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Review

Effects of appraisal and coping on the neuroendocrine response to extreme stress

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Abstract

Although many people are exposed to extreme stress, only some of them develop psychobiological disturbances that can lead to posttraumatic stress disorder (PTSD) or other posttrauma psychopathology. This paper examines the effects of different types of appraisal and coping to find clues to how individuals differ in their neuroendocrine responses to extreme stress. It proposes a conceptual model for components of the adult response to stressors. Threat appraisal and defensive coping may play crucial roles in determining the neuroendocrine response to trauma with potential mental health consequences, particularly PTSD.

Keywords: Stress; Trauma; Posttraumatic stress disorder; Cognitive appraisal; Coping; Cortisol

Contents

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1. Introduction

In the general population 5–6% of men and 10–12% of women suffer from PTSD at some point in their lives, making it the fourth most common psychiatric disorder (Breslau et al., 1991; Resnick et al., 1993; Kessler et al., 1995). In patients with PTSD, studies have shown alterations in brain structures and functions, dysregulation in the neuroendocrine system, psychophysiological abnormalities as well as increased somatic symptoms and illnesses. Although 50–60% of individuals experience a traumatic event in their life, only a minority develops PTSD, depression or other psychiatric or physical disorders (Kessler et al., 1995). It is yet unclear why some people show extreme psychobiological dysregulation due to traumatic events whereas others do not, or why people develop different types of psychopathology.

Our hypothesis is that individual differences in the specific appraisal of the traumatic event and in subsequent coping behavior are crucial in determining outcome. In the stress literature cognitive theories do identify appraisal and coping as critical mediators of stressful person–environment relations and their psychobiological outcomes (Lazarus, 1996; Olff et al., 1993; Ursin and Olff, 1993; McEwen, 1998; Biondi and Picardi, 1999; Olff, 1999; Ursin and Eriksen, 2004). However, these effects have rarely been established in the context of extreme or traumatic stressors (Spaccarelli, 1994; Aldwin and Yancura, 2004).

In the trauma literature there is evidence for the fact that the development of PTSD is associated with specific appraisals (McNally, 2003; Ehlers et al., 1998) and coping styles (Aldwin and Yancura, 2004). It has also been shown that peritraumatic dissociation and specific acute physiological reactions to extreme stressors are implicated in the development of posttraumatic stress disorder (PTSD) or depression (Bryant, 2003; Shalev et al., 1998a,b, 2000). However, there is very little information on how appraisal and coping affect the neuroendocrine responses to trauma. In the present paper the focus is on this latter issue. First, the neuroendocrine response to normal and traumatic stress will be described. Secondly, the (traumatic) stress literature will be reviewed with regard to associations between stress appraisals and neuroendocrine responses to stressors. Subsequently, findings concerning relationships between different types of coping and neuroendocrine data are described. Finally, these findings will be integrated in a model of the psychobiology of traumatic stress-coping in adults.

2. The neuroendocrine stress response

Two neuroendocrine response systems have been described for specific stress-coping patterns. In the event the subject has to 'fight or flight' or has to put an effort to control the situation, adrenergic mechanisms in both central nervous system (CNS) and the peripheral sympathetic nervous system (SNS) are activated. These systems respond with in seconds to threats to homeostasis with the release of neurotransmitters (catecholamines) in the adrenergic system. This sympathetic stress response causes increases in heart rate, blood pressure and in blood glucose levels in muscles and vital organs in order to have enough energy to fight or flee. The other stress response system, involving the hypothalamus pituitary–adrenocortical (HPA) system, requires a slightly longer time frame (i.e. minutes) for engagement. Brain neuropeptides stimulate the paraventricular nucleus of the hypothalamus to release corticotropin factor (CRF) and other neuromodulators from the hypothalamus. CRF stimulates the anterior pituitary gland to release adrenocorticotrophin hormone (ACTH) which in turn stimulates the adrenal glands to release cortisol. Cortisol has an important role in shutting down the sympathetic activation and to suppress the HPA axis by a negative feedback mechanism on the pituitary, hippocampus, hypothalamus and amygdala. Once there is no more threat perception, the negative feedback mechanisms help to restore basal hormone levels.

Maintenance of the integrity of the HPA system is crucial for normal adaptation (Seyle, 1956). The HPA response is activated if a perception of uncertainty and a sense of emotional distress are present in a stressful condition (see Pacák and Palkovits, 2001). In cases where efficient coping is not possible as with severe prolonged stress and the experience of negative affect, dysregulation of the HPA AXIS may appear, implying that cortisol is inhibiting the SNS to maintain physiological homeostasis (Olff, 1999).

Dysregulations of the HPA axis are associated with several psychiatric disorders (Ehlert et al., 2001). In trauma survivors with PTSD most neuroendocrine studies have demonstrated low basal cortisol levels and an augmented cortisol response to dexamethasone administration reflecting a more sensitive cortisol negative feedback inhibition of the HPA axis (e.g. Yehuda, 1997, 2002). Although in both PTSD and major depressive disorder hypersecretion of central CRF is observed, in a large proportion of depressed patients, especially the melancholic subtype, the opposite patterns are found: higher cortisol levels and a blunted negative feedback for the HPA-axis. Intriguing is the fact that although neuroendocrine patterns are so divergent in PTSD and depression following trauma, there is up to about 50% co-morbidity of PTSD and major depressive disorder (Kessler et al., 1995). Until now, however, there is no satisfactory explanation for the discrepancy between neuroendocrine findings and clinical pictures. It is unclear why after trauma in some individuals the acute activation of the HPA axis reverses to a relative deficiency of cortisol when PTSD has developed while in others it rises to higher levels as in melancholic depression. As suggested by Ehlert et al. (2001), it may depend on (inadequate) coping strategies. In their cognitive activation theory of stress (CATS) Ursin and Eriksen (2004) mention helplessness

and hopelessness as non-adaptive forms of coping in order to explain neuroendocrine differences between PTSD and depression, respectively.

3. Stress-coping

Cognitive appraisal refers to the perception, interpretation and evaluation of the stressor. It is the first step in a cascade of responses, as shown in the stress model in Fig. 1 adapted from Olff (1999). After the appraisal phase the emotional, behavioral and physiological responses are determined by the subject's specific coping and defense strategies. Defensive coping will by definition affect the appraisal of the stressor. The distorted perception of reality serves to keep the threatening character of the stressful encounter unconscious or at least minimizes the negative affective response to the stressor. This may be beneficial in the short term, but may be dangerous in situations where reality testing is important (Vaernes et al., 1982). Defensive strategies are described here as a form of emotion-focused stress-coping.

Coping may consist of more instrumental or problem-focused coping strategies where the individual tries to attack the stressor in an active way and the subjects experience control over the situation. This type of coping has also been defined in terms of positive response outcome expectancies within the cognitive activation theory of stress (Ursin and Eriksen, 2004). This may be more effective in the long term than the defensive strategies because the source of stress is being dealt with. Emotion-focused strategies further include for instance expressing emotions and seeking social support. Palliative coping styles are aimed at reducing the arousal provoked by the stressor, for instance, by alcohol or tobacco use. The present review tries to integrate stress literature on appraisal and coping in relationship to neuroendocrine responses and health outcomes with trauma literature.

4. Cognitive appraisal and neuroendocrine responses

The appraisal process, reflecting the person's subjective perception, interpretation and evaluation of the event, is the crucial first step in the cascade eventually leading to symptoms (Frazier et al., 2002). For instance, negative appraisals of stressors lead to the release of cortisol and increase the vulnerability to depression (Buchanan et al., 1999). Given that cortisol is not released until the situation is perceived as noxious, the cognitive ability to evaluate events is the key in the body's response to threat (Biondi and Picardi, 1999; Chrousos and Gold, 1998; Ehlert and Straub, 1998). Most studies about stressor appraisal demonstrate an effect on physiological reactivity (Peters et al., 1998; Tomaka et al., 1993, 1997, 1999; Maier et al., 2003; Dickerson and Kemeny, 2004).

The magnitude of neuroendocrine stress response depends on whether the stressor is appraised as *threatening* (possibility of damage/harm) or as *challenging* (opportunity for gain) (Tomaka et al., 1993, 1997, 1999; Nicolson, 1992; al'Absi et al., 1997; Epel et al., 1998; Peters et al., 2003). In terms of adaptation, the threat response pattern serves to protect the individual from attack, whereas the challenge response pattern is associated with increased energy mobilization for coping (Dienstbier, 1989). Whether the stressor is appraised as threatening versus challenging is associated with different profiles of endocrine and sympathetic arousal.

The *challenge appraisals* are associated with 'fight or flight' response patterns characterized by short-term increases in catecholamines and, probably strong anabolic

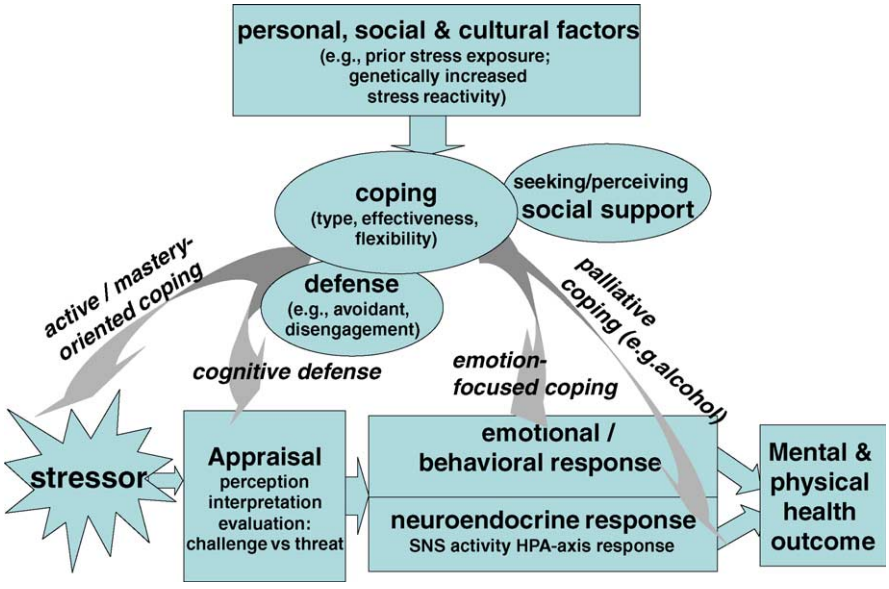

Fig. 1. Stress-coping model.

counterregulatory responses and cortisol adaptation when faced with similar stressors over time (Epel et al., 1998). Cortisol adaptation or habituation to subsequent stressors is a sign of an adaptive stress response. The challenge response is associated with demands within perceived coping ability (non-alarming appraisals), low negative emotion, and enhanced coping, and is accompanied by stronger cardiac activity (Vaernes et al., 1982; Frankenhauser, 1983), and rapid cortisol responses with quick recovery (Epel et al., 1998). Challenge appraisals indicating a shift to perceived positive arousal and perceived controllability can moderate biological stress responses, promoting physical thriving through salutary neuroendocrine responses (Epel et al., 1998; Brosschot et al., 1998).

In contrast, the *threat appraisals* are more strongly associated with demands exceeding perceived coping abilities, high negative emotion, poorer coping, and may lead to increased peripheral vascular resistance (Tomaka et al., 1993, 1997) and higher reactive levels of cortisol (Vaernes et al., 1982; Ursin et al., 1978; Lundberg and Frankenhauser, 1980). The traumatic stress literature notes appraisal of threat as an important peritraumatic predictor of PTSD (McNally, 2003). Appraisal of a stressful situation as a threat might contribute to pathology because it directs coping towards excessive emotional regulation and diverts it from problem solving.

Research has shown that individuals with high tendencies to use *defenses* such as repression, denial and distraction, appraised (laboratory) stressors as less threatening than did individuals with low defensive tendencies (Hansen et al., 1992; Tomaka et al., 1992; Myers, 1996). In a study on coping with rape, repressors recalled fewer details of stressful experiences, suggesting they may be less prone to develop PTSD due to employing selective attention during and after threat (Krahe, 1999). In particular, repressors' avoidance of negative affect may contribute to their tendency to selective inattention when confronted with threatening information (Bonanno et al., 2003).

In the stress literature there is evidence indicating that reduced threat perception can buffer the HPA response to a stressor. Psychological defenses, for instance, were associated with an attenuated neuroendocrine response to an acute laboratory stressor probably due to the reduced experience of negative affect (Olff et al., 1995). Disengagement, like the psychological defense of denial, emotional numbing, avoidance, and withdrawal in coping with psychosocial stress may shield individuals psychologically from threat and result in decreased HPA axis activation and lower cortisol (Morgan et al., 2001; Mason et al., 2001).

However, as yet, relatively few studies have related appraisals of extreme stressors to physiological reactivity. A recent study among combat veterans indicated that cognitive appraisal (i.e. perceived coping ability, but not perceived threat) mediated the observed heart rate recovery–PTSD association (Kibler and Lyons, 2004). Of the other existing trauma studies, some focused on alcohol's impairment or disruption of an initial appraisal of threat (Levenson et al., 1980; Sayette, 1993), resulting in physiological stress response dampening. Study findings do suggest that diminished threat appraisal through alcohol use just before or during a traumatic event gives rise to attenuated peritraumatic physiological arousal reactions (Koss et al., 1996; Maes et al., 2001; Clum et al., 2002). And, an association between reported alcohol use/intoxication and reduced risk for developing PTSD has been found in these studies (Mellman et al., 1998). Also of interest in this respect is that individuals with cerebral damage or who were unconscious in the direct aftermath of trauma also show a reduced risk of PTSD (Adler, 1943; Chemtob et al., 1998; O'Brien and Nutt, 1998; Bryant, 2001). Apparently, a conscious threat perception is necessary to dysregulate the stress response system. In a similar vein, the literature suggests that awareness or perception of life threat during a cardiac arrest increases the risk of developing PTSD (Van Driel and Op den Velde, 1995), whereas sedation during a cardiac arrest reduced the risk of developing PTSD fivefold (Ladwig et al., 1999).

Furthermore, there is some evidence linking *positive appraisal* and perception of control with resilient neuroendocrine functioning. Women who have grown psychologically from trauma, showed quicker cortisol habituation to other stressors, demonstrating greater flexibility in their HPA axis, probably through positive affect (Epel et al., 1998). Changing one's perception of an extreme stressor (positive reappraisal), can be effective in terms of serving as a protector of PTSD, as has been shown for example, in rescue workers after an earthquake (Chang et al., 2003). A recent study among combat veterans indicated that the cognitive appraisal of being able to cope mediated the observed heart rate recovery–PTSD association (Kibler and Lyons, 2004). Furthermore, sex differences in HPA responses to extreme stressors may be explained by gender differences in threat appraisal (Rasmusson and Friedman, 2002) or in subjective arousal (Wallbott and Scherer, 1991). Physiological differences in female and male stress response may help explain gender differences in prevalence rates of stress disorders, particularly PTSD.

5. Coping and neuroendocrine responses

After the appraisal phase, the choice of coping strategies through which individuals either attempt to change the stressful reality or to regulate their emotional reactions codetermines whether an experience is associated with severe symptoms or recovery (Lazarus, 1996). In stress research it is well known that how individuals cope with problems does have an effect on their physiology (Biondi and Picardi, 1999; Aldwin and Yancura, 2004), and subsequently on stress-related mental and physical disorders (Olff et al., 1993; Schnurr and Green, 2004). Individual differences in coping strategies codetermine the

(neuro)endocrine responses to both acute and chronic stress (Olff, 1999).

Findings in stress research can be summarized according to the type of coping strategy used and their effectiveness (Biondi and Picardi, 1999; Aldwin and Yancura, 2004; Ehlert and Straub, 1998; Vickers, 1988; Cramer, 2003). Regarding the *type of coping strategy* used, defensive coping styles based on high levels of avoidance and denial or defensive emotion-inhibiting strategies have been associated with increased neuroendocrine reactivity, implying a more pronounced and prolonged neuroendocrine stress reaction. In contrast, coping styles including active, direct, problem-focused strategies have been associated with reduced neuroendocrine reactivity. For example, subjects with high levels of active or instrumental mastery-oriented coping show little stress response, both on SNS and the HPA axis (Bonanno et al., 2003; Levine and Ursin, 1991). Also, palliative coping such as use of alcohol or other sedatives has been shown to reduce physiological arousal. Additionally, coping including positive emotions produces faster return to baseline levels of cardiovascular activation following negative emotional arousal and prompts toward a flexible use of a wider range of coping strategies (Fredrickson et al., 2000, 2003).

In particular regarding defensive coping, the *effectiveness of defenses* in warding of negative emotions may be crucial for the neuroendocrine reactivity in stressful situations. The effective use of defense mechanisms may dampen the endocrine stress response: individuals using effective defense strategies protect themselves by not getting overwhelmed by negative affect which allows them to maintain an adequate level of functioning. They show lower cortisol levels (Olff et al., 1995; Brown et al., 1996; see Vickers, 1988) which on the short term may be adaptive. However, the reduced cortisol response in defensive subjects has been found to be associated with high noradrenaline levels as well as other signs of SNS activation (heart rate, blood pressure) (Olff et al., 1995), indicating that cortisol may not shut down the sympathetic activation. The resulting sustained activation may—on the long term—be associated with more health problems (Ursin and Olff, 1993).

In trauma research, an association between repressive coping and affective-autonomic response discrepancy (low negative affect and elevated autonomic reactivity) has been reported, namely among females with documented histories of childhood sexual abuse (Bonanno et al., 2003). Furthermore, among PTSD inpatients an association has been reported between lower cortisol levels and higher effectiveness of disengagement coping mechanisms, particularly emotional numbing and avoidance (Mason et al., 2001). In this latter study, marked individual differences and phasic alterations in cortisol levels in PTSD were observed. Results leading the authors to suggest that cortisol levels reflect the balance between undifferentiated emotional arousal (engagement), associated with higher cortisol levels, and disengagement defense mechanisms, associated with lower levels. If this holds true, individuals with PTSD may appear relatively low or high on cortisol levels depending on this balance, supporting the concept of defensive coping as an organizing principle in the interpretation of the inconsistent cortisol findings in PTSD patients (Mason et al., 2002). Other supportive findings for this concept have been reported in accident-related PTSD, namely that greater emotional numbing levels predicted lower cortisol levels at 6-months follow-up after the accident (Hawk et al., 2000).

However, the validity of coping styles obtained in questionnaires has been questioned (Stone et al., 1998). Care should be taken in drawing conclusions based on self-report measures of defensive coping. Part of defensive behaviors show overlap with symptoms of the avoidance cluster of PTSD. It is, therefore, not surprising that PTSD is associated with avoidant coping (Ginzburg et al., 2002). Additionally, as suggested by Olff (1991, 1999), the self-reporting of distress may be contaminated by psychological defense mechanisms which affect the recognition and evaluation of symptoms. Recent trauma studies have looked at these defense mechanisms in interpreting discordance between physiological and self-report measures in individuals with PTSD (Orr and Roth, 2000).

Stress research has shown a role of *seeking social support* (as part of emotion-focused coping) in modulating the (psycho)endocrine reaction to acute stress (Biondi et al., 1986; Kirschbaum et al., 1995). For example, effective social support seeking, i.e. availability of and satisfaction with social support may reduce neuroendocrine reactivity as it is associated with lower heart rate and blood pressure, lower catecholamine levels, use of more coping strategies, increase in positive affect, and better immune functioning (Biondi and Picardi, 1999; Uchino et al., 1996). Specifically, social support and oxytocin, a neurohormone implicated in prosocial behavior and in the nervous control of neuroendocrine responses to stress, seem to interact to suppress cortisol and subjective responses to psychosocial stress (Heinrichs et al., 2003). In fact, the stress literature emphasizes a key role of oxytocin as an underlying biological mechanism for stress-protective effects of positive social interactions (Uvnas-Moberg, 1998; Neumann, 2002; Charney, 2004). If this also holds true for traumatic stress it could explain the protective effect of social support for developing PTSD. We have found no studies examining links between social support and neuroendocrine responses in a context of traumatic stress.

In the traumatic stress literature a few other studies were found examining associations between coping and physiological parameters. First, relations were found between exposure to extreme stress (i.e. the nuclear power plant disaster at Three Mile Island) and urinary cortisol, psychological and physical symptoms and urinary cortisol, but not between coping styles and urinary cortisol (Schaeffer and Baum, 1984). Second, greater coping self-efficacy has been associated with lower norepinephrine to

cortisol ratios in HIV-infected gay men following Hurrican Andrew (Benight et al., 1997), findings concurring with those found among other traumatic stress survivors pointing to the importance of coping self-efficacy in moderating psychological stress responses (Murphy, 1987; Solomon et al., 1991). And third, recent preliminary data among individuals who were in or near the World Trade Center towers at the time of the September 11th attacks suggest that the personality trait of self-enhancement is associated with both active coping styles (especially regarding social networks) and a profile of salivary cortisol levels suggestive of a resilient neuroendocrine response (Bonanno, 2004). Evidently, these latter indications of more adaptive responses to trauma do not characterize the neurobiological adaptation of individuals who develop PTSD.

6. A conceptual model

Evidence does suggest that acute extreme stress and chronic severe stress are associated with the onset of stress-related disorders, possibly through their effects on neuro-endocrine stress systems. More generally, whether a particular response to extreme threats is adaptive or maladaptive is a function of numerous factors of which the environment of the individual is of great importance. Normal adaptive responses to extreme stress can under certain psycho-sociocultural conditions become maladaptive and result in a pathological stress response (McEwen, 2002). The literature clearly indicates that appraisal and coping processes are important (co)determinants of the psychological and physiological stress response. Fig. 2 integrates the reviewed empirical findings on the pathways of appraisal and coping processes to neuroendocrine response and to PTSD. The model builds on the previous one explaining the psychobiology of stress-coping (Fig. 1, Olff, 1999). Presently, the focus is on the crucial mediating role of how a person appraises the traumatic event, what coping strategies they use and how effective these strategies are in preventing the development of posttrauma psycho-pathology, particularly PTSD.

So far, the available evidence from animal and human studies confirms the idea that coping capacity or style helps to determine individual vulnerability for stress-related disease (Koolhaas et al., 1999). And, that one may expect different types of stress-pathology to develop under conditions in which a particular coping style fails. This, because different coping styles are based on differential use of various physiological and neuroendocrine mechanisms. For example, anatomical studies show that different neural circuits mediate active and passive emotional coping (Keay and Bandler, 2001). Research also has made clear that coping style should not be regarded as a rigid characteristic. Rather, flexibility in the use of coping styles or strategies is of crucial importance in understanding health and disease. Factors that have been shown to affect the individual's coping capacity include for example, genotype, development, early experience and social support (Koolhaas et al., 1999).

As depicted in this model, personal, social, and cultural factors as for example, age and gender, genetically increased sensitivity to stress, personality traits, disturbance in early attachment relations, previous trauma, and social support do influence the proposed pathways. The same applies to the type of coping, (perceived) effectiveness of the coping strategy used, the flexibility in the use of different coping strategies, and the possibility to seek and use social support. It is known from human studies that self-esteem, coping efficacy, and ability to maintain positive social relationships may favor adaptation to traumatic experiences (Ozer et al., 2003). On the other hand, it is suggested that

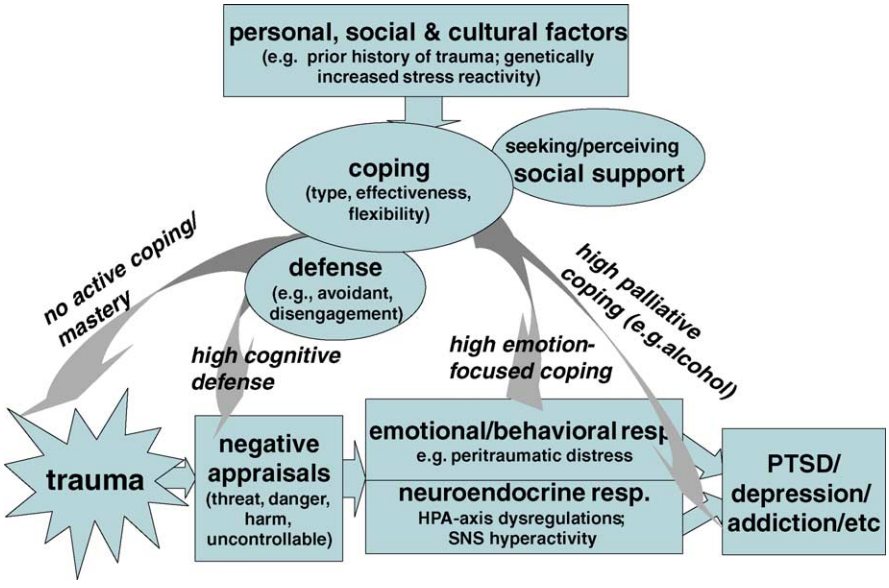

Fig. 2. Traumatic stress-coping model.

those who may fair least successful in terms of risk for developing stress-related psychopathology, may be using coping strategies that interfere with effective coping, such as denial, and avoidance, or may overemploy psychological defense mechanisms such as behavioral disengagement. The question of whether certain coping strategies or defenses are maladaptive can only be answered by considering the context, both external and internal, in which they occur and requires a consideration of the time frame. For example, in the short run, especially when few options are available, defenses may provide the highest level of adaptation possible. However, in the long run, especially if they should interfere with problem-focused coping, defenses are likely to hinder successful adaptation (Pennebaker, 1993). In addition, coping strategies that generate positive affect such as positive reappraisal or coping efforts focusing on the creation of situational meaning in an ongoing (extreme) stressful context, may minimize the negative consequences of traumatic stress and may promote psychological well-being (Janoff-Bulman, 1992; see also Aldwin and Yancura, 2004). In a similar vein, studies concerning depressive disorders have demonstrated that the phenomenon to retain the capacity to engage in meaning-based coping and experiencing positive affect may be critical in understanding how people manage to minimize distress and produce positive outcomes (Folkman and Moskowitz, 2000).

More specifically, the model proposes that cognitive appraisal, the subjective interpretation of the trauma, is crucial in starting the cascade of psychobiological responses to trauma. Virtually by definition a traumatic event is evaluated as a threat. According to DSM IV, traumatic stress is defined as events that involve serious threat to life or physical integrity, either to oneself or significant others. As described earlier, diminished threat appraisal as for instance, through alcohol use just before or during a traumatic event may give rise to attenuated peritraumatic physiological arousal reactions and PTSD. DSM also defines the emotional response to the traumatic event, namely: intense fear, helplessness or horror (American Psychiatric Association, 2001). Extreme acute peritraumatic emotional reactions or peritraumatic distress and accompanying physiological reactions have been shown to be predictive of PTSD (Bryant, 2003; Shalev et al., 1998a,b, 2000). More particularly, the model assumes that effective defensive coping may protect individuals from being overwhelmed and may buffer their HPA acute stress response. Also based on the literature (Olff et al., 1995) a premorbid general defensive coping style is associated with non-adaptive coping with trauma, resulting in short-term relief but not being able to mount an adequate cortisol response it may lead to long-term sustained activation of sympathetic arousal. At the end of the cascade, long-term psychobiological dysregulation gives rise to posttrauma psychopathology.

Alternatively, active coping may help individuals to deal with the traumatic stressor, avoiding long term physiological dysregulation and posttrauma symptoms. A rather consistent pattern of individual characteristics associated with successful adaptation to extreme stress has been reported (Charney, 2004), including an active coping style in confronting a stressor, optimism, effective self-regulation of emotions and attachment behaviors, good intellectual functioning, a positive self-concept, altruism, and a capacity to convert traumatic helplessness into learned helplessness. Of interest in this regard are current neurobiological models of acute stress response, implicating the amygdala and hippocampus as key brain areas that are involved in the registration of potentially dangerous situations and in the latter formation of the memories of such events (LeDoux, 2000). In these brain models the HPA axis is given a central role in both the development of PTSD and its maintenance. Additionally, even more interesting with regard to the current paper, this research based on animal models suggests that it is possible to turn maladaptive responses into more adaptive ones by introducing an active coping response (LeDoux, 2000; LeDoux and Gorman, 2001). Laboratory experiments have shown that the pathways leading from the central nucleus of the amygdala to the brainstem that initiate defensive freezing and associated autonomic and endocrine reactions can be redirected. This 'rerouting' in brain circuits—from a pathway controlling dysfunctional passivity to one controlling successful engagement with the environment—proposes as possible effective psychosocial interventions to improve individuals' coping skills, including those that can be used to generate or sustain positive affect in the context of traumatic stress (LeDoux and Gorman, 2001). Building optimism, for example, prevents clinical disorder of depression (Seligman and Csikszentmihalyi, 2000). It may prove to be effective for preventing PTSD as well.

In sum, the conceptual model proposes that a threat appraisal of a traumatic event or situation is followed by an acute stress response that involves both emotional, behavioral and biological components. A failure to regulate the biological stress response at the time of trauma may result in a cascade of psychobiological alterations that lead to intrusive recollections of the event, avoidance of reminders of the event, and symptoms of hyperarousal. Still very little is known about why in some individuals the acute psychobiological dysregulation leads to PTSD while in others it gives rise to depression, substance abuse, other anxiety disorders, somatic disorders or combinations of disorders. There is some evidence that the particular outcome is the result of individual differences in defense and coping strategies representing important targets for psychotherapy (Gersons et al., 2000). Finally, the conceptual model encourages several lines of new research, which we believe can further increase understanding of the role of coping in adaptive processes.

7. Future research directions

First of all, it should be mentioned that research findings on coping are difficult to draw together due to the wide variety of different operational definitions of coping that have been employed (Skinner et al., 2003). Consensus about how to conceptualize or measure ways of coping would be very helpful in future research concerning links between coping to adaptive processes. Furthermore, trauma research should include measures of new facets of the coping process being discussed in the broader stress literature such as positive affect and positive reappraisal for further exploring the adaptational significance of these variables (Folkman and Moskowitz, 2000). In addition, future psychoneuroendocrine research on extreme stress should include evaluations of effective coping/coping efficacy (Lazarus, 2000). It may well be that the appraisal of one's perceived capability to manage environmental demands is far more important than the coping strategy itself for understanding the psychopathology of stress-related disorders (Ursin and Olff, 1993; Ehlers et al., 1998) (see also a recent paper on a two-dimensional defense system, noting 'defensive distance'—i.e. intensity of perceived threat—as a key factor in stress response systems; McNaughton and Corr, 2004). Further insight into how coping strategies shifted over time is highly relevant. Moreover, prospective, longitudinal studies of stress or trauma in high risk populations are vital to the understanding of the influence of pretrauma (biological) variables in pathological trauma responses. Such studies are also important to determine why a stressor increases the vulnerability to a particular pathology in one individual (e.g. PTSD) but a different pathology in another individual (e.g. panic disorder, major depression, alcoholism) (Ehlert et al., 2001; Yehuda et al., 2004). Research as such can provide us with information regarding clinical interventions.

Over the past several decades, rapid growth in the knowledge of neuroendocrinology and the involvement of neuroendocrine factors in normal brain functioning as well as in psychiatric illness has opened many exciting new lines of inquiry. The challenge that lies ahead is to further expand and refine our knowledge of the neurobiology of stress related disorders, particularly posttraumatic stress disorder. Although stress research has clearly demonstrated that containment of a variety of physiological responses to stress is a key feature of (un)successful adaptation, much remains to be learned about the pathways from perceived stress to the neurochemical and neuroanatomical features of posttraumatic stress disorder in adults (McNally, 2003; LeDoux and Gorman, 2001; McEwen, 2003). In addition, it has been suggested that resiliency may also be manifest in the individual differences in the functioning of the HPA axis (Ozer et al., 2003). Identification of neurochemical profiles that characterize psychobiological resilience are of importance due to their predictive value regarding successful adaptation to extreme stress (Charney, 2004; Curtis and Cicchetti, 2003). Comprehensive understanding of the neurobiology of resilience or stress-resistance may ultimately provide a foundation for new approaches to the successful treatment and prevention of stress-induced psychiatric and physical disorders. Besides, although regulation of the HPA axis could be a target for pharmacological treatment of PTSD, this area has only recently been targeted in research (Aerni et al., 2004).

Extensive animal and human research demonstrates consistent gender differences in stress responsiveness (Rasmusson and Friedman, 2002). For example, one of the most consistent findings in the epidemiology of PTSD is women's greater rates of this disorder (Kessler et al., 1995; Breslau et al., 1997). The reasons for the gender difference remain unclear. Several possible explanations have been suggested for the higher liability of women to develop PTSD such as, gender-specific exposure to various forms of traumatic events, which themselves have different conditional risk for the development of PTSD, and gender-specific psychological and (neuro)biological reactions to trauma. It has been hypothesized that sex differences in physiological stress reactivity under both acute and chronic conditions may be one mechanism underlying gender differences in PTSD (Rasmusson and Friedman, 2002). As our paper shows, the effects of appraisal and coping on psychobiological outcomes have rarely been established in the context of traumatic stress, let alone gender differences in these associations. Based on the available information that appraisal and coping processes are key (co)determinants of the psychological and physiological stress response, and of gender-related differences in neuroendocrine profiles related to coping processes (Peirce et al., 2002), future psychoneuroendocrine research on extreme stress should include measures of the concepts of appraisal and coping (including defensive coping) as well as examine gender differences in response patterns. Clearly, gender-specific patterns of appraisal and coping strategies may moderate the relation of the HPA axis reactivity to extreme stress and contribute to differential adaptive and maladaptive psychobiological responses to extreme stress.

8. Concluding remarks

Acknowledging the important implication of appraisal and coping for interventions designed to alleviate posttraumatic stress (Saakvine et al., 1998; Ehlers and Clark, 2000), because these variables are more amenable to change than neuroendocrine response, further research among trauma victims focusing on these variables is warranted. That cognitive appraisal and coping may represent important targets for trauma treatments, has been indicated by a randomized controlled study of effects of cognitive behavioral stress management on cortisol responses to acute stress in healthy individuals (Gaab et al., 2003) as well as by a recently performed study on the effects of psychotherapy on cortisol levels in PTSD patients (Olff et

al., 2004). More generally the success of targeting appraisal and coping has been shown in studies on cognitive behavioral treatment in preventing (chronic) PTSD (Bryant et al., 1999, 2003; Ehlers et al., 2003) or treating PTSD (Gersons et al., 2000). Also, combinations of psychological and pharmacological interventions may help individuals to turn maladaptive responses into more adaptive ones (Christopher, 2004; Bonne et al., 2004).

Perhaps the primary advantage of the presented theoretical framework lies in its heuristic value, connecting multiple fields of inquiry and as such it may serve to promote further programmatic research. Clearly, additional research is needed examining the associations between appraisal and coping and the neuroendocrine response to further our understanding of the process through which PTSD and other psychopathologies develop. Finally, the conceptual model encourages research efforts focusing on the role of coping in adaptive responses to trauma.

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