

EFFECTS OF TRAUMA REMINDERS ON MEMORY AND ATTENTION
PROCESSES: THE ROLE OF ASSOCIATED EMOTIONAL DISTRESS AND
UNSUPPORTIVE SOCIAL INTERACTIONS

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by

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Abstract

Stressful life events and traumatic experiences have been associated with adverse psychological outcomes. Beyond their pronounced immediate effects, stressors may proactively influence physiological and emotional responses to subsequently encountered stressors (sensitization). These proactive stressor effects appear to extend to cognitive functioning and may be particularly prominent in situations reminiscent of the initial stressor experience. The present investigation examined the influence of trauma reminders, and the ensuing emotional responses, on memory and attention functioning. As coping methods and social support (or conversely ‘unsupport’) may influence the distress individuals experienced, particular consideration was given to the potential moderating effects of unsupportive social interactions individuals encountered during their most distressful experience. In Study 1 ($n = 65$) exposure to trauma images and previous trauma altered memory performance and these effects were most prominent among traumatized individuals that were exposed to trauma related stimuli. These variations were less pronounced in Study 2 ($n = 157$), but decreased memory functioning was associated with increased distress and cortisol reactivity following trauma reminders. Study 3 ($n = 195$) indicated that traumatized individuals having more negative social interactions during their most distressing experience, exhibited greater attention bias toward trauma-related material than traumatized individuals without such social interactions. These findings highlight the dynamic effects of trauma reminders on cognitive processes. Additional studies with clinical populations are required to obtain a more detailed understanding of the processes involved in the relationships between trauma experiences and cognitive functioning.

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EFFECTS OF TRAUMA REMINDERS ON MEMORY AND ATTENTION
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Stressful life events and traumatic experiences, such as war, death of a loved one, car accidents or natural disasters have been implicated in the evolution of numerous physiological and psychopathological outcomes, such as cardiovascular disease, depression, anxiety and posttraumatic stress disorder (PTSD) (Dickinson, deGruy, Dickinson, & Candib, 1999; Leserman et al., 2005; McEwen, 1998, 2003; Raber, 1998; Yehuda, 2002). Additionally, such extreme experiences may induce cognitive alterations, particularly with respect to memory and attention processes (Golier & Yehuda, 2002; Johnsen & Asjornsen, 2008; Stein, Kennedy, & Twanley, 2002; Vedhara, Hyde, Gilchrist, Tytherleigh, & Plummer, 2000).

Intrusive memories (flashbacks) as well as partial forgetfulness (poor intentional recall) of the traumatic experience are prevailing features of memory functioning in traumatized individuals (APA, 1994). Intrusive memories, such as those reported in PTSD, include repetitive and intrusive recollections of the traumatic event, which may be experienced in a vivid and very emotional re-enactment of the event. Generally, involuntary memories are triggered by aspects of situations that are perceptually similar to those surrounding the initial trauma encounter (i.e., present shortly before or during the trauma). Physical cues, (e.g., light, smells, spatial cues), emotional states (e.g., feeling helpless) and internal cues (e.g., being touched on a particular body part) may trigger such memories and are thought to act as reminders of the original traumatic event (Brewin & Andrews, 1998; Brewin, Andrews & Rose, 2000; Brewin & Holmes, 2003).

Trauma-related stimuli appear to selectively bias memory and attention processes and elicit psychological and physiological reactivity in several stress-related disorders (Amir, Kaplan & Kotler, 1996; Bryant & Harvey, 1995, 1997; Coles & Heimberg, 2002; Dougall, Craig, & Baum, 1999; Ehlers, Hackmann, & Michael, 2004; Kaspi, McNally & Amir, 1995; Mathews & MacLeod, 2005; Squire & Zola-Morgan, 1991; Van Oyen Witvliet, 1997). In effect, individuals often display pronounced attention toward stimuli associated with trauma, and exhibit strong memories related to these events. In contrast to this apparently sensitized response to these reminder stimuli, it seems that in other respects memory functioning may be disrupted (APA, 1994), possibly owing to diminished encoding or impaired retrieval abilities. However, little is known about whether trauma reminders interfere with memory processes. Not surprisingly, perhaps, the bulk of research has focused on memory disturbances that might be present among individuals presenting with PTSD symptoms, and much less information is available concerning the impact of stressors and traumatic experiences among those individuals that are not afflicted with PTSD. Yet, there is no reason to assume that traumatic experiences would not affect memory performance in these individuals.

Given the very large number of individuals that encounter trauma without PTSD emerging (or, for that matter, PTSD remaining undiagnosed), it has become particularly relevant to explore the influence of trauma reminders, and the ensuing emotional responses, on memory and attention functioning. One objective of the present research was to determine whether trauma images would affect memory processes (i.e. disrupted or facilitated), and to determine whether such performance alterations would be related to

previous traumatic experiences and the emotional arousal associated with such reminders (Study 1).

The processes by which stressful events, including traumatic experiences and their reminders, might influence memory and other cognitive processes have not been fully elucidated, but several cogent propositions have been advanced. Among these, particular consideration was given to glucocorticoid (GC) release, particularly cortisol, resulting from the stressor-provoked activation of hypothalamic-pituitary-adrenal (HPA) functioning (Lupien & Lepage, 2001). Numerous factors justify the focus on GCs to explain the influence of stressful events on cognition: (a) their release may be related to emotional arousal (Sachar, Hellman, Roffwarg, Halpern, Fukushima, & Gallagher, 1973), (b) they have been implicated in stress-related psychopathologies, such as depression and PTSD (Claes, 2004; Hatzinger, 2000; Holsboer, 2000; Meewisse, Reitsma, de Vries, Gersons, & Olf, 2007; Parker, Schatzberg, & Lyons, 2003; Rasmusson, Vythilingam, & Morgan 2003; Seckl & Meaney, 2006; Southwick, Vythilingam, & Charney, 2005; Steckler, Holsboer, & Reul, 1999; Yehuda, 2002, 2006), (c) in animal and human studies, GCs have been shown to modify cognitive processes, especially memory (Alderson & Novack, 2002; Heffelfinger & Newcomer, 2001; Het, Ramlow & Wolf, 2005; Lupien, Maheu, Tu, Fiocco & Schramek, 2007; Wolf, 2003), and (d) they have been known to affect structure and/or functioning of the hippocampus and the prefrontal cortex (PFC), which have been implicated in these cognitive functions (Aronsson, Fuxe, Dong et al., 1988; Diorio, Viau & Meaney, 1993; Jacobson & Sapolsky, 1991; Lupien & Lepage, 2001; McEwen, 2001, 2002; McEwen, De Kloet & Rostene, 1986; Nelson & Carver, 1998; Reul & De Kloet, 1986 ; Van Eekelen, Jiang, De Kloet, & Bohn, 1988).

Accordingly, a second objective of the present research was to determine whether trauma reminders provoked cortisol release, and to determine whether cortisol mediated the relation between the response to trauma-related images and memory processes as well as between trauma history and memory processes (Study 2).

Obviously, not all individuals that encounter stressors, even those of a traumatic nature, exhibit cognitive disturbances, nor is it the case that the development of cognitive disturbances is necessarily tied to stressful experiences. Interestingly, there appear to be a constellation of factors that increase vulnerability to psychopathology, and conversely resilience factors exist that may protect or buffer the individual from the adverse effects of stressors (Anisman & Matheson, 2005). Among the variables implicated as moderating factors for psychopathology, social support is thought to be particularly pertinent. Inasmuch as social support contributed to stressor responses in other psychological domains, and with respect to cortisol release (Dickerson & Kemeny, 2004; Michaud, Matheson & Anisman, 2008), it is possible that these factors might also be involved in the vulnerability to cognitive impairments following stressor exposure. Thus, the third objective of this research was to evaluate the moderating influence of social support, particularly unsupportive social interactions during the trauma experience, on trauma-related memory disruptions. Further, given that traumatic experiences may impact attention processes (Bryant & Harvey, 1995, 1997; Paunovic, Lundh, & Ost, 2002; Standford, Vasterling, Mathias, Constans, & Houston, 2001; Vythilingam, Blair, & McCaffrey, 2007), by affecting performance and the processing of subsequently encountered stimuli (Amir, Taylor, Bomyea, Badour, 2009; Chemtob, Roitblat, Hamada, Muraoka, Carlson, & Bauer, 1999), another objective of this study was to examine the

contribution of unsupportive social interactions on subsequent attention processes (Study 3).

Trauma and Stressors: Immediate Physiological Effects

Obviously, not all individuals who encounter stressors respond in a uniform fashion nor do all stressors elicit comparable outcomes. Further, although many types of stressors may lead to similar effects, there may also be distinct differences in the outcomes elicited and the specific mechanisms that govern these processes (for a more detailed description, see Michaud et al., 2008 in Appendix P).

Ordinarily, stressors give rise to a stress response that encompasses a cascade of adaptive behavioral and physiological reactions that alter the body's internal milieu (allostasis). Generally, under stressful circumstances certain behaviors, notably those associated with arousal, vigilance, and coping responses, predominate, whereas behaviors that are not productive in a defensive capacity (e.g., sexual and feeding behaviors) may be suppressed. Concurrently, several neurochemical changes are elicited in a variety of stressor-sensitive brain regions, including various amygdala nuclei, medial prefrontal cortex, locus coeruleus (Valentino & Van Bockstaele, 2008), hippocampus, and hypothalamic nuclei; presumably in order for the individual to deal effectively with the ongoing challenge and to restore homeostasis (McEwen, 2006, 2007).

Ultimately, stressors may activate the hypothalamic-pituitary-adrenal (HPA) axis, which is considered to be a primary mechanism for maintaining homeostasis in response to stressors. This section only provides a general overview of the stressor-provoked HPA variations and readers more inclined toward a more detailed description of the biological/neural circuitry involved in the stress response may consult Appendix A.

Essentially, HPA axis activation involves the stimulation of the medial parvocellular paraventricular nucleus (PVN) of the hypothalamus, giving rise to the release of corticotropin releasing hormone (CRH) and arginine vasopressin (AVP) from terminals located at the median eminence, causing the release of adrenocorticotrophic hormone (ACTH) from the anterior pituitary (Antoni, 1986; Whitnall, 1993). This, in turn, stimulates the release of glucocorticoids (GCs), such as cortisol, from the adrenal cortex. Once released, cortisol enters circulation and finds its way to the brain where it stimulates specific corticoid receptors on the hippocampus, which then cause the termination of the hypothalamic CRH activity (Antoni, 1986; Chrousos & Gold, 1992; Herman & Cullinan, 1997; Sapolsky, Romero & Muck, 2000; Whitnall, 1993). Although these responses are thought to be of adaptive value, sustained activation of these processes may cause excessive wear and tear on biological systems (allostatic overload), thereby increasing vulnerability to various pathological outcomes (McEwen, 1998, 2000, 2004, 2006, 2007; McEwen & Wingfield, 2003; Miller & O'Callaghan, 2002).

Stressors & Traumatic Experiences: Physiological, Psychological and Cognitive Outcomes

As mentioned earlier, ongoing stressors as well as previous stressful and/or traumatic experiences may influence physiological and psychological well-being (for a review see McEwen, 1998, 2000, 2004, 2006, 2007; Schneiderman, Ironson, & Siegel, 2005; Wolkowitz, Epel, & Reus, 2001). Among other things, stressors may be associated with immune disturbances (Glaser & Kielcolt-Glaser, 2005; Kielcolt-Glaser, McGuire, Robles & Glaser, 2002), cardiovascular disease (Bunker, Colquhoun, Esler et al., 2003; Tennant, 1999; von Känel, Mills Fainman & Dimsdale, 2001), depressive illness

(Abramson, Seligman & Teasdale, 1978; Bifulco, Bernazzani, Moran & Ball, 2000; Billings & Moss, 1982, 1985; Brown, Harris & Eales, 1996; Cui & Vaillant, 1996; Daley, Hammen & Rao, 2000; Dura, Stukenberg & Kiecolt-Glaser, 1990; Griffith, Ravindran, Merali & Anisman, 2000; Hammen, Davila & Brown, 1992; Hammen, Mayol & deMayo, 1986; Kessler, 1997; Monroe et al., 1983, 1992; Monroe & Simons, 1991; Mundt, Reck, Backenstrass et al., 2000; Paykel, 2001), post-traumatic stress disorder (PTSD) (Dickinson et al., 1999; Yehuda, 2001, 2002), drug addiction (Cleck & Blendy, 2008; Goeders, 2003), as well as neurodegenerative disorders (Anisman, Merali & Hayley, 2008). In addition, there are indications that stressful experiences may also induce long-lasting cognitive disturbances (Cohen, Kamarck & Mermelstein, 1983; Lupien, Gaudreau, Tchiteya et al., 1997; Orem, Petrac, & Bedwell, 2007; Pluck, Lee, David, Macleod, Spence & Sparks, 2010; Wilson, Bennet, Mendes, de Leon, Bienias, Morris and Evans, 2005;), such as those that may be evident in stress-related psychiatric disorders, including depression (Burt, Zembar & Niederehe, 1995; Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari, Lönnqvist, 2008), dissociative disorders (Amrhein, Hengmith, Maragos, Hennig-Fast, 2008; Dorahy, 2001) and post-traumatic stress disorder (PTSD; see section below).

PTSD and Cognitive Functioning

Although the present investigation was primarily concerned with the influence of previous traumatic experiences and their reminders on subsequent cognitive processes (not necessarily on individuals suffering from PTSD), most of the findings regarding the impact of trauma experiences on cognition have emerged from studies conducted with individuals suffering from PTSD (for a review, see Moore, 2009). As PTSD-related

findings may have implications for cognitive disturbances on individuals not necessarily suffering from this disorder, it may be important to consider the findings derived from individuals presenting PTSD symptoms.

Most studies have suggested that individuals presenting with PTSD symptoms experience impaired abilities in tests of attention (Gilbertson, Gurvits, Lasko & Pitman, 1997; Gil, Calev, Greenberg, Kugelmass, & Lerer, 1990; Jenkins, Langlais, Delis, & Cohen, 2000; Sachinvala, von Scotti, McGuire, et al. 2000; Uddo, Vasterling, Brailey & Sutker, 1993; Vasterling, et al. 1998, 2002), learning, memory (Berendt & Moritz, 2005; Bremner, Scott, Delaney et al., 1993; Bremner, Randall, Scott, et al. 1995a; Elzinga & Bremner, 2002; Gil et al., 1990; Gilbertson, Gurvits, Lasko, Orr, & Pitman, 2001; Jenkins, Langlais, Delis, & Cohen, 1998; Vasterling et al. 1998, 2000; Uddo et al., 1993; Yehuda et al., 1995) and executive functioning (Beckham, Crawford & Feldman, 1998; Gil et al., 1990; Sutker, Winstead, Galina, & Allain 1991). However, there have also been studies that revealed few or no impairments in these domains among traumatized individuals (Barrett, Green, Morris, Giles & Croft, 1996; Crowell, Keiffer, Siders, & Vanderploeg, 2002; Dalton, Pederson & Ryan, 1989; Golier et al. 1997; Gurvits et al., 1993; Stein, Hanna, Vaerum, & Koverola, 1999; Zalewski, Thomson & Gottesman, 1994). In part, these discrepancies might be due to difference in methodology (i.e. use of different neuropsychological batteries or tasks to measure cognitive functioning) or the nature of the sample used in these studies (i.e., most of these studies were conducted with individuals suffering from combat-related PTSD). This notwithstanding, the conflicting results have led to the obvious question of whether impairments of cognitive functioning following trauma exposure actually existed, and if so, whether these disturbances

reflected (a) the effect of trauma exposure causing hippocampal neuronal death (Sapolsky, Krey, & McEwen, 1984), (b) psychiatric comorbidities, such as depression (Barret et al., 1996; Moore, 2009) or substance abuse (Stein, Höfler, Perkonigg, et al., 2002) or (c) a pre-morbid condition that increased the risk of developing PTSD. In this regard, twin studies suggested that the smaller hippocampal size (Bremner & Narayan, 1998; Bremner, Randall, Scott et al., 1995b; Gurvits, Shenton, Hokama et al., 1996; Stein, Koverola, Hanna, Torchia, & McClarty, 1997), lower intelligence quotient and/or cognitive impairments associated with PTSD may constitute a premorbid risk factor for PTSD rather than being the result of trauma exposure (Buckley, Blanchard, & Neill, 2000; Gilbertson, Shenton, Ciszewski et al., 2002; Gilbertson Paulus, Wiliston et al., 2006; Gurvits, Gilbertson, Lasko, et al., 2000; Macklin, Metzger, Litz et al., 1998; McNally & Shin, 1995; Moore, 2008; Pitman, Gilbertson, Gurvits et al., 2006; Vasterling et al., 1997; Yehuda, 2001, 2005).

As mentioned earlier, the picture regarding trauma effects on cognitive processes is complicated by the fact that most of these findings emerge from combat-related PTSD. Although there is some indication that some of these neuropsychological changes are likely present in other populations exposed to trauma (Hart, Gunnar & Cichetti, 1996; Samuelson, Krueger, Burnett & Wilson, 2009; Stein, Kennedy & Twamley, 2002), the extent to which these findings are generalizable to other populations and traumas remains uncertain. Given that these clinical samples may differ in important ways (training, anticipation of adverse events) from the general population or sub-clinical populations exposed to traumatic events, a study conducted with a “more general” population like the present study may be timely.

Stressors and Traumatic Experiences: Proactive Effects

In addition to their immediate effects, there is a substantial body of evidence that antecedent stressors may proactively influence the neurobiology of the stress response, particularly with respect to subsequently encountered stressors (Anisman, Zaharia, Meaney & Merali, 1998; Plotsky & Meaney, 1993; Post, 1992; Sapolsky, 1997; Tilders & Schmidt, 1998) and might thus come to affect mood states. It has been suggested that this outcome may result from the sensitization of neuronal processes, so that re-exposure to the same stressor (and even to alternate stressors) at a later time results in neurochemical changes occurring more readily (Anisman et al., 1998). Such sensitization effects have been documented with respect to numerous neurochemical systems, including monoamines, cytokines, CRH and GCs; all of which have been implicated in stress-related pathologies such as depression as well as PTSD (Anisman, 2009; Anisman, Merali & Hayley, 2003; Nemeroff, 1996; Tilders & Schmidt, 1999; Yehuda, 2002) and cognition (Bennet, Ballard, Watson & Fone, 1997; Chamberlain, Müller, Robbins & Sahakian, 2006; Heffelfinger & Newcomer, 2001; Martignoni, Costa, Sinforiani et al., 1992; McAfoose & Baune, 2009; Robbins & Roberts, 2007).

In line with these findings, the proactive effects of stressors are particularly notable among individuals who encountered traumatic experiences in that such individuals exhibited greater psychological and physiological reactivity in response to later stressors (Butler, Braff, Rausch et al. 1990; Elsesser, Sartory, & Tackenberg, 2004; Miller & Litz, 2004; Morgan et al., 1996, 1997) as well as memory and attention biases regarding trauma-related cues (Bryant & Harvey, 1995, 1997; Cassiday, McNally & Zeitlin, 1992; Coles & Heimberg, 2002; Foa, Feske, Murdock et al. 1991; Freeman &

Beck, 2000; Kaspi, McNally & Amir, 1995; McNally, 1997; McNally, Amir & Lipke, 1996; McNally, English, & Lipke, 1993; McNally, Kaspi, Riemann, & Zeitlin, 1990; Moradi, Taghavi, Neshat-Doost, Yule, & Dalgleish, 2000; Paunovic, Lundh & Ost, 2003; Squire & Zola-Morgan, 1991; Thrasher, Dalgleish & Yule, 1994; Vrana, Roodman & Beckman, 1995; Vythilingam et al., 2007; Williams et al., 1996; Zeitlin & McNally, 1991).

Most studies examining attention biases in traumatized individuals involved testing PTSD-affected individuals in the Emotional Stroop test (William et al., 1996). This task assesses the interfering effects of trauma on a concurrently performed non-emotional task by requesting participants to name the color of trauma-related and trauma-unrelated words. A substantial body of literature shows that individuals suffering from PTSD take longer to name words related to their previous trauma experience (i.e., interference effect), but not to words unrelated to their trauma (Beck, Freeman, Shiperd, Hamblen & Lackner, 2001; Bryant & Harvey, 1995, 1996; Cassiday et al., 1992; Foa et al., 1991; Harvey, Bryan & Rapee, 1996; Kaspi et al., 1995; McNally et al., 1990; Moradi et al., 1999; Trasher, Dalgleish, Yule, 1994). Although, these studies suggest that such interference might be specific to the disorder and not the experience itself, other studies have yielded mixed results. For instance, longer time to color name trauma related words naming was found in sexual assault but not physical abuse (Dubner & Motta, 1999) or was present in both trauma words and generally threatening words (Litz et al., 1996). Others have not found delayed color naming of trauma words associated with PTSD (Bremner et al., 2004; Devineni, Blanchard, Hickling & Buckley, 2004; Freeman & Beck, 2000; Naidich & Motta, 2000; Suozzi & Motta, 2004; Shin et al., 2001).

The Visual Probe task is another commonly used task to examine the relationships between attention bias for threat and anxiety, although not specifically PTSD (Bradley et al., 1999; Mogg & Bradley, 1999; 2002). In general, face pairs, or other evocative photographs, are used in this task; participants are presented with two facial expressions: one neutral and the other either angry/threatening or happy. Participants are required to indicate the position of a probe presented immediately after the face pair, thereby allowing for determination of the individuals attention orientation (Bradley et al., 1999; Mogg & Bradley, 1998, 1999, 2000; Mogg, Bradley, Hyare, & Lee, 1998; Mogg, Millar & Bradley, 2000;). Although this paradigm is used extensively in cognitive neuroscience to examine the relationships attention bias for threat and anxiety, no study specific to PTSD was found, nor did existing studies employ other types of images to quantify the effects of trauma material on attention. However, consistent with the idea of an attention bias toward threat, when using the Visual Probe task it was found that anxious individuals made greater errors when the position of the probe was discordant from the position of the angry face, and exhibited faster reaction times when the position of the probe was concordant with that facial expression (Armony & Dolan, 2002; Bradley, Mogg, White, Groom, & de Bono, 1999; Coull, 1998; MacLoeod, Rutherford, Campbell, Ebsworthy & Holker, 2002; Mogg & Bradley, 1998; Mogg, Millar, & Bradley, 2000; Posner, 1986).

Together, the findings suggest that when threat stimuli are present, traumatized individuals exhibit a bias towards the recognition of those threatening stimuli and appear to involuntarily encode trauma-relevant information (Amir et al., 1996; Chemtob et al., 1999; Kaspi et al., 1995; Vrana et al., 1995). Interestingly, it has been proposed that

these automatic biases in processing trauma-related information might contribute to PTSD symptomatology (McNally et al., 1993), particularly hyperarousal (i.e., hypervigilance and exaggerated startle response) and involuntary re-experiencing of symptoms. Indeed, as PTSD affected individuals appear to scan the environment preferentially for trauma-specific threatening stimuli, it is possible that the identification of such stimuli may act as reminders of traumatic events and produce intrusive recollections, flashbacks, and nightmares, which in turn, might interfere with cognitive functioning (Chemtob et al., 1999; Cassiday et al., 1992; Foa & Rothbaum, 1998; Pineles, Shipherd, Mostoufi, Abarmovitz, & Yovel, 2009; Pineles, Shipherd, Welch & Yovev, 2007).

This pattern of findings suggests that these biases toward trauma-related stimuli might also disrupt memory processing or retrieval of other memories. Given that retrieval of information from explicit memory often involves the selection of a particular event over other competing events, a process that frequently gives rise to interference effects, it is possible that biases toward trauma-related material may also interfere with memory processes. Although memory interference has been widely studied (Anderson & Neeley, 1996), little is known about whether and how intrusive memories of previous traumatic experiences interfere with memory functioning.

Stressful Experiences: GCs Effects on Brain Functioning and Subsequent Cognition

Processes involving the release of GCs (i.e., corticosterone in animals and cortisol in humans) might be an important mechanism by which stressors influence brain structure and function, as well as memory processes (Golier & Yehuda, 1998). Indeed, in

both animals and humans, chronic exposure to high levels of GCs have been associated with hippocampal atrophy and impaired memory functioning (Bremner et al., 1995; Bremner & Narayan, 1998; Gurvits et al., 1996; Kirschbaum, Wolf, May et al., 1996; Landfield, Baskin & Pitler, 1981; Landfield, Waymire & Lynch, 1978; Ling, Perry & Tsuang, 1981; Luine, Villegas, Martinez & McEwen, 1994; Lupien et al., 1997, 1998; Sapolsky et al., 1988, 1990; Starkman, Gebarski, Berent, & Schteingart, 1992).

However, an opposite relationship between cortisol reactivity and hippocampal volume, as well as between hippocampal volume and memory impairments, has been recently reported in humans. In healthy young men, a *larger hippocampal volume* (instead of smaller as previously found in animals and humans) was associated with poorer memory, as well as significantly *stronger cortisol reactivity* (Pruessner, Pruessner, Hellhammer, Pike & Lupien, 2007). Furthermore, although a smaller hippocampus has been linked to declarative memory deficits in PTSD individuals (Tischler, Brand, Stavitsky et al., 2006). The smaller hippocampus, lower cortisol levels, and memory impairments commonly associated with this disorder may not be directly linked to one another (Lindauer, Olf, van Meijel, Carlier & Gersons, 2006; Yehuda, Golier, Tischler, et al., 2007).

Acute Effects of GCs on Cognition: Enhancement or Impairment?

Administering HPA hormones or drugs that affect these hormones (i.e. hydrocorticosterone, dexamethasone, prednisolone), or exposing participants to a laboratory stressor (e.g. a videotaped speaking performance, or public speaking (Trier Social Stress Test, TSST)) have been the most commonly used methods to study the influence of acute stressors/GCs on cognitive processes. So far, studies using these methods have yielded inconsistent and sometimes contradictory findings (Lupien &

McEwen, 1997; Mendl, 1999). Indeed, pharmacological and stressor treatments heightened several aspects of cognition in some studies involving rodents and humans (Beckwith et al., 1986; Buchanan & Lovallo, 2001; Cahill, Gorski, & Le, 2003; Chajut & Algom, 2003; de Kloet, Rosenfeld, Van Eekelen et al., 1988; Lupien, Wilkinson, Brière et al. 2002; Micco, McEwen & Shein, 1979; Micco & McEwen, 1980; Mitchell & Meaney, 1991; Roozendaal, 2000; Stansbury, Hayley & Koeneker, 2000; Veldhuis, de Korte & de Kloet, 1985), whereas they had detrimental or no effect on cognition in other studies (Bohnen, Houx, Nicolson, & Joll, 1990; Braunstein-Bercovitz, Dimentman-Ashkenazi, & Lubow, 2003; Domes, Heinrichs, Rimmele et al., 2004; Elzinga, Bakker & Bremner, 2005; Forget, Lacroix, Somma & Cohen, 2000; Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Lupien & McEwen 1997; Lupien et al., 1997; Newcomer et al., 1994, 1999; Skosnik, Chatterton, Swisher, & Park, 2000; Wolf et al., 2001).

Similarly controversial findings were found with respect to memory processes (for a review, see Wolf, 2003). In general, however, high levels of exogenous GCs did not affect implicit memory (Kirschbaum et al. 1996; Lupien et al. 1994; Newcomer et al. 1994, 1999; Seeman et al. 1997) but were detrimental to declarative memory and other hippocampal-dependent types of memory (de Quervain, Roozendaal, Nitsch et al., 2000; Newcomer et al. 1994, 1999). Once again, these studies suggest that cortisol interactions with hippocampal neurons might induce memory deficits.

Several propositions have been offered to explain the discrepant findings concerning the acute effects of stressors/GCs on cognition. One view has been that the acute effects of stressors and/or GCs on cognition follow an inverted U-shaped function with increasing stressor severity. In particular, cognitive processes such as vigilance,

attention, memory and the acquisition of tasks that involve the hippocampus, are enhanced at low to moderate levels of GCs, whereas these processes are disrupted by high GC levels exceeding the optimal level (Conrad, Lupien & McEwen, 1999; Diamond, Bennet, Fleshner & Rose, 1992; Diamond, Fleshner & Rose, 1999; Joels, Pu, Wiegert, Oitzl, & 2006; Lupien & McEwen, 1997; Pugh, Duda, Sitaramayya, & Sharma, 1997).

The lack of consistency concerning the effects of stressors/GCs might also have to do with the effects of stress interacting with the various phases of memory. Indeed, memory processing involves several phases (encoding, consolidation, storage and retrieval) and stress-related factors may differentially affect memory processes at any of these levels (i.e., depending on when the stressor is encountered or when GC is administered or released (for reviews see Joels, 2006; Wolf, 2003; and for a meta-analysis see Het, Ramlow & Wolf, 2005). In this regard, these studies suggest that both stressors and high levels of GCs facilitate consolidation, but impair memory retrieval both in rats and humans (Beckner, Tucker, Delville, & Mohr, 2006; de Kloet et al., 1999; de Quervain, Roozendaal, & McGaugh, 1998; de Quervain et al., 2000, 2003; Kim & Diamond, 2002; Kuhlman et al. 2005a, b; Lupien & Lepage, 2001; McEwen & Sapolsky, 1995; Newcomer et al., 1994, 1999; Roozendaal 2002; Tollenaar, Elzinga, Spinoven, & Everaerd, 2008; Wolf et al. 2001). However, none of the studies to date have demonstrated both of these effects in the same experiment. A few studies have reported impaired retrieval by exogenous GCs, but no effects on consolidation (Wolf et al., 2004), whereas others reported enhanced consolidation by GCs and no effect on retrieval (Abercrombie, Kalin, Thurow, Rosenkranz, & Davidson, 2003; Beckner et al., 2006;

Buchanan & Lovallo, 2001; Cahill et al., 2003; McGaugh, 2000; Okuda, Roozendaal, McGaugh, 2004; Roozendaal, 2002). These inconsistent findings also led Roozendaal (2002, 2003, 2004) to propose that the modulating effects of GCs on cognition (enhanced consolidation, but impaired retrieval) may be dependent on GC interactions with noradrenergic and corticoid receptors located in the amygdala; particularly the basolateral amygdala (BLA), which in turn, modulate the activity of the hippocampus (Roozendaal, 2002; Roozendaal, Barsegyan, & Lee, 2008). Generally, emotional arousal activates the BLA and the interaction of the BLA with the hippocampus and other brain regions, such as the caudate nucleus, nucleus accumbens and cortex, enhances the memory consolidation of emotional material (Akirav & Richter-Levin, 2002; Buchanan et al., 2005, 2006a; Dolcos, LaBar, & Cabeza, 2005; Kensinger & Schacter, 2005; McGaugh, 2002, 2004; McGaugh, McIntyre, & Power, 2002; Pelletier & Paré, 2004; Sharot & Phelps, 2004) but impairs memory retrieval (Roozendaal et al., 2003). Further, the influence of emotional arousal involving the amygdala on declarative memory may vary according to the time elapsed between encoding and recall of the learned material (Bianchin, Souza, Medina, & Izquierdo, 1999). Specifically, short-term declarative recall (i.e., when assessed within 1 hr after learning) was not affected by emotionally arousing material, whereas long-term declarative memory recall (i.e., assessed 1 week later) was enhanced (Quevedo, Sant'Anna, Madruga, et al., 2003).

The influence of stress-related factors on consolidation and retrieval may also depend of the nature of the to-be remembered material as well as the timing of the stressor or GC release (i.e., whether they occur before, during or after learning as well as before recall). For instance, it was reported that GC release *during a given experience*

enhanced the memory of that experience and the information related to that event (Buchanan & Lovallo, 2001; McGaugh, 2000; Roozendaal, 2002). Some findings suggest that the enhancing effects of GCs on consolidation might be specific to highly arousing material or emotional information related to the stressor, whereas others suggest that both emotional and neutral information related to the stressor is enhanced by GC release (Abercrombie et al., 2003; Maheu, Joerber, Beaulieu, & Lupien, 2004). Further, it was suggested that the memory enhancing effects occurred primarily when the stressor or GC changes occurred after learning (i.e., post-learning stress) (Buchanan & Lovallo, 2001; Cahill et al. 2003; Okuda, Roozendaal, McGaugh, 2004).

The importance of emotional stimuli has also been incorporated into analyses of retrieval processes. Specifically, it was suggested that the detrimental effects of stress-related factors on retrieval might be specific to emotionally arousing stimuli experienced before retrieval (i.e., pre-retrieval stress) (Abercrombie, Speck, & Monticelli, 2006; Buchanan & Lovallo, 2001; Cahill et al., 2003; de Quervain, Roozendaal, Nitsch, McGaugh & Hock, 2000; Domes, Heinrichs, Rimmele, Reichwald, & Hautzinger, 2004; Domes, Rothfischer, Reichwald & Hautzinger, 2005; Het et al., 2005; Kuhlman et al. 2005a, b; Maheu, Collicutt, Kornik, Moszkowski, & Lupien, 2005; Roozendaal 2002). In contrast, diverse findings were reported concerning the influence of emotional-material unrelated to stressor experiences. Regardless of when a stressor was presented (i.e., before learning or retrieval of emotional and neutral information unrelated to the stressor), increased GC levels were associated with memory impairments for both positive and negative emotional information, but had no effect on memory for neutral material (Abercrombie et al., 2006; Domes et al., 2004; Elzinga, Bakker, & Bremner,

2005; Kuhlmann, Piel, & Wolf, 2005; Maheu, Collicutt, Kornik, Moszkowski, & Lupien, 2005). However, it is unclear whether or not these findings reflect the effects of stressors on cognition in naturalistic circumstances (Lupien & Schramek, 2006).

Moderators of the Relation Between Stressors and Well-being

As mentioned earlier, not all individuals respond to stressors in the same fashion and not all stressors have the same impact. Several organismic variables (i.e., genetics, age, gender), psychological variables (i.e., coping strategies, social support, appraisals), situational factors, and characteristics of the stressor itself, such as its severity, predictability and chronicity, differentially influence behavioural and neurochemical responses to stressors and their effect on well-being. Among these variables, social support plays a particularly significant role in determining the impact of stressful events on stressor reactivity (Ditzen et al., 2007, 2008; Eisenberger, Taylor, Gable, Hilmert, & Lieberman, 2007; Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Nausheen, Gidron, Gregg, Tissarchondou, & Peveler, 2007; O'Donovan & Hugues, 2008; Rosal, King, Ma & Reed, 2004; Strickland, Deakin, Percival, Dixon, Gater & Goldberg, 2002; Uchino & Garvey, 1997), well-being and the emergence of psychopathology (Anisman & Matheson, 2005; Aspinwall & Taylor, 1997; Kessler, Price, Wortman, 1985; Powers, Ressler, & Bradley, 2008). For instance, the social support perceived by an individual both before and after encountering a traumatic event is an important factor in determining vulnerability to the development of PTSD (Charuvastra & Cloitre, 2008; Pietrzak, Johnson, Goldstein, Malley, Southwick, 2009; Markowitz, Milrod, Bleiberg, Marshall, 2009).

Several reports have emphasized the importance of taking into account the quality of social support when considering the benefits of social support on well-being. Indeed, depending on their reaction, personal relationships may be a source of great comfort and/or great frustration during particularly distressing times. Unsupportive social interactions refer to the upsetting or unhelpful behaviors (such as minimizing the problem or blaming the person) received by an individual from members of his/her social network with respect to a particular stressor (Ingram et al., 2001; Mindes et al., 2003; Reynolds & Perrin, 2004; Rook, 1984, 1992; Schrimshaw, 2003). In this regard, negative social interactions during stressful events may be particularly detrimental to well-being (Ingram, Betz, Mindes, Schmitt, & Grant Smith, 2001; Lincoln, 2000). These experiences have been associated with depression (Ingram, Jones, Fass, Neidig & Song, 1999; Schuster, Kessler, Aseltine, 1990), decreased social functioning and psychological adjustment, and emotional problems (Davis, Brickman, & Baker, 1991; Figueiredo, Fries, & Ingram, 2003; Mindes, Ingram, Kliewer, & James, 2003; Song & Ingram, 2002). Interestingly when both positive elements (e.g., perceptions of emotional and practical support) and negative aspects of support (indifference and criticism) were considered, a negative social environment was a better indicator of PTSD symptomatology than a lack of positive support (Ullman & Filipas, 2001; Zoellner, Foa, & Bartholomew, 1999). Moreover, negative social support predicted subsequent PTSD symptoms (Dunmore et al., 2001) and poorer responses to treatment for PTSD (Tarrier, Sommerfield, & Pilgrim, 1999), and these effects appeared to be stronger for women than for men (Andrews, Brewin, & Rose, 2003).

It would seem that individual difference factors pertaining to the availability and quality of social coping resources might also influence the effects of traumatic experiences on cognitive processes. However, the effect of the quality of social support received might also depend of the nature of the stressor. When the Trier Social Stress Test is experienced in front of either a supportive or unsupportive audience, both conditions elicited significantly increased cortisol reactivity, suggesting that in some instances, the evaluative component may outweigh the positive effects of social support (Taylor et al., 2010). Interestingly, in these laboratory and evaluative settings, cortisol increases do not appear to be due to the quality of the social support, but rather to the presence of explicit negative social evaluations (Bosch et al., 2009; Dickerson, Mycek, & Zaldivar, 2008; Dickerson & Kemeny, 2004; Gruenewald, Kemeny, Aziz, Fahey, 2004).

Overview of the Present Research

The goal of the present research was to assess the relationships between trauma experiences, reminders of trauma and their associated emotional responses, on memory, attention processes, and cortisol reactivity. It was also of particular interest to establish how negative support influenced the impact of stressors on these processes. Study 1 examined the role of trauma images, trauma reminders and the emotional arousal to individuals' most distressing life event in predicting memory performance. Study 2 investigated the contribution of cortisol reactivity in these relations. Finally, Study 3 assessed the relation between trauma reminders and the ensuing emotional and cortisol response on memory processes, and on attention bias toward trauma-related material. As well, this study assessed the potential moderating role of negative social interactions on these processes.

STUDY 1

Stressful events, including traumatic experiences, have been associated with several physiological and psychological disturbances such as depression and PTSD (e.g., McEwen, 2003). Although traumatized individuals exhibit heightened physiological reactivity, emotional arousal and cognitive biases toward trauma-related material compared to controls, other aspects of cognitive functioning, particularly memory and attention processes, appear to be impaired. While the processes by which trauma reminders lead to such outcomes in humans remains to be elucidated; in animal studies it is known that when rodents are exposed to a stressor, later re-experience of these stressors (or cues reminiscent of these stressors) elicits marked neurochemical and hormonal changes (Anisman et al., 2003). It is conceivable that similar processes operate in humans (Post, 1992), and it might be considered that reminders of previous trauma, and the emotional arousal elicited by these reminders, might interfere with memory processes. Thus, one objective of the present research was to further explore the interfering role of previously encountered traumatic experiences and their emotional arousal on memory processing. It was considered that a) reminders of traumatic experiences might impair retrieval of material unrelated to these experiences, and b) the impact of previous trauma on retrieval abilities might be dependent on emotional arousal elicited by these reminders (i.e., distress felt after being reminded of the most distressful event an individual had previously encountered). It was hypothesized that recall of prior trauma would be associated with poorer memory recognition of trauma-unrelated material relative to that evident in participants that had not encountered such experiences. Further, it was expected that participants exposed to trauma images (related to their own

trauma experiences) during the period between encoding and retrieval would exhibit greater memory disturbances compared to those exposed to trauma-unrelated images.

Methods

Participants and Procedures

University students (44 women; 21 men) aged between 18 and 32 years old ($M = 20.35$, $SD = 2.90$) were recruited from an Introductory Psychology course for a study entitled “Images of emotional events in relation to behavioral and hormonal reactivity” (Appendix B). The sample comprised individuals who were Euro-Caucasian ($n = 39$; 60 %), East Asian ($n = 2$; 3.1 %); Asian ($n = 12$; 18.5 %), Middle-Eastern ($n = 11$; 16.9 %) and Black ($n = 1$; 1.5 %). This study was approved by the Carleton University Psychology Ethics Committee and met all ethics guidelines set forth by the (Canadian) Tricouncil.

Participants received experimental credits as incentive. After the study was described (Appendix B), participants completed an informed consent (Appendix C), and were asked to complete the following questionnaires (see Appendix D): Background Information, Beck Depression Inventory (BDI; Beck et al., 1961) and The Traumatic Life Events Questionnaire (TLEQ; Kubany et al. 2000). Importantly, the TLEQ not only served to provide information on past stressor experiences, but also served as a reminder of previous adverse events that had been experienced. Once these questionnaires were completed, participants engaged in a computer-based memory recognition test (Appendix E). Subsequently, students were debriefed, and provided with contact information in case of any concerns or distress (Appendix F).

Measures

The *Beck Depression Inventory* (BDI) is a 21 item self-report rating inventory measuring characteristic attitudes and symptoms of depression (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Participants were asked to select from a set of responses that reflected increasing degrees of problem severity (e.g., from 0 'I am not particularly pessimistic or discouraged about the future' to 3 'I feel the future is hopeless and things cannot improve'). Responses were summed and categorized into three groupings of depressive symptomatology (BDI scores < 9: minimal symptoms; BDI scores between 9 and 18: moderate symptoms; and BDI scores >18: high symptoms). The item-total reliability for the total summed scores on the BDI was high (Chronbach $\alpha = .85$). Depressive symptoms varied appreciably across the sample (BDI range = 0 to 28; $M = 7.06$, $SD = 6.29$). The majority of participants reported minimal symptoms of depression ($n = 44$; 67.7%) and about a third reported moderate ($n = 18$; 27.7%) or high symptoms of depression ($n = 3$; 4.6%). Because of the small sample sizes of participants reporting moderate and high depressive symptomatology, the two categories were collapsed in subsequent analyses of this study.

Trauma History was determined through the *Traumatic Life Events Questionnaire* (TLEQ; Kubany et al. 2000). This 23-item self-report questionnaire assesses exposure to a broad spectrum of potentially traumatic events, ranging from natural disasters, accidents, assaults and childhood abuses. Events are described in behaviourally descriptive terms (consistent with the DSM-IV PTSD criterion A1). The frequency of occurrence of each event was assessed using a 7-point scale on which participants indicated whether each event occurred from never (0) to more than five times (6). When

events were endorsed, respondents were asked if they experienced intense fear, helplessness, or horror (the PTSD stressor criterion A2 in the DSM-IV), and how long ago the event occurred. Trauma history scores were calculated by summing the frequencies of all the traumatic events experienced that participants reported as causing fear, helplessness, and/or horror. These experiences included: shock (e.g. natural disaster, accident; $n = 34$), assault ($n = 33$), death of a loved one ($n = 32$) and witnessing violence done to others ($n = 31$). Thus, high scores reflected multiple experiences of each of these four types of traumatic events (Cronbach's $\alpha = .79$). The majority of the participants experienced at least one of these events ($n = 54$; 83.1%), with many of them experiencing multiple events whereas the remainder of participants reported not having encountered any of the experiences listed in the TLEQ ($n = 12$; 16.9%).

Importantly, respondents were also asked in the TLEQ to indicate which of the events that they experienced elicited the most distress and to rate the intensity of the distress experienced when being reminded of that event on a 6-point Likert scale ranging from 0 'none happened' to 6 'extreme distress'. This item was used in all the subsequent studies as a measure of the *distress* or emotional arousal following trauma reminders.

Memory Recognition Test

The present study adapted the modified forced-choice recognition memory test described by Chandler and Gargano (1991, 1998) to explore the role of emotional interference on memory processing in relation to previously encountered traumatic experiences. In this task, retrieval interference can be manipulated across experimental conditions. The modified forced-choice recognition test consists of three successive tasks: i) a memory-encoding task; ii) an intervening task comprising one of four

conditions: control (no intervening tasks), neutral (intervention consists of being exposed to images unrelated to any sort of trauma (i.e., light bulb, lamp, spoon), trauma-related (being exposed to images related to previous trauma experienced by the participant) or trauma-unrelated (being exposed to trauma images but that are unrelated to participants' previous trauma experiences); and iii) a forced-choice memory recognition test.

During the memory-encoding task, the stimulus materials consisted of 36 images portraying nature scenes and landscapes. Each of the 36 images was presented for 750 ms at a rate of 1 segment/sec. To encourage effective encoding and good retention of these stimuli into memory, participants were instructed to judge the visual complexity of the scenes depicted. On each trial, the participant pressed one button if the scene was complex (e.g., picture including trees, mountains, waterfalls; many focal points of interest) or a second button if the scene was judged to be simple (e.g., picture including only desert sand; only one main focal point of interest).

An intervening task, which followed two minutes after completion of the encoding task, used the same presentation parameters. In this phase, as indicated earlier, participants were randomly assigned to only one of the four conditions: no intervention, presentation of neutral images, trauma-related images or trauma-unrelated images. Given the high frequency of participants having encountered trauma previously (~ 80%), there was a relatively high probability of participants being exposed to trauma-related or -unrelated scenes. As the control condition had no intervening task, participants assigned to this condition performed the forced-choice memory recognition test at an interval equivalent to that of participants who received the intervening test (approximately two

minutes). During this time, participants were instructed to relax and wait for further instructions to appear on the computer screen.

In each of the three intervening conditions, 36 images selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthberg, 1999) were presented in random order, and the participants were asked to rate how stressful each image was to them on a Likert-scale of 1 'not stressful' to 5 'extremely stressful' by pressing on the corresponding keyboard keys. In the neutral condition, participants were presented with 36 emotionally neutral images (e.g., lamp, spoon, iron), whereas the other two conditions (i.e., trauma-related or trauma-unrelated) involved presentation of 10 images of traumatic events interspersed with 26 neutral images. In the latter two conditions, the series of images each included two types of trauma, namely (a) a shocking experience or witnessing a traumatic event happening to another (e.g., car accident, fire, injured soldier), or (b) assault or death of an individual (e.g., a girl being assaulted, couple in a graveyard). It was expected that of those individuals that had experienced trauma (~ 80%), half of these participants would have experienced one of these trauma experiences and by using the two sets of trauma images for each participant, a large proportion would have the images match their own trauma experiences. Importantly, in each of these conditions, none of the images were the same as those presented during the encoding task.

A forced choice recognition test immediately followed the intervening task. This consisted of the successive presentation of 18 images presented during encoding together with an equal number of new images (18) that had not been presented previously in the experiment. Again, the participant was required to indicate by pressing a keyboard key as

to whether the picture had been presented during encoding. Recognition trials were presented at intervals of one second, with each picture appearing for 2000 ms. Appendix E provides a schematic illustration of these different tasks.

Results

Memory Recognition Performance

Table 1 illustrates the performance of participants on the memory recognition task. Preliminary analyses were conducted to assess outliers (i.e., greater or less than ± 3 Z-scores) and missing data, as were a series of diagnostic measures (e.g., normal probability plots, skewness < 3) to ensure that the assumptions necessary for the analyses were met. Levene's Test of Homogeneity of variance revealed that the variances of latencies of correct, incorrect recognition and the difference of latencies between errors and correct recognition were not homogeneous (all Levene's $p < .05$). Thus, these latencies were log transformed to correct for the lack of homogeneity of variance. However, for informative purposes, raw latencies measures are reported in Table 1. Raw memory recognition scores were used for the analyses as none of the assumptions were violated for this measure.

.Table 1.

Overall Error Rates and Response Latencies in the Memory Recognition Test (M ± SD)

	Condition			
	None	Neutral	Shock/Witness trauma	Assault/Death
Errors	5.56 ± 2.23	5.53 ± 2.50	6.46 ± 2.44	7.00 ± 2.15
<i>Response Latencies</i>				
Correct Recognition (ms)	636 ± 178	1152 ± 627	973 ± 359	762 ± 221
Error Recognition (ms)	735 ± 277	1375 ± 721	1292 ± 731	852 ± 287
Difference between Errors and Correct (ms)	99 ± 174	223 ± 493	319 ± 411	89 ± 157

Effect of Gender, Depressive Symptoms, Intervening Condition and Previous Trauma on Recognition Memory Scores and Latencies

In order to assess the influence of gender, depressive symptoms, intervening condition and prior trauma on memory recognition scores, a 2 (Gender) x 2 (Depression Category: minimal or moderate to severe) x 2 (Condition: being exposed to trauma images or not) x 2 (Prior trauma or not) ANOVA was conducted. As seen in Table 2, this univariate analysis revealed that neither gender nor depression categories influenced memory recognition scores. However, participants having experienced at least one trauma made more recognition memory errors than trauma-free individuals. In addition, individuals exposed to trauma images exhibited more recognition memory errors compared to individuals that were not exposed to such images (see Table 2).

Gender and prior trauma did not interact with the other variables to influence recognition memory errors (all $F < 1$, *ns*), whereas depression category significantly interacted with the experimental condition, $F(1, 51) = 5.28$, $p < .05$, $\eta^2 = .094$. In order to specify the nature of this interaction, separate univariate analyses were conducted to assess the effect of condition (i.e., being exposed to trauma images or not) in each of the depression categories. As seen in Table 3, follow-up analyses revealed that participants exhibiting moderate to severe symptoms of depression, and who were exposed to trauma images, made more errors compared to those participants exhibiting the same levels of symptomatology but who had not been exposed to such images. For participants exhibiting minimal symptoms of depression, those exposed to trauma images did not make more errors than participants not exposed to these images.

Table 2.

Overall Error Rates and Response Latencies as a function of Gender, Depression

Category, Intervening Condition and Prior Trauma

Variables	<i>M</i>	<i>SD</i>	<i>F</i> (1,51)	η^2
<i>Gender</i>			1.96	.037
Male (<i>n</i> = 21)	5.77	.56		
Female (<i>n</i> = 44)	5.23	.46		
<i>Depression</i>			1.53	.029
Minimal (<i>n</i> = 44)	5.60	.44		
Moderate to severe (<i>n</i> = 21)	5.38	.61		
<i>Condition</i>			4.81*	.086
Not exposed to trauma image (<i>n</i> = 35)	4.77	.48		
Exposed to trauma images (<i>n</i> = 30)	6.24	.53		
<i>Prior Trauma</i>			8.04**	.136
None (<i>n</i> = 11)	4.03	.72		
More than one (<i>n</i> = 54)	6.61	.33		

* $p < .05$; ** $p < .01$; *** $p < .001$

Table 3

Overall Error Rates and Response Latencies as a function of Depression Category and Intervening Condition¹

	Condition		<i>F</i> (1, 51)	η^2
	Not exposed to trauma images	Exposed to trauma images		
	<i>n</i> , <i>M</i> ± <i>SD</i>	<i>n</i> , <i>M</i> ± <i>SD</i>		
<i>Depression</i>			5.28*	.094
Minimal	<i>n</i> = 25, 5.30 ± .55	<i>n</i> = 19, 5.90 ± .68		
Moderate to severe	<i>n</i> = 10, 4.06 ± .86	<i>n</i> = 11, 6.70 ± .85		

* $p < .05$; ** $p < .01$; *** $p < .001$

¹ The *F* and η^2 values reported in this Table correspond to the values of the interaction between depressive category and condition in the univariate analysis illustrated in Table 2.

A similar 2 (gender) x 2 (depression category) x 2 (condition) x 2 (prior trauma) MANOVA was conducted on the various latencies measured (correct recognition, incorrect recognition or difference between correct and incorrect recognition). This analysis indicated that none of these factors, nor their interactions, influenced response latencies (all F 's ns).

Effect of Distress Elicited by Trauma Reminders on Memory Recognition

As seen in Table 4, correlation analyses revealed that the intensity of the distress caused by the most distressful event reported on the TLEQ was associated with more recognition errors, longer latencies of recognition, longer latencies of errors of recognition and longer latencies of correct recognition. However, the latency difference between errors and correct recognition was not significantly associated with the distress reported.

Effect of Presenting Trauma-Related Images versus Trauma-Unrelated Images on Recognition Memory Scores and Latencies among Traumatized Individuals

It was of interest to assess whether presenting trauma-related images (those that were related to previous trauma experienced by the participant) or trauma-unrelated images (including traumatic events that were unrelated to those previously experienced by the participant) influenced memory recognition inaccuracy. A 2 (trauma-related or not) x 2 (depression category) ANOVA was conducted only on the responses of participants that reported previous traumatic experiences and that were assigned to the conditions in which they were presented with trauma images. Participants were further classified according to the degree of concordance of their previous traumatic history and the images to which they were exposed (i.e. either trauma-related or trauma-unrelated).

As seen in Table 5, this analysis revealed that participants who had experienced trauma that was concordant with the images they were presented with made significantly more errors compared to those participants who had been exposed to trauma-unrelated images. In this analysis, depressive symptoms did not directly influence memory recognition scores, nor did it interact with the degree of concordance of the trauma images with previous trauma. Likewise, the degree of concordance of previous trauma with the images presented did not influence or interact with depressive symptomatology in affecting recognition error latencies, latencies of correct recognition, or the latency difference between errors and correct recognitions, $F_s(1,28) < 1, ns$.

Table 4.

*Pearson Correlation Coefficients of Distress Following Trauma Reminders, Overall
Error Rates and Response Latencies*

	Distress <i>R</i>
Memory Performance	
Recognition errors	.27*
<i>Latencies</i>	
Recognition errors	.29*
Correct recognition	.29*
Difference errors – correct recognition	.11

* $p < .05$

Table 5.

Overall Error Rates and Response Latencies as a function of Depression Category and Being Exposed to Trauma-Related Images

Variable	<i>M</i>	<i>SD</i>	<i>F</i> (1,26)	η^2
<i>Depression</i>			0.29	.011
Minimal (<i>n</i> = 19)	6.20	.52		
Moderate to severe (<i>n</i> = 11)	5.70	.78		
<i>Trauma relatedness</i>			10.10**	.280
Trauma related (<i>n</i> = 7)	4.45	.84		
Trauma unrelated (<i>n</i> = 23)	7.44	.43		

** *p* < .01

Discussion

Experiment 1 assessed whether previous traumatic experiences interfered with memory processes and aimed to identify several factors that might influence such an outcome. To this end, participants were first reminded of their trauma history by completing the TLEQ. Thereafter, participants completed a computer-based memory recognition task. It was observed that poorer performance in a memory recognition task was related to being exposed to trauma images before retrieval. This outcome appeared to reflect the interactive effects of depressive symptomatology and exposure to trauma images. As well, recall of previous traumatic experiences (and the high distress associated with the recall) disrupted memory performance. In this regard, participants exposed to trauma-related images before retrieval displayed more frequent memory recognition errors compared to those participants exposed to trauma-unrelated images. In contrast to recognition errors, none of these variables influenced reaction times, including latencies of correct recognition, latencies of error recognition or the latency difference between correct recognition and recognition errors.

The fact that trauma history was associated with greater memory errors for neutral material compared to that observed among individuals that had not encountered such experiences is consistent with past research linking trauma and impairments in learning and memory (Bremner et al., 1993, 1995a; Gil et al., 1990; Gilbertson et al., 2001; Jenkins et al., 1998; Vasterling et al. 1998, 2000; Uddo et al., 1993; Yehuda et al., 1995). As expected, exposure to trauma images before retrieval was sufficient to induce greater memory errors compared to being exposed to neutral images or not being exposed to any images. Given that exposure to trauma images or TLEQ administration could constitute

potent stressors, especially for individuals that had encountered such experiences (i.e., served as a reminder of previous adverse events), our findings are also consistent with studies showing that laboratory stressors provoke memory impairments for material unrelated to the stressor itself (Domes, Heinrichs, Reichwald, & Hautzinger, 2002; Jelicic, Geraerts, Merckelbach, & Guerrieri, 2004; Lupien, Buss, Schramek, Maheu, & Pruessner, 2005; Lupien, Fiocco, Wan, Maheu, Lord, Schramek, et al., 2005; Sauro et al., 2003; Takahashi et al., 2004; Wolf, Schommer, Hellhammer, Reischies, & Kirschbaum, 2002). Since performance on tasks that tap memory for novel material is facilitated if the material is preceded by something that primes its representation in memory (Marcel, 1983), it might have been expected that individuals having previously experienced trauma would show facilitated recall for trauma-relevant information during implicit or explicit memory tasks and conversely, impaired recall of trauma-unrelated information.

Distress generally appears to have an influential role on cognition. For example, distress has been related to poorer working memory, but not processing speed or episodic memory in aged adults (Stawski, Sliwinski, & Smyth, 2006). Distress has also been linked to prospective memory impairments in individuals with HIV (Woods, Iudicello, Moran, Carey, Dawson, & Grant, 2008), to memory and concentration complaints in women suffering from breast cancer and chronic pain (Shilling & Jenkins, 2007; Munoz & Esteve, 2005); with poor effort on cognitive tests in Mild Traumatic Brain Injury (MTBI) (Stulemeijer, Andriessen, Brauer, Vos, & Van Der Werf, 2007); and have been found to have a predictive role on tests assessing attention, concentration, memory and reasoning abilities (Iezzi, Duckworth, Vuong, Archibald, & Klinck, 2004).

The memory impairments associated with distress as well as with the interaction between depressive symptomatology and exposure to trauma images could be the result of “mood congruency effects”. In this regard, several experiments suggested that memories are more easily retrieved when their valence is congruent with the momentary mood of the participant (Blaney, 1986; Matt, Leuthold, & Sommer, 1992). Specifically, subjects with low mood might be expected to show enhanced memory for negative stimuli and impaired memory for neutral or positive stimuli. Because of the negative affective state induced by reminders of previous traumatic experiences (or their depressive symptomatology interacting with trauma images), it was expected that these participants would be less likely to recall the neutral stimuli that had been presented previously. As mentioned earlier, emotional arousal generally influences memory processes only when the source of the arousal (the emotionally arousing event) is directly related to the information to be remembered (Gore, Krebs, & Parent, 2006; Kuhlman & Wolf, 2006).

Based on the present results, it was not possible to determine whether the detrimental effect of the stressor on memory recognition performance occurred at the encoding versus retrieval levels in traumatized individuals exposed to trauma images. Indeed, participants who had experienced trauma tended to make more memory recognition errors when they were presented trauma images that were concordant with their trauma history (i.e., submitted to pre-learning stress and pre-retrieval stress) compared to participants exposed to neutral images or individuals that had not been exposed to any images (i.e., only submitted to pre-learning stress). Consistent with other studies of attention processes (Chemtob et al., 1999), these results suggest that previous

stressor experiences might result in reminders of stressors impacting memory functioning.

Together, these results are consistent with the idea that exposure to trauma-related material might activate intrusive memories, particularly in depressed individuals, which might then interfere with memory. However, the small sample size of this study represents a major limitation regarding the conclusions that can be drawn, and hence ought to be considered with caution.

STUDY 2

As mentioned earlier, there is ample evidence suggesting that individuals that had experienced traumatic events exhibited greater physiological reactivity as well as cognitive biases toward trauma-related material (Bryant & Harvey, 1995, 1997; Butler, Braff, Rausch et al. 1990; Cassiday, McNally & Zeitlin, 1992; Coles & Heimberg, 2002; Elsesser, Sartory, & Tackenberg, 2004; Foa, Feske, Murdock et al. 1991; Freeman & Beck, 2000; Kaspi, McNally & Amir, 1995; McNally, 1997; McNally, Amir & Lipke, 1996; McNally, English, & Lipke, 1993; McNally, Kaspi, Riemann, & Zeitlin, 1990; Miller & Litz, 2004; Moradi, Taghavi, Neshat-Doost, Yule, & Dagleish, 2000; Morgan et al., 1996, 1997; Paunovic, Lundh & Ost, 2003; Squire & Zola-Morgan, 1991; Thrasher, Dagleish & Yule, 1994; Vrana, Roodman & Beckman, 1995; Vythilingam et al., 2007; Williams et al., 1996; Zeitlin & McNally, 1991). Further, it was proposed that biases toward trauma-related stimuli might interfere with performance in a cognitive task. By showing that the emotional arousal associated with trauma reminders and exposure to trauma-related images interfered with memory processes and induced memory impairments for material unrelated to trauma, the data of Study 1 are consistent with this

proposition. The mechanisms involved in these processes remain to be elucidated, but recent findings suggest that long-term alteration of amygdala neuronal activity and consequent emotional arousal might play a prominent role in this respect (Tsoory, Vouimba, Akirav, Kavushansky, Avital, & Richter-Levin, 2008). Further, as indicated earlier, stress-related glucocorticoid alterations could underlie changes in cognitive functioning, either as a cause or as an effect of amygdala activation and/or hippocampal functioning. It was hypothesized that high levels of distress associated with trauma-reminders would be associated with more memory recognition errors and higher cortisol reactivity. Further, it was expected that cortisol levels would mediate the relation between 1) distress associated with trauma reminders and memory performance and 2) being exposed to trauma-related images and memory performance.

Methods

Participants

Women ($n = 112$) and men ($n = 45$), aged between 18 and 32 years old ($M = 20.58$, $SD = 4.22$) were recruited from Introductory Psychology course for a study on “Images of emotional events in relation to behavioral and hormonal reactivity” (Appendix G). This sample was principally Euro-Caucasian ($n = 116$; 73.9 %) and the remainder was Middle-Eastern ($n = 12$; 7.6 %), East Asian ($n = 12$; 7.6%), Asian ($n = 8$; 5.1%) and Black ($n = 7$; 4.5 %).

Procedure

After the study was described (Appendix G), participants completed an informed consent (Appendix H). Participants were asked to provide four saliva samples at several time-points during the experiment. The initial saliva sample was collected at the

beginning of the experiment once the informed consent was signed. Thereafter, participants were asked to complete the following questionnaires (see Appendix D and I): Background Information, Beck Depression Inventory (BDI; Beck et al., 1961) and the Traumatic Life Events Questionnaire (TLEQ; Kubany et al. 2000). Once these questionnaires were completed, participants provided the second saliva sample. Participants then completed the same computer-based memory recognition test used in Study 1 (Appendix E). They provided the third saliva sample after they completed the memory recognition task, waited a further twenty minutes and then provided the last saliva sample. Thereafter, they were debriefed and provided with contact information in case of any concerns or distress (Appendix J). Given that cortisol follows a well-defined diurnal pattern (Linkowski, Van Onderbergen, Kerkhofs, Bosson, Mendlewicz, & Van Cauter, 1993; Schmidt-Reinwald, Pruessner, Hellhamer et al., 1999; Späth-Schwalbe, Schöller, Kern, Fehm, & Born, 1992; Van Cauter, Sturis, Byrne et al., 1994; Windle, Woods, Shanks, Lightman, & Ingram, 1998), and that time of day might be an important moderating variable of the effects of cortisol/stressors on memory processes (Lupien, Gillin & Hauger, 1999; Lupien et al., 2005; Maheu et al., 2005), all sessions were conducted in the afternoon.

Measures

Given that certain medications may also influence the level of stress hormones, information about medications was collected as part of the Background information. However, the few individuals using prescription drugs (e.g., antidepressants) performed at or near the means for their respective groups, and hence no further efforts were made to control for drug treatments. As in study 1, depressive symptoms were assessed using

the BDI (Beck et al., 1961; Cronbach's $\alpha = .86$). Depressive symptoms varied appreciably in this sample (BDI range = 0 to 28, $M = 8.44$, $SD = 6.90$). The majority of participants reported minimal symptoms of depression (BDI scores < 9 ; $n = 100$, 63.7%) whereas a smaller proportion reported moderate (BDI scores between 9 and 18; $n = 43$, 27.4%) or high symptoms (>18 ; $n = 13$; 8.3 %). Once again, because of the small sample sizes of participants reporting moderate and high symptoms of depression, the latter two categories were collapsed for subsequent analyses.

As in the first study, the TLEQ was used to assess participants previous traumatic experiences and also served as a trauma reminder (Kubany et al., 2000; Cronbach's $\alpha = .65$). Traumatic history scores were calculated by summing the frequencies associated with traumatic events that elicited fear, horror and helplessness. Of the total sample, twenty-eight participants (17.8%) reported no previous traumatic experiences, whereas the vast majority of the participants reported at least one traumatic experience ($n = 129$, 82.2%). Moreover, as in Study 1, participants' ratings of how much distress elicited by the most distressful event that happened to them on the TLEQ (e.g. anxiety, worry, sadness, or grief) served as a measure of *distress* following trauma reminders. Memory functioning was measured using the modified recognition memory task described in Study 1 (Chandler & Gargano, 1991, 1998).

Salivary Cortisol

As indicated earlier, saliva samples were taken on four occasions: upon their arrival to the laboratory; after the completion of the questionnaires (BDI and TLEQ); immediately following the memory task (which might include the presentation of trauma images) and twenty minutes later. Saliva samples were obtained by having participants

chew on a piece of cotton for two minutes. All saliva samples were stored at -80°C until they were analyzed for cortisol levels. Salivary cortisol levels were determined, in duplicate, by means of a solid phase radio-immuno assay using ^{125}I obtained from ICN Biochemicals Inc., CA. The inter- and intra-assay variability was less than 10%.

Results

Effect of Gender, Depressive Symptoms, Intervening Condition and Previous Trauma on Recognition Memory Scores and Latencies

Preliminary analyses were conducted to assess outliers (i.e., greater or less than ± 3 z-scores) and missing data, as were a series of diagnostic measures (e.g., normal probability plots, skewness < 3) to ensure the assumptions for ANOVA were met. In order to assess the influence of gender, depressive symptoms, prior trauma and intervening condition (being exposed to trauma images or not) on recognition memory scores, a 2 (Gender) x 2 (Depression Category) x 2 (Condition) x 2 (Prior trauma or not) ANOVA was conducted. Levene's Test revealed that the variance of memory performance scores were homogeneous, $F(13, 142) = 1.80, p > .05$, and therefore raw memory recognition scores of participants were used. As seen in Table 6, this analysis revealed that neither gender, depression category, prior trauma, nor being exposed to trauma images, influenced or interacted with each other to influence memory recognition scores, all interaction $F_s = ns$. A similar analysis also indicated that recognition latencies (correct and errors of recognition, and difference of latencies between the two) were not influenced by these variables or their interactions, all $F_s = ns$.

Table 6.

Overall Error Rates and Response Latencies as a function of Gender, Depression Category, Prior Trauma and Intervening Condition

Variable	<i>M</i>	<i>SD</i>	<i>F</i> (1, 142)	η^2
<i>Gender</i>			2.81	.019
Male (<i>n</i> = 45)	6.41	.79		
Female (<i>n</i> = 111)	6.99	.57		
<i>Depression</i>			0.38	.003
Minimal (<i>n</i> = 100)	6.90	.48		
Moderate to severe (<i>n</i> = 56)	6.43	.94		
<i>Prior Trauma</i>			0.32	.002
None (<i>n</i> = 27)	6.30	.96		
More than one (<i>n</i> = 129)	7.00	.45		
<i>Condition</i>			2.71	.019
Not exposed to trauma image (<i>n</i> = 77)	6.94	.63		
Exposed to trauma images (<i>n</i> = 79)	6.46	.74		

Effect of Distress Elicited by Trauma Reminders on Memory Recognition Scores and Latencies

As seen in Table 7, correlation analyses indicated that the distress felt from remembering the most distressful event reported by participants in the TLEQ was related to more recognition memory errors, but did not influence the latencies of correct recognition, errors of recognitions or the latency difference between adequate recognition and errors.

Effect of Presenting Trauma-Related Images versus Trauma-Unrelated Images on Recognition Memory Scores and Latencies

To assess whether exposure to trauma-related images influenced memory recognition errors in individuals having experienced previous trauma, univariate analyses were conducted with individuals that were assigned to the conditions in which they were presented with trauma images. Participants were further classified according to the degree of concordance between their previous trauma history and the images to which they were exposed (i.e. either trauma-related ($n = 50$) or trauma-unrelated ($n = 30$)). As seen in Table 8, individuals exposed to trauma images related to their own experiences tended to make more recognition memory errors compared to those individuals exposed to trauma-unrelated images. However, this outcome was just shy of statistical significance, but it did account for almost 5% of the variance in memory performance. In the same fashion, a multivariate analysis revealed that exposure to trauma-related images did not influence latencies of correct recognition, latencies of errors of recognition, or the difference between the two, all $F_s < 1$, *ns*.

Table 7.

*Pearson Correlation Coefficients of Distress Following Trauma Reminders, Overall
Error Rates and Response Latencies*

	Distress
	<i>r</i>
Recognition errors	.29 ^{***}
<i>Latencies</i>	
Recognition errors	-.00
Correct recognition	-.05
Difference errors – correct recognition	-.07

^{***} $p < .001$

Table 8.

Overall Error Rates and Response Latencies as a function of Being Exposed to Trauma-Related Images

Variables	<i>M</i>	<i>SD</i>	<i>F</i> (1,78)	η^2
<i>Trauma Relatedness</i>			3.72 [†]	.046
Trauma related (<i>n</i> = 30)	5.73	.66		
Trauma unrelated (<i>n</i> = 50)	7.34	.51		

[†]*p* < .10

Cortisol/Stress Reactivity

Overall, the four saliva samples collected throughout the experiment were not statistically different across time or as function of gender, depression score or trauma history. Specifically, as seen in Table 9, a mixed measures analysis of variance in which sampling time was considered to be a within group variable and gender, depression category, trauma history and condition were considered as between-group variables, indicated that neither time, gender, depression, trauma history, condition or their interactions, significantly influenced cortisol levels, as measured from the saliva samples collected in the laboratory, $F_s < 1$, *ns*.

A separate set of analyses was conducted assessing cortisol change after the completion of the computer task (ratio 1) and twenty minutes after the TLEQ was completed (ratio 2). The cortisol scores were calculated as a proportion of the cortisol score at baseline (i.e., at arrival in the laboratory). In order to assess the effect of the TLEQ and the computer task on these ratios, a 2 (Gender) x 2 (Depression Category) x 2 (Condition) x 2 (Prior trauma or not) MANOVA was conducted. As seen in Table 10, this analysis revealed that neither ratio 1 nor ratio 2 was influenced by gender, depression, condition, trauma history, or their interactions, all $F_s < 1$, *ns*.

Table 9.

Cortisol Levels in function of Time, Gender, Depression Category, Prior Trauma and Intervening Condition

Variable	<i>F</i>	η^2
<i>Within-subject variable</i>		
	<i>F</i> (3, 420)	
Time	0.36	.003
<i>Between-subjects variables</i>		
	<i>F</i> (1, 140)	
Gender	1.69	.012
Depression	0.34	.002
Prior trauma	0.00	.000
Condition	0.08	.001

Table 10.

Cortisol Reactivity in function of Gender, Depression Category, Prior Trauma and Intervening Condition

	Cortisol Reactivity ²			
	Ratio 1		Ratio 2	
	<i>F</i> (1,135)	η^2	<i>F</i> (1,135)	η^2
<i>Between-subjects variables</i>				
Gender	.34	.002	.62	.005
Depression	.01	.000	.04	.000
Prior trauma	.00	.000	.24	.002
Condition	.13	.001	.01	.000

² Ratio 1 correspond to the cortisol change after the completion of the computer task relative to baseline levels and ratio 2 corresponds to cortisol changes twenty minutes after the TLEQ was completed relative to baseline levels.

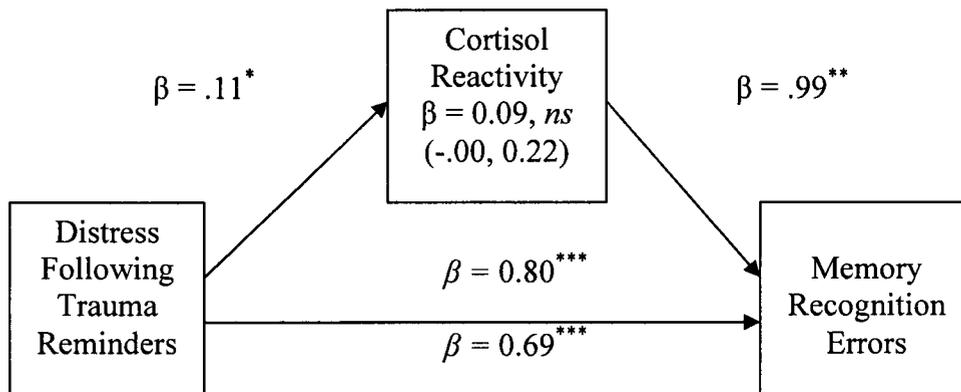
Distress, Cortisol Reactivity and Recognition Memory

It was posited that cortisol levels would be related to the distress reported on the TLEQ as well as to higher memory recognition errors. Correlation analysis indicated that higher cortisol reactivity (ratio1; cortisol levels after the completion of the memory task compared to baseline levels) was related to both high distress ($r = .17, p < .05$) and higher recognition memory errors ($r = .27, p < .001$). Given these relations, it is possible that cortisol levels may serve as a mediating variable in the relation between distress and memory recognition errors. To examine this mediated model, Preacher and Hayes (2004) procedures for assessing multiple mediations were followed. This approach uses bootstrap sampling distribution and the construction of confidence intervals in order to assess the direct, indirect (i.e., through mediator(s)), and the total effects of the predictor variables (i.e., distress felt following trauma reminders). This method is considered to be superior to the Sobel Test (as delineated by Baron and Kenney, 1986), in that it has greater statistical power (Mallinckrodt, Abraham, Wei & Russel, 2006), does not assume a normal distribution, and it is especially useful with smaller samples (i.e., more conservative) (Preacher & Hayes, 2004). Preacher, Rucker and Hayes (2004) SPSS macro for mediation was used for the analysis, with 1000 bootstrap samples and 95% bias corrected and accelerated confidence intervals.

As seen in Figure 1, when the potential mediating role of cortisol reactivity was assessed in the relation between distress following trauma reminders and memory recognition errors, this relation remained significant. Further, the 95% confidence intervals for cortisol reactivity overlapped zero, indicating that cortisol reactivity did not mediate the relation between distress and memory recognition errors.

Figure 1.

Path Diagram depicting the Indirect Effects of Cortisol Reactivity in the Relation between Distress Following Trauma Reminders and Memory Recognition Errors



Note. The unstandardized coefficients for the total effect of distress following trauma reminders on memory recognition errors is presented above the horizontal arrow-line; the unstandardized coefficient for the direct effect of distress following trauma reminders on memory recognition errors (controlling for cortisol reactivity) is presented below the horizontal arrow-line. The unstandardized coefficient for the indirect effect of cortisol reactivity and the bootstrapped 95% corrected and accelerated confidence intervals are presented inside the boxes.

* $p < .05$; ** $p < .01$; *** $p < .001$

Discussion

One objective of Study 2 was to replicate the findings of the first study, using a larger sample size. As in the first study, poorer memory recognition was related to increased distress elicited by remembering the most distressful event reported in the TLEQ. Further, cortisol reactivity following trauma reminders (i.e., cortisol levels after the completion of the TLEQ and the memory task compared to baseline levels) was also related to increased distress as well as higher errors of memory recognition. However, cortisol reactivity did not mediate the relation between distress and memory performance. Both cortisol reactivity and distress constituted significant independent predictors of memory performance.

As mentioned in Study 1, the fact that distress following trauma reminders was related to poorer recognition memory performance is consistent with previous studies suggesting that emotional arousal may be an important factor that influences cognitive processes and particularly memory (Dolan, 2002). Interestingly, Medonça-de-Souza et al., (2007) emphasized that negative affect associated with exposure to unpleasant context increased sensitivity to an acute stressor, and were critical to stimulation of cortisol release by a speech stressor. Indeed, they found that compared to basal levels, a cortisol response in association with public speaking (using TSST paradigm) was only seen among those who had first viewed unpleasant images and scored above the average on the negative affect scale. Further, the fact that higher cortisol levels were associated with distress following trauma reminders is in line with the results of Elzinga et al., (2003) who reported higher cortisol levels following trauma-related reminders.

The finding that higher cortisol levels were related to greater memory impairments in trauma-unrelated material is consistent with studies that have shown that manipulation of cortisol levels influenced memory for both neutral and emotional information (Abercrombie et al., 2003; Andreano & Cahill, 2006; Buchanan, Tranel & Adolphs, 2006; Jelici et al., 2004; Maheu et al., 2004; Tops et al., 2003). These studies generally assessed the impact of exogenous cortisol administration on measures of verbal memory (i.e., word list). The objective of the present investigation was to relate endogenous changes of cortisol to impairments in “visual memory” (i.e., participants had to encode images of landscapes). Further, given that visual or spatial memory is dependent on the hippocampus (Kandell et al., 2000), our findings were also consistent with previous reports suggesting that hippocampal-dependent forms of memory (declarative memory) might be particularly impaired by chronic elevation of corticosteroids (Lupien et al., 1994; Newcomer et al., 1994; Seeman et al. 1997; for a meta-analysis see, Sauro, Jorgensen, & Pedlow, 2003). As well, greater cortisol reactivity following a psychological stressor was associated with lower cognitive performance in a subsequent task (Buchanan et al., 2006; Elzinga & Roleofs, 2005).

Given that cortisol release follows a well-defined diurnal rhythm (Linkowski, Van Onderbergen, Kerkhofs, Bosson, Mendlewicz, & Van Cauter, 1993; Schmidt-Reinwald, Pruessner, Hellhamer et al., 1999; Späth-Schwalbe, Schöller, Kern, Fehm, & Born, 1992; Van Cauter, Sturis, Byrne et al., 1994; Windle, Woods, Shanks, Lightman, & Ingram, 1998), some studies suggested that time of day might be an important moderating variable of the effects of cortisol/stressors on memory processes. Indeed, administrations of exogenous GCs, or psychological stressors administered in the afternoon had no effect

on memory performance, whereas they were detrimental if administered in the morning (Lupien, Gillin & Hauger, 1999; Lupien et al., 2005; Maheu et al., 2005). Our sample appeared to yield cortisol changes that were inconsistent with this view, as the detrimental effect of psychological stressor/increased cortisol reactivity influenced memory performance despite the samples being collected in the afternoon. In fact, in their meta-analysis of the relation between stress, cortisol and memory processes, Sauro et al., (2003) did not find any differences in the effect sizes reported between samples collected in the morning versus the afternoon. Furthermore, it is important to consider that GC administration or laboratory stressors commonly used in these studies are quite different from the stressor in the present investigation (i.e., reminders of trauma history). Indeed, exogenous GC administration or laboratory stressors are of short-term duration and might not reflect the impact of naturalistic stressors or long term effects of previous trauma exposure on cortisol reactivity and memory processes. It is possible that, in the present study, trauma reminders might have been particularly relevant to participants, involved different appraisal processes, been related to higher distress (as shown here), and involved other neurochemical processes, and thus produced different outcomes.

Unfortunately, it is impossible, based on our findings, to disentangle whether the effects of these endogenous elevations of cortisol occurred before learning or retrieval, although the effect of cortisol elevations were more likely to have occurred before retrieval if we take into account the amount of time it takes for cortisol to be released and to provide feedback to the brain. To be sure, the associations between memory performance, cortisol and emotional reactivity following trauma reminders in this study design cannot be interpreted in a causal way.

Several studies suggested that exogenous cortisol administration before retrieval exerted the greatest influence on memory performance, thus altering the retrieval of already acquired memories, compared to when the drug was given before or immediately after learning (de Quervain et al., 2000, 2003; Wolf et al., 2001a; see Het et al., 2005 for a meta-analysis). Although it has been suggested that endogenous stressor-induced increases in cortisol secretion should have memory effects similar to those reported after administration of exogenous glucocorticoids, the empirical evidence in humans is inconsistent. Data from studies with elderly participants showed that a brief stressor can reduce declarative memory performance when subjects are exposed to the stressor immediately before learning (Maheu et al, 2005; Wolf et al., 1998) or retrieval (Lupien et al., 1997). In contrast, three other studies did not detect such effects using a similar laboratory stressor (Domes et al., 2002; Wolf et al., 2001b, 2002).

Further, there is ample support that the ability of PTSD individuals to recall specific autobiographical memories in response to reminder cues is particularly impaired, compared to less-distressed traumatized individuals and with those without trauma exposure (Hauer, Wessel, Geraerts, Merckelbach & Dalgeish, 2008; McNally, Lasko, Macklin, & Pitman, 1995; Moore & Zoellner, 2007; Schonfeld & Ehlers, 2006). Importantly, reduced specificity of autobiographical memory has been linked to poorer symptom outcomes in longitudinal studies (Harvey, Bryant & Dang, 1998; Kleim & Ehlers, 2008; Williams et al., 2007). Although this type of memory impairment may be a consequence of trauma exposure, constituting a way of avoiding those specific aspects that may be distressing (Dalgeish, Rolfe, Golden, Dunn, & Barnard, 2008; Williams et al., 2007; Williams, Stiles & Shapiro, 1999), it was also argued that pre-trauma memory

styles may themselves influence the course of post-traumatic stress reactions (Bryant, Sutherland, & Guthrie, 2007; Hauer, Wessel, Engelhard, Peeters, Dalgeish, 2009; Williams et al., 2007). Inasmuch as the stressor used in the present study involved reminders of past experience, it might have been more appropriate to assess the effects of reminder stimuli on autobiographical memories that could potentially involve neural circuits like those associated with trauma recall.

Unexpectedly, in contrast to the initial study, there was no apparent effect of previous traumatic experiences or of being exposed to trauma-related images on memory recognition. Participant's age, gender or levels of previous trauma experiences did not differ between the two studies. However, participants of Study 2 made more recognition errors, regardless of whether they were exposed to trauma images or not, and reported more distress following trauma reminders compared to participants of Study 1. Whether or not these differences were due to PTSD symptomatology or sensitization effects (i.e., being affected more by previous experiences) is uncertain. Nevertheless, this might be a plausible explanation for the between study difference, particularly as participants of Study 2 reported more early adverse events such as maltreatment and abuse while growing up.

It is known that early life events may have profound long-term ramifications on adult well-being and subsequent stressor reactivity (Anisman & Matheson, 2005; Meaney, 2001). In this regard, it has been proposed that such events may result in the sensitization of neurochemical processes, so that later introduction of stressors elicit exaggerated responses (Anisman et al., 2003; Heim & Nemeroff, 2001; Nemeroff, 1996; Post, 1992) that come to provoke adverse outcomes (Anisman & Matheson, 2005; Post,

1992). Thus, it is possible that the greater distress and lower memory functioning among participants of the second study may be a result of multiple (or chronic) traumatic events, coupled with early life adverse experiences. This proposition is admittedly speculative, but alternative accounts for the differences, other than a spurious outcome in one of the studies, are not readily apparent.

Taken together, the findings of the present study suggest that emotional arousal and stress-related elevations of cortisol levels were associated with impaired memory performance for material unrelated to the stressor (i.e., neutral images). This is consistent with other studies showing that the effects of emotionally arousing and/or stressful events on declarative memory vary according to the nature of to-be remembered material.

STUDY 3

Study 1 and 2 indicated that the degree of memory impairment following reminders of previous traumatic experiences seemed to be related to the psychological distress and cortisol reactivity associated with such reminders. As discussed earlier, it is possible that altered attention and/or motivation after stressor exposure might also have affected memory performance. Indeed, it has been reported that an attention bias directed toward trauma-related stimuli, which is frequently reported in traumatized individuals, might interfere with performance in an attention test (Chemtob et al., 1999; Pineles, Shipherd et al., 2007, 2009).

Given that attention biases directed toward threat stimuli (at the expense of attention toward emotionally neutral information) might be reflective of PTSD symptomatology rather than previous trauma exposure *per se* (Kimble, Kaloupek, Kaufman, & Deldlin, 2000; Michael, Elhers, & Halligan, 2005; Stanford, Vasterling,

Mathias, Contans, & Houston, 2001; Vythilingam et al., 2007), the first objective of this study was to examine whether attention biases toward trauma-related material were present in the general population and in sub-clinical populations that have experienced traumatic events. This was achieved by comparing performance on an attention task using trauma-related, positive or neutral images or words. A second objective of this study was to determine whether such biases were related to (1) distress and cortisol reactivity following trauma reminders (2) depressive and PTSD symptomatology, as well as whether these variables might come to influence memory processes.

As indicated earlier, not all individuals respond to stressors in the same fashion and not all stressors have the same impact. It has been reported that social support, particularly perceived support (i.e., social resources that one believes would be available to him or her if needed) plays a significant role in determining the impact of stressful events on well-being (Sarason, Sarason, & Pierce, 1990) including the development of PTSD (Brewin et al., 2000; Charuvastra & Cloitre, 2008). In the same fashion, unsupportive social interactions in response to a specific stressful event may influence subsequent psychological adjustment, well-being, and health in adults (Ingram, Betz, Mindes, Schmitt, & Smith, 2001; Mindes, Ingram, Kliever, & James, 2003; Reynolds & Perrin, 2004; Rook, 1984; Schrimshaw, 2003).

Inasmuch as unsupportive social interactions play a role in determining the ultimate impact of trauma experiences on well-being, it is possible that individual difference factors pertaining to the quality of social interactions during a stressful encounter might influence the effects of traumatic experiences on cognitive processes. Research addressing the associations of these variables with traumatic history, memory

and attention processes warrants further examination. Thus, a further objective of this study was to explore the moderating role of social support, particularly unsupportive social interactions during previous traumatic experiences, in the relation between traumatic history and stress-related outcomes (i.e. distress, depressive and PTSD symptoms, memory, attention bias toward trauma-related material and cortisol reactivity). It was hypothesized that the influence of trauma history on these outcomes would be moderated by unsupportive social interactions. In particular, those participants having experienced more unsupportive social interactions during their most distressful traumatic event would exhibit greater distress following trauma reminders, depressive and PTSD symptoms and attention bias-toward trauma-related material, as well as a poorer performance on the memory task. Furthermore, it was expected that these effects would be mediated by greater reported distress, as well as higher cortisol reactivity, following trauma reminders compared to those that had more unsupportive social interactions during the traumatic event.

Methods

Participants

University students (135 women; 61 men) aged between 16 and 61 years old ($M = 19.98$, $SD = 4.37$) were recruited from an Introductory Psychology course for a study entitled “Images of emotional events in relation to behavioral and hormonal reactivity” (Appendix K). About two-thirds of the sample was Euro-Caucasian ($n = 132$; 67.3%), whereas the remainder self-identified as Asian ($n = 29$; 14.8%); Middle-Eastern ($n = 13$; 6.6%), Black ($n = 17$; 8.7%) or did not report their ethnicity ($n = 5$; 2.6%).

Procedure and Measures

When beginning the actual testing session, after describing the study, participants completed the informed consent (Appendix L). Thereafter, participants provided the first of four saliva samples and completed a series of background information questions. As in Study 1 and 2, participants were asked to complete the Beck Depression Inventory (BDI; Beck et al., 1961). The item-total reliability for the total summed scores on the BDI was high (Cronbach $\alpha = .87$). Once again, depressive symptoms varied appreciably across the sample (BDI range = 0 to 43; $M = 9.23$, $SD = 7.19$) with more than half of participants reporting minimal symptoms of depression ($BDI \leq 9$; $n = 115$; 58.7%), an appreciable portion reporting moderate ($10 \leq BDI \leq 18$; $n = 59$; 30.1%) or high symptoms of depression ($BDI \geq 19$; $n = 22$; 11.2%). The latter two categories did not differ on any of the test dimensions and hence were pooled in subsequent analyses.

As in Study 1 and 2, the Traumatic Life Events Questionnaire (TLEQ, Kubany et al., 2000) not only assessed trauma history but also served as a trauma reminder in this study. Trauma history scores were calculated by summing the frequencies of all the traumatic events experienced that participants reported as causing fear, helplessness, and/or horror. Thus, high scores reflected multiple experiences of traumatic events (Cronbach's $\alpha = .80$). The majority of the participants experienced at least one of these events ($n = 164$; 84.1%), with many having had experienced multiple events, whereas the remainder reported not having encountered any of the experiences listed in the TLEQ ($n = 31$; 15.9%). As in Study 1 and 2, participants' ratings of the distress elicited by the most distressful event that happened to them on the TLEQ (e.g. anxiety, worry, sadness, or grief) served as a measure of *distress or emotional arousal* following trauma reminders.

Participants were also asked to complete a few additional questionnaires namely the Impact of Event Scale - Revised (IES-R; Weiss & Marmar, 1997) and the Modified Unsupportive Social Interaction Inventory (MUSII; Ingram, Betz, Mindes, Schmitt, & Smith, 2001) (Appendix M). The IES-R is a 22 item self-report questionnaire that was used to assess current specific distress to any specific life event. Six of these items reflect hyperarousal symptoms such as anger, irritability, heightened startle response, difficulty concentrating, hypervigilance; eight items reflect intrusion (in which one item refers to dissociative states when experiencing true flash-backs) whereas the other eight items reflect avoidance symptoms. Participants had to indicate how frequently they experienced each item in the past seven days on a 5-point Likert scale ranging from 0 'not at all' to 4 'extremely'. Summed scores on all the items were used since the item-total reliabilities for the total scores for on the IES-R were very high (Cronbach's $\alpha = .95$), as were the correlations between the subscales and total scores (ranging in magnitude from .68 to .94). The choice to use a total score on the IES-R instead of the separate scores on the three IES-R dimensions (i.e., hyperarousal, intrusion and avoidance) was also based on the fact that when BDI scores were regressed onto the three IES-R dimensions, consideration of the three dimensions only slightly improved predictability of depressive symptoms ($R^2 = .298, p < .001$) in comparison to the relations with total scores on PTSD ($R^2 = .288, p < .001$). Taken together, these findings indicated that it would be reasonable to proceed with the use of a total score in further analyses. High scores on this index therefore reflected high levels of hypervigilance, intrusions and avoidance symptoms. In this study, PTSD symptoms varied appreciably across the sample (IES range = 0 to 76; $M = 20.97, SD = 18.98$).

In addition to the IES-R, participants completed the MUSII (Ingram, Betz, Mindes, Schmitt, & Smith, 2001), which was slightly modified to measure unsupportive social interactions in relation to the most distressful event reported on the TLEQ. This measure involves 24-items rated on a 5-point scale ranging from 0 'none' to 4 'a lot'. For each item of the MUSII, respondents were asked to "indicate how much of that type of response" they received from other people during their most distressful event encountered on the TLEQ. Evidence supporting the construct validity of the instrument included significant correlations with measures of depression and psychological distress, after controlling for the influence of stress and positive social support (Ingram et al., 1999, 2001). Previous exploratory and confirmatory factor analysis revealed 4 subscales, namely distancing (e.g., When I was talking about the issue/situation with someone, this person did not give me enough of his/her time, or made me feel like I should hurry), bumbling (e.g., From the person's tone of voice, expression, or body language, I got the feeling that the person was uncomfortable talking with me about my problem), minimizing (Someone thought I was over-reacting to the situation), and blaming (Someone made "should/shouldn't have" comments about my role in the situation, such as, "You shouldn't have") (Ingram et al., 2001). In the present study, the four subscales were also calculated (reflecting bumbling, distancing, minimizing and blaming), with the factors demonstrating relatively high intercorrelations (ranging from .45 to .61). However, findings similar to those obtained with the PTSD subscales indicated that it would be reasonable to proceed with the use of a total score (Cronbach $\alpha = .92$) in further analyses. Specifically, the item-total reliabilities for the total scores for unsupportive social interactions were very high (Cronbach's $\alpha = .92$), as were the

correlations between the subscales and total scores (ranging in magnitude from .73 to .84). In addition, when BDI scores were regressed onto the four unsupportive social interactions subscales ($R^2 = .229, p < .001$), consideration of the four dimensions only slightly improved predictability of depressive symptoms in comparison to the relations with total scores on unsupportive social interactions ($R^2 = .209, p < .001$). Therefore, high scores on unsupportive social interactions reflected high levels of bumbling, blaming, distancing and minimizing during the most distressful event reported on the TLEQ.

Once these questionnaires were completed, participants provided the second saliva sample, and then executed one memory and two attention computer-based tasks that were administered in a randomized order to each participant. The memory task used in this study was similar to the one used in Studies 1 and 2 (Appendix E). Only the intervening task was slightly changed to include 16 trauma images (instead of 10) and 20 neutral images. Two additional computer-based tasks were used to measure attention bias toward trauma-related material, namely the Visual Probe Task (Mogg & Bradley, 1999) and a Modified Emotional Stroop test (e.g., Paunovic et al., 2002). For a schematic description of these tasks, see Appendix N.

Visual Probe Task

A Visual Probe Task was used to determine whether participants who had previously encountered traumatic experiences had an attention bias toward trauma-related material. The Visual Probe Task involved showing a pair of images for 500 ms. Each pair, taken from the IAPS, was presented together on a computer screen. Every pair comprised a neutral picture and a picture of an emotional experience (positive or

traumatic). Sixty-four pairs of images were presented to the participants. Half (32) of these included both neutral and positive experiences and the other half comprised neutral and traumatic experiences. An additional 16 pairs were used as practice items.

Each trial started with a central fixation cross for 500 ms, followed by the pair of pictures for 500 ms. Following the offset of the pair, a single dot probe was presented in the location of one of the pictures (i.e., at the position of the neutral or emotional stimulus). Participants were asked to indicate *the location of the probe* (left or right) by pressing one of two keys as quickly and accurately as possible. Trauma images and probes appeared on the left or right with equal frequency. The inter-trial interval varied randomly between 500 and 1250 ms. It has been shown, using this paradigm, that attention biases toward emotional stimuli promote faster reaction times when the position of the probe is the same as the position of the emotional picture (Bradley et al., 1999; Mogg & Bradley, 1998, 1999, 2000; Mogg, Bradley, Hyare, & Lee, 1998; Mogg, Millar & Bradley, 2000). Thus, it was expected that if attention was biased toward trauma material, participants would make less errors and be faster to identify the location of the probe when its position was concordant with the position of these images, relative to those cases where the probe and images were discordant.

Modified Emotional Stroop Test

The Stroop Test is a neuropsychological test that assesses cognitive processes such as attention, flexibility and executive functioning. The task takes advantage of our ability to read words more quickly and automatically than we can name colors. This task usually involves three conditions: 1) reading; 2) color naming (i.e. name the color of the ink in which the word is written); and 3) interference condition, in which people are

required to verbally identify the color of each printed word (but in this instance the print is a color different from the color expressed by the word's semantic meaning, hence leading to interference in verbal expression of the color).

In this experiment, an emotional version of the Stroop Test was used. The task used four conditions. The first three conditions were similar to those used in the regular Stroop Test mentioned earlier (i.e., color reading, color naming and color interference) whereas the fourth condition was similar to the one used by Paunovic et al. (2002). In this condition, three categories of words were used: eight positive words (i.e., comfort, safety, secure), eight neutral words (i.e., object, fruit, stamp), and eight trauma-related words (i.e., assault, abuse, accident; see Appendix N) and participants had to name the color in which these words were printed. During the Stroop Test, each word was individually presented three times, once in each of the possible colors (blue, green, and red). Given that the error rate on this condition was very low (< 4 errors), analyses consisted only of correct color naming responses that were made between 200ms and four seconds after word presentation. For each participant, the response accuracy and mean latencies on the color-naming task for each word type (i.e. neutral, positive or traumatic) were calculated. In order to determine whether traumatized individuals exhibited colour-naming interference effects in response to trauma-related words but not to words unrelated to their trauma, trauma words were classified according to their degree of relevance with participants' trauma history. Consequently, mean latencies for trauma-related and trauma-unrelated words were computed.

Once the computer-based tasks were completed, participants were provided with the third saliva sample. Finally, the fourth saliva sample was provided twenty minutes

after the computer tasks were completed. During the 20 minutes period, participants relaxed while reading neutral materials. Participants were debriefed and provided with contact information in case of any concerns or distress (Appendix O).

Salivary Cortisol

As already indicated, saliva samples were taken on four occasions: upon their arrival to the laboratory; after the completion of the questionnaires (BDI, TLEQ, IES, MUSII); immediately following the memory and attention tasks (which might include the presentation of trauma images or words) and 20 minutes later. Saliva samples were obtained by having participants chew on a piece of cotton for two minutes. All saliva samples were stored at -80°C until they were analyzed for cortisol determinations. Salivary cortisol levels were determined, in duplicate, by means of a solid phase radio-immuno assay using ^{125}I obtained from ICN Biochemicals Inc., CA.

Results

Preliminary Analyses

As in Studies 1 and 2, preliminary analyses were conducted to assess outliers and missing data, as well as a series of diagnostic measures to ensure that the assumptions of our planned analyses were met. No assumptions were violated, and thus raw responses were assessed in subsequent analyses.

Behavioral Performance of Participants on Attention Tasks

As mentioned earlier, one objective of this study was to examine whether attention biases toward trauma-related cues were present in the general population or in sub-clinical populations that had experienced traumatic events. Behavioral performance of participants on the Modified Emotional Stroop Test and the Visual Probe Task are

depicted in Tables 11 and 12 respectively. As seen in Table 11, regardless of their trauma history, participants did not take significantly longer to color name trauma or positive words compared to neutral ones (all t s = ns). In the same fashion, as seen in Table 12, participants did not exhibit faster reaction times when the position of the probe was concordant with the trauma positive image than when its position was discordant (the difference between the two conditions indicates if there was a bias toward these emotional images, which was not the case here).

Together, these descriptive statistics suggest that an attention bias toward trauma-related or more generally toward emotionally-related material was not present in the sample *as a whole*. However, subsequent correlation analyses, presented in the next sections, helped to determine whether attention biases toward emotional images or words were more prominent among individuals that had experienced traumatic events.

Table 11

Overall Responses Latencies for Neutral, Positive and Trauma Words in the Modified Emotional Stroop Test (M ± SD)

	Words			
	Neutral	Positive	Trauma- Related	Trauma- Unrelated
Response Latencies ³	755 ± 151	756 ± 154	780 ± 205	769 ± 176

³ As mentioned earlier, response latencies are the time required to identify correctly the color of the ink in which the word is printed.

Table 12

Overall Errors Rates and Responses Latencies for Trauma and Positive Images in the Visual Probe Task (M ± SD)

	Images	
	Positive	Traumatic
Errors	0.56 ± 2.29	0.56 ± 2.29
<i>Response Latencies</i>		
Probe Concordant	409 ± 166	401 ± 140
Probe Non Concordant	417 ± 167	398 ± 141
Attention Bias ⁴	8.00 ± 166	-3.00 ± 140

⁴ Attention bias is the mean reaction time difference between trials in which the probe position is concordant with the position of the trauma images relative to those trials where the probe and these images are discordant.

Cortisol/ Stress Reactivity

Overall, salivary cortisol did not vary as a function of gender, depression or trauma history. Specifically, a mixed measures analysis of variance in which sampling time was considered to be a within group variable and gender, depression category and trauma history were considered as between-group variables, indicated that cortisol levels varied over time, $F(3,528) = 4.82, p < .01, \eta^2 = .027$. It appeared that cortisol levels significantly declined linearly with time, $F(1,176) = 9.59, p < .01, \eta^2 = .052$. However, neither gender, $F(1,176) = 0.81, ns, \eta^2 = .005$, depression, $F(1,176) = 3.49, ns, \eta^2 = .019$, nor trauma history, $F(1,176) = 2.19, ns, \eta^2 = .012$ or their interactions, significantly influenced the cortisol levels, $F_s < 1, ns$.

To determine differences in cortisol reactivity to trauma reminders, cortisol scores were calculated as a proportion of the cortisol score at baseline (i.e., at arrival in the laboratory). Specifically, ratio 1 reflected cortisol change after the completion of the TLEQ; ratio 2 reflected cortisol changes after computer tasks; and lastly, ratio 3 reflected the cortisol change approximately 20 minutes after the TLEQ was completed. As indicated by a 2 (Gender) x 2 (Depression Category) x 2 (Condition) x 2 (Prior trauma or not) MANOVA, these ratios were not influenced by gender, depression, condition, trauma history, or their interactions, all Pillais's Trace $F < 1, ns$.

Relations Among Variables

To assess the relations between the variables measured (i.e. trauma, distress, depressive and PTSD symptoms, unsupportive social interactions, cortisol reactivity and cognitive functioning), correlation analyses were conducted (see Table 13). Once again, errors of recognition were significantly associated with increased distress following

trauma reminders. Increased numbers of errors on the Visual Probe Task was significantly associated with increased levels of past trauma. Further, as seen in Table 13, trauma experiences, unsupportive social interactions, distress following trauma reminders, as well as symptoms of depression and PTSD were all positively associated with each other. Cortisol ratios, however, were not significantly associated with any of these variables and thus, they were dropped from subsequent analyses.

Table 13.

Pearson Correlations Among the Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Trauma	-.												
2. Unsupportive Interactions	.29***	-.											
<i>Psychological Outcomes</i>													
3. Distress	.62***	.19**	-.										
4. PTSD	.48***	.43***	.56***	-.									
5. Depression	.37***	.46***	.36***	.54***	-.								
<i>Memory Task</i>													
6. Errors	.03	.03	.16*	.12	.11	-.							
7. Correct Recognition	.02	-.05	-.07	-.03	.08	-.30**	-.						
8. Recognition Errors	-.00	-.03	-.06	-.06	.06	-.15	.81***	-.					
<i>Stroop (Trauma Words)</i>													
9. Bias ⁵	.00	.14	.10	.08	.17*	.04	.08	.06	-.				
<i>Visual Probe (Trauma Images)</i>													
10. Errors	.24**	.12	.10	.10	.10	.01	-.08	-.07	.05	-.			
11. Bias ⁶	.03	-.03	.05	-.05	.05	-.04	.14	.13	.00	0.00	-.		
<i>Cortisol Reactivity</i>													
12. Ratio 1	.11	.14	.00	.02	.05	-.14	.03	.06	-.09	.01	.01	-.	
13. Ratio 2	-.06	-.07	-.09	-.17	-.12	-.05	.01	.04	-.08	-.01	.13	.58***	-.

Note. * $p < .05$; ** $p < .01$; *** $p < .001$

⁵ Difference of reaction time between trauma-related words and trauma-unrelated words

⁶ Difference of reaction time when the position of the probe is concordant with the trauma image compared to when its' position is discordant.

Effect of Gender, Depressive Symptoms, Prior Trauma, Condition and Being Exposed to Trauma-Related Images on Recognition Memory Scores and Latencies

As expected, ANOVA and MANOVA, similar to those conducted in Studies 1 and 2, indicated that memory performance (i.e., errors of recognition, latencies of correct recognition and latencies of incorrect recognition) did not vary as a function of gender, prior trauma, depression symptoms, condition and the order of the cognitive tasks, all *F*s, *ns*.

As evident in the preceding studies, being exposed to trauma-related images influenced memory performance, however this effect was just shy of significance, even though it explained 6.0% of the variance, $F(1,60) = 3.80$, $p = .06$, $\eta^2 = .060$. Once again, participants exposed to trauma-related images tended to make more recognition errors ($M \pm SD = 7.02 \pm 0.52$) than those exposed to trauma-unrelated images ($M \pm SD = 6.04 \pm 0.97$). Further, neither memory performance nor recognition latencies were influenced by the other variables, or their interactions (all *F*s, *ns*).

Trauma History and Stress-Related Outcomes: The Moderating Role of Unsupportive Social Interactions

The main objective of this study was to assess whether the interactions between trauma history and unsupportive social interactions influenced the stress-related outcomes (i.e. distress, depressive and PTSD symptoms, memory, attention and cortisol reactivity). Moderation was tested separately for each outcome through hierarchical regressions. Specifically, each outcome variable was regressed onto adverse events and unsupportive social interactions on the first step, and the interaction between these

variables on the second step. All these variables were standardized prior to calculating the interactions terms.

Table 14 indicates that overall trauma experiences were significantly associated with distress following trauma reminders in that high trauma history was associated with increased distress. However, neither unsupportive social interactions nor its interaction with trauma history significantly influenced distress.

Table 15 indicates that increased levels of trauma experiences and unsupportive social interactions tended to be associated with increased cortisol levels after trauma reminders relative to baseline levels (i.e., ratio 3) but this trend did not reach significance. In addition, the interaction between prior trauma and unsupportive social interactions predicted cortisol reactivity, although once more this outcome was modest, explaining 1.5% of the variance (see Table 15). Specifically, as seen in Figure 2, it was unexpectedly found that those with high levels of trauma and high unsupportive social interactions tended to have *lower* cortisol levels compared to participants with similar levels of trauma history, whereas high unsupportive social interaction was associated with greater cortisol reactivity at lower levels of traumatic experiences. Table 15 also indicates that the cortisol ratio measured 20 minutes after the completion of the experiment (i.e., ratio 4) was not influenced by these variables or their interactions.

In addition, both overall trauma and unsupportive social interactions, but not their interactions, were significant predictors of symptoms of depression and PTSD. Specifically, high trauma history and increased levels of unsupportive social interactions were predictive of higher depressive and PTSD symptoms (see Table 16 and 17).

Table 14.

*Regression Analysis (Pearson Correlations and Standardized Regression Coefficients)
Assessing the Moderated Relations between Traumatic Experiences, Unsupportive Social Interactions (Predictor Variables) and Distress Following Trauma Reminders (Outcome Variable)*

	Distress		
	<i>R</i>	β^7	R^2_{total}
Overall Trauma	.60***	.61***	.359***
Unsupportive Social Interactions	.19	.04	
Trauma x Unsupportive Social Interactions	.11	-.08	.006

*** $p < .001$

⁷ In this Table, only the Beta standardized coefficients of the second step are reported

Table 15.

Regression Analysis (Pearson Correlations and Standardized Regression Coefficients) Assessing the Moderated Relations between Traumatic Experiences, Unsupportive Social Interactions (Predictor Variables) and Cortisol Reactivity Following Trauma Reminders (Outcome Variable)

	Cortisol Reactivity					
	Ratio 1			Ratio 2		
	<i>r</i>	β^8	R^2_{total}	<i>R</i>	β^9	R^2_{total}
Overall Trauma	.12	.12	.026 [†]	-.05	-.03	.006
Unsupportive Social Interactions	.14	.13 [†]		-.07	-.06	
Trauma x Unsupportive Social Interactions	-.06	-.12 [†]	.015 [†]	-.02	.01	.000

Note. [†] $p < .10$

⁸ In this Table, only the Beta standardized coefficients of the second step are reported

⁹ In this Table, only the Beta standardized coefficients of the second step are reported

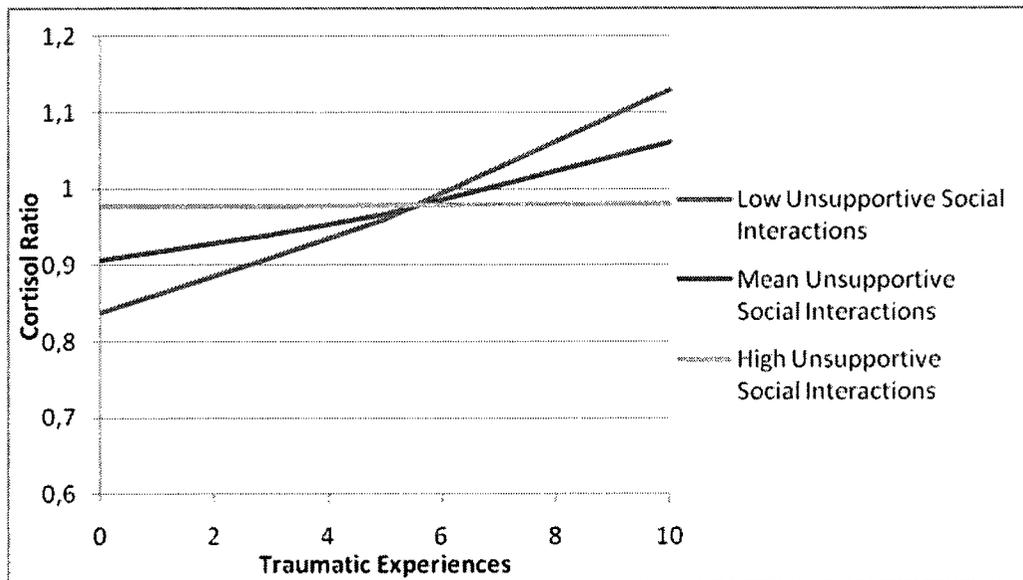


Figure 2. While high unsupportive social interaction was associated with greater cortisol reactivity at lower levels of traumatic experiences, those participants having encountered both high levels of trauma and high unsupportive social interactions tended to have *lower* cortisol levels compared to participants with similar levels of trauma history.

Table 16

*Regression Analysis (Pearson Correlations and Standardized Regression Coefficients)
Assessing the Moderated Relations between Traumatic Experiences, Unsupportive Social
Interactions (Predictor Variables) and Depressive Symptoms (Outcome Variable)¹⁰*

	Depressive Symptoms		
	<i>R</i>	β	R^2_{total}
Overall Trauma	.35***	.23***	.263***
Unsupportive Social Interactions	.46***	.38***	
Trauma x Unsupportive Social Interactions	.18	.03	.001

 $p < .001$

¹⁰ In this Table, only the Beta standardized coefficients (β) of the second step are reported.

Table 17

*Regression Analysis (Pearson Correlations and Standardized Regression Coefficients)
Assessing the Moderated Relations between Traumatic Experiences, Unsupportive Social
Interactions (Predictor Variables) and PTSD Symptoms (Outcome Variable)¹¹*

	PTSD Symptoms		
	<i>R</i>	β	R^2_{total}
Overall Trauma	.47 ^{***}	.38 ^{***}	.314 ^{***}
Unsupportive Social Interactions	.43 ^{***}	.31 ^{***}	
Trauma x Unsupportive Social Interactions	.20	.01	.000

^{***} $p < .001$

¹¹ In this Table, only the Beta standardized coefficients (β) of the second step are reported.

Neither prior trauma, unsupportive social responses received from others, nor their interactions predicted memory performance (see Table 18). In contrast, as seen in Tables 19 and 20, a significant interaction between trauma and unsupportive social interactions emerged with respect to performance on the attention task (i.e., the slopes were significantly different). The interaction between trauma and unsupportive social interactions significantly predicted response bias in the trauma condition of the Emotional Stroop Test as well as the number of errors and the bias in the trauma condition of the Visual Probe Task. As seen in Figure 3, in the Emotional Stroop Test, those individuals that had encountered high levels of trauma and a high degree of unsupportive social interactions took longer to identify trauma-related words than trauma-unrelated words (interference effect) compared to those with both lower levels of prior trauma and unsupportive social interactions. Further, those participants with high trauma experiences but lower levels of unsupportive social interactions were faster to identify the color of the trauma-related words than trauma un-related words (facilitation effect).

In the same fashion, in the trauma condition of the Visual Probe Task, Figure 4 illustrates that those individuals that had encountered high levels of trauma and a high degree of unsupportive social interactions exhibited greater errors when the probe position was the same as that of the previous trial that contained the trauma image compared to: a) those with similar levels of trauma but lower levels of unsupportive social interaction as well as b) those with similar levels of unsupportive social interactions but lower levels of trauma history. Further, as seen in Figure 5 those individuals that had encountered high levels of trauma and a high degree of unsupportive

social interactions also exhibited faster reaction times when the probe position was the same as that of the previous trial that contained the trauma image, compared to those with similar levels of trauma but lower levels of unsupportive social interactions. In contrast, at lower levels of previous trauma, those participants having experienced high unsupportive social interactions exhibited longer reaction times.

In contrast to the effects associated with negative stimuli, trauma and unsupportive social interactions did not explain a significant amount of variance in the error rates or latency bias when participants were exposed to the positive stimuli. This was the case in both the Emotional Stroop Test and the Visual Probe Task, all $R^2 = ns$ (Tables 19 and 20). Taken together, this pattern of findings suggests that trauma history combined with unsupportive social interactions may induce an attention bias toward trauma material, and this is not only attributable to the emotional nature of the images presented.

Table 18

Regression Analysis (Pearson Correlations and Standardized Regression Coefficients)

Assessing the Moderated Relations between Traumatic Experiences, Unsupportive Social Interactions (Predictor Variables) and Memory Recognition Errors (Outcome Variable)¹²

	Memory Recognition Errors		
	<i>R</i>	β	R^2_{total}
Overall Trauma	-.00	-.00	.001
Unsupportive Social Interactions	.03	-.04	
Trauma x Unsupportive Social Interactions	-.03	-.04	.002

¹² In this Table, only the Beta standardized coefficients (β) of the second step are reported

Table 19

*Regression Analysis (Pearson Correlations and Standardized Regression Coefficients) Assessing the Moderated Relations between Traumatic Experiences, Unsupportive Social Interactions (Predictor Variables) and Performance on the Modified Emotional Stroop Test (Outcome Variable)*¹³

	Bias		
	<i>r</i>	β	R^2_{total}
<i>Trauma</i> ¹⁴			
Overall Trauma	.00	-.08	.019
Unsupportive Social Interactions	.14	.09	
Trauma x Unsupportive Social Interactions	.19	.18*	.027*
<i>Positive Condition</i> ¹⁵			
Overall Trauma	-.01	.01	.000
Unsupportive Social Interactions	.00	.03	
Trauma x Unsupportive Social Interactions	-.10	-.11	.010

* $p < .05$

¹³ In this Table, only the Beta standardized coefficients (β) of the second step are reported

¹⁴ As mentioned earlier, performance in this condition corresponds to mean latencies between trauma-related and trauma-unrelated words.

¹⁵ Performance in this condition corresponds to mean latencies between happy words and neutral words.

Table 20

*Regression Analysis (Pearson Correlations and Standardized Regression Coefficients) Assessing the Moderated Relations between Traumatic Experiences, Unsupportive Social Interactions (Predictor Variables) and Performance on the Visual Probe Task (Outcome Variable)*¹⁶

	Errors			Bias		
	<i>R</i>	β	R^2_{total}	<i>R</i>	β	R^2_{total}
<i>Trauma Condition</i>						
Overall Trauma	.24	.18*	.059**	.03	-.01	.002
Unsupportive Social Interactions	.12	.04		-.03	-.07	
Trauma x Unsupportive Social Interactions	.23	.18*	.028*	.16	.18*	.029*
<i>Positive Condition</i>						
Overall Trauma	.13	.12	.017	-.03	.02	.015
Unsupportive Social Interactions	.04	.01		-.12	-.11	
Trauma x Unsupportive Social Interactions	.06	.02	.000	-.08	-.06	.004

Note. * $p < .05$; ** $p < .01$

¹⁶ In this Table, only the Beta standardized coefficients (β) of the second step are reported

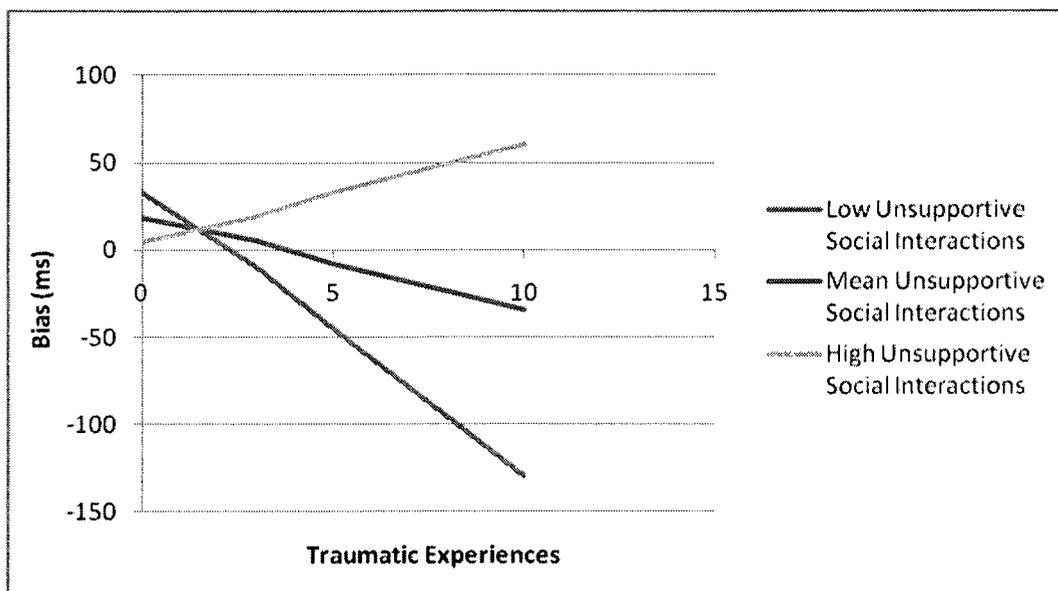


Figure 3. In the Emotional Stroop Test, those individuals that had encountered high levels of trauma and a high degree of unsupportive social interactions took longer to identify trauma-related words than trauma-unrelated words (interference effect) compared to those with both lower levels of prior trauma and unsupportive social interactions. Further, those participants with high trauma experiences but lower levels of unsupportive social interactions were faster to identify the color of the trauma-related words than trauma un-related words (facilitation effect).

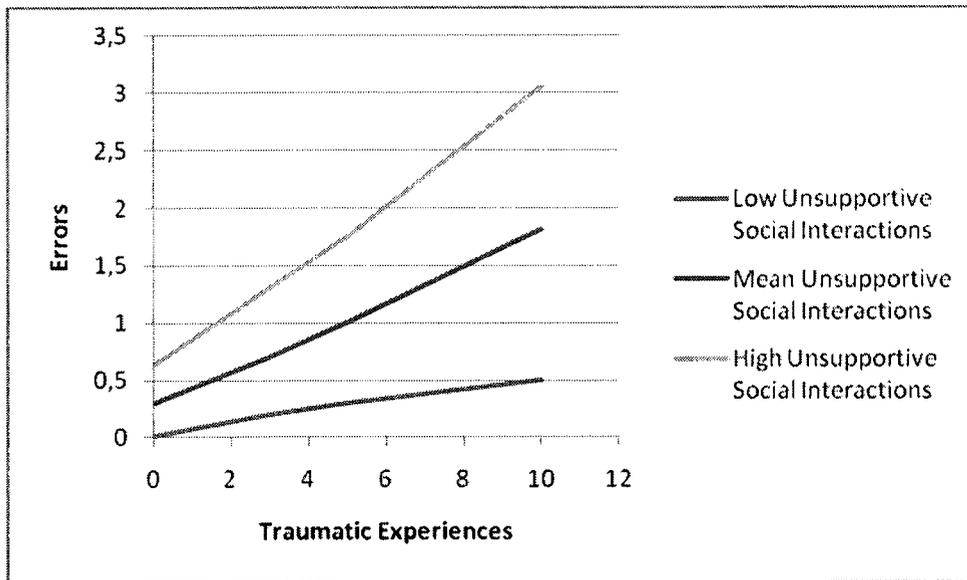


Figure 4. In the trauma condition of the Visual Probe Task, those individuals that had encountered high levels of trauma and a high degree of unsupportive social interactions exhibited greater errors when the probe position was the same as that of the previous trial that contained the trauma image compared to: a) those with similar levels of trauma but lower levels of unsupportive social interaction as well as b) those with similar levels of unsupportive social interactions but lower levels of trauma history.

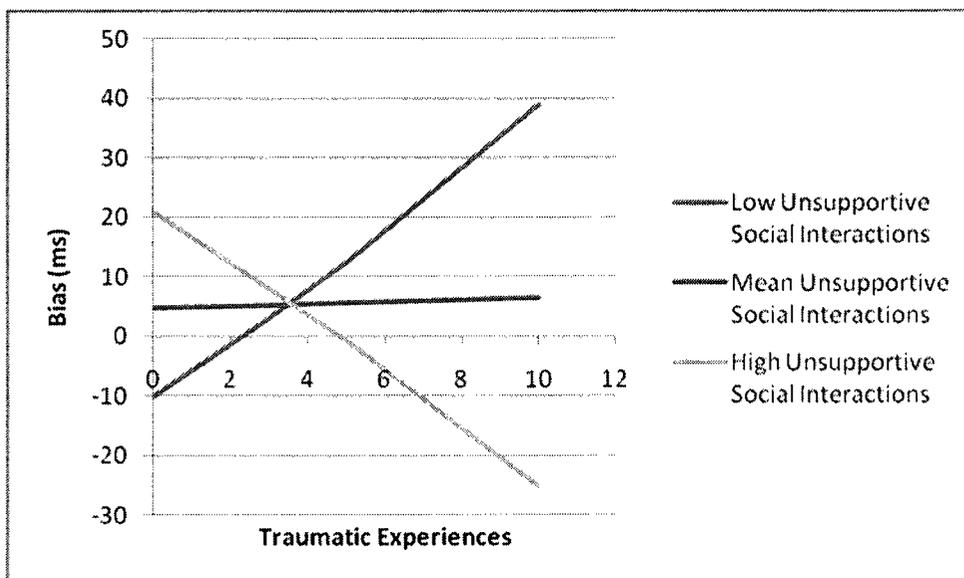


Figure 5. In the trauma condition of the Visual Probe Task, those individuals that had encountered high levels of trauma and a high degree of unsupportive social interactions exhibited faster reaction times when the probe position was the same as that of the previous trial that contained the trauma image compared to those with similar levels of trauma but lower levels of unsupportive social interactions. In contrast, at lower levels of previous trauma, participants having experienced high unsupportive social interactions exhibited longer reaction times.

Discussion

One of the aims of Study 3 was to build on the two previous studies by examining the role of unsupportive social interactions in influencing well-being, emotional distress, cognitive functioning and cortisol reactivity following trauma reminders. In addition to memory processes, this study assessed changes in attention associated with reminders of trauma, as it has been found that traumatic events may induce attention biases toward trauma-related material (Bryant & Harvey, 1995, 1997; Cassiday, McNally & Zeitlin, 1992; Coles & Heimberg, 2002; Foa, Feske, Murdock et al. 1991; Kaspi, McNally & Amir, 1995; McNally, 1997; McNally, Amir & Lipke, 1996; McNally, English, & Lipke, 1993; McNally, Kaspi, Riemann, & Zeitlin, 1990; Moradi, Taghavi, Neshat-Doost, Yule, & Dalgleish, 2000; Paunovic, Lundh & Ost, 2003; Squire & Zola-Morgan, 1991; Thrasher, Dalgleish & Yule, 1994; Vrana, Roodman & Beckman, 1995; Vythilingam et al., 2007; Williams et al., 1996; Zeitlin & McNally, 1991). The present results were consistent with the view that previous trauma experiences influence attention allocation. Moreover, it appeared that unsupportive social interactions during a particularly distressing time in an individual's life not only influenced subsequent psychological well-being, but also interacted with these trauma experiences to influence later attention processes.

Trauma, Unsupportive Social Interactions and Psychological Well-Being

As mentioned earlier, traumatic experiences have been associated with symptoms of depression (e.g., Ducrocq, Vaiva, Ciiencin, Molenda & Bailey, 2001; Heim and Nemeroff, 2001; Laugharne, Lillee & Janca, 2010) and PTSD (e.g., Yehuda, 2002). Further, it had been proposed that exposure to multiple traumatic events might have

cumulative effects on these symptoms (Fincham, Altes, Stein & Seedat, 2009; Suliman, Mkabile, Fincham, Ahmed, Stein & Seidat, 2009), and that the quality of the social support received during these events (and afterward) might be fundamental in determining the ultimate impact of these events on psychological well-being (Anisman & Matheson, 2005; Aspinwall & Taylor, 1997; Kessler, Price, Wortman, 1985; Powers, Ressler, & Bradley, 2009). Consistent with this perspective, the present study indicated that both high levels of trauma history and stressor-specific unsupportive social interactions were independent predictors of depressive and PTSD symptoms. However, contrary to our initial hypothesis, unsupportive social interactions did not moderate the impact of prior trauma on these psychological outcomes.

Although these findings are consistent with the idea that stressor-specific unsupportive social interactions have a detrimental impact on psychological well-being, independent of the effects of trauma experiences (Rook, 1990), the cross-sectional nature of our research design does not allow assumptions to be made about the directionality of these results. While receiving high levels of unsupportive social interactions might contribute to the development of depressive or PTSD symptoms in traumatized individuals, it is also possible that depressive or PTSD symptoms in these individuals intensify social network members' feelings of vulnerability, helplessness, fearfulness and uncertainty about how to respond appropriately, therefore leading to an increase in unsupportive social interactions (Hays, Magee & Chauncey, 1994; Johnston, Stall, & Smith, 1995; Siegel et al., 1997; Wortman & Lehman, 1985).

Alternatively, it is possible that the association between negative social interactions and decreased well-being might be due to a negative cognitive bias (Beck,

1967, 1976; Buckley, Blanchard & Neil, 2000; Moore, 2009; Weber, 2008) or trait negative affectivity (Rook, 1990, 1992; Watson & Clark, 1984). Indeed, such individuals may tend to have a more critical attitude about themselves and others, and to restrict their attention on the negative facets of their experiences (Watson & Clark, 1984). However, consistent with Ingram et al. (1999), this did not appear to be the case in this study as unsupportive social interactions were not associated with increased psychological distress following trauma reminders, which would be likely if the associations observed were due to negative biases or affectivity. Part of this uncertainty could be resolved if the perceptions of unsupportive social interactions were corroborated by social network members. In this regard, it might be useful to obtain behavioural data and reports from network members, understanding, of course, that their perspectives might be influenced by their own experiences.

As one cannot fully dismiss the possibility that retrospective reports of stressor-specific unsupportive social interactions were negatively biased, the present results ought to be considered with a degree of caution. However, this does not imply that the individual's perceptions do not ultimately impact psychological well-being. Indeed, some investigators have considered negative biases as "working models" of these relationships, and that the perceptions associated with these working models may influence adult well-being and psychosocial functioning (see Carnelly, Pietromonaco, & Jaffe, 1994; Elwood, Hahn, Ulatunji & Williams, 2009). Importantly, among individuals with PTSD, negative information processing might even constitute a vulnerability factor to, rather than a consequence of, poor psychological well-being (Bryant & Guthrie, 2005; Moore, 2009).

Trauma, Unsupportive Social Interactions, Distress and Cortisol Reactivity Following Trauma Reminders

Consistent with Studies 1 and 2, high levels of prior trauma predicted increased distress following a reminder of individuals' most distressful experience. However, unsupportive social interactions during that particular event did not predict the distress elicited by remembering the event during the completion of the TLEQ. Although this result might be a bit surprising, it is possible that other factors might have played a substantial role in determining whether or not trauma reminders would still elicit distress. As several individual differences (i.e., early life events, stressor appraisals, and coping strategies endorsed) and characteristics of the stressor itself (i.e., chronicity, severity, controllability, etc.) determine the ultimate impact of stressful events on well-being (Matheson et al., 2005; Michaud et al. 2008), it is possible that these factors also contributed to emotional reactivity following trauma reminders.

Although this trend was shy of significance, unsupportive social interactions tended to moderate the relation between trauma and cortisol reactivity. Specifically, while high unsupportive social interactions were associated with greater cortisol reactivity at lower levels of traumatic experiences, those participants having encountered both high levels of trauma and high unsupportive social interactions tended to have *lower* cortisol reactivity. This result is in line with recent findings that unresolved trauma in abused women, although associated with highest levels of perceived stress, was associated with the most suppressed cortisol reactions following the TSST (Pierrehumbert, et al., 2009). This is also consistent with findings which showed that chronic stressor experiences (e.g. childhood maltreatment, poor relationship functioning)

and PTSD are associated with blunted HPA functioning, as reflected by diminished cortisol reactivity to stressors (Adam & Gunnar, 2001; Boscarino, 1996; Goenjian, et al., 1996; Gurvits et al., 2000; Hart, Gunnar & Cichetti, 1996; Kellner, Baker & Yehuda, 1997; Mason, Giller, Kosten et al., 1986; Pervanidou, 2008; Yehuda, 2002; Yehuda et al., 1993, 1996, 1998; Wang, 1997) and that negative social support might be particularly important in determining whether or not PTSD will subsequently develop. In effect, the present results suggest that the dampened HPA functioning may be the result of multiple (or chronic) traumatic events, coupled with unsupportive social interactions.

Contrary to the results of Study 2, increased cortisol reactivity was not associated with distress or memory impairments. The source for this difference is not immediately evident, although it appeared that participants in the second study exhibited greater distress and experienced more early trauma ($M \pm SD = 0.94 \pm .08$; but not overall trauma; $M \pm SD = 2.76 \pm .20$) compared to those in the third study (early trauma $M \pm SD = 0.39 \pm .07$; overall trauma $M \pm SD = 2.93 \pm 0.18$). Whether the greater cortisol reactivity in the second study was due to a greater number of early life events is uncertain, however, this might be a plausible explanation given that study participants were not different on any of the other variables measured (i.e., age, gender, depressive symptomatology). Indeed, it has been proposed that prior trauma, particularly early life events, alters stressor reactivity by sensitizing neurochemical processes that influence affective states, so that re-exposure to the same stressor (and even to alternate stressors) at a later time, results in the neurochemical changes occurring more readily (Post, 1992). Such sensitization effects have been documented with respect to numerous neurochemical systems,

including monoamines (Anisman, Hayley & Merali, 2003) and corticotropin releasing hormones (Tilders & Schmidt, 1999).

Yet, as studies of stressor evoked neuroendocrine alterations in humans suggest a complex relationship between factors that govern the effects of aversive events (e.g. characteristics of the stressor, stressor appraisal, and environmental factors) and adrenal corticoid release (Michaud et al., 2008), it is possible that the studies differed on other factors known to modulate stressor-evoked cortisol release, which were not measured in the present studies. For instance, it is possible that due to their nature, the most distressful events in the TLEQ reported by participants in the second study might have been more effective in promoting cortisol release than those in the third study. This is a plausible explanation given that post-hoc analyses revealed that the recall of these events induced less distress from participants of the third study. It is also possible that stronger trauma-related reminder cues in another context, notably one that involved social evaluation or narrative processes, might have been necessary and more effective in promoting cortisol release in the third study (Dickerson & Kemeny, 2004; Elzinga et al., 2003).

Attention Bias toward Trauma-Related Material

Given the possibility that an attention bias for objectively threatening stimuli may be common to all individuals, an additional aim of the present study was to examine whether attention biases toward trauma-related material are present in the general population and/or in sub-clinical populations exposed to traumatic events. This was achieved by comparing the cognitive performances of participants on attention tasks using trauma-related, positive or neutral images or words. In this regard, participants'

performances on the trauma condition of both the Emotional Stroop Test and the Visual Probe Task did not differ significantly from the positive or neutral conditions. In effect, these results suggest that participants, irrespective of their trauma history, did not exhibit a specific attention bias toward trauma-related, or a more general bias toward emotionally-based, material. These results are consistent with a recent review suggesting that healthy individuals do not typically show emotional biases (Bar-Haim et al., 2007), and the proposition that biases toward threatening material are reflective of underlying psychopathology (Dagleish, & Power, 2004; Kimble, Kaloupek, Kaufman, & Deldlin, 2000; MacLeod, 2005; Michael, Elhers, & Halligan, 2005; Stanford, Vasterling, Mathias, Contans, & Houston, 2001; Vythilingam et al., 2007).

Attention Bias Toward Trauma-Related Material, Depressive and PTSD Symptoms

While attention bias toward trauma-related material has been widely documented among traumatized individuals diagnosed with PTSD (Bryant & Harvey, 1995, 1997; Cassiday, McNally & Zeitlin, 1992; Coles & Heimberg, 2002; Foa et al. 1991; Kaspi, McNally & Amir, 1995; McNally, 1997; McNally, Amir & Lipke, 1996; McNally, English, & Lipke, 1993; McNally, Kaspi, Riemann, & Zeitlin, 1990; Moradi, Taghavi, Neshat-Doost, Yule, & Dagleish, 2000; Paunovic, Lundh & Ost, 2003; Squire & Zola-Morgan, 1991; Thrasher, Dagleish & Yule, 1994; Vrana, Roodman & Beckman, 1995; Vythilingam et al, 2007; Williams et al., 1996; Zeitlin & McNally, 1991), attention performances in the present study were not associated with PTSD symptoms. The reason why PTSD symptoms were not associated with attention bias toward trauma-related material remains to be elucidated. On the one hand, it is possible that trauma words or images may not have been threatening enough to alter attention orientation and produce

interference. This study did not evaluate the degree of threat or meaning of the words and images to participants. As a consequence, it is not known how threatening the trauma-related words were to subjects. It is possible that PTSD symptoms were not associated with altered attention processes due to the absence of highly threatening stimuli. Indeed, in contrast to existing literature, the stimuli used in this study did not intend to be trauma-specific, and therefore, may not have been adequately threatening. The selection of general trauma related words and images was motivated by the fact that participants had experienced heterogeneous types of trauma, and not only one type of trauma, as in other studies. On the other hand, it is also possible that our sample did not suffer from “clinical” levels of PTSD symptoms, which, as mentioned earlier, might be necessary to be associated with attention biases toward threatening material (Dagleish, & Power, 2004; Kimble, Kaloupek, Kaufman, & Deldlin, 2000; Stanford, Vasterling, Mathias, Contans, & Houston, 2001; MacLeod, 2005; Michael, Elhers, & Halligan, 2005; Vythilingam et al., 2007).

However, depressive symptoms were associated with delayed responses to the color naming of trauma-related words in the trauma condition of the Emotional Stroop Test. This is in line with previous studies demonstrating that attention biases on this task are common among depressive individuals (Bradley et al., 1995; Broomfield, Davies, MacMahon, Ali, Cross, 2007; Kerr, Scoot, & Phillips, 2005; McNeely, Lau, Cristensen, & Alain, 2008; Mitterschiffthaler, et al., 2008; Williams et al., 1996). Why such an association between depressive symptoms and attention biases was not also found in the Visual Probe Task remains to be elucidated, as the bias is usually more clear-cut on this measure than the modified Stroop measure (Dagleish, et al., 2003). Indeed, previous

studies using facial expressions as stimuli in the Visual Probe Task have found that depressed individuals exhibited biases toward angry and sad faces, as compared to healthy controls (Gotlib, Krasnoperova, Yue, & Joorman, 2004; Joorman & Gotlib, 2006, 2007; Lemouth, Joorman, Sherdell, Wright & Gotlib, 2009; Lapanen, 2006). This suggests the possibility that the discrepancies between these findings and those in our study may lie in the nature of the stimuli themselves. Indeed, it is likely that facial expressions interfere more with attention processes than the general trauma images used in this study. It has to be considered that these images may not have been sufficiently attention grabbing compared to facial expressions, which have been proposed to be evolutionarily prepared (e.g., Ohman & Mineka, 2001) and may have become engraved in our brains, and which activate emotional processing modes even at weak strengths or short durations. Further, it is important to consider that although the Emotional Stroop Test and the Visual Probe Task assess attention processes, they are not capturing the same stages of attention. While the Visual Probe Task assesses the influence of trauma images on attention orientation, the Emotional Stroop Test assesses the interfering effect of trauma on a concurrently performed non-emotional task. In addition, even if a failure of response inhibition or disengagement was seen in both tasks (Lezak, Howieson, Lorring, Hannay & Fischer, 2004), it is important to note that the type of disengagement is somewhat different in each task. In the Visual Probe Task, attention has to be shifted from the location of a trauma image to the location of a dot probe that is *presented subsequent to the trauma image*, whereas in the Emotional Stroop Test, attention has to be shifted from the meaning of the word to the color of the ink in which it is printed *on the same trial*. Therefore, it is possible that executive functions might be more implicated in the Stroop

Test compared than the Visual Probe Task, which are especially impaired among depressive individuals (for a review see Castadena, Tuulio-Henriksson, Marttunen, Suvisaari, & Lonnqvist, 2008).

Trauma, Unsupportive Social Interactions and Attention Bias Toward Trauma-Related Material

Interestingly, however, it was found in the present study that the interaction between the number of trauma experiences and unsupportive social interactions during the most distressful event predicted participant performance in the trauma conditions of the Visual Probe Task and the Emotional Stroop Test. In particular, it was found that participants having both high levels of prior trauma and unsupportive social interactions made fewer errors and exhibited faster reaction time to identify the position of the probe on the Visual Probe Task when the position of the probe was concordant with the trauma image, and also took longer to identify the color of trauma-related words than neutral words. These results are consistent with our initial hypothesis that these individuals would exhibit greater attention bias toward trauma-related material. Further, as these results were not found in the positive conditions of the tasks, these results suggest that these biases occur for only threatening stimuli (Bryant & Harvey, 1995; Foa et al., 1991; McNally, 1990), and not emotional stimuli more generally (Cassiday et al., 1992; Martin et al., 1991; Paunovic et al., 2002).

These results also suggest that particular conditions, such as the combination of accumulated trauma exposure and poor social support during stressful experiences, might be potent enough to induce attention biases in individuals who have experienced significant trauma, even though they are not currently exhibiting symptoms of PTSD. In

this regard, attention biases to threat were found in victims of sexual abuse regardless of whether or not they suffered from PTSD (Freeman & Beck, 2000). The following is an explanation of the possible mechanisms involved: According to the cognitive model of emotional attention (Blair et al., 2005; Mitchell et al., 2006; Pessoa & Ungerleider, 2004), the increased attention bias toward threat is a consequence of increased emotional responsiveness to threatening stimuli, which leads to greater priming of the representations of emotional information in the temporal cortex near the amygdala. Consequently, this line of thinking predicts that these emotionally laden representations will compete with the stimulus presented in the task and produce increased interference, which is exhibited through attention biases. In short, the current results are consistent with the idea that participants having both experienced high levels of trauma and unsupportive social interactions might be more emotionally reactive to threatening information, which in turn increases attention towards potential threat.

Taken together, performance in the trauma conditions of the Visual Probe Task and the Emotional Stroop Test suggest that individuals that experienced both high trauma and unsupportive social interactions might perceive trauma-relevant information more rapidly and efficiently than trauma-unrelated or neutral information. In addition, it is noteworthy that these participants exhibited greater attention orientation toward trauma images of the Visual Probe Task, regardless of whether they were related to their previous trauma. These results, consistent with previous studies, suggest that the impact of the trauma (Armony et al., 2005; Rauch et al., 2000) and unsupportive social interactions on attention orientation may generalize to other threatening stimuli, and not necessarily remain specific to only those stimuli which were part of the original trauma.

Memory Functioning

Since altered attention processes might also influence memory performance (e.g., Kandell et al., 2001), it was of particular interest in this study to assess whether memory functioning was associated with attention biases towards trauma-related material. In this regard, this study did not find an association between attention biases toward trauma related material and memory recognition. Once again, similar to the first and second studies, only increased distress following trauma reminders was associated with greater impairments on the memory recognition task. Further, consistent with the first and second studies, participants exposed to trauma-related images tended to make more recognition errors than those exposed to trauma-unrelated images. However, this trend was shy of significance, probably due to the small number of individuals that were exposed to trauma unrelated images ($n = 13$) relative to those exposed to trauma related images ($n = 67$), thus causing largely heterogeneous variances and a lack of power ($P = .136$).

Limitations and Conclusions

There are several limitations to the present study. As indicated earlier, the cross-sectional nature of the data collected does not allow any inferences about causality. Further, all measures were self-report, and it remains to be determined whether these reports could be corroborated by members of the social network. The generalization of the results of this study is also limited. First, the distribution of sex was largely unequal; most of the subjects were female. Second, the sample was very heterogeneous with respect to the type of trauma experiences previously encountered. This diversity of trauma experiences might also have obscured interactive effects of trauma experiences

and unsupportive social interactions on psychological well-being or cognitive function. For example, it is possible that stressor-specific unsupportive social interactions have stress-amplifying effects, but only for certain kinds of traumatic events. Future studies might be able to examine more closely these potential interactive effects by focusing on a particular trauma, such as assault, car accident or the death of a loved one, instead of multiple diverse traumas.

In addition, it would have been useful to include clinical groups of subjects suffering from PTSD, as well as anxiety disorders without PTSD, in order to investigate whether attention and memory processes would be disrupted in the research paradigms used in the present study. Indeed, it has been suggested that attention disruptions might be a cognitive marker of psychopathology rather than the result of trauma exposure (Kimble et al., 2000; Michael et al., 2005; Stanford et al., 2001; Vythilingam et al., 2007), but this is a controversial suggestion, as Freeman & Beck (2000) showed the presence of attention biases toward threatening stimuli in abused women regardless of whether they suffered from PTSD.

It remains to be determined whether PTSD and anxious individuals reports similar levels of unsupportive social interactions as this might be an indication of negative cognitive bias, or alternatively of whether the moderating effect of unsupportive social interactions on the relation between trauma history and attention biases toward trauma material is more pronounced in these samples.

Notwithstanding these limitations, this study emphasizes the importance of unsupportive social interactions in determining the ultimate impact of traumatic experiences not only on well-being (Ingram et al., 2001; Mindes et al., 2003; Reynolds &

Perrin, 2004; Rook, 1984; Schrimshaw, 2003), but also on attention processes. While both trauma experiences and unsupportive social interactions independently predicted depressive and PTSD symptoms, it appeared in this study that high levels of prior trauma had to be combined with high levels of unsupportive social interactions to disrupt attention allocation toward trauma-related material.

GENERAL DISCUSSION

At one time or another, most individuals encounter stressful events that challenge their psychological and/or physiological well-being. Today, the word “trauma” covers a wide variety of stressful events, including severe events, such as naturalistic stressors (earthquakes, hurricanes, fire-storms, ice-storms) that concurrently affect great numbers of individuals, as well as those that affect single individuals, such as physical and psychological experiences (car accidents, assault). In the wake of such events, traumatized individuals may not only develop depression and PTSD (Yehuda, 2002), but they may also suffer from cognitive disturbances, particularly with respect to memory and attention processes (Golier & Yehuda, 2002; Johnsen & Asjornsen, 2008; Stein, Kennedy, & Twanley, 2002; Vedhara, Hyde, Gilchrist, Tytherleigh, & Plummer, 2000).

The vast majority of findings concerning the impact of traumatic experiences on memory functioning have emerged from individuals suffering from PTSD. However, there is no reason to assume that traumatic experiences would not influence memory processes in traumatized or severely stressed individuals who do not present with clinical levels of PTSD. In this regard, the three studies of the present investigation demonstrated that increased emotional arousal associated with the most distressful event experienced was related to decreased memory recognition. In particular, as depicted in Table 21, and as observed previously (Iezzi, Duckworth, Vuong, Archibald, & Klinck, 2004; Munoz & Esteve, 2005; Shilling & Jenkins, 2007; Stawski, Sliwinski, & Smyth, 2006; Woods, Iudicello, Moran, Carey, Dawson, & Grant, 2008), participants who expressed distress when asked to recall past adverse experiences (through the use of the TLEQ) exhibited greater memory recognition impairments. However, the particular mechanisms by which

trauma reminders elicit distress and the way that emotional arousal influences memory processes remain to be elucidated.

Consistent with the view that trauma reminders, especially exposure to trauma-related material, might activate intrusive memories that interfere with performance on a test of memory, Study 1 indicated that memory functioning was altered by exposure to trauma-related stimuli in, but this trend was shy of significance in Studies 2 and 3. It is certainly possible that weak and inconsistent effects observed across studies might be due to the possibility that university students are particularly highly functioning or “cognitively resilient” compared to other (e.g., older) populations or those with PTSD (Twanley, Hami, & Stein, 2004).

Table 21.

Associations of Trauma Reminders and Memory Recognition Across the Three Studies

	Associations with Memory Recognition		
	Study 1	Study 2	Study 3
Distress (TLEQ)	↓	↓	↓
Trauma-related Images	↓	Trend, ↓	Trend, ↓
Cortisol	N/A	↓	<i>Ns</i>

In addition, it is also possible that different types of memory or attention measurement might have yielded more robust results. Indeed, it has been shown that

stressors and trauma experiences may have relatively weak effects on visual memory. As such, assessing this form of memory assessment might have yielded less stressor effects that were less pronounced compared to effects on working (Luethi, Meier & Sandi, 2008), verbal (Johnsen, & Asjornsen, 2008), autobiographical (Harvey et al., 1998; Hauer et al., 2008; Kleim & Ehlers, 2008; McNally et al., 1995; Moore & Zoellner, 2007; Schonfeld & Ehlers, 2006; Williams et al., 2007) and social memory (i.e. socially relevant information) (Mercz, Wolf, & Henning, 2010).

It has been reported that in some populations (e.g., in association with aging), memory functioning may be influenced, in part, by the cortisol levels, and it is possible that cortisol release elicited by stressors might contribute to memory disturbances elicited by trauma reminders (Alderson & Novack, 2002; Heffelfinger & Newcomer, 2001; Het et al., 2005; Lupien & Lepage, 2001; Lupien et al., 2007; Wolf, 2003). Consistent with this proposition, Study 2 indicated that both high cortisol levels and high distress evoked by remembering a previous traumatic event independently predicted higher levels of memory impairments. Although distress and cortisol levels were associated with each other, cortisol did not significantly mediate the relationship between distress and memory performance. Thus, cortisol may be an index of distress associated with the reminder stimuli, but likely did not, in itself, contribute to memory impairment. However, the relation between cortisol and memory performance evident in Study 2 was not replicated in the third study. It may well be the case that different individual attributes (e.g. characteristics of the stressor, stressor appraisal, and environmental factors) between Study 2 and 3 might have buffered or exacerbated the effect of trauma reminders on this neuroendocrine outcome (Dickerson & Kemeny, 2004; Michaud et al., 2008) and on

memory functioning. This also leads to the possibility that trauma reminders in Study 3 were simply insufficiently intense to impair memory functioning or elicit significant cortisol release. It is possible that the use of more potent reminders (i.e., make narratives more salient; use more trauma-specific associated images) or focusing on one particular negative life event, such as bereavement, chronic or acute illness or assault might have yielded different results. Indeed, given that the present studies involved general trauma images and words in an effort to cover the spectrum of traumatic experiences assessed in the TLEQ, these stimuli were not sufficiently specific to elicit intrusive memories that might have impaired performance. In retrospect, it would be particularly relevant to have participants rate the meaningfulness of trauma cues and to determine whether it moderates the impact of trauma experiences on cognitive function and cortisol reactivity. Likewise, the relevance of these cues was not rated by other subjects (Lavy et al., 1994; Unoki et al., 1999) or experts (Moritz, Jacobsen et al., 2004).

In the present studies, we were interested in the neuroendocrine response elicited by trauma reminders, and focused on the absolute changes in cortisol levels at a particular point in time. Inasmuch as cortisol may influence memory processes (e.g., Lupien & Lepage, 2001), particularly if cortisol variations were sustained (McEwen, 2001; 2006), it would have been particularly relevant to assess the diurnal variations of cortisol associated with trauma in relation to memory and attention processes. As indicated earlier, cortisol release ordinarily follows a well-defined diurnal rhythm. Specifically, in humans, glucocorticoid release, already high at awakening, rises about 40-60% over the first 30-60 minutes following awakening (irrespective of wake time), and decreases slowly thereafter (Linkowski et al., 1993; Schmidt-Reinwald et al., 1999). Although this

pattern appears to be independent of age, time of awakening, quality of sleep, physical activity or morning routine, factors including gender, use of oral contraceptives and stressor experiences may influence free cortisol levels (Schmidt-Reinwald et al., 1999). Among individuals with elevated life stress the rise of morning cortisol is particularly notable, and it has been suggested that the 30-minute period after awakening may be unique in detecting stressor-related cortisol variations.

Importantly, variations of diurnal cortisol rhythmicity, compared to acute stressor-induced changes, might provide more insight about the neuroendocrine functioning associated with stressor-related pathological states. In contrast to the effects of moderate acute stressors, there is reason to believe that traumatic events (or chronic stressor experiences) may be associated with the blunting of the diurnal cortisol curve (Aardal-Eriksson et al., 2001; Abercrombie, et al., 2004; Lauc, Zvonar, Vuksic & Flogel, 2004; Pico-Alfonzo, Garcia-Linares, Celda-Navarro, Herbert & Martinez, 2004; Pruessner, Hellhammer & Kirschbaum, 1999). It might be expected that memory and attention would be most impaired among individuals who exhibit the greatest cortisol rise, or perhaps in those individuals that show abnormal rhythms, such as patients with PTSD who tend to display a flattened diurnal profile (Bremner et al., 2003; Gunnar-Vasquez, 2001; Suglia, Staudenmayer, Cohen, & Wright, 2010; Yehuda, Golier, & Kaufman, 2005), and especially among those individuals who also experienced unsupportive social interactions when they encountered adverse events. Further to this, a stressor challenge might also have more dramatic effects among individuals where elevated cortisol levels are sustained (e.g., in the elderly).

It has been suggested that the quality of the social support received by traumatized individuals may be fundamental in determining the development of psychopathology (Brewin et al., 2000; Charuvastra & Cloitre, 2008; Sarason et al., 1990). Consistent with previous studies showing an association between unsupportive social interactions on psychological adjustment and well-being (Ingram et al., 2001; Mindes et al., 2003; Reynolds & Perrin, 2004; Rook, 1984; Schrimshaw, 2003), Study 3 found that unsupportive social interactions during a traumatic event could be associated with increased depressive and PTSD symptoms. The mechanism by which stressor-specific unsupportive social interactions contribute to increased adverse psychological symptoms remains to be elucidated. For instance, it is possible that unsupportive social interactions would lead to increased depressive and PTSD symptoms by promoting greater use of avoidant coping (Lepore et al., 1996; Lepore & Helgeson, 1998), or by feeling greater levels of shame and increased indulgence in self-critical thinking (Harman & Lee, 2010). It is also possible that a person may feel more powerless and helpless during a traumatic event if a member of their social network reacts negatively by blaming, minimizing, distancing or bumbling, feelings which in turn, may promote the development of depression and PTSD (Abramson, Seligman, & Teasdale, 1978; Abramson, Metalsky & Alloy, 1989; Alloy, Abramson, Metalsky & Hartalge, 1988; APA, 1994; Overmier, 2002; Scher, & Resick, 2005).

The present investigation did not only examine the association between unsupportive social interactions during the most distressing traumatic event experienced and well-being, but also considered its influence on cognitive functioning and cortisol reactivity. In this regard, it was found that although trauma and unsupportive social

interactions did not by themselves predict memory and attention functioning or cortisol reactivity, they interacted to influence attention bias toward trauma-related material. Specifically, it was found that those individuals that experienced both high levels of trauma and unsupportive social interactions exhibited greater bias toward trauma-related material. It might be the case that such a bias would be particularly adaptive for individuals without quality support from other members of their network as this would allow them to “spot” trauma cues more easily and hence take appropriate defensive responses.

The present investigation primarily measured selective attention, but it might have been profitable to assess the role of unsupportive social interactions on the performance of traumatized individuals in sustained attention tasks that involved repeated exposure to threatening stimuli. Although there is some evidence available suggesting that PTSD is associated with deficits in sustained attention, the majority of studies used general measures such as the Paced Auditory Serial Addition Task (PASAT) and Continuous Performance Tests (CPT) (Jenkins et al., 2000; Koso & Hansen, 2006; Meewise, Nidjam, De Vries, et al., 2005; Stein, Kennedy & Twanley, 2002; Toomey, Alpern, Vasterling et al., 2009; Vasterling et al., 1998; 2002; Yehuda et al., 1995b). Sustained attention tasks involving threatening or trauma-related stimuli may be more reflective of traumatized individuals' daily experiences of vigilance in environments rich in threat cues, and thus may offer more applicable results.

Although most of the research in this domain, including the present investigation, has focused on the individual's perceptions of the quality and nature of support received by members of their social networks, it remains necessary to corroborate and assess the

reliability of these perceptions by exploring the helpers' perceptions of the support they gave to their friend. Retrospective studies are, of course, fraught with biases and one cannot fully dismiss the possibility that a negative view of interactions with others might be reflective of negative affectivity commonly seen in traumatized individuals, rather than a true representation of the actual help received. Further, it remains to be determined how social network members' unsupportive interactions vary over time and which factors, such as certain personality (i.e., self-esteem) or stressor characteristics, determine the type of unsupportive social interactions chosen.

Finally, the possibility remains to be considered that stronger effects of unsupportive social interactions could have been observed by focusing on particular negative life events. The diversity of stressful events assessed in the present research might not only have influenced the "meaningfulness" of trauma reminders, but may also have obscured the effects of trauma, unsupportive social interactions, and their interactive effects on the various physiological, psychological and cognitive outcomes assessed. For example, it is possible that stressor-specific unsupportive social interactions have stress-amplifying effects, but only for some kinds of stressful events (e.g., those involving interpersonal as opposed to intrapersonal stressors). By studying a sample of individuals who have experienced the same stressful life event, such as assaultive experiences, it might be possible to assess the potential effects of trauma reliably.

Limitations and Direction of Future Studies

Several limitations of the present research should be noted. First, this study involved a relatively restricted sample that limits the generalizability of these findings.

Indeed, the majority of participants were female, relatively young, and from an advantaged educational background (i.e. they were all university students). It is possible, as indicated earlier, that our participants were generally cognitively resilient in the face of adversity compared to other groups of traumatized individuals (e.g., non-university populations). By example, one could imagine that recall of adverse events might have more profound effects among the elderly whose memory functioning may be diminishing. In effect, the present findings may not be generalizable to more traumatized or less resilient populations or among individuals at high risk for showing impairments.

It is possible that symptom levels and duration or intensity of trauma exposure in our participants were, on average, less severe than those diagnosed with PTSD, thus leading to a lower likelihood of cognitive dysfunction. Further, it is also possible that traumatized individuals had remained particularly distressed about their previous traumatic experiences, and might have been more prone to avoid experiments that involved a stressful component.

Notwithstanding these limitations, this research points to novel and fruitful directions for research particularly in the context of the role that unsupportive social interactions play in determining the outcome of trauma experiences on subsequent well-being and cognitive functioning. This would necessarily entail the use of prospective studies that might facilitate (a) the identification of causal relationships between stressful events, unsupportive social interactions and negative psychological and physiological outcomes (e.g. PTSD), (b) assessment of the stability of these interactions over time, and (c) evaluation of the evolution of attention biases and memory changes over time (Bradley, Mogg et al, 1999).

Implications of this Research

Consistent with previous research indicating that unsupportive social interactions may be particularly detrimental to psychological well-being during a stressful encounter (Ingram et al., 2001; Mindes et al., 2003; Reynolds & Perrin, 2004; Rook, 1984; Schrimshaw, 2003), the results of the present investigation suggest that those having more unsupportive social interactions might be disadvantaged when dealing with traumatic events. These detrimental effects may not only be observable with respect to psychological well-being, but may also extend to attention processes. From a clinical perspective, these findings highlight the importance of investigating not only the availability of social resources, but also the quality of the social interactions offered by the social network. Information about unsupportive social interactions and their potential impact on well-being could be included in prevention and intervention programmes for traumatized individuals. For instance, cognitive, affective and behavioural strategies, such as specific communication skills for responding to unsupportive social interactions, could be given to traumatized individuals. Not only should health professional promote the use of social support, they must encourage traumatized individuals to seek “good” support from others. Further, it might also be relevant to raise significant others’ awareness about the potential impact of their interventions, thus facilitating the use of more positive social interaction strategies in daily situations.

Across studies, altered memory functioning was consistently associated with increased distress following trauma reminders and tended to be associated with exposure

to trauma-related images. While some of these findings have implications for the ruminative and intrusive sequelae of traumatic events that feed into trauma symptoms and PTSD, inconsistencies between the studies highlight the dynamic nature of the effects of trauma reminders on cortisol and cognitive processes. One major contribution of this preliminary research stems from its integrative perspective, namely the simultaneous inclusion and exploration of several variables (psychological, interpersonal, biological and cognitive). In the recent years, social psychological research involving biological processes has tended to focus either on the link between psychosocial variables and the activity in specific regions in the brain (Lieberman, 2007; Ochsner, 2004), or on the link between psychosocial variables and common stress-related processes such as immune or endocrine reactivity (i.e., psychoneuroendocrinology or psychoneuroimmunology; Kemeny, 2009; Kudielka, Hellhammer & Wust, 2009). The present research extended these findings by focusing on cognitive processes. Additional controlled studies with clinical populations are required to obtain a more detailed understanding of the processes involved in the relationships between trauma experiences and cognitive functioning.

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Appendix A

Description of DSM-IV Diagnostic Criteria for PTSD

A. The person has been exposed to a traumatic event in which both of the following have been present:

1. The person has experienced, witnessed or been confronted with an event or events that involved actual or threatened death or serious injury, or the threat of the physical integrity of oneself or others.
2. The person's response involved intense fear, helplessness and horror. Note: In children, this may be expressed as disorganized or agitated behaviour.

B. The traumatic event is persistently re-experienced in at least one of the following:

1. Recurrence and distressing recollections of the event, including images, thoughts, or perceptions. Note: in young children, repetitive play may appear which themes or aspects are expressed.
2. Recurrent distressing dreams of the event. Note: in children, there may be frightening dreams without recognizable content.
3. Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur upon awakening or when intoxicated). Note; in young children, trauma-specific re-enactment may occur.
4. Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of the general responsiveness (not present before the trauma), as indicated by at least three of the following:

1. Efforts to avoid thoughts, feelings, or conversations associated with the trauma
2. Efforts to avoid activities, places or people that arouse recollections of the trauma
3. Inability to recall an important aspect of the trauma
4. Markedly diminished interest or participation in significant activities
5. Feeling of detachment or estrangement from others
6. Restricted range of affect (e.g. unable to have love feelings)
7. Sense of foreshortened future (e.g. does not expect to have a career, marriage, children, or a normal life span).

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by at least two of the following:

1. Difficulty of falling or staying asleep
2. Irritability or outbursts of anger
3. Difficulty concentrating
4. Hypervigilance
5. Exaggerated startle response

E. Duration of the disturbance (symptoms B, C and D) is more than one month

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important area of functioning.

Specify if: Acute: if duration of symptoms is less than three months

Specify if: With delayed onset: onset of symptoms is at least six months after the stressor.

Appendix B

SONA Recruitment Notice (Study 1)

Images of emotional events and reactivity in relation to memory, hormone and immune changes

This study involves seeing images of emotional events and providing your reactions to such images. You will be randomly be exposed to *either* neutral images or potentially distressing events such as scenes of violence. You will also complete questionnaires regarding your personal history and well-being, as well as a series of questions that asks you about potentially traumatic events that you might have experienced (e.g. loss of a loved one, abuse, war, accident, etc.) and when these events occurred. Some individuals may experience discomfort or distress when viewing or recalling a stressful experience. This study will take about 15-20 min, and you will be given 0.5% grade increase for your participation.

Appendix C

Informed Consent Form (Study 1)

The purpose of an informed consent is to ensure that you understand the purpose of the study and the nature of your involvement. The informed consent has to provide sufficient information such that you have the opportunity to determine whether you wish to participate in the study.

Pilot Study of the study untitled “Images of emotional events and reactivity in relation to memory, hormone and immune changes”.

Research Personnel: The following people are involved in this research and may be contacted at any time if you have any questions about the project, what it means, or concerns about how it was conducted:

Dr. Kim Matheson (Faculty Investigator, 520-2648)

Dr. Hymie Anisman (Faculty Investigator, 520-2699)

Kathy Michaud (Researcher, 520-2600 ext. 7513)

If you have any ethical concerns about how this study please contact Dr. J. Mantler (Chair of the Carleton University Research Ethics Committee for Psychological Research, 520-2600, ext 4173) or Dr. M. Gick, Chair of the Department of Psychology, Carleton University, 520-2648.

Purpose, Task Requirements and Time for Participation: In this short study, you’ll be we will be asking you to fill out a few questions about yourself (e.g. gender, age), your well-being as well as a series of questions that asks you about potentially traumatic events (e.g. loss of a loved one, abuse, war, accident, etc.) that you might have experienced and when these occurred. Once those questionnaires are completed, we will be asking you to complete a task that requires you to see a series of images and indicate your emotions and reactions to these images. Some people will be randomly assigned to a condition that includes pictures of distressing scenes such as assault, war violence or loss of a loved one. Given the nature of the questions and pictures and the fact that they may evoke distress for some individuals, remember that your participation is completely voluntary and that you may withdraw from the study at anytime. This will take approximately 10-15 minutes to complete, and you will receive 0.5 % grade increase or M&M’s candies for your participation.

Potential Risk and Discomfort: There are no physical risks in this study. Some individuals may experience discomfort when asked to respond to personal, sensitive questions, or recalling or viewing a stressful experience.

Right to Withdraw: Your participation in this study is entirely voluntary. At any point during the study you have the right to choose to withdraw entirely without penalty. Likewise, even after you’ve provided answers, should you change your mind and want your answers destroyed, you need only call us or drop by and indicate your preference.

Anonymity/Confidentiality: The data collected in this study will be kept anonymous and confidential. Your informed consent form will be separated from your data collected and kept in a separate and secured file by one of the research investigators. No individual results will be made available – all results will be reported in aggregate

I have read the above description of the study concerning images of emotional events and reactivity. The data collected will be used in research publications and/or for teaching purposes. My signature indicates that I agree to participate in the study, and this in no way constitutes a waiver of my rights.

Full Name (please print): _____

Participant _____ Researcher: _____

Date: _____ Date: _____

Appendix D

Measures (Study 1)

BACKGROUND

Age _____

Sex _____

Ethnic/racial background _____

Marital status _____

BECK DEPRESSION INVENTORY

On this questionnaire are groups of statements. Please read the entire group of statements of each category. Then pick out ONE statement in that group which best describes the way you feel. Check off the number beside the statement you have chosen.

1. 0 = I do not feel sad
 1 = I feel sad or blue
 2a = I am blue or sad all of the time and I can't snap out of it
 2b = I am so sad or unhappy that it is very painful
 3 = I am so sad or unhappy that I can't stand it

2. 0 = I am not particularly pessimistic or discouraged about the future
 1 = I feel discouraged about the future
 2a = I feel I have nothing to look forward to
 2b = I feel I won't every get over my troubles
 3 = I feel that the future is hopeless and things cannot improve

3. 0 = I do not feel like a failure
 1 = I feel I have failed more than the average person
 2a = I feel I have accomplished very little that is worthwhile or that means anything
 2b = As I look back on my life, all I can see is a lot of failures
 3 = I feel I am a complete failure as a person

4. 0 = I am not particularly dissatisfied
 1a = I feel bored most of the time
 1b = I don't enjoy things the way I used to
 2 = I don't get satisfaction out of anything anymore
 3 = I am dissatisfied with everything

5. 0 = I don't feel particularly guilty
 1 = I feel bad or unworthy a good part of the time
 2a = I feel quite guilty
 2b = I feel bad or unworthy practically of the time now
 3 = I feel as though I am very bad or worthless

6. 0 = I don't feel I am being punished
 1 = I have a feeling that something bad may happen to me
 2 = I feel I am being punished or will be punished
 3a = I feel I deserve to be punished
 3b = I want to be punished

7. 0 = I don't feel disappointed in myself
 1a = I am disappointed in myself
 1b = I don't like myself
 2 = I am disgusted with myself
 3 = I hate myself

8. 0 = I do not feel I am any worse than anybody else
 1 = I am very critical of myself for my weaknesses or mistakes
 2a = I blame myself for everything that goes wrong
 2b = I feel I have many bad faults

- ___ 0 = I don't have thoughts of harming myself
 ___ 1 = I have thoughts of harming myself but I would not carry them out
 ___ 2a = I feel I would be better off dead
 ___ 2b = I have definite plans about committing suicide
 ___ 2c = I feel my family would be better off if I were dead
 ___ 3 = I would kill myself if I could
10. ___ 0 = I don't cry anymore than usual
 ___ 1 = I cry more now than I used to
 ___ 2 = I cry all the time now. I can't stop it
 ___ 3 = I used to be able to cry but now I can't cry at all even though I want to
12. ___ 0 = I am no more irritated now than I ever am
 ___ 1 = I get annoyed or irritated more easily than I used to
 ___ 2 = I get irritated all the time
 ___ 3 = I don't get irritated at all the things that used to irritate me.
12. ___ 0 = I have not lost interest in other people
 ___ 1 = I am less interested in other people than I used to be
 ___ 2 = I have lost most of my interest in other people and I have little feeling for them
 ___ 3 = I have lost all my interest in other people and don't care about them at all
13. ___ 0 = I make decisions about as well as ever
 ___ 1 = I am less sure of myself now and try to put off making decisions
 ___ 2 = I can't make decisions anymore without help
 ___ 3 = I can't make decisions at all anymore
14. ___ 0 = I don't feel I look any worse than I used to
 ___ 1 = I am worried that I am looking old or unattractive
 ___ 2 = I feel that there permanent changes in my appearance and they make me look unattractive
 ___ 3 = I feel that I am ugly or repulsive looking
15. ___ 0 = I can work about as well as before
 ___ 1a = It takes extra effort to get started at doing something
 ___ 1b = I don't work as well as I used to
 ___ 2 = I have to push myself very hard to do anything
 ___ 3 = I can't do any work at all
16. ___ 0 = I can sleep as well as usual
 ___ 1 = I wake up more tired in the morning than I used to
 ___ 2 = I wake up 1-2 hours earlier than usual and find it hard to get back to sleep
 ___ 3 = I wake up early every day and can't get more than 5 hours sleep
17. ___ 0 = I don't get anymore tired than usual
 ___ 1 = I get tired more easily than I used to
 ___ 2 = I get tired from doing anything
 ___ 3 = I get too tired to do anything
18. ___ 0 = My appetite is no worse than usual
 ___ 1 = My appetite is not as good as it used to be
 ___ 2 = My appetite is much worse now

- ___ 3 = I have no appetite at all any more
19. ___ 0 = I haven't lost much weight, if any, lately
___ 1 = I have lost more than 5 pounds
___ 2 = I have lost more than 10 pounds
___ 3 = I have lost more than 15 pounds
20. ___ 0 = I am no more concerned about my health than usual
___ 1 = I am concerned about aches and pains or upset stomach or constipation or other unpleasant feelings in my body
___ 2 = I am so concerned with how I feel or what I feel that it's hard to think of much else
___ 3 = I am completely absorbed in what I feel
21. ___ 0 = I have not noticed any recent change in my interest in sex
___ 1 = I am less interested in sex than I used to be
___ 2 = I am much less interested in sex now
___ 3 = I have lost interest in sex completely

LIFE EVENTS QUESTIONNAIRE

The purpose of this questionnaire is to identify significant life experiences in one's life. The events listed below are far more common than many people realize. Please read each question carefully and circle the answers that best describe your experience.

1. Have you ever experienced a natural disaster (a flood, hurricane, earthquake, etc.)?

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago
 _____ 10-15 years ago; _____ when you were less than 5
 years old

Did you experience fear, helplessness, or horror at what happened? yes / no

Were you seriously injured? yes / no

Was someone you cared about or close by seriously injured or killed? yes / no

Did you think you or a loved one was in danger of being killed by the disaster? yes / no

2. Were you involved in a motor vehicle accident for which you received medical attention or that badly injured or killed someone?

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago
 _____ 10-15 years ago; _____ when you were less than 5
 years old

Did you experience fear, helplessness, or horror when it happened? yes / no

Were you seriously injured? yes / no

3. Have you been involved in any other kind of accident where you or someone else was badly hurt? (examples: a plane crash, a drowning or near drowning, an electrical or machinery accident, an explosion, home fire, chemical leak, or overexposure to radiation or toxic chemicals)

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago
 _____ 10-15 years ago; _____ when you were less than 5
 years old

Did you experience fear, helplessness, or horror when it happened? yes / no

Were you seriously injured? yes / no

4. Have you lived, worked, or had military service in a war zone? yes / no
 If yes, were you ever exposed to warfare or combat? (for example: in the vicinity of a rocket attack or people being fired upon; seeing someone getting wounded or killed)

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago
 _____ 10-15 years ago; _____ when you were less than 5
 years old

Did you experience fear, helplessness, or horror when it happened? yes / no

Were you seriously injured? yes / no

5. Have you experienced the unexpected and sudden death of a close friend or loved one?

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago
 _____ 10-15 years ago; _____ when you were less than 5
 years old

Did you experience fear, helplessness, or horror when it happened? yes / no

Were you seriously injured? yes / no

6. Has a loved one (who is living) ever experienced a life threatening or permanently disabling accident, assault, or illness? (examples: spinal cord injury, rape, life threatening virus)

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago
 _____ 10-15 years ago; _____ when you were less than 5
 years old

Did you experience fear, helplessness, or horror when it happened? yes / no

7. Have you ever had a life threatening illness?

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago
 _____ 10-15 years ago; _____ when you were less than 5
 years old

Did you experience fear, helplessness, or horror when it happened? yes / no

8. Have you been robbed or been present during a robbery – where the robber(s) used or displayed a weapon?

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago
 _____ 10-15 years ago; _____ when you were less than 5
 years old

Did you experience fear, helplessness, or horror when it happened? yes / no

Were you seriously injured? yes / no

9. Have you ever been hit or beaten up and badly hurt by a stranger or someone you didn't know very well?

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago
 _____ 10-15 years ago; _____ when you were less than 5
 years old

Did you experience fear, helplessness, or horror when it happened? yes / no

Were you seriously injured? yes / no

- 10. Have you seen a stranger (or someone you didn't know very well) attack or beat up another someone and seriously injure or kill them?**

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago
 _____ 10-15 years ago; _____ when you were less than 5
 years old

Did you experience fear, helplessness, or horror when it happened? yes / no

- 11. Has anyone threatened to kill you or cause you serious physical harm?**

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago
 _____ 10-15 years ago; _____ when you were less than 5
 years old

Did you experience fear, helplessness, or horror when it happened? yes / no

Was this person a stranger? yes / no **friend or acquaintance?** yes / no

relative? yes / no **intimate partner?** yes / no

- 12. While growing up, were you physically punished in a way that resulted in bruises, burns, cuts, or broken bones?**

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

Did you experience fear, helplessness, or horror when it happened? yes / no

- 13. While growing up, did you see or hear family violence? (such as your father hitting your mother; or any family member beating up or inflicting bruises, bruises, or cuts on another family member)**

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

Did you experience fear, helplessness, or horror when it happened? yes / no

- 14. Have you ever been slapped, punched, kicked, beaten up, or otherwise physically hurt by your spouse (or former spouse), a boyfriend/girlfriend, or some other intimate partner?**

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

When did it happen? _____ In the past year; _____ 2-5 years ago; _____ 6-10 years ago

Did you experience fear, helplessness, or horror when it happened? yes / no

Were you seriously injured? yes / no

Has more than one intimate partner physically hurt you? yes / no

If yes, how many have hurt you? _____

- 15. Before your 13th birthday: Did anyone – who was at least 5 years older than you – touch or fondle your body in a sexual way or make you touch or fondle their body in a sexual way?**

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

Did you experience fear, helplessness, or horror when it happened? yes / no

Were you seriously injured? yes / no

Was the person a stranger? yes / no **friend or acquaintance?** yes / no

parent or caregiver? yes / no **other relative?** yes / no

Was threat or force used? yes / no

Was there oral, anal, or vaginal penetration? yes / no

- 16. Before your 13th birthday: Did anyone close to your age touch sexual parts of your body or make you touch sexual parts of their body –against your will or without your consent?**

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

Did you experience fear, helplessness, or horror when it happened? yes / no

Were you seriously injured? yes / no

Was the person a stranger? yes / no **friend or acquaintance?** yes / no

parent or caregiver? yes / no **other relative?** yes / no

Was threat or force used? yes / no

Was there oral, anal, or vaginal penetration? yes / no

- 17. After your 13th birthday and before your 18th birthday: Did anyone touch sexual parts of your body or made you touch sexual parts of their body – against your will or without your consent?**

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

Did you experience fear, helplessness, or horror when it happened? yes / no

Were you seriously injured? yes / no

Was the person a stranger? yes / no **friend or acquaintance?** yes / no

parent or caregiver? yes / no **other relative?** yes / no

Was threat or force used? yes / no

Was there oral, anal, or vaginal penetration? yes / no

- 18. After your 18th birthday: : Did anyone touch sexual parts of your body or made you touch sexual parts of their body – against your will or without your consent?**

never once twice 3 times 4 times 5 times more than 5 times

If this happened:

Did you experience fear, helplessness, or horror when it happened? yes / no

23. Have you experienced (or seen) any other events that were life threatening, caused serious injury, or were highly disturbing and distressing? (examples: lost in the wilderness; a serious animal bite; violent death of a pet; being kidnapped and held hostage; seeing a mutilated body or parts)

never once twice 3 times 4 times 5 times more than 5 times

Please describe: _____

If this happened:

Did you experience fear, helplessness, or horror when it happened? yes / no

Were you seriously injured? yes / no

24. If any of the events (listed above) happened to you, which one event CAUSES YOU THE MOST DISTRESS?

Indicate Item #: _____

When did this event (last) happen (your age or date)? _____

How much distress (anxiety, worry, sadness, or grief) does this event cause you?

None happened	no distress	slight distress	moderate distress	considerable distress	extreme distress
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Appendix E

Schematic Illustration of the Modified-Recognition Memory Task

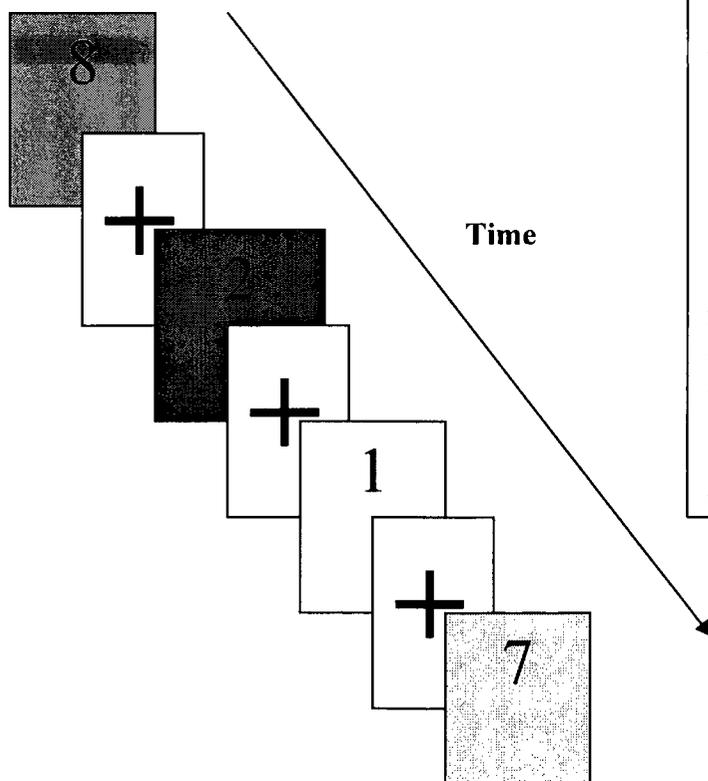
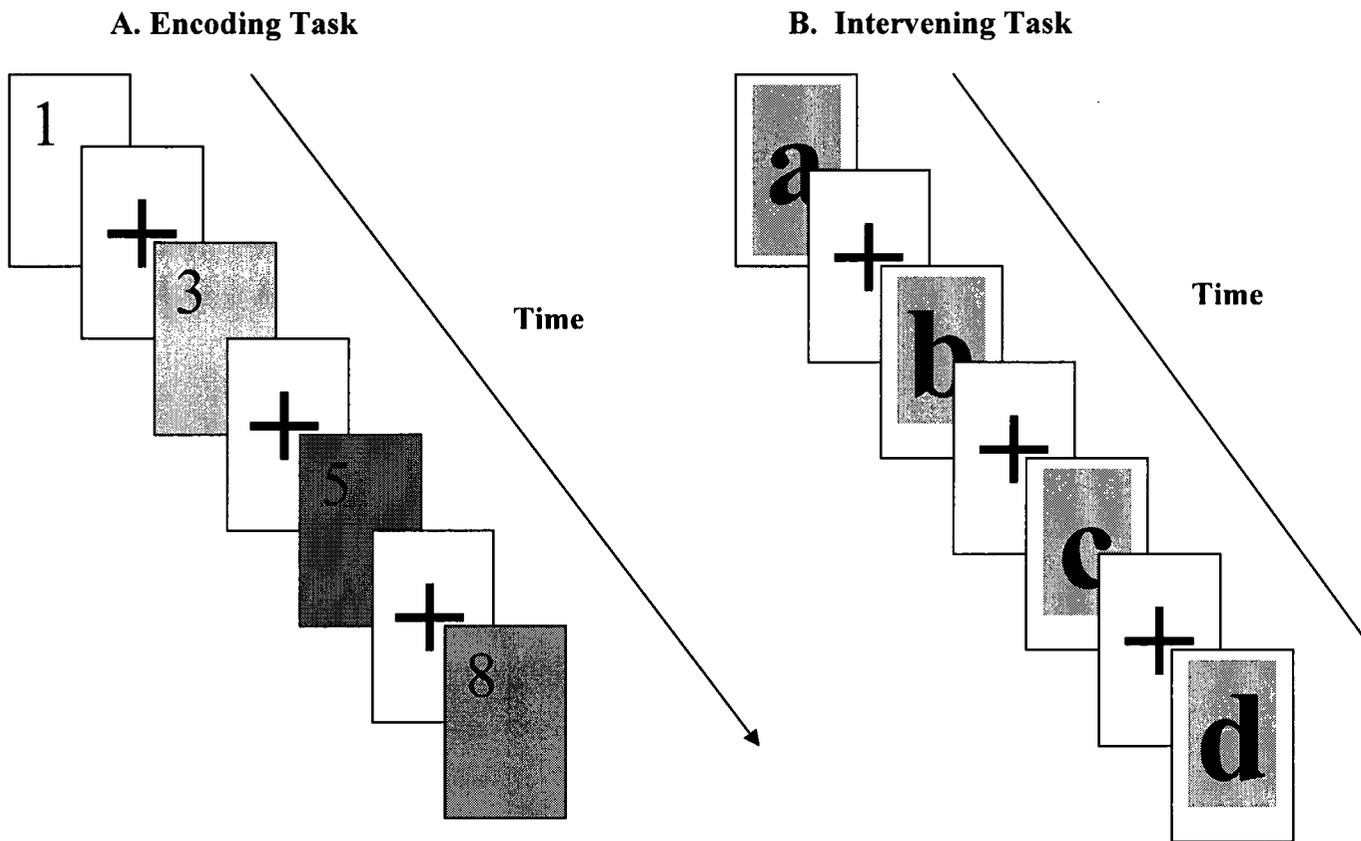


Fig.1 Illustration of the modified recognition test. Panels A, B, C illustrate the three successive tasks that will be administered, for the three trial types (neutral, trauma-related or unrelated), and presents the schematic presentation of the tasks. During encoding, a series of images will be presented (as indicated by 1,3,5,8). The intervening task will follow, in which 36 pictures will be presented as interference stimuli. During the forced-recognition task, half of the pictures presented during encoding will be presented along with other images that have never been seen before interspersed. Participants will be asked to indicate if they have previously seen the picture.

Appendix F

Debriefing Form (Study 1)

Memory plays a central role in human life. It touches almost everything we do and is involved in many of our daily activities. We want to remember people's names, where we put things, when to pay our bills, when to take our medicine, when to acknowledge our friends' and family birthdays—the list goes on and on. Given its importance in daily life, an improved understanding of the factors that may interfere with memory processes may be particularly important to daily living and well-being.

In this study we were primarily interested in knowing how factors such as the experience of previous stressful events, current well-being as well as the emotions that are raised by the various images influence your memory processes, particularly memory recall of images you have previously seen. In order to do this, we asked you to complete a few questionnaires about your well-being and previous traumatic history and complete a memory task. This task was constituted of three successive tasks that required you to: 1) judge a series of images according to their level of complexity; this procedure is meant to help you encode the images (i.e., we want you to remember them); 2) indicate your emotional reactivity to another series of images either entailing stressful experiences or not; 3) recognize the first series of images among others that have never been previously seen. We are especially interested in seeing if the trauma pictures and your emotional reactions elicited by the second series of images influenced your ability to remember the first series of images and how this might be related to your personal history and well-being.

Contacts

The following people are involved in this research project and may be contacted at any time if you have any further questions about the project, what it means, or concerns about how it was conducted:

Dr. H. Anisman, Faculty Member, Department of Psychology, 520-2699

Dr. K. Matheson, Faculty Member, Department of Psychology, 520-2684

Kathy Michaud, Researcher, 520-2600 ext. 7513,

kmichaud@connect.carleton.ca

If you have any ethical concerns about how this study was conducted, please contact either of the following:

Dr. J. Mantler, Chair of the Carleton University Research Ethics Committee for Psychological Research, 520-2600, ext. 4173

Dr. M. Gick, Chair of the Department of Psychology

Carleton University, 520-2648.

If you are experiencing distress, or if you feel unhappy or depressed, then it is advisable that you contact your family physician, Carleton Health and Counseling Services at 520-6674 or Student Life Services at 520-6600. It is not a good idea to allow problems to fester, as ruminating over these problems will typically not make them go away. Your family physician or counselor will usually be able to help you or to refer you to someone who can.

Appendix G
SONA Recruitment Notice (Study 2)

Images of emotional events in relation to behavioral and hormonal reactivity

This study involves seeing images of emotional events and providing your reactions to such images. You will be randomly be exposed to *either* neutral images or potentially distressing events such as scenes of violence. You will also complete questionnaires regarding your personal history and well-being, as well as a series of questions that asks you about potentially traumatic events that you might have experienced (e.g. loss of a loved one, abuse, war, accident, etc.) and when these events occurred. Some individuals may experience discomfort or distress when viewing or recalling a stressful experience.

Finally, because stress can affect how we perceive situations and how we react, we will be asking you for several measures of physiological stress. This study will take about 75 min, and you will be given 1.5% grade increase for your participation.

Appendix H

Informed Consent Form (Study 2)

The purpose of an informed consent is to ensure that you understand the purpose of the study and the nature of your involvement. The informed consent has to provide sufficient information such that you have the opportunity to determine whether you wish to participate in the study.

Study Title: Images of emotional events and reactivity in relation to memory, hormone and immune changes.

Research Personnel: The following people are involved in this research and may be contacted at any time if you have any questions about the project, what it means, or concerns about how it was conducted:

Dr. Kim Matheson (Faculty Investigator, 520-2684)

Dr. Hymie Anisman (Faculty Investigator, 520-2699)

Kathy Michaud (Researcher, 520-2600 ext. 4199)

If you have any ethical concerns about how this study please contact Dr. J. Mantler (Chair of the Carleton University Research Ethics Committee for Psychological Research, 520-2600, ext 4173) or Dr. M. Gick, Chair of the Department of Psychology, Carleton University, 520-2648.

Purpose, Task Requirements and Time for Participation: This study comprises two parts. In the first part of the study, we will be asking you to fill out questionnaires regarding several personal issues, including your psychological health. In addition, you will be given a series of questions that asks you about some potentially traumatic events you may have experienced (e.g. assault, war, or loss of a loved one) that may have affected how you deal with other events in your life. Once the questionnaires are completed, you'll be asked, in the second part, to see and judge a series of images that are presented to you on a computer screen, and to indicate your emotional reaction to them. Assignment to conditions is random, and there is a 50% chance of being assigned to a condition that includes pictures of distressing scenes including war and violence. Given the nature of the questions and pictures and the fact that they may evoke distress for some individuals, remember that your participation is completely voluntary and that you may withdraw from the study at anytime.

Once this task is finished, you will be asked to complete a few other questionnaires regarding your emotional reactions toward the stimuli and your coping strategies.

Finally, as one's own stress levels can affect their reactions and performance, we will also be asking you to provide 4 saliva samples throughout the study from which we can extract various stress hormones, including cortisol, and indicators. This study will take approximately 75 minutes, and you will receive 1.5 % grade increase for your participation.

Potential Risk and Discomfort: There are no physical risks in this study. Some individuals may experience discomfort or distress when asked to respond to personal, sensitive questions, or when recalling or viewing a stressful experience.

Right to Withdraw: Your participation in this study is entirely voluntary. At any point during the study you have the right to choose to not answer any questions, or to withdraw entirely without penalty. Likewise, even after you've provided answers or saliva, should

you change your mind and want your answers and the saliva destroyed, you need only call us or drop by and indicate your preference.

Anonymity/Confidentiality: The data collected in this study will be kept anonymous and confidential. Your informed consent form will be separated from your questionnaire and kept in a separate and secured file by one of the research investigators. A code placed on your written measures will be used to match your responses with the physiological measures. No individual results of the saliva will be made available – all results will be reported in aggregate. Once biological samples are analyzed, the remnants or remaining sample is destroyed.

I have read the above description of the study concerning images of emotional events and reactivity. The data collected will be used in research publications and/or for teaching purposes. My signature indicates that I agree to participate in the study, and this in no way constitutes a waiver of my rights.

Full Name (please print): _____

Participant _____ Researcher: _____
Date: _____ Date: _____

Appendix I
Additional Measures (Study 2)

BACKGROUND

Age _____

Sex _____

Ethnic/racial background _____

Marital status _____

The following questions are important for our analysis of stress hormones:

Are you currently being treated for any physical condition?

No _____ Yes _____ If yes, please specify _____

Are you on any of the following medications (please check all that apply):

_____ Birth control pill

_____ Anti-inflammatories (please specify) _____

_____ Anti-depressives (please specify) _____

_____ Anti-anxieties (please specify) _____

_____ Other Prescription drugs (please specify) _____

Appendix J

Debriefing Form (Study 2)

Memory plays a central role in human life. It touches almost everything we do and is involved in many of our daily activities. We want to remember people's names, where we put things, when to pay our bills, when to take our medicine, when to acknowledge our friends' and family birthdays—the list goes on and on. Given its importance in daily life, an improved understanding of the factors that may interfere with memory processes may be particularly important to daily living and well-being.

In this study, we are particularly interested in knowing how an intervening task and emotions influence your memory of images you have previously seen. In order to do this, we asked you to complete three successive tasks: 1) judge a series of images according to their level of complexity; this procedure is meant to help you encode the images (i.e., we want you to remember them); 2) Indicate your emotional reactions to a separate series of images entailing either neutral scenes or stressful experiences; 3) recognize among many images which scenes were present in the first series of images. We are especially interested in seeing if the trauma pictures and your emotional reactions elicited by this second series of images influenced your ability to remember the first series of images. Clearly, there are many other factors that may influence memory processes including age, motivation and so on. In this study we were primarily interested in factors such as the experience of previous traumatic experiences, current well-being as well as the emotions that are raised by the various images.

We also wish to assess whether the body's response to stress and psychological well-being (e.g., depression) is influenced by an individual's past traumatic history and reactivity to emotional images influences. Examining physiological indices does this, in this case by measuring the levels of a stress hormone, cortisol, which appears in saliva. While stress hormones normally aid the body to deal with stress, having high levels of stress hormones for extended periods of time can have harmful effects on the body. Thus, knowing the relationship between this hormone, previous life experiences, and the type and severity of stressors experienced may allow us to understand the processes through which stressful events induce effects on well-being. We hope the results of this study will provide some insight regarding some of the factors that might influence memory processes, and in particular those situations that comprise stressful experiences. Thank you for your participation in this study. The information you have provided is of great value to us.

Contacts

The following people are involved in this research project and may be contacted at any time if you have any further questions about the project, what it means, or concerns about how it was conducted:

Dr. H. Anisman, Faculty Member, Department of Psychology, 520-2699

Dr. K. Matheson, Faculty Member, Department of Psychology, 520-2684

Kathy Michaud, Researcher, 520-2600 ext. 4199, kmichaud@connect.carleton.ca

If you have any ethical concerns about how this study was conducted, please contact either of the following:

Dr. J. Mantler, Chair of the Carleton University Research Ethics Committee for Psychological Research, 520-2600, ext. 4173

Dr. M. Gick, Chair of the Department of Psychology, Carleton University, 520-2648.

If you are experiencing distress, or if you feel unhappy or depressed, then it is advisable that you contact your family physician, Carleton Health and Counseling Services at 520-6674 or Student Life Services at 520-6600. It is not a good idea to allow problems to fester, as ruminating over these problems will typically not make them go away. Your family physician or counselor will usually be able to help you or to refer you to someone who can.

Appendix K

SONA Recruitment Notice (Study 3)

Images of emotional events and reactivity in relation to attention, memory, hormones and immune changes

This study involves seeing images of emotional events and providing your reactions to such images. You will be exposed to *both* neutral images and images of potentially distressing events such as scenes of violence. You will also complete questionnaires regarding your personal history and well-being, as well as a series of questions that asks you about potentially traumatic events that you might have experienced (e.g. loss of a loved one, abuse, war, accident, etc.) and when these events occurred. Some individuals may experience discomfort or distress when viewing or recalling a stressful experience.

Finally, because stress can affect how we perceive situations and how we react, we will be asking you for several measures of physiological stress. This study will take about 75 min, and you will be given 1.5% grade increase for your participation.

Appendix L

Informed Consent Form (Study 3)

The purpose of an informed consent is to ensure that you understand the purpose of the study and the nature of your involvement. The informed consent has to provide sufficient information such that you have the opportunity to determine whether you wish to participate in the study.

Study Title: Images of emotional events and reactivity in relation to attention, memory, hormones changes.

Research Personnel: The following people are involved in this research and may be contacted at any time if you have any questions about the project, what it means, or concerns about how it was conducted:

Dr. Kim Matheson (Faculty Investigator, 520-2684)

Dr. Hymie Anisman (Faculty Investigator, 520-2699)

Kathy Michaud (Researcher, 520-2600 ext. 4199)

If you have any ethical concerns about how this study please contact Dr. A. Parush (Chair of the Carleton University Research Ethics Committee for Psychological Research, 520-2600, ext 6026) or Dr. A. Bowker, Chair of the Department of Psychology, Carleton University, 520-8218.

Purpose, Task Requirements and Time for Participation: This study comprises two parts. In the first part of the study, we will be asking you to fill out questionnaires regarding several personal issues, including your psychological health. In addition, you will be given a series of questions that asks you about some potentially traumatic events you may have experienced (e.g. assault, war, or loss of a loved one) that may have affected how you deal with other events in your life and your coping strategies.

Once the questionnaires are completed, you'll be asked to complete three computerized tasks for the second part of this study. In the first task, you will see and judge a series of images that are presented to you on a computer screen, and be asked to indicate your emotional reaction to them. Assignment to conditions of this task is random, and there is a 50% chance of being assigned to a condition that includes pictures of distressing scenes including war and violence. The second task is a modification of the Stroop Test, which involves four conditions: 1) reading color words; 2) naming the colors of circles presented; 3) naming the color in which color words are written (which is different in different) and 4) naming the color in which trauma-related, happy or neutral words are written. The third task involves seeing pair of images of neutral and distressing scenes including war and violence presented to you very quickly on a computer screen. These pairs of images are followed by the presentation of a "*" at the right or left of the screen and you will be asked to indicate the location of the "*". Given the nature of the questions and pictures and the fact that they may evoke distress for some individuals, remember that your participation is completely voluntary and that you may withdraw from the study at anytime.

Once these tasks are finished, you will complete a few other questionnaires regarding your emotional reactions toward the stimuli. Finally, your stress levels can affect your reactions and performance, we will also be asking you to provide 4 saliva samples throughout the study from which we can extract various stress hormones, including cortisol, and indicators. This study will take approximately 75 minutes, and you will receive 1.5 % grade increase, OR \$10 for your participation.

Potential Risk and Discomfort: There are no physical risks in this study. Some individuals may experience discomfort or distress when asked to respond to personal, sensitive questions, or when recalling or viewing a stressful experience.

Right to Withdraw: Your participation in this study is entirely voluntary. At any point during the study you have the right to choose to not answer any questions, or to withdraw entirely without penalty. Likewise, even after you've provided answers or saliva, should you change your mind and want your answers and the saliva destroyed, you need only call us or drop by and indicate your preference.

Anonymity/Confidentiality: The data collected in this study will be kept anonymous and confidential. Your informed consent form will be separated from your questionnaire and kept in a separate and secured file by one of the research investigators. A code placed on your written measures will be used to match your responses with the physiological measures. No individual results of the saliva tests will be made available – all results will be reported in aggregate. Once biological samples are analyzed, the remnants or remaining sample is destroyed.

I have read the above description of the study concerning images of emotional events and reactivity. The data collected will be used in research publications and/or for teaching purposes. My signature indicates that I agree to participate in the study, and this in no way constitutes a waiver of my rights.

Full Name (please print): _____

Participant _____ Researcher: _____

Date: _____ Date: _____

Appendix M

Additional Measures (Study 3)

IMPACT OF EVENT SCALE – REVISED

The following is a list of difficulties people sometimes have after stressful life events. Please read each item and then indicate how distressing each difficulty has been for you **during the past seven days** as a result of the event that happened to you that you indicated that caused you the most distress in the last question (TLEQ 24)

During the past seven days, how much have you been distressed or bothered with these difficulties?

	<i>Not at All</i>	<i>A little bit</i>	<i>Moderately</i>	<i>Quite a bit</i>	<i>Extremely</i>
1. Any reminder brought back feelings about it.	0	1	2	3	4
2. I had trouble staying asleep.	0	1	2	3	4
3. Other things kept making me think about it.	0	1	2	3	4
4. I felt irritable and angry.	0	1	2	3	4
5. I avoided letting myself get upset when I thought about it or was reminded of it.	0	1	2	3	4
6. I thought about it when I didn't mean to.	0	1	2	3	4
7. I felt as if it hadn't happened or wasn't real.	0	1	2	3	4
8. I stayed away from reminders about it.	0	1	2	3	4
9. Pictures about it popped into my mind.	0	1	2	3	4
10. I was jumpy and easily startled.	0	1	2	3	4
11. I tried not to think about it.	0	1	2	3	4
12. I was aware that I still had a lot of feelings about it but I didn't deal with them.	0	1	2	3	4
13. My feelings about it were kind of numb.	0	1	2	3	4
14. I found myself acting or feeling like I was back at this time.	0	1	2	3	4
15. I had trouble falling asleep.	0	1	2	3	4
16. I had waves of strong feelings about it.	0	1	2	3	4
17. I tried to remove it from my memory.	0	1	2	3	4
18. I had trouble concentrating.	0	1	2	3	4

	<i>Not at All</i>	<i>A little bit</i>	<i>Moderately</i>	<i>Quite a bit</i>	<i>Extremely</i>
19. Reminders of it caused me to have physical reactions, such as sweating, trouble breathing, nausea, or a pounding heart.	0	1	2	3	4
20. I had dreams about it.	0	1	2	3	4
21. I felt watchful and on guard.	0	1	2	3	4
22. I tried not to talk about it.	0	1	2	3	4

Modified USSI – A

Please think about times when you've turned to other people (e.g., friends, partner, family, etc.) for support in regards the **event that happened to you that you indicated that caused you the most distress in the last question of the TLEQ (TLEQ 24)**. If you did not experience any situation mentioned in the TLEQ, please think of an experience situation that was bothering you (i.e. frustrations or disappointments with friends, family, school, health, work or anything else that is important to you).

Item of TLEQ 24 or Situation: _____

For each of the statements below, please indicate how frequently other people responded in this way when you went to them for support.

	None				I
	0	1	2	3	4
1. Someone thought I was over-reacting to the situation	0	1	2	3	4
2. When I was talking about the issue/situation with someone, this person did not give me enough of his/her time, or made me feel like I should hurry	0	1	2	3	4
3. Someone made "should/shouldn't have" comments about my role in the situation, such as, "You shouldn't have"	0	1	2	3	4
4. Someone didn't seem to know what to say, or seemed afraid of saying/doing the "wrong" thing	0	1	2	3	4
5. Someone refused to provide the type of help or support I was looking for	0	1	2	3	4
6. After becoming aware that I was dealing with something that I found difficult or distressing, someone responded with uninvited physical touching, such as hugging	0	1	2	3	4
7. Someone said I should look on the bright side	0	1	2	3	4
8. Someone said "I told you so." Or made some similar comment to me about my situation	0	1	2	3	4
9. Someone seemed to be telling me what he thought I wanted to hear	0	1	2	3	4
10. In responding to me about my situation, someone seemed disappointed in me	0	1	2	3	4
11. When I was talking to someone about my situation, this person changed the subject before I wanted to	0	1	2	3	4

	None			A lot	
	0	1	2	3	4
12. Someone felt that I should stop worrying about the situation and just forget about it	0	1	2	3	4
13. Someone asked me “why” questions, such as, “Why did/didn’t you ...”	0	1	2	3	4
14. Someone felt that I should focus on the present and/or future, and that I should forget about what’s happened and get on with my life.	0	1	2	3	4
15. Someone tried to cheer me up when I was not ready to cheer up about the situation	0	1	2	3	4
16. Someone refused to take me seriously	0	1	2	3	4
17. Someone told me to be strong, to keep my chin up, or that I shouldn’t let it bother me	0	1	2	3	4
18. When I was talking to someone about what was bothering me, this person did not seem to want to hear about it	0	1	2	3	4
19. Someone told me that I had gotten myself into the situation in the first place, and that now I must deal with the consequences	0	1	2	3	4
20. Someone did something for me that I wanted to do and could have done for myself, as if the person thought I was no longer capable	0	1	2	3	4
21. Someone discouraged me from expressing feelings about my situation, such as anger, hurt or sadness	0	1	2	3	4
22. Someone felt that ‘it could have been worse’ or that ‘it was not as bad as I thought’	0	1	2	3	4
23. From the person’s tone of voice, expression, or body language, I got the feeling that the person was uncomfortable talking with me about my problem	0	1	2	3	4
24. Someone made comments which blamed me or tried to make me feel responsible	0	1	2	3	4

Appendix N
Attention Measures (Study 3)

Words used in the Emotional Stroop Test

Trauma-related words	Positive words	Neutral words
Aggression	Comfort	Object
Assault	Safety	Fruit
Shock	Secure	Stamp
Horror	Joyful	Circle
Attack	Delight	Table
Helpless	Happy	Graphic
Murder	Hopeful	Computer
Victim	Humour	Hammer
Pain	Friendly	Towel
Terror	Divine	House
Violent	Harmony	Pencil
Disaster	Loving	Street
Brutal	Faithful	Blouse
Despair	Harmless	Stone
Threaten	Goodness	Factory
Abuse	Peaceful	Cupboard
Stalked	Trusting	Library
Nightmare	Courage	Science
Tragedy	Affection	Tool
Death	Loyal	Memory
Robbery	Grateful	Ankle
Accident	Freedom	Kettle
Illness	Healing	Clothing
Crisis	Helpful	edition

Illustration of the Visual Probe Test

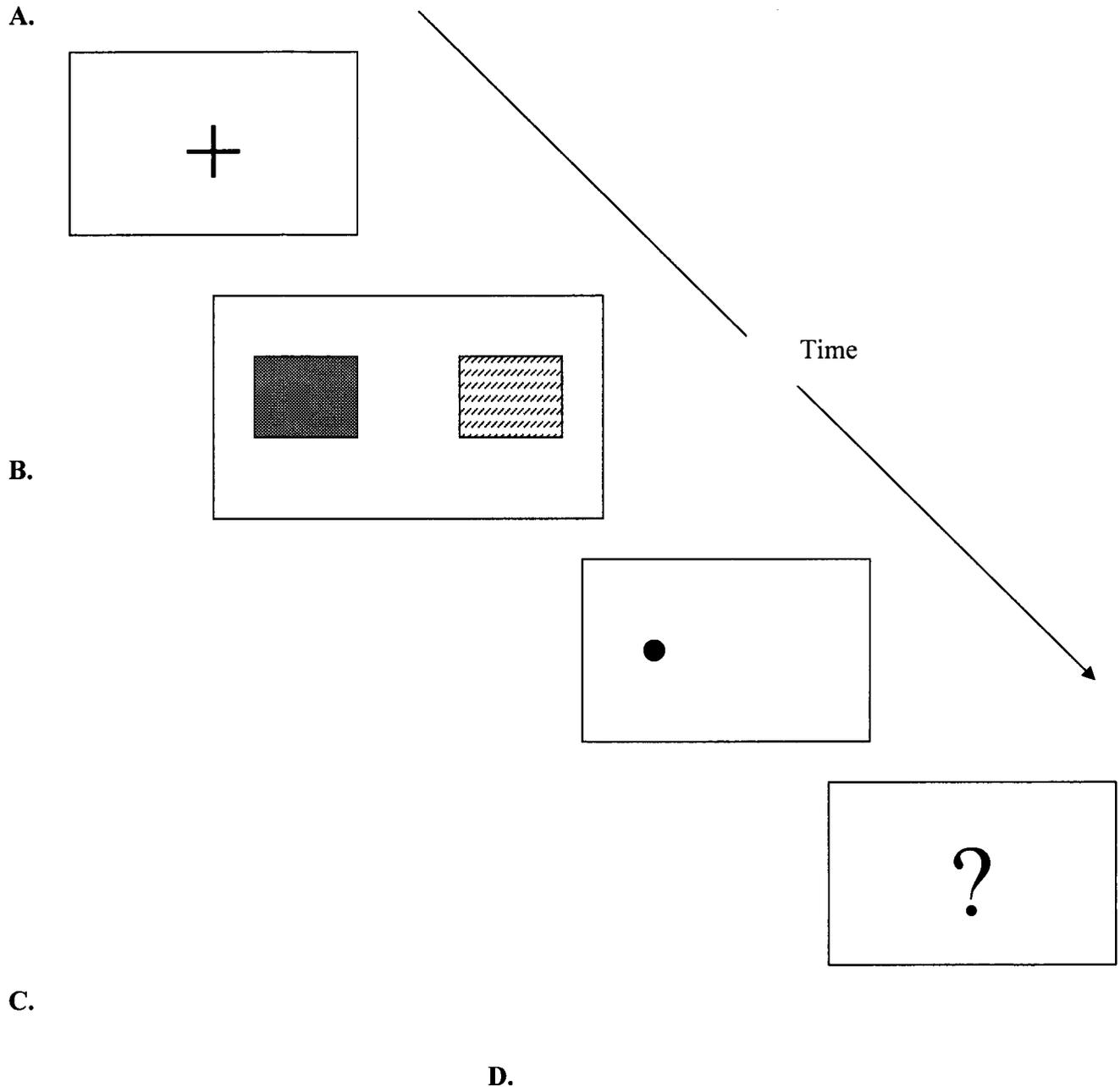


Fig.2 Illustration of the visual probe test. Panels A, B, C, D illustrate the sequence of events appearing successively on the computer screen on each trial. Each trial begins by central fixation cross for 500 ms (A). This is followed by a pair of photographs for 500 ms (B). A single probe is presented at the location of one of the picture for 500 ms (C) and participants have to indicate as quickly as possible where the probe was presented (D).

Appendix O

Debriefing Form (Study 3)

Memory and attention processes play a central role in human life. It touches almost everything we do and is involved in many of our daily activities. We want to pay attention to and remember people's names, where we put things, when to pay our bills, when to take our medicine, when to acknowledge our friends' and family birthdays—the list goes on and on. Given its importance in daily life, an improved understanding of the factors that may interfere with memory and attention processes may be particularly important to daily living and well-being.

In this study, we are particularly interested in knowing how trauma related pictures or words influence your memory and attention processes. In order to do this, we asked you to complete three computerized tasks. The first task you completed was the memory task. It involved three successive tasks: 1) to judge a series of images according to their level of complexity; this procedure is meant to help you encode the images (i.e., we want you to remember them); 2) Indicate your emotional reactions to a separate series of images entailing either neutral scenes or stressful experiences; 3) recognize among many images which scenes were present in the first series of images. We are especially interested in seeing if the trauma pictures and your emotional reactions elicited by this second series of images influenced your ability to remember the first series of images.

The two other computerized task were concerned with attention processes. In order to know if trauma-related words influence your attention, the modified version of the Stroop test was used. In the last condition, you had to name the color in which happy (i.e. joyful), neutral (i.e. table) and trauma-related words (i.e. assault) are written. This task takes advantage of our ability to read words more quickly and automatically than we name colors. Thus, if trauma-related words influenced your attention, naming the color in which it is written would have been harder for you compared to neutral words. In addition to know whether trauma-related images also influence you attention, we presented to you a series of pair of images containing either neutral-happy or neutral-trauma images. These images were followed by the presentation of a "*" located at the right or the left of the screen. Once the "*" had disappeared, you had to indicated where the "*" appeared. If your attention was biased by trauma-related pictures, you answered more rapidly to "*" that replaced traumatic pictures.

Clearly, there are many other factors that may influence memory and attention processes including age, motivation and so on. In this study we were primarily interested in factors such as the experience of previous traumatic experiences, current well-being as well as the emotions that are raised by the various images.

We also wish to assess whether the body's response to stress and psychological well-being (e.g., depression) influences attention and memory processes. Examining physiological indices does this, in this case by measuring the levels of a stress hormone, cortisol, which appears in saliva, and other hormones and indicators in blood (if you agreed to also provide a blood sample). While stress hormones normally aid the body to deal with stress, having high levels of stress hormones for extended periods of time can

have harmful effects on the body and these have been associated with memory impairments. Thus, knowing the relationship between this hormone, memory, attention and previous life experiences may allow us to understand the processes through which stressful events induce effects on well-being and cognitive processes. We hope the results of this study will provide some insight regarding some of the factors that might influence memory processes, and in particular those situations that comprise stressful experiences. Thank you for your participation in this study. The information you have provided is of great value to us.

Contacts

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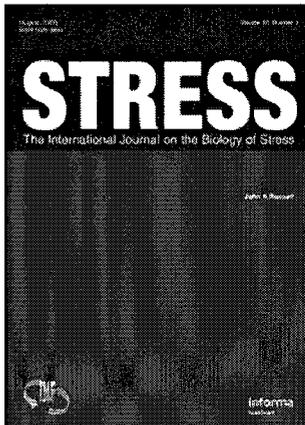
Dr. H. Anisman, Faculty Member, Department of Psychology, 520-2699
Dr. K. Matheson, Faculty Member, Department of Psychology, 520-2684
Kathy Michaud, Researcher, 520-2600 ext. 7513
kmichaud@connect.carleton.ca

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Dr. A. Parush, Chair of the Carleton University Research Ethics Committee for Psychological Research, 520-2600, ext. 6026
Dr. A. Bowker, Chair of the Department of Psychology, Carleton University, 520-8218.

If you are currently or later on (i.e., in the next few days) experiencing distress, or if you feel unhappy or depressed, then it is advisable that you contact your family physician, Carleton Health and Counseling Services at 520-6674 or Student Life Services at 520-6600. Similarly, please contact these resources if you are distressed, unhappy or depressed later on (i.e., in the next few days). Although, it is not common, it is possible that some vulnerable people may experience a delayed reaction. It is not a good idea to allow problems to fester, as ruminating over these problems will typically not make them go away. Your family physician or counsellor will usually be able to help you or to refer you to someone who can.

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Impact of stressors in a natural context on release of cortisol in healthy adult humans: A meta-analysis

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Impact of stressors in a natural context on release of cortisol in healthy adult humans: A meta-analysis

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Abstract

Increased hypothalamic–pituitary–adrenal (HPA) activation, culminating in elevated circulating cortisol levels is a fundamental response to stressors. In animals, this neuroendocrine change is highly reliable and marked (~5–10-fold elevations), whereas in humans, the increase of cortisol release is less pronounced, and even some potent life-threatening events (anticipation of surgery) only elicit modest cortisol increases. Meta-analysis of factors that influenced the increase of cortisol release in a laboratory context pointed to the importance of social evaluative threats and stressor controllability in accounting for the cortisol rise. The present meta-analysis, covering the period from 1978 through March 2007, was undertaken to identify the factors most closely aligned with cortisol increases in natural settings. It appeared that stressor chronicity was fundamental in predicting cortisol changes; however, this variable is often confounded by the stressor type, the stressor's controllability, as well as contextual factors, making it difficult to disentangle their relative contributions to the cortisol response. Moreover, several experiential factors (e.g. previous stressor experiences) may influence the cortisol response to ongoing stressors, but these are not readily deduced through a meta-analysis. Nevertheless, there are ample data suggesting that stressful events, through their actions on cortisol levels and reactivity, may influence psychological and physical pathology.

Keywords: *Cortisol, corticosterone, stress, naturalistic, ACTH*

Introduction

At one time or another, most individuals encounter stressful events that challenge their psychological and/or physiological well-being. Obviously, not all individuals who encounter stressors respond in a uniform fashion nor do all stressors elicit comparable outcomes. Ordinarily, stressors will give rise to several behavioral, cognitive and emotional changes, some of which are aimed at contending with the challenge. Concurrently, a concatenation of brain neurochemical and hormonal changes are provoked, whose function is one of enhancing the effects of other neuroendocrine processes, preparing the organism to cope with the insult, and blunting the physiological and

psychological impact of the stressor (de Kloet et al. 1999; McEwen 2000a,b; Sapolsky et al. 2000). Although stressor-induced neurochemical alterations may be adaptive, it has been suggested that when the stressor is sufficiently protracted, the strain on endogenous systems might become excessive (allostatic overload), thereby increasing vulnerability to disturbances, such as depression (McEwen 2003; McEwen and Wingfield 2003).

Of the many physiological changes that emerge in response to stressors, one of the most fundamental, and most frequently studied, is the increase of cortisol concentrations in blood, saliva or urine that accompanies stressful experiences. Increased secretion of cortisol by the adrenal cortex is thought

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to be a fundamental feature of the stress response with multiple beneficial effects. However, in humans, this hormone is not elicited under all stressor conditions, and even some fairly strong stressors elicit only moderate cortisol variations (Biondi and Picardi 1999). The goal of the present meta-analysis was to define the contribution of several factors in determining cortisol release in humans in response to naturalistic stressors. In this regard, it was of particular interest to establish the contribution of variables such as stressor controllability, predictability and chronicity, as well as social evaluative threats in promoting these cortisol changes. These factors are known to affect stress responses in animals (in the case of the former three variables), and social evaluative threat has been implicated as being particularly cogent in promoting cortisol changes in a laboratory context (Dickerson and Kemeny 2004). Thus, it was hypothesized that such factors might also be pertinent in moderating the cortisol response in humans in real world settings.

Physiological responses to stressors

In response to stressful experiences, certain behaviors, notably those associated with arousal, vigilance and coping processes predominate, whereas behaviors that are not productive in a defensive capacity (e.g. sexual and feeding behaviors) ought to be suppressed, although in humans, there are instances where eating, especially carbohydrates, may be a response to stressors (Levine and Marcus 1997; Dallman et al. 2003; Peters et al. 2007). Concurrently, neuronal functioning is increased within several stressor-sensitive brain regions. In this regard, studies in animals indicated that neuropeptide and monoamine changes are evident across multiple extrahypothalamic regions (including various amygdala nuclei, medial prefrontal cortex, locus coeruleus) and in hypothalamic nuclei, presumably to facilitate the ability to deal effectively with the ongoing challenge (Munck and Naray-Fejes-Toth 1994; de Kloet et al. 1999; McEwen 2000; Sapolsky et al. 2000).

To a considerable extent, organismic variables (age, sex, genetics) moderate the neurochemical changes elicited by stressors, as do experiential variables (previous stressful experiences, maternal factors) (Anisman and Matheson 2005). As well, some neurochemical stress responses (e.g. monoamine turnover in limbic regions) are influenced by factors such as stressor controllability (Weiss et al. 1981; Petty and Sherman 1982; Heinsbroek et al. 1989; Anisman et al. 1991; Bolanos-Jimenez et al. 1995; Nankai et al. 1995), chronicity, ambiguity and predictability (Matthews et al. 1980; Osuna 1985; Baker and Stephenson 2000) and it seems that different types of stressors may be selective in activating particular neural circuits (Anisman and Merali 1999). Whether this holds true in humans

is uncertain, although some types of stressors (e.g. anticipation of adverse events) are particularly effective in promoting anxiety (Paykel 1982; Reno and Halaris 1990), whereas others (e.g. loss, social conflict) are more aligned with depression (Roy 1983, 1985; Brown et al. 1987; Monroe and Depue 1991). Moreover, these particular effects may vary with gender (Harris 2001; Kendler et al. 2001; Mazure and Maciejewski 2003).

Stressor provoked variations of hypothalamic–pituitary–adrenal (HPA) activity

Psychological (psychogenic) or physical (neurogenic) stressors (both classed as “processive” stressors as they involve appraisal of the stimulus and the context in which this stimulus is presented), as well as systemic insults (e.g. immune activation associated with infection), stimulate hypothalamic–pituitary–adrenal (HPA) functioning, although they may do so through different neural circuits (Herman and Cullinan 1997; Yokoyama and Sasaki 1999; Sawchenko et al. 2000). By example, animal studies revealed that neurogenic stressors, innate psychogenic insults and learned psychogenic stressors may differentially influence neuropeptide processes (Merali et al. 2004). It is equally possible that, in humans, different types of stressors might not engage all of the same processes, and hence certain stressors may be more likely to influence HPA functioning.

Ordinarily, when a processive stressor is encountered, various brain regions may be activated. Some regions may be involved in the development or elicitation of fear and/or anxiety (e.g. central and medial amygdala and bed nucleus of the stria terminalis) (Lee and Davis 1997; LeDoux 2000), whereas others may be more important in the appraisal of the stressor or in executive functioning (e.g. medial and orbital prefrontal cortex) (Fuster 1989, 1995) or the learning/memory of fear/anxiety (Nader et al. 2000). After emotional meaning is assigned to the sensory information, the amygdala guides emotional behavior, likely through projections to the hypothalamus, hippocampus and prefrontal cortex (LeDoux 1986; Fellous 1999; Vertes 2006). Ultimately, the paraventricular nucleus (PVN) of the hypothalamus is activated, giving rise to the release of corticotropin-releasing hormone (CRH) from terminals located at the median eminence, thus promoting the release of adrenocorticotrophic hormone (ACTH) from the anterior pituitary, which then stimulates cortisol release (or corticosterone in rodents) from the adrenal cortex.

As prolonged cortisol increases may increase vulnerability to immunosuppression, and to autoimmune-related and metabolic disorders, it is essential that an individual be able to terminate cortisol release appropriately (Munck et al. 1984; Sapolsky et al. 2000). Once released into the circulation, cortisol activates corticoid receptors in the hippocampus

(Jacobson and Sapolsky 1991; Herman 1993; McEwen 2000), or may directly impact the hypothalamus to attenuate further neuroendocrine activity. In the latter instance, cortisol action on the PVN down-regulates CRH activity, and attenuates ACTH secretion (Uht et al. 1988; Whitnall 1993). In addition, given that stressors activate several limbic sites, it is not surprising that brain regions involved in emotional responses or appraisal processes also contribute to HPA regulation (Viau and Meaney 1991; Buijs et al. 1993; Diorio et al. 1993; Larsen et al. 1994; Suemaru et al. 1995; Herman and Cullinan 1997). Interestingly, unlike the suppression of hypothalamic CRH release elicited by cortisol (stemming from negative feedback), in limbic regions, particularly the central amygdala and bed nucleus of the stria terminalis, cortisol acts to increase CRH release, as well as GABA_A-mediated neuronal functioning, which may be fundamental in the promotion of various anxiety states (Schulkin et al. 1998, 2006; Duvarci and Pare 2007).

Glucocorticoid functions

Glucocorticoids (GC) have multiple actions that facilitate the ability to deal with stressors; GCs influence glucose metabolism, lipolysis, alterations in regional blood flow and may prevent overshoot of immune reactions (Sapolsky et al. 2000). Moreover, their function in dealing with adverse events can take several forms; they may act in a *permissive* (exert an effect prior to stressor and prime defence mechanisms), *suppressive* (arise from stress-activated defence reactions and prevent them from overshooting), *stimulating* (enhance the effects of the first wave of hormonal responses to stress) and finally *preparatory* capacity (do not affect the immediate response to a stressor but modulate the organism's response to a subsequent stressor (Munck et al. 1984; Sapolsky et al. 2000). Despite the adaptive and highly beneficial effects associated with cortisol release, as indicated earlier, sustained activation of these processes might culminate in excessive wear and tear on biological systems (allostatic overload), and hence might increase vulnerability to various pathological outcomes (McEwen 2000). Indeed, protracted increases of cortisol levels have been associated with depression and cognitive impairments (Lupien et al. 2005) as well as hippocampal cell loss (McEwen 2000).

Corticosterone (or cortisol, the primary GC in humans) has been taken to be the prototypical stress hormone, and it has frequently been assumed (incorrectly so) that GC changes provide an index of distress (witness for instance that cortisol levels may be reduced among those with post-traumatic stress disorder, PTSD; Yehuda 2002). In studies conducted in humans, at least within a laboratory context (e.g. using the Trier Social Stress Test, TSST), cortisol

elevations as high as 2–4-fold (much lower than the ~10-fold increase seen in rodents) have been reported, but changes of this magnitude are infrequent (Biondi and Picardi 1999). The factors governing the magnitude of cortisol changes elicited by stressors within a laboratory situation remain to be fully elucidated, although in their recent meta-analysis of laboratory-based studies, Dickerson and Kemeny (2004) indicated that cortisol variations are most pronounced in tests that involve uncontrollable social-evaluative threat (i.e. assessment by others).

The analysis of endocrine reactivity to psychological stress within a laboratory setting has been exceptionally valuable as it is permitted standardization of the stressor and control of potential confounding factors. Yet, little is known about the generalizability of these studies to actual life circumstances. However, field studies have been conducted to determine those conditions that favor cortisol changes that occur in response to day-to-day stressors or those of a more severe or chronic nature. These studies have included analyses of cortisol changes associated with daily hassles, events such as academic and occupational stressors, athletic stressors and various social stressors. Some naturalistic stressors (e.g. surgery) reliably increased HPA activity, but cortisol fluctuations in response to other stressful events have been less consistent, typically increasing from 25 to 100%, or have not been detected at all (Biondi and Picardi 1999). Of course, meaningfulness of these variations is difficult to compare across studies, as the stressors were qualitatively different from one another (e.g. differing in their relative severity/emotional impact, controllability, predictability, chronicity), as were the fluids in which cortisol was determined (i.e. blood, saliva, urine). Moreover, as already indicated, several organismic (genetic, age, gender) and experiential variables may also contribute in this regard.

HPA diurnal rhythm associated with stressful experiences

Ordinarily, cortisol release follows a well-defined diurnal rhythm, and is secreted from the adrenal cortex in a pulsatile manner (Deuschle et al. 1987; Mershon et al. 1992; Windle et al. 1998). In humans, cortisol release, already high at awakening, increases to reach a morning peak during the ensuing 30–60 min, and declines precipitously thereafter, reaching an evening nadir at about midnight (Linkowski et al. 1993; Schmidt-Reinwald et al. 1999). Although, it has been suggested that the increase of free cortisol (~50–60%) within the first 30 min after awakening is largely independent of the time of awakening, sleep duration, sleep quality, physical activity or morning routines (Spath-Schwalbe et al. 1992; Wust et al. 2000; Wilhem et al. 2007), there have been several reports indicating that time of awakening may, indeed, influence the course of the morning cortisol

rise (Edwards et al. 2001; Kudielka and Kirschbaum 2003; Kudielka et al. 2006; Federenko et al. 2004). Nevertheless, it does appear that the magnitude of the increase is related to general levels of distress (Pruessner et al. 1999; Schmidt-Reinwald et al. 1999) and may also be predictive of the response to other challenges, including CRH challenge and the TSST (Schmidt-Reinwald et al. 1999).

It has been suggested that the factors that moderate the morning cortisol rise have not been sufficiently examined, and it is unclear whether this increase is functionally significant (Clow et al. 2004). Indeed, although increasing stressful experiences have been associated with elevated morning cortisol release, relatively severe trauma has been associated with a blunting of the morning cortisol response (e.g. in patients affected by PTSD; Yehuda 2002). This may be coupled with an increase of the afternoon cortisol levels, so that the diurnal profile of cortisol release is flattened (Caplan et al. 1979; Hart et al. 1996; Adam and Gunnar 2001). Furthermore, there is evidence suggesting that chronic stressful experiences may be associated with diminished cortisol levels (Pruessner et al. 1999), although the case for such an outcome is not unequivocal (Melamed et al. 1999; Steptoe et al. 2000; Grossi et al. 2001), and defining what constitutes a chronic stressor has not been satisfactorily addressed. This should not be taken to imply that stressors do not favor elevated morning cortisol secretion, but that such an outcome is subject to the moderating influence of other factors, including a backdrop of traumatic experiences that provoke PTSD. In their recent meta-analysis regarding the influence of chronic stressors on HPA functioning, Miller et al. (2007) indicated that much of the variability that has been reported concerning cortisol changes elicited by chronic stressors could be attributed to features of the stressor, individual factors and the time since the stressor was experienced. It was suggested that those stressors that involve trauma, are uncontrollable, and threaten personal integrity tend to produce a high, flat diurnal profile, although the degree of morning cortisol release tends to be somewhat lower than that associated with stressors that do not promote PTSD.

The present analysis

There clearly exists an array of variables that could account for the individual differences of cortisol reactivity that typically occur in response to stressful experiences. Factors such as stressor predictability, controllability (with respect to stressor onset, stressor termination, and the consequences of stressor experiences) and chronicity have long been thought to play a pivotal role in behavioral disturbances and central neurochemical changes (reviewed in Anisman and Matheson 2005). Likewise, as already indicated,

in their meta-analysis, Dickerson and Kemeny (2004) indicated that within a laboratory context stressor controllability and social evaluative threats were fundamental in determining stressor-provoked cortisol increases. However, a comparative analysis is unavailable concerning the influence of different types of stressors on the cortisol response under naturalistic settings, or whether common denominators exist that moderate this stress response. In the present analysis, it was of interest to establish whether evaluative stressors (e.g. examinations, oral presentation, athletic competition), as well as stressor controllability, predictability and chronicity are fundamental in determining cortisol increases under naturalistic conditions. These variables were selected largely on the basis of animal studies that have identified them to be particularly important in determining behavioral and neurochemical changes associated with stressors (Anisman and Matheson 2005) as well as the meta-analysis reported by Dickerson and Kemeny (2004) concerning the factors that influence cortisol output in a laboratory context.

Methods

Literature search

Articles for consideration were identified through a computer-based search using Pubmed and PsycInfo databases from 1978 through March 2007. The key words were cortisol, HPA, neuroendocrine, hydrocortisone, psychoneuroimmunology, psychoimmunology, psychoneuroendocrinology and psychoendocrinology with naturalistic terms such as stressor, natural stress, psychological stress, as well as more specific terms such as caregiving, academic examination, competition and surgery. Additional articles were also obtained from the reference lists of these articles and several reviews (Kirschbaum and Hellhammer 1994; Biondi and Picardi 1999; Kiecolt-Glaser et al. 2002; Miller and O'Callaghan 2002; de Kloet 2003; Burke et al. 2005).

Study inclusion criteria

Articles were included only if they met the following criteria: (1) used naturalistic stressors, defined as stressful events or situations likely to be encountered by many or most individuals in the context of their everyday lives (e.g. academic examination, job strain, marital conflict, work-related noise exposure, anticipation of a competition or medical procedure) as well as stressful events of a chronic nature (e.g. caregiving). Inasmuch as severe stressors that might promote PTSD (e.g. rape, war) may involve processes distinct from the more common stressors, these were not included in the present analysis, but were recently reviewed by Miller et al. (2007); (2) the sample involved healthy adult participants. This criterion

allowed control for the effect of confounding variables with cortisol levels, namely age and the presence of physical and psychological pathologies that may also influence cortisol reactivity. Thus, studies involving severe illness or those maladies that require drug treatments that directly or indirectly affect HPA functioning (e.g. cancer, heart disease, diabetes, autoimmune disorders, depression and so forth), were excluded, as were those that examined stress reactivity in children and/or adolescents (i.e. where the mean age was under 18 years or the age range included participants under 18 years old); (3) reported or provided data from which an effect size was provided in the text or could be extracted or extrapolated by inferential statistics. Studies involving the prediction of cortisol levels from various personality characteristics were excluded given that it was impossible to determine from these data whether the stressor itself evoked significant changes of cortisol levels.

Coding

Demographic characteristics of participants as well as several features of the methodology and the nature of the stressor itself were coded. Reliabilities of the coding schema were calculated with the intraclass correlation (r_1) for the continuous variables and kappa (κ) for categorical variables (Orwin 1994) by randomly selecting 15% of the studies to be coded by a second independent, trained judge. As will be seen in ensuing sections, the interclass correlations were relatively high, attesting to the inter-rater reliability with respect to the evaluation of the characteristics of the stressors.

Participant characteristics. Participant characteristics included (a) sample size ($r_1 = 1.00$), (b) mean age of participants ($r_1 = 1.00$), (c) gender composition (coded as percent female, $r_1 = 1.00$) and whether exclusion criteria (e.g. depression, inflammatory diseases, medications used, $\kappa = 1.00$) were absent (coded 0) or present and reported in the studies (coded 1).

Methodological characteristics. Various aspects of the methodology of each study were coded. In this regard, studies were coded for the time of day that cortisol was collected ($\kappa = 1.00$). As described earlier, cortisol levels follow a diurnal rhythm, wherein an increase in level of the hormone is evident within the first 30–60 min following awakening, and then declines over the course of the day. Furthermore, in a laboratory context, stressors tend to have greater effects in the afternoon (when basal cortisol secretion is relatively low), thus it was deemed important to evaluate studies on the basis of the time of day in which they were conducted. Even though there is variability over the

course of the morning and over the course of the afternoon, like in Dickerson and Kemeny (2004), those studies in which the cortisol samples were collected before 12 p.m. were coded as morning studies (AM coded 1), whereas studies in which samples (saliva or plasma) for cortisol determination were collected after 12 p.m. were coded as afternoon studies (PM coded 2). Studies in which samples were collected at several prescribed times of the day were coded as diurnal (coded 3). Those studies in which saliva or plasma sample collection occurred at varied times of day, and were too broad to fit into a circumscribed code (e.g. between 9 a.m. and 4 p.m.) coded as AM/PM (coded 4). Finally, when the study did not report the time of day at which samples for cortisol measurement were collected, it was coded as non-specific (coded 5).

Given that the method by which samples for cortisol measurement were collected may also differentially influence cortisol values, this variable was also coded. Specifically, cortisol can be measured either in saliva (coded 1), blood (coded 2) or urine (coded 3). Not only do these methods vary in their degree of intrusion (e.g. venepuncture is a more intrusive method of collection that may, itself, constitute a distressing factor thereby influencing cortisol values) but they also assess different cortisol fractions. Plasma samples reflect levels of cortisol bound to protein as well as biologically active free cortisol (unbound), whereas urine and salivary samples reflect only the levels of free cortisol (Peters et al. 1982; Kaye and Crapo 1990; Bonnin et al. 1993).

Characteristics of the stressor. A stressor classification was used ($\kappa = 1.00$) so that each stressor fell into one of four types: Occupation-based stressors (coded 1; including job strain, burnout, work related noise exposure, unemployment or academic stressors); social stressors (coded 2; including events such as marital conflict, loneliness, interpersonal stress and caregiving); medical procedures (coded 3; including surgery and dental procedures); sports (coded 4). It should be underscored that this does not mean that caregiving is being equated with, say, marital conflict. For the purpose of the present investigation caregiving was considered a chronic stressor, whereas marital conflict was considered to be acute (or subchronic) because it was conducted within an experimental setting.

Additionally, whether or not the stressor was: (1) acute/chronic [$\kappa = 1.00$ (acute was coded as 0 and chronic was coded as 1)]; (2) predictable [$\kappa = 0.78$ (no was coded as 0, and yes was coded as 1)]; (3) had an uncontrollable or unpredictable *outcome* [$\kappa = 0.77$ (no was coded as 0, and yes was coded as 1) and included and (4) an evaluative component [$\kappa = 0.77$

(defined as 'being judged or evaluated' with no being coded as 0, and yes coded as 1]) was also coded.

Statistical analysis

Effects sizes: Meta-analytical technique. For cortisol analyses, we calculated the effect size of the difference between conditions (e.g. cortisol concentrations at baseline compared to those following a stressor) or between groups (e.g. cortisol concentrations of those individuals not suffering from burnout compared to those of participants suffering from burnout) using the standardized-mean change statistic, Cohen's *d*, which is appropriate for repeated measures effect size estimates (Becker 1988; Dunlap et al. 1996; Morris 2000). This statistic can be interpreted as the magnitude of the difference between pre- and post-stressor cortisol values in standard deviation units [$d = (\text{Mean post stressor level} - \text{Mean pre stressor level}) / \text{pooled SD}$] and were further corrected for sample size bias (Hedges and Olkin 1985). The direction of the effect size was positive if the study reported an increase of cortisol levels from pre- to post-stressor. Cohen classifies an effect size (*d*) of 0.20 as small, 0.50 as moderate, and 0.80 as large (Cohen 1977).

To the extent that they could be, effect sizes were calculated using the means and standard deviations provided. However, for studies in which these statistics could not be found in the article or directly obtained from the author, inferential statistics were used (Hedges and Olkin 1985; Rosenthal 1991). Specifically, for the between-subject designs, the effect size *r* was first computed from *t* or *F* statistics with 1 df in the numerator using the formulae provided by Rosenthal and DiMatteo (2001) and then transformed into Cohen's *d*, [$r = (t^2/t^2 + df)^{1/2}$ or $r = (F/F + df_{\text{error}})^{1/2}$; $d = (4r^2/(1 - r^2))^{1/2}$]. For within-subjects designs, the formula $d = tc\{2/[n(1 - r^2)]\}^{1/2}$ or $d = (F)^{1/2}\{2/[n(1 - r^2)]\}^{1/2}$ was used in order to control for possible effect size overestimation (Dunlap et al. 1996). *F* and *tc* correspond to the values reported in the text comparing the pre- and post stressor conditions. Frequently, the value of *r* was not provided in the text. Therefore, a value of 0.40 was used to calculate the effect sizes. Additional analyses indicated that *r* values from 0.20 to 0.60 did not alter the results of the analyses.

For those studies that only reported significant results, we assumed $p < 0.05$ and this value was used to calculate the effect size, whereas those studies reporting "no significant effect" were given an assumed effect size $d = 0.00$ (Rosenthal 1991). Such inferences were made for 40 studies; importantly, omitting these studies did not alter the results of the analyses. Even if diurnal studies comprised several cortisol samples, separate *ds* were calculated for each stressor-related cortisol assessment relative to a control group not exposed to the naturalistic stressor in question. In order

to maximize the condition of independence in the meta-analysis, when saliva samples were collected on multiple days from the same participants (e.g. job strain among bus drivers; Aronsson and Rissler 1998), only effect sizes from the first day of the study were calculated and used in the analyses.

The hypotheses were tested through regression analyses, comparisons of means (*t*-tests to determine that effect sizes differed from 0), analyses of variance (ANOVAs) to compare stressor types and characteristics. Effect sizes served as the dependent variable in all the analyses, whereas methodological factors and participant characteristics served as predictor variables in the regression analyses.

Results

Sample of studies selected

A total of 140 studies met the inclusion criteria for the present analysis resulting in 181 effects sizes. In total, 10,976 participants (mean age = 38.3 years old, SD = 14.0) of whom 47% were females (range = 0–100%, SD = 0.37), contributed to this study. Saliva collection was the most frequent method of cortisol assessment ($n = 112$; 61.9%), followed by plasma measurements ($n = 47$; 26.5%) and urine measurements ($n = 19$; 10.5%). The majority of studies were conducted in the morning (AM; $n = 80$; 44.2%), whereas some were conducted in the afternoon (PM; $n = 35$; 19.3%), throughout the day (Diurnal; $n = 19$; 10.5%), at varied times throughout the day (AM/PM; $n = 16$; 8.8%) or at unspecified times ($n = 31$; 17.1%).

Methodological factors: Time of day and method of cortisol collection

As alluded to earlier, given that the time of day that samples were collected may influence the observed fluctuations, coupled with the fact that the method of collection (saliva, blood, urine) influences different cortisol fractions, the effect sizes were first regressed separately onto dummy coded variables representing the four time periods (i.e. time of day was dummy coded using three variables and type of cortisol collection dummy coded using two variables).

Time of day did not influence the effect size associated with the stressor experience, $R^2 = 0.001$, $F(3,143) < 1$, ns, even though this variable explained 5.0% of the between-study variance. Furthermore, the method of sampling for cortisol measurement was not a significant predictor of the effect size, $R^2 = 0.012$, $F(2,176) = 1.05$, ns.

Participant characteristics

A regression of the effect sizes for stressor-induced cortisol variations as a function of participant

characteristics (number of participants, mean age and gender composition) was also conducted. This regression analysis was not significant, $R^2 = 0.035$, $F(3,128) = 1.57$, ns, indicating that neither the number of participants, the gender composition, nor the mean age of the sample were significant predictors of the effect sizes. Given that none of the methodological factors (type of sample for cortisol collection, time of day) or participant characteristics qualified as significant predictors of the effect sizes, these variables were not considered in subsequent analyses.

Types of stressors used

The majority of the naturalistic studies involved occupational stressors ($n = 112$; 61.9%), whereas the remaining studies comprised social stressors ($n = 33$; 18.2%), medical procedures ($n = 13$; 7.2%), and sports-related stressors ($n = 23$; 12.7%). Table I provides a summary of the studies used in the present analysis as a function of the type of stressor used, the characteristics of the stressor, and the time of day at which the study was conducted. The average effect size and the primary coded dimensions for each study are provided in Table II.

Effect sizes and stressor types

A summary of the effect sizes for all stressor types is provided in Table III. As indicated by t -tests and the 95% confidence intervals involving the various stressor categories, it appeared that average effect size across studies differed from 0 (i.e. the stressors had a significant effect), and this was the case for each of the stressor categories.

Furthermore, univariate analyses of effect sizes indicated a significant effect of type of stressor, $F(3,177) = 6.00$, $p < 0.001$, $\eta^2 = 0.092$. Follow-up comparisons indicated that there were significant differences between medical and occupational stressors (Mean Diff = 1.09, $p < 0.05$), with medical stressors demonstrating the strongest effects on cortisol levels. As well, sports stressors promoted stronger effects on cortisol levels than did occupational stressors (Mean Diff = 0.96, $p < 0.01$). No further differences were found between stressor types.

Stressor characteristics

In order to determine which stressor characteristics explained additional variance of effect sizes, a 2 (chronicity) \times 2 (predictability) \times 2 (controllability) \times 2 (evaluative) between subjects ANOVA was conducted. A main effect of chronicity was found, $F(1,167) = 8.21$, $p < 0.01$, $\eta^2 = 0.047$, in that acute stressors had greater effect sizes than did chronic stressors (Table IV). Levene's test of homogeneity of variances indicated that error variance across stressor categories were not equal, $F(8,167) = 3.49$, $p < 0.001$. Specifically, in order to determine whether the inclusion of medical and sports stressors in the sample biased the results (and indeed, these were shown to have greater effects than occupational stressors), a second analysis was conducted in which these two categories of stressors were excluded [i.e. the analysis only included occupational stressors (i.e. academic stressors, job strain) and social stressors]. A similar 2 (chronicity) \times 2 (predictability) \times 2 (controllability) \times 2 (evaluative) ANOVA was

Table I. Characteristics of the studies.

Type of stressor task	No. of effect sizes	Subjects	Methodological characteristics		Stressor characteristics			
			Time of day	Collection	Acute	Unpredictable	Uncontrollable	Evaluative
Occupational	112	7744	47 a.m.	65 saliva	30	22	58	29
			17 p.m.	28 blood				
			16 a.m./p.m.	17 urine				
			13 diurnal	2 ns				
Social	33	2304	19 ns		3	30	32	31
			15 a.m.	24 saliva				
			11 p.m.	8 blood				
			6 diurnal	1 urine				
Medical procedures	13	574	1 ns		13	10	13	0
			7 a.m.	5 saliva				
			2 p.m.	7 blood				
			4 ns	1 urine				
Sports	23	354	11 a.m.	18 saliva	21	0	0	21
			5 p.m.	4 blood				
			7 ns	1 ns				
Total	181	10,976						

Table II. Methodological characteristics and average effect sizes for the studies included in the meta-analysis.

	Author/date	N	Gender*	Age	Time [†]	Collection [‡]	d [§]
<i>Occupational</i>							
<i>Academic Examination</i>	Frankenhaeuser et al. (1978)	30	0.61	19	a.m.	Urine	1.06
	Johansson et al. (1983)	2	0.50	31	Diurnal	Urine	0.27
	Kirkeby et al. (1984)	9	1.00	26	–	Blood	0.17
	Hellhammer et al. (1985)	9	0.00	–	–	Saliva	2.14
	Lovallo et al. (1986)	28	0.00	23	a.m.	Blood	1.12
	Meyerhoff et al. (1988)	11	0.00	25	a.m.	Blood	2.33
	Herbert et al. (1986)	38	0.00	–	a.m.	Blood	1.97
	Semple et al. (1988)	9	–	21	p.m.	Blood	0.69
	Johansson et al. (1989)	27	1.00	27	a.m.	Blood	0.05
	Evans et al. (1994)	7	–	–	Diurnal	Saliva	0.87
	Glaser et al. (1994)	45	0.00	–	p.m.	Blood	0.00
	Malarkey et al. (1995)	55	0.00	22	Diurnal	Blood	0.00
	Guidi et al. (1999)	28	0.29	–	a.m.	Blood	1.19
	Song et al. (1999)	38	0.66	–	p.m.	Saliva	0.06
	Lacey et al. (2000)	15	0.50	27	a.m./p.m.	Blood	2.38
	Vedhara et al. (2000)	60	0.40	22	Diurnal	Saliva	0.20
	Ennis et al. (2001)	36	0.76	24	a.m.	Urine	0.86
	Ng et al. (2003a)	11	0.18	33	a.m.	Saliva	0.94
	Ng et al. (2003b)	31	0.32	23	a.m.	Saliva	– 4.52
	Al-Ayadhi et al. (2005)	48	1.00	20	a.m.	Blood	1.78
	Droogleever Fortuyn et al. (2004)	15	0.20	35	p.m.	Blood	0.19
	Gaab et al. (2006)	15	0.40	24	a.m.	Saliva	1.39
	Weekes et al. (2006)	67	0.51	20	p.m.	Saliva	0.28
<i>Driving examination</i>	Dugue et al. (2001)	7	0.00	18	–	Blood	0.21
<i>Job Strain</i>	Caplan et al. (1979)	85	0.00	40	Diurnal	Urine	0.49
	Rose et al. (1982)	195	0.00	–	–	Blood	0.22
	Harenstam and Theorell (1990)	1034	–	–	a.m.	Blood	0.00
	Schreinicke et al. (1990)	77	0.00	43	a.m.	Saliva	0.50
	Zeier (1994)	22	0.03	–	a.m.	Saliva	0.57
<i>Job Strain (continued)</i>	Zeier et al. (1996)	16	0.00	42	a.m.	Saliva	0.61
	Burton et al. (1996)	115	0.57	–	p.m.	Saliva	0.00
	Fujigaki and Mori (1997)	10	0.00	28	p.m.	Saliva	0.00
	Aronsson and Rissler (1998)	10	0.50	30	a.m.	Urine	1.10
	Schulz et al. (1998)	12	0.51	26	a.m./p.m.	Saliva	0.00 – 0.61
	Sluiter et al. (1998)	10	0.00	47	a.m.	Urine	0.18 – 1.15
	Steptoe et al. (1998)	61	0.62	35	–	Saliva	– 0.55
	Fischer et al. (2000)	139	0.81	–	Diurnal	Saliva	0.27
	Hanson et al. (2000)	77	0.44	40	a.m.	Saliva	0.00
	Rissen et al. (2000)	31	1.00	44	–	Saliva	0.15
	Sluiter et al. (2000)	115	0.00	37	–	Urine	0.00
	Steptoe et al. (2000)	105	0.61	39	a.m.	Saliva	0.50
	Ganster et al. (2001)	198	1.00	–	–	Saliva	0.95
	Sluiter et al. (2001)	55	0.00	40	Diurnal	Urine	0.41
	Ohlson et al. (2001)	103	0.92	–	a.m.	Blood	0.00
	Yang et al. (2001)	73	–	30	a.m./p.m.	Saliva	0.75
	Lundberg and Hellstrom (2002)	82	–	–	a.m.	Urine	0.69
	Lundberg and Frankenhaeuser (1999)	60	0.50	44	a.m./p.m.	Urine	0.05 – 0.51
	Hansen et al. (2003)	101	1.00	41	a.m.	Blood	3.43
	Sluiter et al. (2003)	20	0.00	42	–	Saliva	0.19
	Weibel et al. (2003)	8	0.88	43	Diurnal	Saliva	0.50
	Fujiwara et al. (2004)	8	1.00	28	a.m./p.m.	Saliva	– 0.13
	Kunz-Ebrecht et al. (2004a)	124	0.46	52	a.m.	Saliva	0.54
	Kunz-Ebrecht et al. (2004b)	75	0.49	–	Diurnal	Saliva	0.67
	Otte et al. (2005)	76	0.13	28	–	Saliva	0.59
	Schlotz et al. (2004)	214	0.53	49	a.m.	Saliva	0.04
	Steptoe et al. (2004)	91	0.47	–	–	Saliva	0.59
	Dahlgren et al. (2005)	25	0.63	47	Diurnal	Saliva	1.68

Table II – continued

	Author/date	N	Gender*	Age	Time†	Collection‡	d [§]
	Ritvanen et al. (2006)						
	Low vs. high stress (young)	14	1.00	31	a.m.	Blood	0.19
	Low vs. high stress (old)	14	1.00	54	a.m.	Blood	-0.05
	Smith et al. (2006)	32			a.m.	Blood	4.00
<i>Commuting</i>	Evans and Wener (2006)	208	0.48		a.m.	Saliva	0.38
<i>Burnout</i>	Melamed et al. (1999)	89	0.04	43	a.m./p.m.	Saliva	0.81
	Pruessner et al. (1999)	66	0.64	44	a.m.	Saliva	-0.95
	de Vente et al. (2003)	46	0.36	42	a.m.	Saliva	0.13 - 0.59
	Moch et al. (2003)	16	1.00	38	a.m.	Blood	-2.30
	Grossi et al. (2003)	63	-	48	a.m.	Blood	0.00
	Grossi et al. (2005)	42	0.52	40	a.m.	Saliva	0.27
	Mommersteeg et al. (2006a)						
	Control vs. burnout (awakening)	94	0.55	43	a.m.	Saliva	-0.05
	Control vs. burnout (15 min after Awakening)	94	0.55	43	a.m.	Saliva	0.14
	Mommersteeg et al. (2006b)	33	0.68	45	a.m.	Saliva	1.20
	Mommersteeg et al. (2006c)	109	0.28	43.9	a.m.	Saliva	0.07
	Langelaan et al. (2006)	88	0.00	45	a.m.	Saliva	0.00
<i>Unemployment/socioeconomic status</i>	Kapuku et al. (2002)	24	0.00	19	-	Blood	-1.00
	Brenner and Levi (1987)	110	1.00	-	-	-	0.24
	Arnetz et al. (1991)	227	0.75	35	-	-	0.00
	Ockenfels et al. (1995)	60	-	-	am,pm	Saliva	0.25-0.50
	Toivanen et al. (1996)	162	-	-	a.m.	Blood	0.27
	Grossi et al. (1998)	11	0.37	42	a.m.	Saliva	0.27
	Decker (2000)	31	0.00	-	a.m./p.m.	Saliva	0.00
	Steptoe et al. (2005)	160	0.45	55	am,pm	Saliva	-0.44-0.07
	Wright and Steptoe (2005)	139	0.49	71	a.m.	Saliva	0.07
	Maier et al. (2006)	71	0.40	42	p.m.	Blood	0.38
	Steptoe et al. (2003)						
	Low vs. high grade (men)	73	0	53	Diurnal	Saliva	0.51
	Low vs. high grade (female)	63	1	52	Diurnal	Saliva	-0.59
<i>Noise chronic</i>	Melamed and Bruhis (1996)	35	-	-	-	Saliva	0.27
	Evans and Johnson (2000)	38	1.00	37	-	Saliva	0.02
<i>Public lecture</i>	Basset et al. (1985)	29	0.24	29	Diurnal	Urine	0.19
	Bolm-Audorff et al. (1986)	10	-	-	a.m./p.m.	Blood	1.42
	Lucas et al. (2006)	11	0.73	38	-	Blood	-0.18
<i>Medical procedures</i>							
<i>Surgery</i>	Manyande et al. (1992)	40	0.45	42	p.m.	Blood	1.47
	Manyande et al. (1995)	48	0.41	45	p.m.	Blood	1.06
	Dahanukar et al. (1996)	23	-	-	-	Blood	0.65
	Smith-Hanrahan (1996)	35	1.00	-	a.m.	Saliva	0.65
	Augustin et al. (1999)	45	0.56	34	a.m.	Saliva	0.27
	Kain et al. (1999)	56	0.50	62	a.m.	Blood	4.89
	Ozarda-Ilcol et al. (2002)	16	-	-	-	Blood	9.58
	Ilcol et al. (2004)	26	1.00	49	-	Blood	1.21
	Pearson et al. (2005)	39	0.31	71	a.m.	Urine	3.15
	Nicholson et al. (2005)	-	0.25	31	-	Blood	-3.23
<i>Dental procedures</i>	Benjamins et al. (1992)	13	0.46	35	a.m.	Saliva	1.87
	Miller et al. (1995)	196	0.00	35	a.m.	Saliva	2.75
	Brand (1999)	31	0.00	42	a.m.	Saliva	0.46
<i>Severe physical injury</i>	Offner et al. (2002)	22	0.38	35	-	Blood	4.15
<i>Social stressors</i>							
<i>Marital conflict</i>	Miller et al. (1999)	-	0.50	31	a.m.	Blood	0.27
	Kiecolt-Glaser et al. (2003)	138	0.50	25	Diurnal	Saliva	0.24
<i>Marital Conflict</i>	Heffner et al. (2004)	85	0.50	26	a.m.	Blood	-0.27
	Kiecolt-Glaser et al. (1996)	90	0.50	26	a.m.	Blood	0.27
<i>Abuse</i>	Pico-Alfonso et al. (2004)	162	1.00	46	a.m./p.m.	Saliva	0.00 - 0.61
<i>Loneliness</i>	Pressman et al. (2005)						
	Low vs. high loneliness (morning)	83	0.55	18	a.m.	Saliva	0.54
	Low vs. high loneliness (afternoon)	"	"	"	p.m.	"	0.53

Table II – continued

	Author/date	N	Gender*	Age	Time [†]	Collection [‡]	d [§]
<i>Interpersonal Stress Caregiving</i>	Hansen et al. (2006)	393	0.64	44	Diurnal	Saliva	-0.20
	Irwin et al. (1997)	133	0.58	70	a.m.	Blood	-0.32
	Bauer et al. (2000)	116	0.49	72	Diurnal	Saliva	0.49
	Cacioppo et al. (2000)	27	1.00	67	a.m.	Blood	-2.82
	Da Roza and Cowen (2001)	58	0.60	69	am,pm	Saliva	0.38 - 2.47
	Davis et al. (2004)	30	1.00	71.4	Diurnal	Saliva	1.31
	Provinciali et al. (2004)	75	0.79	54	-	Blood	0.00
	de Vugt et al. (2005)	112	0.65	60	Diurnal	Saliva	0.03
<i>Caregiving (continued)</i>	Wilcox et al. (2005)	35	1.00	65	a.m.	Saliva	-
	Gallagher-Thompson et al. (2006a)	22	1.00	51	a.m.	Saliva	0.67 - 1.93
<i>Bereavement</i>	Vedhara et al. (2002)	103	0.34	43	Diurnal	Saliva	0.92
	Jacobs et al. (1987)	56	0.50	62	Diurnal	Urine	3.22
<i>Sports</i>	Gerra et al. (2003)	14	0.57	38	a.m.	Blood	-0.42
	Urhausen and Kindermann (1987)	8	0.00	25	a.m.	Blood	4.04
	Booth et al. (1989)	6	-	-	-	Saliva	0.00
	Passelergue et al. (1995)	6	0.00	26	p.m.	Saliva	1.47
	Filaire et al. (1996)	31	1.00	19	a.m.	Saliva	0.27
	Kugler et al. (1996)	17	0.00	40	-	Saliva	3.29
	Eubank et al. (1997)	5	0.00	24	p.m.	Saliva	-0.48-2.41
	Gonzalez-Bono et al. (1999)	16	0.00	23	p.m.	Saliva	0.00
	Salvador et al. (1999)	28	0.00	18	a.m.	Blood	0.00
	Carre et al. (2006)	17	0.00	18	p.m.	Saliva	1.03
	Suay et al. (1999)	26	0.00	18	a.m.	Blood	3.41
	Bonifazi et al. (2000)	8	0.00	-	a.m.	Blood	2.29
	Filaire et al. (2001)	12	0.00	22	-	Saliva	4.13
	Wagner et al. (2002)	8	0.00	-	-	Saliva	0.57
	Bateup et al. (2002)	17	1.00	20	-	Saliva	0.33
	Iellamo et al. (2003)	7	0.43	27	-	Saliva	1.51
	Filaire et al. (2003)	20	0.00	25	a.m.	Saliva	0.00
	Salvador et al. (2003)	17	0.00	-	a.m.	Saliva	3.11
	Kivlighan et al. (2005)	12	0.50	-	-	Saliva	1.30
	Edwards et al. (2006)	40	0.45	20	p.m.	Saliva	0.00

* Denotes % female; † a.m. = morning; p.m. = afternoon; a.m./p.m. = during the day; ‡ Medium of cortisol collection; § Effect size estimate.

conducted and no significant main effects of stressor characteristics or interactions were observed (Table V). Levene's test of homogeneity of variances indicated that the error variance across stressor categories were not equal, $F(7,134) = 2.51$, $p < 0.05$. However, in order to keep the theoretical meaningfulness of the effect sizes, these data were not transformed.

In the analyses there were no other significant interactions involving the different stressor characteristics (i.e. stressor controllability, predictability,

chronicity and evaluative component). From this perspective, it seems that these variables did not promote synergistic actions, although this does not imply that they did not have additive effects with respect to cortisol variations.

Discussion

The objective of the present meta-analysis was to identify those factors that were fundamental in determining increased cortisol levels under naturalistic

Table III. Effect sizes of different stressor types.

Type of stressor task	No. of effect sizes	Subjects	d	Confidence interval (95%)
Occupational	112	7744	0.33**	0.12-0.56
Social Stressors	33	2304	0.63**	0.20-1.06
Medical Procedures	13	574	1.42*	0.16-2.68
Sports	23	354	1.29***	0.72-1.86
Total	181	10,976	0.58***	0.39-0.77

t-Tests indicated that effect sizes were significantly different from 0 at * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Table IV. Effect sizes of stressor characteristics across stressor types.

Stressor characteristic	Mean $d \pm SD$
<i>Chronicity**</i>	
Acute	0.76 \pm 0.40
Chronic	0.22 \pm 0.35
<i>Predictability</i>	
No	0.42 \pm 0.47
Yes	0.60 \pm 0.32
<i>Controllability</i>	
No	0.57 \pm 0.30
Yes	0.36 \pm 0.65
<i>Evaluative threat</i>	
No	0.68 \pm 0.27
Yes	0.21 \pm 0.61

** $p < 0.01$.

stressor conditions. It is understood, of course, that unlike discrete stressors employed in a laboratory context, stressors experienced *in situ* typically comprise compound adverse events and stimuli (e.g. medically related stressors may have implications for employment, and stressor experiences are often accompanied by rumination that may exacerbate the adverse experience). As these stressors share a common source, as well as several fundamental characteristics, they might be highly correlated with one another. Not surprisingly, the contribution of different elements of compound stressors often cannot readily be disentangled from one another, making it difficult to conclude whether observed neuroendocrine alterations are specific to a particular element of the stressor.

Factors such as time of day, gender, age and the medium used for cortisol analyses (blood, saliva, urine) have been found to influence hormone levels in human studies that involved laboratory stressors (Seeman and Robbins 1994; Dickerson and Kemeny 2004; Kudielka et al. 2004; Otte et al. 2005a,b; Burke et al. 2005). Although such factors are known to influence basal cortisol levels, they did not appear to influence the effect sizes of cortisol changes elicited by stressors in natural settings.

Table V. Effect sizes of stressor characteristics without medical and sports stressors.

Stressor characteristic	Mean $d \pm SD$
<i>Chronicity</i>	
Acute	0.53 \pm 0.46
Chronic	0.22 \pm 0.32
<i>Predictability</i>	
No	0.41 \pm 0.46
Yes	0.33 \pm 0.32
<i>Controllability</i>	
No	0.38 \pm 0.32
Yes	0.36 \pm 0.59
<i>Evaluative threat</i>	
No	0.53 \pm 0.30
Yes	0.11 \pm 0.55

A variety of different naturalistic stressors influenced cortisol levels ($d = 0.61$), but the overall effect size was moderate (Cohen 1988). Indeed, the magnitude of the cortisol increase was generally much smaller than that evident in stressed rodents (where 8–10-fold elevations are common), or in response to a laboratory challenge such as TSST, where ~2–4-fold increases have been reported (Kirschbaum et al. 1992, 1993, 1995, 1996; Biondi and Picardi 1999; Al'Absi et al. 2000; Gerra et al. 2001; Wolf et al. 2001; Dickerson and Kemeny 2004). Indeed, across studies it appeared that in response to naturalistic stressors, cortisol increase ranged from 0 to 180%.

Medical and sports stressors were particularly potent in stimulating HPA activity relative to occupational and social stressors. Given their nature, threat and potential severity, it is not surprising that medical stressors were associated with greater increases of cortisol compared to other type of stressors (Ellis and Humphrey 1982; Kehlet 1984; Douglas and Shaw 1989; Kincey and Satmore 1990). In addition to constituting a source of distress, anticipation and anxiety (Johnston 1986, 1988; Doering et al. 2000; Pearson et al. 2005), medical stressors may involve a pre-existing disease that acts to sensitize reactivity to stressful aspects of medical procedures, particularly surgery. Similarly, as indicated earlier, medical procedures might be associated with other experiential or circumstantial factors that place an additional burden on the individual (e.g. possible financial and personal burden; hospitalization may represent a marked change of environment, sleep cycle, eating habits, and so forth). Of course, it is also necessary to consider individual differences related to surgical history, the nature of the surgery (risk, success rate), and the individual's age (as a risk factor).

The relatively large cortisol increase in association with sports-related events might, at first, be considered surprising. However, the distress associated with such events may involve multiple factors. Among other things, these include concerns regarding the impact of a mediocre performance on an individual's career or status on the team, and the fear of embarrassment or of being condemned following a poor performance. Furthermore, sports-related events often entail a social-evaluative threat (performing in front of an audience) and anticipatory arousal, and thus might have been particularly potent in eliciting cortisol secretion (Dickerson and Kemeny 2004). Moreover, training leading up to the athletic competition itself, may place a strain on some physiological systems (e.g. cardiovascular system, pulmonary system), and hence cortisol changes may be secondary to such factors rather than just the psychogenic aspects of the stressor. The difficulties of assessing the impact of sports stressors on cortisol variations are compounded by the fact that some studies involved well-trained and experienced athletes,

whereas others used novices. Thus, differences in experience performing in public, self-confidence and self-esteem may all have contributed to variations of the cortisol response. Finally, it will be recognized that sustained exercise (especially among athletes) may result in hypothalamic–pituitary–gonadal neuroendocrine variations (e.g. in association with amenorrhea in females) that might not only influence basal cortisol levels (Ding et al. 1988; Brundu et al. 2006), but might also affect the response to stressors (McComb et al. 2006).

Although stimulated cortisol secretion for limited periods can be beneficial, as indicated earlier, prolonged elevations can promote several adverse physiological and psychological disturbances (e.g. hyperlipidemia, hypertension, chronic immunosuppression, dysphoria, affective disorders, cognitive disturbances and sleep disorders) (McEwen 2000; Sapolsky et al. 2000; McEwen and Wingfield 2003). Thus, when faced with a chronic stressor, it would be adaptive for cortisol functioning to be down-regulated. Indeed, the present meta-analysis revealed that across stressor types, those of an acute nature elicited greater cortisol changes than did chronic stressors. Furthermore, in studies that assessed the impact of acute and chronic stressors, a similar outcome was observed. By example, the cortisol rise associated with parachuting diminished over successive jumps (Deinzer et al. 1997), as did the response to chronic exercise (Wittert et al. 1996). Further, it seems that with chronic illness, biphasic changes of circulating cortisol level occur. The distress initially leads to high levels of cortisol, coupled with a reduction of the cortisol binding protein, corticosteroid-binding globulin, hence resulting in greater free cortisol levels. With continued distress hypocortisolemia may ensue (Beishuizen and Thijs 2004; Johnson and Rn 2006). These findings are in keeping with animal studies that indicated that the initially high levels of cortisol elicited by a stressor are abated with chronic exposure (Weiss et al. 1975; Nankova et al. 1993; Haleem and Parveen 1994; Armario et al. 2004). What is less clear, however, is whether the adaptation, such as that evident in chronic illness, represents an adaptation that reflects diminished distress and has beneficial value, or is actually a reflection of exhaustion of those processes governing HPA functioning (Beishuizen and Thijs 2004).

In humans, including the studies reviewed here, certain types of stressors tended to be more chronic than others (e.g. caregiving or job strain vs. academic examination or sports event), and frequently stressor chronicity and stressor type were confounded. Further to this same issue, certain stressors have a lengthy anticipatory period and/or a ruminative period following the actual experience. Thus, it is difficult in some instances to define when a stress experience began and when it ended. Finally, determining what constitutes a chronic stressor is dependent on a host

of individual difference factors as well as those related to the characteristics of the stressor itself (i.e. severity, controllability, predictability, threat). Thus, although it generally appeared that the cortisol response was diminished with chronic stressors, it needs to be re-emphasized that this may depend on other factors, including the severity of the stressor as well as other ongoing stressors being encountered.

In most studies that assessed cortisol levels associated with stressful experiences, these were done at specific time(s) within a day, but relatively few studies assessed diurnal cortisol variations, including the early morning increase of cortisol secretion. Yet, it will be recalled that the rise of morning cortisol (i.e. over the first 30 min following awakening) appears to be particularly sensitive to the influence of ongoing life stressors (Melamed et al. 1999; Pruessner et al. 1999; Schmidt-Reinwald et al. 1999). Just as chronic stressors have been associated with a dampening of the morning cortisol rise, it was also reported that morning cortisol release was diminished among individuals experiencing PTSD (Yehuda et al. 1995a,b, 1996; Boscarino 1996; Goenjian et al. 1996; Anisman et al. 2001; Abercrombie et al. 2004; Lauc et al. 2004; Pico-Alfonso et al. 2004; Griffin et al. 2005; Wessa et al. 2006), although contradictory data have been reported in this regard (DeBellis et al. 1994; Lemieux and Coe 1995; Young and Breslau 2004; Young et al. 2004; Inslicht et al. 2006). Among the studies showing attenuated morning cortisol increase (or a flattened diurnal cortisol profile), it did not seem that this effect was unique to any given stressor that involved threats to the person, having been reported among war veterans with PTSD (Lauc et al. 2004), severely abused women (Pico-Alfonso 2005), and women undergoing the chronic distress of metastatic breast cancer (Abercrombie et al. 2004).

Based on a meta-analytic review regarding the cortisol changes associated with chronic stressors, Miller et al. (2007) concluded that a variety of factors related to characteristics of the stressor, as well as person variables, determined the cortisol profile that emerged. Specifically, it was observed that, in general, chronic stressors were associated with reduced morning cortisol release, coupled with greater afternoon/evening secretion (and hence a flatter diurnal cortisol curve), resulting in a higher overall daily cortisol output. These outcomes were most pronounced for traumatic stressors and those that entailed physical threats. In contrast, stressors that influenced the social self (including those that elicited shame) tended to promote elevated morning and afternoon/evening cortisol levels. These cortisol variations tended to diminish with time following the stressful experience.

Given the apparently protracted effects of chronic stressors, even if these are diminished relative to acute

insults, the possibility ought to be considered that such events may give rise to a variety of pathological conditions associated with cortisol alterations, and these may be evident well after the stressor experience has ended. Indeed, these data raise the possibility that the processes leading to allostatic overload, and hence vulnerability to pathological outcomes, may be less related to the *magnitude* of the cortisol changes induced by the stressor than to the *chronicity* of these variations (McEwen 1998; McEwen and Seeman 1999). However, as far as we are aware, the possibility that chronic stressors, rather than stressor severity, may be most closely aligned with allostatic overload has not been reported in animals or in humans.

The present meta-analysis, as well as the discussion to this point, focused on the immediate effects of acute and chronic stressors on cortisol release. However, in assessing the impact of ongoing life stressors on cortisol reactivity, it might be considered that beyond their immediate effects, stressors may proactively augment neurochemical responses to subsequently encountered challenges (sensitization) (Anisman et al. 2003). This is particularly pertinent as such experiences may influence the recurrence of depressive symptoms, as well as development of PTSD (Post 1992; Breslau et al. 1999; Kendler et al. 2000; Heim and Nemeroff 2001; Penza et al. 2003). Essentially, stressors might influence vulnerability to later affective changes as the neurochemical substrates of the illness may evolve over time and repeated illness episodes. Studies in animals have indicated that although neuroendocrine adaptation occurs in association with a chronic stressor (although this effect may vary with the nature of the chronic stressor paradigm employed), when chronically stressed animals are subsequently exposed to a novel (heterotypic) stressor, an exaggerated cortisol response may be engendered (Armario et al. 2004; Dallman et al. 2004). The processes responsible for such an outcome have not been fully deduced and may involve either altered HPA feedback mechanisms or increased co-expression of ACTH secretagogues (CRH and arginine vasopressin (AVP)) at the external zone of the median eminence, that synergistically stimulate HPA activity (Tilders and Schmidt 1999; Anisman et al. 2003). For the present purposes the essential point is that stressors have ramifications on cortisol functioning that may persist long after the initial stressor experience has ended, provided that individuals again encounter a stressor (even if it differs from the initial traumatic or chronic event). Thus, although cortisol variations were reported to decline over time following a chronic stressor experience (Miller et al. 2007), this should not necessarily be interpreted as HPA reactivity being normalized. To the contrary, among some individuals, increased HPA reactivity may persist for extended periods.

Intuitively, one might expect that uncontrollable stressors would be more aversive and threatening than controllable stressors, and hence would lead to greater neuroendocrine variations. Ordinarily, when faced with an acute stressor, it would be adaptive for certain neuronal responses to be elicited rapidly, irrespective of the attributes of the stressors (e.g. controllable vs. uncontrollable; predictable vs. unpredictable). After all, when a stressor is initially encountered, especially if it is one that has not previously been experienced (making appraisal of the stressor difficult), a robust neuroendocrine response would assure adequate resources to deal with the stressor. Once appropriate appraisals of the situation have been made, and the threat is deemed to be modest or controllable, then the biological response might taper off. Indeed, those systems that are necessary for immediate responses to stressors (i.e. those that threaten the well-being of the organism), including activation of the sympathetic nervous system and even fundamental immune responses that act against pathogenic stimuli, should react rapidly and comparably to both controllable and uncontrollable stressors (Sapolsky et al. 2000). However, in actuality, it is often difficult to dissociate controllability or uncontrollability of a stressor from the chronicity of the stressor. In most instances, controllable stressors might be deemed to be acute (after all, if a stressor is perceived as being controllable, by definition it ought to be possible for the individual to terminate it, and hence its duration should be brief), whereas uncontrollable stressors can be either acute or chronic.

Summary and limitations

It appears that stressors experienced in natural settings were associated with increased cortisol release, but the magnitude of the effects were generally smaller than those observed in certain laboratory contexts. Not surprisingly, stressors related to medical procedures were most apt to promote elevated cortisol levels. Interestingly, the effect size associated with chronic stressors was smaller than that associated with acute stressors. Yet, given the possibility that stressors may engender sensitization of neuronal processes leading to changes in cortisol regulation, it should be considered that subsequent stressor experiences might elicit pronounced neuroendocrine responses.

The influence of social-evaluative threat found in the meta-analysis of laboratory stress studies by Dickerson and Kemeny (2004) might also manifest itself in the individual's self-evaluation, cognition, or beliefs about the self and might thus influence behavioral and neurochemical changes (Dickerson et al. 2004; Gruenewald et al. 2006). Such factors have not been intensively examined within a naturalistic setting, and hence their contribution to variations in cortisol secretion is uncertain. Nevertheless, it may

be significant that internalized racism (i.e. where the individual accepts the racist views of others as being correct) was associated with increased cortisol levels (Tull et al. 2005), which likely reflects low self esteem of these individuals. It was likewise reported that threats to one's group was associated with elevated levels of cortisol, particularly among individuals who tended to express higher levels of anger (Matheson and Cole 2004).

Despite the relatively large number of studies that have evaluated stressor-provoked changes in cortisol secretion, the influence of a great number of variables remains to be determined. In this regard, as already indicated, most life stressors are complex, typically comprising multiple strains. Thus, it is difficult to identify the unique contributions of each element of the stressor mosaic, although based on the present analysis it did not seem that experiencing stressors with multiple components (i.e. those stressors with several attributes that might augment cortisol levels, including stressors that are chronic, uncontrollable and unpredictable) synergistically enhanced cortisol levels. As well, limited information is available concerning the influence of individual difference factors (personality factors), stressor appraisal and coping strategies, as well as previous stressor experiences (including trauma) in determining HPA functioning. It is conceivable that the use of oral contraceptives influences cortisol levels in natural settings just as they were reported to do so in laboratory tests (Kirschbaum et al. 1999). However, as most studies did not report this information, nor was there typically any mention of phase of the menstrual cycle, their influence could not be assessed. These limitations notwithstanding, the present findings reinforce the view that changes in HPA activity represent dynamic processes to accommodate challenges, and may contribute to the provocation or exacerbation of various stress-related illnesses.

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