely unpleasant; it is about the forgetting of vital threat – threat whose intensity simply cannot be reproduced in the laboratory. Even so, to psychoanalysts, repression is obvious in their consulting rooms, and experimental evidence is not necessary. As Freud himself put it in his 1934 note to Saul Rosenzweig, an experimental psychologist quite sympathetic to psychoanalytic thinking (Mackinnon & Dukes, 1964):

> I have examined your experimental studies for the verification of the psychoanalytic assertions with interest. I cannot put much value on these confirmations because the wealth of reliable observations on which these assertions rest make them independent of experimental verification. Still, it can do no harm. (p. 703)

The new dissociation(s)

By contrast with repression, the concept of dissociation has fared somewhat better in its later history. As noted earlier, Hilgard's (1977) neo-dissociation theory of divided consciousness was instrumental in reviving interest in the phenomenon. One important aspect of Hilgard's argument was that divisions of consciousness could occur in normal mental life, without being instigated by trauma. We see dissociative divisions of consciousness all the time in hypnosis, for example (Kihlstrom, 2005). In posthypnotic amnesia, highly hypnotizable subjects cannot remember what they did or experienced while they were hypnotized – yet they show both repetition and semantic priming effects related to those experiences, revealing a dissociation between explicit and implicit

memory. Furthermore, they recover the critical memories when the amnesia suggestion is canceled by a prearranged reversibility cue. Spared priming is also observed in hypnotically suggested blindness, revealing a dissociation between explicit and implicit perception. Posthypnotic suggestion also has some of the character of implicit learning: by virtue of posthypnotic amnesia, subjects typically forget that they received the suggestion; yet they respond appropriately to the posthypnotic cue.

Of course, the very existence of the category of *dissociative disorder* in the *DSM* also indicates that the concept of dissociation has attained general acceptance (Kihlstrom, 1992b, 1994). And again, we can see dissociations in the dissociative disorders. The interpersonality amnesia observed in multiple personality disorder, for example, impairs explicit memory but spares at least some forms of implicit memory (Eich et al., 1996). Still, we have to be careful here, because the term "dissociation" can be used in two quite different senses – one descriptive, the other explanatory.

Neuropsychologists use the word in a purely descriptive manner, to refer to a situation where a single independent variable has different effects on two dependent variables. When we say that the amnesic syndrome or posthypnotic amnesia dissociate explicit and implicit memory, we are using the term "dissociation" as a kind of synonym for statistical interaction. This usage goes back at least to William James (Taylor, 1999), who coined the term to refer to a disruption in the normal association between two processes (James, 1890/1980). This is what Janet had in mind when he referred to *désagrégation*, translated as *dissociation* – a usage which entered English in the last years of the nineteenth century. And it's also what *DSM* means, at least on the surface, when it defines the dissociative disorders as entailing disruptions in the normally integrated functions of consciousness.

In either case, dissociation simply describes a situation where some percept, memory, or thought is not accessible to conscious awareness. But *dissociation* can also refer to a psychopathological process. This use of the term as an explanation, rather than a description, began appearing in the 1990s, as the dissociative disorders began to be viewed as syndromes of trauma. Thus, van der Kolk and others have argued that stress-induced increases in corticosteroids interfere with hippocampal function, and thus the storage of an explicit memory of the stressful event, but have no effect on the storage of implicit emotional associations, which is mediated by the amygdala (van der Kolk, 1994; van der Kolk & Fisler, 1993). As a result, trauma victims will respond emotionally to objects and events that somehow resemble the original trauma, without consciously remembering the trauma itself. In this usage, dissociation is an explanatory construct, not a descriptive label. Trauma causes dissociation, which renders the victim amnesic – an amnesia that in turn is characterized by dissociations between explicit and implicit memory.

TRAUMATIC MEMORY: NOT SO SPECIAL AFTER ALL?

The only problem with this assertion, which after all goes back at least as far as Janet, is that everything we know about emotion and memory tells us that emotional involvement makes events *more* memorable, not less (e.g., see Reisberg, this volume). At least, this is true in the mundane circumstances of the *affective intensity effect* familiar in laboratory studies of verbal learning (Kihlstrom et al., 2000). Of course, higher levels of stress, or different kinds of stress, might have different effects. In this way, proponents of the trauma-memory argument and recovered-memory therapy often argue that traumatic memories have special properties that render the usual rules of memory processing inapplicable. For example, it has been proposed that traumas of terror are well remembered, but traumas of betrayal are subject to dissociative amnesia because detecting such betrayals threatens the attachment-based dependency of the victim on his or her primary caregivers (Freyd, 1996). Reviewing this literature in 1997, Shobe and I questioned whether traumatic memories were special, and concluded that the major theories of traumatic memory were either incoherent or inadequately supported by empirical evidence (Shobe & Kihlstrom, 1997).

An anatomy of traumatic memory?

In apparent response to our critique, Nadel and Jacobs (1998) took another tack to support the idea that traumatic memories are special. According to them, different aspects of memories are processed by different memory modules. Furthermore, they argued that emotion, and particularly traumatic stress, affects the function of these memory modules differently. The differential effects of emotion on different memory modules provide the mechanism by which traumatized individuals can forget some aspects of their experiences but not others, resulting in the kinds of fragmentary memories described by van der Kolk and Fisler (1995) in a widely cited paper. Specifically, Nadel and Jacobs propose that traumatic stress enhances the emotional component of the traumatic memory, but impairs the contextual component – resulting in the free-floating "feeling memory" described by Fredrickson (1992): "What distinguishes these intrusive memory states is the absence of the time-and-place contextual information that typically characterizes autobiographical episode memory" (p. 156).[1]

In support of their first proposition, Nadel and Jacobs cited a number of animal studies showing that "various aspects of an episode memory are represented and stored in dispersed neocortical modules," collected by a

"hippocampal ensemble" (p. 155). As far as it goes, this view of the neural substrates of memory is unobjectionable. The distributed nature of memory processing – in which, for example, emotional valence is contributed by the amygdala, recognition mediated by the rhinal cortex, spatial context by the parahippocampal gyrus, and the whole trace bound together by the hippocampal formation – is widely accepted within cognitive neuropsychology and cognitive neuroscience.

In principle, the fact that different aspects of a memory are processed by different brain structures could provide the foundation for the fragmentary nature of emotional memory noted by van der Kolk and Fisler, among other proponents of the trauma-memory argument and recovered-memory therapy. If one module were impaired (for example, by the biological consequences of traumatic stress), the information processed by that module might well be missing from the resulting memory. However, this proposition must be considered purely speculative, because Nadel and Jacobs offer no evidence in support of the second proposition, that the functioning of these cortical subsystems, or the representational components generated by them, are differentially impaired by traumatic stress. On the contrary, there are good reasons to think that the amygdala is activated by stress, resulting in robust, lasting memory for emotional events, as demonstrated convincingly by Cahill and his colleagues, in widely known research that Nadel and Jacobs failed to cite (e.g., Cahill & McGaugh, 1996, 1998; Cahill et al., 2004). The trauma-memory argument seeks to explain why people forget trauma; it cannot be supported by evidence that the involvement of the amygdala makes events *more* memorable.

Nadel and Jacobs do cite a number of animal studies showing that increased stress impairs hippocampal functioning, and thus memory. But the stress in question is chronic stress (e.g., 21 days of restraint) and the memory task in question is unrelated to the stressful event (e.g., maze learning). It is highly plausible, as Sapolsky (1996) and others (e.g., Bremner, 2002) have suggested, that exposure to chronic stress releases neurotoxins which damage the hippocampus and consequently impair memory. But this memory impairment would be general in nature, not specific to the trauma, and would include memories unrelated to the trauma. The amnesia would be anterograde in nature, not including the initial trauma itself (the retrograde effects of hippocampal damage remain highly controversial), and it would be progressive, producing denser amnesic lacunae as the stress continued. There is no evidence from controlled research on either humans or animals that stress specifically impairs memory for the central details of the stressful event itself – which is what the claim of "repressed" or "dissociated" traumatic memories is all about.

Fragmentary memories?

Nadel and Jacobs' third proposition, that traumatic memory is fragmentary, is the outcome to be explained by the first two propositions. But if the second of these propositions is invalid, what is there to be said about the third? In fact, Nadel and Jacobs seem to base their third proposition entirely on the work of van der Kolk and Fisler:

> Within a certain range, stress could enhance all forms of explicit memory, but high levels of stress could enhance some aspects of explicit memory while impairing others. And here is the critical point: When stress is high enough to impair the function of the hippocampus, resulting memories will be different from those formed under more ordinary circumstances. These empirical data may be available as isolated fragments rather than as coherently bound episodes (e.g., van der Kolk & Fisler, 1995). This hypothesis contrasts with the position espoused by Shobe and Kihlstrom (1997), who did not take into account the differential effects of stress on the various memory modules. (p. 156)

Note, first, that the material quoted is presented only as a "hypothesis" and it is qualified with hedge words such as "could" and "may." In fact, nowhere in their paper do Nadel and Jacobs offer any evidence that stress has the predicted effect on memory; it is not clear why they should criticize us for failing to take into account evidence that did not exist at the time, and apparently still does not exist.

It is ironic that, in bolstering their "hypothesis" concerning the fragmentary nature of traumatic memories, Nadel and Jacobs rely heavily on van der Kolk and Fisler (1995), because – as Shobe and I discussed in some detail – this study is badly confounded, and no conclusions about the qualities of traumatic memory should be drawn from it. These investigators recruited subjects for their study by advertising in a local newspaper for individuals who were "haunted by memories of terrible life experiences" (p. 514), which they then compared to self-selected memories for events such as weddings and graduations. To make things worse, most of the traumatic events were reported to have occurred in childhood, while most of the nontraumatic events occurred in adulthood. As Shobe and I wrote: "the poor narrative quality of the traumatic memories, and even the periods of amnesia, may have been due to normal processes associated with infantile and childhood amnesia, rather than any special qualities of traumatic memory" (Shobe & Kihlstrom, 1997, p. 72). In fact, subsequent, more carefully constructed comparisons of traumatic and nontraumatic memories have largely failed to confirm the assertions of van der Kolk and Fisler (e.g., Berntsen, 2001; Berntsen, Willert, & Rubin, 2003;

Bohanek, Fivush, & Walker, 2005; Byrne, Hyman, & Scott, 2001; Peace & Porter, 2004; Porter & Birt, 2001). Traumatic memories differ from positive memories in some respects, but they are far from fragmentary.[2]

"Hot" and "cool" memory systems?

Nadel and Jacobs also cited a paper by Metcalfe and Jacobs (1996; see also Metcalfe & Jacobs, 1998, 2000) which proposes that there are separate "cool" and "hot" memory systems that are affected differently by stress. Metcalfe and Jacobs propose:

> As stress increases, the cool-memory system at first becomes increasingly responsive but then, as the level continues to grow, becomes less responsive until, at traumatic levels of stress, it becomes dysfunctional . . . In contrast, the hot system becomes increasingly responsive to increasing levels of stress in a monotonic manner up to and including very extreme levels, breaking down only at extremely high levels. (pp. 205–6)

Presumably, the cool memory system processes information about the context in which the trauma occurred, while the hot memory system processes information about its emotional content.

Like Nadel and Jacobs, Metcalfe and Jacobs seem to be invoking something like the Yerkes-Dodson Law, the famous inverted U-shaped function that relates arousal to performance (Yerkes & Dodson, 1908). According to the law, there is some moderate level of arousal that leads to optimum performance on any task. Extremely high levels of arousal impair performance, perhaps by activating competing responses that are ordinarily inhibited (their original idea), or perhaps by reducing the number of cues that the organism can process (Easterbrook, 1959). Either way, high levels of arousal might produce just the kinds of fragmentary memories described by van der Kolk and Fisler. But fragmentary memory is not the absence of memory. High levels of arousal might impair the victim's memory for peripheral details, but there is no reason to think they might impair memory for central details, like the event itself.

Still, even the assertion of a new Yerkes-Dodson Law takes the form of a hypothesis. Metcalfe and Jacobs (1998) review available evidence that low levels of stress can enhance processing of both the "cool" (hippocampus-based) and "hot" (amygdala-based) memory systems, as well as available evidence that moderate levels of stress can enhance "hot" memory while impairing "cool" memory to some degree. However, they offer no evidence for the critical prediction that traumatic stress can produce profound amnesia for the spatiotemporal components of memory processed by the "cool" system. Aside

from two anecdotal case reports of unknown representativeness, Metcalfe and Jacobs, like Nadel and Jacobs, rely entirely on the study by van der Kolk and Fisler (1995) – the same study that Shobe and I had already criticized in considerable detail, and that subsequent research has undermined.

It is important to understand precisely what is going on here. Nadel and Jacobs offer no empirical support for their prediction that individuals subjected to traumatic levels of stress can retain strong memories for their emotional state while forgetting the spatiotemporal context in which this emotion was aroused. Although they refer to papers by Jacobs et al. (1996) and Metcalfe and Jacobs (1998) as if they presented such evidence, these papers both rely solely on the highly questionable study by van der Kolk and Fisler (1995). As such, their hypothesis remains just that – a hypothesis that has no grounding in actual empirical data. A hypothesis (Nadel & Jacobs, 1998) cannot be supported merely by repetitions of the same hypothesis in other publications (Jacobs et al., 1996; Metcalfe & Jacobs, 1998).

Nevertheless, six years later Jacobs, Nadel, and their colleagues repeated essentially the same argument (Payne et al., 2004). Once again, they conflated memory and cognitive difficulties that might be caused by the neurotoxic effects of chronic stress with traumatic amnesia mediated by repression or dissociation. Once again, they relied on the van der Kolk and Fisler (1995) study for evidence that "traumatic events are remembered initially as disconnected images and waves of disjointed sensations" (p. 97) – plus a description of a "hypothetical traumatic war experience" (p. 95). Once again, they proposed that "If high levels of cortisol disrupt normal neuronal function [of the hippocampus] . . . then coherent memories of trauma will be rendered inaccessible" (p. 97). This time, at least, the proposal by Metcalfe and Jacobs of two memory systems, one hot and one cool, is clearly labeled "speculative" (p. 98), and the notion of traumatic amnesia is acknowledged to be "controversial" (p. 113). Nevertheless, these authors conclude that "Intense stress is associated with memory disruption, either partial or complete, relating to the trauma itself or to episodic memory in general" (p. 102), and that "Trauma appears to disrupt memory for the context and the details of experienced events" (p. 111).

Recovery and reconstruction

Despite the lack of pertinent evidence, Nadel and Jacobs (1998) concluded that "Traumatic stress can cause amnesia for the autobiographical context of stressful events, but stronger than normal recall for the emotional memories produced by them" (p. 156). But they went even further, concluding that despite the allegedly fragmentary nature of traumatic memories, "an autobiographical memory eventually emerges" by a process of "inferential narrative smoothing

whereby disembodied fragments are knit together into a plausible autobiographical episode . . . The present analysis suggests that at least some memories 'recovered' during therapy should be taken seriously" (p. 156). The evidence provided that memory fragments can be knitted into autobiographical narratives was, once again, the study by van der Kolk and Fisler (1995). Unfortunately, as noted by Shobe and myself, van der Kolk and Fisler did not attempt to corroborate their subjects' narratives against independent records of the traumatic events in question. Accordingly, we have no idea how much of their subjects' narratives were accurate accounts of the traumatic events in question – or, frankly, even that the events occurred at all.

Nadel and Jacobs (1998) concede as much: "The narratives associated with [recovered traumatic] memories are less likely to be veridical in their entirety" (p. 156). Given that, according to their own hypothesis, the contextual information underlying these narratives was not properly encoded at the time that the events ostensibly occurred, this would seem to be an understatement. If, as they propose, extremely high levels of arousal impaired the processing of central as well as peripheral details, and included the emotional response to the event as well as the contextual features of the event, such a massive encoding failure would render attempts to recover traumatic memories pointless: there would be no traumatic memory available to recover.

It is a cardinal principle of memory that encoding constrains retrieval (Morris, Bransford, & Franks, 1977; Tulving & Thomson, 1973). To put it bluntly, if something is not encoded in memory at the outset, it cannot be retrieved later (see also Uttl & Graf, this volume). If the Yerkes-Dodson law impairs encoding to such a degree as to render someone amnesic for even the central details of a traumatic event, then these details are simply not available in memory for later retrieval. To speak of such memories as "recovered" is, therefore, to make a category mistake – which Ryle (1949, p. 17) referred to as "one big mistake and a mistake of a special kind." If some sort of Yerkes-Dodson process impairs the encoding of the various features of a traumatic event, then the prospects of recovering such an event are nil. Any such "recovery" would be a reconstruction, based more on imagination than on the retrieval of trace information. No matter how "plausible" such memories may be, they should not be "taken seriously" (Nadel & Jacobs, 1998, p. 156) unless and until they have been subjected to independent corroboration.

DOES TRAUMA CAUSE AMNESIA AFTER ALL?

Much of the literature on the effects of very high levels of stress cited by Nadel and Jacobs (1998) and other theorists sympathetic to the trauma-memory

argument is animal research, and studies of animal memory are often open to interpretation as pertaining to *implicit*, or at least nonverbal, memories. Under this gambit the very literature that McGaugh (2004) cites as evidence that stress improves memory can be cited in favor of the trauma-memory argument, on the assumption that high levels of stress impair the encoding of explicit, consciously accessible memories of the sort that might be expressed in free recall, but enhance the encoding of implicit, unconscious ones that might be expressed in priming and other aspects of behavior. Of course, such a reconstrual risks confusing unconscious implicit memories with conscious explicit memories that are simply nonverbal in nature. Setting this nontrivial problem aside, in the final analysis, it all comes down to a simple question: Is there any evidence that psychological trauma actually causes functional or psychogenic amnesia – that is, a profound loss of explicit memory for traumatic and peritraumatic events?

Although traumatic amnesia has been part of the folklore of psychiatry and clinical psychology since the nineteenth century (Janet and Freud both made it a central part of their theories of neurosis and psychotherapy), the best that can be said is that after more than 100 years even the best evidence favoring the existence of traumatic amnesia is highly debatable (Crews, 1995, 2004). Even amnesia in war neurosis – perhaps the prototype of stress-induced functional or psychogenic amnesia (Arrigo & Pezdek, 1997; Brown, Scheflin, & Hammond, 1998) – rests on an unsecured empirical base. As Pendergrast (1998) has pointed out, the classic monographs of Kardiner and Spiegel (1941/1947) and Grinker and Spiegel (1943/1945) each include only a single detailed case report of traumatic amnesia (for detailed critiques of the war neurosis literature, see also Giglio, 1998; Lilienfeld, 1998; Piper, 1998). Amnesia may occasionally occur in the context of war neurosis, but apparently it does not happen often enough to permit clinicians who were actively looking for it to report a series of cases. Moreover, as Pendergrast also pointed out, the general failure of clinicians to independently corroborate memories of war-related trauma recovered through such means as hypnosis and the amytal interview (e.g., Fisher, 1945) raises the possibility that many, if not most, of these memories are confabulations.

Disaster and terror

In view of the problem of obtaining independent corroboration of retrospective reports of trauma, it would seem that the best evidence for trauma-induced amnesia would be provided by prospective studies of actual trauma survivors. In a comprehensive review of the available literature, Pope and his colleagues surveyed 63 studies of more than 10,000 trauma survivors, everything from the Holocaust and war to accidents and natural disasters, and concluded that they recorded "not a single instance" of a psychogenic amnesia caused by

processes such as repression or dissociation (Pope et al., 1998, p. 213). Most victims apparently remembered their experiences all too well. Those who did not could be accounted for by organic amnesia, or by normal memory processes such as infantile and childhood amnesia or time-dependent forgetting (Pope, Oliva, & Hudson, 2000).

By contrast, Brown and his colleagues reviewed much of the same literature in a massive tome of almost 800 pages, leading toward quite the opposite conclusion (Brown, Scheflin, & Hammond, 1998). For example, they concluded that "A significant subpopulation of traumatized individuals retain no or little narrative memory for the trauma" (p. 200). Although the exigencies of publication prevented Brown and his colleagues from confronting Pope's analysis directly, they did so in a further paper, more than 150 pages long, just the next year (Brown, Scheflin, & Whitfield, 1999). After rejecting 42 of the 63 studies as "irrelevant" to the question of amnesia, Brown et al. concluded that "all 21 [of the remaining studies] show that trauma significantly affected memory – and 18 demonstrate amnesia either for the traumatic event or for injuries related to the trauma" (p. 29). As it happens, the 21 allegedly positive studies were a mixed bag, including evidence for general memory disturbance of the sort often seen in PTSD, or for "cognitive avoidance" of the trauma, rather than amnesia per se. But Brown et al. did assert that "Nine studies actually present *data in favor of the existence of traumatic amnesia* [emphasis added]" (p. 28).

In response, Piper and his colleagues re-analyzed those nine studies and reaffirmed the conclusions of Pope et al. (see also McNally, 2003; Piper, Pope, & Borowiecki, 2000). For example, two individuals who were amnesic for a lightning strike were "side-flash" victims who probably received the equivalent of electroconvulsive shock. Some of the children who forgot a flood disaster were as young as two years old at the time of the incident. And while approximately one-third of older children who were earthquake survivors were reported as showing psychogenic amnesia for the event, more than two-thirds of a control group of children who were not directly exposed to the trauma met the same criterion. One study did report a high rate of dissociative symptoms, as measured by the DES, among those who experienced the Loma Prieta earthquake of 1989, but these were most likely common experiences of depersonalization and derealization; there was no evidence that any subject forgot the earthquake.

Incest and child sexual abuse

In the face of such evidence, the trauma-memory argument is sometime revised to take special note of trauma associated with incest and other childhood sexual abuse (Brown, Scheflin, & Hammond, 1998; Scheflin & Brown, 1996). For example, Freyd (1996) has proposed that traumas of terror, such as natural

disasters, are remembered well, while traumas of betrayal, such as incestuous sexual abuse, are dissociated from conscious recollection. Brown and his colleagues reviewed the literature on "naturally occurring dissociative or traumatic amnesia for childhood sexual abuse," and concluded that "Not a single one of the 68 data-based studies failed to find it" (Brown, Scheflin, & Whitfield, 1999, p. 127; see also p. 67).

Unfortunately, these studies suffer from a host of methodological problems, including, in many cases, an unhealthy reliance on self-reports – both that the trauma in question actually occurred, and that it was actually forgotten (Kihlstrom, 1996, 1998; Loftus, Garry, & Feldman, 1994; Pendergrast, 1996; Pope & Hudson, 1995a). Even where there is independent verification of the abuse, there is often a failure to distinguish between amnesia and the normal forgetting that occurs with the passage of time – not to mention infantile and childhood amnesia. Typically, there is also a failure to distinguish between actual forgetting and failures of self-disclosure. For example, Brown, Scheflin, and Hammond (1998) cited a massive study by Widom and Morris (1997) of "full amnesia" in 37 percent of 1,114 adult survivors of "court-substantiated" childhood sexual abuse (p. 196) – despite the fact that Widom and Morris themselves specifically disavowed such an interpretation, and attributed their reporting failures as lack of disclosure rather than amnesia. In view of these problems, perhaps a more accurate statement would be that *not a single one of the studies in question convincingly showed it.*

When researchers take account of these sorts of factors, the incidence of "amnesia" for child abuse goes way down. In a study of victims of documented child sexual abuse (CSA), Goodman and her colleagues reported that only 15.5 percent of abuse victims failed to report the target incident during a telephone interview conducted an average of 13 years after the events in question (Goodman et al., 2003). Nondisclosure dropped to 8.3 percent after follow-up by a mailed questionnaire and a telephone interview. Both rates of reporting failure are markedly lower than that yielded by earlier, less rigorous studies (e.g., Williams, 1994).

Although an 8 percent disclosure failure might be interpreted in terms of traumatic amnesia, it should be understood that this figure represents an upper limit, because the failure to report may be for reasons other than repression or dissociation. Goodman's detailed analysis supports more prosaic interpretations in terms of infantile and childhood amnesia, depth of processing, time-dependent forgetting, and a simple unwillingness to disclose personal tragedy to a stranger. Accordingly, Goodman et al. concluded:

> These findings do not support the existence of special memory mechanisms unique to traumatic events, but instead imply that normal cognitive operations underlie long-term memory for CSA. (p. 117)

Goodman (this volume) has provided a further analysis of these data that provides more evidence against the trauma-memory argument. When directly questioned, 21 of the 142 respondents who completed the telephone interview reported that there had been some period of time when they had forgotten about the abuse. Of these, most engaged in conscious suppression: only 5 of 138 subjects who answered the question indicated that they would not have remembered the target incident if they had been asked directly about it, and a sixth was not sure – yielding a reduced estimate of 3.6 percent for the incidence of traumatic amnesia.

Although this estimate is an order of magnitude below Williams' figure of 38 percent, it should be underscored that even this vastly reduced estimate is an upper bound. Goodman and her colleagues have reported that at least two of these subjects were very young at the time the target incident occurred – again raising the likelihood of infantile and childhood amnesia. Another victim was asleep at the time of the incident, and thus was not even aware of it at the time it occurred. That leaves only two individuals, at most 1.5 percent of the sample, who might conceivably have suffered from traumatic amnesia. Similarly, Porter and Birt (2001) reported that 14 of 306 subjects (4.6 percent) reported an extended time period during which they did not recall their traumatic experience. Excluding cases of normal forgetting or deliberate non-recall, only three instances – slightly less than 1 percent – remained that could be attributed to unconscious repression (interestingly, 2.6 percent of subjects also reported forgetting a positive experience for an extended period of time).

Any of these figures – 3.5 percent, 1.5 percent, less than 1 percent – is far below the figures given, suggested, or implied by proponents of the trauma-memory argument. For all we know, this level of co-occurrence of trauma and amnesia might be nothing more than sheer coincidence. In any event, the estimate is so low as to refute the claim, or implication, that trauma causes amnesia with anything like the regularity claimed by advocates of the trauma-memory argument and recovered-memory therapy.

What about recovered memories?

Sometimes, recovered memories of abuse and other trauma are offered as evidence of repression or dissociation (Brown, Scheflin, & Hammond, 1998; Gleaves et al., 2004; Kihlstrom, 2004; Sivers, Schooler, & Freyd, 2002). It is a telling point that, more than a century after the emergence of the trauma-memory argument, the most convincing evidence that its promulgators can offer are individual cases of doubtful generality. And even these are precious few in number, far between – and often of doubtful validity. For example, Karon and Widener (1997, 1998) offered, as an example of repression in battlefield

neurosis, the case of a World War II veteran who had apparently repressed an airplane crash in which he rescued the pilot and earned a medal for his bravery. But it is not at all clear whether the episode actually represented the lifting of amnesia, as opposed to the patient's more causal report that he had once received a medal (for detailed critiques, see Lilienfeld, 1998; Pendergrast, 1998; Piper, 1998). Although there are serious questions about whether this recovered memory is accurate (Giglio, 1998; for a reply, see Karon & Widener, 1998), the fact remains that Karon and Widener did not even corroborate the one absolutely confirmable detail – that the patient in question did, in fact, receive a medal for bravery.

Other reported cases of recovered memory suffer from similar problems. For example, Cheit (1998) claimed to have validated 35 cases of recovered memory of child sexual abuse, only to have the claim systematically dismantled by Piper (for a reply, see Cheit, 1999; Piper, 1999). Corwin and Olafson (1997) presented the case of Jane Doe, whose recovery of a memory of childhood sexual abuse was actually captured on videotape. Although this case study was compelling to many (if not all) researchers who were asked to comment on it, closer scrutiny raises the question of whether the subject in question recovered a memory of *abuse*, or only a memory of her previous *testimony about* abuse. The difference is critical – especially since the victim in question was the object of a vigorous custody dispute between divorcing parents, and there are serious questions about whether any abuse actually took place at all (Loftus & Guyer, 2002a, 2002b).

With respect to trauma and memory, the argument seems to be that because repression and dissociation can be reversed *in theory*, the recovery of memories shows that they were repressed or dissociated in the first place. At first blush, this would seem to be a textbook example of the logical fallacy of *affirming the consequent*: if repressed memories can be recovered, then recovered memories were repressed. But the recovery of a traumatic memory, even one that is independently corroborated (Schooler, 2001), does not by itself imply that the event was forgotten due to repression or dissociation. Researchers must be careful to distinguish between recoveries mediated by the lifting of repression or breaching of dissociation from other causes of remembering, including the normal effects of shifting retrieval cues, reminiscence effects, and hypermnesia. The recovery of a forgotten trauma may be no different in kind than the recovery of one's memory for where one put the car keys, or the name of one's third-grade teacher. Remembering a forgotten event may be traumatic, even if the trauma did not cause the forgetting.

Of course, self-reported amnesia requires confirmation, just as self-reported trauma does. Two of the seven instances of "discovered" memories corroborated by Schooler (2001) are problematic in this respect: both TW and MB (also referred to as "WB") discussed the events with their spouses during the

time they supposedly had forgotten them. It is also important to distinguish between the recovery of a forgotten memory of trauma and a reinterpretation of an event that had always been remembered. As Schooler notes, "Rather than discovering the existence of the memory itself, these individuals may be discovering the emotionally disturbing understanding of the experience. Nevertheless, because of the profound sense of discovery, individuals may conclude that they must have just remembered a long inaccessible memory" (p. 113). In any event, it should surprise no one if traumatic events are occasionally forgotten, and if recovered memories of trauma are occasionally corroborated. Neither empirical fact demands interpretation in terms of trauma-induced repression or dissociation.

What about dissociative amnesia?

Sometimes, proponents of the trauma-memory argument point to the existence of the dissociative disorders as evidence that trauma can be repressed, or dissociated, and thus lost to conscious recollection. As was the case with recovered memories, the argument verges on the circular: dissociative disorders exist, dissociative disorders are instigated by trauma, therefore trauma causes dissociative amnesia. Moreover, the argument seems to confuse the two meanings of the term *dissociation* discussed earlier: the dissociative disorders are "dissociative" in the descriptive sense of entailing a loss of the integrative functions of consciousness, and not because they are *caused* by stress-induced "dissociation." It is in this descriptive sense that the conversion disorders can also be described as dissociative in nature, although the dissociations in question affect sensory-perceptual and motor functioning, rather than memory (Kihlstrom, 1992b, 1994).

In fact, dissociative disorder is a valid diagnostic category (Kihlstrom, 2005) and functional amnesia a genuine phenomenon (Kihlstrom et al., 2000; see also Kopelman, 2002; Markowitsch, 2000) – although genuine cases appear to be vanishingly rare. Thigpen and Cleckley (1954, 1957), who treated and reported the famous *Three Faces of Eve*, never saw another valid case despite repeated referrals over the next 30 years – even at the height of the multiple personality "epidemic" (Thigpen & Cleckley, 1984). Given the apparent incidence of child sexual abuse, if the trauma-memory argument – and particularly arguments about betrayal trauma (Freyd & Gleaves, 1996) – were correct, we should see more cases of dissociative amnesia than we do.

Although there are undoubtedly some cases of dissociative disorder which present histories of trauma, including incestuous child abuse, apparently this is not always, or even often, the case. For example, the eponymous Eve denied ever having been abused, and complained that people who attended

her speaking engagements insisted that she was in denial about having been abused (Ganaway, 1995). Even when a patient with dissociative disorder presents a documented history of child sexual abuse, inferring a causal link between abuse and dissociation is fraught with difficulty (Pope & Hudson, 1995b; Rind, 2003; Sbraga & O'Donohue, 2003). For example, the most widely cited evidence for an association between child sexual abuse and dissociative amnesia is retrospective in nature, and retrospective studies by their very nature necessarily overestimate the relation between antecedent and consequent variables (Dawes, 1993; Swets, Dawes, & Monahan, 2000). Moreover, such studies often rely on excessively liberal definitions of abuse and trauma to begin with.

In point of fact, there is actually no good evidence that trauma plays a particular role in the etiology of these disorders – that is, evidence based on random or prospective samples (Kihlstrom et al., 2000). In such studies, the fact is that examples of multiple personality disorder and other dissociative disorders simply do not figure prominently among the sequelae of documented child sexual abuse (see also Bailey & Shriver, 1999; Beitchman et al., 1991, 1992; Kendall-Tackett & Marshall, 1998; Kendall-Tackett, Williams, & Finkelhor, 1993; Lange et al., 1999; Rind, 1998). The dissociative disorders cannot be cited as evidence that trauma impairs memory unless and until there is convincing evidence that such traumas as child sexual abuse actually cause dissociative disorder. Occasional cases of dissociative disorder who also have a history of trauma have helped keep the trauma-memory argument alive. But even then they are the exceptions that test, one might even go so far as to say *prove*, the rule that emotion – even negative emotion, even at extremely high levels – generally enhances memory, such that trauma is typically remembered particularly well by those who have actually experienced it.

"Organic" and "functional," "somatogenic" and "psychogenic"

A word is in order about the distinction between "organic" and "functional" (or "somatogenic" and "psychogenic") amnesia and other mental disorders. In medicine, the term "functional" often means that the biological basis of a disorder is not yet known, and there is no necessary implication that the disorder has no organic basis. For example, general paresis, a form of dementia, was considered functional until the discovery of the syphilis spirochete, at which point the illness was reclassified from functional to organic. On the other hand, the term "psychogenic" implies that the underlying etiology is psychological rather than biological in nature. Some forms of depression may reflect anomalies of neurotransmitter function, and thus be properly classified as somatogenic in nature. On the other hand, other forms of depression may be

considered psychogenic because they are caused by certain beliefs held by the patient – the "depressogenic" schemata of Beck (1967), perhaps, or the pessimistic attributional style that lies at the core of the reformulated hopelessness theory (Abramson, Metalsky, & Alloy, 1989).

The memory disorder envisioned by the trauma-memory argument is, in theory, both functional and psychogenic. Traumatic amnesia is "functional" in that it "is attributable to an instigating event or process that does not result in damage to brain structures, and produces more forgetting than would normally occur in the absence of the instigating event or process" – in this case, trauma (Kihlstrom & Schacter, 2000, p. 409). And it is "psychogenic" in that it is caused by a mental state, namely mental trauma, or mental processes of repression or dissociation.

The fact that dissociative amnesia might have biological correlates (Markowitsch, 1999) does not mean it is really "organic" in nature after all: it is axiomatic in contemporary psychology and cognitive science that all mental states have their neural correlates, and this is no less true for dissociative amnesia than it is for conscious recollection. What makes dissociative amnesia functional, at least in principle, is the absence of demonstrable brain insult, injury, or disease as an instigating factor. There are many other disorders of memory that are functional in just the same way (Kihlstrom & Evans, 1979a). These include posthypnotic amnesia, which is also psychogenic because it only occurs in response to suggestion (Kihlstrom, 1979, 1985, 2005; Kihlstrom & Evans, 1979b). But the category also includes infantile and childhood amnesia, which is not psychogenic.

By contrast, the memory dysfunction commonly associated with PTSD is "organic" because it is (ostensibly) caused by a stress-induced "glucocorticoid cascade" (Bremner, 2002; O'Brien, 1997) that damages the hippocampus; but it is also "psychogenic" because stress is defined psychologically by the experience of unpredictable and/or uncontrollable events (Mineka & Kihlstrom, 1978). To be clear: a functional amnesia is one that is not associated with brain insult, injury, or disease; a psychogenic amnesia has a mental, as opposed to an organic cause. The two categories overlap, but they are different.

A WILL-O'-THE-WISP, A MYTH, AN URBAN LEGEND

The trauma-memory argument and recovered-memory therapy have been with us for more than 100 years, and have embedded themselves deeply into both our professional practices and our wider culture. But there was never any good evidence for either, and there still is none. Maybe such evidence will be forthcoming in the future. There are occasional cases in which traumatic stress and

amnesia *seem* to go together, inviting interpretations in terms of repression or dissociation. But nothing in the case literature justifies assertions that trauma impairs memory as a matter of course, or in the majority of cases, or often – even *sometimes* seems too extreme. In view of the paucity of evidence that trauma causes amnesia, discussion of implicit memories of trauma seems pointless. Laboratory analogs of traumatic amnesia are models in search of a phenomenon; theories of traumatic amnesia are explanations in search of facts.

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NOTES

1 In a sense, Nadel and Jacobs revived an even earlier concept of dissociation, one that arose in nineteenth-century chemistry, referring to the separation by heat of compounds into their constituent elements. Traumatic stress, in this view, impairs some aspects of memory, while simultaneously enhancing others: applying the psychological heat of mental stress, the various elements of an integrated memory become separated from each other.

2 Nadel and Jacobs attempt to bolster their position further by citing an earlier paper as supporting the notion that "even in the presence of extensive autobiographical amnesia, intrusive emotions or images associated with the trauma (and related events) may appear" (Jacobs et al., 1996, p. 156; see also Thomas et al., 1995). Unfortunately, the paper cited presents no empirical data to support this or any other notion about traumatic memory. Instead, these authors offer yet another hypothesis about "the conditions under which a memory for a traumatic event has a high, medium, or low probability of accurately reflecting the target event," as well as a series of fictional (*sic*) cases illustrating the basic points of the proposed model. This hypothesis was not tested in the Jacobs et al. (1996) paper, and as such remains highly speculative.

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