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Psychobiology of PTSD in the Acute Aftermath of Trauma: Integrating Research on Coping, HPA Function and Sympathetic Nervous System Activity

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Abstract

Research on the psychobiological sequelae of trauma has typically focused on long-term alterations in individuals with chronic posttraumatic stress disorder (PTSD). Far less is known about the nature and course of psychobiological risk factors for PTSD during the acute aftermath of trauma. In this review, we summarize data from prospective studies focusing on the relationships among sympathetic nervous system activity, hypothalamic-pituitary-adrenal function, coping strategies and PTSD symptoms during the early recovery (or non-recovery) phase. Findings from pertinent studies are integrated to inform psychobiological profiles of PTSD-risk in children and adults in the context of existing models of PTSD-onset and maintenance. Data regarding bidirectional relations between coping strategies and stress hormones is reviewed. Limitations of existing literature and recommendations for future research are discussed.

Keywords

trauma; PTSD; coping; hypothalamic-pituitary-adrenal axis; sympathetic nervous system

1. Introduction

Traumatic stressors are defined by the direct experience, witnessing, or confrontation by an event involving actual or threatened danger and evoking responses that include intense fear, helplessness or horror (American Psychiatric Association, 2000). Epidemiological studies indicate that up to 60%-90% of individuals will experience at least one traumatic event in their lifetime (Breslau et al., 1998; Kessler et al., 1995; Sledjeski et al., 2008). Of these

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Contributors

Matthew Morris managed literature searches and was involved in writing and editing the manuscript.

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Both authors contributed to and have approved the final manuscript.

Conflict of Interest

The authors have no conflicts of interest.

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individuals, many will experience transient symptoms of posttraumatic stress disorder (PTSD) in the immediate aftermath of trauma, and they often fully recover without treatment (e.g., Kessler et al., 1995). However, approximately 8% to 18% of trauma-exposed individuals will go on to develop PTSD (Breslau et al., 1998; Kessler et al., 1995; Sledjeski et al., 2008). Whereas many studies have identified pretraumatic, peritraumatic, and posttraumatic factors that characterize these high-risk individuals (for reviews, see Brewin et al., 2000; McNally, 2003; Ozer et al., 2003; Shalev, 1996), far less attention has focused on psychobiological mechanisms of risk operating in the early stages of trauma recovery (Delahanty and Nugent, 2006). Understanding trauma-response patterns associated with PTSD-vulnerability early in their trajectories will help to refine psychoneuroendocrine models of PTSD-vulnerability, identify those individuals who are least likely to recover without treatment, and to guide the development of early and more effective interventions.

Increased vulnerability to traumatic stress is determined by a complex interplay of preexisting risk factors, mediators of stress reactivity and regulation, and moderating factors. A theoretical model outlining relations among these vulnerability factors is presented in Figure 1.

In this article, we review empirical data for alterations in peripheral indicators of two stress response systems and coping strategies that comprise the acute reaction to trauma. To identify potential mediators of risk, we focus on findings from prospective studies that assessed predictors of PTSD symptom severity over time. We begin by reviewing data regarding the time course of natural recovery from trauma. Next, we evaluate cross-sectional and prospective studies examining relations between PTSD symptoms and sympathetic nervous system (SNS) activity, hypothalamic-pituitary-adrenal (HPA) function, and coping strategies. We place particular emphasis on the timing of coping to address the question: which coping strategies appear effective at what times during the course of recovery? After summarizing findings from these literatures individually, we review research focusing on coping strategies and neuroendocrine function simultaneously. Prior reviews have examined cognitive-appraisal and coping strategies as determinants of traumatic stress responses (Olf et al., 2005a, 2005b), placing particular emphasis on psychobiological factors that contribute to gender differences in rates of PTSD (Olf et al., 2007). This review will focus primarily on the adult literature as there has been very limited prospective research on the psychobiology of PTSD in children. However, findings from pediatric samples will be discussed where relevant. Finally, we make recommendations for future research regarding the examination of additional biological factors involved in stress reactivity and regulation.

2. Methods

Pertinent studies for this review were identified through searches of PsycINFO, Web of Knowledge, and PubMed databases. Initial searches crossed keywords reflecting traumatic stress (*abuse, accidents, assault, combat, loss, maltreatment, neglect, rape, refugees, terrorism, torture, trauma, veteran, and war*) and posttraumatic stress symptoms (*posttraumatic stress disorder, PTSD*) with those reflecting indicators of SNS activity (*noradrenergic, norepinephrine, epinephrine, sympathetic nervous system, SNS*), HPA function (*adrenocortical, cortisol, glucocorticoid, HPA*), and coping (*coping, emotion regulation, stress response*). In addition, we searched the reference sections of qualifying articles as well as reviews (Brewin et al., 2000; Buckley and Kaloupek, 2001; de Kloet et al., 2006; Delahanty and Nugent, 2006; McNally, 2003; Olf et al., 2005a, 2005b, 2007; Ozer et al., 2003; Pervanidou and Chrousos, 2010; Pole, 2007; Shalev, 1996; Spaccarelli, 1994; Yehuda, 2002a). This review article focused on studies that included either a continuous measure of PTSD symptom severity or a semi-structured interview assessing PTSD diagnosis.

3. Acute Changes in PTSD Symptom Severity

Prospective studies of PTSD symptoms in the immediate aftermath of trauma help to elucidate rates of natural recovery. One study conducting weekly assessments of sexual ($n = 64$) and non-sexual ($n = 49$) assault revealed that the rate of PTSD among rape victims was 94% at two weeks (excluding the duration criterion) and fell to 47% at three months (Rothbaum et al., 1992). These findings paralleled those of another study of rape ($n = 96$) and non-sexual assault ($n = 100$) victims (Foa, 1995), which showed high rates of PTSD approximately two weeks after the incident (92% and 74% for sexual and non-sexual assault victims, respectively) and similar rates of recovery at three months (47% and 27% for sexual and non-sexual assault victims, respectively). Moreover, the rate of PTSD was 38% for rape victims and 13% for non-sexual assault victims at six months following the initial assault (Foa, 1995). Another study found that the rate of severe PTSD symptoms was 66% for rape victims and 33% for robbery victims at one month, dropping to 15% and 10% at 18 months for rape and robbery victims, respectively (Resick, 1988). Finally, prospective studies suggest that PTSD symptom severity beginning one to two weeks after trauma exposure is a stronger predictor of chronic PTSD (e.g., Koren et al., 1999) than symptom severity within the first few days (e.g., Shalev, 1992). Taken together, these findings indicate that the majority of trauma-exposed individuals will recover within three to six months, with rates of recovery differing according to trauma type. Nevertheless, a sizeable minority of trauma-exposed individuals continue to meet criteria for PTSD up to 18 months after the traumatic event.

4. Acute SNS Trauma Responses

The SNS and HPA axis are both activated as part of the stress response and work in concert to promote adaptation, or *allostasis*, by enabling organisms to accommodate to changing conditions in their environment (McEwen and Seeman, 1999). Within seconds of exposure to a stressor, norepinephrine and epinephrine are released from the adrenal medulla as part of the SNS response. Within minutes, cortisol is released from the adrenal cortex, initially amplifying the SNS response and then curtailing it through negative feedback mechanisms (Jacobson and Sapolsky, 1991; Munck et al., 1984; Sapolsky et al., 1986). Thus, the SNS and HPA axis are critical for both stress reactivity and recovery processes.

According to classical conditioning theory, traumatic events serve as unconditioned stimuli and intense fear, helplessness, and horror comprise the unconditioned responses. The onset and maintenance of PTSD is linked to the development of conditioned fear responses (reexperiencing the trauma and hyperarousal symptoms) to conditioned stimuli (reminders of the traumatic event). Researchers have emphasized the acute unconditioned response to trauma in PTSD development (Feinstein and Dolan, 1991), placing particular emphasis on the role of peritraumatic SNS activity in enhanced fear conditioning (Pitman, 1989) and consolidation of emotional memories (McCleery and Harvey, 2004). Findings from animal and human studies reveal that catecholamines facilitate acquisition of conditioned fear responses (e.g., Cahill et al., 1994; Roozendaal et al., 1997); further, the effects of catecholamines on memory storage are sensitive to stress-related increases in glucocorticoids (Roozendaal et al., 1997). Alterations of noradrenergic activity have been implicated in the hyperarousal and reexperiencing symptoms of PTSD (O'Donnell et al., 2004). In addition, overconsolidation of trauma-related memories has been linked to intrusive thoughts, images, flashbacks, and repetitive nightmares in PTSD patients (Southwick et al., 2002).

4.1. Cross-sectional Research on SNS Trauma Responses

Cross-sectional studies of SNS activity in PTSD have reported increased central and peripheral noradrenergic activity in both children and adults with PTSD symptoms or disorder (for reviews, see Delahanty and Nugent, 2006; O'Donnell et al., 2004; Pervanidou and Chrousos, 2010). Meta-analytic findings regarding peripheral indicators of noradrenergic activity indicate that PTSD is associated with elevated resting heart rate, skin conductance, and blood pressure (both systolic and diastolic), exaggerated heart rate, eyeblink and skin conductance to acoustic startle responses, slower skin conductance habituation to acoustic startle, increased heart rate and skin conductance responses to standardized trauma cues, and elevated heart rate, skin conductance, diastolic blood pressure and facial muscle responses to idiographic trauma cues (Pole, 2007). A meta-analysis examining basal cardiovascular activity among individuals with PTSD, trauma-exposed individuals without PTSD (TE), and never traumatized controls (NTC), found elevated resting heart rate in PTSD relative to TE and NTC groups (Buckley and Kaloupek, 2001). Data regarding greater eyeblink electromyogram and heart rate responses to acoustic startle in PTSD compared to TE groups suggest that enhanced SNS reactivity may be associated with PTSD symptoms in particular, and not trauma-exposure in general (Griffin, 2008).

4.2. Prospective Research on SNS Trauma Responses

Findings of prospective studies examining relations between SNS activity and PTSD symptom severity varied according to the timing of SNS assessment (see Table 1). Overall, results were mixed for adult studies examining SNS function during the peritraumatic risk period (within 24 hours of trauma exposure). While some studies found that increased heart rate was associated with subsequent PTSD symptoms or disorder (Kassam-Adams et al., 2005; Shalev et al., 1998; Zatzick et al., 2005), other studies found that decreased heart rate was associated with subsequent PTSD (Blanchard et al., 2002), or that heart rate was unrelated to PTSD (Buckley et al., 2004; Ehrling et al., 2008b; Kuhn et al., 2006). Whereas decreased diastolic blood pressure was associated with subsequent PTSD in two studies (Blanchard et al., 2002; Ehrling et al., 2008b), blood pressure was not associated with PTSD in another study (Buckley et al., 2004). Finally, studies examining epinephrine, norepinephrine, or methoxy-4-hydroxyphenylglycol (a metabolite of norepinephrine) within 24 hours of trauma exposure generally showed no associations with PTSD symptoms or disorder (Heinrichs et al., 2005; Pervanidou et al., 2007; Videlock et al., 2008; Yehuda et al., 1998). However, epinephrine was negatively associated with subsequent PTSD in adults (Delahanty et al., 2000, 2003b) and positively associated with PTSD symptoms in children (Delahanty et al., 2005).

A slightly more consistent pattern of results emerges from studies examining SNS activity in adults during the acute risk period (within one month of trauma exposure). Increased heart rate at one week was associated with subsequent PTSD in several studies (Bryant et al., 2000, 2003; Shalev et al., 1998), although heart rate at one month was not associated with PTSD (Griffin, 2008). Whereas one study found that elevated epinephrine and norepinephrine at one month was associated with PTSD symptoms in men, but not women (Hawk et al., 2000), another study found lower norepinephrine at 10 days and one month in the PTSD group (Videlock et al., 2008). Startle responses were not associated with PTSD during this acute risk period (Griffin, 2008; Shalev et al., 2000). However, one study found that increased skin conductance startle response before the traumatic event was associated with increased PTSD symptoms in the first month after exposure (Guthrie and Bryant, 2005). A study of children found that higher norepinephrine was associated with PTSD (Pervanidou et al., 2007).

Studies examining SNS activity in adults during the intermediate risk period (1-11 months after trauma exposure) showed that increased heart rate reactivity and systolic blood pressure reactivity between one and four months was associated with subsequent PTSD (Blanchard et al., 1996). Increased heart rate startle response was associated with PTSD at one and four months (Shalev et al., 2000), increased heart rate and eyeblink startle responses were observed in PTSD groups at six months (Griffin, 2008), and decreased habituation to startle (skin conductance, eyeblink) was seen in PTSD groups at four months (Shalev et al., 2000); one study found that heart rate at four months did not distinguish PTSD from non-PTSD (Shalev et al., 1998). Whereas one study found that higher epinephrine was positively associated with PTSD symptoms at 6 months for men but not women (Hawk et al., 2000), another study found lower norepinephrine in adults with PTSD at five months (Vidlock et al., 2008). One study of children revealed that higher norepinephrine was associated with PTSD at 6 months (Pervanidou et al., 2007). Finally, results were mixed for studies examining SNS activity in adults during the enduring risk period (12-24 months after trauma exposure). Whereas higher peritraumatic heart rate was associated with increased PTSD symptoms 12 months later (Zatrack et al., 2005) and higher heart rate at one week was associated with PTSD two years later (Bryant et al., 2003), lower heart rate and diastolic blood pressure at one month were also associated with PTSD 13 months later (Blanchard et al., 2002).

Summary—Overall, support for associations between SNS activity and risk for PTSD in adults during the peritraumatic, acute and enduring risk periods was inconsistent. However, increased reactivity of SNS indicators to trauma scripts and acoustic startle tasks were reliably associated with a diagnosis of PTSD. In adults, epinephrine and norepinephrine levels were positively associated with subsequent PTSD symptoms, though these relations may exhibit sex differences (Hawk et al., 2000). The authors of one study showing a negative association between norepinephrine and PTSD (Vidlock et al., 2008) attributed this finding to their more stringent definition of traumatic events (including DSM-IV Criterion A2: intense fear, helplessness, or horror) and measurement of plasma norepinephrine, which may not accurately capture sustained adrenergic trauma reactivity; the authors concluded that resting measurements of hormone levels may have limited utility as markers of risk for PTSD. This conclusion is supported by studies assessing hormone levels in responses to various challenges during the intermediate risk period, which have consistently demonstrated higher SNS responses in PTSD groups. Studies administering psychosocial stress tasks to trauma-exposed individuals have demonstrated exaggerated SNS responses in both TE children (Saltzman et al., 2005) and adults with PTSD (Liberzon et al., 1999). Finally, epinephrine was positively associated with PTSD symptoms in children during the peritraumatic risk period and norepinephrine was positively associated with PTSD symptoms during the acute and intermediate risk periods.

5. Acute HPA Trauma Responses

Research examining acute biological risk factors for PTSD has implicated HPA hypoactivity in addition to SNS hyperactivity. These two stress response systems may have independent effects on the development of PTSD symptoms, with lower cortisol levels associated with avoidance behaviors (Yehuda et al., 1995) and higher catecholamines associated with reexperiencing and hyperarousal symptoms (Charney et al., 1993; Southwick et al., 1993; Yehuda et al., 1992). Transient cortisol elevations in response to trauma may protect against PTSD symptoms by terminating the initial surge of catecholamines via negative feedback mechanisms (Yehuda, 2002b). Reduced cortisol-mediated containment of SNS activity during traumatic events can impact memory consolidation and retrieval via prolonged norepinephrine availability in the brain (Pacak et al., 1995), and thereby increase the risk for PTSD. According to Yehuda's (2002b) two-factor model of acute biological risk for PTSD,

either SNS hypersecretion or cortisol hyposcretion during trauma exposure are capable of triggering PTSD symptoms.

5.1. Cross-sectional Research on HPA Trauma Responses

Findings from cross-sectional studies of HPA function in PTSD have been inconsistent and dogged by methodological issues such as the inclusion of TE individuals in control groups, lack of discrimination between individuals with PTSD only and those with PTSD and comorbid major depressive disorder, assessment of diurnal secretion without controlling for the time of measurement, and variability in gender composition, type of trauma, and the time interval between traumatic episode and assessment (for reviews, see de Kloet et al., 2006; Yehuda, 2002a). Meta-analytic studies of HPA function attempting to account for these factors have generally found lower daily cortisol output in PTSD groups relative to NTC (Meewisse et al., 2007; Miller et al., 2007; Morris et al., 2012; see also Klaassens et al., 2012). Findings regarding diurnal cortisol secretion in TE groups have been more variable, with two meta-analyses reporting no differences in daily output between TE and NTC groups (Klaassens et al., 2012; Morris et al., 2012) and another reporting lower daily output in TE and PTSD groups compared to NTC groups (Meewisse et al., 2007). Finally, meta-analyses suggest enhanced cortisol suppression in PTSD and TE groups relative to NTC (Klaassens et al., 2012; Miller et al., 2007; Morris et al., 2012). The majority of studies assessing trauma-related HPA and SNS alterations in PTSD have been cross-sectional and focused on chronic PTSD. Hence, questions regarding biological correlates of acute PTSD remain unanswered.

5.2. Prospective Research on HPA Trauma Responses

Results of prospective studies examining relations between HPA activity and PTSD symptom severity have varied according to the timing of assessment (see Table 2). Taken together, studies of the peritraumatic period indicate that lower cortisol levels were associated with subsequent PTSD in adults (Delahanty et al., 2000; Ehling et al., 2008b). Findings for adult studies were mixed during the acute risk period. Whereas some studies found that higher cortisol levels were associated with subsequent PTSD symptoms (Cieslak et al., 2011), others have found no association between cortisol and PTSD symptoms (Bonne et al., 2003; Pervanidou et al., 2007). One possible explanation for discrepancies across these studies may be variability in the time of day when cortisol was assessed. For example, one study found that PTSD symptoms five days after trauma exposure were linked to lower morning and higher afternoon cortisol levels (Aardal-Eriksson et al., 2001). In addition, sex differences may also account for the mixed findings (e.g., Hawk et al., 2000). Enhanced suppression of cortisol in response to the dexamethasone suppression test (DST) was also associated with PTSD in adults during the acute risk period (McFarlane et al., 2011). In contrast to the findings from adult samples, studies in children showed that higher peritraumatic cortisol levels were associated with subsequent PTSD symptoms (Delahanty et al., 2005; Pervanidou et al., 2007). Discrepancies between children and adults in trauma-related HPA function may be explained by the maturation of neurobiological systems, developmental timing of trauma-exposure, and/or by differences in the time elapsed since the traumatic event (De Bellis et al., 1999; Gunnar and Quevedo, 2007; Miller et al., 2007).

Findings of studies assessing HPA function during the intermediate risk period were mixed for adults. Whereas several studies found no association between cortisol and PTSD symptoms (Hawk et al., 2000; Pervanidou et al., 2007; Turner-Cobb et al., 2010), one study found that higher cortisol levels were associated with PTSD (Maes et al., 1998). Similar to research on HPA function in adults during the acute risk period, these results may be influenced by the time of assessment. For example, lower morning cortisol (Bonne et al., 2003) and higher afternoon cortisol levels (Aardal-Eriksson et al., 2001) were associated

with increased PTSD symptoms during the intermediate risk period. Finally, one study found that cortisol levels were not associated with PTSD symptoms during the enduring risk period (Anisman et al., 2001).

Summary—Overall, findings from prospective studies of HPA function and PTSD symptoms revealed that increased risk for subsequent PTSD was related to lower cortisol levels for adults and higher cortisol levels for children during the first 24 hours after trauma exposure. Although results were mixed for adults during later risk periods, there were some data suggesting that lower morning cortisol levels and higher afternoon cortisol levels were associated with increased PTSD symptoms. Given diurnal patterns of cortisol secretion (Bailey and Heitkemper, 1991), these findings emphasize the importance of measuring HPA function over the course of the day to capture time-sensitive alterations associated with the risk for PTSD (Yehuda et al., 1996).

6. Acute Coping with Trauma

Cognitive appraisal processes involve an individual's perception, interpretation, and evaluation of experiences, and are important for determining psychobiological responses to stressful events (Lazarus and Folkman, 1984). Thus, cognitive appraisal plays an important role in signaling whether, and what type, of coping strategies may be enacted in response to traumatic stress. Theories of coping vary according to whether they consider involuntary or automatic responses to stress as coping (Eisenberg et al., 1997; Lazarus and Folkman, 1984; Skinner and Wellborn, 1994). For the purposes of the present review, we adopt the following operational definition of coping: "conscious, volitional efforts to regulate emotion, cognition, behavior, physiology, and the environment in response to stressful events or circumstances" (Compas et al., 2001, p. 89). According to Compas and colleagues (2001), volitional coping responses include primary-control engagement (i.e., efforts to change the situation or one's emotional response to it, such as problem-solving, emotional regulation, and emotional expression), secondary-control engagement (i.e., efforts to adapt to the situation, such as cognitive-restructuring, acceptance, distraction, and positive thinking), and disengagement coping (i.e., efforts to relinquish control over the situation, such as avoidance, denial, and wishful thinking). The present review will not examine associations between PTSD symptoms and involuntary stress responses, which include involuntary engagement (e.g., rumination, intrusive thoughts, emotional/physiological arousal, and impulsive action) and involuntary disengagement (e.g., emotional-numbing, inaction, escape, and cognitive interference), because our primary focus is to identify coping strategies that can be incorporated into early interventions for PTSD. Nevertheless, we acknowledge that these involuntary responses are important targets for future research on trauma recovery due to their overlap with current PTSD symptom criteria and the need for prospective research demonstrating links between these automatic stress responses and PTSD-onset and maintenance (e.g., rumination; Ehling et al., 2008a; peritraumatic dissociation; Ozer et al., 2003).

6.1. Cross-sectional Research on Coping with Trauma

Individuals cope with traumatic stress in a variety of ways (Aldwin and Yancura, 2004). Data from cross-sectional studies of coping with traumatic events indicates that PTSD symptoms are associated with a variety of coping strategies, although findings have been inconsistent (see Table 3). Research examining primary control engagement coping strategies has found problem-focused strategies to be negatively associated with PTSD symptoms (e.g., Sutker et al., 1995), positively associated with PTSD symptoms with greater time since trauma exposure (e.g., Kanninen et al., 2002), or not significantly associated with PTSD symptoms at all (e.g., Goldenberg and Matheson, 2005). To the extent

that social support is sought as a means of emotional expression or to aid in problem-solving, it could be considered a type of primary control coping (social support with the intent of distraction would be considered secondary control engagement coping). Research assessing social support has examined different dimensions, including perceived social support, received social support (e.g., number of confidants, size of network), type of support (e.g., family, military unit), and satisfaction with support. While many studies suggest that social support after traumatic events is associated with decreased PTSD symptoms (e.g., Astin et al., 1993; King et al., 1998; Kramer and Green, 1991; Perrin et al., 1996; Solkoff et al., 1986; Solomon et al., 1987; Weiss et al., 1995), others have found that social support-seeking is positively associated with PTSD symptoms (e.g., Fairbank et al., 1991), or that social support is unrelated to PTSD symptoms (e.g., DePrince et al., 2011). Research examining secondary control engagement coping strategies has found lower PTSD symptoms associated with increased acceptance (e.g., Pietrzak et al., 2009; Tull et al., 2007; Vujanovic et al., 2009) and decreased distraction (Steil and Ehlers, 2000). Finally, cross-sectional research examining disengagement coping has found positive associations between PTSD symptoms and disengagement (e.g., Santello and Leitenberg, 1993), wishful thinking (e.g., Clohessy and Ehlers, 1999; Fairbank et al., 1991; Sutker et al., 1995), behavioral avoidance (e.g., Dempsey et al., 2000), experiential avoidance (e.g., Gold et al., 2007, 2009; Kashdan et al., 2009; Morina, 2007; Morina et al., 2008; Orcutt et al., 2005; Thompson and Waltz, 2010; Tull et al., 2004), and avoidant coping (e.g., Harvey and Bryant, 1998; Littleton and Grills-Taquechel, 2011; Matthews et al., 2009).

Trauma-related coping research has been limited by cross-sectional study designs, lack of consensus on coping definitions, and a plethora of coping measures. Inconsistent results have also been linked to the tendency for individuals to simultaneously employ multiple coping strategies and for higher stress levels to be associated with greater utilization of all types of coping (Connor-Smith et al., 2000; Holahan and Moos, 1987; Marmar et al., 1996; Norris et al., 2002). These findings have led some researchers to suggest that “coping strategies [are] really expressions of symptoms of disorder as much as attempts to manage external threat and disruption” (Spurrell and McFarlane, 1993, p. 199). One solution to this problem is to assess the degree to which an individual employs a particular coping strategy relative to other strategies by computing proportion scores (Connor-Smith et al., 2000; Holahan & Moos, 1990, 1991; Valentiner et al., 1994; Wolfe et al., 1998). In addition, because whether a particular coping method is adaptive likely depends on the context in which it is used, prospective studies are needed to address the question: which coping strategies are most useful (or detrimental), and at what times?

6.2. Prospective Research on Coping with Trauma

The manner in which individuals cope with traumatic stress is not static; rather, coping efforts are ongoing and dynamic, adjusting to contextual factors and changing over time (Lazarus and Folkman, 1984). Whereas primary control engagement coping may be used when trauma-related threat is controllable or escapable, secondary control engagement coping or disengagement coping may be used when the threat is uncontrollable or inescapable. Hence, trauma features (e.g., type and duration) likely influence the timing and selection of coping strategies. Findings from prospective studies examining relations between coping and PTSD symptom severity are consistent for certain strategies but not others (see Table 4).

Primary control engagement coping—Studies examined different aspects of social support, including perceived support, received support, and support-seeking. Perceived social support was adaptive during the first year after trauma exposure. One study found that perceived support at one week was negatively related to PTSD symptoms at two, six, and 12

months (Perry et al., 1992). Received social support was adaptive during the 1.5 years following trauma exposure. One study found that received support was negatively associated with PTSD symptom severity at two weeks, one month, three months, and six months (Ehring et al., 2008c). Another study found that received support between three and 14 months was associated with decreased avoidance type of PTSD symptoms at 18 months (Joseph et al., 1993). In another investigation, no association between received social support and PTSD symptoms was detected at three or six years after the traumatic event (Dagleish et al., 1996). Social support-seeking was maladaptive. One study found that support-seeking was positively associated with PTSD at 11 and 29 months after trauma (McFarlane, 1989), and another found that support-seeking within one month of trauma exposure was associated with increased odds of PTSD symptoms over the next six months (Silver et al., 2002). Results of studies examining problem-focused coping after trauma were mixed. Whereas one study found that problem-focused coping 1-2 years after the end of the war was negatively associated with PTSD symptoms two years after the end of the war (Solomon et al., 1988), another found greater problem-focused coping in PTSD compared to TE groups at 42 months after trauma exposure (Spurrell and McFarlane, 1993). A third study found that active problem-focused coping declined more rapidly in full or subsyndromal PTSD groups one to 12 months after trauma exposure (Hepp et al., 2005). Finally, task-focused coping 2-3 weeks after trauma-exposure was negatively associated with PTSD symptoms at four months (Johnsen et al., 2002).

Secondary control engagement coping—Thought suppression was maladaptive during the first year after trauma exposure. Thought suppression was positively associated with PTSD symptom severity at two weeks (Ehring et al., 2008c), one month (Ehring et al., 2008c), three and six months (Ehlers et al., 2003, Ehring et al., 2008c); moreover, thought suppression at three months was associated with PTSD symptom severity at six months (Ehlers et al., 2003) and one year (Ehlers et al., 1998). Results of studies examining distraction after trauma exposure were mixed. Whereas one study found that repressive coping (defined as “cognitive and emotional effort to ignore or divert attention from threatening stimuli, whether internal or external”) was negatively associated with PTSD symptoms at one week and 7 months (Ginzburg et al., 2002, p. 748), another study found that self-distraction within one month of trauma exposure was associated with increased odds of PTSD symptoms over the subsequent six months (Silver et al., 2002). Acceptance was adaptive in the first month after trauma exposure and was associated with decreased odds of PTSD symptoms over the first six months (Silver et al., 2002).

Disengagement coping—Disengagement from coping efforts and denial within one month of trauma exposure were associated with increased odds of PTSD symptoms over the subsequent six months (Silver et al., 2002). Studies examined different aspects of avoidance, including avoidant coping, escape-avoidance coping, behavioral avoidance, and experiential avoidance; all were maladaptive up to five years after trauma exposure. Avoidant coping was associated with maintenance of PTSD symptoms from two weeks until 12 months after the trauma (Eid et al., 2001; Johnsen et al., 2002; Eid, 2003), and avoidance coping 14 months after trauma was associated with PTSD symptoms 13 months later (Benotsch et al., 2000). Escape-avoidance coping within one week of trauma exposure was associated with PTSD at six weeks (Nightingale and Williams, 2000). Behavioral avoidance was positively associated with PTSD symptoms (Resick, 1988). Experiential avoidance before trauma exposure was positively associated with PTSD symptoms at 27 days, and experiential avoidance at 27 days was associated with PTSD symptoms at 35 weeks (Kumpula et al., 2011); in addition, experiential avoidance between one month and five years after trauma exposure was associated with increased PTSD symptom severity four to eight weeks later (Marx and Sloan, 2005). Wishful thinking was maladaptive up to one year after trauma

exposure. Wishful thinking was positively associated with PTSD symptom severity at three months after trauma exposure (Valentiner et al., 1996), and higher rates of ‘downplaying’ and wishful thinking were found in full or subsyndromal PTSD groups one to 12 months after trauma (Hepp et al., 2005; Spurrell and McFarlane, 1993).

Summary—Findings from prospective studies of coping and PTSD symptoms revealed that social support (perceived and received) and acceptance were adaptive coping strategies in the immediate aftermath of trauma. Social support-seeking, thought suppression, avoidance, denial, and wishful thinking were all maladaptive trauma-related coping strategies (for a discussion of how maladaptive coping strategies are linked to appraisals of traumatic events, see Ehlers and Clark, 2000). Studies examining problem-focused coping and distraction yielded inconsistent findings. Taken together, these results suggest that perceived and received social support as soon as one week after trauma exposure is associated with improved trauma recovery. However, social support-seeking measures may reflect *attempted* coping rather than *adaptive* coping, and could be considered as indices of posttraumatic distress (e.g., Spurrell and McFarlane, 1993). Of note, support was stronger for acceptance than problem-focused coping strategies in the acute trauma recovery phase.

Findings from the trauma coping literature are consistent with existing treatment approaches for PTSD. Models of PTSD drawing from information and emotional processing theories (e.g., Foa et al., 1989; Lang, 1977) posit the development of trauma-related fear networks in memory that elicit escape or avoidance behaviors. Empirically supported treatment approaches grounded in these models, such as Prolonged Exposure (Foa et al., 2007), emphasize repeated exposure (imaginal and in vivo) to traumatic memories in order to overcome avoidance and promote habituation of fear responses and reorganization of fear networks. Models of PTSD drawing from social-cognitive theories emphasize the development of maladaptive beliefs about the traumatic event, the world, the self, and others, leading to escape/avoidance behaviors including avoidance of thoughts or reminders, suppression of emotions, substance abuse, and social withdrawal. Empirically supported treatment approaches based on social-cognitive models, such as Cognitive Processing Therapy (Resick et al., 2007), seek to identify and modify these maladaptive beliefs. For individuals who have difficulty with treatments involving trauma-processing (e.g., Becker and Zayfert, 2001), mindfulness-based therapies promoting awareness of the present moment and nonjudgmental acceptance of thoughts, emotions and sensations (e.g., Kabat-Zinn, 1994; Linehan, 1993) may also be efficacious in decreasing PTSD symptoms (Kimbrough et al., 2010). Finally, reviews of early intervention programs for individuals recently exposed to trauma suggest that individual cognitive-behavioral therapies that include psychoeducation about trauma sequelae, imaginal exposure, cognitive restructuring, and decreasing avoidance, are effective in preventing chronic PTSD symptoms (e.g., Ehlers and Clark, 2003).

7. Summary of Findings

The present article reviewed prospective studies focusing on acute biological (SNS and HPA function) and psychological (coping) predictors of PTSD symptom severity. A summary of findings is presented in Table 5. Studies of SNS activity revealed a consistent pattern of increased reactivity to challenge (trauma scripts and acoustic startle tasks) associated with higher PTSD symptom severity. In both adults and children, positive associations were found between epinephrine and norepinephrine levels and PTSD symptom severity. Studies of HPA function showed that lower peritraumatic cortisol levels for adults and higher peritraumatic cortisol levels for children were associated with increased risk for subsequent PTSD. In addition, lower morning cortisol levels and higher afternoon cortisol levels were associated with higher PTSD symptom severity in adults. Prospective studies of primary

control engagement coping revealed that perceived and received social support were associated with lower PTSD symptom severity, whereas social support-seeking was associated with increased odds of PTSD. Studies of secondary control engagement coping revealed that thought suppression was maladaptive at all time points, whereas acceptance in the acute risk period was associated with decreased odds of subsequent PTSD. Distraction was adaptive in one study and maladaptive in another. Finally, studies of disengagement coping showed that avoidance, denial, and wishful thinking were consistently associated with higher PTSD symptom severity.

7.1. Psychobiological Profiles of PTSD Risk

Researchers have proposed that a pattern of increased SNS function and decreased HPA function in the acute risk phase after trauma exposure could contribute to PTSD onset and maintenance through the formation of “overconsolidated” memories (Pitman, 1989; Pitman et al., 1993). Elevated catecholamine release unrestrained by cortisol may trigger intrusive PTSD symptoms via disruption of memory consolidation and retrieval processes (Yehuda and Harvey, 1997; Yehuda et al., 1998). However, alterations of SNS and HPA function in PTSD have typically been examined separately without considering potential synergistic or inhibitory interactions between these stress response systems (for a review of preclinical and clinical research, see O'Donnell et al., 2004). Consistent with predictions from these acute biological risk models of PTSD (Pitman, 1989; Yehuda, 2002b), the present review showed that increased SNS activity, enhanced SNS reactivity to challenge, and decreased HPA activity were associated with higher PTSD symptom severity in adults. Studies have also demonstrated increased cortisol reactivity to psychosocial stressors in adults with PTSD (for a review, see de Kloet et al., 2006). Enhanced SNS/HPA stress reactivity in the context of lower circulating cortisol levels suggests a “sensitized” neuroendocrine system capable of rapid and robust responses to perceived threat (Post, 1992; Yehuda et al., 1996). Increased risk for PTSD during the one-to-six-month window after trauma exposure was also positively associated with social support-seeking, thought suppression, avoidance, denial and wishful thinking, and negatively associated with perceived and received social support and acceptance.

Among children, several studies showed that higher risk for PTSD was linked to increased SNS and HPA activity in the first six months after trauma exposure. According to the developmental traumatology model (De Bellis et al., 1999), differences in HPA findings between studies of children and adults could be explained by long-term adaptation of the HPA axis after trauma exposure: that is, an initial period of HPA hyperactivity may be followed by compensatory negative feedback inhibition of the pituitary and an adaptive downregulation of pituitary corticotropin-releasing hormone receptors, eventually resulting in diminished basal HPA activity. In support of this model, prospective studies have shown that diurnal cortisol levels in children are elevated shortly after trauma exposure and decreased over the first six months (Pervanidou et al., 2007). Some studies have also demonstrated increased SNS reactivity to psychosocial stressors in trauma-exposed children; however, findings regarding HPA reactivity to challenge have not been consistent and may differ according to the nature of the threat and availability of coping resources (e.g., Gunnar et al., 2009; Ivanov et al., 2011). Due to a paucity of studies employing pharmacological or psychological challenge paradigms with trauma-exposed children, it remains unclear whether these children exhibit enhanced SNS or HPA stress reactivity, and if a profile of higher circulating cortisol levels reflects a “desensitized” neuroendocrine system that exhibits blunted responses to perceived threat. Acute biological risk models for PTSD in adults emphasizing cortisol hypoactivity (Yehuda, 2002b) may require modification for children; for example, would we expect the conditional probability of developing PTSD

after trauma exposure to be lower in children due to the inhibitory effects of cortisol on SNS activity?

7.2. Relations between Coping and HPA/SNS Trauma Responses

How are coping and HPA/SNS responses related? Studies have shown that primary control coping is associated with decreased cortisol output (e.g., Nicolson, 1992; O'Donnell et al., 2008; Thorsteinsson and James, 1999). Emotional expression and social support are associated with lower levels of stress hormones and decreased risk for stress-related psychopathology (Taylor, 2006; Taylor et al., 2000). However, consistent with data reviewed in this article, not all types of social support are considered adaptive. For example, interactions with friends involving co-rumination (i.e., extensive discussion of problems and focus on negative affect) may lead to prolonged activation of stress response systems and increased risk for stress-related psychopathology (Rose, 2002; Rose et al., 2007). Both secondary control coping (Nicolson, 1992) and disengagement coping (e.g., Knight et al., 1979; Sapolsky, 1992; Schulkin et al., 1998; Vaernes et al., 1982) have been linked to increased cortisol output. However, previous reviews have also reported negative associations between disengagement coping and cortisol following trauma-exposure (Olf et al., 2005a, 2005b). Although these studies highlight associations between basal neuroendocrine function and coping strategies, they cannot disentangle potential bidirectional influences.

7.2.1. Do coping strategies regulate stress hormones?—Research examining stress response activation following treatment conditions that target coping strategies is relevant to this question (for a review, see Adam et al., 2008). Studies employing laboratory stress tasks have shown that the presence of social support figures and improvement in mood during testing sessions predict lower cortisol responses (Heinrichs et al., 2003; Kirschbaum et al., 1995). In addition, random assignment to a group cognitive-behavioral intervention including cognitive restructuring, problem-solving and progressive muscle relaxation, predicted lower cortisol responses to a psychosocial stress task; changes in threat appraisals partially mediated this effect (Gaab et al., 2003; Hammerfald et al., 2006). Another study examining the impact of this group intervention on diurnal cortisol levels before an exam found an association between cortisol awakening responses and perceived stress levels in the intervention group but not in the control group (Gaab et al., 2006). Research has also examined the impact of brief cognitive interventions on HPA response to pharmacological challenges. One study employing a pentagastrin infusion challenge showed that experimental manipulations enhancing a sense of control, reducing novelty and coaching participants to make more adaptive appraisals, reduced cortisol responses to the challenge (Abelson et al., 2008). Another study employing a corticotropin-releasing hormone challenge and a similar experimental manipulation showed that enhanced control, reduced novelty and appraisal-coaching reduced adrenocorticotrophic hormone responses but not cortisol responses (Abelson et al., 2010). Taken together, these findings suggest that changes in coping strategies can impact cortisol reactivity. Future studies are needed to examine relations between specific coping strategies and HPA/SNS function in the early stages of trauma recovery.

7.2.2. Do stress hormones facilitate or constrain specific coping strategies?—According to Hobfoll (1989), individuals exposed to stressors who engage in maladaptive coping strategies will deplete their available resources, leading to increased stress levels. Research suggests that higher stress levels may be associated with greater reliance on maladaptive coping strategies (e.g., Solomon et al., 1988). In addition, higher PTSD symptom severity predicts decreased problem-focused coping (Solomon et al., 1988) and increased avoidance coping (Benotsch et al., 2000) in combat veterans. Results of these

studies suggest that stress levels have the capacity to influence coping strategies. Research examining the impact of stress hormones on coping suggests that stress-related increases in cortisol levels serve an adaptive function by restoring goal-directed processing of emotional information following a period of automatic and stimulus-driven processing; this could lead to greater approach-driven behavior in healthy individuals or it could enhance threat-avoidance behavior in anxious individuals (Putman and Roelofs, 2011). Higher cortisol levels may be necessary for the goal-directed processing involved in active/voluntary coping strategies. In contrast, we might expect lower cortisol levels to be associated with more automatic and stimulus-driven processing characteristic of involuntary stress responses. It is unclear whether investigations of single cortisol administration on cognitive processing (Putman and Roelofs, 2011) extend to diurnal cortisol levels, and how these findings apply to trauma-exposed individuals. Although the present review found that diurnal cortisol levels were lower in adults at risk for PTSD, studies have also demonstrated increased cortisol stress reactivity in adults with PTSD (de Kloet et al., 2006). Exaggerated HPA responses to perceived threat may facilitate disengagement coping strategies among adults at risk for PTSD. In contrast to adults, diurnal cortisol levels in children were initially higher; data regarding SNS and HPA stress reactivity in children is inconsistent and may be influenced by developmental factors (e.g., Gunnar et al., 2009). Future studies are needed to assess the impact of HPA and SNS activity on trauma-related coping, to examine whether increased HPA stress reactivity in adults could be co-opted by psychotherapeutic interventions to encourage adaptive coping strategies, and to explore whether development influences the relation of traumatic stress hormone responses to coping.

8. Recommendations

Research examining coping and neuroendocrine function in the immediate aftermath of trauma would benefit from a number of methodological improvements. First, given the findings that individuals exposed to trauma tend to utilize more coping strategies of all types, it is critical for future studies to include measures that assess relative use of different coping strategies within individuals (e.g., Connor-Smith et al., 2000). Second, there is a need for more detailed assessment of potentially adaptive primary control and secondary control coping strategies (e.g., cognitive restructuring, emotion regulation, acceptance), as the bulk of research to this point has focused on maladaptive disengagement strategies. As illustrated in the present review, there is also a need to identify the 'active ingredients' of social support that are associated with adaptive outcomes. Third, although prospective studies have examined coping as a predictor of PTSD symptom severity, there is a need to identify within-individual changes in coping after trauma-exposure and to examine relations between coping and PTSD symptom trajectories. Individual differences in trauma responses can be examined using latent growth mixture modeling approaches to capture prototypical trajectories (e.g., Bonanno and Mancini, 2012). Ecological momentary assessment studies (Tennen et al., 2000) may also be used to examine more nuanced relations between coping and PTSD symptoms after trauma. Fourth, the majority of prospective studies assessing trauma-related SNS or HPA function have focused on individuals who experienced motor vehicle accidents. Future studies should assess acute biological predictors of PTSD symptoms in individuals exposed to different types of trauma, such as interpersonal violence. Fifth, studies should assess both morning and afternoon/evening HPA activity during the early recovery period due to findings suggesting that these may be differentially related to PTSD risk. Sixth, there is a dearth of studies assessing patterns of acute trauma-related coping in children. Longitudinal data of neuroendocrine function in trauma-exposed children can help to disentangle the relative influence of maturational factors and biological adaptation over time. Seventh, future research should investigate bidirectional relations between coping factors and HPA/SNS activation in the aftermath of trauma to identify patterns of mutual facilitation or inhibition that could inform combined psychosocial and

pharmacological interventions for PTSD. Finally, research on trauma-related coping should be extended beyond peripheral indicators of HPA and SNS function to investigate structural and functional properties of brain regions involved in stress reactivity and regulation as well as interactions between genetic and environmental factors.

9. Summary and Conclusions

The bulk of research on neuroendocrine alterations and coping strategies associated with PTSD has assessed individuals long after their exposure to focal traumatic events. Relatively fewer studies have captured dynamic relations between stress hormones, coping and PTSD symptoms in the acute aftermath of trauma. The present article reviewed prospective data on acute biological (SNS and HPA activity) and psychological (coping) predictors of PTSD. Given that many individuals exhibit transient PTSD symptoms following trauma-exposure and recover without treatment, we placed particular emphasis on the timing of SNS/HPA alterations and coping strategies that were associated with subsequent PTSD to help characterize psychobiological profiles of risk and resilience. Our findings suggest that acute biological models of PTSD-risk in adults may require modification for children due to distinct patterns of peritraumatic HPA activity. Whereas disengagement coping strategies have been frequently assessed and universally acknowledged as maladaptive following trauma exposure, much less attention has focused on potentially adaptive primary control and secondary control coping strategies. Advancement of existing PTSD risk models will require careful consideration of the timing of psychobiological alterations during the early recovery phase and interactions between the stress response and coping efforts.

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Highlights

- SNS activity is positively associated with PTSD symptoms in children and adults.
- Lower peritraumatic HPA activity is linked to PTSD risk in adults.
- Higher peritraumatic HPA activity is linked to PTSD risk in children.
- Adaptive coping includes social support (received and perceived) and acceptance.
- Maladaptive coping includes avoidance, denial and thought-suppression.

Fig. 1.
Traumatic Stress Vulnerability Model

Table 1

Prospective Relations Between SNS Activity and PTSD Symptoms.

Adult Studies	Trauma Type	TE (n)	PTSD measure	SNS Measure	Risk Period		
					Peritraumatic (< 24 hrs)	Acute (< 1 month)	Enduring (~12-24 months)
Blanchard et al. (1996)	MVA	125	CAPS, CAPS-2	HRR BPR			T1 ↑HRR (1-4 months) predicts PTSD at T2. T2 PTSD associated with ↑HRR and ↑SBPR at T1.
Blanchard et al. (2002)	MVA	76	CAPS	HR/BP	T1 ↓HR predicts higher probability of PTSD at 1 and 13 months.	PTSD at 1 month associated with T1 ↓HR and ↓DBP.	PTSD at 13 months associated with T1 ↓HR and ↓DBP.
Bryant et al. (2000, 2003)	MVA	146	CIDI	HR/BP		↑HR (~1 week) predicts PTSD at 6 months.	↑HR (~1 week) predicts PTSD at 2 years.
Buckley et al. (2004)	MVA	65	SCID IES	HR/BP	HR/BP not associated with PTSD at 1 month.		
Delahanty et al. (2000, 2003b)	MVA	99	SCID IES	15-hr urinary EPI/NE; HR/BP	Dissociation associated with lower EPI and NE.	PTSD at 1 month associated with lower EPI at T1.	
Ehring et al. (2008b)	MVA	53	SCID PDS	HR/BP	PTSD (6 months) predicted by ↓T1 DBP (< 9 hrs), not HR.		
Griffin (2008)	Sexual or physical assault	40	CAPS	Acoustic startle (EMG, HR)		Similar eyeblink EMG and HR responses for PTSD and non- PTSD at 1 month.	PTSD had larger HR and eyeblink EMG responses compared to non-PTSD at 6 months.

Adult Studies	Trauma Type	TE (n)	PTSD measure	SNS Measure	Risk Period		
					Peritraumatic (< 24 hrs)	Acute (< 1 month)	Intermediate (~1-11 months) Enduring (~12-24 months)
Guthrie & Bryant (2005)	Firefighters	35	CAPS ASDI IES	Acoustic startle (EMG, SC)		↑IES at T2 (< 4 weeks) linked to ↑SC response to startle at T1 (pre-trauma).	
Hawk et al. (2000)	MVA	55	SCID IES	150hr urinary EPI/NE		↑PTSD symptoms at 1 month associated with ↑EPI and ↑NE for men, not women.	↑PTSD symptoms at 6 months associated with ↑EPI for men, not women.
Heinrichs et al. (2005)	Firefighters	43	PTSS	24-hr urinary EPI/NE	EPI/NE not linked to PTSS.		
Kuhn et al. (2006)	MVA	50	CAPS PCL	HR	↑HR in ED associated with peritraumatic dissociation.	HR in ED not associated with PTSD at 1 or 3 months.	↑HR in ED predicted PTSD symptom severity at 6 months.
Shalev et al. (1998)	MVA	86	CAPS SCID IES	HR BP	↑HR (< 12 hrs) in PTSD vs. non-PTSD. ↑HR (< 12 hrs) predicted PTSD at 4 months.	↑HR (1 week) in PTSD compared to non-PTSD.	HR (1 and 4 months) did not distinguish PTSD from non-PTSD.
Shalev et al. (2000)	Accidents (e.g., MVA)	218	CAPS IES	Acoustic startle (EMG, SC, HR)		No differences between PTSD and non-PTSD in startle responses at 1 week.	↑HR and ↓habituation (SC, EMG) in PTSD vs. non-PTSD at 1 and 4 months.
Vidlock et al. (2008)	Accidents (e.g., MVA)	155	CAPS IES-R	Plasma and urinary NE, HR	Plasma NE (ED) not associated with PTSD in ER or	↓plasma NE in PTSD compared to non-PTSD	↓plasma NE in PTSD compared to non-PTSD group at 5 months.

Adult Studies	Trauma Type	TE (n)	PTSD measure	SNS Measure	Risk Period		
					Peritraumatic (< 24 hrs)	Acute (< 1 month)	Intermediate (~1-11 months) Enduring (~12-24 months)
Yehuda et al. (1998)	Rape	20	SCID-P	Plasma MHPG	at 5 months. PTSD (ED) not associated with ↑NE:cortisol ratio or HR.	group at 10 days and 1 month.	
Zatzick et al. (2005)	Injured surgical patients	161	PCL	HR	MHPG (< 51 hrs) did not predict PTSD at T2 (mean = 90 days).	↑PCL at 1 month associated with ↑HR (95) in ER.	↑PCL at 4-6 months associated with ↑HR (95) in ER. ↑PCL at 12 months linked to ↑HR in ER.
Child Studies	Trauma Type	TE (n)	PTSD measure	SNS Measure	Risk Period		
					Peritraumatic (< 24 hrs)	Acute (< 1 month)	Intermediate (~1-11 months) Enduring (~12-24 months)
Delahanty et al. (2005)	MVA (71%)	82	CAPS-CA	12-hr urinary EPI/NE		PTSD symptoms at 6 weeks linked to ↑T1 EPI.	
Kassam-Adams et al. (2005)	Traffic-related injury	190	CAPS-CA	HR	Higher HR predicted PTSD at 6 months.		
Pervanidou et al. (2007)	MVA	TE = 60; NTC = 40	KSADS-PL IES	Plasma NE	No differences in NE between PTSD, non-PTSD, and NTC < 24 hrs.	↑NE in PTSD (T2 and T3) vs. non-PTSD and T3 (6 months).	↑NE in PTSD (T2 and T3) vs. non-PTSD and NTC at T3 (6 months).

Note: TE = trauma-exposed; PTSD = posttraumatic stress disorder; SNS = sympathetic nervous system activity; HR = heart rate; HRR = heart rate reactivity to stressor; BP = blood pressure; BPR = blood pressure reactivity to stressor; SBPR = systolic blood pressure reactivity; DBP = diastolic blood pressure; EPI = epinephrine; NE = norepinephrine; MVA = motor vehicle accident; CAPS = Clinician-administered PTSD Scale; IES = Impact of Events Scale; CIDI = Composite International Diagnostic Interview; SCID = Structured Clinical Interview for DSM-IV; PDS = Posttraumatic Diagnostic Scale; EMG = left orbicularis oculi electromyogram (eye blink); SC = skin conductance; ED = emergency department; ASDI = Acute Stress Disorder Interview; PTSS = PTSD Symptom Scale; PCL = Post-Traumatic Stress Disorder Checklist – Civilian Version; MHPG = 3-methoxy-4-hydroxyphenylglycol (metabolite of NE); NTC = never-traumatized control; KSADS-PL = Kiddie Schedule for Affective Disorders and Schizophrenia – Patient Version; CAPS-CA = CAPS for Children and Adolescents.

Table 2

Prospective Relations Between HPA Function and PTSD Symptoms.

Adult Studies	Trauma Type	TE (n)	PTSD measure	HPA Measure	Peritraumatic (< 24 hrs)	Risk Period		
						Acute (< 1 month)	Intermediate (~ 1 -11 months)	Enduring (~ 12 -24 months)
Aardal-Eriksson et al. (2001)	Mine accident	31	IES PTSD	Salivary cortisol (am, pm)	$\uparrow$ IES linked to $\downarrow$ am cortisol and $\uparrow$ pm cortisol at 5 days.	$\uparrow$ IES associated with $\uparrow$ pm cortisol at 2 and 9 months.		
Anisman et al. (2001)	Ice storm	115	IES	Salivary cortisol (am)	Overall, TE had $\uparrow$ IES and $\downarrow$ cortisol relative to NTC at T1 at 1 month. However, TE with highest IES had lowest cortisol.		Cortisol similar in TE and NTC at 1 year. No relation between T2 IES and T2 cortisol	
Bonne et al. (2003)	Accident (e.g., MVA)	21	CAPS IES	Plasma cortisol (am)	Cortisol (1 week) not linked to IES at 1 week or 6 months.	$\uparrow$ IES associated with $\downarrow$ am cortisol at 6 months in PTSD group.		
Cieslak et al. (2011)	MVA	30	IES-R	Salivary cortisol (am, pm)	$\uparrow$ Cortisol output at 1 month predicted $\uparrow$ IES-R at 1 and 3 months.	$\uparrow$ IES -R at 1 week and 1 month predicted lower cortisol sensitivity at 3 months.		
Delahanty et al. (2000, 2003a, 2003b)	MVA	99	SCID IES	15-hr urinary cortisol	Dissociation not related to cortisol at T1 (< 15 hrs).	PTSD at 1 month associated with lower cortisol at T1 (< 15 hrs). Cortisol mediated relations of trauma history and injury severity to IES.		
Ehring et al. (2008b)	MVA	53	SCID PDS	Salivary cortisol (am, pm)	$\downarrow$ T1 cortisol (< 12 hrs) predicted PTSD and MDD symptoms at 6 months. $\downarrow$ cortisol associated with prior trauma.	Higher pm cortisol at T1 (< 12 hrs) linked to more severe PTSD and MDD symptoms at 6 months.		

Adult Studies	Trauma Type	TE (n)	PTSD measure	HPA Measure	Risk Period		
					Peritraumatic (< 24 hrs)	Acute (<1 month)	Intermediate (~1-11 months) Enduring (~12-24 months)
Hawk et al. (2000)	MVA	55	SCID IES	15-hr urinary cortisol		↑PTSD symptoms at 1 month associated with ↑cortisol for men, not women.	PTSD not associated with cortisol at 6 months.
Heinrichs et al. (2005)	Firefighters	43	PTSS	Salivary cortisol (am, pm)	Cortisol not associated with PTSS.		
Maes et al. (1998)	Hotel fire, MVA	10	CIDI	24-hr urinary cortisol			PTSD associated with ↑cortisol at 6-9 months.
McFarlane et al. (1997)	MVA	40	CAPS IES	Plasma cortisol (mean time = 2pm)	Higher cortisol at T1 (ED admission) predicted MDD symptoms at 6 months.		PTSD at 6 months linked to ↓cortisol than MDD at T1 (ED admission); PTSD did not differ from non-PTSD.
McFarlane et al. (2011)	Accident (e.g., MVA)	48	CAPS-II IES-R	24-hr urinary cortisol, salivary cortisol (am, pm), DST (0.5mg)	↓am cortisol on day 2 associated with increased odds of PTSD at 1 and 6 months.	Hyper-suppression of cortisol associated with PTSD at 1 month.	↑IES-R at 6 months linked to ↓am cortisol and ↑pm cortisol on day 2. Trend for hyper-suppression in PTSD group at 6 months.
Resnick et al. (1995)	Rape	37	SCID-P	Plasma cortisol (timing NR)	Cortisol (< 51 hrs) did not predict PTSD at T2 (mean = 90 days). Prior trauma associated with ↓cortisol and ↑PTSD risk.		
Shalev et al. (2008)	Accidents (e.g., MVA)	155	CAPS IES-R	Plasma, salivary (timing NR), and urinary cortisol	Cortisol measures did not distinguish PTSD and non-PTSD at any time point. Women had ↓plasma		PTSD symptoms at 5 months associated with ER and 1 week ACTH levels in women.

Adult Studies	Trauma Type	TE (n)	PTSD measure	HPA Measure	Risk Period		
					Peritraumatic (<24 hrs)	Acute (<1 month)	Intermediate (~1-11 months) Enduring (~12-24 months)
Turner-Cobb et al. (2010)	Close relatives of persons with severe brain injury	15	IES	Salivary cortisol (am, pm)	ACTH at all time points. ↑IES associated with ↑cortisol at T1 (admission to rehabilitation facility).		IES not associated with cortisol output at 6 months.
Child Studies	Trauma Type	TE (n)	PTSD measure	HPA Measure	Risk Period		
					Peritraumatic (<24 hrs)	Acute (<1 month)	Intermediate (~1-11 months) Enduring (~12-24 months)
Delahanty et al. (2005)	MVA (71%)	82	CAPS-CA	12-hr urinary cortisol		PTSD symptoms at 6 weeks associated with higher cortisol at T1 (< 12 hrs).	
Pervanidou et al. (2007)	MVA	TE = 60 NTC = 40	KSADS-PL	Serum cortisol (am), Salivary cortisol (am, pm)	↑ 12 pm, ↑6 pm, and ↑9 pm salivary cortisol in PTSD (T2 and T3) vs. non-PTSD and NTC at T1 (< 24 hrs).	No differences in salivary cortisol between PTSD (T2 and T3), non-PTSD, and NTC groups at T3 (6 months).	

Note: TE = trauma-exposed; NTC = non-traumatized controls; PTSD = posttraumatic stress disorder; HPA = hypothalamic-pituitary-adrenocortical axis; am = morning (< 12:00 pm); pm = afternoon (> 12:00 pm); MVA = motor vehicle accident; CAPS = Clinician Administered PTSD Scale; IES = Impact of Events Scale; IES-R = IES-Revised; PTSS = Post Traumatic Symptom Scale; PTSS = PTSD Symptom Scale; SCID = Structured Clinical Interview for DSM-III-R; DST = dexamethasone suppression test; ED = emergency department; PDS = Posttraumatic Diagnostic Scale; CIDI = Composite International Diagnostic Interview; ACTH = adrenocorticotrophic hormone; NR = not reported; SCID-P = Structured Clinical Interview for DSM-III-R – Patient Version; CAPS-CA = Clinician Administered PTSD Scale for Children and Adolescents; KSADS-PL = Kiddie Schedule for Affective Disorders and Schizophrenia – Lifetime Version.

Table 3

Cross-Sectional Relations Between Coping and PTSD Symptoms.

Adult Studies	Trauma Type	TE (n)	PTSD measure	Coping Measure	Time Since Focal Trauma	Key Findings
Amir et al. (1997)	Mixed	42	IES	AECOM	NR	↑Suppression associated with ↑IES.
Astin et al. (1993)	Interpersonal violence	53	IES PSC	SSQ	~ 8 months	↑Social support associated with ↓PTSD symptoms.
Besser & Neria (2012)	Missile threat	135	PTSD-I	MSPSS	NR	↑Perceived social support associated with ↓PTSD symptoms. Perceived social support mediated relation of attachment anxiety to PTSD symptoms.
Blake et al. (1992)	Combat	64	MMPI-PK	WCC-R	NR	PTSD associated with ↑emotion-focused coping, including ↑accepting responsibility and ↑escape-avoidance.
Bryant et al. (2000)	Severe TBI	96	PTSD-IN	CSQ	6 months	↑Avoidant and active behavioral coping associated with ↑PTSD symptom severity.
Bryant & Harvey (1995)	MVA	56	IES	CSQ	12 months	↑Avoidant coping associated with ↑IES-Intrusion.
Chang et al. (2003)	Earthquake	84	IES	WCQ	5 months	↑Escape-avoidance coping associated with ↑IES. ↑Positive reappraisal associated with ↓IES.
Chung et al. (2008)	Myocardial Infarction	96	PDS	COPE	~10 years	PTSD associated with ↑suppression, ↑restraint coping, ↑focus on and venting of emotion, ↑ mental disengagement.
Clohessey & Ehlers (1999)	Emergency service incidents	56	PSS	COPE (extended)	NR	↑Wishful thinking associated with ↑PTSD severity.
DePrince et al. (2011)	Nonsexual partner abuse	236	PDS	ISEL	26 days	Social support not associated with PTSD symptoms.
Fairbank et al. (1991)	POW	30	MMPI-PK	WCC-R (for memories of captivity)	NR	PTSD linked to ↑self-isolation, ↑wishful thinking, ↑self-blame, and ↑social support seeking.

Adult Studies	Trauma Type	TE (n)	PTSD measure	Coping Measure	Time Since Focal Trauma	Key Findings
Gold et al. (2007)	Sexual assault	74	PDS	AAQ	NR	PTSD linked to ↑experiential avoidance.
Gold et al. (2009)	Sexual assault	72	PDS	AAQ	NR	PTSD linked to ↑experiential avoidance.
Goldenberg & Matheson (2005)	Mixed	95	TSI	WCC-R	NR (> 1 month)	↑PTSD symptoms associated with ↑passive coping (self-blame, avoidance, wishful thinking), not associated with active coping (problem-focused, seeking social support).
Harvey & Bryant (1998)	mTBI caused by MVA	48	CIDI-P ASDI	CSQ	< 18 days	↑PTSD symptoms associated with ↑avoidant coping.
Hough et al. (1990)	Sniper massacre community	290	DIS	Social support (confidant)	~ 6 months	Severe PTSD symptoms more common for individuals with a confidant than for those without.
Kanninen et al. (2002)	Political prisoners	103	HTQ	Coping items from Frijda et al. (1989)	Range: 3 to 180 months	↑Emotion-focused coping associated with ↑vigilance and ↑intrusion symptoms. ↑Problem-focused coping associated with ↑avoidance symptoms. ↑Emotion-focused coping associated with ↓PTSD symptoms for those with more recent trauma exposure and ↓PTSD symptoms for those with more distant trauma exposure. ↑Problem-focused coping associated with ↓PTSD symptoms for more recent trauma exposure and ↑PTSD symptoms for more distant trauma exposure.
Kashdan et al. (2009)	Survivors of Kosovo War	74	MINI	AAQ	~ 7 years	PTSD associated with ↑experiential avoidance.
King et al. (1998)	Vietnam veterans	1,632	DIS	Social support (functional)	> 20 years	↑Social support associated with ↓PTSD symptoms.
Kramer & Green (1991)	Sexual assault	30	Interview IES	Social support (network)	NR	↑Social support associated with ↓PTSD symptoms.
Littleton & Grills-Taquechel (2011)	Sexual assault	340	PSS	CSI	NR	↑PTSD symptoms associated with ↑avoidant coping and ↓approach coping.

Adult Studies	Trauma Type	TE (n)	PTSD measure	Coping Measure	Time Since Focal Trauma	Key Findings
Mathews et al. (2009)	Accidental injury (MVA)	69	PCL	CSQ	~8 months	PTSD associated with ↑avoidance coping.
Morina (2007)	Survivors of Kosovo War	152	HTQ IES-R	AAQ	~ 6 years	↑Experiential avoidance associated with ↑PTSD symptoms.
Morina et al. (2008)	Survivors of Kosovo War	84	HTQ MINI IES-R	AAQ	~ 6 years	↑Experiential avoidance associated with current, but not past, PTSD.
Orcutt et al. (2005)	Interpersonal trauma	229	DEQ	AAQ	NR	↑Experiential avoidance associated with ↑PTSD symptoms. Experiential avoidance partially mediated relation of interpersonal trauma to PTSD symptoms.
Perrin et al. (1996)	Domestic abuse	69	MMPI-PK	ISEL	NR	↑Social support associated with ↓PTSD symptoms.
Pietrzak et al. (2009)	OEF/OIF veterans	272	PCL-M	CD-RISC; USS; PSSS	NR	PTSD associated with ↓PSSS and ↓USS. ↑PTSD symptoms associated with ↓personal control and ↓acceptance of changes.
Santello & Leitenberg (1993)	Sexual assault (acquaintance)	106	PTSD symptom checklist	CSI	~ 2 years	↑PTSD symptoms associated with ↑disengagement coping (problem-avoidance, social-withdrawal, self-criticism).
Shipherd & Beck (1999)	Sexual assault	36	PTSD-IN IES	Experimental thought suppression	~ 74 months	PTSD associated with increase in rape-related thoughts following suppression phase; non-PTSD showed no increase.
Solkoff et al. (1986)	Combat	50	PTSD symptom checklist	Interview	NR	PTSD associated with ↓perceived support from family and spouses after return home.
Solomon et al. (1987)	Combat	684	PTSD-IN	MCEI	~ 12 months	↑Social support associated with ↓PTSD symptoms.
Steil & Ehlers (2000)	MVA	Study 1 159 Study 2 138	PSS	PAQ; Cognitive control of intrusions	~ 7 years	↑PTSD symptom severity associated with ↑avoidance or reminders, ↑thought suppression, and ↑distraction.

Adult Studies	Trauma Type	TE (n)	PTSD measure	Coping Measure	Time Since Focal Trauma	Key Findings
Sutker et al. (1995)	Persian Gulf War veterans	581	MISS PCL-M	WCC SSQ FRI	35-287 days after Persian Gulf duty	PTSD linked to ↑blaming self, ↑wishful thinking, ↑avoidance, ↓problem-focused coping, ↑social support (number, satisfaction), and ↓family support (cohesion, expressiveness) vs. no distress group.
Thompson & Walz (2010)	Mixed trauma	191	PDS	FFMQ AAQ WBSI CISS	NR	↑PTSD avoidance symptoms linked to ↑experiential avoidance, ↑thought suppression, ↑emotion-oriented coping, and ↓mindfulness.
Tull et al. (2004)	Sexual assault	160	PCL	AAQ WBSI	NR	↑PTSD symptom severity linked to ↑experiential avoidance and ↑PTSD thought suppression.
Tull et al. (2007)	Mixed trauma	116	PCL	DERS	NR	↑PTSD symptom severity linked to ↑emotion regulation difficulties (i.e., lack of emotional acceptance, lack of emotional clarity, limited access to effective emotion-regulation strategies, difficulties engaging in goal-directed behavior when upset, impulse-control difficulties).
Vázquez et al. (2008)	Civilians exposed to Madrid terrorist attack	503	PCL-C	WBSI Coping questions based on Schuster et al. (2001)	2-3 weeks	↑PTSD symptoms associated with ↑thought suppression and ↑coping behaviors (talking with others about thoughts/feelings, focus on religion/praying, social engagement related to attack, helping victims, supporting/comforting those who are close, avoid thinking about attack, avoid reminders)
Vujanovic et al. (2009)	Mixed trauma	239	PDS	KIMS	NR	↑Accepting without judgment associated with ↓PTSD symptoms.
Weiss et al. (1995)	Emergency medical service personnel	367	MISS	Current Social Support Scale	Mixed	↑Social support associated with ↓PTSD symptoms.
Child Studies	Trauma Type	TE (n)	PTSD measure	Coping Measure	Time Since Focal Trauma	Key Findings
Bal et al. (2003)	Sexual abuse	96	CAPS-CA TSCC	HICUPS	NR	↑Avoidant coping associated with ↑PTSD symptoms.

Adult Studies	Trauma Type	TE (n)	PTSD measure	Coping Measure	Time Since Focal Trauma	Key Findings
Dempsey (2002)	Exposure to violence	120	CCDS	CHSE		↑Negative coping (ignoring problem, crying, hitting or fighting, screaming or yelling) linked to ↑PTSD symptoms.
Dempsey et al. (2000)		70		KidCope	NR	↑Behavioral avoidance coping linked to ↑PTSD reexperiencing symptoms. Relation of violence to PTSD hyperarousal moderated by behavior avoidance. Relation of violence to PTSD reexperiencing moderated by cognitive distraction.
Merrill et al. (2001)	Childhood sexual abuse	1,134	TSI	"How I Deal With Things" scale (Burt & Katz, 1987)	NR	↑TSI associated with ↑self-destructive coping, ↑avoidant coping, and ↓constructive coping.
Weisenberg et al. (1993)	Missile attacks	492	SRQ	Coping behavior questionnaire	3 weeks after war	PTSD associated with ↑checking, ↑reassurance request, and ↓verbal distraction.

Note: TE = trauma-exposed; PTSD = posttraumatic stress disorder; NR = not reported; MVA = motor vehicle accident; TBI = traumatic brain injury; CAPS = Clinician Administered PTSD Scale; PTSD-I = PTSD Inventory; PTSD-IN = PTSD Interview; PDS = Posttraumatic Stress Diagnostic Scale; PSS = Posttraumatic Stress Symptom Scale; PSC = PTSD Symptom Checklist; IES = Impact of Events Scale; MMPI-PK = Minnesota Multiphasic Personality Inventory – PTSD scale; AECOM = Albert Einstein College of Medicine Coping Styles Questionnaire; CSQ = Coping Style Questionnaire; WCQ = Ways of Coping Questionnaire; WCC = Ways of Coping Checklist; WCC-R = WCC-Revised; SSQ = Social Support Questionnaire; mTBI = mild traumatic brain injury; POW = prisoner of war; TSI = Trauma Symptom Inventory; CID-I-P = Composite International Diagnostic Interview – PTSD module; ASDI = Acute Stress Disorder Interview; DIS = Diagnostic Interview Schedule/Disaster Supplement; ISEL = Interpersonal Support Evaluation List; AAQ = Acceptance and Action Questionnaire; HTQ = Harvard Trauma Questionnaire; IES-R = IES - Revised; PCL = PTSD Checklist Civilian Version; MINI = MINI International Neuropsychiatric Interview; CSI = Coping Strategies Inventory; OEF/OIF = Operations Enduring Freedom and Iraqi Freedom; PCL-M = PCL – Military Version; DEQ = Distressing Events Questionnaire; CD-RISC = Connor-Davidson Resilience Scale; USS = Unit Support Scale; PSSS = Postdeployment Social Support Scale; MCEI = Military Company Environment Inventory; PCL-M = PTSD Checklist Military Version; MISS = Mississippi Scale for Combat-Related PTSD; PAQ = Postaccident Avoidance Questionnaire; FRI = Family Relationships Index; FFMQ = Five Facet Mindfulness Questionnaire; WBSI = White Bear Suppression Inventory; CISS Coping in Stressful Situations; DERS = Difficulties in Emotion Regulation Scale; PCL-C = PTSD Checklist Civilian Version; KIMS = Kentucky Inventory of Mindfulness Skills; CAPS-CA = CAPS for Children and Adolescents; TSCC = Trauma Symptom Checklist for Children; CCDS = Checklist of Children's Distress Symptoms; SRQ = Stress Reaction Questionnaire; HICUPS = How I Cope Under Pressure Scale; CHSE = Coping in the Home and School Environments.

Table 4

Prospective Relations Between Coping and PTSD Symptoms.

Adult Studies	Trauma Type	TE (n)	PTSD measure	Coping Measure	Time Since Focal Trauma	Key Findings
Benotsch et al. (2000)	Gulf War veterans	348	PCL	WCC SSQ FRI	T1 = 14 months after end of hostilities; T2 = 13 months after T1	↑T1 avoidance coping predicted ↑T2 PTSD symptoms. ↑T1 PTSD symptoms also predicted ↑T2 avoidance coping and ↓T2 family cohesion.
Dalgleish et al. (1996)	Ferry sinking disaster	37	IES	CSS (received support)	T1 = retrospective trauma report T2 = 3 years T3 = 6 years	No association between IES and crisis support at T2 or T3. ↑T1 crisis support predicted ↓T3 avoidance.
Ehlers et al. (1998)	MVA	967	PSS	Thought suppression	3 months, 1 year	Thought suppression correlated with concurrent PTSD symptoms at 3 months and 1 year. ↑Thought suppression at 3 months predicted ↑PTSD symptoms at 1 year.
Ehring et al. (2008c)	MVA	147	PDS	RIQ CSS	2 weeks, 1 month, 3 months, 6 months	↑Social support associated with ↓PTSD severity at all time points. ↑Thought suppression associated with ↑PTSD severity at all time points.
Eid (2003); Eid et al. (2001); Johnsen et al. (2002)	Military training fatalities	122	IES PTSS-10	CSQ	2-3 weeks, 4 months, 12 months	↑Emotion-focused coping at 2-3 weeks predicted ↓PTSD symptoms at 12 months. ↑IES linked to ↑avoidant coping at 3 weeks and 4 months. ↑Task-focused coping at 2-3 weeks predicted ↓PTSD symptoms at 4 months. ↑Avoidant coping linked to maintenance of PTSD symptoms over time.
Ginzburg et al. (2002)	Myocardial Infarction	116	PTSD-I	RCS	1 week, 7 months	↑Repressive coping associated with ↓PTSD symptoms at 1 week and 7 months.
Hepp et al. (2005)	Accidental injury	106	CAPS-2	FQCI	1, 6, and 12 months	Full or subsyndromal PTSD associated with more rapid decreases in active coping over time and ↑downplaying and wishful thinking.
Joseph et al. (1993)	Cruise ship disaster	17	IES	CSS	T1 = 3-9 months T2 = 14 months T3 = 18 months	↓IES-avoidance symptoms at T3 predicted by ↑crisis support at T1 and T2.
Kumpula et al. (2011)	Campus shooting	532	DEQ	AAQ-II	T1 = pre-trauma T2 = ~27 days T3 = 35 weeks	↑T1 experiential avoidance predicted ↑T2 PTSD symptoms. ↑T2 experiential avoidance predicted ↑T3 PTSD symptoms.

Adult Studies	Trauma Type	TE (n)	PTSD measure	Coping Measure	Time Since Focal Trauma	Key Findings
Marx & Sloan (2005)	Mixed	185	PDS	AAQ	1 month – 5 years after trauma. T1 = baseline T2 = 4 weeks T3 = 8 weeks	↑Experiential avoidance at baseline predicted ↑PTSD symptom severity at T2 and T3.
McFarlane (1989)	Bushfire disaster	469	IES	Retrospective report of coping at 11 months	4, 11, and 29 months	PTSD at 11 and 29 months associated with ↑social support seeking.
Spurrell & McFarlane (1993)		(147)	DIS	WCQ	42 months	PTSD group used more problem-focused coping and wishful thinking than group with no disorder.
Nightingale & Williams (2000)	MVA	60	IES PDS	WCQ	T1 < 1 week. T2 = 6 weeks	↑Escape-avoidance coping predicted T2 PTSD.
North et al. (2001)	Survivors of mass murder spree	136	DIS	RTE	T1: 3-4 months T2: 1 year T3: 3 years	PTSD at T1 linked to ↓active outreach and ↓informed pragmatism coping. PTSD (T2 and T3) linked to ↓informed pragmatism coping.
Perry et al. (1992)	Burn victims	51	SCID	ISEL	T1 = 1 week T2 = 2 months T3 = 6 months T4 = 12 months	↓T1 perceived social support predicted ↑PTSD symptoms at 2, 6, and 12 months.
Plumb et al. (2004)	Mixed	Study 2 160 Study 3 37	PDS CAPS	AAQ	Time since trauma NR. T1 = baseline T2 = 8 weeks	↑T1 experiential avoidance predicted ↑T2 PTSD symptom severity.
Resick (1988)	Sexual assault, robbery	59	IES	Social support (perceived, number, network size)	T1: 1 month T2: 3 months T3: 6 months T4: 12 months T5: 18 months	↑Behavioral avoidance coping associated with ↑PTSD symptoms. ↑Perceived support and ↑network size associated with better recovery for robbery victims.
Schuster et al. (2011)	Mixed trauma	70	PDS	Brief COPE	2 assessments 15 months apart	↑T1 avoidant coping (denial, behavioral disengagement, substance use, self-blame) linked to ↑PTSD symptoms at T1 and T2.
Sharkansky et al. (2000)	Combat	1,058	MISS	CRI	T1 = return to United States (CRI administered < 5 days); T2 = 18-24 months	↑Approach coping in war zone associated with ↓T1 PTSD symptoms. Approach coping did not predict change in PTSD symptoms from T1-T2.
Silver et al. (2002)	September 11, 2001, attacks	1,069	SARSQ	Brief COPE (administered at T1)	T1 = 9-23 days T2 = 2 months T3 = 6 months	↑Odds of PTSD symptoms associated with ↑denial, ↑self-distraction, ↑self-blame, ↑social support seeking, ↑disengagement from coping efforts.

Adult Studies	Trauma Type	TE (n)	PTSD measure	Coping Measure	Time Since Focal Trauma	Key Findings
Solomon et al. (1988)	Combat	255	PTSD-I	WCC (administered at T2 retrospectively)	T1 = 1 year after end of war T2 = 2 years	T2 PTSD associated with ↑emotion-focused coping, ↑distancing, and ↓problem-focused coping at T2. In addition, coping (emotion-focused, distancing, help-seeking) interacted with life events to predict T2 PTSD.
Ticehurst et al. (1996)	Earthquake	3,007	IES	Social support (Tucker, 1982); coping (Billings & Moos, 1981)	T1 = 27 weeks T2 = 50 weeks T3 = 86 weeks T4 = 114 weeks	↑IES associated with ↑behavioral coping and ↑avoidance coping.
Valentiner et al. (1996)	Sexual and nonsexual assault	215	PSS	WCI-A (assessed at T2)	T1 < 2 weeks T2 = 3 months	↑T2 PTSD severity associated with ↑T2 wishful thinking and ↓T2 positive distancing.
Wolfe et al. (1998)	Mixed (combat, sexual assault)	160	MISS	CRI Social Support	T1 = return from Persian Gulf War T2 = 18 months T3 = 24 months	↑Leader support associated with ↓PTSD symptoms.
Child Studies	Trauma Type	TE (n)	PTSD measure	Coping Measure	Time Since Focal Trauma	Key Findings
Ehlers et al. (2003)	Traffic accident	86	IES-C RI	Thought suppression, avoidance	2 weeks, 3 months, 6 months	Thought suppression at 2 weeks predicted PTSD severity at 3 and 6 months. Parental avoidant attitude at 2 weeks predicted PTSD severity at 6 months. Thought suppression at 3 months predicted PTSD severity at 6 months.

Note: TE = trauma-exposed; PTSD = posttraumatic stress disorder; MVA = motor vehicle accident; PDS = Posttraumatic Stress Diagnostic Scale; PSS = Posttraumatic Stress Symptom Scale; PCL = PTSD Checklist Civilian Version; IES = Impact of Events Scale; PTSS-10 = Post-Traumatic Symptom Scale – 10 item version; PTSD-I = PTSD Inventory; WCC = Ways of Coping Checklist; RIQ = Response to Intrusions Questionnaire; CSS = Crisis Support Scale; SSQ = Social Support Questionnaire; FRI = Family Relationships Index; CSQ = Coping Style Questionnaire; RCS = Repressive Coping Scale; CAPS = Clinician Administered PTSD Scale; DEQ = Distressing Events Questionnaire; DIS = Diagnostic Interview Schedule/Disaster Supplement; FQCI = Freiburg Questionnaire of Coping with Illness; AAQ = Acceptance and Action Questionnaire; WCQ = Ways of Coping Questionnaire; RTE = Response to Traumatic Events inventory; MISS = Mississippi Scale for Combat-Related PTSD; SCID = Structured Clinical Interview for DSM-III-R; SARSQ = Stanford Acute Stress Reaction Questionnaire; ISEL = Interpersonal Support Evaluation List; CRI = Coping Responses Inventory; IES-C = IES children's version; RI = Children's Post-traumatic Stress Reaction Index; WCI-A = Ways of Coping Inventory – Abbreviated.

Table 5

Summary of Findings for Associations between PTSD symptoms and HPA, SNS, and Coping.

		Peritraumatic (< 24 hrs)	Acute (< 1 month)	Intermediate (1-11 months)	Enduring (12-24 months)
HPA	Adult	↓	↓a.m, ↑p.m.	↓a.m, ↑p.m.	?
	Child	↑	↑↓	?	?
SNS	Adult	↑↓	↑	↑	↑↓
	Child	↑	↑	↑	?
Coping	Adaptive	?	↓	↓	↓
	Maladaptive	?	↑	↑	↑

Note: ↑ = Positive association with PTSD symptoms; ↓ = Negative association with PTSD symptoms; ↑↓ = equivocal findings; ? = insufficient evidence; HPA = hypothalamic-pituitary-adrenal axis; SNS = sympathetic nervous system activity; Adaptive coping strategies = social support (received, perceived), acceptance; Maladaptive coping strategies = thought suppression, social support-seeking, avoidance, denial, wishful thinking; a.m. = morning cortisol levels; p.m. = afternoon/evening cortisol levels.