Theoretical Review

RESILIENCE: RESEARCH EVIDENCE AND CONCEPTUAL CONSIDERATIONS FOR POSTTRAUMATIC STRESS DISORDER

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The growing recognition and occurrence of traumatic exposure in the general population has given increased salience to the need to understand the concept of resilience. More than just the "flip side" of a risk factor, the notion of resilience encompasses psychological and biological characteristics, intrinsic to an individual, that might be modifiable and that confer protection against the development of psychopathology in the face of stress. In this review, we provide some perspective on the concept of "resilience" by examining early use of the term in research on "children at risk" and discuss the relationship between risk and resilience factors. We then review psychological and biological factors that may confer resilience to the development of posttraumatic stress disorder (PTSD) following trauma, examine how resilience has been assessed and measured, and discuss issues to be addressed in furthering our understanding of this critical concept going forward. Depression and Anxiety 24:139–152, 2007.
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INTRODUCTION

Increased understanding of the impact of trauma and the characteristics that promote resilience in individuals has recently become more critical given heightened tensions in the international political environment, which have included increased exposure to threats of terrorism and armed conflict. Most of the research on posttraumatic stress disorder (PTSD) has focused on variables that confer risk factors for developing this disorder after a trauma, despite the fact that the majority of trauma victims do not develop PTSD. Far fewer studies have focused on variables that buffer risk or serve as resilience factors. There is a growing interest in determining and understanding factors that promote resilience to psychopathology in individuals after they experience a traumatic event. Our aim in this article is to review research on resilience, first looking at historical perspectives on the concept of resilience itself, then examining resilience from the perspective of decreased vulnerability to PTSD in reaction to trauma. We review both psychological and biological variables representing resilience, as well as

tools that have been used to measure this concept. Last, we suggest that resilience in the context of PTSD includes personal psychological and biological variables or "strengths" that protect against the development of psychopathology in the face of trauma.

The term "resilience" has been used to describe "the positive pole of individual differences in people's response to stress and adversity" [Rutter, 1987]. Alternative terms have been used, such as "protective factors," "invulnerability" or "hardiness." Most of the

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early research on resilience focused on children determined to be "at risk" for later difficulties in life because of adverse life circumstances such as poverty, loss of family cohesion, and parental mental illness. Researchers followed these children over many years and measured their mental health, social, economic, and occupational status, and identified variables that seemed to promote health and well-being, such as good self-esteem and social support. For example, Rutter and colleagues [1976] examined children living in inner-city London and on the Isle of Wight and measured variables, such as low socioeconomic status, severe marital discord, and paternal criminality. The definition of "resilience" in this area of research was not clearly defined, and referred to personal attributes, as well as environmental factors, but was generally used to refer to the children who had lower rates of psychopathology later in life. Factors associated with resilience defined this way included having an easy temperament, self-mastery, planning skills, and a warm, close relationship with an adult [Rutter 1985, 1987].

Garmezy and colleagues followed children of mentally ill parents, as well as children with behavioral disturbance, for more than 10 years. In writing about this research and reviews of the literature on resilience, Garmezy stated that three types of factors in children at risk promote resilience, or success later in life: (1) temperamental or dispositional factors of the individual, (2) family ties and cohesion, and (3) external support systems [Garmezy 1993; Garmezy et al., 1984].

The first category, temperamental factors, includes items found to be relevant in many studies of resilience and protective factors. For example, a number of studies have found that intelligence or cognitive skills are associated with resilience [Garmezy et al., 1984; Werner, 1989; Zimrin, 1986], whereas other studies have indicated that in times of stress, intelligence can be related to vulnerability; this latter finding has been explained by the hypothesis that intelligent children may have a higher sensitivity to certain types of stress [Luthar, 1991]. Another factor that has been examined is sociability or positive response to others [Garmezy et al., 1984; Rubenstein et al., 1989; Werner, 1989]. Luthar [1991] examined several aspects of sociability in adolescents and found that the most significant protective effect resulted from "social expressiveness," or verbal fluency of communication.

Two widely recognized factors that also fall under this descriptive category are the related concepts of positive self-esteem and an internal locus of control, or a belief in self-efficacy [Luthar, 1991; Rutter, 1987; Werner and Smith, 1982]. Positive self-esteem refers to a sense of self-worth. For example, Zimrin [1986], who followed abused children over 14 years and at follow-up, examined measurements of academic and social success, symptoms of mental illness, and self-reported measures of fulfillment and constructive plans for the future. Subjects who scored higher on the positive measures of functioning at follow-up had higher self-esteem as children.

"Internal locus of control" is defined as the belief that forces shaping one's life are largely within one's control [Luthar, 1991]. This belief may influence a person to make more active attempts to overcome difficult situations. The opposite of this concept is found in the theory of "learned helplessness," in which people who believe themselves to be powerless becomes more passive and restricted in their coping abilities [Abramson et al., 1978]. Luthar [1991] examined inner-city adolescents under varying amounts of life stress, and assessed later academic and interpersonal functioning. She found that a belief in an internal locus of control was protective against stress. This finding has been replicated by others [Werner and Smith, 1982; Zimrin, 1986]. Rutter [1987] wrote that both self-esteem and self-efficacy, or a feeling of control over one's circumstances, are enhanced by close relationships with others, such as parents or spouses. His research on children also revealed that self-esteem and self-efficacy are enhanced by the successful accomplishment of tasks, for example, "taking on positions of responsibility, and success in nonacademic pursuits (sports, music, craftwork)." Factors related to self-esteem and self-efficacy include positivity, hope, or optimism [Zimrin, 1986].

The second and third types of factors listed by Garmezy [1993] involve ties to family and other social support systems, such as school or church. Characteristics of these factors include a feeling of warmth, and supportiveness and closeness in the family or other social structure, along with "the presence of some caring adult," including a neighbor, parents of peers, or a teacher; the protective effect of family and familylike relationships was confirmed by additional research [Bifulco et al., 1987; Werner, 1989]. These resilience factors are summarized in Table 1.

Resilience has also been examined in adults. Early research measured resilience in populations with significant medical problems or undergoing stressful life experiences. Kobasa [1979b] introduced the concept of "hardiness," which has been defined as a stable personality resource that consists of three psychological attitudes and cognitions: commitment, challenge, and control. "Commitment" refers to an ability to turn events into something meaningful and important; "control" refers to the belief that, with effort, individuals can influence the course of events around them, and "challenge" refers to a belief that fulfillment in life results from the growth and wisdom gained from difficult or challenging experiences [Maddi and Khoshaba, 1994]. The composite variable of hardiness has been assessed with various scales measuring these three factors in a number of populations, such as adults experiencing stressful life events [Bartone et al., 1989; Ford et al., 2000; Lang et al., 2003; Northouse et al., 2002; Williams and Lawler, 2001]. For example, in a study examining patients with HIV-AIDS, a high

TABLE 1. Resilience factors in high-risk children

Stress	Population	Outcome protected against	Resilience factors
Physical abuse leading to hospitalization	Children	Poor "adjustment" (low school and work achievement, psychopathology)	Feeling of control (internal locus of control), self-esteem, cognitive abilities, hope and fantasy (mental images of safety) [Zimrin, 1986]
"High-risk": poverty, parental psychopathology, family discord	Children	Delinquency, mental health problems, teenage pregnancy	Positive self-concept, internal locus of control, sociability, social support outside of family [Werner, 1992]
Family discord, parental psychopathology, overcrowding due to poverty	Children from low-income families	Delinquency	Close relationship with an adult, positive self-concept, taking responsibilities for younger siblings, easy temperament, self-mastery, planning skills [Rutter, 1985]
Childhood psychiatric disorder	Children	Worse symptoms at follow-up	Family cohesion (not being placed in temporary state care) [Rutter et al., 1976]
Chronic stress (poverty, parental disability, illnesses)	Children with mentally ill parents or with behavioral problems	Low "competence" (poor school and social performance)	Higher IQ, higher socioeconomic status, family cohesion, external support systems [Garmezy et al., 1984]
Individual life stressors, poverty, disruptive events	Inner-city adolescents	Poor competence (academic performance, interpersonal performance)	Intelligence, internal locus of control, social skills (verbal fluency of communication), ego development, positive life events [Luthar, 1991]
Reared in institution	Girls	Teenage pregnancy, poorer functioning in adulthood (unstable marriage, job)	Close, confiding relationships, future planning, self-esteem, self-efficacy [Rutter, 1987]

score in hardiness was associated with lower psychological distress and higher quality of life [Farber et al., 2000]. In another study, hardiness was associated with decreased time loss due to injury in athletes [Ford et al., 2000]. Several of the factors in the construct of hardiness overlap with attributes listed in the developmental psychology literature cited earlier. For example, the personality style of "control," the feeling that one can influence the course of events around him or her, is similar to the concept of internal locus of control described earlier; aspects of "challenge" sound very much like "optimism."

RESILIENCE AND PTSD

More recently, the term "resilience" has been used in the context of acute trauma, such as combat, assault, accidents, or natural disasters. In this context, resilient individuals are those who experience a trauma but do not develop PTSD. About 50–60% of Americans are exposed to significant traumatic events over the course of their lifetime; of those exposed, 8–20% develop PTSD [Kessler et al., 1995]. We now review some of the factors already discussed as contributing to resilience that are also relevant in this context.

RISK FACTORS FOR PTSD

A number of risk factors for PTSD have been identified and include pretrauma, peritrauma, and posttrauma variables. Pretrauma variables exist before the trauma occurs. Examples include lower educational level [Kessler et al., 1999]; lower intelligence [Orr

et al., 1998]; neurodevelopmental delays, such as delayed onset of walking and speech, as well as learning disabilities [Orr et al., 1998]; previous history of mental disorders [Kessler et al., 1999]; and female gender [Brewin et al., 2000]. Relevant peritraumatic variables include the magnitude of the stressor [Carlier et al., 1997] and immediate reactions to the stressor, such as fear of threats to one's safety [Basoglu et al., 2005] or dissociation [Marmar et al., 1994]. Pertinent posttraumatic variables include perceived social support [King et al., 1998; Koenen et al., 2003], subsequent life stress [Green and Berlin, 1987], and ongoing threat to safety [Basoglu et al., 2005].

Brewin et al. [2000] performed a meta-analysis of PTSD risk factors identified in 85 different studies. Effect sizes were combined to provide scores representing the strength of each predictive effect. They found the strongest combined effect sizes for (in order) (1) lack of perceived social support, (2) subsequent life stress, (3) trauma severity, (4) adverse childhood, and (5) low intelligence.

Peritraumatic dissociation has also been suggested as a possible predictor and risk factor for PTSD. In a meta-analysis of studies on PTSD, Ozer et al. [2003] found that, of the seven predictors examined, peritraumatic dissociation had the largest effect size for predicting PTSD symptoms. In a study of crew members who survived a flooding and fire incident on a submarine, investigators found that peritraumatic dissociation was associated with an increased likelihood of subsequent PTSD [Berg et al., 2005], and in young adult survivors of community violence, peritraumatic dissociation was highly correlated with the develop-

ment of PTSD [Marshall and Schell, 2002]. This retrospective research has been criticized on the grounds that it relied on the tenuous ability of patients to recall their dissociative symptoms well after the trauma occurred [Candel and Merckelbach, 2004]. Additionally, some subsequent studies did not find dissociation to be predictive of PTSD. For example, a study evaluating posttraumatic symptoms after the September 11, 2001, terrorist attacks found that higher rates of dissociation did not predict a higher rate of PTSD [Simeon et al., 2003], and Zoellner et al. [2003] reported that dissociative symptoms within 1 month after the trauma did not predict the subsequent development of PTSD in 79 mixed-trauma patients.

RISK FACTORS VERSUS RESILIENCE FACTORS

The question of whether a factor associated with resilience can be considered the converse of a risk factor has been raised. Intuitively, some risk factors seem to tap similar domains as resilience factors, whereas others do not. For example, "social support" could represent a person's ability to gather social resources, a possible "resiliency" characteristic. However, since neurodevelopmental delays in childhood appear to represent a risk factor for the development of PTSD, it does not seem useful to think of *a lack of* neurodevelopmental delays as conferring particular resilience. Similarly, though being female is a risk factor for developing PTSD after a trauma, it does not seem particularly informative to think that being male represents a resiliency factor.

Rutter [1987] has argued that resilience is more than just the "flip-side" of risk factors, but rather represents qualities encompassing process and mechanisms that confer protection. Risk factors "lead directly to disorder," whereas resiliency factors "operate indirectly, with their effects apparent only by virtue of their interactions" with other variables. For example, a positive coping strategy that confers resilience may not be apparent except when a stressful event causes it to be revealed.

Another way to conceptualize whether a variable associated with PTSD is a resiliency or risk factor is to examine whether it influences the positive pole (i.e., creating protective changes), or whether it affects the individual in a negative way, adding harmful influences or removing protective ones. However, even this conceptualization is not without difficulties, because an individual's characteristics can be viewed from multiple perspectives. For example, social support (measured as perceived social support) may be seen either as a potential risk factor for PTSD or as a manifestation of resilience, representing the presence or absence of an individual's ability to garner a critical resource to buffer the impact of traumatic stress.

If we define a resiliency characteristic as a factor that is intrinsic to the individual and that might be modifiable (e.g., unlike gender or neurodevelopmental delays), then we can describe a set of psychological and biological factors that may be interrelated and that confer protection against psychopathology in the face of stress—in this case, PTSD. Using this definition, we can examine the previously described psychological variables, as well as biological variables associated with resilience. Although some variables seem to indicate environmental factors, such as social support and family cohesion, it is individuals' contribution to these factors that confers their status as characteristics of resilience. For example, a resilient person may have the ability to seek and extract support from others, and enhance his or her social support; similarly, the person contributes to the cohesion in his or her family. The examination of group behavior that may confer protection, or "community resilience," is beyond the scope of this review. "Protective factors" in PTSD might include environmental factors outside of the individual's control, such as socioeconomic status and lower levels of subsequent life stress; however, it is difficult to determine the relative contribution that an individual makes to these factors.

PSYCHOLOGICAL RESILIENCE TO PTSD

Psychological resiliency variables with regard to PTSD would ideally be measured before the onset of trauma, and correlated with the subsequent development of PTSD. However, this is almost never the case in empirical studies. Most of the data describe attributes of the segment of the group exposed to the trauma that *did not* end up developing PTSD, or that developed comparatively fewer symptoms. However, in some cases, the presence or absence of resiliency characteristics may be expressed only after a trauma occurs; thus, these characteristics may not be detectable in the unstressed state. Alternatively, it is possible that a protective psychological factor, such as an internal locus of control, may be learnable or improvable after a trauma and before the onset of PTSD a month or more later. Further research is needed in this area.

Coping styles have been examined in traumatized populations, with better outcomes associated with positive or action-oriented styles. For example, Johnsen et al. [2002] measured three dimensions of coping styles in soldiers caught in an avalanche. They found that "task-focused coping" (e.g., "I make a plan of action") *or* "emotion-focused coping" (e.g., "I let my feelings out"), as opposed to an "avoidant coping style" (e.g., "I refuse to believe that it happened"), was associated with lower PTSD symptom scores. Similarly, research examining coping strategies in fire service personnel found that an avoidant coping style was associated with a higher level of PTSD symptoms following trauma exposure [Beaton et al., 1999]; however, retrospective assessment of avoidance as a coping strategy may be confounded by the fact that

avoidance is a common symptom in PTSD and may thus represent a result of PTSD rather than a predictor. Other coping strategies were nonsignificantly associated with protection from PTSD symptoms, such as "cognitive positive self-talk" (i.e. "I remind myself that I am providing help" and "I remind myself that things could be worse"). In a study of women who had been assaulted, Valentiner et al. [1996] correlated PTSD symptoms with higher scores on a factor construct that they called "wishful thinking" (i.e., "You wished that you could change the way you felt") and negatively correlated with "positive distancing" (i.e., "You accepted the next best thing to what you wanted"). Hope, optimism, and religious behavior (i.e., prayer and faith), were also associated with fewer symptoms of anxiety and depression after the September 11, 2001, events, although PTSD symptoms were not specifically assessed [Ai et al., 2005b].

Another variable also associated with resilience in the child development and adult life stress literature reviewed earlier is the belief in an internal locus of control, which is one of the three concepts measured in the Hardiness Scale [Kobasa, 1979b]. An internal locus of control (feeling that one can generally influence one's life circumstances) has been associated with lower levels of PTSD symptoms in several [Bolstad and Zinbarg, 1997; Casella and Motta, 1990; Kuterovac-Jagodic, 2003; Marmar et al., 1996; Soet et al., 2003] but not all [Kilpatrick and Williams, 1998] studies. For example, locus of control was measured prior to trauma in a study of women going through childbirth [Soet et al., 2003]. Out of 103 subjects, 14 women reported PTSD symptoms from the experience 1 month later. Greater premorbid internal locus of control significantly predicted the lack of PTSD symptoms [Soet et al., 2003]. Similarly, in another study, children with higher levels of internal locus of control who were exposed to war-related events such as violence or loss of home had lower rates of PTSD symptoms at a 3-year follow-up [Kuterovac-Jagodic, 2003]. Similarly, the concept of "hardiness" we discussed earlier includes the measurement of perceived control as a critical cognitive variable. An examination of hardiness in a group of Vietnam veterans after war-related trauma was associated with a lower likelihood of developing PTSD symptoms [King et al., 1998]. Consistent with the concept of locus of control, a "sense of control" over the specific traumatic situation may also protect against PTSD. Although this has not been prospectively tested, it has been examined in individuals undergoing treatment for PTSD and is associated with improved maintenance of treatment gains [Livanou et al., 2002].

Psychological preparedness may promote a sense of control over the trauma: In a study examining psychopathology in individuals who were tortured, those who were political activists appeared to have more resilience. These individual were thought to be protected by their commitment to a cause, training in

stoicism, and prior knowledge about torture techniques [Basoglu et al., 1997]. Other researchers have also found that prior training in emergency work appears to enhance resilience [Alvarez and Hunt, 2005; Hagh-Shenas et al., 2005].

Similar to findings in the childhood resiliency research, multiple studies of adult trauma have found that perceived social support and family cohesion are associated with greater resilience [King et al., 1998; Koenen et al., 2003; Perry et al., 1992], although some authors did not find this effect [e.g., Hyman, 2004]. For example, firefighters were asked about various job-related traumas and the amount of social support they received from their employer, union, family, spouse, and friends. Perceived support from all sources examined was associated with lower psychological distress [Regehr et al., 2000]. Similarly, King et al. [1998], who examined resilience variables in a group of 1,632 Vietnam veterans, found that several factors, including higher levels of both perceived ("functional") and structural (i.e., membership in organizations) social support were associated with a lower likelihood of PTSD. In a study of Kosovan Albanian refugees living in England [Turner et al., 2003], family cohesion was associated with lower risk for PTSD symptoms. Although these factors may appear to be protective factors rather than resiliency factors, outside of the control of the individual, there is evidence that the individual's active engagement in relationships, rather than just the increased availability of support, helps mediate the protection from PTSD. In the firefighter study described earlier, relational capacity was strongly associated with lack of symptoms, and high scores on the interpersonal styles of insecurity and alienation were related to PTSD symptoms, suggesting that the protection against symptoms involves the individual's ability to use and sustain such support [Regehr et al., 2000]. This is similar to findings in the child development literature that positive interpersonal response to others is associated with resilience to stressful life events [Garmezy et al., 1984; Werner, 1989]. However, as with most variables in the trauma literature, perceived social support has generally been measured after the trauma occurred; this limitation presents the possible confound that the disorder interferes with the individual's ability to activate and utilize social support.

Previous exposure to trauma or stress has a complicated relationship to resilience. A history of prior exposure to trauma, such as child abuse, is generally associated with the development of more severe PTSD symptoms after a new trauma [Chang et al., 2005; Fullerton et al., 2004]. However, it appears that certain types of exposure to stress may have a protective effect for individuals later exposed to new trauma. Rutter [1987] writes that the impact of the stress depends on how the individual experiences it. He states that "protection [from adverse sequelae] in this case resides not in the evasion of the risk, but

TABLE 2. Resilience factors in PTSD studies

Stress	Population	Resilience factors
Fleeing homeland	Adult refugees	Family cohesion [Turner et al., 2003]
Avalanche	Soldiers	Task-focused or emotion-focused coping style, as opposed to avoidant coping style [Johnsen et al., 2002]
Assault	Women	Coping styles using "positive distancing" vs. 'wishful thinking' [Valentiner et al., 1996]
War-zone stressors	Vietnam veterans	Resilience–recovery factors: hardiness (control, commitment, challenge), social support, fewer additional stressful life events [King et al., 1998, 1999]
Torture	Adults	Psychological preparedness for arrest and torture (i.e., prior knowledge of torture events, training in stoicism) [Basoglu et al., 1997]
Childbirth trauma	Women (pre- and postchildbirth)	Internal locus of control [Soet et al., 2003]
War-related events	Children	Internal locus of control [Kuterovac-Jagodic, 2003]
Firefighters	Work-related traumatic events	Ability to use and sustain social support, higher number of traumatic exposures (with the effect of increasing internal locus of control and self-efficacy) [Regehr et al., 2000]
Burn injury	Adults	Perceived social support [Perry et al., 1992]
September 11th attacks in NY	Adults	Hope and optimism [Ai et al., 2005a] and religious behavior [Ai et al., 2005b]

in successful engagement with it." Interestingly, in one of the studies of firefighters cited earlier, Regehr et al. [2000] found that a higher number of traumatic exposures actually increased subjects' sense of internal locus of control and self-efficacy. Thus, trauma exposure may represent a protective rather than vulnerability factor for adverse posttraumatic sequelae, if it is associated with an increased sense of mastery or growth. Table 2 summarizes some of the resilience factors that have been identified in the PTSD studies we have mentioned.

BIOLOGICAL MEASURES OF RESILIENCE

A growing body of research has focused on determining physiological or biological differences in individuals who experienced a trauma and either developed or did not develop PTSD. Similar to the studies of psychological variables, studies of biological variables have relied mostly on assessments of groups of people only after a traumatic event has occurred. However, determination of true biological resilience factors would involve assessing these variables *before* the onset of a trauma. Unfortunately, very few prospectively derived data exist. Most studies have examined differences in individuals with PTSD compared to a normal population; few data exist examining the relative differences in a trauma-exposed population that did not develop PTSD. However, examination of the neurobiology of PTSD may inform our understanding of the critical factors underlying resilience and shape future lines of inquiry.

Hypothalamic–pituitary–adrenal (HPA) axis. Much of the research on the pathophysiology of PTSD has focused on dysregulation of the HPA axis. Evidence suggesting a role for HPA axis dysregulation in the development of PTSD is supported by clinical studies demonstrating decreased 24-hour urinary cortisol levels in subjects with PTSD. For example, Yehuda et al. [1995] examined 22 Holocaust survivors with PTSD and 25 without PTSD. Subjects with PTSD had an average of 32.6 μg of cortisol in their 24-hour urine sample, compared to 62.7 μg of cortisol in those without PTSD. This finding replicates data from a number of studies [Mason et al., 1986; Yehuda et al., 1990, 1993], although other studies have reported higher than normal cortisol levels in individuals with PTSD [De Bellis et al., 1999; Lemieux and Coe, 1995; Maes et al., 1998; Pitman and Orr, 1990; Rasmusson et al., 2001]; in all, nine of the 12 studies examining 24-hour urinary cortisol have found abnormal cortisol levels, either high or low, in patients with PTSD. The discrepancies in findings reported in different studies may be attributable, in part, to differences in illness severity among the patient populations studied: with lower cortisol levels associated with more severely ill patients (i.e., hospitalized) and higher cortisol levels associated with less ill patients (i.e., outpatients; Rasmusson et al., 2003]. Other factors contributing to these discrepancies include differences in the timing of measurements relative to the acuity of the disorder (acute vs. chronic), and differences in measurement techniques, with some researchers measuring plasma or saliva cortisol at fixed times during the day, whereas others measured it after waking or after performance of a dexamethasone suppression test. However, even studies employing similar methodology have yielded discrepant results, suggesting the potential importance of broadening the area of inquiry to include alternative measurements of neuroendocrine function.

Relevant to the issue of cortisol and PTSD are results from the study by Schelling et al. [2001], in which hydrocortisone administration to patients shortly after the onset of septic shock decreased the subsequent development of PTSD symptoms. This

seems to indicate a protective effect of glucocorticoids at the time of trauma. Reduction in the development of PTSD associated with hydrocortisone administration has been replicated in patients undergoing cardiac surgery [Schelling et al., 2004] and was postulated to confer a protective effect by interfering with retrieval of traumatic memories, an effect of glucocorticoids that has been demonstrated in animal models [Roozendaal, 2003]. This observation led to a small trial in which cortisol was used as a treatment for chronic PTSD. Three patients were administered low-dose (10 mg/day) cortisol orally in a 3-month, placebo-controlled, crossover design study. All three patients experienced reductions in reexperiencing symptoms, and one had decreased avoidance symptoms [Aerni et al., 2004].

In addition to cortisol, dehydroepiandrosterone (DHEA) and its metabolite dehydroepiandrosterone sulfate (DHEAS) are also adrenal gland products that are secreted in response to ACTH. Animal studies have indicated that DHEAS is released under stressful conditions and exhibits memory-enhancing, anti-depressant, anxiolytic, and antiaggression properties [Morgan et al., 2004]. In cell culture experiments, DHEA and DHEAS enhanced neuronal survival in the face of *N*-methyl-D-aspartate (NMDA) receptor–induced toxicity, leading the authors to conclude that DHEA and DHEAS are important protectors of cells undergoing damage or stress [Kimonides et al., 1998]. These studies suggest that DHEA and DHEAS are involved in modulating an organism's vulnerability to stress. Studies examining levels of DHEA and DHEAS in PTSD have also generated mixed results; some researchers have found higher than normal levels in subjects with PTSD, whereas others found lower than normal levels in subjects with PTSD and depression [Sondergaard et al., 2002; Spivak et al., 2000].

DHEA appears to have an antagonistic relationship with cortisol. Whereas cortisol is a glucocorticoid, DHEA has antiglucocorticoid properties that appear to protect neurons [Blauer et al., 1991; Browne et al., 1992]. For example, research on metabolites of DHEA in the brain suggests that they interfere with uptake of activated glucocorticoid receptors [Morfin and Starka, 2001]. In addition, administration of DHEA in humans resulted in a decrease of plasma cortisol [Kroboth et al., 2003]. Given this antagonistic relationship of DHEA(S) with cortisol, and because cortisol is associated with improved emotional memory encoding [Buchanan and Lovallo, 2001; Cordero et al., 2002], DHEA(S) may antagonize these enhanced memory effects, resulting in decreased PTSD symptoms of reexperiencing, such as nightmares or flashbacks. Because of the antagonistic relationship, levels of DHEA(S) may be more meaningful when examined in relationship with cortisol—indeed, preclinical and clinical data have found the DHEAS:cortisol ratio associated with improved functioning [Morgan et al., 2004]. For example, in a study of HIV+ men,

psychotherapy improved the DHEA:cortisol decline seen in this population [Cruess et al., 1999]. In a recent study of soldiers enrolled in military survival school, Morgan et al. [2004] found that DHEAS:cortisol ratios during stress were higher in those subjects who were able to perform better under intense stress.

Catecholamines. The stress response involves both the sympathetic nervous system and the HPA axis; consequently, there has also been an interest in norepinephrine and epinephrine in patients with PTSD. For example, Kosten et al. [1987] measured urinary norepinephrine and epinephrine levels in hospitalized patients with PTSD, and found higher levels of norepinephrine than in other hospitalized psychiatric control groups with depression, bipolar disorder, and schizophrenia. This finding has been replicated by some [Lemieux and Coe, 1995; Yehuda et al., 1992, 1998] but not all [McFall et al., 1992; Murburg et al., 1995] investigators. Other groups reported an increased release of catecholamines in PTSD subjects after exposure to combat-related stimuli, such as films or evocative sounds [Blanchard et al., 1991; Liberzon et al., 1999; McFall et al., 1990].

A few studies have examined norepinephrine or epinephrine levels at the time of trauma, to investigate their potential relationship with later development of PTSD. Delahanty et al. [2005] examined urinary epinephrine levels in children admitted to the hospital after an acute trauma and found that elevated epinephrine levels were associated with higher levels of PTSD symptoms 6 weeks later. Schelling [2002] reported that patients who received epinephrine as part of their intensive care unit (ICU) medical care had more types of trauma memories 6 months later compared to similarly severely ill patients in the ICU who had not received epinephrine.

Adrenergic stimulation has been shown to enhance memory consolidation in animals and humans. For example, administering amphetamine to human subjects before learning of word lists enhances memory of those words [Soetens et al., 1995]. A significant part of the memory consolidation process appears to continue *after* exposure to the memory task, which is also enhanced by noradrenergic stimulation. For example, Southwick et al. [1993] administered yohimbine, which stimulates norepinephrine release, to subjects *after* they viewed slides of an emotionally arousing story. Increased 3-methoxy-4-hydroxyphenyl-glycol (MHPG, a noradrenergic metabolite) levels were associated with enhanced memory of the story. In another study, among normal subjects, memory was greater for emotionally arousing stories than for neutral stories. Propranolol, a β-adrenergic receptor antagonist, blocked the enhanced memory for emotionally arousing stories [Cahill et al., 1994]. The findings of increased memory consolidation associated with increased adrenergic tone may have significant implications for the pathogenesis of PTSD, which is hypothesized to be attributable to an overconsolidation

of the trauma memory, resulting in the intrusive recollections and reexperiencing symptoms in PTSD [Pitman et al., 2002].

Neuropeptide Y (NPY). NPY, a 36-amino acid peptide that is colocalized with neurons containing norepinephrine, may be a natural resiliency factor for stress. NPY is anxiolytic when administered intraventricularly in animals [Heilig et al., 1989]. NPY administration promotes sleep, as well as decreases cortisol and ACTH production in humans [Antonijevic et al., 2000]. Widdowson et al. [1992] reported lower NPY levels in the brains of individuals who had committed suicide. Individuals trained to remain unperturbed under stress manifest increased levels of NPY after a stressful event: Morgan et al. [2004] measured NPY 24 hours after Special Forces soldiers and non–Special Forces soldiers underwent a simulated prisoner-of-war experience. The Special Forces soldiers regained their normal NPY levels 24 hours later, whereas levels in the non–Special Forces were significantly lower. This finding was hypothesized to suggest that Special Forces soldiers had physiologically adjusted to handle stress better, either as a result of their native endowment or by dint of their training, and the normalized NPY levels served as a marker of this resilience. Supporting this hypothesis, individuals with PTSD have been found to have lower NPY levels at baseline and a blunted yohimbine-induced NPY increase [Rasmusson et al., 2000]. NPY may exert its stress-buffering effect by way of its counterregulatory effects on the locus coeruleus–norepinephrine system (NPY appears to inhibit norepinephrine release) and on corticotropin-releasing hormone [CRH; NPY blocks CRH's anxiogenic effects; Britton et al., 2000]. In addition, NPY may disrupt fearful memory consolidation; injection of NPY in the amygdala of mice impaired memory in a foot shock task [Flood et al., 1989].

Allopregnanolone. Allopregnanolone, another product of the adrenal gland associated with the stress response, may be a potential resiliency factor relevant to the development of PTSD. Stress lowers γ-aminobutyric acid A (GABA-A) transmission, causing neuronal overexcitation; allopregnanolone modulates the GABA-A receptor, counteracting this effect of stress [Higashi et al., 2005]. For example, Bitran et al. [1995] showed that administration of the allopregnanolone precursor progesterone in rats produces a decrease in anxiety similar to that observed after the administration of benzodiazepine compounds, and that allopregnanolone's effects on the GABA-A receptor mediate this effect. Similarly, Higashi et al. [2005] found that allopregnanolone increased in rats exposed to recorded distress cries of other rats, as a putative homeostatic mechanism to counteract stress. Allopregnanolone increased in students undergoing test-taking stress [Droogleever Fortuyn et al., 2004]. Perhaps resilience corresponds, in part, to an ability to increase allopregnanolone levels during stress.

Interestingly, allopregnanolone levels decrease when mice are socially isolated and placed in individual living quarters [Dong et al., 2001]. Because social contact and support in humans promotes resilience and protects against anxiety and depression, this effect may be mediated in part by changes in levels of allopregnanolone and other neuroactive steroids.

MEASUREMENT OF RESILIENCE

Numerous scales for measuring resilience and hardiness have been developed over the years; however, there has been little consensus on which scales most effectively describe and quantitatively evaluate resiliency characteristics.

Some early studies examining resilience in children and adolescents used combinations of scales examining specific resilient characteristics and risk factors, such as locus of control [Luthar, 1991]. Some scales that have been commonly used to measure the locus-of-control component of resilience include the Locus of Control Scale [Nowicki and Strickland, 1973] and the Internal–External Locus of Control Scale [Rotter, 1966]. Other scales have been used collectively to assess competence or coping skills, two qualities that may be related to resilience. Measures of social competence used in Luthar's study [1991] included the Teacher – Child Rating Scale [Hightower et al., 1986], the Revised Class Play Scale [for peer assessment; Masten et al., 1985], and school grades.

In addition, a number of instruments have been developed to measure hardiness—a concept related to resilience—based on Kobasa's [1979b] original description of the core elements of commitment, control, and challenge. As with resilience scales, there has been little consistency either in the scales selected for use in studies or in the way the scales have been applied to measure hardiness [Funk and Houston, 1987]. An early instrument, the Hardiness Scale [Kobasa et al., 1982], comprises five different scales—including the Alienation from Self scale, the Alienation from Work scale, the Powerlessness scale, all three scales from the Alienation Test [Maddi et al., 1979]; the Security scale of the California Life Goals Evaluation Schedule [cited by Kobasa, 1979a]; and the External Locus of Control Scale [Rotter et al., 1962]—all containing items corresponding to different aspects of hardiness. Commitment is measured by the Alienation from Self and Alienation from Work scales; control is measured by the External Locus of Control and Powerlessness scales; and challenge is assessed by the Security scale [Hull et al., 1987]. These scales provide an important context for the resiliency scales, which incorporate the core concepts of hardiness, as previously discussed.

Following criticisms of the design and validity of the original Hardiness Scale [Funk and Houston, 1987; Hull et al., 1987], a new measure, the Personal Views Survey (PVS), was developed by Maddi and Khoshaba [1994]. This new and shorter measure

includes 45 rating-scale items selected from the original composite Hardiness Scale. Selection of items was based on an evaluation of each item's psychometric validity by Bartone et al. [1989]. The scale also contains a smaller number of items (i.e., 45) than the Hardiness Scale (which contains 90 items between the different subscales) and may therefore be a more practical clinical tool. The PVS exhibits a positive correlation with health status [Campbell et al., 1989] and a negative correlation with psychopathological tendencies, as assessed by the Minnesota Multiphasic Personality Inventory [after controlling for negative affectivity; Maddi and Khoshaba, 1994]. Another research group [King et al., 1998] incorporated 11 items that roughly corresponded to the commitment, control, and challenge elements of hardiness into a larger survey, the National Survey of the Vietnam Generation of the National Vietnam Veterans Readjustment Study [NVVRS; Kulka et al., 1990]. They examined the relationship of these measurements with war zone stressors and PTSD symptomatology in Vietnam veterans [King et al., 1998] as part of the NVVRS. As detailed earlier, researchers have created new scales by selecting from the previously established items or by choosing items for larger surveys that correspond to the three hardiness components.

The relationship of hardiness to resilience has not been clearly defined, but the hardiness characteristics of commitment, control, and challenge appear to be considered features of resilience, and resilience scales have used the construct of hardiness as a key point of quantitative evaluation. For example, Connor and Davidson [2003] included the three aspects of hardiness in a resilience scale that is discussed next.

One of the earliest scales to include the term "resilience" in its title was the Resilience Scale [RS; Wagnild and Young, 1993], which includes 25 items and evaluates qualities of personal competence and acceptance of self and life. This scale was used in a study that examined resilience in sheltered, battered women [Humphreys, 2003]. The Resilience Scale for Adults (RSA), independently developed by Friborg et al., contains items related to five components of resilience: personal competence, social competence, family coherence, social support and personal structure [Friborg et al., 2003; Hjemdal et al., 2001]. Each of the five components corresponded to one of the three key categories of resilience described by Werner 1989, 1993], Rutter [1990], and Garmezy [1993]: dispositional attributes, family cohesion, and external support systems, respectively.

The most recently developed instrument for measuring resilience is the 25-item Connor–Davidson Resilience Scale [CD-RISC; Connor and Davidson, 2003]), which incorporates items from earlier resiliency research, including those corresponding to Kobasa's hardiness model and Rutter's list of resilient characteristics from his 1985 review, including self-esteem, "action orientation," adaptability to change, the ability to develop strategies around a clear goal, skillfulness in social problem solving, sense of humor during stressful events, and a number of other items. In addition, the scale also measures patience and endurance as introduced by Lyons [1991], as well as components of "faith" and "belief in benevolent intervention" or "optimism" adapted from the biographical literature describing the explorer Shackleton's expeditions [Connor and Davidson, 2003]. Subject responses to the list of 25 statements cover a 5-point range, from *not true at all* to *true nearly all the time*. This scale was validated with samples of the general population, primary care outpatients, psychiatric outpatients, patients with generalized anxiety disorder, and patients with PTSD [Connor and Davidson, 2003]. Ideally, a scale of resilience would be examined in a trauma-exposed control group that did *not* develop PTSD, because these are probably the individuals most likely to be truly resilient. Unfortunately, data assessing resilience instruments in this population are not available. To give a rough estimate of the frequency of use of these resilience and hardiness scales in the psychiatric literature, the number of Medline abstract mentions is provided in Table 3.

The variety of approaches that have attempted to capture the concept of resilience reflects the difficulty of defining this notion. The *American Heritage Dictionary* [2000] defines *resilience* as "the ability to recover quickly from illness, change, or misfortune; buoyancy." From this definition of resilience, which incorporates the idea of a response to some event or situation, the question emerges about whether resilience can be measured by a set of questions at a single point in time, as opposed to observing a subject during a stressful experience and then determining how well he or she returns to normal functioning. Otherwise, a set of questions at a single time point may end up only capturing state characteristics, such as positive attitudes or mood. Using this reasoning, a true resilience scale would measure an individual's reaction to an experimental stress paradigm or to stressful life events or traumas over time. With PTSD, a resiliency-measuring instrument would need to be administered to subjects prior to experiencing a trauma and then demonstrated to predict resistance to the development of stress-related disorders. Unfortunately, these kinds of prospectively derived data are not currently available.

In addition, it is not clear whether a resiliency scale can truly measure improvement in resilience, independent from improvement in symptoms of PTSD or other disorders, because the term "resilience" defines individuals who were protected from developing the disorder in the first place. For example, it may not be valid to describe subjects who improve after pharmacological or psychotherapeutic treatment as "more resilient," separately from simply being successfully treated for their disorder. To use the term "resilience" in this context, the measurement of resilience should

TABLE 3. Resilience scales

Resilience scale	Number of items	Core concepts	Frequency of use[a]
CD-RISC [Connor and Davidson, 2003]	25	Hardiness (control, commitment, change viewed as challenge), developing strategy with a clear goal, action orientation, strong self-esteem, adaptability to change, social problem-solving skills, humor, strengthening effect of stress, acceptance of responsibility in dealing with stress, secure emotional relationships, previous achievement, patience, ability to endure stress, faith, belief in benevolent intervention	2
HS [Kobasa, 1979b]; (composed of previously validated subscales)—a newer version of the scale is the PVS, also known as the HardiSurvey	90 (varies for PVS and HardiSurvey)	Subscales: Alienation from Self scale and Alienation from Work scale (commitment); Security and Cognitive Structure (challenge); Powerlessness scale and External Locus of Control scale (control)	15
RS [Wagnild and Young, 1993]	25	Personal competence, acceptance of Self and Life	5
RSA [Friborg et al., 2003]	45	Personal competence, social competence, Family coherence, social support, personal structure	0

[a]Number of studies using the scale according to Medline abstract searches.

show that the subjects are better able to handle stress or trauma, independent of their treatment response.

CONCLUSION

Many variables associated with the absence of PTSD are considered features of "resilience." This term has been used in various contexts and populations, and a clarification of the meaning of this term in the context of PTSD would be helpful. In this article, we propose that the term "resilience" refers to psychological or biological factors that may have been alterable at some point, and that confer protection from PTSD. Many psychosocial variables, including positive or action-oriented coping styles, internal locus of control, cognitive abilities, and social support, have been examined in individuals who have experienced trauma or other hardship. Several biological variables appear to be promising lines of inquiry for further research of resilience, such as DHEA(S), allopregnanolone, and NPY. As more knowledge is gained in the area, possible connections between the psychological and biological variables can be explored.

Ideally, these variables should be measured prospectively, before a trauma, or before the onset of PTSD, then correlated with rates of later PTSD symptoms. In addition, the use of the term "resilience" is less meaningful when applied to changes seen after successful treatment of a psychiatric disorder; it does not seem valid to link the idea of resilience to decreasing symptoms without establishing that that a decrease in symptoms is associated with a better ability to handle stress or trauma. For example, a medication treatment study showing an improvement in symptoms does not necessarily demonstrate that resilience is enhanced, as any measures of resilience examined in the absence of further trauma exposure may be confounded with the treatment effect. Indeed, it is rare to find a study showing additional trauma exposure following treatment for PTSD symptoms. In one study however, earthquake survivors treated successfully with cognitive-behavioral therapy (CBT) maintained therapeutic gains despite ongoing earthquake aftershocks, suggesting a possible enhancement of resilience, although exposure to aftershocks was not systematically measured [Basoglu et al., 2003]. Researchers have also examined the effect of early treatment after a trauma (i.e., before the onset of PTSD, but after the individual has started to experience some traumatic stress symptoms). In several studies, CBT, which included education, exposure, cognitive restructuring, and relaxation training, decreased PTSD symptoms more than did supportive counseling [Ehlers and Clark, 2003].

Further research is warranted to better explore the relationships between such interventions and later symptoms. In addition, prospective studies should examine psychological and biological resilience characteristics in populations likely to experience trauma, or in the interval between trauma exposure and symptom development. These approaches will help define resilience characteristics and guide the development of interventions to enhance them.

REFERENCES

2000. The American Heritage Dictionary of the English Language. 4th ed. Boston: Houghton Mifflin.

Abramson LY, Seligman ME, Teasdale JD. 1978. Learned help-lessness in humans: Critique and reformulation. J Abnorm Psychol 87:49–74.

Aerni A, Traber R, Hock C, Roozendaal B, Schelling G, Papassotiropoulos A, Nitsch RM, Schnyder U, de Quervain DJ. 2004. Low-dose cortisol for symptoms of posttraumatic stress disorder. Am J Psychiatry 161:1488–1490.

Ai AL, Cascio T, Santangelo LK, Evans-Campbell T. 2005a. Hope, meaning, and growth following the September 11, 2001, terrorist attacks. J Interpers Violence 20:523–548.

Ai AL, Tice TN, Peterson C, Huang B. 2005b. Prayers, spiritual support, and positive attitudes in coping with the September 11 national crisis. J Pers 73:763–791.

Alvarez J, Hunt M. 2005. Risk and resilience in canine search and rescue handlers after 9/11. J Trauma Stress 18:497–505.

Antonijevic IA, Murck H, Bohlhalter S, Frieboes RM, Holsboer F, Steiger A. 2000. Neuropeptide Y promotes sleep and inhibits ACTH and cortisol release in young men. Neuropharmacology 39:1474–1481.

Bartone PT, Ursano RJ, Wright KM, Ingraham LH. 1989. The impact of a military air disaster on the health of assistance workers: A prospective study. J Nerv Ment Dis 177:317–328.

Basoglu M, Livanou M, Crnobaric C, Franciskovic T, Suljic E, Duric D, Vranesic M. 2005. Psychiatric and cognitive effects of war in former Yugoslavia: Association of lack of redress for trauma and posttraumatic stress reactions. JAMA 294:580–590.

Basoglu M, Livanou M, Salcioglu E, Kalender D. 2003. A brief behavioural treatment of chronic post-traumatic stress disorder in earthquake survivors: Results from an open clinical trial. Psychol Med 33:647–654.

Basoglu M, Mineka S, Paker M, Aker T, Livanou M, Gok S. 1997. Psychological preparedness for trauma as a protective factor in survivors of torture. Psychol Med 27:1421–1433.

Beaton R, Murphy S, Johnson C, Pike K, Corneil W. 1999. Coping responses and posttraumatic stress symptomatology in urban fire service personnel. J Trauma Stress 12:293–308.

Berg JS, Grieger TA, Spira JL. 2005. Psychiatric symptoms and cognitive appraisal following the near sinking of a research submarine. Mil Med 170:44–47.

Bifulco AT, Brown GW, Harris TO. 1987. Childhood loss of parent, lack of adequate parental care and adult depression: A replication. J Affect Disord 12:115–128.

Bitran D, Shiekh M, McLeod M. 1995. Anxiolytic effect of progesterone is mediated by the neurosteroid allopregnanolone at brain GABAA receptors. J Neuroendocrinol 7:171–177.

Blanchard EB, Kolb LC, Prins A, Gates S, McCoy GC. 1991. Changes in plasma norepinephrine to combat-related stimuli among Vietnam veterans with posttraumatic stress disorder. J Nerv Ment Dis 179:371–373.

Blauer KL, Poth M, Rogers WM, Bernton EW. 1991. Dehydroepiandrosterone antagonizes the suppressive effects of dexamethasone on lymphocyte proliferation. Endocrinology 129:3174–3179.

Bolstad BR, Zinbarg RE. 1997. Sexual victimization, generalized perception of control, and posttraumatic stress disorder symptom severity. J Anxiety Disord 11:523–540.

Brewin CR, Andrews B, Valentine JD. 2000. Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. J Consult Clin Psychol 68:748–766.

Britton KT, Akwa Y, Spina MG, Koob GF. 2000. Neuropeptide Y blocks anxiogenic-like behavioral action of corticotropin-releasing factor in an operant conflict test and elevated plus maze. Peptides 21:37–44.

Browne ES, Wright BE, Porter JR, Svec F. 1992. Dehydroepiandrosterone: Antiglucocorticoid action in mice. Am J Med Sci 303:366–371.

Buchanan TW, Lovallo WR. 2001. Enhanced memory for emotional material following stress-level cortisol treatment in humans. Psychoneuroendocrinology 26:307–317.

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

Campbell J, Amerikaner M, Swank P, Vincent K. 1989. The relationship between the Hardiness Test and the Personal Orientation Inventory. J Res Pers 23:373–380.

Candel I, Merckelbach H. 2004. Peritraumatic dissociation as a predictor of post-traumatic stress disorder: A critical review. Compr Psychiatry 45:44–50.

Carlier IV, Lamberts RD, Gersons BP. 1997. Risk factors for posttraumatic stress symptomatology in police officers: A prospective analysis. J Nerv Ment Dis 185:498–506.

Casella L, Motta RW. 1990. Comparison of characteristics of Vietnam veterans with and without posttraumatic stress disorder. Psychol Rep 67:595–605.

Chang CM, Connor KM, Lai TJ, Lee LC, Davidson JR. 2005. Predictors of posttraumatic outcomes following the 1999 Taiwan earthquake. J Nerv Ment Dis 193:40–46.

Connor KM, Davidson JR. 2003. Development of a new resilience scale: The Connor–Davidson Resilience Scale (CD-RISC). Depress Anxiety 18:76–82.

Cordero MI, Kruyt ND, Merino JJ, Sandi C. 2002. Glucocorticoid involvement in memory formation in a rat model for traumatic memory. Stress 5:73–79.

Cruess DG, Antoni MH, Kumar M, Ironson G, McCabe P, Fernandez JB, Fletcher M, Schneiderman N. 1999. Cognitive-behavioral stress management buffers decreases in dehydroepiandrosterone sulfate (DHEA-S) and increases in the cortisol/DHEA-S ratio and reduces mood disturbance and perceived stress among HIV-seropositive men. Psychoneuroendocrinology 24:537–549.

De Bellis MD, Baum AS, Birmaher B, Keshavan MS, Eccard CH, Boring AM, Jenkins FJ, Ryan ND. 1999. Developmental traumatology: Part I. Biological stress systems. Biol Psychiatry 45:1259–1270.

Delahanty DL, Nugent NR, Christopher NC, Walsh M. 2005. Initial urinary epinephrine and cortisol levels predict acute PTSD symptoms in child trauma victims. Psychoneuroendocrinology 30:121–128.

Dong E, Matsumoto K, Uzunova V, Sugaya I, Takahata H, Nomura H, Watanabe H, Costa E, Guidotti A. 2001. Brain 5-alpha-dihydroprogesterone and allopregnanolone synthesis in a mouse model of protracted social isolation. Proc Natl Acad Sci USA 98:2849–2854.

Droogleever Fortuyn HA, van Broekhoven F, Span PN, Backstrom T, Zitman FG, Verkes RJ. 2004. Effects of PhD examination stress on allopregnanolone and cortisol plasma levels and peripheral benzodiazepine receptor density. Psychoneuroendocrinology 29:1341–1344.

Ehlers A, Clark D. 2003. Early psychological interventions for adult survivors of trauma: A review. Biol Psychiatry 53:817–826.

Farber EW, Schwartz JA, Schaper PE, Moonen DJ, McDaniel JS. 2000. Resilience factors associated with adaptation to HIV disease. Psychosomatics 41:140–146.

Flood JF, Baker ML, Hernandez EN, Morley JE. 1989. Modulation of memory processing by neuropeptide Y varies with brain injection site. Brain Res 503:73–82.

Ford IW, Eklund RC, Gordon S. 2000. An examination of psychosocial variables moderating the relationship between life stress and injury time-loss among athletes of a high standard. J Sports Sci 18:301–312.

Friborg O, Hjemdal O, Rosenvinge JH. 2003. A new rating scale for adult resilience: What are the central protective resources behind healthy adjustment? Int J Methods Psychiatr Res 12:65–76.

Fullerton CS, Ursano RJ, Wang L. 2004. Acute stress disorder, posttraumatic stress disorder, and depression in disaster or rescue workers. Am J Psychiatry 161:1370–1376.

Funk SC, Houston BK. 1987. A critical analysis of the Hardiness Scale's validity and utility. J Pers Soc Psychol 53:572–578.

Garmezy N. 1993. Children in poverty: Resilience despite risk. Psychiatry 56:127–136.

Garmezy N, Masten AS, Tellegen A. 1984. The study of stress and competence in children: A building block for developmental psychopathology. Child Dev 55:97–111.

Green MA, Berlin MA. 1987. Five psychosocial variables related to the existence of post-traumatic stress disorder symptoms. J Clin Psychol 43:643–649.

Hagh-Shenas H, Goodarzi MA, Dehbozorgi G, Farashbandi H. 2005. Psychological consequences of the Bam earthquake on professional and nonprofessional helpers. J Trauma Stress 18: 477–483.

Heilig M, Soderpalm B, Engel JA, Widerlov E. 1989. Centrally administered neuropeptide Y (NPY) produces anxiolytic-like effects in animal anxiety models. Psychopharmacology (Berl) 98: 524–529.

Higashi T, Takido N, Shimada K. 2005. Studies on neurosteroids: XVII. Analysis of stress-induced changes in neurosteroid levels in rat brains using liquid chromatography-electron capture atmospheric pressure chemical ionization-mass spectrometry. Steroids 70:1–11.

Hightower AD, Work WC, Cowen EL, Lotyczewski BS, Spinell AP, Guare JC, Rohrbeck CA. 1986. The Teacher–Child Rating Scale: A brief objective measure of elementary school children's school problem behaviors and competencies. School Psychol 15:393–409.

Hjemdal O, Friborg O, Martinussen M, Rosenvinge JH. 2001. Preliminary results from the development and validation of a Norwegian scale for measuring adult resilience. J Norw Psychol Ass 38:310–317.

Hull JG, Van Treuren RR, Virnelli S. 1987. Hardiness and health: A critique and alternative approach. J Pers Soc Psychol 53:518–530.

Humphreys J. 2003. Resilience in sheltered battered women. Issues Ment Health Nurs 24:137–152.

Hyman O. 2004. Perceived social support and secondary traumatic stress symptoms in emergency responders. J Trauma Stress 17: 149–156.

Johnsen BH, Eid J, Laberg JC, Thayer JF. 2002. The effect of sensitization and coping style on post-traumatic stress symptoms and quality of life: Two longitudinal studies. Scand J Psychol 43: 181–188.

Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. 1995. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry 52:1048–1060.

Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB, Breslau N. 1999. Epidemiological risk factors for trauma and PTSD. In: Yehuda R, editor. Risk factors for post traumatic stress disorder. Washington, DC: American Psychiatric Association Press. p 23–59.

Kilpatrick KL, Williams LM. 1998. Potential mediators of post-traumatic stress disorder in child witnesses to domestic violence. Child Abuse Negl 22:319–330.

Kimonides VG, Khatibi NH, Svendsen CN, Sofroniew MV, Herbert J. 1998. Dehydroepiandrosterone (DHEA) and DHEA-sulfate (DHEAS) protect hippocampal neurons against excitatory amino acid-induced neurotoxicity. Proc Natl Acad Sci USA 95: 1852–1857.

King DW, King LA, Foy DW, Keane TM, Fairbank JA. 1999. Posttraumatic stress disorder in a national sample of female and male Vietnam veterans: Risk factors, war-zone stressors, and resilience–recovery variables. J Abnorm Psychol 108:164–170.

King LA, King DW, Fairbank JA, Keane TM, Adams GA. 1998. Resilience–recovery factors in post-traumatic stress disorder among female and male Vietnam veterans: Hardiness, postwar social support, and additional stressful life events. J Pers Soc Psychol 74:420–434.

Kobasa SC. 1979a. Personality and resistance to illness. Am J Community Psychol 7:413–423.

Kobasa SC. 1979b. Stressful life events, personality, and health: An inquiry into hardiness. J Pers Soc Psychol 37:1–11.

Kobasa SC, Maddi SR, Kahn S. 1982. Hardiness and health: A prospective study. J Pers Soc Psychol 42:168–177.

Koenen KC, Stellman JM, Stellman SD, Sommer JF Jr. 2003. Risk factors for course of posttraumatic stress disorder among Vietnam veterans: A 14-year follow-up of American Legionnaires. J Consult Clin Psychol 71:980–986.

Kosten TR, Mason JW, Giller EL, Ostroff RB, Harkness L. 1987. Sustained urinary norepinephrine and epinephrine elevation in post-traumatic stress disorder. Psychoneuroendocrinology 12: 13–20.

Kroboth PD, Amico JA, Stone RA, Folan M, Frye RF, Kroboth FJ, Bigos KL, Fabian TJ, Linares AM, Pollock BG, Hakala C. 2003. Influence of DHEA administration on 24-hour cortisol concentrations. J Clin Psychopharmacol 23:96–99.

Kulka RA, Schlenger WE, Fairbank JA, Hough RL, Jordan BK, Marmar CR, Weiss DS. 1990. Trauma and the Vietnam War generation: Report on the findings from the National Vietnam Veterans Readjustment Study. New York: Brunner/Mazel.

Kuterovac-Jagodic G. 2003. Posttraumatic stress symptoms in Croatian children exposed to war: A prospective study. J Clin Psychol 59:9–25.

Lang A, Goulet C, Amsel R. 2003. Lang and Goulet Hardiness Scale: Development and testing on bereaved parents following the death of their fetus/infant. Death Stud 27:851–880.

Lemieux AM, Coe CL. 1995. Abuse-related posttraumatic stress disorder: Evidence for chronic neuroendocrine activation in women. Psychosom Med 57:105–115.

Liberzon I, Abelson JL, Flagel SB, Raz J, Young EA. 1999. Neuroendocrine and psychophysiologic responses in PTSD: A symptom provocation study. Neuropsychopharmacology 21: 40–50.

Livanou M, Basoglu M, Marks IM, De SP, Noshirvani H, Lovell K, Thrasher S. 2002. Beliefs, sense of control and treatment outcome in post-traumatic stress disorder. Psychol Med 32:157–165.

Luthar SS. 1991. Vulnerability and resilience: A study of high-risk adolescents. Child Dev 62:600–616.

Lyons J. 1991. Strategies for assessing the potential for positive adjustment following trauma. J Traumatic Stress 4:93–111.

Maddi SR, Khoshaba DM. 1994. Hardiness and mental health. J Pers Assess 63:265–274.

Maddi SR, Kobasa SC, Hoover M. 1979. An alienation test. J Humanistic Psychol 19:73–75.

Maes M, Lin A, Bonaccorso S, van Hunsel F, Van Gastel A, Delmeire L, Biondi M, Bosmans E, Kenis G, Scharpe S. 1998. Increased 24-hour urinary cortisol excretion in patients with post-traumatic stress disorder and patients with major depression, but not in patients with fibromyalgia. Acta Psychiatr Scand 98:328–335.

Marmar CR, Weiss DS, Metzler TJ, Delucchi K. 1996. Characteristics of emergency services personnel related to peritraumatic dissociation during critical incident exposure. Am J Psychiatry 153(Suppl 7):94–102.

Marmar CR, Weiss DS, Schlenger WE, Fairbank JA, Jordan BK, Kulka RA, Hough RL. 1994. Peritraumatic dissociation and posttraumatic stress in male Vietnam theater veterans. Am J Psychiatry 151:902–907.

Marshall GN, Schell TL. 2002. Reappraising the link between peritraumatic dissociation and PTSD symptom severity: Evidence from a longitudinal study of community violence survivors. J Abnorm Psychol 111:626–636.

Mason JW, Giller EL, Kosten TR, Ostroff RB, Podd L. 1986. Urinary free-cortisol levels in posttraumatic stress disorder patients. J Nerv Ment Dis 174:145–149.

Masten AS, Morison P, Pellegrini DS. 1985. A revised class play method for peer assessment. Dev Psychol 21:523–533.

McFall ME, Murburg MM, Ko GN, Veith RC. 1990. Autonomic responses to stress in Vietnam combat veterans with posttraumatic stress disorder. Biol Psychiatry 27:1165–1175.

McFall ME, Veith RC, Murburg MM. 1992. Basal sympathoadrenal function in posttraumatic distress disorder. Biol Psychiatry 31:1050–1056.

Morfin R, Starka L. 2001. Neurosteroid 7-hydroxylation products in the brain. Int Rev Neurobiol 46:79–95.

Morgan CA III, Southwick S, Hazlett G, Rasmusson A, Hoyt G, Zimolo Z, Charney D. 2004. Relationships among plasma dehydroepiandrosterone sulfate and cortisol levels, symptoms of dissociation, and objective performance in humans exposed to acute stress. Arch Gen Psychiatry 61:819–825.

Murburg MM, McFall ME, Lewis N, Veith RC. 1995. Plasma norepinephrine kinetics in patients with posttraumatic stress disorder. Biol Psychiatry 38:819–825.

Northouse LL, Mood D, Kershaw T, Schafenacker A, Mellon S, Walker J, Galvin E, Decker V. 2002. Quality of life of women with recurrent breast cancer and their family members. J Clin Oncol 20:4050–4064.

Nowicki S, Strickland BR. 1973. A locus of control scale for children. J Consult Clin Psychol 40:148–154.

Orr SP, Lasko NB, Metzger LJ, Berry NJ, Ahern CE, Pitman RK. 1998. Psychophysiologic assessment of women with posttraumatic stress disorder resulting from childhood sexual abuse. J Consult Clin Psychol 66:906–913.

Ozer EJ, Best SR, Lipsey TL, Weiss DS. 2003. Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. Psychol Bull 129:52–73.

Perry S, Difede J, Musngi G, Frances AJ, Jacobsberg L. 1992. Predictors of posttraumatic stress disorder after burn injury. Am J Psychiatry 149:931–935.

Pitman RK, Orr SP. 1990. Twenty-four hour urinary cortisol and catecholamine excretion in combat-related posttraumatic stress disorder. Biol Psychiatry 27:245–247.

Pitman RK, Sanders KM, Zusman RM, Healy AR, Cheema F, Lasko NB, Cahill L, Orr SP. 2002. Pilot study of secondary prevention of posttraumatic stress disorder with propranolol. Biol Psychiatry 51:189–192.

Rasmusson AM, Hauger RL, Morgan CA, Bremner JD, Charney DS, Southwick SM. 2000. Low baseline and yohimbine-stimulated plasma neuropeptide Y (NPY) levels in combat-related PTSD. Biol Psychiatry 47:526–539.

Rasmusson AM, Lipschitz DS, Wang S, Hu S, Vojvoda D, Bremner JD, Southwick SM, Charney DS. 2001. Increased pituitary and adrenal reactivity in premenopausal women with posttraumatic stress disorder. Biol Psychiatry 50:965–977.

Rasmusson AM, Vythilingam M, Morgan CA III. 2003. The neuroendocrinology of posttraumatic stress disorder: New directions. CNS Spectr 8:651–667.

Regehr C, Hill J, Glancy GD. 2000. Individual predictors of traumatic reactions in firefighters. J Nerv Ment Dis 188:333–339.

Roozendaal B. 2003. Systems mediating acute glucocorticoid effects on memory consolidation and retrieval. Prog Neuropsychopharmacol Biol Psychiatry 27:1213–1223.

Rotter JB. 1966. Generalized expectancies for internal versus external control of reinforcement. Psychol Monogr 80:Whole No. 609.

Rotter JB, Seeman M, Liverant S. 1962. Internal vs. external locus of control of reinforcement: A major variable in behavior study. In: Washburne NF, editor. Decisions, values, and groups. London: Pergamon. p 473–516.

Rubenstein JL, Heeren T, Housman D, Rubin C, Stechler G. 1989. Suicidal behavior in "normal" adolescents: Risk and protective factors. Am J Orthopsychiatry 59:59–71.

Rutter M. 1985. Resilience in the face of adversity: Protective factors and resistance to psychiatric disorder. Br J Psychiatry 147:598–611.

Rutter M. 1987. Psychosocial resilience and protective mechanisms. Am J Orthopsychiatry 57:316–331.

Rutter M. 1990. Psychosocial resilience and protective mechanisms. In: Rolf JM, Masten AS, Cicchetti D, Nucherlein KH, Weintraub S, editors. Risk and protective factors in the development of psychopathology. New York: Cambridge University Press. p 181–214.

Rutter M, Tizard J, Yule W, Graham P, Whitmore K. 1976. Research report: Isle of Wight Studies, 1964–1974. Psychol Med 6:313–332.

Schelling G. 2002. Effects of stress hormones on traumatic memory formation and the development of posttraumatic stress disorder in critically ill patients. Neurobiol Learn Mem 78:596–609.

Schelling G, Briegel J, Roozendaal B, Stoll C, Rothenhausler HB, Kapfhammer HP. 2001. The effect of stress doses of hydrocortisone during septic shock on posttraumatic stress disorder in survivors. Biol Psychiatry 50:978–985.

Schelling G, Kilger E, Roozendaal B, de Quervain DJ, Briegel J, Dagge A, Rothenhausler HB, Krauseneck T, Nollert G, Kapfhammer HP. 2004. Stress doses of hydrocortisone, traumatic memories, and symptoms of posttraumatic stress disorder in patients after cardiac surgery: A randomized study. Biol Psychiatry 55:627–633.

Simeon D, Greenberg J, Knutelska M, Schmeidler J, Hollander E. 2003. Peritraumatic reactions associated with the World Trade Center disaster. Am J Psychiatry 160:1702–1705.

Soet JE, Brack GA, DiIorio C. 2003. Prevalence and predictors of women's experience of psychological trauma during childbirth. Birth 30:36–46.

Soetens E, Casaer S, D'Hooge R, Hueting JE. 1995. Effect of amphetamine on long-term retention of verbal material. Psychopharmacology (Berl) 119:155–162.

Sondergaard HP, Hansson LO, Theorell T. 2002. Elevated blood levels of dehydroepiandrosterone sulphate vary with symptom load in posttraumatic stress disorder: Findings from a longitudinal study of refugees in Sweden. Psychother Psychosom 71:298–303.

Southwick SM, Krystal JH, Morgan CA, Johnson D, Nagy LM, Nicolaou A, Heninger GR, Charney DS. 1993. Abnormal noradrenergic function in posttraumatic stress disorder. Arch Gen Psychiatry 50:266–274.

Spivak B, Maayan R, Kotler M, Mester R, Gil-Ad I, Shtaif B, Weizman A. 2000. Elevated circulatory level of GABA(A)—antagonistic neurosteroids in patients with combat-related posttraumatic stress disorder. Psychol Med 30:1227–1231.

Turner SW, Bowie C, Dunn G, Shapo L, Yule W. 2003. Mental health of Kosovan Albanian refugees in the UK. Br J Psychiatry 182:444–448.

Valentiner DP, Foa E, Riggs DS, Gershuny BS. 1996. Coping strategies and posttraumatic stress disorder in female victims of sexual and nonsexual assault. J Abnorm Psychol 105:455–458.

Wagnild G, Young HM. 1993. Development and psychometric evaluation of the resilience scale. J Nurs Meas 1:165–178.

Werner EE. 1989. High-risk children in young adulthood: A longitudinal study from birth to 32 years. Am J Orthopsychiatry 59:72–81.

Werner EE. 1992. Overcoming odds: High risk children from birth to adulthood. Ithaca, NY: Cornell University Press.

Werner EE. 1993. Risk, resilience and recovery: Perspectives from the Kauai longitudinal study. Dev Psychopathol 5:503–515.

Werner EE, Smith RS. 1982. Vulnerable but invincible: A study of resilient children. New York: McGraw-Hill.

Widdowson PS, Ordway GA, Halaris AE. 1992. Reduced neuropeptide Y concentrations in suicide brain. J Neurochem 59:73–80.

Williams D, Lawler KA. 2001. Stress and illness in low-income women: The roles of hardiness, John Henryism, and race. Women Health 32:61–75.

Yehuda R, Boisoneau D, Mason JW, Giller EL. 1993. Glucocorticoid receptor number and cortisol excretion in mood, anxiety, and psychotic disorders. Biol Psychiatry 34:18–25.

Yehuda R, Kahana B, Binder-Brynes K, Southwick SM, Mason JW, Giller EL. 1995. Low urinary cortisol excretion in Holocaust survivors with posttraumatic stress disorder. Am J Psychiatry 152:982–986.

Yehuda R, Siever LJ, Teicher MH, Levengood RA, Gerber DK, Schmeidler J, Yang RK. 1998. Plasma norepinephrine and 3-methoxy-4-hydroxyphenylglycol concentrations and severity of depression in combat posttraumatic stress disorder and major depressive disorder. Biol Psychiatry 44:56–63.

Yehuda R, Southwick S, Giller EL, Ma X, Mason JW. 1992. Urinary catecholamine excretion and severity of PTSD symptoms in Vietnam combat veterans. J Nerv Ment Dis 180:321–325.

Yehuda R, Southwick SM, Nussbaum G, Wahby V, Giller EL Jr, Mason JW. 1990. Low urinary cortisol excretion in patients with posttraumatic stress disorder. J Nerv Ment Dis 178:366–369.

Zimrin H. 1986. A profile of survival. Child Abuse Negl 10:339–349.

Zoellner LA, Jaycox LH, Watlington CG, Foa EB. 2003. Are the dissociative criteria in ASD useful? J Trauma Stress 16:341–350.