Behavior Modification
Volume 31 Number 1
January 2007 80-116
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10.1177/0145445506295057
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A Critical Analysis of Approaches to Targeted PTSD Prevention

Current Status and Theoretically Derived Future Directions

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Although efforts to prevent posttraumatic stress disorder (PTSD) have met with relatively limited success, theoretically driven preventive approaches with promising efficacy are emerging. The current article critically reviews investigations of PTSD prevention programs that target persons at risk for being exposed to a traumatic event or who have been exposed to a traumatic event. This review uniquely extends prior reviews in this area by using theories of PTSD to suggest future directions in the area of PTSD prevention. The authors first discuss the primary mechanisms of action believed to account for the failure for PTSD symptoms to remit among a substantial minority of traumatic event–exposed individuals. Second, empirical progress in PTSD prevention efforts is reviewed. Third, the authors consider how existing prevention programs target these mechanisms of action. Finally, the authors consider directions for future research in the area of targeted PTSD prevention.

Keywords: *posttraumatic stress disorder; trauma; prevention; risk*

Posttraumatic stress disorder (PTSD) is a common (Kessler, Berglund, Demler, Jin, & Walters, 2005), often chronic (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995) and debilitating (Zatzick et al., 1997) condition. Although efficacious treatments for PTSD have been developed (Bradley, Greene, Russ, Dutra, & Westen, 2005; McDonagh et al., 2005; Resick, Nishith, Weaver, Astin, & Feuer, 2002) and continue to be modified (McFall et al., 2005; Wald & Taylor, 2005), there has been comparably less

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progress in the domain of PTSD prevention. The current article reviews tests of PTSD prevention programs aiming to reduce the incidence of PTSD among persons exposed to a traumatic event in terms of theories of the pathogenesis of PTSD. This discussion uniquely extends prior reviews in this area by using theories of PTSD to suggest future directions in the area of PTSD prevention. By doing so, the current discussion highlights several novel programs of research that have the potential to significantly advance our ability to reduce the likelihood of PTSD among trauma-exposed individuals. In line with these overarching aims, we first discuss the primary mechanisms of action that cut across multiple theoretical perspectives on why posttraumatic symptoms fail to remit among a substantial minority of trauma-exposed individuals. Second, empirical progress in targeted PTSD prevention is reviewed. Third, we consider how these empirical tests may (or may not) target these mechanisms of action. Finally, we consider several potential directions for future investigation in the area of PTSD prevention based on contemporary theories of PTSD etiology. Figure 1 provides a graphic depiction of this organization.

First, it is critical to define what constitutes prevention. The Institute of Medicine (IOM) has outlined a taxonomy designed to delimit what falls under the purview of prevention, because traditional typologies (i.e., primary, secondary, and tertiary) encompass all intervention research, including what can be considered risk factor and treatment outcome research (Mrazek & Haggerty, 1994). Noting that, as such, this traditional typology risks making the concept of prevention so inclusive it has little meaning, it was suggested that prevention exclusively focus on reducing the *incidence* (rather than prevalence) of a disorder. With this in mind, three categories of prevention programs were outlined. A *universal* intervention is applied to all members of the population, regardless of their risk for developing a disorder. A *selective* intervention targets only persons at risk for developing, but showing no signs of, a disorder. An *indicated* intervention is aimed at individuals demonstrating aspects of a disorder but who are subsyndromal

Authors' Note: This article was supported by an Arkansas Biosciences Institute grant awarded to Dr. Feldner, and portions of this article were presented at the First Annual Winter Anxiety Summit. The authors would like to thank (in alphabetical order) participants of the summit, including, but not necessarily limited to, David Barlow, Chris Hayward, Ellen Leen-Feldner, Carl Lejuez, Brad Schmidt, Jasper Smits, Sherry Stewart, Carl Weems, Richard Zinbarg, and Michael Zvolensky, for their helpful comments during discussion of a subset of the ideas presented herein. The authors also would like to thank Mark Bouton and members of both the Executive Division of the National Center for PTSD and the Anxiety and Health Research Laboratory for their input at various stages of this project.

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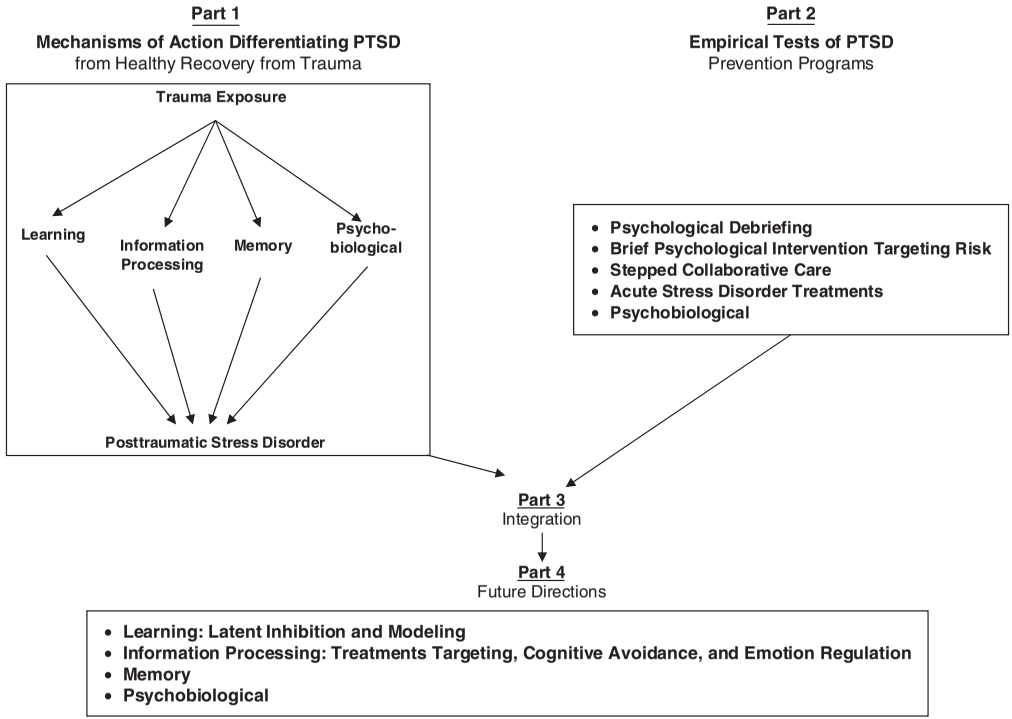
Figure 1
Graphical Overview of the Organizational Structure of the Current Discussion



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or subclinical in terms of diagnosis. Studies selected for review in the current article met the IOM's system explicit focus on risk processes, as opposed to disorder prevalence and related disability. As such, the approach corresponds well with the current discussion's focus on applying our understanding of risk processes to the development of novel theoretically informed approaches to improving posttraumatic event outcomes among high-risk groups.

Suspected Mechanisms of Action Differentiating Healthy Recovery From Exposure to a Traumatic Event and PTSD

Theories of PTSD seek to explain the failure to cope successfully with traumatic stress as well as the failure for posttraumatic symptoms to remit among a substantial minority of individuals exposed to traumatic events (Kessler et al., 1995). Core mechanisms of action involved in such a failure to recover that are explicated in these theories will now be considered in order to draw out potentially fruitful targets for preventive interventions. See Brewin and Holmes (2003) for a more detailed review.

Learning

One process thought to be critical to PTSD is learning. Two key components have been emphasized in these theories: learning and related avoidance that maintains the learning (Keane, Zimering, & Caddell, 1985). In terms of conditioning, fear-based responses are theorized to become associated with traumatic event–related cues during the event (Mineka & Zinbarg, 2006). Emotion network theorists have drawn on Lang's bio-informational theory of emotion-relevant learning (e.g., P. J. Lang, 1979) to suggest that associations learned during a traumatic event form a "fear network" (Foa & Rothbaum, 1998). A network of associations is formed, consisting of information about (a) the stimulus; (b) verbal, physiological, and overt behavioral responses; and (c) interpretation of the meaning of the stimulus and response elements. Activation of such fear networks, via exposure to traumatic event cues, initiates a "program" to escape danger. These fear networks become pathological, as in PTSD, when there are excessive response elements that (a) are relatively resistant to change (Foa & Kozak, 1986) and (b) persist in the presence of realistically nondangerous cues. Traumatic event–related learning is theorized to be particularly robust (Ehlers & Clark,

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2000; Pitman, Shalev, & Orr, 2000), thereby accounting for certain symptoms of PTSD, such as chronic reexperiencing and stimulus generalization. Learning to fear internal cues (interoceptive conditioning), such as a racing heart, has been highlighted as a potentially important association developed during traumatic event exposure (Jones & Barlow, 1990). Specifically, interoceptive cues become conditioned stimuli that may elicit traumatic event–related responses, such as fear and memories of the event. In terms of the avoidance component of learning theories, it is thought that fear responses conditioned during a traumatic event elicit subsequent anxious apprehension about experiencing a similar emotional upset and associated avoidance of traumatic event–relevant cues, which functions to maintain the conditioned fear (Bouton & Waddell, in press; Keane et al., 1985). Specifically, behavioral avoidance prevents exposure to traumatic event cues, which prevents the extinction of the learned fear.

Information Processing

Learning-based accounts of PTSD have been complemented by information processing theories about the cognitive structures and processes involved in the origins and maintenance of PTSD. Social–cognitive theories have focused in part on the content of these structures and processes (i.e., attributions, cognitions, schema content). Horowitz (1986) initially argued that there is a "completion tendency" in which traumatic event–exposed individuals wish to integrate new information into their existing beliefs and/or schemas about one's self, others, and the world. When this information is not incorporated, the memory is thought to remain in "active memory" and manifest in PTSD symptoms. In a related vein, Janoff-Bulman (1992) theorized that traumatization results in the "shattering" of assumptions about the world, including the "just world" belief. McCann and Pearlman (1990) also outline cognitive content relevant to PTSD, including beliefs about safety, trust, power and/or control, intimacy, and esteem. A few theorists have brought together the above implicated cognitive content and processes to yield more unified information processing theories of PTSD. For instance, Brewin, Dalgleish, and Joseph (1996) argue that there are more and less effortful cognitive dimensions that relate to meaning-making versus relatively automatically activated conditioned responses. Here it also is suggested that emotion processing may become chronic, resulting in preoccupation with traumatic-related information (e.g., memories) or be prematurely inhibited, resulting in vulnerability to reactivation of traumatic event–related memory. Ehlers and Clark (2000) also offer this type of

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unified theory of PTSD that emphasizes *problematic processing* of traumatic event–related information, wherein problematic appraisals that individuals with PTSD make about events and their own PTSD symptoms (e.g., catastrophic misinterpretation) are highlighted.

Memory

Traumatic event–related memory formation also has been highlighted in both psychological (e.g., Brewin et al., 1996; Ehlers & Clark, 2000; Foa, Steketee, & Rothbaum, 1989; Spiegel, 1996) and psychobiological (e.g., Pitman, 1989) conceptualizations of PTSD. Specifically, traumatic experiences are believed to result in disorganized and fragmented memories of the event. Situationally encoded memories are thought to be overly accessible due to strong associations with traumatic event cues. Verbally accessible memories are often fragmented and difficult to recall (Brewin et al., 1996), which is thought to be due, at least in part, to high levels of peri- and posttraumatic arousal (McCleery & Harvey, 2004). That is, high levels of arousal *during* a traumatic event results in vivid memories that are highly resistant to forgetting, despite their fragmented nature, and *posttraumatic* arousal results in overconsolidation of traumatic event–related memories, thereby yielding abnormally strong traces to these memories and chronic reexperiencing (McCleery & Harvey, 2004). This contention has been supported by evidence of the memory-enhancing effects of arousal as well as PTSD treatment resulting in defragmentation of memory. Brewin and colleagues (1996) also have highlighted differential retrieval processes involved with situationally versus verbally accessible memory systems. Situationally accessible memories that are vividly remembered are argued to be unavailable to voluntary recall but hyperaccessible to contextual cues (e.g., mood). Verbally accessible memories, which are believed to be encoded in a separate memory system, are available to voluntary recall, yet are fragmented. Finally, inadequate elaboration of memories of a traumatic event and poor integration of them into autobiographical memory has been theorized as critical to PTSD (Ehlers & Clark, 2000).

Psychobiology

Multiple distinct, yet perhaps not mutually exclusive, mechanisms of action involved in PTSD have been suggested in the psychobiological domain. The major neurobiological system within which to understand adaptive and maladaptive responses to stress involves the amygdala, hippocampus, and

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medial prefrontal cortex (mPFC). The amygdala processes the emotional information associated with traumatic event exposure, the hippocampus contextualizes such information, and the mPFC is the key structure that can inhibit excessive amygdala activation associated with acute and chronic stress reactions. Once this circuitry is activated, corticotropin releasing factor (CRF) mediates both the adrenergic (fight, flight, or freeze) response as well as the hypothalamic–pituitary–adrenocortical (HPA) response, which produces increased cortisol release from the adrenal cortex. An adaptive psychobiological response to traumatic stress is one that mobilizes these mechanisms for adequate coping and adaptation but which returns to normal function when the demands of traumatic event exposure have ceased. In general, PTSD may result from an (a) excessive stress reaction, (b) inadequate stress response, (c) inability to terminate the stress response when it is no longer adaptive, and/or (d) inability to calibrate the magnitude of a stress response to match the demands of a traumatic event (Friedman, 2002). Individuals with PTSD exhibit dysregulation of adrenergic, HPA, and other key systems that mediate the human response to stress. Psychobiological models, invoked to explicate this reaction include fear conditioning, resistance to extinction, and psychological sensitization (Charney, Deutch, Krystal, Southwick, & Davis, 1993; Friedman, 2002; Friedman & McEwen, 2004; Griffen, Resick, & Yehuda, 2005; McEwen, 1998).

Summary

Survivors of traumatic events who do not recover from traumatization, compared to those who do, may (a) learn greater or less readily extinguished fear responses to traumatic event–associated stimuli, (b) demonstrate problems in processing traumatic event–introduced information, (c) have disorganized traumatic event–related memory systems, and/or (d) have alterations in adrenergic, HPA, and other key psychobiological mechanisms. Each of these theories is supported by a host of psychobiological, memory, learning, and clinical research, and they continue to be the focus of empirical examination. It is important to note, there may be a complex interplay among these mechanisms. For example, information processing problems may moderate the effects of fear-related learning. Here, catastrophic misinterpretation of fear responses learned during a traumatic event (e.g., increased heart rate elicited by men after a male-perpetrated sexual assault) may increase the likelihood of PTSD relative to learned fear responses in the absence of such problematic processing.

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Empirical Tests of PTSD Prevention Programs

Below we review several tests of PTSD prevention-oriented interventions, highlighting their strengths and limitations, as well as possible reasons for the relatively limited progress in this domain. In addition to confining our review to investigations that fit the IOM's definition of prevention, we only discuss studies examining PTSD-relevant criteria, as opposed to studies that examine the prevention of nonspecific anxiety psychopathology (e.g., Barrett & Turner, 2001; Dadds et al., 1999; Lowry-Webster, Barrett, & Dadds, 2001). Within this domain, there have been five primary foci: (a) psychological debriefing, (b) brief psychosocial interventions targeting risk, (c) stepped collaborative care, (d) acute stress disorder (ASD) treatments, and (e) psychobiological interventions.

Psychological Debriefing

The vast majority of PTSD prevention programs have targeted persons at risk for developing PTSD, as indexed by traumatic event exposure, regardless of early symptom levels. The corpus of tests of psychological debriefing falls within this category. These studies typically test a variation of Critical Incident Stress Debriefing (CISD) (Mitchell, 1983). The CISD intervention generally consists of individual or group discussion of a traumatic event, delivered quickly within the wake of traumatic event exposure, which allows victims to verbally express their cognitive and emotional experience of a traumatic event. These discussions are guided and structured by a CISD facilitator who offers support (Everly, Flannery, & Eyler, 2002), but they do not include standardized cognitive–behavioral exposure or cognitive restructuring techniques. Another aim is to mobilize resources for victims to cope with repercussions of the traumatic event (e.g., job loss). It has been suggested the mechanisms of action include provision of a time-limited early support system wherein verbal ventilation of emotion allows for cognitive reconstruction of memories of the traumatic event, thereby reducing stress, rumination, and strain on homeostatic bodily mechanisms and facilitating meaning-making about the event (Everly et al., 2002; Mitchell & Everly, 1995). A detailed review of individual studies of psychological debriefing procedures is not included as there have been at least 15 reviews of this literature in the past 5 years (e.g., Bisson, 2003; Bryant, 2002; Ehlers & Clark, 2003; Everly et al., 2002; Gray & Litz, 2005; Litz, 2004; McNally, Bryant, & Ehlers, 2003; Rose, Bisson, & Wessely, 2003).

These reviews reveal that on balance, CISD does not appear to effectively prevent PTSD (Bryant, 2002; Litz, Gray, Bryant, & Adler, 2002;

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McNally et al., 2003; Rose et al., 2003; van Emmerik, Kamphuis, Hulsbosch, & Emmelkamp, 2002; Watson et al., 2003). In fact, one randomized controlled trial indicated that CISD may increase the likelihood of PTSD (Bisson, Jenkins, Alexander, & Bannister, 1997), and another suggested that it may maintain PTSD symptoms (Mayou, Ehlers, & Hobbs, 2000). Thus, CISD may interfere with natural (typical) recovery from a traumatic event (McNally et al., 2003), perhaps by increasing postevent arousal, which may facilitate overencoding and/or exacerbation of traumatic event–related memories. However, Everly and colleagues (Everly & Boyle, 1999; Everly, Boyle, & Lating, 1999; Everly et al., 2002) have reported findings from meta-analyses that suggest psychological debriefing does result in statistically significant reductions of PTSD development. Although not unanimous, it appears that psychological debriefing, delivered to recently traumatized persons regardless of their immediate response to the traumatic event, does not reduce PTSD incidence.

Brief Psychological Intervention Targeting Risk

Two studies have examined the effects of brief psychologically focused interventions that target risk for developing PTSD among individuals recently exposed to a traumatic event. First, Resnick and colleagues (Resnick, Acierno, Holmes, Kilpatrick, & Jager, 1999) targeted anxiety experienced during a gynecological forensic exam conducted shortly after sexual victimization, which is an empirically supported risk factor for PTSD development. A total of 205 adult women victims of forced sexual victimization were randomly assigned to either standard treatment or standard treatment preceded by a 17-minute video. The prevention video (a) described the procedures involved in the forensic exam, (b) provided a model patient demonstrating a relaxed response to the exam, and (c) provided instructions for self-directed exposure, education regarding how to identify and stop avoidance behavior, and strategies to improve mood and control anxiety. Standard treatment consisted of a "brief" session with a rape crisis counselor followed by a forensic gynecological exam. Of the initial sample, 123 (60%) returned for a 6-week follow-up assessment (attrition rates did not differ as a function of treatment group). Participants in the prevention condition reported a greater decrease in self-reported anxiety from preforensic to postforensic exam and fewer anxiety symptoms postexam than those in the control condition. Although there was not an overall difference between the two interventions on PTSD incidence, among the 46 women who reported a prior history of rape, PTSD diagnoses were significantly less likely in the intervention condition (33%) than the control

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condition (72%) at 6-week follow-up (Resnick, Acierno, Kilpatrick, & Holmes, 2005). Participants in the intervention, relative to control, were also significantly less likely to abuse marijuana by the follow-up (Acierno, Resnick, Flood, & Holmes, 2003).

The second study in this domain aimed to facilitate information processing among 17 traffic accident victims within 24 hours of the accident (Gidron et al., 2001). A phone-based intervention, which aimed to integrate nonverbal and verbal memory, was compared to a supportive listening condition in terms of PTSD symptom frequency at a 3- to 4-month follow-up. The intervention consisted of two sessions. The following components were included in Session 1: (a) A therapist worked with participants to clarify organizational (factual, affective, and sensory) details of the traumatic event, (b) participants were read the organized narrative, (c) participants repeated the organized narrative, and (d) participants were instructed to repeat the narrative to friends and family. During Session 2, participants repeated the narrative again and were educated about the importance of social support in healthy recovery from traumatic event exposure. The supportive listening condition consisted of two phone calls during which participants were asked to describe the traumatic event, but did not receive therapist assistance with organizing the narrative, instructions to practice recounting the narrative, or education regarding the role of social support in recovery. More patients in the control ($n = 4$; 44%) versus intervention ($n = 1$; 12%) condition met criteria for PTSD at follow-up. Participants in the control condition also reported significantly higher levels of total PTSD symptoms, a difference primarily attributable to differences in intrusion and hyperarousal symptoms. Importantly, due to the relatively small sample size, caution is warranted in interpreting these findings.

Stepped Collaborative Care

A third approach consists of stepped collaborative care, where the symptoms of individuals exposed to a traumatic event are monitored, and the level of care is adjusted accordingly. Zatzick and associates (2004) tested this model, wherein a case manager coordinated a stepped care approach for persons reporting to a Level I trauma center for intentional (e.g., physical assault) or unintentional (e.g., motor vehicle accidents) injuries. Prior to their discharge from the hospital, a case manager developed a postinjury care plan with them to address medical and psychosocial injury–related complications. The case manager was then available to patients 24 hours per day. Patients with a positive alcohol toxicology test on admission or

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who had abused alcohol in a potentially hazardous fashion also received an evidence-based motivational interviewing intervention, and patients with high levels of psychological distress immediately postinjury received psychiatric evaluations. Those demonstrating sustained psychological distress (e.g., severe distress for 24 hours) received evidence-based pharmacological intervention. If patients met criteria for PTSD at 3 months postinjury, they were offered their choice of evidence based cognitive–behavioral therapy (CBT), pharmacological treatment, or a combination. These treatments were provided throughout the rest of the 12-month follow-up period. Results suggested stepped collaborative care resulted in no increases in PTSD whereas there was a 6% increase ($p < .05$) in PTSD in the usual care condition.

ASD Treatments

Prevention programs also have targeted the reduction of early signs of PTSD (e.g., ASD) to prevent the development of chronic PTSD. The most common approach here has been CBT for ASD. These interventions have varied from 4 to 16 sessions in length and typically include the following components: psychoeducation, anxiety management training, cognitive restructuring, and exposure (Bryant, 2005). One study compared the effects of repeated assessment alone to a brief four-session CBT intervention for assault victims delivered shortly after the assault (Foa, Hearst-Ikeda, & Perry, 1995). CBT resulted in significantly lower rates of PTSD at 2 months (10% to 70%, respectively), with no differences in terms of diagnostic status by 5 months. Kilpatrick and Veronen (1984) compared the efficacy of group-based CBT to a repeated assessment control and a delayed assessment control condition. The intervention was 4 to 6 hours of group therapy consisting of (a) a rape-focused interview targeting elicitation of affect, (b) psychoeducation regarding normative responses to traumatic event exposure, (c) instructions to explore how they are made to feel guilty for being raped (e.g., messages in the media) and the implications of such guilt (e.g., self-limitations to avoid a similar experience), and (d) training in assertiveness, relaxation, thought stopping, and avoidance reduction. There were no significant effects of the intervention (Resnick et al., 1999). In contrast, three studies compared supportive counseling to brief CBT (approximately 5 sessions) delivered to civilians meeting criteria for ASD (Bryant, Harvey, Dang, Sackville, & Basten, 1998; Bryant, Moulds, Guthrie, & Nixon, 2005; Bryant, Sackville, Dang, Moulds, & Guthrie, 1999). Here, fewer persons receiving CBT (approximately 20%) met criteria for PTSD at a 6-month follow-up than

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those receiving supportive counseling (57%-67%). These effects appear to maintain for up to 4 years (Bryant, Moulds, & Nixon, 2003).

A separate approach to ASD treatment consisted of a pilot study of 25 children and adolescents who were severely burned (Robert, Blakeney, Villareal, Rosenberg, & Meyer, 1999). Tricyclic antidepressant treatment (imipramine), compared to sedative–hypnotic treatment (chloral hydrate), resulted in significantly greater reductions in ASD severity by 6-week follow-up. Although these findings are promising, methodological limitations, such as discontinuing the trial at 7 days postinitiation and not monitoring intervention effects on PTSD development, render conclusions regarding PTSD prevention speculative.

Psychobiological

One focus of psychobiological PTSD prevention is early "correction" of psychobiological responses to traumatic event exposure. Based on the work of Yehuda and associates (1990) showing an association between lower cortisol levels and PTSD, it has been hypothesized that PTSD might be prevented by preventing a reduction in cortisol levels. Schelling administered hydrocortisone prior to, during, and following cardiac surgery (Schelling, Briegel, et al., 2001; Schelling, Stoll, et al., 2001). For instance, when 20 patients were interviewed approximately 31 months postsurgery, the rates of PTSD were significantly lower in the hydrocortisone treatment (11%) than placebo (64%) group (Schelling, Briegel, et al., 2001). See Schelling, Stoll, and colleagues (2001), Schelling, Kilger, et al. (2004), and Weis et al. (2006) for comparable results. Four possible, nonmutually exclusive, mechanisms of action have been suggested to explain these findings. First, administration of hydrocortisone reduced the likelihood of HPA alterations by preventing the upregulation of glucocorticoid receptors. Second, the intervention may ensure a sufficient amount of circulating cortisol to reduce sympathetic arousal (Friedman, 2002; Schelling, Roozendaal, & de Quervain, 2004). Third, retrieval of traumatic event–related memory may be impaired, thereby preventing excessive retrieval as observed in PTSD (Schelling, Roozendaal, et al., 2004). Finally, high glucocorticoid levels may facilitate extinction of fearful responding learned during traumatic event exposure (Schelling, Roozendaal, et al., 2004).

A separate line of research has tested the effects of administering β adrendergic antagonists immediately after traumatic event exposure. This intervention aims to prevent posttraumatic arousal and to simultaneously

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interrupt psychopathological peri- and posttraumatic reconsolidation of traumatic event–related memories facilitated by excessive arousal (McCleery & Harvey, 2004; see also Cahill, 1997). A pilot investigation ($n = 41$) of β adrendergic-blocker administration within 6 hours of a motor vehicle accident for 10 days (Pitman et al., 2002) tested this conceptualization. Compared to the placebo control group, persons receiving the β adrendergic-blocker did not significantly differ in terms of PTSD symptoms or diagnoses at a 1- or 3-month follow-up assessment (although there was very low statistical power to detect such differences). However, at the 3-month follow-up, those receiving the β adrendergic-blocker evidenced significantly less autonomic reactivity to individualized script-driven images of their traumatic events, which can differentiate those with versus without PTSD (Orr et al., 1998).

Finally, benzodiazepine administration has been utilized as an early intervention among individuals recently exposed to a traumatic event to target sleep quality and overconsolidation of event-related learning. For example, Mellman, Bustamante, David, and Fins (2002) tested the efficacy of temazepam administration for 7 nights among 22 recently traumatic event–exposed (on average, 14 days postevent) adults in a randomized placebo-controlled trial. Approximately 6 weeks after the trial began, 6 (55%) of those receiving temazepam and 3 (27%) receiving placebo met criteria for PTSD. Similarly, Gelpin, Bonne, Peri, and Brandes (1996) administered a benzodiazepine shortly after exposure to a traumatic event and reported increased PTSD incidence at 6-month follow-up (69%) compared to a gender and acute distress–matched control group (15%).

Summary

The vast majority of research on PTSD prevention, consisting primarily of tests of CISD, has yielded relatively little efficacy in preventing PTSD. However, newly emerging prevention programs are beginning to demonstrate promising findings in terms of efficacy. In particular, CBT and reducing acute posttraumatic event elevations in adrenergic or HPA activity appear promising. Importantly, the relatively short follow-up periods in some of the existing studies render conclusions tentative given evidence of delayed onset PTSD (e.g., Gray, Bolton, & Litz, 2004). Moreover, evidence suggesting posttraumatic event CISD and benzodiazepine administration may have negative iatrogenic effects necessitate caution when developing and testing early interventions among recently traumatized persons.

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Relation of Core Mechanisms of Action to Extant Empirical Tests

Taken together, there is a degree of uncertainty about how the core theoretically relevant processes believed to be involved in recovery from traumatic event exposure have been targeted in the many of the extant PTSD prevention programs. There are some exceptions, mostly with regard to those programs that explicitly target psychobiological processes implicated in PTSD. To advance future prevention efforts, we believe it is fruitful to consider how potentially efficacious interventions that have not explicitly targeted processes articulated in PTSD theories may affect such mechanisms. Consideration of the active mechanisms in existing programs may stimulate empirical examination of these mediators in future tests of these programs, which would allow for statements regarding which mechanisms may be usefully targeted in preventive interventions.

Resnick and colleagues' (2005) program may reduce problematic overconsolidation of memory by reducing posttraumatic arousal. Similarly, the first level of intervention in Zatzick and colleagues' (2004) stepped care program was typically administration of selective serotonin reuptake inhibitors to persons exhibiting high levels of distress shortly after traumatic event exposure. This intervention also may target posttraumatic arousal and therefore prevent overconsolidation of traumatic event–related memories. Also, constant access to a care manager in this intervention may have facilitated processing traumatic event–related information via social support, which Brewin and colleagues (1996) have predicted would reduce PTSD development. CBT for ASD also may target multiple mechanisms of action simultaneously. Cognitive restructuring components may facilitate the processing of traumatic event–related information by correcting problematic interpretations of trauma-related events and/or reducing thought and/or emotion suppression of related information. Also, exposure-based components may alter, via extinction, traumatic event–related fear conditioning. To date, the mechanisms of action resulting in promising effects of these interventions have not been tested.

Future Directions

In addition to testing mechanisms of action involved in existing prevention programs to refine these protocols, several other programs of research may be helpful in advancing the science of PTSD prevention. Because there

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are numerous possible approaches in this domain, we focus on interventions that seem to hold the most promise in preventing PTSD. These possibilities, which are based on a large backdrop of empirical work, are provided to stimulate future research and discussion, as opposed to exhaustively outlining possible approaches to PTSD prevention.

Learning

There is a relative paucity of programs that explicitly target learning that occurs during traumatic events despite the centrality of learning in theories of PTSD (e.g., Ehlers & Clark, 2000; Foa & Rothbaum, 1998; Jones & Barlow, 1990; Pitman et al., 2000). As noted above, exposure-based components of CBT approaches, as well as instructions for self-guided exposure, may have preventive effects by way of facilitating extinction of traumatic event–related learning. However, direct tests of the mediating role of extinction in the effects of these components are needed. For instance, laboratory-based methodologies that can index learned emotional reactivity to trauma-cues (e.g., individualized script-driven imagery procedures; Orr et al., 1998) could be used to index reductions in such reactivity over the course of these preventive interventions. This type of index could then be examined statistically as a mediator of the relation between an intervention and associated outcomes. In addition, there likely are other pathways to target traumatic event–related learning. In addition to examining the role of learning in existing prevention programs, perhaps three of the most promising approaches in need of further examination are latent inhibition of fear-related conditioning, reducing peritraumatic arousal to reduce traumatic event–related learning, and modeling nonfearful responses to traumatic event–related stimuli.

Latent inhibition. Latent inhibition is a process by which preexposure to a stimulus without consequences retards learning about the stimulus, relative to a stimulus that has not been preexposed, during future conditioning trials (Lubow, 1973). Interventions based on latent inhibition have demonstrated efficacy in preventing the development of dental phobias (Davey, 1989; De Jongh, Muris, Ter Horst, & Duyx, 1995). Prevention of fear-based learning via latent inhibition also has been examined in rhesus monkeys (e.g., Mineka & Cook, 1986), in laboratory paradigms of human affective learning (e.g., Díaz & De la Casa, 2002; see also Lubow and Gewirtz, 1995, for a review), and has been recommended as a prophylactic procedure for preventing the incidence of fear-based psychopathology (Lubow, 1998;

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Mineka & Zinbarg, 2006). However, there has been no such test in the area of PTSD.

At least two factors that modulate the effects of latent inhibition procedures need to be considered in the development of such an approach. Specifically, the extent to which learning will be prevented is dependent on the degree (a) the preexposed stimuli resemble the traumatic event–related stimuli and (b) the preexposure context resembles the context in which a traumatic event occurs (see Lubow, 1998). Together, preexposing persons who are at risk for exposure to a potentially traumatic event to a situation that, as closely as ethically possible, resembles the impending event may prevent learning to fear cues present during the event. For instance, utilization of virtual reality techniques to mimic combat exposure, such as those demonstrating efficacy in exposure therapy for PTSD (e.g., Rothbaum, Hodges, Ready, Graap, & Alarcon, 2001; Rothbaum, Ruef, Litz, Han, & Hodges, 2003), may hold promise for preventing combat-related PTSD. Of note, although the PTSD treatment literature suggests stimulus exposure procedures can efficaciously modulate relevant learning related to different types of trauma (e.g., combat, sexual assault; Bryant et al., 2003; Taylor et al., 2003), virtual reality protocols used in prevention programs may be more readily applied to certain traumatic event types (e.g., combat, natural disaster) than others (rape, physical assault). For instance, it may be unnecessary to use virtual reality protocols to realistically preexpose young women to sexual assault–related stimuli. Instead, the careful and sensitive use of standardized script-driven imagery procedures, such as those used in laboratory studies of PTSD (e.g., Orr et al., 1998), or film clips from the popular media, such as the sexual assault scene in Paramount studio's *The Accused*, to preexpose rape-related stimuli may be similarly effective and less distressing, and therefore preferable given the overarching priority of protecting human participants.

Peritraumatic arousal. Arousal experienced during a traumatic event (i.e., peritraumatic) has been widely recognized as involved in problems recovering from exposure to the event (e.g., Ehlers & Clark, 2000; Foa & Rothbaum, 1998; Jones & Barlow, 1990; McCleery & Harvey, 2004; Ozer, Best, Lipsey, & Weiss, 2003; Pitman et al., 2000). For instance, peritraumatic panic attacks, which are characterized by high levels of arousal, have been associated with increased PTSD incidence (e.g., Galea et al., 2002). Moreover, strong emotional responses (e.g., fear, helplessness, horror, anger) during a traumatic event are related to an increased likelihood of PTSD (Brewin, Andrews, & Rose, 2000; Ozer et al., 2003). These data are also consistent with research suggesting that the stronger an emotional

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(unconditioned) response (e.g., fear) elicited in conditioning trials, the greater the learning (e.g., greater magnitude of conditioned response, more resistant to extinction) about a previously unconditioned stimulus (Forsyth & Eifert, 1998). This suggests a traumatic event that elicits a highly arousing emotional response is more likely to result in learned fear of cues present during a traumatic event (e.g., crowds of people) than an event that elicits less emotional arousal.

These data collectively suggest that peritraumatic arousal may be important in the development of learned fear of traumatic event–relevant cues (see below for a discussion of the possible role of arousal in traumatic event–related memory problems). It follows, then, that reduction of peritraumatic arousal may reduce traumatic event–related learning, which may reduce the incidence of PTSD. Here, research on factors that reduce the degree to which one responds fearfully to an unconditioned stimulus (e.g., traumatic event) is informative. Although a number of factors are thought to influence such responding (e.g., developmental histories characterized by dyscontrol, traumatic event severity; see Brewin, Andrews, and Valentine, 2000, and Ozer and colleagues, 2003, for detailed reviews), only those that may be malleable will be discussed, as these factors are most pertinent to preventive interventions.

Two particularly well-researched factors are predictability of, and control over, unconditioned emotion-eliciting stimuli. Indeed, both uncontrollability and unpredictability have been theorized as important factors in failure to recover from traumatic event exposure (Foa, Zinbarg, & Rothbaum, 1992). An array of animal (e.g., Mineka, Cook, & Miller, 1984; see Mineka & Kihlstrom, 1978, for a review) and human (e.g., Lejuez, Eifert, Zvolensky, & Richards, 2000; Zvolensky, Eifert, Lejuez, & McNeil, 1999) research suggests unpredictable and uncontrollable stressors elicit greater fearful responding than predictable and/or controllable stressors. Even *perceived* control over unconditioned stimuli can reduce fearful responding (Sanderson, Rapee, & Barlow, 1989), which may be of particular relevance to the current discussion. For instance, in a comparison of torture victims who were either "psychologically prepared" political activists or unprepared nonpolitically active victims, nonactivists were significantly more likely to meet criteria for both current and lifetime PTSD despite reporting being subjected to relatively less severe torture (Basoglu, Mineka, Paker, Aker, Livanou, & Gök, 1997). This pattern of findings may be due to activists being more able to predict the torture, perhaps due to knowledge of torture tactics, and/or they had higher levels of perceived control during the torture (Basoglu et al., 1997).

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Preventive interventions that increase appropriate perceptions of control and predictability over traumatic events may decrease peritraumatic emotional arousal, thereby reducing PTSD incidence subsequent to traumatic event exposure. For example, educating people who live in areas vulnerable to natural disaster about the risk for such a disaster and what experiencing such a disaster looks and feels like may increase the predictability of the event. This, in turn, may decrease fear experienced during the traumatic event and decrease related learning compared to an area without such preparation. Prior to developing broad-based preventive interventions based on increasing perceived control and predictability, future research that systematically examines the effects of predictability of, and control over, traumatic event exposure is needed to determine the extent to which these factors can be utilized to proximally reduce peritraumatic arousal and distally reduce PTSD incidence.

A third malleable factor shown to modulate emotional responding to unconditioned fear-relevant stimuli is anxiety sensitivity, defined as fear of anxiety and anxiety-related sensations (Taylor, 1999). High, relative to low, levels of anxiety sensitivity predict greater fear-based responses when faced with fear-eliciting events (e.g., Zvolensky, Feldner, Eifert, & Stewart, 2001). Thus, persons high in anxiety sensitivity may experience greater emotion-related arousal during traumatic event exposure. Indeed, cross-sectional (Fedoroff, Taylor, Asmundson, & Koch, 2000) and prospective (Keogh, Ayers, & Francis, 2002) investigations suggest that persons higher, compared to those lower, in anxiety sensitivity respond more symptomatically to traumatic events (see Taylor, 2003, for a discussion). Moreover, the effects of exposure to multiple traumatic event types, a well-documented risk factor for PTSD symptomatology (Schnurr, Spiro, Vielhauer, Findler, & Hamblen, 2002; Zatzick et al., 2002), are moderated by anxiety sensitivity. Specifically, among individuals high in anxiety sensitivity, greater traumatic event exposure is associated with higher levels of PTSD symptoms, whereas number of traumatic event type exposures has relatively less of an effect on PTSD symptoms among individuals low in anxiety sensitivity (Feldner, Lewis, Leen-Feldner, Schnurr, & Zvolensky, 2006). Taken together, pretraumatic event reductions in anxiety sensitivity may reduce fear during event exposure, thereby preventing fear-relevant learning and associated PTSD symptomatology. Brief psychosocial interventions, such as interoceptive exposure and psychoeducation, have demonstrated efficacy in reducing anxiety sensitivity (Maltby, Mayers, Allen, & Tolin, 2005) and therefore may hold promise as elements of PTSD preventive strategies.

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Modeling nonfearful responses. Finally, modeling a relaxed response to a potentially traumatic event may also help prevent fear-related learning. Mineka and Cook (1986) demonstrated that exposing Rhesus monkeys to a relaxed monkey model in the presence of a to-be-conditioned stimulus (snake) significantly reduced the acquisition of a fear response to the snake during subsequent conditioning compared to monkeys not exposed to such a model. Studies may examine the preventive implications of this work by, for instance, modeling the use of relaxation strategies prior to, or during, a traumatic event to groups at high risk for traumatic event exposure (e.g., emergency response personnel). Interventions of this sort also could examine the mechanisms by which such modeling interferes with fear-related learning by examining the role of peritraumatic arousal. It may be that modeling a relaxed response reduces peritraumatic arousal and thereby prevents learning (or overconsolidation of event-related memories).

Information Processing

To the best of our knowledge, there is little empirical work on the effects of premorbid interventions on anxiety-relevant information processing. Yet there are at least three research domains that may inform the development of novel prevention programs targeting these processes: PTSD treatment, emotion and/or thought regulation, and the role of drug use and social support in PTSD.

Treatments targeting information processing. Cognitive-focused PTSD treatments have demonstrated efficacy (Ehlers, Clark, Hackmann, McManus, & Fennell, 2005; Monson et al., 2006; Resick et al., 2002), and recent data suggest this effect may be due, at least in part, to the modification of problematic information processing (Ehlers et al., 2005). These treatments typically identify problems patients are having with processing information, such as attentional biases for threat-relevant information, and modify them with contemporary cognitive restructuring interventions (Resick, 2001). Cognitive restructuring interventions typically provide alternatives to problematic processing and support these alternative approaches by instructing patients to collect data testing (e.g., is there evidence that your bias to perceive threat is accurate?) and challenging (e.g., if there is little supporting evidence, how else might you think of this "threatening" event?) problematic processing. Given evidence of the malleability of problematic processing in clinical trials, prevention scientists may

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examine the efficacy of developing cognitive restructuring skills among those at risk for exposure to a traumatic event, or among recently traumatic event–exposed persons, to promote recovery by preventing or correcting problematic information processing.

There are several possibly efficacious training programs and intervention points to consider along these lines. For instance, young-adult women in college, a group at significant risk for developing sexual assault–related PTSD, may be taught the likely information processing problems resulting from such a traumatic event (e.g., self-blame, overgeneralization of fear of the rapist to fear of being in a social situation). Such a training program could include teaching participants to identify problems with information processing and to utilize cognitive restructuring techniques (e.g., thought monitoring) to modify them.

Cognitive avoidance and emotion regulation. Several converging lines of evidence have implicated cognitive avoidance in PTSD. First, high levels of cognitive avoidance are endorsed by persons with (a) a history of sexual abuse (Batten, Follette, & Aban, 2001) and (b) ASD (Harvey & Bryant, 1998a) or PTSD (Ehlers & Steil, 1995). Second, high levels of cognitive avoidance predict greater posttraumatic event symptom severity among persons with PTSD (Bryant & Harvey, 1995), ASD (Harvey & Bryant, 1998a), and a history of traumatic event exposure (Marx & Sloan, 2005). Third, cognitive avoidance mediates the relation between a history of childhood sexual abuse and subsequent distress (Marx & Sloan, 2002). Finally, efforts to suppress traumatic event–relevant thoughts appear to increase the frequency of both the suppressed thoughts (Davies & Clark, 1998; Harvey & Bryant, 1998b; Shipherd & Beck, 2005) and concurrent physiological arousal (Roemer & Salters, 2004). These data argue that cognitive avoidance may maintain PTSD symptoms.

An area that has been differentiated from thought suppression (Zvolensky, Feldner, Leen-Feldner, & Yartz, 2005) and received less empirical attention in relation to PTSD is the role of *emotion* inhibition. Additional research on the role of emotion regulation in terms of the information processing implicated in PTSD seems warranted for two reasons. First, persons with PTSD, compared to those without, report more frequently and intensely withholding emotions (Roemer, Litz, Orsillo, & Wagner, 2001). Second, emotion inhibition may occupy significant levels of limited cognitive resources, thereby leaving few cognitive resources for processing information during inhibition attempts (Muraven, Tice, & Baumeister, 1998). Indeed, chronic efforts aimed at inhibiting emotion among persons with PTSD may prematurely

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inhibit the processing of traumatic event–related information, which is consistent with contemporary accounts of PTSD (Brewin et al., 1996).

Further examination of the role of emotion regulation also would complement extant research on cognitive avoidance. Cognitive avoidance (e.g., thought suppression) has typically been considered a mechanism underlying the intrusive thoughts inherent to PTSD. Examining the effects of emotion suppression within a PTSD-relevant paradigm would elucidate the role of such a strategy in terms of intrusive thoughts, as well as other PTSD-relevant domains. For instance, data suggest emotion suppression results in increased activation of the cardiovascular system during (Gross, 1998, 2002) and after (Feldner, Zvolensky, Stickle, Bonn-Miller, & Leen-Feldner, 2006) suppression attempts. These findings suggest emotion suppression may, in part, maintain PTSD-related hyperarousal.

Two other factors that likely are functionally related to the processing of traumatic event–related information are drug use and social support. First, models of drug use–anxiety comorbidity suggest that drug use principally aimed at escaping or avoiding anxiety-related states may be an affect regulation strategy that maintains fear-related problems (Otto, Safren, & Pollack, 2004; Stewart & Conrod, 2003; Zvolensky, Bernstein, Marshall, & Feldner, 2006). Indeed, the presence versus absence of nicotine dependence prior to traumatic event exposure increases the likelihood of meeting criteria for PTSD subsequent to combat exposure (Koenen et al., 2005) and smoking to reduce negative affect is positively associated with posttraumatic stress symptoms among trauma-exposed young adults (Feldner et al., in press). Health behaviors such as drug use may make useful proximal targets in prevention programs as targeting use directed at avoiding anxiety-related states may decrease avoidant-type affect regulation, thereby promoting processing of traumatic event–related information and allow for integration of this information into preexisting informational networks.

Second, the presence of social support appears protective; traumatic event–exposed persons with, versus without, social support are less likely to be diagnosed with PTSD (Brewin, Andrews, Valentine, 2000). Several different mechanisms have been put forth to explain the buffering effects of social support. Instrumental and emotional support may reduce negative affect through associated behavioral activation that reinforces healthy behaviors and attenuates avoidance behaviors that maintain PTSD and negative mood (e.g., Lewinsohn, Hoberman, Teri, & Hautzinger, 1985). Social support can also facilitate disclosure about a traumatic event, which directly enhances emotional processing of traumatic events. Disclosure of the traumatic event to trusted others provides opportunities for extinction of

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learned fear, as well as modifications in the maladaptive cognitions that may inhibit processing of the traumatic event (e.g., Brewin et al., 1996; Monson, Stevens, & Schnurr, 2005). Therefore, interpersonal interventions aimed at developing social support networks may aid posttraumatic event recovery. Only a few studies have investigated the role of significant others in PTSD treatment (Glynn et al., 1999; Monson, Guthrie, & Stevens, 2003), and to our knowledge, no studies have specifically investigated social support interventions in prevention efforts. Mobilization of social support in preventive programs likely will be most effective (e.g., reach the most at-risk individuals) within naturalistic settings, such as schools, religious communities, veterans' organizations, the workplace, or community organizations. Given the avoidance inherent to PTSD, those experiencing the highest levels of traumatic event–related distress may avoid these organizations. Therefore, greater formal integration of these support networks into the assessment, prevention, and treatment process may be necessary to the success of this type of preventive effort.

Collectively, prevention programs delivered prior to exposure to a traumatic event or immediately after such exposure that target reduced cognitive avoidance, emotion inhibition, avoidant-type drug use, and increased social support may prove fruitful in promoting healthy recovery from traumatic event exposure by facilitating information processing. For instance, interventions designed to reduce emotion suppression subsequent to traumatic event exposure may reduce PTSD development by promoting processing of trauma-related information, directly reducing hyperarousal, and indirectly allowing for extinction of traumatic event–related learning by promoting engagement with event-related cues and associated emotional responses. Several existing cognitive and emotion-focused regulation training programs may be utilized as models for such programs. For instance, training nonclinical participants in mindfulness, a strategy that appears opposed to avoidance and suppression (Zvolensky et al., 2005), has demonstrated promising results in terms of immune system functioning and increased left-sided anterior brain activation, which is consistent with increased positive affect (Davidson et al., 2003). Moreover, anxiety disorder treatments are continually being developed to target mindfulness and acceptance (see Baer, 2003, for a review).

Memory

There also are multiple interventions that may prevent the development of problematic memories implicated in PTSD. Innovative psychological programs that explicitly target peritraumatic and posttraumatic event arousal

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to reduce overconsolidation of traumatic event–related memory may be indicated. Several examples of interventions that may do so have already been suggested, such as targeting the controllability and predictability of a traumatic event among emergency response personnel or providing modeling and training in the use of relaxation strategies during a traumatic event. Also, pretraumatic event psychological inoculation strategies may reduce fearful arousal experienced during a traumatic event, thereby reducing overconsolidation of related memories. For instance, reducing anxiety sensitivity levels by preexposing individuals to traumatic event cues may reduce peritraumatic arousal, thereby reducing consolidation of traumatic event–related memories. Posttraumatic event strategies also may be helpful in this domain. For instance, relaxation training soon after traumatic event exposure may reduce postevent arousal and related memory overconsolidation.

Psychobiological

There are multiple investigations within this domain that likely would enhance our understanding of the processes involved in psychobiological preventive interventions. To date, it is difficult to draw conclusions about the mechanisms of action targeted by these interventions. Specifically, the effects observed in these studies may be due to reduced arousal during traumatic event exposure, thereby affecting learning and memory, or they may be exerting their effects via reduction of sympathetic arousal subsequent to exposure. One possibly fruitful line of inquiry would be varying the timing of glucocorticoid administration, which would allow for teasing apart these different mechanisms of action. For instance, if glucocorticoid administration conducted only prior to traumatic event exposure (e.g., surgery) results in effects comparable to administration prior to, during, and after event exposure, it would seem that the intervention is targeting processes functioning prior to or during the event (e.g., learning) rather than after the event (memory consolidation or posttraumatic arousal).

Two other approaches may also prove helpful in future investigations. First, programs may be developed to bolster resilience among persons at risk for posttraumatic problems due to specific genotypes that early evidence suggests are linked to such problems. For example, the presence of the A1 allele of the D(2) dopamine receptor has been linked to greater severity of PTSD symptoms among persons with PTSD (Lawford, Young, Noble, Kann, & Ritchie, 2006). Also, evidence suggests the functional polymorphism in the promoter region of the serotonin transporter gene (5-HTT) moderates the effects of childhood maltreatment, such that childhood maltreatment among those with a short allele of this gene are particularly

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likely to develop adulthood depression (Caspi et al., 2003; Kaufman et al., 2006). These genotypes also may be examined as moderators of the effects of PTSD prevention programs, generally, to more fully understand their role in PTSD and related interventions. Second, other avenues toward bolstering psychobiological resilience may be helpful for persons at risk for PTSD (e.g., likely to be exposed to a traumatic event). For instance, if an individual is deficient in his or her capacity to mobilize neuropeptide Y or mobilizes too much CRF, pretraumatic event adjustment of these reactions may be protective (see Friedman, 2002, for a discussion).

Summary

Collectively, prevention programs within the domains of learning, information processing, memory, and psychobiology could target the primary mechanisms thought to differentiate PTSD from healthy recovery from traumatic event exposure. Moreover, many of these theoretically derived programs could consist of administering protocols either pre- or post-traumatic event (or both). Large bodies of research support the potential efficacy of these programs and scientists may be able to capitalize on newly emerging technology, such as virtual reality and genotyping, to advance these programs beyond that which has previously been possible. Importantly, the application of these new technologies requires careful consideration to ensure the safe and ethical treatment of human participants. Also, collaborations between clinical researchers and those involved in research at more basic levels of analysis, such as learning theory, the laboratory study of emotion, and genotyping, would improve the development and conduct of these types of sophisticated programs.

Broad-Based Directions for Future Research

We have thus far outlined several potentially advantageous areas of future research for PTSD prevention scientists to begin developing novel theoretically driven prevention programs. In addition to these recommendations, this review raises three broad considerations for research in this area.

Risk and Protective Factor Research

Given high rates of traumatic event exposure, targeting all traumatic event–exposed individuals or those at risk for traumatic event exposure may

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be particularly costly, as such interventions are resource intensive (Cuijpers, 2003). For example, traumatic event exposure estimates among the general U.S. population range from 55% (Kessler et al., 1995) to 69% (Norris, 1992), with higher estimates among high-risk groups, such as women (69%; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993) and military veterans (79%; Schnurr et al., 2002). Thus, there is a need for continued research on risk and protective factors that may affect recovery from traumatic event exposure. In particular, research on malleable factors within these domains will be critical.

There are at least three distinct directions for future research in this domain. First, additional prospective research linking malleable risk and protective factors to PTSD symptom maintenance is needed. This literature has linked several factors to PTSD symptoms, such as genetic vulnerability, anxiety sensitivity, emotion and/or thought regulation, social support, and dysregulation of key biological mechanisms that mediate and moderate the human stress response. However, to further substantiate these factors as targets for prevention programs, the prospective relations between many of these factors and posttraumatic stress symptom maintenance subsequent to traumatic event exposure are needed. Second, research on other theoretically predicted risk and protective factors is needed. Several factors have been suggested, all of which require further empirical exploration to determine their roles in PTSD development as well as the degree to which they can be modified. These include, but are not limited to, substance use (Stewart & Conrod, 2003), mental defeat (Ehlers & Clark, 2000), and peritraumatic dissociation (Brewin & Holmes, 2003). Finally, research should examine the mediating role of changes in these factors on PTSD development, which will provide critical tests of the causal role of these factors in PTSD development (Kellam, Koretz, & Moscicki, 1999). Similarly, these types of tests will allow for examination of the utility of theories of PTSD relative to each other. For example, Janoff-Bulman's (1992) information processing theory and learning theory would make contradictory predictions regarding the likelihood of PTSD in the case of a woman with a relatively safe and extensive history of combat zone exposure who incurs a combat-related injury. The former would predict a high likelihood of PTSD because of the resulting "shattered" belief about the safety of being in a combat zone, whereas the latter, based on findings in the latent inhibition domain, may predict a relatively low likelihood of PTSD development due to a long history of stimulus preexposure, resulting in inhibition of fear-relevant learning. A preventive intervention that incorporates latent inhibition procedures to experimentally manipulate virtual preexposure to dangerous combat zone

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situations may prove fruitful in shedding light on this type of differential prediction.

Identifying Individuals at Greatest Risk

Research also should continue to focus on improving methods for identifying traumatic event–exposed persons who are at high risk for PTSD. Research has pointed to multiple domains, within which specific factors have been identified that appear to increase the risk for developing PTSD. For instance, in terms of individual difference characteristics, female gender (Kessler et al., 1995), family history of psychopathology (Brewin, Andrews, & Valentine, 2000), and history of exposure to multiple traumatic event types (Schnurr et al., 2002) increase risk for PTSD. Peritraumatic factors including panic (Galea et al., 2002), traumatic event severity (Norris et al., 2002), interpersonal traumatic event types (Kessler et al., 1995), dissociation (Ozer et al., 2003), and posttraumatic factors such as lack of social support and additional life stress (Brewin, Andrews, Valentine, 2000) also appear to increase risk of PTSD. However, our ability to specifically identify those at risk remains limited, which hampers efforts to provide what currently are resource intensive prevention programs to those at highest risk for PTSD.

This recognition has led to theoretically derived recommendations, such as the assessment of (a) serum and urinary indicators of psychobiological abnormality (e.g., HPA axis) and (b) stress system provocations (e.g., yohimbine provocation, startle paradigms) to identify abnormal recovery from stress (Friedman, 2002). Also, researchers continue to focus on developing brief screening assessments that can be implemented in settings with little available time, such as primary care settings (A. J. Lang & Stein, 2005). This research, in combination with continued research on integrative models of PTSD prediction among traumatic event–exposed individuals, will improve our ability to develop prevention programs that vary in terms of intensity and can be titrated according to risk level among those exposed to a traumatic event.

Examining and Improving Implementation

The degree to which the majority of the targeted PTSD prevention programs tested to date can be implemented on a relatively large scale remains unclear. Three approaches to advancing our understanding of how current prevention programs can be implemented within a health care system that already is challenged by limited resource (budgetary, time) constraints

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are recommended. First, cost-effectiveness analyses of programs that are ongoing are needed. For instance, it is unclear if programs that require the assignment of a case manager to all traumatic event–exposed people reporting to an emergency department (see Zatzick et al., 2004) will offset the costs associated with the development of PTSD, which would be predicted only among a minority of these individuals. Model cost-effectiveness analyses of this sort can be found in other literatures (Aos, Lieb, Mayfield, Miller, & Pennucci, 2004; Katon, Roy-Byrne, Russo, & Cowley, 2002), and applying these methods to targeted PTSD prevention program development would help outline the parameters, in terms of resource allotment, that would need to be considered. Second, given the relative intensity of several programs tested to date (e.g., ASD treatments and stepped collaborative care approaches), additional research into the dissemination of these programs is needed. For instance, promising results have emerged from tests of Internet-based anxiety disorder treatments (Kenardy, McCafferty, & Rosa, 2003), and preliminary evidence suggests these types of interventions are feasible in the context of postdisaster intervention efforts (Ruggiero et al., 2006). Integrating these cutting-edge technologies into the widespread dissemination of targeted PTSD prevention programs may significantly improve cost-effectiveness. Finally, assessments of the effects of PTSD prevention programs should be broad enough to capture the effects of these interventions on the broad array of psychopathological outcomes that can follow from traumatic event exposure. For example, traumatic event exposure can increase drug use, such as alcohol use, cigarette smoking, and marijuana use (Vlahov et al., 2002), and PTSD is related to increased rates of physical health problems (Friedman & Schnurr, 1995) and associated health care utilization (Ford et al., 2004). Indeed, PTSD-focused prevention programs can have effects on these other outcomes (Acierno et al., 2003; Zatzick et al., 2004).

Concluding Remarks

Prevention scientists currently are faced with exciting developments and related challenges in the area of PTSD prevention. Continuing theoretical work has implicated fear-related learning, problems processing traumatic event–introduced information, disorganized memory systems related to traumatic events, and/or alterations in key psychobiological mechanisms in the pathogenesis of PTSD. Although promising prevention programs have been tested within the past decade, the degree to which factors implicated in the

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pathogenesis of PTSD are targeted in, and affected by, existing prevention programs is typically unclear. Thus, research on the prevention of PTSD would benefit from a more explicit incorporation of theoretical accounts of PTSD into intervention development and testing. As we have discussed, there are several possible avenues for the development of these types of novel, theoretically driven prevention programs. Moreover, a large body of research in the areas of contemporary learning theory, information processing, thought and emotion regulation, PTSD treatment, and psychobiology supports the development and testing of several such programs. Testing these types of programs, in combination with continued research on risk and resilience related to posttraumatic stress, risk identification, and prevention program implementation likely will advance the area of PTSD prevention as well as our understanding of the factors that result in posttraumatic stress problems.

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