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A Dual Representation Theory of Posttraumatic Stress Disorder

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A cognitive theory of posttraumatic stress disorder (PTSD) is proposed that assumes traumas experienced after early childhood give rise to 2 sorts of memory, 1 verbally accessible and 1 automatically accessible through appropriate situational cues. These different types of memory are used to explain the complex phenomenology of PTSD, including the experiences of reliving the traumatic event and of emotionally processing the trauma. The theory considers 3 possible outcomes of the emotional processing of trauma, successful completion, chronic processing, and premature inhibition of processing. We discuss the implications of the theory for research design, clinical practice, and resolving contradictions in the empirical data.

Posttraumatic stress disorder (PTSD) has recently become the focus for a great deal of empirical and theoretical work. The fact that it is one of the few psychiatric disorders to be associated with a relatively specific etiological agent in the form of a traumatic event, along with its distinctive clinical feature of repeated intrusive memories, has proven a magnet for researchers. Already a number of valuable psychological theories of the disorder have been proposed, many couched in terms of an information-processing analysis (Chemtob, Roitblat, Hamada, Carlson, & Twentyman, 1988; Creamer, Burgess, & Pattison, 1992; Foa, Steketee, & Rothbaum, 1989; Foa, Zinbarg, & Rothbaum, 1992; Litz & Keane, 1989). In this article we attempt to establish explicit criteria for a theory, review existing formulations, and propose a new cognitive model of PTSD.

We believe an adequate theory of PTSD should address a number of issues. First, the theory should account for the clinical characteristics of the disorder, including the range of associated symptoms and the time course of the disorder. Second, it should indicate whether the symptoms of PTSD are themselves automatically indicative of an abnormal process whenever they occur. If they are not, the theory should explain how normal and abnormal processing differ within a comprehensive model. Third, the theory should outline the conditions that are associated with the severity and outcome of PTSD and should explain these relations. Fourth, the theory should discriminate PTSD from other related disorders and account for any evidence of comorbidity. Fifth, it should explain the experimental data on information processing in PTSD. Finally, ideally, the theory should do a better job of accounting for the available data than existing theories and make novel predictions that can be tested empirically.

We will briefly summarize the state of knowledge in the first five areas described above. Then we will describe existing theories and how they address the available knowledge. Finally, we present our own theory, indicate in what respects we believe it goes beyond existing theories, and describe a number of novel predictions that it generates.

Existing Knowledge About PTSD

Clinical Characteristics

The major clinical features of PTSD are described in the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*; American Psychiatric Association, 1994) and include the reexperiencing symptoms, such as intrusive memories and nightmares; the protective reactions, such as emotional numbing, amnesia, and cognitive avoidance; and the arousal symptoms, such as the startle response and hypervigilance. In addition, PTSD is commonly accompanied by a wide range of negative emotions, such as sadness and anger, and by negative cognitions, such as guilt.

Perhaps the hallmark characteristic of PTSD is the alternation between reexperiencing and avoiding trauma-related memories. The memories that are particularly associated with PTSD appear rapidly and spontaneously, often intruding into consciousness with high frequency. In many cases the intrusive memories consist of images accompanied by high levels of physiological arousal and are experienced as reenactments of the original trauma ("flashbacks"). Other memories, particularly those associated with traumas in the distant past, may be more fragmentary, consisting of isolated visual, auditory, olfactory,

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or tactile sensations. They may have many of the qualities of flashbacks, without necessarily corresponding to an easily recognizable event.

Flashbacks can be distinguished from memories of the trauma retrievable through a normal search of long-term memory. Whereas a great deal of deliberate and spontaneous cognitive activity typically involves individuals recalling aspects of trauma and the accompanying emotions they remember having experienced at the time, these deliberately retrievable memories are qualitatively different from the spontaneous reenactments, in which emotions often appear to be resynthesized in their original intensity (Janet, 1889; Terr, 1991; Van der Kolk & Fisler, 1995). Herman (1992b) has referred to these as *frozen memories*, a term that captures their repetitive, unchanging quality. It is also evident that, over time, trauma survivors are able to deliberately recall and describe some aspects of their experiences in a dispassionate way, without provoking full-scale flashbacks, whereas flashbacks may still be triggered by recalling certain details of the trauma or by unexpected reminders of the trauma.

Green (1994) concluded from an extensive review of the literature that rates of PTSD, given exposure to a traumatic event, average around 25 to 30% in the general population, although certain stressors, such as rape, are routinely associated with much higher rates. Prevalence rates tend to decrease with time elapsed, although up to one half of the individuals diagnosed as having PTSD may continue to have it for many years.

Longer term studies have shown PTSD lasting up to 40 years posttrauma in World War II combat veterans and POWs (e.g., Davidson, Kudler, Saunders, & Smith, 1990; Hierholzer, Munson, Peabody, & Rosenberg, 1992) and Jewish survivors of the Holocaust (Kuch & Cox, 1992). However, although epidemiological data suggest that PTSD can be prevalent many years subsequent to an event, survivors often report being only intermittently troubled by their symptoms (Zeiss & Dickman, 1989). In addition to this varied course, initial onset may occasionally be delayed by many years (Blank, 1993; McFarlane, 1988).

Normal or Abnormal Process?

If the symptoms of PTSD do not occur except in the context of a specific diagnosable condition, then it is appropriate for a theory of PTSD to focus on explaining this distinctive pathology. If PTSD symptoms are found more generally, albeit perhaps at a lesser intensity, a more inclusive theory is required that distinguishes between normal and abnormal responses to extreme stress. To date, with few exceptions (e.g., Horowitz, 1986), theories of PTSD have not adopted this broader focus. This may be connected with the claims made in the *Diagnostic and Statistical Manual of Mental Disorders*, third edition, revised (*DSM–III–R;* American Psychiatric Association, 1987) that the hallmark symptoms of PTSD, such as intrusive reexperiencing, are associated with events outside the range of human experience and are not found in other stress-related conditions, such as adjustment disorder. Theories of the stress response that do span normal and abnormal aspects have, however, been developed, particularly applied to bereavement and victimization, and we will review them briefly.

Among influential theories of the bereavement process are those of Bowlby (1980), Parkes (1971), and Horowitz (1990). In Bowlby's attachment theory, the typical response to loss of an attachment figure is a phase of protest and anger, which, if it does not succeed in restoring the loss, is followed by a phase of despair. During this period there is an oscillation between conscious preoccupation with the loss and defensive exclusion of related thoughts and reminders. Bowlby described phases of grief involving numbness, pining, disorganization–despair, and reorganization.

This theory was elaborated by Parkes (1971, 1986), who formulated loss in terms of *psychosocial transitions*. Psychosocial transitions are major life changes that involve disruption to basic assumptions about one's nature, needs, goals, and sources of support. Bereavement, Parkes suggested, consists of the difficult and painful process of abandoning long-held assumptions and adopting new and more appropriate ones. Similarly, Horowitz (1990) formulated bereavement in terms of changes to *person schemas*, cognitive structures summarizing information about oneself, close others, and relationships. In addition to the repeated descriptions of anger, denial, and numbness, bereavement researchers have consistently found anxiety symptoms indicative of heightened arousal, such as muscle tension, panic, and insomnia (Maddison & Viola, 1968; Parkes, 1970; Worden, 1982). Preoccupation accompanied by intrusive images of a bereaved spouse are also extremely common (Horowitz, 1990; Rees, 1975).

General theories of response to problematic experiences or severe life events (e.g., Janoff-Bulman, 1992; Klinger, 1975; Lazarus & Launier, 1978; Rachman, 1980; Shontz, 1965; Stiles et al., 1990) have many similarities to the theories of bereavement outlined above. Most of these theories explicitly recognize that following severe events there can frequently be observed intrusive cognitions or intrapsychic defences such as denial and avoidance that help to mitigate the intensity of distress. It has been argued that a similar syndrome may result from exposure to repeated minor stressors (Prolonged Duration Stress Disorder or PDSD; Scott & Stradling, 1994), and recent research has shown that intrusive memories are a relatively common everyday experience (Brewin, Christodoulides, & Hutchinson, 1996). This considerable degree of overlap indicates that PTSD can be partly understood in terms of general theories of adjustment. However, these theories do not describe the characteristic repetitive flashbacks involving startle responses and high degrees of associated physiological arousal and, thus, do not provide a full account of PTSD. For example, the intrusive memories associated with bereavement often involve positive recollections of the lost person.

Variables Associated With Severity and Outcome

Although there is evidence that bereavement is especially associated with more severe and chronic disturbance (e.g., Green, Grace, Lindy, Tichener, & Lindy, 1983; Joseph, Yule, Williams, & Hodgkinson, 1994; Shore, Tatum, & Vollmer, 1986), bereavement does not fully account for the individual differences in reactions. Exposure variables, such as personal injury and life-threat, have also been found to influence the course of symptomatology in combat veterans (e.g., Fontana, Rosenheck, & Brett, 1992; Foy, Resnick, Sipprelle, & Carroll, 1987; Kulka

et al., 1990; Ørner, Lynch, & Seed, 1993; Yehuda, Southwick, & Giller, 1992) as well as in civilian survivors of a variety of events (e.g., Cluss, Boughton, Frank, Stewart, & West, 1983; Ellis, Atkeson, & Calhoun, 1981; Gleser, Green, & Winget, 1981; Green et al., 1983; Joseph, Yule, Williams, & Hodgkinson, 1994; Maida, Gordon, Steinberg, & Gordon, 1989; Resnick, Kilpatrick, Best, & Kramer, 1992; Shore et al., 1986; Smith, Robins, Pryzbeck, Goldring, & Solomon, 1986). However, much of this work has relied on the subjective assessment of exposure variables, and other work has attempted to demonstrate an objective exposure–response relationship. For example, Bromet, Parkinson, Schulberg, Dunn, and Gondek (1982) showed that mothers living within 5 miles of the Three Mile Island plant had poorer psychological health than those outside this radius at 3 months after the leak. Pynoos and Nader (1988) also demonstrated that following a fatal sniper shooting in a California school, children trapped in the playground had the strongest posttraumatic reactions; those who did not attend school that day showed the least reaction.

A number of studies have shown a history of psychological or behavioral problems to be predictive of PTSD symptoms in adults after a natural disaster (McFarlane, 1989); personal injury or violent attack (Breslau, Davis, Andreski, & Peterson, 1991; Helzer, Robins, & McEvoy, 1987); and, in women, following rape (Atkeson, Calhoun, Resick, & Ellis, 1982; Burgess & Holmstrom, 1978; Frank & Anderson, 1987; Frank, Turner, Stewart, Jacob, & West, 1981; Ruch, Chandler, & Harter, 1980). Other researchers have not found prior clinical history to be associated with outcome (Kilpatrick, Veronen, & Best, 1985; Madakasira & O'Brien, 1987; Solkoff, Gray, & Keill, 1986; Speed, Engdahl, Schwartz, & Eberly, 1989). On the other hand, the important role of prior experience of traumatic events in general has been confirmed in numerous studies (Breslau et al., 1991; Burgess & Holmstrom, 1978; Kilpatrick et al., 1985; Roth, Wayland, & Woolsey, 1990; Ruch & Leon, 1983). Thus, prior trauma may account for the association between previous clinical history and the severity of PTSD.

Postdisaster life events have been associated with poorer outcome following disaster (Joseph, Yule, Williams, & Hodgkinson, 1994; McFarlane, 1988) and rape (Ruch et al., 1980). Higher levels of social support have been found to be adaptive after rape (Burgess & Holmstrom, 1974; Kilpatrick et al., 1985), disaster (Bartone, Ursano, Wright, & Ingraham, 1989; Cook & Bickman, 1990; Green, Grace, & Gleser, 1985; Joseph, Andrews, Williams, & Yule, 1992; Joseph, Yule, Williams, & Andrews, 1993; Madakasira & O'Brien, 1987), toxic exposure (Bromet et al., 1982; Fleming, Baum, Gisriel, & Gatchel, 1982), and combat (Foy et al., 1987; Foy, Sipprelle, Rueger, & Carroll, 1984; Frye & Stockton, 1982; Keane, Scott, Chavoya, Lamparski, & Fairbank, 1985).

Work with combat veterans has shown an association between better outcome and a more internal locus of control (Frye & Stockton, 1982; Solomon, Mikulincer, & Benbenishty, 1989) and a more internal and controllable attributional style for positive events (McCormick, Taber, & Kruedelbach, 1989; Mikulincer & Solomon, 1988). Conversely, in survivors of the *Herald of Free Enterprise* and *Jupiter* ship disasters, internal or controllable attributions for disaster-related events were associated with poorer outcome (Joseph, Brewin, Yule, & Williams, 1991, 1993).

Comorbidity

PTSD shares a number of clinical characteristics with other psychiatric disorders and is rarely diagnosed in isolation (Davidson & Foa, 1991; McNally, 1992). Typically, around 80% of PTSD sufferers receive an additional diagnosis (McFarlane, 1992), although work on combat veterans has suggested that lifetime comorbidity rates may be as high as 99% (Kulka et al., 1990). Epidemiological surveys have reported that rates of somatization disorder, psychosis, anxiety disorder, and depression are substantially elevated in PTSD sufferers (Davidson, Hughes, Blazer, & George, 1991; Helzer et al., 1987; Shore, Vollmer, & Tatum, 1989). Studies of Vietnam veteran populations indicate that depression, generalized anxiety disorder, and substance abuse are the most frequent codiagnoses (Davidson & Foa, 1991).

Numerous authors, noting the high comorbidity of PTSD with anxiety disorder and depression, have pointed to the substantial symptom overlap. For example, Farmer, Tranah, O'Donnell, and Catalan (1992) and McNally (1992) have noted that many depressive symptoms appear in Sections C and D of the *DSM–III–R* criteria for PTSD: C4, markedly diminished interest in significant activities; C5, feelings of estrangement or detachment from others; C6, restricted affect; C7, sense of foreshortened future; D1, difficulty in staying or falling asleep; D2, irritability or outbursts of anger; and D3, difficulty in concentrating. Other symptoms, such as guilt, are commonly found in both conditions.

It would not be fair to conclude, however, that symptom overlap is an adequate explanation of comorbidity. Noting the various similarities between depression and PTSD, McNally (1992) suggested that the distinctive features of PTSD are the exaggerated startle, the reexperiencing symptoms (such as nightmares or intrusive memories of the trauma), and physiological reactivity to trauma-related cues. He also reviewed a number of differences in biological variables and drew attention to various ways in which PTSD differed from panic disorder and phobia (i.e., in the latter, psychic numbing and reexperiencing phenomena are unusual).

Experimental Data

As well as providing an explanatory framework for the wealth of self-report data, any comprehensive theory of PTSD needs to account for the findings from laboratory-based paradigms that do not rely on subjective reports of conscious states. Such experimental methodologies can potentially demonstrate biases in the underlying cognitive processes of perception, attention, judgment, and memory in PTSD sufferers; each of these cognitive mechanisms is considered in turn.

McNally et al. (1987) investigated auditory threshold for combat-related and neutral material both in participants with combat-related PTSD and in controls. Although all of the participants exhibited a lower threshold for the combat-related words, relative to the neutral control stimuli, McNally et al. found some suggestive evidence that detection of threat targets

was accompanied by enhanced skin conductance responding in PTSD, relative to control, participants.

Several studies of attentional processing in PTSD have used a modified Stroop (Stroop, 1935) color-naming paradigm. However, there is some debate about whether this task is a true measure of attentional bias (Dalgleish, 1994; MacLeod, 1990). In the modified Stroop task, participants have to name (as fast as they can) the ink color in which words of personal or emotional significance are written, while attempting to ignore the words themselves. McNally, Kaspi, Riemann, and Zeitlin (1990) and McNally, English, and Lipke (1993) found that Vietnam veterans with PTSD were slower to color-name Vietnam-related words relative both to control words and to the performance of a group of veterans without PTSD. Kaspi, McNally, and Amir (1995) found that this Stroop effect was strongest for threatening information with a strong degree of self-relevance, although only in individuals with PTSD. Similar effects have been found in other studies of combat veterans (Vrana, Roodman, & Beckham, 1995), as well as in studies of rape victims with PTSD (Cassiday, McNally, & Zeitlin, 1992; Foa, Feske, Murdock, Kozak, & McCarthy, 1991) and PTSD sufferers who have survived a major disaster (Thrasher, Dalgleish, & Yule, 1994).

Memory biases have been found on an autobiographical memory task with Vietnam war veterans (McNally, Lasko, Macklin, & Pitman, 1995; McNally, Litz, Prassas, Shin, & Weathers, 1994). Veterans with PTSD, especially those who wore combat regalia in daily life, exhibited deficits in retrieving specific autobiographical memories to a set of cue words. Finally, Dalgleish (1993) examined judgmental bias in survivors of a major disaster with and without PTSD. It was shown that only those survivors with PTSD generated elevated judgments of the probability of a range of negative events happening in the future.

Cognitive Theories of PTSD

In addition to biological theories (e.g., Kolb, 1987; Van der Kolk, Boyd, Krystal, & Greenburg, 1984), a number of psychological paradigms provide frameworks for understanding PTSD, for example, the psychodynamic (Freud, 1919; Kardiner, 1941; Krystal, 1968), learning theory (Fairbank & Brown, 1987; Keane, Fairbank, Caddell, Zimering, & Bender, 1985; Keane, Zimering, & Caddell, 1985; Kilpatrick et al., 1985), and cognitive (e.g., Horowitz, 1986). Other authors have proposed models that integrate psychological and biological perspectives (Jones & Barlow, 1990). Although all of these approaches encompass theories that offer interesting insights into the nature of the disorder, it is the cognitive one that is perhaps the most fully developed and offers the greatest explanatory and predictive power. For this reason, in this review, we will focus on the various cognitive theories of PTSD.

Cognitive theories of PTSD share certain core theoretical assumptions. They propose that individuals bring to the traumatic experience a set of preexisting beliefs and models of the world. The experience of trauma provides information that is both highly salient and incompatible with these preexisting models. The attempt to assimilate this new information with the existing models leads, it is argued, to the various phenomena that characterize the posttraumatic reaction. Successful information processing occurs when the new information is integrated into the existing models (often by virtue of changes in those same models). Unsuccessful information processing occurs when the individual is unable to bring the new trauma-related information into accord with the current models of the world. This can lead to pathological posttraumatic reactions such as PTSD.

The strength of the cognitive approach is that each of the various theories addresses the majority of the data about PTSD that we have reviewed in the preceding sections. However, each theory endeavors to explain only a circumscribed set of empirical and clinical findings. Data that fall outside of the theoretical spotlight, although addressed, are often merely referred to or redescribed, with no explication of how the theory in question would explain the findings.

Cognitive theories of PTSD fall into two distinct camps. Social–cognitive theories, such as those of Horowitz and Janoff-Bulman (see below), emphasize the impact of the trauma on individuals' lives and highlight the massive readjustments that often need to be made to integrate the traumatic experience into an individual's preexisting views of the world. By emphasizing the wider impact of the trauma and its consequences, they are able to explain other reactions, such as anger, anxiety, and depression, which often accompany PTSD. In contrast, information-processing theories, such as that of Foa and colleagues (e.g., Foa et al., 1989), focus more specifically on trauma-related threat, on how trauma-related information is represented in the cognitive system, and how it is subsequently processed. In the next section, a number of social–cognitive and information-processing theories of PTSD are briefly described and areas are discussed in which the theories have some explanatory power and in which they offer no more than a descriptive or embryonic analysis of the data.

Horowitz's (1973, 1976, 1979, 1986; Horowitz, Wilner, Kaltreider, & Alvarez, 1980) formulation of stress-response syndromes offers perhaps the most comprehensive and influential social–cognitive theory of PTSD. Although influenced by classical psychodynamic psychology (notably Freud, 1920), Horowitz's theory is principally concerned with the cognitive processing of traumatic information (i.e., ideas, thoughts, images, affects, and so on). Horowitz (1976) argued that the main impetus for such processing comes from a *completion tendency*: The psychological need for new information to be integrated with existing cognitive world models or schemata (Horowitz, 1986).

Horowitz (1986) proposed that, subsequent to an experience of trauma, there is an initial crying out or stunned reaction followed by a period of information overload, in which the thoughts, memories, and images of the trauma cannot be reconciled with current schemata. As a result, a variety of psychological defense mechanisms come into operation to keep the traumatic information unconscious and the individual experiences a period of numbing and denial. However, the completion tendency helps maintain the trauma-related information in active memory, causing it to break through these defenses and intrude into consciousness in the form of flashbacks, nightmares, and unwanted thoughts as the individual endeavors to merge the new information with existing models. This tension between the

completion tendency, on the one hand, and the psychological defense mechanisms, on the other, causes the person to oscillate between phases of intrusion and denial-numbing as he or she gradually integrates the traumatic material with his or her long-term schematic representations. According to Horowitz (1986), failures of information processing can mean that the partially processed traumatic information remains in active memory without ever being fully integrated, thus leading to chronic posttraumatic reactions.

Horowitz (1986) has developed an extremely comprehensive theory of stress response. His discussion of the processes underlying completion, intrusion, and denial has considerable explanatory potential for PTSD phenomenology. The theory indicates clearly the ways in which normal reactions to trauma can become chronic or pathological. However, Horowitz's theory has a number of limitations that are important to highlight. First, there is little discussion of why some individuals develop PTSD, whereas others, after ostensibly similar traumatic experiences, show little or no symptomatology. Indeed, there is little detail on the nature of existing schema structure and the exact ways in which it fails to accommodate new information from the traumatic experience. Second, Horowitz's formulation struggles to account for epidemiological data regarding the frequency of late onset, although this could be ascribed to a long period of denial that later breaks down. Third, although Horowitz provides a clear description of the time course of posttraumatic reactions, it is far from certain that all individuals do experience an initial period of denial or later oscillations between denial and intrusion. In fact, Creamer et al. (1992), whose ideas are discussed below, argued for an initial episode of intrusive symptoms. Fourth, although Horowitz highlights processes such as social support, there is little explanation within the theory of how such factors might operate. Finally, Horowitz's theory is somewhat passive. Little credit is given to the power of the individual's attributions and interpretations of the traumatic experience and the effect that these have on outcome.

The cognitive–appraisal theory of Janoff-Bulman (1985, 1992; Janoff-Bulman & Frieze, 1983) focused principally on the nature of the preexisting models of the world that the individual carries into the traumatic situation. Janoff-Bulman argued that PTSD is the result of certain basic assumptions about the world that are shattered: the assumption of personal invulnerability, the perception of the world as meaningful or comprehensible, and the view of the self in a positive light.

Janoff-Bulman's (1985, 1992; Janoff-Bulman & Frieze, 1983) work is important in that it describes the ways in which trauma-related information is incongruent with the usual models and assumptions about the world that people possess. There is little attempt, however, to explain how such models are represented or what processes are involved when they are shattered. An additional problem is the ubiquitous finding (e.g., Kilpatrick et al., 1985) that individuals with a premorbid psychiatric history are more likely to develop PTSD after a trauma. Such individuals would presumably be characterized by assumptions of personal vulnerability and views of the self in a negative light. Such premorbid assumptions are unlikely to be shattered by a traumatic experience (in fact, they are more likely to be confirmed), and the high incidence of PTSD following a trauma in this population has yet to be addressed by cognitive–appraisal theories, such as that of Janoff-Bulman.

Applying Lang's (1979, 1985) concept of fear structures, Foa and her colleagues (Foa & Kozak, 1986; Foa & Riggs, 1993; Foa et al., 1989, 1992) have put forward an information-processing theory of PTSD that centers around the formation of a fear network in memory. This network encompasses stimulus information about the traumatic event; information about cognitive, behavioral, and physiological reactions to the trauma; and interoceptive information that links these stimulus and response elements. Activation of this fear network by triggering stimuli (i.e., reminders of the trauma) causes information in the network to enter consciousness (the intrusion symptoms of PTSD). Attempts to avoid and suppress such activation lead to the cluster of avoidance symptoms. Successful resolution of the trauma can occur only by integrating the information in the fear network with existing memory structures. Such integration requires, first, the activation of the fear network so that it becomes accessible for modification and, second, availability of information that is incompatible with the fear network so that the overall memory structure can be modified. A number of factors make such integration problematic; Foa and her colleagues (Foa & Kozak, 1986; Foa & Riggs, 1993; Foa et al., 1989, 1992) argued that the unpredictability and uncontrollability of the traumatic event make it difficult to assimilate into existing models in which the world is controllable and predictable. In addition, factors such as the severity of the event disrupt the cognitive processes of attention and memory at the time of the trauma. They argued that this disruption leads to the formation of a disjointed and fragmented fear network that is consequently difficult to integrate with existing organized models.

By outlining an information-processing architecture, within which some of Horowitz's and Janoff-Bulman's social–cognitive ideas can be instantiated, Foa and her colleagues (Foa & Kozak, 1986; Foa & Riggs, 1993; Foa et al., 1989, 1992) have made considerable progress toward a greater understanding of how the cognitive processes underlying PTSD operate. Furthermore, in stressing factors such as the predictability and controllability of the trauma, they have highlighted one important role of the individual's attributions and interpretations of the traumatic event. Also, the proposal that the availability of information incompatible with the trauma is necessary for successful information processing provides a framework for understanding both the role of social support as a vehicle for the provision of such information and the processes underlying the success of exposure-based treatments for PTSD. What is less clear is whether network theory provides an architecture powerful enough to cope with the range of PTSD phenomenology. For example, Foa and her colleagues described in any detail only a network with a single level of representation. Such an analysis struggles with even basic concepts such as denial, numbing, and psychogenic amnesia, which require a higher level of representation that cannot access the information in memory. The term *consciousness* is used (Foa et al., 1989) to refer to this higher stratum, but it is unclear whether consciousness is represented by a network or by some other form of cognitive architecture. Furthermore, there is little discussion of how the existing models of the world, which are such a feature of the social–

cognitive theories (Foa & Riggs, 1993), are represented by networks, how integration of new information with such models might take place, or of why fear networks develop in some individuals but not in others.

The cognitive action theory of Chemtob et al. (1988) is based on work with veterans of the Vietnam war. It presents a similar perspective to that of Foa et al. (1989), although with more detailed analysis of the structure of the fear network, which is formulated as a parallel-distributed, hierarchical system. Chemtob et al. argued that, in individuals with PTSD, the fear network is permanently activated, causing them to function in a survival mode that has proved adaptive during the traumatic incident. This permanent activation leads to the symptoms of hyperarousal and intrusion.

The cognitive action theory of Chemtob et al. (1988) provides another potential processing framework within which to understand PTSD. It suffers slightly from being overly narrow in its emphasis on combat-related trauma. Also, as with the other theories discussed above, it offers little insight into why some individuals remain in a survival mode, whereas others do not. Similarly, there is no discussion of other variables of known importance, such as attributions and social support.

Creamer et al.'s (1992) cognitive processing theory of PTSD is presented as a "synthesis and reconceptualization of existing formulations" (p. 453). It combines the central ideas of Horowitz with the network architecture of Foa et al. (1989) and Chemtob et al. (1988). Creamer et al. proposed—as did Foa—that the fear network must be activated for recovery to take place—a mechanism referred to as *network resolution processing*. This concept is similar to Horowitz's (1986) completion tendency. However, as noted above, Horowitz and Creamer et al. differed in the details of such resolution or completion. Horowitz (1986) suggested an initial period of denial-numbing, followed by oscillating experiences of intrusion and denial-numbing. In contrast, Creamer et al. argued for an initial period of intrusion (because of activation of the fear network), which the individual copes with by bringing a range of defensive and avoidant strategies into play. Creamer et al. suggested that the extent of initial intrusive symptomatology is an index of the degree of network resolution processing that is occurring. Thus, high levels of initial intrusion are a predictor of successful recovery, whereas low levels of initial intrusion are a predictor of poor outcome and chronic pathology. Creamer et al.'s longitudinal follow-up of the victims of an office block shooting supports these predictions. However, other data (e.g., McFarlane, 1989) has suggested that prior levels of high intrusion are predictive of a poorer outcome.

Creamer et al.'s (1992) theory is significant in that it is based on longitudinal data and makes clear predictions about outcome. However, it has a number of drawbacks. First, the theory has limited explanatory value; it is an attempt at presenting some interesting correlational data in a loose theoretical framework. Second, the theory gives no indication as to why some individuals develop PTSD and others do not, nor does it account for the effects of factors of known importance, such as social support and the individual's attributions and interpretations of the event. Finally, the strong predictions concerning outcome are only partially supported by the literature (Horowitz, 1986; McFarlane, 1989).

In summary, cognitive theories of PTSD fall into two distinct groups. The primary focus of one is the impact of the trauma on the person's general assumptions and goals, the generation of complex emotions, and the sometimes profound changes that will be necessary to adjust to the consequences of the trauma. The primary focus of the other is the intrusion of trauma-related flashbacks, the generation of the same intense emotions felt during the trauma, and the need to habituate to trauma-related images and stimuli. The two groups of theories appear to be focusing on phenomena that, although clearly related, may have different underlying mechanisms.

Dual Representation Theory and Trauma Processing

What Is Trauma?

By *trauma* we mean any experience that by its occurrence has threatened the health or well-being of the individual. Just as physical trauma may extend from minor abrasions to severe tissue damage, there is no implication that psychological trauma must involve an event outside the ordinary range of human experience, although it may do so. As various authors have suggested (e.g., Horowitz, 1986; Janoff-Bulman, 1992; Parkes, 1986), trauma generally involves a violation of basic assumptions connected with survival as a member of a social group. These include assumptions (not necessarily conscious ones) about personal invulnerability from death or disease, status in a social hierarchy, the ability to meet internal moral standards and achieve major life goals, the continued availability and reliability of attachment figures, and the existence of an orderly relation between actions and outcomes. The types of events likely to violate these assumptions will frequently involve indications that the world is uncontrollable or unpredictable (Foa et al., 1992), including major illness or disability; physical or sexual assault; social humiliation; transgression of one's own moral code; loss of employment; divorce and separation; bereavement; and involvement in actual or potential accidents, conflict, and natural disasters. Where such assumptions have already been violated, events that confirm the violation may also be traumatic.

The Representation of Trauma in Memory

A number of cognitive psychologists researching "flashbulb memories" have proposed that a special mechanism exists for encoding emotion-laden memories of events such as the death of President John F. Kennedy (e.g., Brown & Kulik, 1977; Conway, 1995). In general, however, this research has not considered the special properties of memory for personal trauma, particularly the experience of reliving the event. Clinical researchers have attempted to account for the often severe impact of trauma by proposing that it leads to the formation of particularly durable representations in memory. They have suggested that these emotional memories contain a record of the stimulus elements of the event, the person's physiological and motor responses, and the individual meaning of the event (Foa & Kozak, 1986; Lang, 1979). However, the concept of a single emotional memory does not appear adequate to capture the full range of observed phenomena reviewed earlier in this article, for exam-

ple, the distinction between verbally retrievable memories and flashbacks.

Research in several areas of cognitive psychology, social psychology, clinical psychology, and neuropsychology supports the view that sensory input is subject to both conscious and nonconscious information processing (see Brewin, 1988, 1989; Epstein, 1994, for reviews). It has been persuasively argued that the characteristics of nonconscious processing (e.g., extreme rapidity, parallel processing of multiple inputs) permit far more detailed and extensive computations than does conscious processing, which is limited by its slowness, serial nature, and our inability to hold more than a small amount of information in memory at one time. Similarly, it has been proposed that the output of these different forms of processing are stored in different locations or different codes (e.g., Broadbent, FitzGerald, & Broadbent, 1986; Tulving & Schacter, 1990). Neuroanatomical research has located a variety of pathways that might permit sensory data associated with an emotionally significant event to be stored in memory without being subjected to cortical processing (LeDoux, 1992).

In applying these ideas for the first time to the experience of trauma, Brewin (1989) and Terr (1991) suggested that from the outset there will be more than one type of representation of an experience of single or repeated trauma. This proposal is mirrored in related theoretical work, with theories such as Teasdale and Barnard's (1993) interacting cognitive subsystems and Johnson and Multhaup's (1992) multiple-entry modular memory system, emphasizing the need for more than one level of representation in order to understand the complex relationship between emotion and cognition. The theory presented here builds on earlier work (Brewin, 1989) by proposing dual representations in memory of traumatic experiences as the minimum cognitive architecture within which the complex data reviewed earlier can be understood. As illustrated in Figure 1, one representation or set of representations will be of the person's conscious experience of the trauma. Brewin (1989) termed this *verbally accessible knowledge* because it can in principle be deliberately retrieved from the store of autobiographical experiences. These memories, although they are likely to be reasonably detailed, may be highly selective because anxiety increases attentional selectivity and decreases short-term memory capacity (Eysenck & Keane, 1990). The verbally accessible memories (VAMs) will contain some information about the sensory features of the situation, the emotional and physiological reactions experienced, and the perceived meaning of the event.

The output of the more extensive nonconscious processing of the traumatic situation will form a second representation or set of representations that cannot be deliberately accessed. Brewin (1989) termed this *situationally accessible knowledge* because the representations may be accessed automatically when the person is in a context in which the physical features or meaning are similar to those of the traumatic situation. This context may be internal, such as consciously thinking about the trauma, or external, such as hearing about a similar trauma on television. The particular form that such representations may take (schemas, mental models, distributed networks, etc.) is a matter of controversy and is not specified by the theory. Although the two representations are likely to share a number of similarities, they will also differ in important respects. This is because people are able to manipulate to some degree their autobiographical memories, and also because situationally accessible memories (SAMs) are not subject to the same processing capacity limitations as verbally accessible knowledge. Moreover, in SAMs, the meaning automatically ascribed to events may not correspond to verbally retrievable meanings in terms of consciously held goals and plans (see below).

According to a number of authors (e.g., Jacobs & Nadel, 1985), the hormonal effects of acute trauma may act to diminish neural activity in anatomical structures serving conscious processing and to enhance activity in structures serving nonconscious perceptual and memory processes. We propose that the sensory (visual, auditory, olfactory, etc.), physiological, and

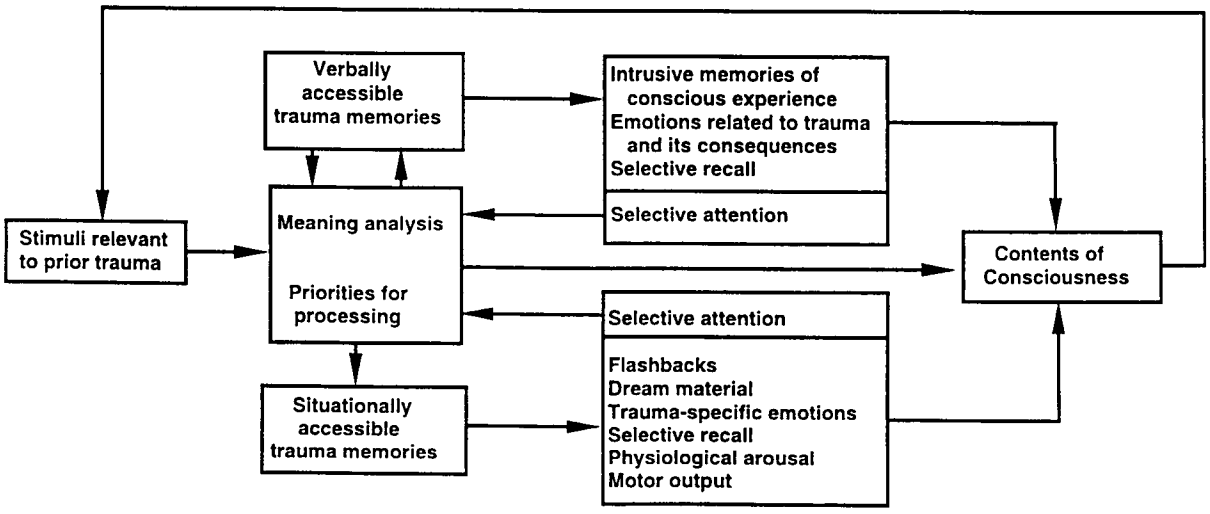

Figure 1. Cognitive processing of stimuli relevant to prior trauma. Adapted from "Cognitive Change Processes in Psychotherapy," by C. R. Brewin, 1989, *Psychological Review, 96*, p. 383. Copyright 1989 by the American Psychological Association.

motor aspects of the traumatic experience are represented in situationally accessible knowledge in the form of analogical codes that enable the original experience to be recreated. These codes would be part of an overall representation that contained (a) stimulus information automatically coded for its ability to discriminate the trauma from other previous nontraumatic situations, using criteria such as novelty (Gray, 1982); (b) meaning information derived from prior associative learning and from innate, nonconscious appraisal mechanisms concerned with the achievement of universal goals such as attachment to caregivers or the regulation of status within a social hierarchy (e.g., Gilbert, 1989); and (c) information about the person's state of consciousness, for example, their degree of detachment (dissociation) from the traumatic situation. This account, although elaborated in various respects, is similar to Leventhal's (1984) description of schematic emotional memories. The person may only become aware that these representations have been accessed when they experience symptoms such as emotional arousal, motor impulses, spontaneous intrusive images, or dissociative states.

In contrast, verbally accessible knowledge of the trauma consists of a series of autobiographical memories that can be deliberately and progressively edited. For example, autobiographical knowledge can be ordered on a hierarchy of generality from broad lifetime themes, to general descriptions of events, to detailed event-specific knowledge (Conway, 1996; Williams, 1992). Immediately posttrauma, these memories are likely to be dominated by detailed information concerning the conscious perception of sensory detail and of bodily reactions. There will also be an initial attempt to assign meaning to the trauma in terms of verbally accessible constructs and categories, and to consider the implications for valued life goals. As time goes on, however, more generic memories may be created that encompass the fact of having experienced the trauma but without the same level of detail. Similarly, repeated recall of certain aspects of the experience may give opportunities to focus on and embellish some features, whereas the ability to recall other features may be impaired (e.g., Hashtroudi, Johnson, Vnek, & Ferguson, 1994).

There is persuasive evidence from the clinical literature documenting a variety of other, less common memory phenomena. Although they are not the primary focus of this article, they will be briefly mentioned. Thus, under conditions of extreme stress, attentional narrowing may be so great that VAMs contain significant gaps, for example, omitting certain moments in time or information from a particular sensory modality. Herman (1992a) and Terr (1991) have suggested a distinction between Type 1 traumas, usually involving a single episode, and Type 2 traumas, where there is repeated extreme stress. They proposed that Type 2 traumas may lead to the development of dissociative reactions, for example, where the person is able to observe him or herself from an external (out-of-body) perspective. It is likely that these disturbances of consciousness may in some cases create additional barriers to the registration and recall of verbal memories. Finally, there are fugue states in which there is impaired access not just to specific VAMs but to autobiographical memory as a whole. Theoretically, we would predict, however, that SAMs would remain intact, despite the impairment in VAMs. Studies of functional amnesia after trauma confirm that individuals may demonstrate implicit memory for relevant details even though they have little explicit memory (Christianson & Nilsson, 1989).

Emotional Processing of Trauma

The idea that trauma is normally followed by a period of emotional processing is, as we have seen, a characteristic of social–cognitive and information-processing accounts, as well as of more general explanations of how cognitive–behavior therapies work (e.g., Rachman, 1980). Accounts differ, however, in the nature of the intrusive memories they describe and in the mechanisms they put forward to explain what drives emotional processing. In the information-processing theories (e.g., Foa & Kozak, 1986), it is the activation of traumatic memories by appropriate cues, whereas in the social–cognitive theories (e.g., Janoff-Bulman, 1992), it is the discrepancy between the trauma and the prior assumptions. We will attempt to synthesize the accounts of this process that we reviewed earlier and explain them in terms of a dual representation theory.

We use the term *emotional processing* broadly to denote a largely conscious process in which representations of past and future events, and awareness of associated bodily states, repeatedly enter into and are actively manipulated within working memory. We suggest that this process, which may or may not lead to a satisfactory outcome, has at least two elements. One element, described best by information-processing theories, involves the activation of highly specific SAMs, whose function is to aid the process of cognitive readjustment by supplying detailed sensory and physiological information concerning the event (flashbacks). The second element, described best by social–cognitive theories, is the conscious attempt to accommodate the conflicting information supplied by the trauma by searching for meaning and making judgments of cause and blame. The end point of this process is to reduce negative affect by restoring a sense of safety and control and by making appropriate adjustments to expectations about the self and the world. This process often has the unwanted effect of creating internal cues that tend to trigger the spontaneous intrusion of SAMs into consciousness. Both elements are further facilitated by the information-processing system giving high priority to trauma-related cues in the form of attentional and memory biases.

Emotional processing will therefore involve, we suggest, different kinds of emotional reactions. First, there will be conditioned emotional reactions corresponding to the activation of specific emotional states (predominantly fear but also other emotions such as anger) experienced during the trauma, as recorded in the person's SAMs of the event. In conjunction with these, we, along with numerous other authors (e.g., Oatley & Johnson-Laird, 1987; Weiner, 1985), suggest that emotions such as sadness, anger, and fear for the future will be generated by ongoing threat to safety, the disruption of plans, and the loss of valued goals. Other emotions, such as guilt and remorse (and also anger), may be generated through responsibility attributions. All of these emotions, which we term *secondary emotions*, follow from the consequences and implications of the trauma. The increased cognitive accessibility of trauma-related information can account for other features of PTSD, such as startle

responses and an elevated subjective probability of aversive events occurring.

The aim of this period of emotional processing may, similarly, be seen to consist of two elements. On the one hand, the person needs to actively reduce the secondary negative affects generated by the implications of the trauma by consciously reasserting perceived control, reattributing responsibility, and achieving an integration of the new information with preexisting concepts and beliefs. This process, described in detail by the social–cognitive theories of Horowitz (1986) and Janoff-Bulman (1992), may involve substantial editing of autobiographical memory (VAMs) in order to bring perceptions of the event into line with prior expectations. For example, the behavior of an attacker previously believed to be trustworthy may be reinterpreted, excused, or explained away, or aspects of what was done or said may be forgotten. Alternatively, previous expectations may be adjusted in line with the event, for example, behaviors, neighborhoods, or locations believed to be safe may be reclassified as dangerous or life goals may be abandoned in favor of less ambitious ones.

The second aspect to emotional processing is to prevent the continued automatic reactivation of situationally accessible knowledge about the trauma. It has been suggested that alterations in situationally accessible knowledge may be brought about by incorporating new knowledge into the original SAMs (e.g., Foa & Kozak, 1986) or by creating new SAMs that block access to the original ones (Brewin, 1989).[1] Following activation and emergence into consciousness, SAMs will be automatically altered or added to whenever some or all of the information they contain happens to be paired with changes in concurrent bodily states or contents of consciousness. Changes in bodily states may consist of states of reduced arousal and reduced negative affect. These affective and arousal changes may be brought about by a number of means, including spontaneous or programmed habituation to the traumatic images. Similar changes would also be expected to follow the conscious restoration of a sense of safety (reduced fear), the abandonment of now unattainable goals (reduced sadness), the absolution of others from responsibility for the trauma (reduced anger), the absolution of self from responsibility (reduced guilt), and other attempts to integrate the new information into preexisting expectations.

As this process of conscious cognitive restructuring continues, the trauma images can be paired with progressive representations of effective action–outcome sequences and reduced negative affect. Reminders of the trauma will then, hopefully, lead to the accessing of more recent representations rather than the original traumatic memories. In the absence of the negative affect, there will also be a reduction in attentional and memory biases and, hence, in the accessibility of the memory.

Conversely, situational representations of trauma-related threat may be strengthened if the person continues to be anxious and fearful about the consequences of the trauma. Threat representations may also be altered as a result of stimulus revaluation (Davey, 1989). Subsequent information that increases the perception of threat—for example, that an attacker has previously or subsequently killed someone—may exacerbate attentional and memory biases and result in an increased likelihood that SAMs will be activated by relevant cues. Equally, information that decreases the perception of threat may result in a reduction in selective cognitive biases and the decreased accessibility of situational memories.

Table 1
Outcomes of Emotional Processing

Outcome	Description
Completion/integration	No memory bias No attentional bias No symptoms
Chronic emotional processing	Memory biases Attentional biases Phobic state Depression Panic Anxiety Substance abuse
Premature inhibition of processing	Attentional biases Avoidance schema Impaired memory Phobic state Dissociation Somatization

As has frequently been noted, the affect accompanying trauma can be so overwhelming that processing may be involuntarily suspended and the individual may experience emotional numbing. Alternatively, people may attempt to deliberately interfere with this processing by distracting themselves and systematically avoiding either reminders of the trauma in general or reminders of specific aspects of the trauma. The various processes of information retrieval, conscious reappraisal, emotional numbing, and cognitive and behavioral avoidance allow for a number of quite different outcomes to the processing of any particular trauma.

Three Endpoints of Emotional Processing

A variety of well-known strategies are commonly used for resolving discrepancies between preexisting schemas and new information. These include externalization of responsibility, disengagement of self-evaluative processes, victim blaming, denial of the validity of the new information, and revaluation of previously held goals (see Fiske & Taylor, 1991, for a review). The nature of trauma is such, however, that previously successful strategies may no longer be effective and more radical solutions may be required. As we have seen, the outcome of the period of emotional processing is likely to be influenced by a number of factors, including the severity and length of the trauma, its meaning to the person, accompanying emotions such as guilt or shame, and the availability of appropriate social support that enables the survivor to confide. Three possible outcomes may be distinguished: completion/integration, chronic emotional processing, and premature inhibition of processing (see Table 1). We describe these outcomes in more detail.

[1] Recent research in animal learning is consistent with the idea that it is not possible to "unlearn" conditioned emotional reactions and that, even after its extinction, fear may be reinstated by the appropriate contextual cues (Bouton & Swartzentruber, 1991).

Completion/integration. Completion or integration represents the ideal stage in which the memories of the trauma have been fully processed, or worked through, and integrated with the person's other memories and sense of self in the world. In part, this involves reducing negative affect by restoring a sense of control and by resolving discrepancies with preexisting expectations and goals. There must be sufficient repetition of the incident in memory for the person to accept the reality of what has happened to them and its consequences without being overcome by the accompanying emotions. The threat of recurrence in the future can be assessed realistically, and experiences that induced guilt or shame and have implications for the self-concept lead to adjustments in self-expectations. Horowitz (1979) noted that before major interpersonal loss can be accepted, the survivor has to adjust his or her internal model of self and of the relationship to the lost person. However, as noted by Janoff-Bulman (1992), as a result of these adjustments, quite profound changes may occur and the person may feel that he or she is no longer the same as before the trauma occurred.

Reductions in negative affect may permit the person to tolerate the intrusion of situational memories and to allow habituation or stimulus revaluation to take place. Habituation is likely to be brought about by repeated exposure to the memories in states of gradually increasing calm and relaxation. Social support has an obvious role ~~to play here~~, in directly reducing negative affect by offering physical comfort and emotional support, in providing opportunities for the repeated rehearsal of the traumatic memories, and in assisting in the process of cognitive reappraisal.

Theoretically, we would predict that successful integration would be marked by the absence of attentional and memory biases for trauma-related stimuli; in other words, these stimuli would no longer be deemed sufficiently important to be accorded preferential processing. Successful integration should be achieved under the following conditions: small discrepancies between trauma information and prior assumptions (a result of less intense and nonrepeated stressors, less negative self-evaluation, lack of previous trauma, less extreme assumptions, and less rigid goals), adequate cognitive development, good social support, and ability to tolerate the intrusion of SAMs into consciousness.

Chronic emotional processing. For a variety of reasons complete integration may represent an ideal that is not possible to attain. The trauma may have been so severe and so prolonged, or have such profound consequences for the person's sense of self and of future safety, that the discrepancy between the trauma and the prior assumptions is too great and the memories cannot be integrated. Alternatively, the person may not have been able to process memories of the trauma effectively because of (a) competing demands, (b) aversive secondary emotions, (c) lack of an appropriate confidant or unwillingness to confide, (d) being too young to appreciate the meaning and circumstances of the event, and (e) the presence of ongoing trauma or threat that continually reactivates trauma memories.

One possible result is that VAMs and SAMs concerning the trauma are chronically processed, with the result that the person is permanently preoccupied with the consequences of the trauma and with intrusive memories. The processing may be largely repetitive with little or no change being effected to existing representations. There is now considerable evidence (reviewed earlier) that a proportion of war veterans and survivors of rape or of man-made disasters develop chronic PTSD that may last for months or years.

In these circumstances, the characteristic features of acute emotional processing, such as heightened arousal and attentional and memory biases, are likely to be chronically present. Also, secondary reactions such as depression, cognitive and behavioral avoidance, or anxiety and panic may develop, reflecting generalization of the impact of chronic processing. For example, patients may experience anxiety phenomena thought to be associated with defensive processes such as obsessional thoughts (Salkovskis, 1985) or worrying (Borkovec & Lyonfields, 1993). These cognitions may not necessarily have any direct trauma-relevant content. Substance abuse may also develop as a coping response. We suggest, therefore, that in many cases comorbidity of PTSD with depressive, anxiety, or substance abuse disorders is likely to reflect the effect of chronic emotional processing.

Chronic processing is likely under the following conditions: large discrepancies between trauma information and prior assumptions (a result of more intense or repeated stressors, more negative self-evaluation, presence of previous trauma, more extreme assumptions, and more rigid goals), inadequate cognitive development, poor social support, and inability to prevent the intrusion of SAMs into consciousness. This inability may be linked to resolutions of the discrepancy in which previous assumptions and beliefs have changed in the direction of seeing the self and the world in much more negative terms. Ehlers and Steil (1995) also presented evidence suggesting that distressing misinterpretations of the PTSD symptoms themselves (e.g., interpreting the occurrence of intrusive recollections as a sign of brain damage or impending breakdown) lead to more chronic PTSD.

Premature inhibition of processing. The other possible outcome is that emotional processing will be prematurely inhibited. Usually, inhibition is the result of sustained efforts to avoid the reactivation of unpleasant SAMs and VAMs. Trauma victims frequently describe strategies they use for avoiding thinking about the trauma and hence escaping the accompanying emotional arousal. Sufficient repetition of these strategies may result in this process becoming automatic. For example, an avoidance schema may develop that monitors sensory input for trauma-related stimuli and directs conscious attention away from them. Similarly, the person may develop trauma-related scripts that enable him or her to incorporate the autobiographical fact of the trauma into his or her VAMs and to have limited communication about it without reactivating the SAMs.

In this state there will no longer be any active emotional processing, intrusive memories, or deliberate attempts to avoid intrusions, but the SAMs concerning the trauma should still be accessible in the right circumstances. We therefore predict that it should be distinguishable from the state of completion/integration in the following ways: (a) Attentional biases should still be present, indicating that trauma-related stimuli are still being accorded high priority; (b) the person may have impaired memory for the trauma or for trauma-related stimuli; (c) the person should show phobic avoidance of trauma-related situations; and (d) the person may show evidence of somatization.

The significance of this type of outcome is that, although the

person may appear to have recovered from the effects of the trauma, the unprocessed memories remain vulnerable to reactivation later in life. This may occur when the person encounters similar situations or enters similar mood states. Premature inhibition of processing is likely under the following conditions: large discrepancies between trauma information and prior assumptions, inadequate cognitive development, poor social support, and the ability to prevent the intrusion of SAMs into consciousness. This ability may be linked to the use of dissociative defences during the trauma or to a general facility to avoid processing negative information. People with a repressive coping style appear particularly adept at this, showing attentional avoidance (Fox, 1993), impaired memory for negative stimuli (Myers & Brewin, 1994, 1995), and unrealistically optimistic assumptions and beliefs (Myers & Brewin, in press).

A large volume of research has now been carried out on the physiological effects of not expressing deeply felt emotions (e.g., Pennebaker, Kiecolt-Glaser, & Glaser, 1988). It appears that inhibiting the expression of emotion leads to impaired immune function and poorer health status on a variety of indices.

Relation of Dual Representation Theory to Existing Evidence

Although the dual representation theory of PTSD has not been directly tested, it is appropriate to review briefly its relation to existing findings.

Clinical characteristics of PTSD. The theory deals most directly with the intrusions and avoidance that are typical of PTSD. Unlike other theories, however, it distinguishes different types of intrusive phenomena, represented by repeated accessing of VAMs and SAMs. Repeated accessing of VAMs is accompanied by emotional reactions such as anger, sadness, and guilt linked to loss, future threat, and the assignment of blame. VAMs may be edited in various ways to control affect by emphasizing positive or negative aspects or by creating more or less detailed representations of the trauma. In contrast, SAMs tend to be highly detailed, repetitive memories (flashbacks) that are difficult to edit and that are accompanied by emotional and physiological changes experienced during the trauma. The greatly enhanced accessibility of VAMs and SAMs and their associated emotional reactions lead to the familiar symptoms of arousal.

The idea that conscious emotional processing may be prematurely inhibited is another feature of our dual representation theory that has not previously received much attention. This feature means that the theory can account more easily than other theories for the unpredictable time course of PTSD and with the frequent observation that, with exposure to appropriate cues, emotional processing may resume years after it appeared to have ceased. It also accounts for clinical observations of patients who appear never to have consciously processed a trauma but have subsequently experienced chronic nightmares. This would represent an extreme example of the inhibition of conscious processing.

Symptoms not addressed in detail by the theory include emotional numbing and dissociative reactions. In common with other authors (e.g., Herman, 1992b; Van der Kolk & Fisler, 1994), we view emotional numbing as a kind of psychic analgesia, analogous to the accompanying physical analgesia, that protects the person from overstimulation. Whereas emotional numbing appears to be a preprogrammed response that is not under conscious control, dissociation may be a learned response to prolonged or repeated trauma (Herman, 1992a). Our dual representation theory suggests that both of these responses to a specific trauma may be coded within the corresponding SAMs and, when triggered by an appropriate cue, may be reinstated along with other aspects of situationally accessible knowledge.

Normal or abnormal process? Unlike other theories of PTSD, our dual representation theory explicitly includes consideration both of a general response to trauma and of mechanisms that are specific to PTSD. Symptom duration of 1 month, necessary for a diagnosis of PTSD in *DSM–IV,* is likely to capture individuals who, in terms of our theory, are in the process of successfully completing their trauma processing, as well as those who will go on to experience chronic emotional processing or who will prematurely inhibit this processing.

In this way the theory can account for, and indeed predicts, the conflicting findings concerning the relation between intrusive memories and subsequent symptoms (Creamer et al., 1992; Foa & Rothbaum's, 1990, study, as cited in Rothbaum & Foa, 1993; Joseph, Yule, & Williams, 1994; McFarlane, 1992; Shalev, 1992). It suggests that there are two relevant moderator variables, the degree of trauma and the length of time elapsed since the trauma. With mild trauma, only a proportion of individuals are likely to experience intrusive memories for more than a few days, and more prolonged intrusions may therefore indicate an increased probability of later psychiatric disorder. In contrast, the presence of intrusive memories immediately after substantial trauma is a normal reaction that, as Foa and Rothbaum's (1990) study (as cited in Rothbaum & Foa, 1993) and Shalev (1992) found, will not predict subsequent adjustment. After some weeks or months, however, continuing emotional processing is likely to signal a failure to resolve discrepancies in expectations and goals and an inability to curtail unwanted memory reactivation. The longer the time elapsed since the trauma, therefore, the more likely this symptom is to predict a poor outcome (e.g., Joseph, Yule, et al., 1994; McFarlane, 1992).

It should be noted, however, that individuals who, following recent significant trauma, score low on measures of intrusion may have prematurely inhibited emotional processing. This group is predicted to be at enhanced risk of later psychiatric disorder. It is not clear whether such a process can account for Creamer et al.'s (1992) data, in which measures of memory intrusion taken 4 months after an office shooting were related to later outcome. This is because their raw correlations indicated that more intrusions related to a worse outcome, whereas structural equation modeling suggested the opposite relation.

Other predictors of severity. Many factors predictive of severity appear to relate to both of the aspects of emotional processing described in this article. Thus, the intensity of the stressor may impede both appraisal of the implications of the trauma, because of the greater discrepancy with prior assumptions, and the person's ability to tolerate flashbacks. Prior adversity and prior psychiatric disorder may affect both conscious and nonconscious appraisals of the degree of threat posed by the trauma. The content of SAMs is likely to be strongly influ-

enced by similarities between the content and context of current and prior adversity. Social support, as already indicated, may aid both in the reappraisal of the present and the future and make the occurrence of flashbacks more accepted and tolerable.

Of particular interest to the theory are reports that emotions such as guilt and anger predict the maintenance of PTSD symptoms (Riggs, Dancu, Gershuny, Greenberg, & Foa, 1992). This is consistent with the proposal that aversive secondary emotions interfere with habituation to trauma SAMs and are associated with more chronic emotional processing.

Comorbidity. According to this dual representation theory, PTSD is distinguished from other disorders by the existence and current levels of activation of trauma-related SAMs. Other disorders, such as depression, phobia, or generalized anxiety disorder, do not require that there be trauma-related memories, although these may be frequently present (whether currently more or less accessible). PTSD also requires that these memories are currently activated, as indicated by the presence of spontaneous intrusive phenomena or conscious efforts at avoidance, or both.

It seems likely that the theory can account for recent observations by Kuyken and Brewin (1994) and Brewin, Phillips, Carroll, and Tata (in press) that patients with a diagnosis of major depressive episode also experience frequent intrusive memories, particularly of past stressors, such as childhood trauma. Our theory requires that the processing of these events was prematurely inhibited, allowing emotional memories to remain dormant and to subsequently be reactivated by later depression or later life events. This is plausible in that numerous factors are likely to make it difficult for children to process abusive memories. Even if children disclose abuse (and there are often powerful pressures on them not to disclose), they may not be believed, and even if they are believed there may be no adult sufficiently knowledgeable, empathic, and responsible to facilitate this process.

There are many possible reasons for the high rate of comorbidity with other disorders. For example, familial aggregation of psychiatric disorders in the relatives of patients with PTSD may implicate more general biological and psychological vulnerabilities that are important in the genesis of PTSD (Jones & Barlow, 1990). Unlike other information-processing theories, however, dual representation theory offers additional specific suggestions to explain the high rate of comorbidity with other disorders. For example, depression may result as an emotional reaction to the actual or symbolic loss consequent to the trauma. This may consist of the loss of another person or type of relationship, a highly valued role, health, physical and mental capacities, a future goal, or a sense of a good and effective self. Loss of a belief in the self as good (e.g., caring, altruistic, moral) or effective (e.g., strong, self-controlled) may lead to depressions characterized by feelings of guilt or shame. Depression may also develop more slowly as a secondary reaction to prolonged emotional processing and the accompanying feelings of powerlessness and loss of mental capacity.

One of the well-known effects of depression is to enhance access to negative memories and to reduce the accessibility of positive memories (e.g., Brewin, Andrews, & Gotlib, 1993; Dalgleish & Watts, 1990). Teasdale (1983) has argued that the greater accessibility of negative memories further potentiates depression, leading to a vicious circle. It seems likely that depression preexisting a trauma could also operate on traumatic memories and tend to prolong emotional processing, resulting in more chronic PTSD.

According to the theory, initial anxiety reactions immediately posttrauma reflect both emotional reactions to continuing or subsequent threat and conditioned fear reactions contained within reactivated trauma SAMs. This double dose of anxiety, also noted by Janoff-Bulman (1992), results in extremely high levels of arousal, sleep disturbance, and startle responses, and may automatically elicit emotional numbing. These initial levels of anxiety generally begin to decline as the person develops strategies of cognitive avoidance to titrate their exposure to trauma cues.

After this initial period, anxiety reactions directed at trauma-related stimuli will usually signal unsuccessful emotional processing, whether chronic or prematurely inhibited. Whereas these reactions will tend to take the form of a phobia (or possibly obsessional thoughts or worrying) if the cues are avoidable, somatic anxiety and possibly panic may develop if the person is exposed to unavoidable trauma cues. Depending on the success with which trauma cues can be cognitively and behaviorally avoided, there may be an increased incidence of substance abuse as the person makes efforts to restore control over his or her mental processes.

Experimental findings. Dual representation theory accounts for the experimental findings using laboratory tasks by proposing that the content of both VAMs and SAMs is able to bias a wide range of cognitive processes. Perception and attention processes are biased toward information that is congruent with the individual's active plans, goals, and concerns (Dalgleish, 1994; Eysenck, 1992). According to dual representation theory such information concerning plans, goals, and concerns is represented as both VAMs and SAMs. Consequently, following a traumatic event, trauma-related information would be represented in both VAM and SAM formats, and both types of information would be able to drive perception and attention processes to information in the environment that is congruent with the trauma. Biases of perception and attention toward trauma-related information would therefore be expected in individuals suffering from PTSD (e.g., McNally et al., 1990).

Extant research using experimental tasks to investigate perception and memory has rarely distinguished between different groups of individuals who do not have PTSD. However, dual representation theory, unlike other cognitive theories of the disorder, would make differential predictions dependent on whether an individual had successfully integrated the traumatic event or had prematurely inhibited processing of the trauma. The theory would predict that those who had successfully integrated the traumatic experience would show no biases in perception and attention, as memory of the trauma would not be accompanied by high levels of emotion in either VAM or SAM. In contrast, for those individuals who have inhibited emotional processing prematurely, the memory of trauma would not be accompanied by high levels of emotion in VAM, but would be accompanied by high levels of emotion in SAM. In this case, dual representation theory would predict that these individuals

would persist in exhibiting biases of attention and perception for trauma-related information.

Dual representation theory proposes that judgment and memory biases for trauma-related information in individuals with PTSD on laboratory tasks (e.g., Dalgleish, 1994) are a function of the increased accessibility of such information in VAM. Individuals without PTSD, whether they had successfully integrated the trauma-related information or had prematurely inhibited processing, would not experience increased accessibility of trauma-related VAMs. However, those who had prematurely inhibited processing would experience decreased accessibility of trauma-related VAMs.

Predictions and Recommendations

Our dual representation theory makes a number of unique, testable predictions. Some of these have already been outlined: for example, the role of severity of trauma and time elapsed since the trauma in moderating the relations between measures of reexperiencing and subsequent psychiatric adjustment. We will discuss first those additional predictions that are connected with the distinction between VAMs and SAMs. According to the theory, SAMs contain a large amount of detailed information selected partly on criteria that are not available to conscious inspection. Like the implicit memories studied by cognitive psychologists, they cannot be deliberately retrieved and, in addition, are repetitive and difficult to modify. VAMs, on the other hand, like explicit memory, are less detailed and easy to edit. This leads to the prediction that verbal descriptions of the trauma should differ, depending on whether the corresponding SAMs have been activated. Verbal accounts that are unaccompanied by the subjective experience of intense fear or of reliving the trauma should be more variable and should contain less detail than verbal accounts accompanied by the sensation of reliving the trauma. Whereas the former should become more schematic and less specific over time, the latter should remain highly consistent, even after many years.

We would also predict that conditioned emotional reactions (flashbacks) could be elicited by aspects of the traumatic situation that were not part of survivors' initial verbal descriptions of the trauma. Flashbacks should also be more readily elicited by aspects of the traumatic situation prominent in SAMs than by aspects prominent only in VAMs.

A further prediction is that behaviorally based treatment simply involving exposure to the traumatic memories should be effective only at extinguishing emotional reactions, predominantly fear, experienced during the trauma itself. It should not extinguish secondary emotional reactions arising from subsequent conscious appraisal, which are not expected to habituate and should respond only to cognitive therapy techniques. Thus, exposure treatment should be more effective when recalling the trauma does not give rise to aversive secondary or complex emotions such as anger or guilt. According to the theory, if the trauma SAMs activated in therapy are repeatedly paired with aversive secondary emotions arising from conscious appraisal, there will be little or no overall reduction in negative affect and, hence, the habituation of fear will be blocked. The theory suggests, therefore, that aversive secondary emotions should be addressed using cognitive techniques (e.g., Resick & Schnicke, 1993) before exposure treatment is used. It is of interest in this regard that patients who reported more anger prior to therapy and displayed less fear during exposure treatment tended to have a poor outcome (Foa, Riggs, Massie, & Yarczower, 1995).

Other specific predictions relate to the idea that emotional processing may be prematurely inhibited. We predict that among people who have formerly had PTSD but now show no evidence of active emotional processing, there will be a substantial subgroup who (a) show a preattentional bias to attend to trauma-related stimuli; (b) show strong priming effects in response to such stimuli; (c) avoid elaborative processing of trauma-related stimuli, resulting in impaired memory for this material; (d) show phobic avoidance of trauma-related stimuli; (e) show enhanced sensitivity to life events; (f) report more dissociation at the time of the trauma; (g) have unrealistically positive assumptions and beliefs; and (h) show evidence of impaired health status.

The most important recommendation following from the theory is that the cessation of active emotional processing cannot be taken as a guarantee that successful integration of the trauma has taken place. Clinically, therefore, apparent improvement may be misleading. The theory implies a need for careful assessment of phobic avoidance and of attentional and memory biases before concluding treatment for PTSD. Equally, however, the theory implies that individuals entering treatment for other disorders may have inhibited the processing of traumatic events. A careful history of traumatic events should therefore be carried out in all cases, and evidence should be sought for successful or unsuccessful emotional processing. Once again, evidence of cognitive or behavioral avoidance may be a useful marker of incomplete processing.

The research implications are equally wide ranging. If correct, the theory implies that use of traditional outcome measures in naturalistic follow-up studies or treatment trials for PTSD may lead to serious underestimation of the extent of pathology and possible false conclusions concerning the efficacy of different therapeutic techniques. For example, some techniques may only be effective in inhibiting active emotional processing and giving the impression of improvement, rather than in leading to a complete integration of the trauma. Equally, prognostic factors thought to be associated with a good outcome may in fact be associated with premature inhibition of trauma processing. This may help to account for the failure to identify the same prognostic factors in different studies.

The theory would also caution against designing theoretical studies in which individuals with current PTSD are compared with individuals exposed to the same trauma but without current PTSD. This group could contain a mixture of individuals who had successfully completed emotional processing and others who had prematurely inhibited it. As each subgroup would be predicted to have different cognitive characteristics, misleading conclusions could be drawn. We suggest that individuals without current PTSD are screened for the presence of the indicators of incomplete processing described earlier.

A final recommendation concerns measures of intrusive memories. The theory suggests that there may be different types of memory, VAMs and SAMs, but existing scales such as the widely used Impact of Event Scale (Horowitz, Wilner, & Alvarez, 1979) do not capture this distinction. We suggest that it

may be profitable to examine the phenomenology of intrusive memories in more detail, with a view to developing more comprehensive measures.

Conclusions

In dual representation theory, PTSD is viewed as a particular type of unsuccessful adaptation to trauma. By emphasizing its relation to trauma processing in general, the theory attempts to build on the current understanding of the disorder offered by other theories and to highlight the overlap with other disorders. The two innovatory elements are a minimum cognitive architecture involving the distinction between verbally and situationally accessible knowledge and the idea of prematurely inhibited processing. We believe the first is necessary to explain the different types of memory associated with PTSD and to integrate existing social–cognitive and information-processing theories. The second idea also appears heuristic in several respects. Not only does it account for a range of clinical and experimental data and generate novel predictions, but it draws attention to the underlying connection between what may appear to be two unrelated therapeutic tasks. One task is to terminate the distress occasioned by the chronic processing of trauma found in PTSD. The other, often far less obvious, is to detect a history of trauma, encouraging the individual to overcome inhibitory processes and restart the painful processing of traumatic memories. We believe that a focus on trauma processing has the potential to forge interesting links between areas of clinical practice and research that have hitherto been regarded as separate.

References

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Atkeson, B., Calhoun, K., Resick, P., & Ellis, E. (1982). Victims of rape: Repeated assessment of depressive symptoms. *Journal of Consulting and Clinical Psychology, 50,* 96–102.

Bartone, P. T., Ursano, R. J., Wright, K. M., & Ingraham, L. H. (1989). Impact of a military air disaster on the health of assistance workers. *Journal of Nervous and Mental Disease, 177,* 317–328.

Blank, A. S. (1993). The longitudinal course of posttraumatic stress disorder. In J. R. T. Davidson & E. B. Foa (Eds.), *Posttraumatic stress disorder: DSM–IV and beyond* (pp. 3–22). Washington, DC: American Psychiatric Press.

Bouton, M. E., & Swartzentruber, D. (1991). Sources of relapse after extinction in Pavlovian and instrumental learning. *Clinical Psychology Review, 11,* 123–140.

Borkovec, T. D., & Lyonfields, J. D. (1993). Worry: Thought suppression of emotional processing. In H. W. Krohne (Ed.), *Attention and avoidance* (pp. 101–118). Toronto, Ontario, Canada: Hogrefe & Huber.

Bowlby, J. (1980). *Attachment and loss: Vol. 3. Loss: Sadness and depression.* London: Hogarth Press.

Breslau, N., Davis, G. C., Andreski, P., & Peterson, E. (1991). Traumatic events and posttraumatic stress disorder in an urban population of young adults. *Archives of General Psychiatry, 48,* 216–222.

Brewin, C. R. (1988). *Cognitive foundations of clinical psychology.* London: Erlbaum.

Brewin, C. R. (1989). Cognitive change processes in psychotherapy. *Psychological Review, 96,* 379–394.

Brewin, C. R., Andrews, B., & Gotlib, I. H. (1993). Psychopathology and early experience: A reappraisal of retrospective reports. *Psychological Bulletin, 113,* 82–98.

Brewin, C. R., Christodoulides, J., & Hutchinson, G. (1996). Intrusive thoughts and intrusive memories in a nonclinical sample. *Cognition and Emotion, 10,* 107–112.

Brewin, C. R., Phillips, E., Carroll, F., & Tata, P. (in press). Intrusive memories in depression. *Psychological Medicine.*

Broadbent, D. E., Fitzgerald, P., & Broadbent, M. H. P. (1986). Implicit and explicit knowledge in the control of complex systems. *British Journal of Psychology, 77,* 33–50.

Bromet, E. J., Parkinson, D. K., Schulberg, H. C., Dunn, L. O., & Gondek, P. C. (1982). Mental health of residents near the Three Mile Island reactor: A comparative study of selected groups. *Journal of Preventive Psychiatry, 1,* 225–274.

Brown, R., & Kulik, J. (1977). Flashbulb memories. *Cognition, 5,* 73–99.

Burgess, A. W., & Holmstrom, L. L. (1974). Rape trauma syndrome. *American Journal of Psychiatry, 131,* 981–986.

Burgess, A. W., & Holmstrom, L. L. (1978). Recovery from rape and prior life stress. *Research in Nursing and Health, 1,* 165–174.

Cassiday, K. L., McNally, R. J., & Zeitlin, S. B. (1992). Cognitive processing of trauma cues in rape victims with post-traumatic stress disorder. *Cognitive Therapy and Research, 16,* 283–295.

Chemtob, C., Roitblat, H. L., Hamada, R. S., Carlson, J. G., & Twentyman, C. T. (1988). A cognitive action theory of post-traumatic stress disorder. *Journal of Anxiety Disorders, 2,* 253–275.

Christianson, S.-Å., & Nilsson, L.-G. (1989). Hysterical amnesia: A case of aversively motivated isolation of memory. In T. Archer & L.-G. Nilsson (Eds.), *Aversion, avoidance, and anxiety: Perspectives on aversively motivated behavior* (pp. 289–310). Hillsdale, NJ: Erlbaum.

Cluss, P. A., Boughton, J., Frank, L. E., Stewart, B. D., & West, D. (1983). The rape victims: Psychological correlates of participation in the legal process. *Criminal Justice and Behaviour, 10,* 342–357.

Conway, M. A. (1995). *Flashbulb memories.* Hove, England: Erlbaum.

Conway, M. A. (1996). Autobiographical memories and autobiographical knowledge. In D. C. Rubin (Ed.), *Remembering our past: Studies in autobiographical memory* (pp. 67–93). Cambridge, England: Cambridge University Press.

Cook, J. D., & Bickman, L. (1990). Social support and psychological symptomatology following natural disaster. *Journal of Traumatic Stress, 3,* 541–556.

Creamer, M., Burgess, P., & Pattison, P. (1992). Reaction to trauma: A cognitive processing model. *Journal of Abnormal Psychology, 101,* 452–459.

Dalgleish, T. (1993). *The judgement of risk in traumatised and non-traumatised disaster survivors.* Unpublished masters thesis, University of London.

Dalgleish, T. (1994). The appraisal of threat and the process of selective attention in clinical and sub-clinical anxiety states: I. Theoretical issues. *Clinical Psychology and Psychotherapy, 1,* 153–164.

Dalgleish, T., & Watts, F. N. (1990). Biases of attention and memory in disorders of anxiety and depression. *Clinical Psychology Review, 10,* 589–604.

Davey, G. C. L. (1989). UCS revaluation and conditioning models of acquired fear. *Behaviour Research and Therapy, 27,* 521–528.

Davidson, J. R. T., & Foa, E. B. (1991). Diagnostic issues in posttraumatic stress disorder: Considerations for the *DSM–IV. Journal of Abnormal Psychology, 100,* 346–355.

Davidson, J. R. T., Hughes, D., Blazer, D. G., & George, L. K. (1991). Posttraumatic stress disorder in the community: An epidemiological study. *Psychological Medicine, 21,* 713–721.

Davidson, J. R. T., Kudler, H. S., Saunders, W. B., & Smith, R. D.

(1990). Symptom and comorbidity patterns in World War II and Vietnam veterans with posttraumatic stress disorder. *Comprehensive Psychiatry, 31,* 162–170.

Ehlers, A., & Steil, R. (1995). Maintenance of intrusive memories in posttraumatic stress disorder: A cognitive approach. *Behavioural and Cognitive Psychotherapy, 23,* 217–249.

Ellis, E. M., Atkeson, B. M., & Calhoun, K. S. (1981). An assessment of long-term reaction to rape. *Journal of Abnormal Psychology, 90,* 263–266.

Epstein, S. (1994). Integration of the cognitive and the psychodynamic unconscious. *American Psychologist, 49,* 709–724.

Eysenck, M. W. (1992). *Anxiety: The cognitive perspective.* Hove, England: Erlbaum.

Eysenck, M. W., & Keane, T. M. (1990). *Cognitive psychology.* Hove, England: Erlbaum.

Fairbank, J. A., & Brown, T. A. (1987). Current behavioral approaches to the treatment of posttraumatic stress disorder. *The Behavior Therapist, 3,* 57–64.

Farmer, R., Tranah, T., O'Donnell, I., & Catalan, J. (1992). Railway suicide: The psychological effects on drivers. *Psychological Medicine, 22,* 407–414.

Fiske, S. T., & Taylor, S. E. (1991). *Social cognition* (2nd ed.). New York: McGraw-Hill.

Fleming, R., Baum, A., Gisriel, M., & Gatchel, R. (1982). Mediating influences of social support on stress at Three Mile Island. *Journal of Human Stress, 8,* 14–22.

Foa, E. B., Feske, U., Murdock, T. B., Kozak, M. J., & McCarthy, P. R. (1991). Processing of threat-related material in rape victims. *Journal of Abnormal Psychology, 100,* 156–162.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99,* 20–35.

Foa, E. B., & Riggs, D. S. (1993). Post-traumatic stress disorder in rape victims. In J. Oldham, M. B. Riba, & A. Tasman (Eds.), *American Psychiatric Press review of psychiatry* (Vol. 12, pp. 273–303). Washington, DC: American Psychiatric Press.

Foa, E. B., Riggs, D. S., Massie, E. D., & Yarczower, M. (1995). The impact of fear activation and anger on the efficacy of exposure treatment for posttraumatic stress disorder. *Behavior Therapy, 26,* 487–499.

Foa, E. B., Steketee, G., & Rothbaum, B. O. (1989). Behavioral/cognitive conceptualization of post-traumatic stress disorder. *Behavior Therapy, 20,* 155–176.

Foa, E. B., Zinbarg, R., & Rothbaum, B. O. (1992). Uncontrollability and unpredictability in posttraumatic stress disorder: An animal model. *Psychological Bulletin, 112,* 218–238.

Fontana, A., Rosenheck, R., & Brett, E. (1992). War zone traumas and posttraumatic stress disorder symptomatology. *Journal of Nervous and Mental Disease, 180,* 748–755.

Fox, E. (1993). Allocation of visual attention and anxiety. *Cognition and Emotion, 7,* 207–215.

Foy, D. W., Resnick, H. S., Sipprelle, R. C., & Carroll, E. M. (1987). Premilitary, military, and postmilitary factors in the development of combat-related stress disorders. *The Behavior Therapist, 10,* 3–9.

Foy, D. W., Sipprelle, R. C., Rueger, D. B., & Carroll, E. M. (1984). Etiology of posttraumatic stress disorder in Vietnam veterans: Analysis of premilitary, military, and combat exposure influences. *Journal of Consulting and Clinical Psychology, 52,* 79–87.

Frank, E., & Anderson, B. P. (1987). Psychiatric disorders in rape victims: Past history and current symptomatology. *Comprehensive Psychiatry, 28,* 77–82.

Frank, E., Turner, S. M., Stewart, B. D., Jacob, M., & West, D. (1981). Past psychiatric symptoms and the response of sexual assault. *Comprehensive Psychiatry, 22,* 479–487.

Freud, S. (1919). *Introduction to the psychology of the war neuroses* (Standard ed., Vol. 18). London: Hogarth Press.

Freud, S. (1920). *Beyond the pleasure principle* (Standard ed., Vol. 18). London: Hogarth Press.

Frye, J., & Stockton, R. A. (1982). Discriminant analysis of posttraumatic stress disorder among a group of Vietnam veterans. *American Journal of Psychiatry, 139,* 52–56.

Gilbert, P. (1989). *Human nature and suffering.* Hove, England: Erlbaum.

Gleser, G. C., Green, B. L., & Winget, C. N. (1981). *Prolonged psychosocial effects of disaster.* New York: Academic Press.

Gray, J. A. (1982). *The neuropsychology of anxiety.* Oxford, England: Oxford University Press.

Green, B. L. (1994). Psychosocial research in traumatic stress: An update. *Journal of Traumatic Stress, 7,* 341–362.

Green, B. L., Grace, M. C., & Gleser, C. G. (1985). Identifying survivors at risk: Long-term impairment following the Beverly Hills Supper Club fire. *Journal of Consulting and Clinical Psychology, 53,* 672–678.

Green, B. L., Grace, M. C., Lindy, J. D., Tichener, J. L., & Lindy, J. G. (1983). Levels of functional impairment following a civilian disaster: The Beverly Hills Supper Club Fire. *Journal of Consulting and Clinical Psychology, 51,* 573–580.

Hashtroudi, S., Johnson, M. K., Vnek, N., & Ferguson, S. A. (1994). Aging and the effects of affective and factual focus on source monitoring and recall. *Psychology and Aging, 9,* 160–170.

Helzer, J. E., Robins, L. N., & McEvoy, L. (1987). Post-traumatic stress disorder in the general population. *New England Journal of Medicine, 317,* 1630–1634.

Herman, J. L. (1992a). Complex PTSD: A syndrome in survivors of prolonged and repeated trauma. *Journal of Traumatic Stress, 5,* 377–391.

Herman, J. L. (1992b). *Trauma and recovery.* New York: Basic Books.

Hierholzer, R., Munson, J., Peabody, C., & Rosenberg, J. (1992). Clinical presentation of PTSD in World War II combat veterans. *Hospital and Community Psychiatry, 43,* 816–820.

Horowitz, M. J. (1973). Phase-oriented treatment of stress response syndromes. *American Journal of Psychotherapy, 27,* 506–515.

Horowitz, M. J. (1976). *Stress response syndromes.* New York: Jason Aronson.

Horowitz, M. J. (1979). Psychological response to serious life events. In V. Hamilton & D. M. Warburton (Eds.), *Human stress and cognition* (pp. 235–263). New York: Wiley.

Horowitz, M. J. (1986). *Stress response syndromes* (2nd ed.). New York: Jason Aronson.

Horowitz, M. J. (1990). A model of mourning: Changes in schemas of self and others. *Journal of the American Psychoanalytic Association, 38,* 297–324.

Horowitz, M. J., Wilner, N., & Alvarez, W. (1979). Impact of Event scale: A measure of subjective stress. *Psychosomatic Medicine, 41,* 209–218.

Horowitz, M. J., Wilner, N., Kaltreider, N., & Alvarez, W. (1980). Signs and symptoms of post-traumatic stress disorder. *Archives of General Psychiatry, 37,* 85–92.

Jacobs, W. J., & Nadel, L. (1985). Stress-induced recovery of fears and phobias. *Psychological Review, 92,* 512–531.

Janet, P. (1889). *L'automatisme psychologique.* Paris: Alcan.

Janoff-Bulman, R. (1985). The aftermath of victimization: Rebuilding shattered assumptions. In C. R. Figley (Ed.), *Trauma and its wake: The study and treatment of post-traumatic stress disorder* (pp. 15–35). New York: Brunner/Mazel.

Janoff-Bulman, R. (1992). *Shattered assumptions: Towards a new psychology of trauma.* New York: Free Press.

Janoff-Bulman, R., & Frieze, I. H. (1983). A theoretical perspective for

understanding reactions to victimization. *Journal of Social Issues, 39,* 1–17.

Johnson, M. K., & Multhaup, K. S. (1992). Emotion and MEM. In S.-Å. Christianson (Ed.), *Handbook of emotion and memory* (pp. 33–66). Hillsdale, NJ: Erlbaum.

Jones, J. C., & Barlow, D. H. (1990). The etiology of post-traumatic stress disorder. *Clinical Psychology Review, 10,* 299–328.

Joseph, S. A., Andrews, B., Williams, R., & Yule, W. (1992). Crisis support and psychiatric symptomatology in adult survivors of the Jupiter cruise ship disaster. *British Journal of Clinical Psychology, 31,* 63–74.

Joseph, S. A., Brewin, C. R., Yule, W., & Williams, R. (1991). Causal attributions and psychiatric symptoms in survivors of the Herald of Free Enterprise disaster. *British Journal of Psychiatry, 159,* 542–546.

Joseph, S. A., Brewin, C. R., Yule, W., & Williams, R. (1993). Causal attributions and post-traumatic stress in children. *Journal of Child Psychology and Psychiatry, 34,* 247–253.

Joseph, S. A., Yule, W., & Williams, R. (1994). The Herald of Free Enterprise disaster: The relationship of intrusion and avoidance to subsequent depression and anxiety. *Behaviour Research and Therapy, 32,* 115–117.

Joseph, S. A., Yule, W., Williams, R., & Andrews, B. (1993). Crisis support in the aftermath of disaster: A longitudinal perspective. *British Journal of Clinical Psychology, 32,* 177–185.

Joseph, S. A., Yule, W., Williams, R., & Hodgkinson, P. (1994). The Herald of Free Enterprise disaster: Correlates of distress at thirty months. *Behaviour Research and Therapy, 32,* 521–524.

Kardiner, A. (1941). *The traumatic neuroses of war.* New York: Hoeber.

Kaspi, S. P., McNally, R. J., & Amir, N. (1995). Cognitive processing of emotional information in post-traumatic stress disorder. *Cognitive Therapy and Research, 19,* 433–444.

Keane, T. M., Fairbank, J. A., Caddell, R. T., Zimering, R. T., & Bender, M. E. (1985). A behavioral approach to assessing and treating PTSD in Vietnam veterans. In C. R. Figley (Ed.), *Trauma and its wake* (pp. 257–294). New York: Brunner/Mazel.

Keane, T. M., Scott, W. O., Chavoya, G. A., Lamparski, D. M., & Fairbank, J. A. (1985). Social support in Vietnam veterans: A comparative analysis. *Journal of Consulting and Clinical Psychology, 53,* 95–102.

Keane, T. M., Zimering, R. T., & Caddell, R. T. (1985). A behavioral formulation of PTSD in Vietnam veterans. *The Behavior Therapist, 8,* 9–12.

Kilpatrick, D. G., Veronen, L. J., & Best, C. L. (1985). Factors predicting psychological distress among rape victims. In C. R. Figley (Ed.), *Trauma and its wake* (pp. 113–141). New York: Brunner/Mazel.

Klinger, E. (1975). Consequences of commitment to and disengagement from incentives. *Psychological Review, 82,* 1–25.

Kolb, L. C. (1987). A neuropsychological hypothesis explaining post-traumatic stress disorder. *American Journal of Psychiatry, 144,* 989–995.

Krystal, H. (Ed.). (1968). *Massive psychic trauma.* New York: International Universities Press.

Kuch, K., & Cox, B. J. (1992). Symptoms of PTSD in 124 survivors of the Holocaust. *American Journal of Psychiatry, 149,* 337–340.

Kulka, R. A., Schlenger, W. E., Fairbank, J. A., Hough, R. L., Jordon, B. K., Marmar, C. R., & Weiss, D. S. (1990). *Trauma and the Vietnam war generation: Report of findings from the National Vietnam Veterans Readjustment Study.* New York: Brunner/Mazel.

Kuyken, W., & Brewin, C. R. (1994). Intrusive memories of childhood abuse during depressive episodes. *Behaviour Research and Therapy, 32,* 525–528.

Lang, P. J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology, 16,* 495–512.

Lang, P. J. (1985). The cognitive psychophysiology of emotion: Fear and anxiety. In A. H. Tuma & J. D. Maser (Eds.), *Anxiety and the anxiety disorders* (pp. 131–170). Hillsdale, NJ: Erlbaum.

Lazarus, R. S., & Launier, R. (1978). Stress-related transactions between person and environment. In L. A. Pervin (Ed.), *Perspectives in interactional psychology* (pp. 287–327). New York: Plenum.

LeDoux, J. E. (1992). Emotion as memory: Anatomical systems underlying indelible neural traces. In S.-Å. Christianson (Ed.), *Handbook of emotion and memory* (pp. 269–288). Hillsdale, NJ: Erlbaum.

Leventhal, H. (1984). A perceptual-motor theory of emotion. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 17, pp. 117–182). Orlando, FL: Academic Press.

Litz, B. T., & Keane, T. M. (1989). Information processing in anxiety disorders: Application to the understanding of post-traumatic stress disorder. *Clinical Psychology Review, 9,* 243–257.

MacLeod, C. (1990). Mood disorders and cognition. In M. W. Eysenck (Ed.), *Cognitive psychology: An international review* (pp. 9–56). Chichester, England: Wiley.

Madakasira, S., & O'Brien, K. F. (1987). Acute posttraumatic stress disorder in victims of a natural disaster. *Journal of Nervous and Mental Disease, 175,* 286–290.

Maddison, D. C., & Viola, A. (1968). The health of widows in the year following bereavement. *Journal of Psychosomatic Research, 12,* 297–306.

Maida, C. A., Gordon, N. S., Steinberg, A., & Gordon, G. (1989). Psychosocial impact of disasters: Victims of the Baldwin fire. *Journal of Traumatic Stress, 2,* 37–48.

McCormick, R. A., Taber, J. I., & Kruedelbach, N. (1989). The relationship between attributional style and post-traumatic stress disorder in addicted patients. *Journal of Traumatic Stress, 2,* 477–487.

McFarlane, A. C. (1988). The longitudinal course of post-traumatic morbidity: The range of outcomes and their predictors. *Journal of Nervous and Mental Disease, 176,* 30–39.

McFarlane, A. C. (1989). The aetiology of post-traumatic morbidity: Predisposing, precipitating, and perpetuating factors. *British Journal of Psychiatry, 154,* 221–228.

McFarlane, A. C. (1992). Avoidance and intrusion in post-traumatic stress disorder. *Journal of Nervous and Mental Disease, 180,* 439–445.

McNally, R. J. (1992). Psychopathology of post-traumatic stress disorder (PTSD): Boundaries of the syndrome. In M. Başoğlu (Ed.), *Torture and its consequences: Current treatment approaches* (pp. 229–252). Cambridge, England: Cambridge University Press.

McNally, R. J., English, G. E., & Lipke, H. J. (1993). Assessment of intrusive cognition in PTSD: Use of the modified Stroop paradigm. *Journal of Traumatic Stress, 6,* 33–41.

McNally, R. J., Kaspi, S. P., Riemann, B. C., & Zeitlin, S. B. (1990). Selective processing of threat cues in post-traumatic stress disorder. *Journal of Abnormal Psychology, 99,* 398–402.

McNally, R. J., Lasko, N. B., Macklin, M. L., & Pitman, R. K. (1995). Autobiographical memory disturbance in combat-related posttraumatic stress disorder. *Behaviour Research and Therapy, 33,* 619–630.

McNally, R. J., Litz, B. T., Prassas, A., Shin, L. M., & Weathers, F. W. (1994). Emotional priming of autobiographical memory in post-traumatic stress disorder. *Cognition and Emotion, 8,* 351–368.

McNally, R. J., Luedke, D. L., Beysner, J. K., Peterson, R. A., Bohm, K., & Lips, O. J. (1987). Sensitivity to stress-relevant stimuli in post-traumatic stress disorder. *Journal of Anxiety Disorders, 1,* 105–116.

Mikulincer, M., & Solomon, Z. (1988). Attributional style and combat-related posttraumatic stress disorder. *Journal of Abnormal Psychology, 97,* 308–313.

Myers, L. B., & Brewin, C. R. (1994). Recall of early experience and the repressive coping style. *Journal of Abnormal Psychology, 103,* 288–292.

Myers, L. B., & Brewin, C. R. (1995). Repressive coping and the recall of emotional material. *Cognition and Emotion, 9,* 637–642.

Myers, L. B., & Brewin, C. R. (in press). Illusions of well-being and the repressive coping style. *British Journal of Social Psychology.*

Oatley, K., & Johnson-Laird, P. N. (1987). Towards a cognitive theory of emotions. *Cognition and Emotion, 1,* 29–50.

Ørner, R. J., Lynch, T., & Seed, P. (1993). Long-term traumatic stress reactions in British Falklands war veterans. *British Journal of Clinical Psychology, 32,* 457–459.

Parkes, C. M. (1970). The first year of bereavement: A longitudinal study of the reaction of London widows to the death of their husbands. *Psychiatry, 33,* 444–467.

Parkes, C. M. (1971). Psychosocial transitions: A field for study. *Social Science and Medicine, 5,* 101–115.

Parkes, C. M. (1986). *Bereavement: Studies in grief in adult life* (2nd ed.). London: Tavistock.

Pennebaker, J. W., Kiecolt-Glaser, J. K., & Glaser, R. (1988). Disclosure of traumas and immune function: Health implications for psychotherapy. *Journal of Consulting and Clinical Psychology, 56,* 239–245.

Pynoos, R. S., & Nader, K. (1988). Psychological first aid and treatment approach for children exposed to community violence: Research implications. *Journal of Traumatic Stress, 1,* 243–267.

Rachman, S. (1980). Emotional processing. *Behaviour Research and Therapy, 18,* 51–60.

Rees, W. D. (1975). The bereaved and their hallucinations. In B. Schoenberg, I. Gerber, A. Wiener, A. H. Kutscher, D. Peretz, & A. C. Carr (Eds.), *Bereavement: Its psychosocial aspects* (pp. 66–71). New York: Columbia University Press.

Resick, P. A., & Schnicke, M. K. (1993). *Cognitive processing therapy for rape victims.* Newbury Park, CA: Sage.

Resnick, H. S., Kilpatrick, D. G., Best, C. L., & Kramer, T. L. (1992). Vulnerability–stress factors in development of posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 180,* 424–430.

Riggs, D. S., Dancu, C. V., Gershuny, B. S., Greenberg, D., & Foa, E. B. (1992). Anger and post-traumatic stress disorder in female crime victims. *Journal of Traumatic Stress, 5,* 613–625.

Roth, S., Wayland, K., & Woolsey, M. (1990). Victimization history and victim–assailant relationships as factors in recovery from sexual assault. *Journal of Traumatic Stress, 3,* 169–180.

Rothbaum, B. O., & Foa, E. B. (1993). Subtypes of post-traumatic stress disorder and duration of symptoms. In J. R. T. Davidson & E. B. Foa (Eds.), *Posttraumatic stress disorder: DSM–IV and beyond* (pp. 23–35). Washington, DC: American Psychiatric Press.

Ruch, L. O., Chandler, S. M., & Harter, R. A. (1980). Life change and rape impact. *Journal of Health and Social Behavior, 21,* 248–260.

Ruch, L. O., & Leon, J. J. (1983). Sexual assault trauma and trauma change. *Women and Health, 8,* 5–21.

Salkovskis, P. M. (1985). Obsessional–compulsive problems: A cognitive–behavioural analysis. *Behaviour Research and Therapy, 23,* 571–583.

Scott, M. J., & Stradling, S. G. (1994). Post-traumatic stress disorder without the trauma. *British Journal of Clinical Psychology, 33,* 71–74.

Shalev, A. Y. (1992). Posttraumatic stress disorder among injured survivors of a terrorist attack. *Journal of Nervous and Mental Disease, 180,* 505–509.

Shontz, F. C. (1965). Reactions to crisis. *The Volta Review, 67,* 364–370.

Shore, J. H., Tatum, E. L., & Vollmer, W. M. (1986). Psychiatric reactions to disaster: The Mount St. Helens experience. *American Journal of Psychiatry, 143,* 590–595.

Shore, J. H., Vollmer, W. M., & Tatum, E. L. (1989). Community patterns of posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 177,* 681–685.

Smith, E. M., Robins, L. N., Pryzbeck, T. R., Goldring, E., & Solomon, S. D. (1986). Psychosocial consequences of a disaster. In J. Shore (Ed.), *Disaster stress studies: New methods and findings* (pp. 49–76). Washington, DC: American Psychiatric Press.

Solkoff, N., Gray, P., & Keill, S. (1986). Which Vietnam veterans develop posttraumatic stress disorders? *Journal of Clinical Psychology, 42,* 687–698.

Solomon, Z., Mikulincer, M., & Benbenishty, R. (1989). Locus of control and combat-related post-traumatic stress disorder: The intervening role of battle intensity, threat appraisal, and coping. *British Journal of Clinical Psychology, 28,* 131–144.

Speed, N., Engdahl, B., Schwartz, J., & Eberly, R. (1989). Post-traumatic stress disorder as a consequence of the POW experience. *Journal of Nervous and Mental Disease, 177,* 147–153.

Stiles, W. B., Elliott, R., Llewelyn, S. P., Firth-Cozens, J. A., Margison, F. R., Shapiro, D. A., & Hardy, G. (1990). Assimilation of problematic experiences by clients in psychotherapy. *Psychotherapy, 27,* 411–420.

Stroop, J. R. (1935). Studies of interference in serial verbal reaction. *Journal of Experimental Psychology, 18,* 643–662.

Teasdale, J. D. (1983). Negative thinking in depression: Cause, effect, or reciprocal relationship? *Advances in Behaviour Research and Therapy, 5,* 3–25.

Teasdale, J. D., & Barnard, P. J. (1993). *Affect, cognition, and change: Re-modelling depressive thought.* Hove, England: Erlbaum.

Terr, L. C. (1991). Childhood traumas: An outline and overview. *American Journal of Psychiatry, 148,* 10–20.

Thrasher, S. M., Dalgleish, T., & Yule, W. (1994). Information processing in post-traumatic stress disorder. *Behaviour Research and Therapy, 32,* 247–254.

Tulving, E., & Schacter, D. L. (1990). Priming and human memory systems. *Science, 247,* 301–306.

Van der Kolk, B. A., Boyd, H., Krystal, J., & Greenburg, M. (1984). Post-traumatic stress disorder as a biologically based disorder: Implications of the animal model of inescapable shock. In B. A. Van der Kolk (Ed.), *Post-traumatic stress disorder: Psychological and biological sequelae* (pp. 124–134). Washington, DC: American Psychiatric Press.

Van der Kolk, B. A., & Fisler, R. E. (1994). Childhood abuse and neglect and loss of self-regulation. *Bulletin of the Menninger Clinic, 58,* 145–168.

Van der Kolk, B. A., & Fisler, R. E. (1995). Dissociation and the fragmentary nature of traumatic memories: Overview and exploratory study. *Journal of Traumatic Stress, 8,* 505–525.

Vrana, S. R., Roodman, A., & Beckham, J. C. (1995). Selective processing of trauma-relevant words in posttraumatic stress disorder. *Journal of Anxiety Disorders, 9,* 515–530.

Weiner, B. (1985). *An attributional theory of motivation and emotion.* New York: Springer-Verlag.

Williams, J. M. G. (1992). Autobiographical memory and emotional disorders. In S.-Å. Christianson (Ed.), *Handbook of emotion and memory* (pp. 451–477). Hillsdale, NJ: Erlbaum.

Worden, J. W. (1982). *Grief counselling and grief therapy.* London: Tavistock.

Yehuda, R., Southwick, S. M., & Giller, E. L. (1992). Exposure to atrocities and severity of chronic posttraumatic stress disorder in Vietnam combat veterans. *American Journal of Psychiatry, 149,* 333–336.

Zeiss, R., & Dickman, H. (1989). PTSD 40 years later: Incidence and person–situation correlates in former POWs. *Journal of Clinical Psychology, 45,* 80–87.

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