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Relations between Early Life Bonding and Adverse Experiences with both Symptoms of Depression and Diurnal Cortisol Patterns: Contributions of Contingencies of Self-Worth and Coping

A Thesis submitted to the Department of Psychology and the Faculty of Graduate Studies and Research of Carleton University in partial fulfillment of the requirements for the degree of

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Abstract

Previous stressful experiences, including those encountered early in life, may affect quality of life, well-being, and mood states. Although many variables may influence the impact of a potential stressor, it has been suggested that early life events, including poor parental bonding, may be fundamental in this respect. The present investigation demonstrated that perceived poor parental bonding was correlated with depressive mood and this relation was mediated by the coping styles endorsed. Although poor parental bonding was associated with increased propensity to use emotion-focused coping and a lower propensity to use problem-focused coping, the pathways by which poor maternal and paternal bonding lead to depressive symptomatology appeared to be different. Indeed, while poor paternal bonding seemed to be related to competition-based self-worth that may promote the use of emotion-focused strategies, the greater propensity to use emotion-focused strategies associated with poor maternal bonding was unrelated to competition-based self-worth. Furthermore, the present investigation indicated that previous stressful experiences influenced diurnal cortisol patterns as well as cortisol reactivity to a stressor. Specifically, participants that reported five or more traumatic experiences exhibited lower cortisol levels over the course of the day relative to participants reporting fewer traumatic experiences. In contrast, within a laboratory context, participants reporting early life distress exhibited heightened neuroendocrine stressor reactivity in response to reminder stimuli. Thus, although both early life experiences including poor parental bonding was associated with adult vulnerability to stressor-related pathology, the present investigation reflected different plausible pathways leading to such an effect. Indeed, while poor parenting styles appeared to strictly influence depressive symptoms, early life events appeared to be restricted to neuroendocrine activity.

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Introduction

Current stressors as well as previous stressful experiences, including those encountered early in life, may influence the individual's quality of life and may instigate affective illness. Indeed, both retrospective and prospective studies have indicated that depressive mood might be associated with antecedent stressor event (Abramson, Seligman & Teasdale, 1978; Anisman & Zacharko, 1992; Bifulco, Bernazzani, Moran et al. 2000; Billings & Moos, 1982, 1985; Brown, Harris & Eales, 1996; Cui & Vaillant, 1996; Daley, Hammen & Rao, 2000; Dura, Stukenberg, & Kiecolt-Glaser 1990; Griffith Ravindran, Merali, et al. 2000; Hammen, Davila & Brown, 1992; Hammen, Mayol & deMayo, 1986; Kessler, 1997; Monroe & Depue, 1991; Monroe et al., 1983, 1992; Monroe & Simons, 1991; Mundt, Reck & Backenstrass, 2000; Paykel, 2001). Certainly, not all individuals that encounter stressors, even those of a traumatic nature, fall into depression, nor is it the case that the development of depression is necessarily tied to stressful experiences (Judd, Akiskal & Paulus, 1997). Rather, there appear to be a constellation of factors that increase vulnerability to depressive illness, and conversely resilience factors exist that may protect or buffer the individual from the adverse effects of stressors (Anisman & Matheson, 2005).

Among the many variables implicated as vulnerability factors for depression, adverse early life events (poor parental bonding and traumatic experiences) are thought to have particularly marked effects in this respect. The processes by which such events influence adult vulnerability to pathology have not been fully elucidated, but several cogent propositions have been advanced. These have ranged from neurochemical explanations, wherein early life adversity sensitizes neuronal processes, leading to greater

reactions to subsequently encountered stressors (Meaney, 2001), through to those that focus on stressor appraisal processes (e.g. Lazarus & Folkman, 1984). Further, there is reason to believe that early life events may influence self-perception (trait self-esteem), self-esteem under certain conditions (state self-esteem) or with respect to certain functional domains of esteem (contingencies of self-worth), and these might mediate the response to stressors and the course of depressive illness. One purpose of the present investigation was to examine the relation between early life events and depression, with particular consideration given to the contribution of contingencies of self-worth in influencing this relation.

A second objective of the present investigation was related to diurnal variations in the release of the stress hormone, cortisol, and whether variations of cortisol would be related to previous stressful experiences. While it is well documented that stressful events give rise to the release of cortisol (Sapolsky, Romero & Munck, 2000), the occasions wherein stressful events provoke such an outcome are actually relatively limited, and the magnitude of the effects observed are far less pronounced than those seen in animal studies. Furthermore, although the literature concerning the effects of life stressors have generally not revealed adverse events to elicit proactive effects on cortisol levels, it does appear that ongoing stressors, such as job strain, may be associated with elevated morning cortisol levels (Steptoe, Cropley, Griffith & Kirschbaum, 2000). In contrast to such stressors, however, traumatic events that culminate in posttraumatic stress disorder (PTSD), have been associated with reduced cortisol levels (Yehuda, 2002). The processes responsible for these divergent effects are unknown, but there is reason to believe that both traumatic and chronic stressors may attenuate the diurnal

pattern of cortisol release (ordinarily high in the morning and then declining over the course of the day) (Preussner, Hellhammer & Kirschbaum, 1999).

In the present investigation, it was of interest to establish whether early life stressors and/or poor parental boding would influence diurnal cortisol pattern as well as contingencies of self-worth and coping methods used to deal with stressors, and whether the latter would mediate the relation between early life events, depressive symptoms and cortisol reactivity. It was hypothesized that stress reactions might be particularly pronounced if the stressor challenges the individual's self worth. Moreover, the stress reaction might be more pronounced among individuals that had encountered early life adversity, as such events could promote the sensitization of stressor-related neuronal circuits.

Stress: Immediate and Protracted Effects

Stress: Immediate Effects

Stressors give rise to a stress response that encompasses a variety of adaptive behavioural and physiological outputs that alter the body's internal milieu (allostasis) Ordinarily, under stressful circumstances certain behaviors, notably those associated with arousal, vigilance, and coping processes predominate, while behaviors that are not productive in a defensive capacity (e.g., sexual and feeding behaviours) may be suppressed. Concurrently, stressor-sensitive brain regions, including various amygdala nuclei, medial prefrontal cortex, locus coeruleus, and hypothalamic nuclei are activated, presumably in order for the individual to deal effectively with the ongoing challenge. As well, hypothalamic-pituitary-adrenal (HPA) activation is elicited, culminating in the

release of glucocorticoids (GC) by the adrenal glands (Chrousos & Gold, 1992; Herman & Cullinan, 1997). 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, 2000).

Of course, not all individuals respond to stressors in a unitary fashion and not all stressors have the same impact. Several organismic variables (genetic, age, gender), situational factors, and characteristics of the stressor itself, such as its severity, predictability and chronicity, differentially influence behavioural and neurochemical responses (Anisman, Zalcman, Shanks & Zacharko, 1991; Sapolsky et al., 2000). For example, in animal studies, the impact of chronic stressors is very different from those associated with acute stressors. While acute stressors provoke a reduction of monoamines (norepinephrine and serotonin) within several brain regions, chronic stressors may promote increased amine synthesis and/or moderation of utilization, and as a result amine levels may increase, equalling or exceeding basal levels (Anisman et al., 1991; Anisman & Zacharko, 1982; Haleem & Parveen, 1993; Pitman, Natelson, Ottenweller et al. 1995). Further to the same point, in mice there are some strains that are hyper-reactive to stressors, exhibiting profound variations of HPA functioning and monoamine activity, whereas other strains appear to be fairly resilient and exhibit relatively modest neurochemical changes (Anisman et al., 1991). In the same fashion, it is thought that in humans, previous stressor experiences, as well as individual difference factors, influences the neurochemical consequences of acute stressors, and hence may affect vulnerability to pathology.

Stress: Protracted Effects

In addition to their immediate effects, antecedent stressors may proactively influence the response to subsequently encountered stressors (Post, 1992; Tilders & Schmidt, 1998) and might thus come to affect mood states. The processes by which previous aversive events give rise to alterations of stressor reactivity and the emergence of psychological disturbances remain to be elucidated. It has been suggested that this outcome might result from the sensitization of the 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 hormone (Tilders & Schmidt, 1999) that have been implicated in depression as well as PTSD (Maes & Meltzer, 1995; Nemeroff, 1996; Yehuda, 2002).

Protracted Effects and Adverse Early Life Events

The proactive effects of stressors are particularly notable among individuals that had encountered distress early in life (Meaney, 2001; Anisman, Zaharia, Meaney & Merali, 1998), and such experiences are considered as important risk factors for the development and persistence of both physiological (e.g. immune disturbances, heart disease, hypertension, diabetes) and psychological pathologies (e.g. depression, PTSD, anxiety disorders) in adulthood (Brown, 1993; Cohen, Miller & Rabin, 2001; Herbert & Cohen, 1993; Sapolsky et al., 2000). Furthermore, it is thought that in humans various forms of early distress, such as maltreatment (e.g. Famularo, Kinscherff & Fenton, 1992; Pelcovitz, Kaplan, Goldenberg et al. 1994), childhood abuse (e.g. Felitti, Anda,

Nordenberg et al. 1998; Mullen, Martin, Anderson et al. 1996; Saunders, Villeponteaux, Lipovsky et al. 1992; Stein, Murray, Cooper et al. 1996), early parental loss (Agid, Shapira, Zislin et al. 1999; Kendler Neale, Kessler, et al, 1992) and even prenatal distress (Hulshoff et al. 2000), may be associated with increased rates of depression and post-traumatic disorder (PTSD) (Saunders et al., 1992). To be sure, there is no reason to believe that the effects of these divergent stressors are equipotent in promoting pathology, nor that any given stressor is more potent than another across all individuals. Nevertheless, there is a substantial body of literature that suggests that stressful events experienced in childhood may have protracted ramifications on adult well being and on the ability to contend with stressors encountered during adulthood (Meaney, 2001).

Early life Bonding

It has been frequently argued that adverse parent-child relations may increase adult vulnerability to depression (Bifulco et al., 2000; Bowlby, 1980; Kessler, Davis & Kendler, 1997; Sroufe, 1997). This not only refers to relatively intense adverse experiences (e.g., neglect), but also perceived impoverished parental care-taking, particularly the degree of care and affectionless control exerted by parents (Martin & Waite, 1994; Parker, 1979, 1993; Rodgers, 1996). Indeed, both clinical and experimental (community-based) studies have supported the view that risk of depression was associated with dysfunctional parenting (Fergusson, Woodward & Horwood, 2000; Kerver, van Son & de Groot, 1992; Lara, Klein & Kasch, 2000; Lizardi & Klein, 2000; Parker, Hadzi-Pavlovic, Greenwald & Weissman, 1995). As these studies were retrospective in nature, it is possible that the perceptions of depressed individuals colored the memories or perceptions of early life or those individuals were casting for someone to

blame for their current distress. However, the relationship between dysfunctional parenting and depressive illness was not based solely on the perceptions of the depressed respondents, but also on the testimony of corroborative witnesses (Parker, 1981; Parker, Gladstone, Wilhem et al., 1997). As well, correspondence was obtained in the recollections of parental styles reported in twin sets (Parker, 1986).

The relation been dysfunctional parenting and depression can potentially be attributed to any number of factors. Particular attention was, however, given to the proposition that insecure attachment style favored the individual developing diminished self-esteem, which then predisposed them to depressive illness (Matheson, Cole, Tannenbaum, Dodd & Anisman, 2005; Miller, Warner, Wickramaratne & Weissman, 1999; Parker, 1993; Roberts, Gotlib & Kassel, 1996; Strage, 1998).

The view has been expressed that the development of a secure relationship between parents and children may influence the individual's interaction with the environment, affecting exploration and discovery, development of social skills, and ultimately self-confidence (Ainsworth, 1985). Having a secure base permits individuals to engage in activities that promote the development of effective coping strategies, such as seeking social support or problem-solving (Compas, Connor-Smith, Saltzman, Thomsen & Wadsworth, 2001; Sarason, Sarason & Pierce, 1990). In contrast, poor parenting and the resultant reduction of self-esteem could result in the adoption of ineffective or inappropriate methods of coping with stressors (Compas et al., 2001; Strage, 1998).

Although it is often thought that the early development is critical in the formation of attitudes and coping, parental attachment throughout adolescence may have important implications for well being, including mood disorders, substance abuse, eating disorders,

and personality disorders (Burge, Hammen, Davila et al., 1997; Garrison, Waller, Cuffe et al., 1997).

Stressful Events and Depression

There is an abundance of information implicating stressful events in the precipitation of exacerbation of depressive symptoms. The stressors that precede depression may involve a series of slight hassles (Kanner, Coyne, Schaefer et al. 1981; Monroe & Simons, 1991) or they may involve relatively severe stressors, particularly those of a psychosocial nature (Goodyer, Herbert, Tamplin, et al. 2000; Monroe, Rohde, Seeley et al. 1999; Paykel, 2001; Williamson, Birmaher, Frank et al. 1998). Interestingly, however, while first episodes of depression are associated with antecedent stressful events, later episodes are not tied to obvious stressors (Kendler, Thornton, & Gardner, 2000; Kendler, Thornton & Prescott, 2001; Kessler, 1997; Lewinsohn, Allen, Seeley et al. 1999; Solomon, Keller, Leon et al. 2000). From the neurochemical perspective, it is possible that once sensitized, even innocuous events may promote neurochemical alterations that come to provoke adverse outcomes (Anisman & Matheson, 2004; Post, 1992). It is equally possible, however, that the initial experience shatters beliefs and assumptions about the world (Janoff-Bulman, 1992), hence altering appraisals of later stressor experiences, thus leading to depression.

Importantly, not all stressors have the same impact and some seem to be more efficient than others in eliciting depressive symptoms. Social loss tends to be closely related to depression (Brown, Bifulco & Harris, 1987; Monroe & Depue, 1991;Roy, 1983; 1985), whereas anticipation of adverse events tends to be related to anxiety (Paykel, 1982; Reno & Hillaris, 1990). Furthermore, adverse achievement-related events (e.g., work-related stress) seem to be more effective than interpersonal events (e.g., death

of a loved one) in provoking depression (Mazure, Bruce, Maciejewski et al. 2000; Tennant, 2001). Interestingly, psychosocial stressors may have more dramatic consequences among females than in males, whereas those related to job strain/competition generally have greater effects in males (Mazure et al. 2000; Kendler et al., 2001).

Coping and Personality Variables

Stressful Events and Coping Strategies

Coping strategies play a significant role in determining the ultimate impact of stressful life events on well-being. According to the transactional model of stress proposed by Lazarus and Folkman (1984), the behavioural and cognitive processes involved in response to stressors as well as in vulnerability to psychopathology are influenced by the individual's subjective appraisals, and the perceived availability of coping methods. Appraisals, from this perspective, refer to "the cognitive interpretation or representation an individual assigns to a potentially stressful (or positive) event" (Lazarus & Folkman, 1984), while coping has been defined as "the cognitive and behavioural efforts to manage external and internal demands that are appraised as taxing or exceeding an individual's resources" (Lazarus & Folkman, 1984).

According to this framework, the relation between appraisal and coping is interactive, wherein appraisals influence the coping strategies selected and perceived coping efficacy influences appraisal. Essentially, individuals evaluate the possibility of contending with the stressor by engaging in a risk assessment (determining what is at stake when confronted with a challenge) and evaluating coping resources available. Ordinarily, individuals appraise a stressor and the risk to their well being based on their

attribution style (Abramson, Seligman & Teasdale, 1978; Needle & Abramson, 1990), their perception of control (Alloy & Clemens, 1992) and their ability to cope (Lazarus & Folkman, 1984, Lazarus, 1993). The situation is perceived as stressful, if the risk to well being exceeds available resources. In effect, a stressful experience reflects the subjective perception of environmental threats as exceeding one's coping capabilities.

While coping styles (coping as a trait characteristic) may be an important element in determining the coping efforts individuals express, it seems that coping is a dynamic process subject to change as the situation demands. That is, while coping styles set the stage for the specific methods used to deal with stressors, the coping strategies endorsed may actually be situation-specific. Presumably, any number of factors could determine coping styles and strategies, including certain personality characteristics, such as self-efficacy (Cozarelli, 1993; Major, Richard, Cooper et al.1998), self-esteem (Matheson & Anisman, 2005) and optimism (Scheier, Weintraub & Carver, 1986).

Coping strategies have frequently been described as falling into either *emotion-focused coping* (efforts to reduce the distress and negative feelings that arise from the stressful situation) or *problem-focused coping (*efforts to overcome the threatening event or diminish its impact) (Lazarus & Folkman, 1984). Typically, it has been concluded that that emotion-focused strategies are detrimental to well-being, whereas problem-focused coping strategies are beneficial (Billings & Moos, 1981; Carver, Scheier & Weintraub, 1989; Folkman & Lazarus, 1980, 1985). Although most stressors elicit both types of coping, problem-focused efforts tend to predominate when individuals feel that something constructive can be done, whereas emotion-focused coping tends to

predominate when it is felt that the stressor is something that must be endured (Folkman & Lazarus, 1980).

This distinction, as well as others that attempt to categorize coping into limited "sets", may be somewhat simplistic. Although these two categories are easily distinguished in principle, some coping strategies cannot easily be classified into emotion versus problem-focused categorizations. Certain coping strategies may have multiple functions that fit into both categories. For instance, social support may be used to vent emotions (emotional expression), or as a source of information (problem solving). Furthermore, coping strategies endorsed may co-occur, and may have additive or non-additive effects in promoting or buffering against the development of psychopathology. For example, individuals may employ rumination in conjunction with strategies such as problem solving, cognitive restructuring (re-evaluating the relation between the person and the threat), wishful thinking, religion, humor, emotional expression, and so forth (Carver et al. 1989; Endler & Parker, 1994; Nolen-Hoekseman et al. 1994; O'Brien & de Longis, 1996). If rumination is used together with problem focused coping it may result in adaptive coping efforts. In contrast, if used in conjunction with emotional expression or wishful thinking, the strategy may not have redeeming qualities. Thus, coping effectiveness may not depend so much on whether problem or emotion-focused coping styles are used, but rather on the specific combination of coping strategies endorsed. Given that coping represents a dynamic process, likely varying across situations and over time, Matheson & Anisman (2003) suggested that in order to understand the relation between coping and pathology a dynamic conceptualization of stress and coping is necessary, including the broad profile of responses endorsed.

Poor Coping and Depressive Symptoms

As already indicated, depression has been associated with poor or ineffective coping, including the diminished use of problem-focused coping, greater use emotion-focused strategies, reduced social support and satisfaction with the emotional component of this support (Holahan & Moos, 1991; Holahan, Moos, Holohan, & Cronkite, 1999; McNaughton et al. 1992; Parker Brown & Blignault, 1986; Ravindran, Griffiths, Merali & Anisman, 1996, 1998), and high levels of rumination (Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, 1998; Nolen-Hoeksema & Davis, 1999). Importantly, depression may be a cause as well as a consequence of poor coping strategies endorsed by depressed individuals (e.g., Griffith, Ravindran, Merali & Anisman, 2000). As mentioned earlier, stressful events might be antecedents of depression (e.g. Kessler, 1997) and the adoption of particular coping styles (e.g. rumination) has been predictive of the later depressive episodes (Nolen-Hoeksema, 1998). Further, it will be recalled that various experiential and individual factors could influence coping styles and hence the influence of such factors on depressive affect may be mediated by these factors.

Self-Esteem

Self-esteem, an important element that influences quality of life, refers to *global* judgments of self-worth, self-respect, or self-acceptance (Rosenberg, 1965) as well as *domain-specific* evaluations of aspect of the self in areas such as appearance, academics, and other's approval. Furthermore, both global and domain-specific self-esteem may be thought of as either a *trait*, being relatively stable over time (e.g., how do I feel about myself in general), or as a *state*, which fluctuates according to the immediate circumstance or situation (e.g., how do I feel about myself right now) (Rubin &

Hewstone, 1998). Although these subtypes can be differentiated on a conceptual basis, it should be considered that they might impact upon one another in a variety of ways. Importantly, state and trait self-esteem may influence appraisal of stressful events, especially those comprising threat or challenge to individual's self-worth (Rubin & Hewstone, 1998) and may influence the coping strategies that will be employed in certain circumstances (Matheson & Anisman, 2003).

Importantly, self-esteem has repeatedly been invoked as a contributing or an explanatory factor for several behaviors (Rosenberg, 1995). High self-esteem has been associated with more positive affect, more life satisfaction, less anxiety, less hopelessness and fewer depressive symptoms, whereas low self-esteem has been implicated in a several socially problematic and negative behavioural outcomes, such as depression, substance abuse, teenage pregnancy and eating disorders (Crocker, Luhtanen, Cooper et al., 2001; Crocker & Wolfe, 2001; Dawes, 1994; Mecca, Smelser & Vasoconcellos, 1989; Overholser, Adams, Lehnert et al. 1995). However, it was suggested that the relationship between low self-esteem and negative behavioural outcome has often been overstated, and the relationship is actually weaker and more scattered than typically assumed (Mecca, Smelser & Vasconcellos, 1989).

The role of self-esteem and perceived self-worth in depressive illnesses remains elusive (Maciejewski, Prigerson & Mazure, 2000; Shahar and Davidson, 2003). To be sure, self-esteem is diminished among depressed individuals, but it is less clear whether low self-esteem is a symptom of depression, an etiological factor, or a predictor of a later depressive episode (for reviews, see Bernet, Ingram & Johnson, 1993; Brown, Bifulco & Veiel, 1990). In particular, depressive symptoms may be predictive of diminished self-

esteem (Shahar & Davidson, 2003). Even after recovery, previously depressed individuals seemed to maintain their impaired self-esteem compared to never depressed individuals (Coyne & Calarco, 1995). These data suggest that depressive illness may promote diminished self-esteem, possibly owing to the depressive negative interpretations about him/herself and the world around them, as well as impaired social functioning.

In contrast to this view, low or unstable self-esteem has been posited to be predictive of depression, especially for individuals who experience life stress (Brown et al. 1990; Butler, Hokanson, & Flynn, 1994; Kernis, Whisenhunt, Waschull et al., 1998; Roberts & Kassel, 1997). Of course, self-esteem and depression may reciprocally influence one another. For example, while low self-worth may initially precipitate symptoms of depression, subsequent negative interpretations of both environmental and social cues may facilitate even greater decreases in self-esteem, which in-turn, may reinforce depressive symptomatology (Maciejewski et al., 2000).

Contingencies of Self-Worth

Certain domains may be more important than others for an individual's self-esteem and these may be quite different from one individual to the next. Some individuals may feel worthwhile and have good self-esteem if they are successful at school/work, attractive and competitive, whereas others may feel the same way about themselves if they are being virtuous, loved by God and have good relationships. Crocker and Wolfe (2001) defined the domains to which self-esteem is tied as contingencies of self-worth. From this perspective, when self-esteem is invested in a certain area, people become preoccupied with the meaning of events for their own self-

worth and value. Self-esteem is enhanced by success and threatened by setbacks and failure in these specific domains, which may then generalize to the worth and value of the whole person (Crocker, Karpinski, Quinn et al., 2003; Crocker et al. 2002). In effect, self-esteem may be particularly unstable when people experience challenges in domains important to their self-worth (Crocker, 2002; Crocker, Sommers & Luhtanen, 2002; Crocker & Wolfe, 2001) and this instability may be predictive of depression (De Man, Gutierrez & Sterk, 2001; Roberts & Kassel, 1997; Robert et al., 1995).

Some domains of self-worth may be grounded externally, largely relegated to factors beyond the individual's realm of control, while others are more grounded internally and perceived as being under the influence of the self (e.g. virtue and God's love). Still others reflect the influence of both internal and external factors (Crocker, & Wolfe, 2001). Self-esteem may be especially unstable if it is largely based on external contingencies such as appearance, other's approval, academics, etc. Indeed, an externally based contingency of self-worth may render self-esteem more vulnerable and reactive to daily boosts and threats. The instability of self-esteem, affect and belonging may take a toll on overall well being, resulting in the emergence of depressive symptoms over time (Gable & Nezlek, 1998; Kernis, Whisenhunt, Waschull et al. 1998; Roberts & Gotlib, 1996; Roberts, Kassel & Gotlib, 1995). In addition to over-reliance on external sources of self-esteem among depressive-prone individuals, a role for self-esteem instability (also referred to as lability) was suggested by Barnett and Gotlib (1988). Self-esteem lability refers to the excessive reactivity of self-esteem to daily threats and boosts. As lability increases, so does the effectiveness of life stressors to precipitate depressive symptoms.

High lability theoretically reflects marked risk for the onset of major depressive episode in the face of life stressors, such as the loss of a meaningful relationship or a job.

Early Life Events and Contingencies of Self-Worth

It is thought that contingencies of self-worth develop over the course of time in response to varied socialization and social influences (Bandura, 1986, 1991), ranging from parent child interactions (e.g. Bartholomew, 1990; Moretti & Higgins, 1990) to cultural norms and values (Solomon, Greenberg & Pyszczynski, 1991b). As such, it might be expected that adverse early life events might not only influence state and trait self-esteem, but might also influence the development of contingencies of self-worth and hence the domains in which self-esteem are invested in adulthood. To date, few studies have examined the relations between early life events and contingencies of self-esteem in relation to depressive symptoms. Accordingly, the influence of this combination of factors was assessed in the present investigation. Moreover, as will be described in the ensuing section, there is reason to suppose that variations of stressor-sensitive endocrine factors might also be influenced by early events and might be affected by dispositional factors such as self-esteem.

Stress and Physiological Changes

Neuroendocrine Response and Hypothalamic-Pituitary-Adrenal (HPA) axis

In response to stressors, several neurochemical changes are elicited in a variety of brain regions. Some of these (e.g., corticotropin release in different aspects of the amygdala and the bed nucleus of the stria terminalis) are involved in fear and/or anxiety (Lee & Davis, 1997; LeDoux, 2000), other are involved in appraisal or executive

functioning (e.g., within different aspects of the prefrontal cortex) (Doherty & Gratton, 1996), or the learning/memory of fear/anxiety (Nader, Schafe & Ledoux, 2000). For instance, after assigning emotional meaning to the sensory information, the amygdala guides emotional behaviour through projections to the hypothalamus, hippocampus, and basal forebrain (LeDoux, 1986; Pitman, 1989; Squire & Zola-Morgan, 1991).

Ultimately, stressors come to promote activation of the paraventricular nucleus (PVN) of the hypothalamus, giving rise to the release of corticotropin releasing hormone (CRH) from terminals located at the median eminence, causing the release of adrenocorticotropic hormone (ACTH) from the anterior pituitary, which in turn stimulates the release of cortisol from the adrenal cortex (Sapolsky et al., 2000). Once glucocorticoids (GC), such as cortisol, are released into circulation, they may activate glucocorticoid receptors (GR) on hippocampal neurons, or may directly influence hypothalamic activity at the level of the PVN, to restrict further glucocorticoid release (i.e., regulation of cortisol release is subject to a negative feedback loop).

Early Life Stress, Depression and Stress Reactivity into Adulthood

There are numerous biochemical similarities between depression and the effects of early life stressors. Specifically, both depression and stressors are associated with increased plasma GC levels. Indeed, stressors, including early stressors such as maternal deprivation, may result in chronic increase in plasma glucocorticoid levels (Dallman & Jones, 1973; Sapolsky, Alberts & Altmann, 1997), and may be associated with the sensitization of glucocorticoid and CRH responsiveness to subsequent stressors (Coplan, Andrews, Rosenblum et al. 1996; Francis, Caldji, Champagne et al. 1991; Fride, Dan, Feldon et al. 1986; Ladd et al. 1996; Levine, Weiner & Coe, 1993; Makino, Smith &

Gold, 1995; Plotsky & Meaney, 1993; Stanton, Guitierrez & Levine, 1988). Likewise, major depression is characterized by a marked hypersensitivity of the HPA axis (for a review see Plotsky, Ownes & Nemeroff, 1998). Increased levels of CRH, ACTH and enlargement of the adrenal gland often accompany this apparent up-regulation in the release of cortisol (Holsboer, Gerken, Stalla & Muller, 1987; Parker, Schatzberg & Lyons, 2003; Plotsky, Cunnigan & Widmaier, 1989; Rubin, Phillips, Sadow & McCracken, 1995). Furthermore, chronically stressed animals and depressed individuals have also been shown to develop an inability to terminate the glucocorticoid response to stressors (Sapolsky, Krey & McEwen, 1984a, b). Indeed, depressed individuals exhibit altered neuroendocrine activity when administered diagnostic tests used to assess specific aspects of HPA axis reactivity. For example, the stress hormone axis in major depression is relatively resistant to suppression by the synthetic glucocorticoid dexamethasone (Carrol, Martin & Davis, 1968).

Deficits of feedback inhibition of HPA activity in response to dexamethasone could be related to early life stressors (as well as depression) by provoking diminished GC receptor binding in the hippocampus (Makino et al. 1995; Sapolsky et al. 1984b; Smith, Kim, VanOers et al. 1997). For instance, it has been demonstrated that elevated levels of glucocorticoids produce dendritic atrophy and cell death in hippocampal neurons (Brown, Rush & McEwen, 1999; McEwen, 1998; Sapolsky, 2000). In this regard, chronically depressed individuals may display a marked decrease in hippocampal volume, which can persist following successful resolution of the depressive episode (Sapolsky, 2001). This effect appears to be related to duration of the depression rather than age per se (Bremner, Narayan, Anderson et al. 2000). It is not clear whether the

reduction in hippocampal volume observed in major depression precedes depressive symptoms or whether it follows the disorder. However, it has been proposed that glucocorticoid-induced atrophy erodes the ability of the hippocampus to regulate neuroendocrine activity, and hence, buffer against the negative effects of increased cortisol on depression (e.g., altered serotonergic function) (Lee, Ogle & Sapolsky, 2002).

As already described, there is considerable evidence that early life stressors are associated with increased risk for depressive and anxiety disorders in adulthood. Given that early adverse life events provoke long-lasting alterations in stress reactivity and HPA axis functioning, it is possible that these factors serve in an etiological capacity in the evolution of pathology. Among other things, GCs may precipitate symptoms of depression by impacting directly upon components of the central nervous system. Whether or not the stressor reactivity and the central neurotransmitter alterations are related to personality factors, and whether these were shaped by early life trauma, is uncertain.

Traumatic Experiences and Glucocorticoids Diurnal Pattern

Although it is well established that acute stressors generally provoke a transient increase of glucocorticoid release, less information is available as to how traumatic experiences impact on the diurnal pattern of cortisol release. Ordinarily, the release of glucocorticoids from the adrenal cortex, which occurs in a pulsatile fashion, follows a well-defined diurnal pattern (Windle, Woods, Shanks et al., 1998). This pattern is reflected in blood and in saliva (the latter reflecting free cortisol as opposed to bound cortisol). In humans, glucocorticoid concentrations are high at awakening, rise to reach a morning acrophase during the ensuing 30-60 minutes, and decreases rapidly thereafter to

an evening nadir at about midnight (Linkowski, Van Onderbergen, Kerkhofs et al. 1993; Schmidt-Reinwald, Pruessner, Hellhamer et al., 1999; Spath-Schwalbe, Scholler, Kern et al. 1992; Van Cauterm, Sturis, Byrne et al. 1994). The rise of free cortisol during the first 30 minutes after awakening, which reaches about 50-60%, was suggested to be a reliable biological marker for assessment of adrenocortical reactivity (Jobst, Kaspers & Kirschbaum, 1997). It was found to be largely independent of the time of awakening, sleep duration, sleep quality, physical activity or morning routines (Pruessner, Wolff, Hellhammer et al. 1997; Schulz, Kirschbaum, Pruessner et al. 1998; Schmidth-Reinwald et al. 1999; Wust, Federenko, Hellhammer et al. 2000), although it was recently reported that time of awakening may have effects under certain limited circumstances (e.g., shift work). However, this rhythm may be influenced by gender, use of oral contraceptives and stressor experiences may influence free cortisol levels (Schmidt-Reinwald et al., 1999).

Despite the dogma that stressors reliably promote elevated cortisol levels, studies of the relation between seemingly potent stressors (e.g. unemployment, job strain) and the diurnal pattern of cortisol have yielded conflicting results. For example, while Melamed, Ugarten, Shirom et al. (1999) indicated that burnout was associated with increased cortisol levels in the morning and afternoon, Pruessner, Hellhammer & Kirschbaum (1999) found that it was associated with lowered cortisol secretion upon awakening. Furthermore, although financial strain was associated with low levels of cortisol in the evening (Grossi, Perski, Lundberg & Soares, 2001), analysis of the effects of job strain on diurnal cortisol yielded opposite results (Steptoe et al., 2000).

Importantly, there is some indication that the neuroendocrine profile associated with stressor-related pathological states, including PTSD, may, in fact, comprise changes of rhythmicity of HPA functioning. It had been shown that cortisol levels are reduced in patients suffering PTSD. However, several studies suggested that PTSD was, in fact, associated with a change in the diurnal cortisol levels (Yehuda, 2002). For instance, morning cortisol levels were reduced among victims showing symptoms of PTSD (measured 5 days after trauma). Interestingly, evening cortisol levels were elevated in these same individuals. Moreover, with the attenuation of symptoms over a 9 months period, cortisol levels normalized (Aardal-Eriksson, Eriksson & Thorell, 2001). Paralleling these findings, Brunet and Meaney (personal communication) observed that among individuals that had encountered a trauma (motor vehicle accident) and then developed PTSD, the normal circadian cortisol changes were not evident. Instead, these individuals exhibited a relatively flat profile over the course of the day, essentially showing cortisol below control levels in the morning, but higher than those of control participants in the afternoon (recall that cortisol levels ordinarily decline over the day in non-distressed individuals). The flattened diurnal pattern of GC release (i.e., lower levels of cortisol shortly after awakening and higher levels in the evening) was not restricted to PTSD affected individuals. Indeed, a flattened cortisol profile was observed among individuals who reported childhood maltreatment (Hart, Gunnar, Cichetti, 1996), poor-relationship functioning (Adam & Gunnar, 2001), and increased workload (Caplan, Cobb & French, 1979), other studies have reported more varied diurnal patterns in response to stressful events. In effect, it may be constant strain (in addition or instead of severe

strain) that is responsible for the down regulation of cortisol release associated with traumatic events.

In contrast to the cortisol rhythms associated with PTSD, major depression is associated with elevated GC levels across the day, including a marked increase in neuroendocrine activity in the evening when the levels of cortisol are usually low (Deuschle et al. 1998; Plotsky et al., 1998; Young, Haskett Grunhaus et al. 1994). Given the data suggesting that variations of diurnal rhythm of GCs seem to be important to explain the neuroendocrine profile associated with psychopathology, its association with previous stressor experiences as well as with depression was assessed in the present investigation.

Objective of the study

It is possible that early life events, including parental bonding and traumatic experiences, may influence depressive symptoms during adulthood. The present study sought to evaluate the relative role of early adverse experience as well as stressor experiences in adulthood in the prediction of depressive symptoms and neuroendocrine stress reactivity in university students. Inasmuch as dysfunctional coping may be related to depressive symptoms, and adverse life events and external contingencies of self-worth are associated with self-reported depressive mood, the present investigation examined the processes that might mediate the bonding-depression and trauma-depression relations. Specifically, it was hypothesized that (a) both poor bonding and experiences of major early life trauma may be positively related to depressive symptoms, (b) the relationship between the early experiences and depressive symptoms would be mediated by contingencies of self worth, and (c) coping styles would mediate between contingencies

of self worth and depressive characteristics (see Figure 1). In addition, it was hypothesized that (d) cortisol diurnal patterns and stressor reactivity in response to an acute laboratory stressor challenge would be associated with mood alterations and these variations would be predicted by previous trauma, coping styles and the contingencies of self-worth endorsed (see Figure 2).

Figure 1

Early Adverse Events and Depression: Mediated Models

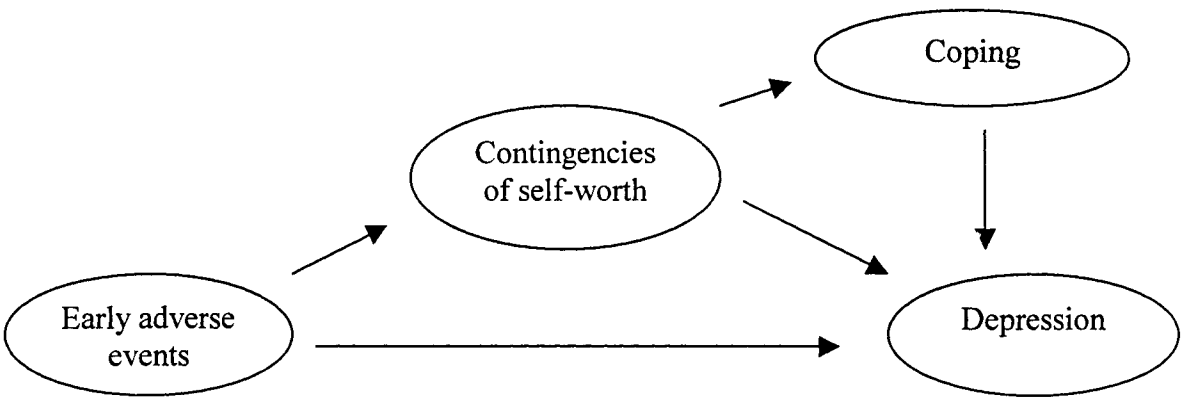
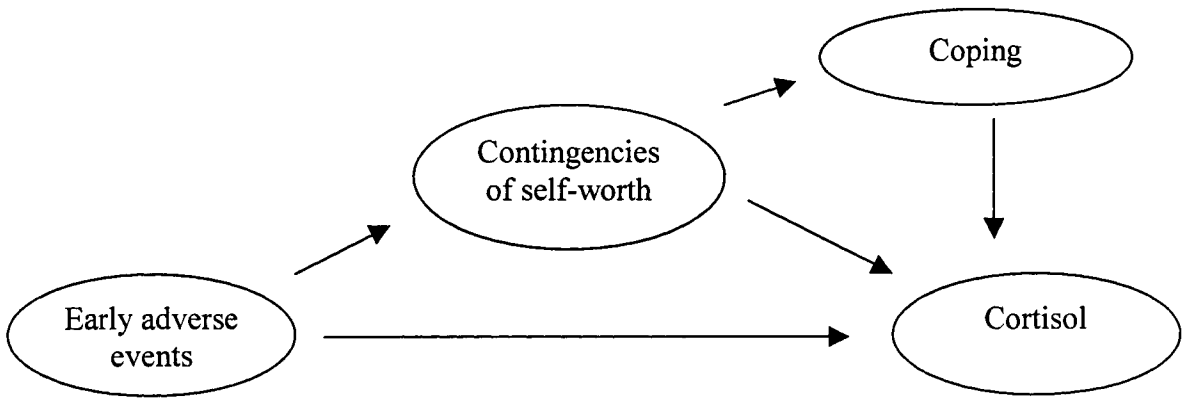


Figure 2

Early Adverse Events and Cortisol: Mediated Models



Method

Participants & Procedure

In the first phase of the study, participants were contacted through sign-up sheets, phoned or emailed, and invited to participate in the study. Participants recruited for this first phase comprised 173 females and 55 males (M age = 19.5, SD = 2.67) and based on the responses of participants reporting racial background, this sample was 77.4% (n=151) Caucasian, 4.1% Middle-Eastern (n=8), 5.5% Black (n=11), 1.5% Asian (n = 3), and 11.3% East Asian (n=22). After written informed consent was obtained, participants completed the Beck Depression Inventory (BDI, Beck, 1961), and the SCOPE, a multidimensional coping survey (Matheson & Anisman, 2003) and Contingencies of Self-Worth scale (CSW; Crocker, Luhtanen, Cooper, & Bouvrette, 2001). Once this had been completed, students were verbally debriefed and scheduled to complete Phase 2 on a day mutually agreed upon by the participant and investigator.

The second phase of the study was largely self-administered. Participants who agreed to return for this phase comprised a subset of the original sample, consisting of 155 females and 50 males (M age = 19.7, SD = 2.84) and based on the responses of participants reporting racial background, this subset was 78.5% (n=135) Caucasian, 3.5% Middle-Eastern (n=6), 5.8% Black (n=10), 1.2% Asian (n=2), and 11% East Asian (n=19). Participants were asked to provide cortisol samples upon awakening, and again at 0.5, 1, 4 and 6 hours later. They were then asked to come to the laboratory 1 hr after the last sample was taken, where they provided 2 further saliva samples 15 min apart. Thereafter, participants were asked to complete several relatively brief questionnaires, which included the Traumatic Life Events Questionnaire (TLEQ; Kubany, Hanes, Leisen

et al., 2000), after which 2 further saliva samples were taken (0 and 15 min after the task). The TLEQ served to as a reminder of previous adverse events they had experienced. In addition to the saliva samples, participants were asked to record a "diary of events" they had experienced the day before and on the morning that saliva samples were taken. This was done in the event that saliva samples were contributed during a particularly stressful period in their lives. At the end of the study participants were debriefed verbally and in writing, and provided the appropriate contact information. For each completed phase, all participants received an experimental credit in an undergraduate Introduction to Psychology course.

Measures

The Beck Depression Inventory (BDI) is a 21 item self-report rating inventory measuring characteristic attitudes and symptoms of depression (Beck et al., 1961). Internal consistency for the BDI ranges from .73 to .92 with a mean of .86. (Beck, Steer, & Garbin, 1988). The BDI demonstrates high internal consistency, with alpha coefficients of .86 and .81 for psychiatric and non-psychiatric populations, respectively (Beck et al., 1988). The BDI has a split-half reliability co-efficient of .93. The 21-item inventory uses a 4-point scale; responses of 0 indicate a lack in symptomatology while 3 indicate high depressive symptomatology. The highest possible total for the whole test is sixty-three. Responses were summed and a total score ranging from 5-9 is considered as reflecting normal ups and downs; 10-18 Mild to moderate depression; 19-29 moderate to severe depression and total score of 30–63; severe depression (Beck et al., 1988; Steer & Beck, 1997).

Coping styles were assessed using a 50-item scale that was found to be useful in assessing and conveying an overall profile of coping strategies individuals may adopt as a general style, and/or in response to specific situations (Survey of Coping Profile Endorsement (SCOPE), Matheson & Anisman, 2003). The SCOPE reliably assesses 13 strategies, including cognitive/behavioural responses (problem-solving, cognitive restructuring, active and cognitive distraction, and rumination) and socio-emotional responses (humor, social-support seeking, emotional expression, other- and self-blame, emotional containment, passive resignation, and religion). Respondents indicated their endorsement of each of the behaviors as a way of dealing with problems or stresses in recent months on a 5-point scale ranging from never (0) to always (4). Summing the scores of affirmative responses and dividing by the number of items for each subscale created a continuous index of strategy endorsement. Although specific coping strategies are often combined to form two to three superordinate categories of coping, analysis of the unique properties of all 13 strategies provides a richer understanding of the coping responses of individuals with stress-related symptoms, and facilitates assessment of how coping processes evolve over time and across situations (Matheson & Anisman, 2003). Thus, all 13 strategies were retained for analyses in the present study.

The psychometric properties of this scale have been previously reported (Matheson & Anisman, 2003). Given that these coping subscales were significantly correlated with those assessed in a commonly used scale developed by Carver Scheier and Weintraub (1989), the SCOPE demonstrates concurrent and construct validity. In particular, problem-focused coping scores using the SCOPE were highly related to Carver et al.'s active coping (r = .56) and planning (r = .59), cognitive restructuring was

associated with positive reinterpretation and growth (r = .51), social support seeking was positively related to the subscales of social support seeking for instrumental (r =.52) and emotional purposes (r = .62), and emotional expression was significantly related to venting of emotions (r = .46).

Participants were asked about their relationship with both their mother and father, using the care and overprotection subscales of the Parental Bonding Instrument (PBI) (Parker, Tupling and Brown, 1979). The PBI is a 25-item, retrospective measure of the individual's perceptions of their mother and father parenting attitudes and behaviors toward them during childhood, especially during the first 16 years of their childhood. The care scale measures the extent to which a parent was perceived to be supportive, affectionate and nurturing, whereas the overprotection scale measures the extent to which the parent was perceived to be protective and controlling. Examples of items from the care scales include: Was affectionate to me and could make me feel better when I was upset. Examples of the overprotective scale include: tried to control everything I did; tend to baby me. Items were rated on a four-point Likert rating scale ranging from *very like (3) to very unlike (0) how I remember my mother or father.* It has been suggested that the PBI is aligned with a 3-factor solution, although the factors identified across studies were not identical to one another (cf. Chambers, Power, Loucks, & Swanson, 2000; Kendler, Sham, & MacLean, 1997). In the present study, a three-factor solution was also derived (reflecting parental warmth, over-protective control, and autonomy), with the factors demonstrating modest intercorrelations (ranging from .19 to .57). However, the item-total reliabilities for the total scores for maternal (Cronbach's α = .91) and paternal (Cronbach's α = .89) relationships were very high, as were the correlations

between the subscales and total scores (ranging in magnitude from .62 to .88). In addition, when BDI scores were regressed onto the three maternal (R^2 = .156, p<.001) or paternal bonding subscales (R^2 = .159, p < .001), consideration of the three dimensions only slightly improved predictability of depressive symptoms in comparison to the relations with total scores on maternal (R^2 = .152, p < .001) and paternal bonding (R^2 = .128, p < .001). These findings indicated that it would be reasonable to proceed with the use of total scores in further analyses. High scores on these indices therefore reflected high levels of perceived parental warmth and autonomy, and low levels of over-protective parental control. Perceptions of maternal and paternal bonding were moderately correlated, $r(228) = .50, p < .001$.

Contingencies of Self-Worth Scale (CSW; Crocker et al., 2001) was determined using a 35-item self-report questionnaire developed to assess seven contingencies in which individuals may stake their self-worth: academic competence, appearance, competition, family support, God's love, other approvals and virtue. Each item was scored on a 5-point Likert scale ranging from 1 = strongly disagree to 5 = strongly agree. Internal consistencies (Cronbach's α) ranging from .82 to .96 was found for each of the seven subscales. Over a period of 8.5 months, test-retest reliability yielded correlation coefficients of .51 to .96 (Crocker et al. 2001; Crocker & Wolfe, 2001). In accordance with confirmatory analyses, each subscale was scored individually.

Trauma History was determined through the Traumatic Life Events Questionnaires (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 associated with traumatic events experienced by participants that caused feelings of fear, helplessness, and/or horror (based on Breslau et al. 1999). Thus, high scores reflected multiples experiences of each of these types of traumatic events. Those events reported as occurring prior to the age of 5 years were considered as adverse early life events.

Results

It was of interest to determine the effects of early life events, such as past trauma and parental bonding, on depressive symptoms and on the diurnal pattern of cortisol, and to identify some of the process by which such effects evolved. In this regard, it was posited that the functional domains in which individuals invest their self-esteem as well as how they cope with stressors mediate the relation between poor parental bonding or previous trauma and subsequent depressive symptoms.

Bonding and Depressive Symptoms

Although depressive symptoms varied appreciably (BDI range = 0 to 44; M = 8.73, SD = 7.27), the majority of participants reported minimal symptoms of depression (BDI scores < 9; n = 157, 68.9%). A sizable, but smaller proportion, reported moderate (scores between 9 and 18; n = 48, 21.1%) or high symptoms (> 18; n = 23, 10.1%).

Depressive symptoms were regressed simultaneously onto maternal and paternal bonding. Parental bonding explained 19.2% of the variance of depressive symptoms, $F(2,215) = 25.53$, $p < .001$, and maternal ($r = -.40$; $\beta = -.29$, $p < .001$) and paternal bonding ($r = -.36$; $\beta = -.22$, $p <. 05$) had additive effects in predicting lower symptomatology (see Table 1). A subsequent analysis indicated that sex was not significantly related to, nor did it interact with, parental bonding in the prediction of depressive symptoms.

Contingencies of Self-Worth and Depressive Symptoms

Table 2 presents the intercorrelations between the contingencies of self-worth subscales. The highest correlations obtained were between academic competence and competition (r = .51) and appearance and approval from others (r = .52). Family based self-worth was not significantly related to competition and appearance based self-worth, whereas perception of self worth through God's love was not related to competition or appearance. Thus, although most of the contingencies were moderately correlated, they did not seem to measure the same constructs.

To assess the relation between contingencies of self-worth and depressive symptoms, regression and correlational analyses were conducted. Overall, contingencies of self worth was a significant predictor of depressive symptoms, $R^2 = 0.126$, $F(7,220) = 4.54$, $p < .001$, but none of the contingencies of self-worth were themselves unique predictors of depression (see Table 3). However, examination of the zero order correlations indicated that higher depressive symptoms were related to increased self-worth based on competition, appearance, academic and approval from others.

Relations between Bonding and Contingencies of Self-Worth

It was proposed that secure parental bonding would be associated with more internally based contingencies of self-worth and effective styles of coping, and it may be through the provision of these resources or skills that the individual becomes more resilient to depressive symptoms. To assess this possibility, the next analytical step was to assess the interrelations between bonding and each of these potential mediating variables.

To assess whether parental bonding provided a basis for contingencies of self-worth, each of maternal and paternal bonding were regressed onto the 7 contingencies of self-worth, entered on the same step. As seen in Table 4, contingencies of self-worth were associated with both maternal, $R^2 = .143$, $F(7,218) = 5.19$, $p < .001$, and paternal bonding, $R^2 = .105$, $F(7,218) = 3.52$, $p < .001$. Regression coefficients indicated that poor maternal bonding was associated with decreased self-worth through family and increased self-worth through perception of God's love. In addition, the zero-order correlations confirmed that poor maternal bonding was associated with decreased self-worth based on family, but also indicated that increased self-worth based on competition was related to maternal bonding. Interestingly, although a similar pattern of correlations was noted in relation to paternal bonding, regression coefficients indicated that poor paternal bonding was not only significantly related to decreased self-worth based on family and increased self-worth based on competition, but also on being virtuous.

Parental Bonding, Contingencies of Self-worth and Depression: A Mediated Model

Given the strength of associations observed, contingencies of self-worth may serve as a mediating variable in the relation between bonding and depressive symptoms. This

possibility was assessed following the approach delineated by Baron and Kenny (1986). Only the contingencies of self-worth that demonstrated significant zero-order correlations with maternal or paternal bonding, and with depression scores, were considered in these analyses (see Table 3 and 4). Thus, depressive symptoms were regressed simultaneously onto contingencies of self-worth based on competition and maternal or paternal bonding (the latter two variables in separate analyses).

With respect to maternal bonding, the relation to depressive symptoms was not mediated by self-worth contingent on competition. Specifically, when controlling for competition based on self-worth, maternal bonding continued to account for 13.1% of unique variance in depressive symptoms. Indeed, the R^2 dropped from .149 to .131, and the mediated path was not significant (Sobel t = 1.98, *ns*). In contrast to maternal bonding, when controlling for competition-based self-worth, paternal bonding continued to account for 9.9% of unique variance in depressive symptoms. Specifically, the R^2 dropped from .128 to .099, suggesting a partially mediated model, and the Sobel's test indicated that the mediated path for paternal bonding was significant (Sobel t =2.12, $p < .05$) (see Table 5). Thus, it appears that although both paternal and maternal bondings were related to depressive symptomatology, the pathways leading to such an effect were different from one another.

Table 1

Regression Analysis (Pearson Correlations and Standardized Regression Coefficients) Assessing the Relations Between Bonding (Predictor Variables) and Depressive Symptoms (Outcome Variable)

	Depressive symptoms		
	R	β	R^2_{total}
Bonding			.438***
Maternal	.396***	.287***	
Paternal	.358***	.216**	

Table 2

Pearson's Correlations Coefficients Assessing the Relations Between the Contingencies of Self-Worth subscales.

	Family	Competition	Appearance	God	Academics	Virtue
Family						
Competition	.089					
Appearance	.070	.373***				
God	.187**	- .001	- .054			
Academic	.208**	.505***	.419***	.146*		
Virtue	.208**	.082	.079	.324***	.275***	
Other	.194**	.381***	.516***	- .030	.399***	.126

$^{+}p < .10$; *$p < .05$; **$p < .01$; ***$p < .001$

Table 3

Regression Analysis (Pearson Correlations and Standardized Regression Coefficients) Assessing the Relations Between Contingencies of Self-Worth (Predictor Variables) and Depressive Symptoms (Outcome Variable).

	Depressive symptoms		
	R	β	R^2_{total}
Contingencies of Self-Worth			
			0.126***
Family	- .069	- .126^{+}	
Competition	.256***	.134^{+}	
Appearance	.254***	.112	
God	- .075	- .074	
Academics	.230***	.098	
Virtue	.066	.057	
Other	.231***	.08	

$^{+}$ p <.10; *p<.05; **p<.01; ***p<.001

Table 4

Regression Analysis (Pearson Correlations and Standardized Regression Coefficients) Assessing the Relations Between Contingencies of Self-Worth (Predictor Variables) and Bonding (Outcome Variable)

	Maternal Bonding			Paternal Bonding		
	R	β	R^2_{total}	r	β	R^2_{total}
Contingencies of Self-Worth			0.143***			.105**
Family	-.275***	-.341***		-.176**	-.233**	
Competition	.132*	.096		.192**	.171*	
Appearance	.084	.047		.097	-.006	
God	.115$^+$	.145*		-.017	-.024	
Academics	.125$^+$	.102		.107$^+$	-.007	
Virtue	.066	.050		.130*	.165*	
Other	.020	-.022		.123	.083	

$^+p<.10$; * $p<.05$; ** $p<.01$; *** $p<.001$

Table 5

Regression Analyses (Pearson Correlations and Standardized Regression Coefficients) and Sobel Tests Assessing the Mediated Relations Between Parental Bonding (Predictors) and Depressive Symptoms (Outcome)

	Maternal Bonding			Paternal Bonding		
Mediating Variables	r	β[a]	Sobel t	r	β[a]	Sobel t
Competition-based	-.243***	-.195**	1.68	-.259***	.197**	2.12*
Self-Worth						
Bonding[b]						
Maternal	.390***	.365***	-			-
Paternal	-	-	-	.358***	.320***	-

$^{+}p < .10$; $^{*}p < .05$; $^{**}p < .01$; $^{***}p < .001$

a. The standardized regression coefficients represent final step weights.

b. Maternal and paternal bonding scores were entered in separate regression analyses.

Coping Strategies and Depressive Symptoms (Factors)

In order to reduce the coping strategies, which were possibly correlated, to a smaller number of uncorrelated components, a principal component analysis with a varimax rotation with Kaiser Normalization was conducted on the 13 coping strategies. Once the scree plot of the eigenvalues was obtained, four principal components were obtained and the importance and contribution of each component to the explained variance was computed. Coping strategies, in order to be considered part of a particular factor, had to have a factor loading of at least .4 on that component. Furthermore, if a specific coping strategy was found to be involved in several factors, it was considered to be part of the factor in which it had the highest score.

The first of the four factors constituted emotion-focused strategies (rumination, emotional expression, self-blame, other-blame, and wishful thinking) explaining 24.56% of the variance; the second factor was problem-focused strategies (problem-solving, social support) explaining 16.78%; the third factor mainly comprised avoidant strategies (active distraction, emotional containment, avoidance and humor) accounting for 10.26% of the variance; finally, the fourth factor, cognitive restructuring (composed of cognitive restructuring, passiveness and religion), explained 7.54% of the variance (see Table 6).

To assess the relation between the four factors making up the coping strategies and depressive symptoms, regression and correlational analyses were conducted using individual's scores on each factor. The regression revealed that coping factors were significantly related to depressive symptoms $R^2 = .424$, $F\ (4,223) = 40.96$, $p < .001$. Examination of the zero order correlations and regression coefficients indicated that higher depressive symptoms were related to a higher propensity to cope through emotion-

focused strategies and a lower propensity to cope through problem-focused strategies (see Table 7).

To assess whether parental bonding provided a basis for adaptive coping, each of maternal and paternal bonding was independently regressed onto 4 factors, entered on the same step. As seen in Table 8, these factors were associated with both maternal, R^2 = .151, $F(4,221) = 9.79$, $p<.001$, and paternal bonding, $R^2 = .108$, $F(4,214) = 6.48$, $p < .001$. As indicated by the zero-order correlations, poor maternal bonding was associated with a greater propensity to endorse emotion-focused coping strategies and cognitive restructuring and a lower propensity to endorse problem-focused strategies and avoidance. A similar pattern of correlations was noted in relation to poor paternal bonding with higher emotion-focused and lower problem-focused strategies being related to depressive symptoms. However, a lower propensity to endorse avoidance was not significantly related to paternal bonding.

Parental Bonding, Coping and Depressive Symptoms: A Mediated Model

Given the strength of associations observed, coping factors may serve as a mediating variable in the relation between bonding and depressive symptoms. To assess this possibility, those factors that demonstrated significant zero-order correlations with maternal or paternal bonding, and with depression scores, were considered in these analyses (see Table 8 and 9). Thus, depressive symptoms were regressed simultaneously onto these common factors and maternal or paternal bonding (the latter two variables in separate analyses).

As seen in Table 9, the relation between maternal bonding and depressive symptoms appeared to be partially mediated by the higher propensity to use emotion-focused and

lower propensity to use problem-focused coping. When controlling for these coping strategies, maternal bonding the R^2 dropped from .152 to .039 suggesting a partially mediated model. Sobel's tests indicated that the mediated paths through emotion-focused strategies (Sobel t = 2.12, p < .05) as well as through problem-focused strategies were significant (Sobel t = 3.80, p < .001).

The relation of paternal bonding with depressive symptoms also appeared to be partially mediated by emotion- and problem-focused coping. Specifically, when controlling for these factors, R^2 dropped from .124 to .026 and paternal bonding continued to account for unique variance in depressive symptoms. Furthermore, the mediated paths through emotion-focused coping and problem-focused coping were significant (Sobel $t = 2.94, p < .005$ and Sobel $t = 3.13, p < .005$ respectively).

Contingencies of self-worth and coping

It was posited that certain contingencies of self-worth may influence the adoption of coping strategies. As competition-based self-worth was related to bonding and depression, an analysis was undertaken to determine whether it provided a basis for maladaptive coping strategies. Thus, this contingency of self-worth was regressed independently onto the 4 coping factors simultaneously. This analysis revealed that coping factors were significantly related to competition based self-worth, $R^2 = .127$, $F(4,223) = 8.08$, $p < .001$. Specifically, examination of the zero-order correlations, as seen in Table 10, indicated that competition-based self-worth was significantly related to higher propensity to use emotion-focused strategies.

Table 6

Principal Component Analyze with Varimax Rotation of the Coping Strategies.

Factor	Components	Factor Loading	% Variance explained
Emotion-focused	Rumination	.85	24.6
	Emotional Expression	.77	
	Self-blame	.62	
	Other-blame	.45	
	Wishful thinking	.79	
Problem-focused	Problem-solving	.74	16.8
	Social support	.83	
Avoidant	Active distraction	.79	10.3
	Emotional containment	.50	
	Avoidance	.53	
	Humor	.43	
Restructuring	Cognitive restructuring	.74	7.5
	Passiveness	.67	
	Religion	.45	

Table 7

Regression Analysis (Pearson Correlations and Standardized Regression Coefficients) Assessing the Relations Between Coping Strategies (Predictor Variables) and Depressive Symptoms (Outcome Variable)

	Depressive symptoms		
	r	β	R^2_{total}
Coping Strategies			.424***
Factor Emotion-focused	.48***	.48***	
Factor Problem-focused	- .44***	- .44***	
Factor Avoidance	- .03	- .03	
Factor Cognitive Restructuring	.07	.07	

$^{+}p < .10$; $*p < .05$; $**p < .01$; $***p < .001$

Table 8

Regression Analysis (Pearson Correlations and Standardized Regression Coefficients) Assessing the Relations Between Coping Strategies (Predictor Variables) and Bonding (Outcome Variable)

	Maternal Bonding			Paternal Bonding		
	r	β	R^2_{total}	r	β	R^2_{total}
Coping Strategies			.151***			.108***
Emotion-Focused	.15*	.15*		.22***	.22***	
Problem solving	- .31***	- .31***		- .24***	- .24***	
Avoidance	- .11	- .11		- .04	- .04	
Cognitive restructuring	.15	.15		.04	.04	

$^{+}$ p < .10; *p<.05; **p<.01; ***p<.001

Table 9

Regression Analyses (Pearson Correlations and Standardized Regression Coefficients) and Sobel Tests Assessing the Mediated Relations Between Parental Bonding (Predictors) and Depressive Symptoms (Outcome)

	Maternal Bonding			Paternal Bonding		
Mediating Variables	r	β[a]	Sobel t	r	β[a]	Sobel t
Factor 1: Emotion-Focused	.46***	.43***	2.17*	.46***	.42***	2.94**
Factor 2: Problem-Focused	- .45***	- .38***	3.30**	- .44***	- .40***	3.13**
Bonding[b]						
Maternal	.39	.21***		-	-	
Paternal	-	-		.36***	.17**	

$^{+}$ $p < .10$; *$p<.05$; **$p<.01$; ***$p<.001$

a. The standardized regression coefficients represent final step weights.
b. Maternal and paternal bonding scores were entered in separate regression analyse.

Table 10

Regression Analysis (Pearson Correlations and Standardized Regression Coefficients) Assessing the Relations Between Coping Strategies (Predictor Variables) and Contingency of Self-Worth based on Competition (Outcome Variable)

	Competition		
	R	β	R^2_{total}
Coping Strategies			.127***
Problem solving	.34***	.34***	
Emotion Focused	- .10	- .10	
Avoidance	.02	.02	
Cognitive restructuring	- .04	- .04	

$^{+}$ p < .10; *p<.05; **p<.01; ***p<.001

Given the strength of associations observed, self worth contingent on competition may serve as a mediating variable in the relation between bonding and coping. Only the coping factors that demonstrated significant zero-order correlations with maternal or paternal bonding, and with depression scores, were considered in the mediating analyses (see Table 1). Each of these coping factors was regressed simultaneously onto competition-based self-worth and maternal or paternal bonding (the latter two variables in separate analyses).

When competition self-worth was controlled for, maternal bonding was no longer a significant predictor of emotion-focused coping strategies, $R^2_{change} = .011$, $F(1,223) = 2.68$, *ns*. Not surprisingly, given that maternal bonding had small predictive power regarding competition-based self-worth, $R^2 = .017$, $F(1,224) = 3.96$, $p < .05$, the mediated path between maternal bonding and emotion-focused strategies through competition based self-worth was not significant, (Sobel $t = 1.84$, *ns*). In contrast, when competition self-worth was controlled for, paternal bonding was still a significant predictor of emotion-focused coping strategies and the mediated path through competition self-worth was significant, (Sobel $t = 2.69$, $p < .01$).

Thus, the pathways by which poor maternal and paternal bonding lead to depressive symptomatology appear to be different. While poor paternal bonding seems to be related to a competition-based self-worth and this may promote the use of emotion-focused strategies, the greater propensity to use emotion-focused strategies associated with poor maternal bonding is unrelated to competition-based self-worth.

Mediating Role of Contingencies of Self-Worth and Coping in the Relations Between Bonding and Depressive Symptoms

A primary goal of the study was to assess the mediating role of contingencies of self-worth and coping in the relation between parental bonding and depressive symptoms. To assess this mediated model, two multiple regressions were conducted in which depressive symptoms were regressed simultaneously onto contingencies of self-worth, the coping styles that demonstrated significant zero-order correlations with maternal and paternal bonding and BDI scores, respectively (see Tables 3, 4, 7, 8), and onto each of maternal and paternal bonding (in separate analyses).

Table 11 indicates that when competition-based self-worth and emotion-focused coping and problem-focused coping factors were controlled for, maternal bonding was still a significant predictor of depressive symptoms, $R^2 = .451$, $F(1,221) = 15.29$, $p < .001$. The mediated path through competition-based self-worth was not significant, whereas the mediated paths through emotion and problem-focused coping were significant (Sobel ts, all $p < .05$). Thus, these coping variables appear to be partially mediating the relation between maternal bonding and depressive symptoms.

With respect to paternal bonding, Table 11 indicates that when competition-based self-worth and emotion-focused coping and problem-focused coping factors were controlled for, paternal bonding was still a significant predictor of depressive symptoms, $R^2 = .432$, $F(1,214) = 9.21$, $p < .005$. The mediated path through competition-based self-worth was not significant (Sobel $t = .72$, *ns*), whereas the mediated paths through emotion and problem- focused coping strategies were significant (Sobel ts, all $p < .01$).

Table 11

Regression Analyses (Pearson Correlations and Standardized Regression Coefficients) and Sobel Tests Assessing the Mediated Relations Between Parental Bonding (Predictors) and Depressive Symptoms (Outcome)

	Maternal Bonding			Paternal Bonding		
Mediating Variables	r	β[a]	Sobel t	r	β[a]	Sobel t
Competition-based	.243***	.041	.72	.259***	.047	.82
Self-Worth						
Coping Styles						
Emotion-focused	.458***	.412***	2.12*	.458***	.409***	2.98**
Problem-focused	- .451***	- .380***	4.04***	- .437***	- .396***	3.26**
Bonding[b]						
Maternal	.390***	.208***		-	.166**	
Paternal	-	-		.358***	-	

$^{+}$ p<.10; $^{*}p$<.05; $^{**}p$ <.01; $^{***}p$ <.001

a. The standardized regression coefficients represent final step weights.

b. Maternal and paternal bonding scores were entered in separate regression analyses.

In summary, emotion and problem-focused strategies appeared to be a partial mediating variable in the relation between parental bonding and depressive symptoms. Furthermore, in contrast to maternal bonding, in the case of paternal bonding and depressive symptoms, self-worth contingent on competition appeared to partially mediate the use of emotion-focused strategy associated with depressive symptoms.

Early Life Trauma

As described earlier, there is reason to believe that early life trauma may affect self-esteem and might thus affect depressive symptoms in adulthood. Accordingly, analyses were undertaken to assess relations related to trauma experienced prior to 5 years of age. Adverse early life events scores were calculated by summing the frequencies associated with traumatic events experienced prior to 5 years of age (see Table 12). The majority of the participants reported no early adverse traumatic experience (scored no traumatic events, n = 163, 71.5%), 47 participants reported 1 early adverse life events (20.6%) and while 18 reported 2 or more early traumatic experiences (7.9%).

Table 13 illustrates the types of adverse early life events based on four categories: shocking experiences (e.g. natural disasters, war), death of someone close to them, assaultive experiences and trauma experienced by others. The majority of early adverse events consisted of the experience of a shocking or unexpected event (n = 51) where the most commonly reported experience was that of experiencing a natural disaster (n = 17) and being involved in any other kind of accident where you or someone else was badly hurt (n = 14). Witnessing violence done to others, being assaulted, or the unexpected death of someone close was a less common experience. The most commonly reported

assaultive experiences were being hit or beaten up and badly hurt by a stranger or someone you didn't know very well (n = 7), whereas all the traumatic events involving others consisted of seeing a loved one (who is living) experiencing a life threatening or permanently disabling accident, assault, or illness.

Table 13 also illustrates the overall (i.e., irrespective of age) trauma history of the participants calculated by summing the frequencies associated with each of the four types of traumatic events that were reported to cause fear, helplessness and/or horror. Experience of a shocking or unexpected event (natural disaster, accident, living in a war zone, witnessing a violent event, experienced a life-threatening illness, miscarriage or abortion) was the most common traumatic experience, with the most frequent being the experience of seeing a stranger or someone they didn't know very well attack or beat up someone and seriously injure or kill them (n = 50). Experiencing the unexpected death of someone close to them (n = 89), assaultive experiences (n = 101) (childhood physical or sexual abuse, spousal assault, rape, stalked, or life threatened) and exposure to traumatic events occurring to others were relatively common experiences in this sample (n = 96). The most frequent reported assaultive experiences were being stalked which caused them to feel intimidated or concerned for their safety (n = 40) and being threatened with death or serious physical harm (n = 32). In addition, while the majority of traumatic experiences involving others concerned a loved one (who is living) experiencing a life threatening or disabling accident, assault, or illness (n = 73), 41 participants reported seeing or hearing family violence while growing up.

Table 12

Distribution of Adverse Early life Events by Gender

	No Early Life Event	Early Life Events		
		1	2	More than 2
Male	41	10	4	2
Female	122	37	8	4
Total	163	47	12	6

Table 13

Distribution of the Type of Early Traumatic Events Experienced by Gender

	Assault	Death	Shock	Other
Early life events				
Male	7	0	13	1
Female	6	9	38	9
Total	13	9	51	10
Over life				
Male	28	19	19	14
Female	73	70	102	82
Total	101	89	121	96

Gender differences

Overall, women experienced more traumatic experiences than did men, $F(1,226) = 5.30$, $p < .05$, and also tended to report more death and traumatic experiences involving others, although the latter effects were not statistically significant $F(1,226) = 3.60$, *ns*; $F(1,226) = 3.49$, *ns,* respectively. In contrast, gender differences were not reported with respect to traumatic experiences experienced early in life or with respect to parental bonding, (see Table 14).

Adverse early life events, overall traumatic history and well-being

To ascertain whether early adverse events were associated with depressive symptoms, correlation and regression analyses of depressive symptoms with overall number of early life events and specific types of early traumatic experiences was conducted. Examination of the zero-order correlations indicated that early assaultive experiences were the unique traumatic experience related to depressive symptoms. Neither the number of early life experiences, overall trauma history or specific types of adverse experience, whether prior to 5 years of age or overall, was related to depressive symptomatology in adulthood (see Table 15). Given that the sample of participants reporting early assaultive experiences was very small, there was insufficient power to conduct further analyses regarding potential mediating factors.

Table 14

Gender Differences in Traumatic Experiences and Depressive Symptoms

(Mean ± SD)

	Gender	
	Males	Females
Early life events	.75 ± 1.13	.84 ± 1.33
Shock	1.62 ± 1.39	1.66 ± 1.43
Assault	2.00 ± 1.83	1.06 ± .14
Death	-	1.56 ± 1.67
Others	1.00[c]	1.44 ± .53
Bonding		
Maternal	1.81± .42	1.83 ± .45
Paternal	1.82 ± .43	1.89 ± .60
Overall trauma	1.95 ± 1.78	2.84 ± 2.69
Shock	1.85 ± 1.36	1.58 ± 1.21
Assault	2.04 ± 1.45	1.76 ± 1.36
Death	1.37 ± .60	2.03 ± 1.30
Others	2.32 ± 1.88	2.18 ± 1.54
Depressive symptoms	8 ± 6	9 ± 8

a. Cannot be computed because individual are all females.
b. Correlations are not significant because n too small, n=10 and n=13.
c. Standard deviation cannot be computed because n=1.

Table 15

Pearson Correlations Assessing the Relations Between Traumatic Experiences and Depressive Symptoms

	Depressive symptoms
	r
Early life events	- .054
Shock	- .077
Assault	.630*
Death	- .369
Others	- .478
Overall trauma	.114
Shock	.022
Assault	.006
Death	- .093
Others	.135

$^{+}p < .10$; $*p < .05$; $**p < .01$; $***p < .001$

Diurnal Cortisol Profile

Ordinarily, salivary cortisol levels increase rapidly over the first hour following awakening and then decline precipitously over the ensuing 3-4 hours (Pruessner et al., 1997; Schmidt-Reinwald et al., 1999). Figure 3 shows that male (n = 44) and female (n = 144) participants exhibited the typical profile of salivary cortisol levels following awakening. Given that gender differences may occur with respect to stress perception and neuroendocrine responses to stressors, a set of analyses was first conducted to ascertain the possible moderating influence of gender on neuroendocrine alterations. As depicted in Figure 3 males tended to exhibit lower cortisol concentrations compared to females, but the repeated measures analyses indicated no gender differences in diurnal pattern of cortisol, $F(1,189) = .61$, *ns* (see Figure 3). Similarly, although Figure 4 suggests that participants reporting low maternal bonding exhibited higher cortisol concentrations at awakening and lower concentrations at the other time points following awakening relative to participants reporting high maternal bonding, the repeated measures ANOVA indicated that difference based on bonding were not statistically significant, $F(1,96) = 1.10$, *ns*. Figure 5 shows that those participants that had encountered early life distress tended to exhibit higher cortisol concentrations at all the time points following awakening relative to participants that had not encountered such experiences. Once again, however, the repeated measures analyses indicated that the differences between groups were not significant, $F(1,189) = .91$, *ns*.

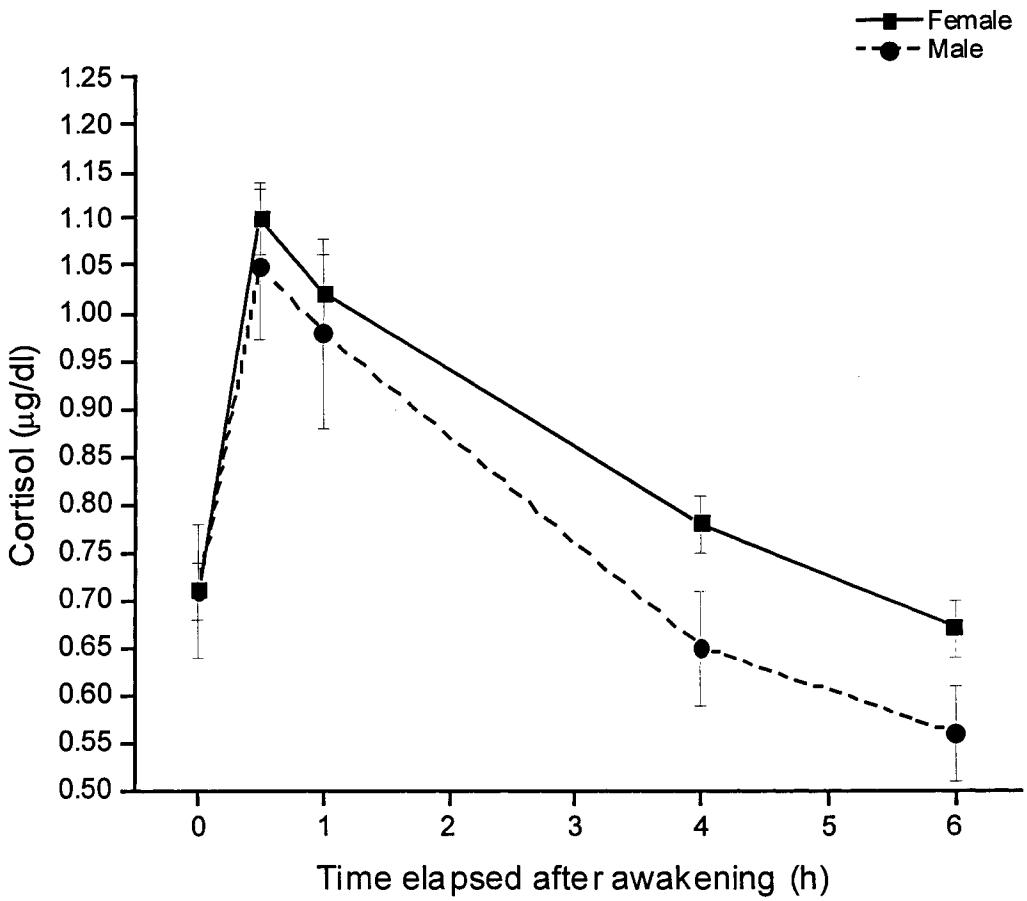


Figure 3. This figure shows the diurnal cortisol patterns for women (solid line) and men (dotted line). Although this trend is not significant, men tended to exhibit lower cortisol levels compared to females. Note that cortisol levels rise markedly 30 minutes after awakening and decline thereafter. Although the difference between males and females was appreciable during the later times of the day, the overall difference between the sexes was not significant.

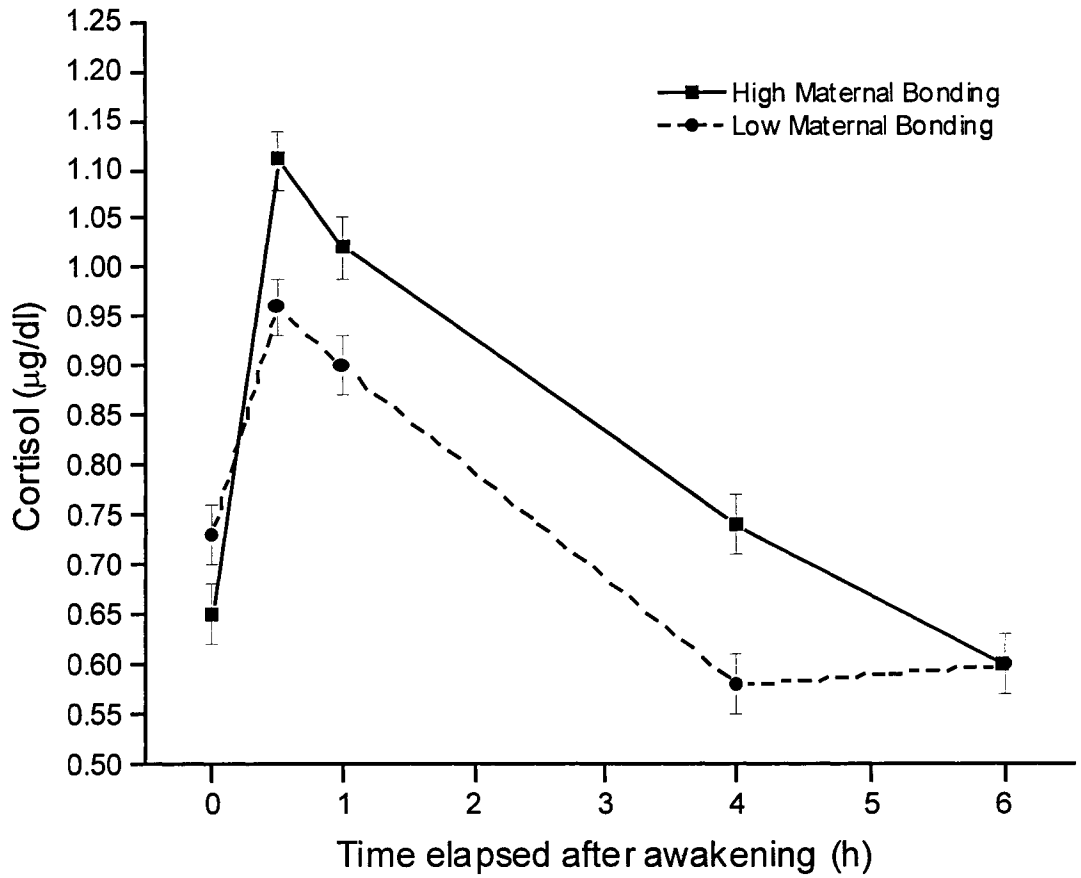


Figure 4. This figure shows the diurnal cortisol patterns for participants reporting high (solid line) and low maternal bonding (dotted line). Except at awakening, participants reporting poor perceived maternal bonding tended to exhibit lower cortisol levels at all times over the course of the day compared to participants reporting high maternal bonding.

Figure 5

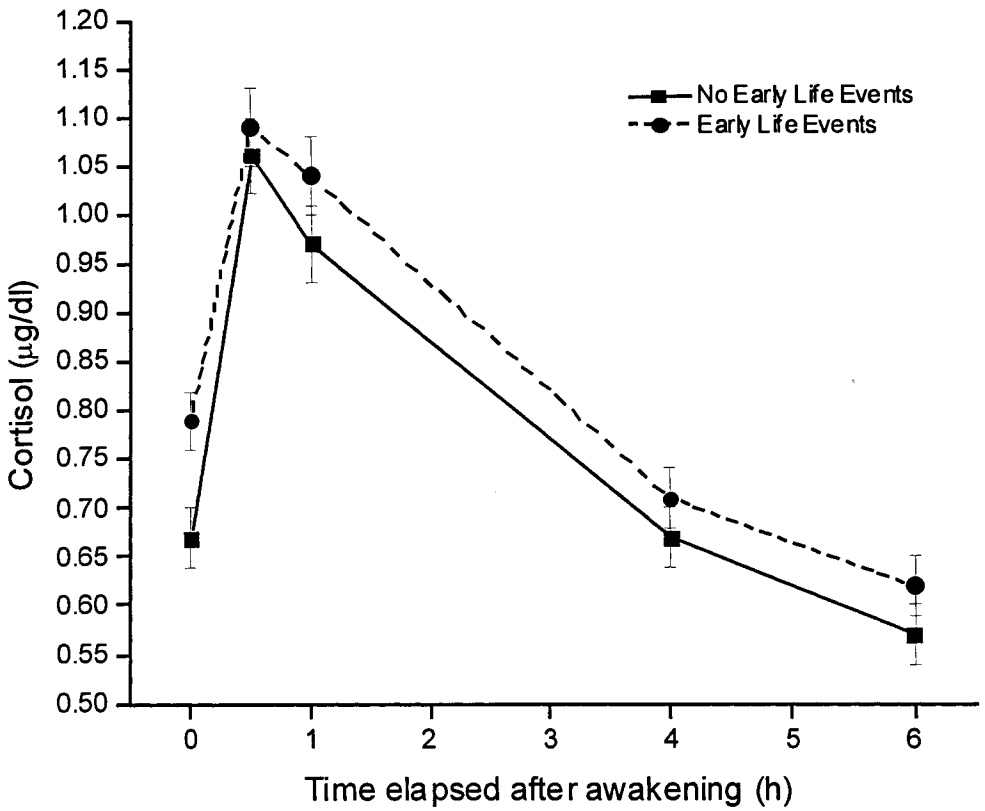


Figure 5. This figure shows the diurnal cortisol patterns for participants reporting no early life events (solid line) or more than one early life event (dotted line). Over the course of the day, participants that had encountered early life distress tended to exhibit higher cortisol levels compared to participants who reported no such event.

In order to assess whether previous traumatic experiences influenced diurnal cortisol patterns, a mixed measures analysis was used in which gender (male coded as 0 and female coded as 1), and their previous trauma history (in which participants that reported more than 5 traumatic events were coded as 1, those reporting between 3 and 5 traumatic events were coded as 2, those reporting 1 and 2 traumatic events were coded as 3 and those reporting no traumatic events coded as 0). Of course, it would have been preferable to divide the number's of trauma experienced into 15%, 35, 35, and 15%, thereby approximating the normal distribution. This segmentation was used because when the data were examined, it was discovered that this was not possible based on the number of participants reporting a given number of traumas. For example, amending the groupings of participants reporting 1 to 2 traumas to reflect those participants reporting 1 to 3 traumas would have resulted in a single category representing 51% of participants. The mixed measures analysis revealed no differences or gender interaction with trauma categories. However, although this difference did not reach significance, as depicted in Figure 6, participants reporting 5 or more traumatic experiences tended to exhibit lower cortisol levels compared to the participants reporting fewer traumatic experiences. Additional orthogonal comparisons comparing cortisol level at each of the sampling intervals for those participants reporting 6 or more traumatic experiences with the combined mean of the other groups (0, 1 to 2, 3 to 5) indicated significantly lower cortisol levels at the first three sampling intervals when compared to the combined levels for the other groups ($ps < .05$).

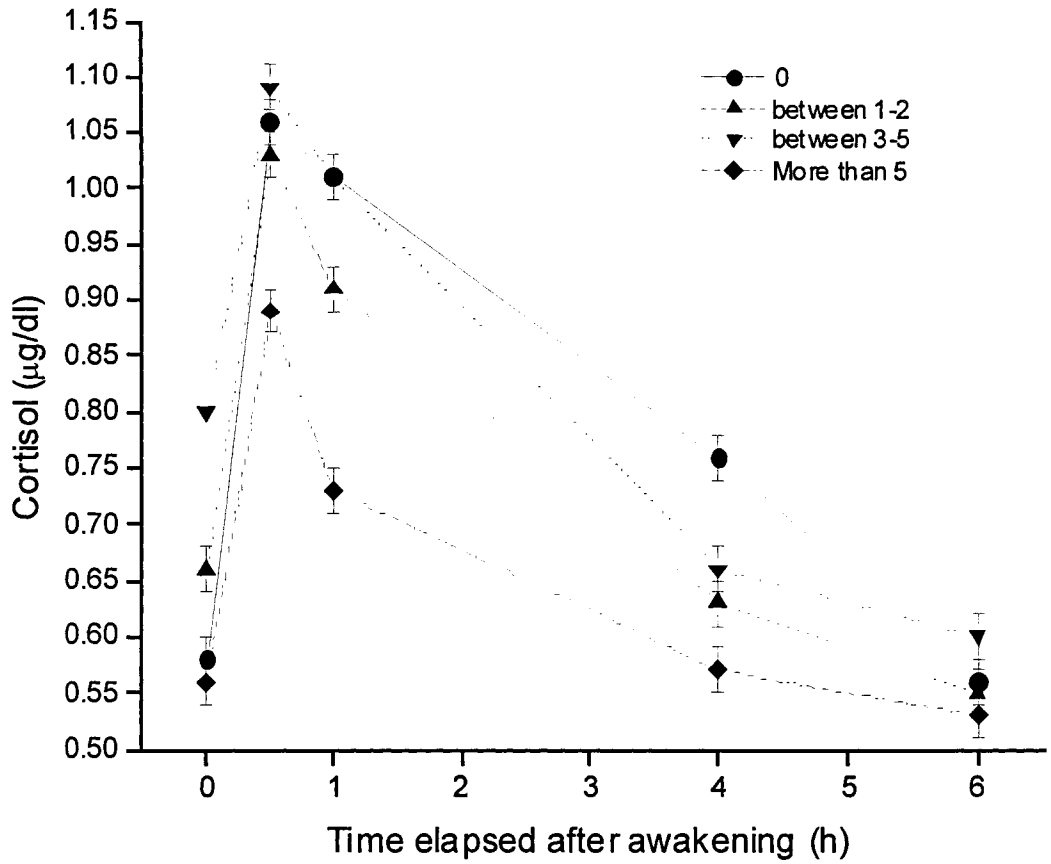


Figure 6. This figure shows the diurnal cortisol patterns for participants reporting no previous history of traumatic experiences (solid line, circle); between one and 2 traumatic experiences (dotted line, triangle); between 3-5 traumatic experiences (dotted line, inverted triangle) and more than 5 traumatic experiences (dotted line, diamond). Participants reporting 6 or more traumatic experiences with the combined mean of the other groups (0, 1 to 2, 3 to 5) exhibited significantly lower cortisol levels at the first three sampling intervals when compared to the combined levels for the other groups.

Cortisol- Stress Reactivity

In the present study, participants displayed increased cortisol levels over the two baseline saliva samples collected in a laboratory context over a 20 min period, possibly reflecting anticipation of the experimental procedures to follow, especially as they were aware that they would be responding to a questionnaire (TLEQ) concerning past trauma. Figure 7 shows that both genders showed an increase of cortisol following their entry to the lab and this effect appeared to be particularly prominent in males relative to females. Indeed, a mixed measures analysis of variance in which sampling time was considered to be a within group variable and gender a between-group variable, indicated significant gender differences in the saliva samples collected in the laboratory, $F(1,914) = 7.15$, $p < .01$, $\eta^2 = .04$. Further univariates analyses with gender as a between-subjects variables indicated that this difference might be due to differences in the two saliva samples collected prior to the TLEQ being completed. Cortisol levels from sample collected upon arrival to the laboratory, and upon subsequent sampling 15 min later, were higher in males than in females, $F(1,190) = 4.06$, $p<.05$, $\eta^2 = .02$ and $F(1,192) = 6.43$, $p < .05$, $\eta^2 = .03$ respectively. In contrast, there were no gender differences in the sample collected 5 or 15 min after the completion of the TLEQ, $F(1,193) = 1.16$ and 0.03, *ns*.

A separate set of analyses was conducted in which cortisol change was calculated as a proportion of the cortisol score just prior to the completion of the TLEQ. In order to assess the effect of the TLEQ on cortisol, a mixed measures analysis of variance was conducted in which sampling ratios were considered to be a within group variable and gender as a between-group variable. The analysis revealed that the effect of the TLEQ on cortisol levels was not influenced by gender, $F(1,182) = .54$, *ns* (see Figure 8).

Stress Reactivity and Overall Trauma

In order to assess whether previous traumatic experiences influenced stress reactivity in response to the TLEQ, a separate set of analyses of variance, using the Greenhouse-Geisser correction, was conducted to evaluate variations of endocrine ratios relative to the baseline measure taken upon arrival at the laboratory. Gender and previous trauma history trauma categories (in which participants that reported more than 5 traumatic events were coded as 1, those reporting between 3 and 5 traumatic events were coded as 2, those reporting 1 and 2 traumatic events were coded as 3 and those reporting no traumatic events coded as 4) were considered as between-group variables. Figure 9 suggests that participants with more traumatic experiences exhibited lower cortisol levels compared to the participants reporting less traumatic experiences, but once again this difference was not statistically significant, $F(6,272) = .36$, *ns*.

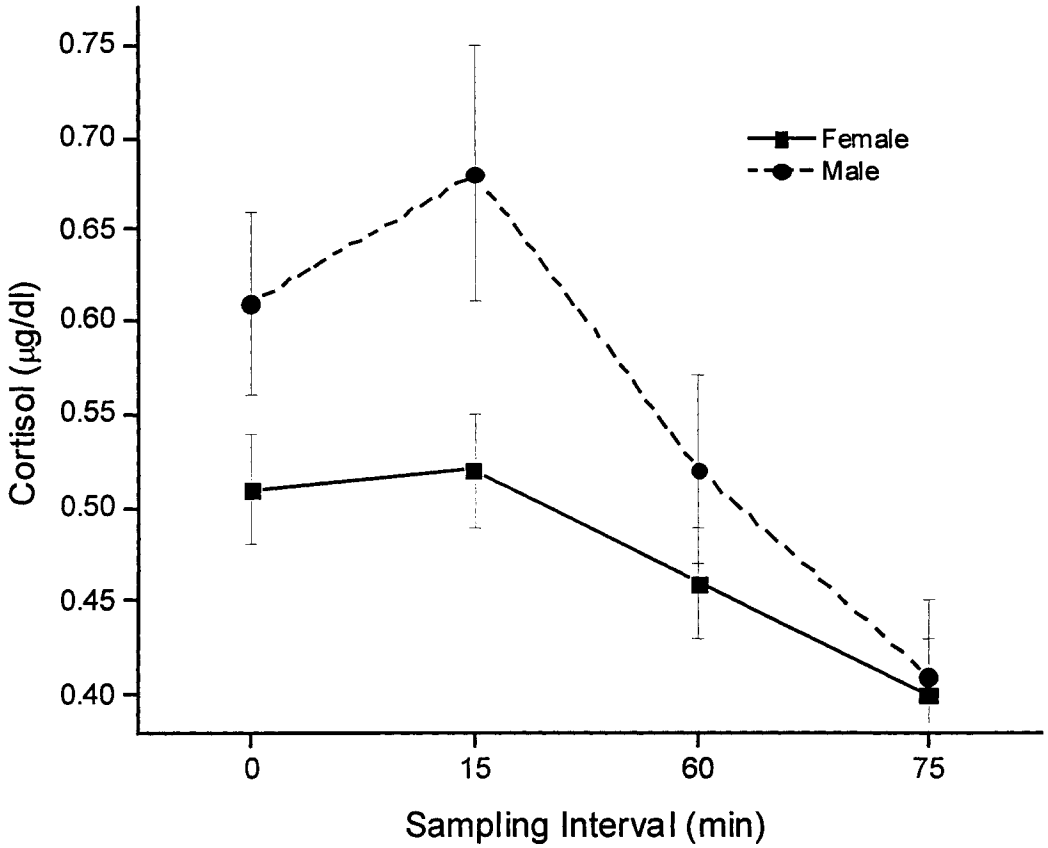


Figure 7 shows the relation between gender and cortisol changes following the completion of the trauma recall measures. Both genders exhibited an increased in baseline levels of cortisol following their arrival to the laboratory, and this effect was particularly prominent in males (dotted line) relative to females (solid line).

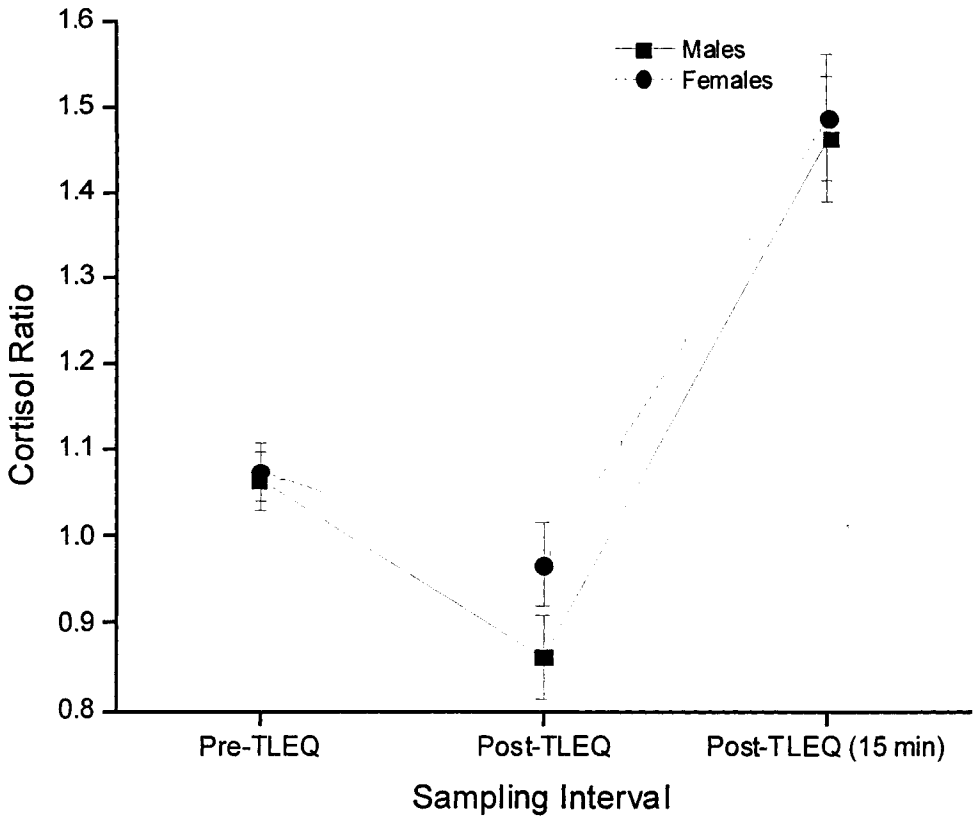


Figure 8 shows the cortisol changes in males (solid line) and females (dotted line) following the completion of the trauma recall measures, as a proportion of the cortisol score relative to baseline levels.

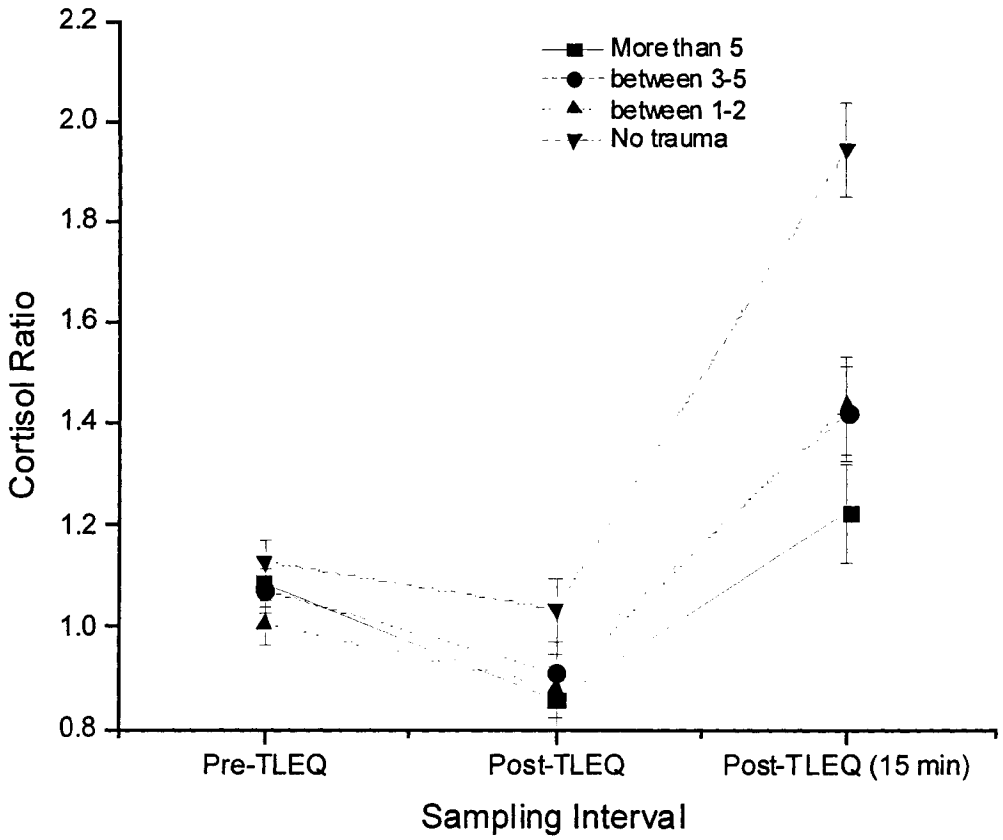


Figure 9 shows the relation between previous traumatic history and cortisol changes after the completion of trauma recall measures. Although the difference as a function of traumatic experiences was not significant, participants with more traumatic experiences tended to exhibit lower cortisol levels compared to the participants reporting fewer traumatic experiences.

Stress Reactivity and Early Life Trauma

In response to completing the questionnaires concerning early life trauma history (i.e., prior to age 5), a mixed measures analysis of variance, using the Greenhouse-Geisser correction, was conducted in which early life events (participants reporting no early life events were coded 1 and those reporting more than 1 adverse early life event were coded 2) and gender were considered as between-subjects variables and cortisol ratios as a within-subject variable. This analysis revealed a significant interaction between gender, the overall cortisol and early life distress, $F(2,360) = 1.81$, $p < .05$, $\eta^2 = .03$. Further comparisons of the means of simple effects of significant interactions were conducted using Tukey's multiple comparisons ($\alpha = .05$). Specifically, males that had encountered early life distress tended to exhibit lower cortisol levels and a more profound decline following the TLEQ compared to males that did not, $F(2,84) = 2.03$, $p<.05$, $\eta^2 = .12$ (see Figure 10). Although Figure 11 suggests that females that had encountered distress in early life tended to exhibit higher cortisol levels following the recall of their previous traumatic experiences compared to females that did not, no significant differences were found, $F(2,276) = .397$, *ns*.

Stress Reactivity and Bonding

Examination of the zero-order correlations indicated a significant relation between stress reactivity and maternal bonding, $r = .16$, $p < .05$, but not paternal bonding, $r = .04$, $p > .05$. Thus, participants were assigned to 2 categories: a) low maternal bonding, coded 0; in order to be included in this category participants had to obtain a maternal bonding score greater than the mean + 2/3 S.D) and b) high maternal bonding, coded 1; in order to be included in those categories, participants had to obtain a maternal

bonding scores lower than the mean - 2/3 S.D. Repeated measures analysis of variance revealed that the overall cortisol change among participants reporting good maternal bonding was not significantly different than that seen among participants that had reported poor maternal bonding, nor did gender interact with bonding to influence cortisol levels following the TLEQ (see Figure 12).

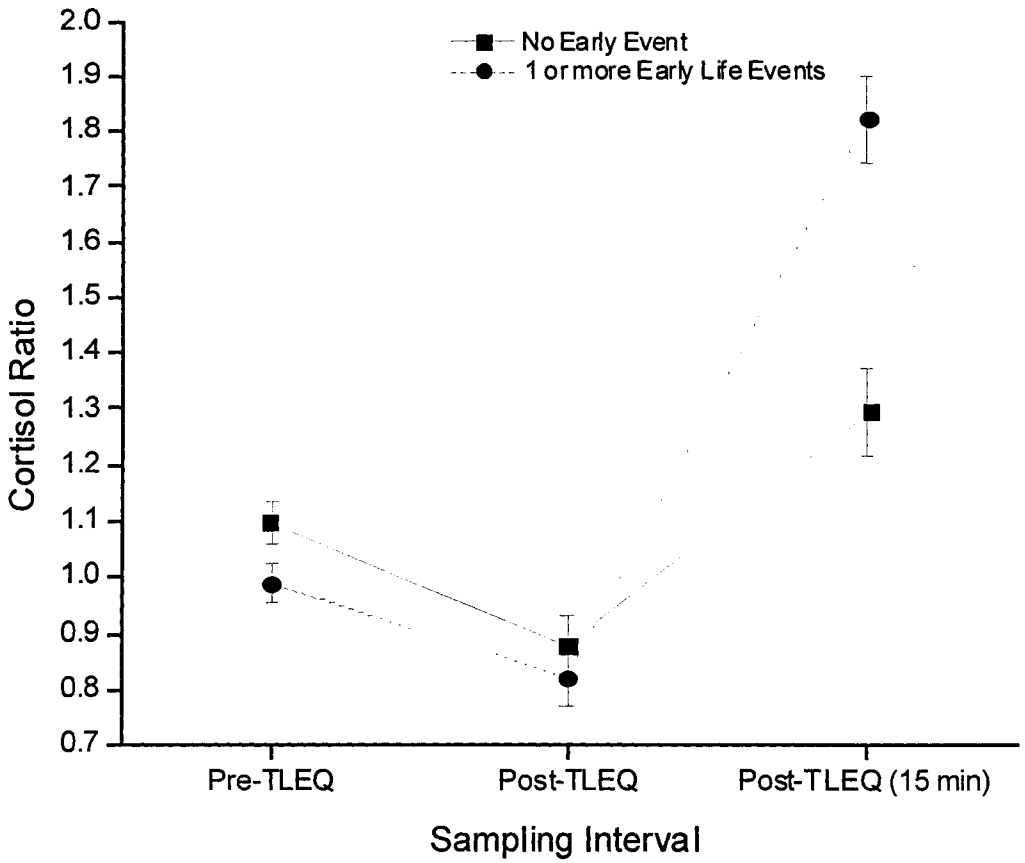


Figure 10 indicates that males that had encountered early life distress (dotted line) tended to exhibit lower cortisol levels and a more profound increase following the TLEQ compared to males that did not (solid line).

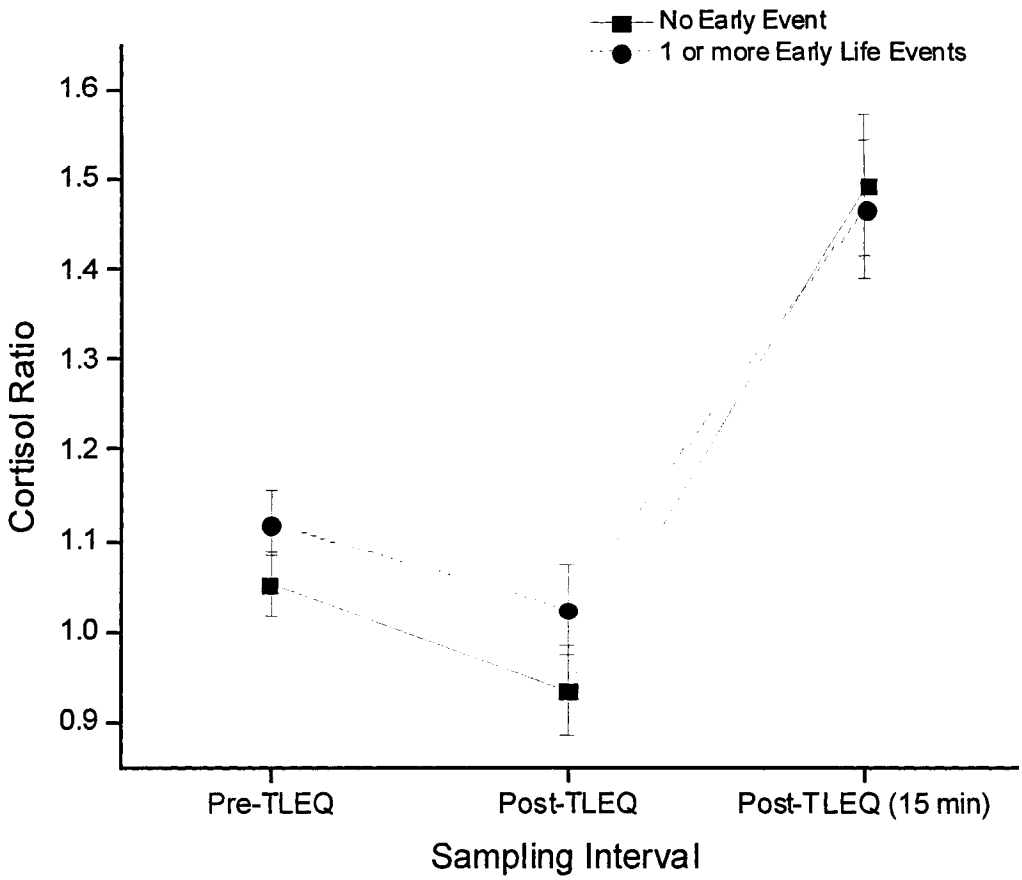


Figure 11 indicates that females that had encountered distress in early life (dotted line) tended to exhibit higher cortisol levels following the recall of their previous traumatic experiences compared to females that did not (solid line).

Figure 12

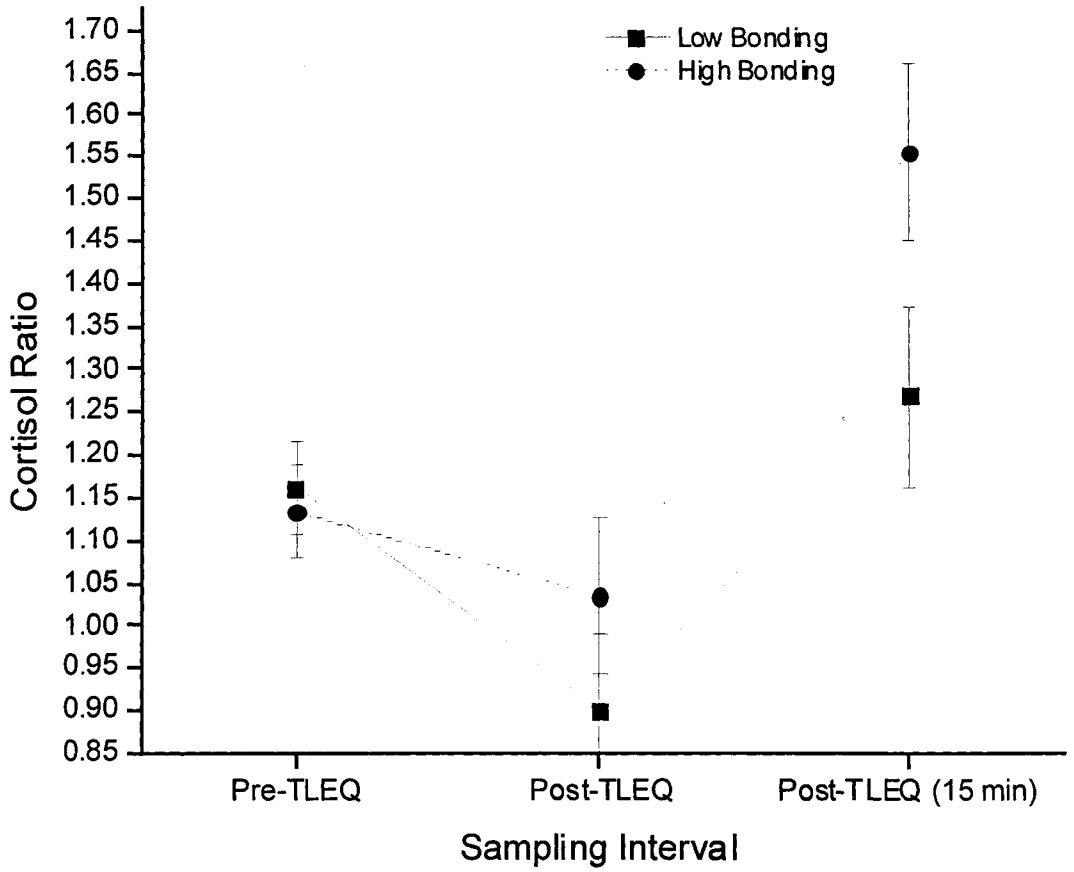


Figure 12 indicates that even if this difference is not significant, participants reporting good maternal bonding (dotted line) tended to exhibit higher cortisol changes following the completion of the TLEQ compared to those seen among participants that had reported poor maternal bonding (solid line).

Discussion

Early life experiences have been shown, both in humans and in animals, to profoundly influence adult behavior and particularly the response to stressors (Meaney, 2001). In this regard, adverse early life experiences have been associated with increased vulnerability to adult depression as well as anxiety disorders (Bifulco et al., 2000; Hammen et al., 1992; Kessler, Davis, & Kendler, 1997; Roy, 1983, 1985; Weiss, Longhurst, & Mazure, 1999). Consistent with animal studies that have reported that the interaction between a dam and her pups is fundamental in predicting adult stress reactivity and vulnerability to depressive-like behaviors, retrospective studies in humans indicated a strong relationship between poor parental bonding and adult depression (Alnaes & Torgerson, 1990; Fergusson et al., 2000; Kerver, van Son, & de Groot, 1992; Lara et al., 2000; Lizardi & Klein, 2000; Martin & Waite, 1994; Meaney, 2001; Parker, 1993; Parker et al., 1995; Rodgers, 1996). Consistent with these findings, in the present study perceived poor maternal and paternal bonding was associated with greater depressive symptoms in adulthood.

In contrast to bonding, a significant relation was not evident between depression and trauma experienced prior to the age of 5 years. This included trauma experiences such as assault, natural disaster, and witnessing violence while growing up. Moreover, the absence of an effect of early life trauma occurred in both men and women. Of course, it is possible that traumatic events experienced when young, especially those that occurred repeatedly or were present on a chronic basis (e.g. sexual abuse or severe neglect), may have profound long-term ramifications provided that adult stressor

experiences were subsequently encountered (Anisman & Matheson, 2005; Meaney, 2001).

While these findings are consistent with research indicating that early life experiences may profoundly influence adult well-being (Bowlby, 1980; Meaney, 2001), it is possible that the negative cognitive bias common among depressed individuals may have influenced the way they perceived their early-life relations, thus resulting in their indication of poor parental bonding. One might also assume that among depressed individuals their colored view of world might also influence their perceptions of traumatic experiences prior to the age of 5. However, this was not the case as depressed and non-depressed individuals reported comparable trauma experiences. It is possible that the unambiguous nature of traumatic events would not be influenced by a negative cognitive bias, whereas something as ephemeral as "perceived parental bonding" may be more vulnerable to these biases. Yet, as indicated earlier, twin studies indicated a high degree of concordance as to perceptions of parenting styles, and it has also been found that the perceptions of depressed individuals regarding their parents could be corroborated and were highly reliable (Parker, 1981, 1986; Parker et al., 1997). Thus, there is reason believe that retrospective accounts of poorer parental bonding are not merely a reflection of a negative evaluative bias (Duggan, Sham, Minne et al., 1998). Furthermore, this negative perception of parents does not seem to result from the greater propensity of depressed individuals to blame others for their difficulties. Indeed, when the coping strategy comprising other-blame was controlled for, the proportion of variance in depressive symptoms explained by bonding was virtually unchanged.

Despite the suggestion that parental bonding may, in fact, be related to depressive symptoms, the present data ought to be considered with a degree of caution. One cannot fully dismiss the possibility that retrospective reports of parental bonding were negatively biased. However, this suggestion does not necessarily imply that these perceptions do not impact adult well-being and psychosocial functioning. Indeed, some investigators have considered retrospective reports of parental bonding to represent individuals' "working models" of their relationships with their parents, and that the perceptions associated with these working models may influence adult well-being and psychosocial functioning (Carnelly, Pietromonaco, & Jaffe, 1994). In this regard, Preussner, Champagne, Meaney et al. (2004) found that several physiological indices of stress reactivity, including changes of brain functioning detected through positron emission tomography, were associated with retrospective reports of parental bonding.

In addition, consistent with previous research (Matheson et al. 2005; Uehara, Sakado, Sato et al., 1999), data from the present study suggested that the pathway by which perceived parenting styles lead to depressive symptomatology in adulthood involved the coping strategies used to contend with stressors. In this regard, it was shown that poor parental bonding was associated with increased endorsement of emotion-focused strategies and diminished use of problem-focused coping and social-support seeking. These altered coping strategies constitute the prototypical coping profile of depressed individuals (Billings & Moos, 1981; Carver et al., 1989; Matheson & Anisman, 2003). It is important to note that when these coping strategies were controlled for, the relations between parental bonding and depressive affect were largely or entirely explained. As such, it seems likely that parental factors may affect the way in which

individuals cope with stressors and hence, may influence the emergence of mood changes in response to stressors. Of course, the present findings are correlational in nature and the possibility cannot be ruled out that alternate routes exist between bonding and depressive symptoms.

It is, of course, well documented that mothers and fathers differentially influence the development and socialization of their children (Collins & Russell, 1991). In a like fashion, the influence of the two parents in the promotion of depressive symptoms may not be identical. In fact, the results of the present study highlight the fact that this was the case. Specifically, it was found the pathway by which parental bonding influenced coping strategies and consequently favor depressive symptomatology, were somewhat different for mothers and fathers. In this regard, the relation between paternal bonding, coping and depressive symptoms seemed to be partially explained by competition-based self-worth.

There are reasons to believe that the differential influence of the mother and father on competition-based self-worth may be explained by the differences in the parent's interactions with their children. Indeed, it was generally shown that father-child interactions tend to place greater emphasis on autonomy, involve more recreation and problem-solving, and tend to be more goal-oriented, whereas the interactions with the mother tend to be more frequent, nurturing, empathic and intimate (Bames & Olsen, 1985; Caplow, Bahr, Chadwick, Hill & Williamson, 1982; Olsen, McCubbin, Bames et al. 1983). In particular, men seem to have a tendency to excite, surprise, and momentarily destabilize children; they also tend to encourage children to take risks, permitting them to learn to be brave in unfamiliar situations, as well as to stand up for

themselves. Thus, it was postulated that father-child interactions encourages the development of competitive skills and openness to the world (Paquette, 2004), which may be reflected in the contingencies of self-worth. Thus, it appears that a greater emphasis on failures and successes in the competition domains in order to define their self-worth is specific to a weaker paternal bond, whereas less emphasis on family's love to define self-worth is related to a weaker bond with the both parents.

Cortisol Diurnal Pattern

It will be recalled that under non-stressor conditions, cortisol release follows a well-defined diurnal rhythm. Specifically, in humans, glucocorticoid release, already high at awakening, rises during the ensuing 30-60 minutes, and decreases slowly thereafter (Linkowski et al., 1993; Schmidt-Reinwald et al., 1999). While this pattern appears to be independent of an individual's 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). In the present study, as previously observed (Klimes-Dougall, Hastings, Granger et al., 2001), females showed similar morning cortisol levels compared to males, but higher cortisol levels at midday and late afternoon.

Most of the research concerning the neuroendocrine response elicited by stressors has focused on absolute changes of cortisol levels at a particular point in time. Indeed, major depression has been associated with elevated cortisol levels (Nemeroff, 1998), whereas PTSD and other chronic stressor experiences (e.g. childhood maltreatment, poor relationship functioning) have been associated with blunted HPA functioning, reflected by diminished cortisol levels (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; Yehuda, 2002; Yehuda et al., 1993, 1996, 1998; Wang, 1997). However, recent evidence suggests that variations of the diurnal rhythmicity of cortisol might provide more information and insight about the neuroendocrine functioning associated with stressor-related pathological states. It will be recalled that cortisol levels ordinarily rise by about 40-60% over the first 30 min following awakening (irrespective of wake time), and then decline precipitously over the rest of the day (Linkowski et al., 1993; 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.

In contrast to the effects of moderate stressors, there is reason to believe that traumatic events (or chronic stressor experiences) may be associated with the blunting of the diurnal cortisol curve. For instance, morning cortisol levels were reduced among victims showing symptoms of PTSD (measured 5 days after trauma), but evening cortisol levels were elevated in these same individuals. Moreover, with the attenuation of symptoms over a 9-month period, cortisol levels normalized (Aardal-Eriksson et al., 2001). Paralleling these findings, it was observed that the normal diurnal cortisol profile was flattened in 29% of non-hospitalized veterans with PTSD and 42% of hospitalized veterans with PTSD (Lauc, Zvonar, Vuksic & Flogel, 2004). Similarly, women who experienced intimate partner violence exhibited elevated afternoon, but not morning cortisol levels (Pico-Alfonzo, Garcia-Linares, Celda-Navarro, Herbert & Martinez, 2004). Brunet and Meaney (personal communication) likewise observed that among

individuals that had encountered a trauma (motor vehicle accident) and then developed PTSD, the normal circadian cortisol changes were not evident. Instead, these individuals exhibited a relatively flat profile over the course of the day, essentially showing cortisol below control levels in the morning, but higher than those of control participants in the afternoon. Like severe stressors, a flattened cortisol curve was evident in association with the chronic distress of the appearance of metastatic breast cancer (Abercrombie, Giese-Davis, Sephton, Eppel, Turner-Cobb & Spiegel, 2004).

In line with the PTSD findings, the present investigation revealed that participants that reported five or more traumatic experiences exhibited flatter cortisol profiles and lower cortisol levels relative to participants reporting fewer traumatic experiences. Whether or not these differences were due to PTSD symptomatology is uncertain, however, this might be a plausible explanation given the fact that the participants reporting more than 5 traumatic experiences also reported also more early adverse events such as maltreatment and abuse while growing up. In effect, the flattened diurnal cortisol rhythm may be a result of multiple (or chronic) traumatic events, coupled with early life adverse experiences.

Cortisol-Stressor Reactivity

Studies of stressor evoked neuroendocrine alterations in humans suggest a complex relationship between several factors that govern the effects of stressors (e.g. characteristics of the stressor, stressor appraisal, and environmental factors) and adrenal corticoid release. In this regard, gender differences in biobehavioral response to stressors, including the endocrine system, have frequently been reported in both animals and human studies (Frankenhaeuser, Rauste VonWright, Collins et al. 1978; Hellhammer

& Wade, 1993; Kirschbaum, Wust & Hellhammer, 1992; Schaeffer & Baum, 1984). Specifically, it has typically been reported that, in humans, males were more stressor-reactive, exhibiting a greater cortisol changes in response to public speaking and mental arithmetic compared to females. In contrast, exercise to exhaustion or administration of CRH elicited similar cortisol elevations in males and females (Kirschbaum et al., 1992). These data raise the possibility that HPA functioning in males and females are comparable, but that males are more sensitive to some stressors than are females. Alternatively, it might be that cortisol release may be a less sensitive indicator of psychological stress in females (Biondi & Picardi, 1999).

Consistent with previous research, the present study indicated a more prominent cortisol change for males than females. Moreover, as reported by Ennis, Kelly and Lambert (2001), males exhibited higher cortisol changes after their arrival to the laboratory. Similarly, Spangler (1997) reported that males rated as High-Ego control, but not females, exhibited increased salivary cortisol in response to an oral examination. In contrast, Lacey, Zaharia, Griffith et al. (2000) demonstrated that it was predominately female graduate students who exhibited elevated levels of cortisol 1 hour prior to their PhD dissertation oral examination relative to matched controls. While the sex-dependent plasma cortisol variations in anticipation of the previous traumatic experiences may have been spurious, it is equally possible that this might reflect differences in the way males and females perceive or cope with this type of event, and comparable sex-dependent effects would not occur in other types of stressful situations. In this regard, Taylor, Klein, Lewis et al. (2000) postulated the tend-and-befriend theory that suggests that in response to stress, women may be more likely to engage in nurturing and network

building activities to promote safety and reduce distress. In this study, even if women tended to be more likely to use social support compared to men, this difference did not reach significance.

As alluded earlier, the HPA response is not only influenced by several organismic variables, but is also affected by characteristics of the stressor itself. Indeed, certain stressors may elicit outcomes dramatically different from one another and within a laboratory context, it does not seem that all stressors effectively influence the HPA response. In particular, Dickerson and Kemeny (2004) argued that "social evaluative challenges", such as public speaking, are most effective in eliciting and adrenal corticoid response, whereas mood induction procedures or passively viewing negative images (e.g., films) are particularly ineffective. In the present investigation, the corticoid response was limited despite the fact that the method did not comprise a simple mood induction procedure, but essentially involved recall of previous events, notably traumatic experiences. It is possible that reminder cues concerning their trauma, in another context, notably one that involved social evaluation, might have been more effective in promoting cortisol release.

Like acute stressors, traumatic events may also promote increased physiological changes, including elevated neuroendocrine activation, presumably to deal effectively with the ongoing events (Sapolsky et al., 2000). Given that cortisol levels in plasma, urine or saliva (all of which reflect adrenal release) are reduced among individuals presenting with PTSD (Boscarino, 1996; Goenjian, et al., 1996; Gurvits et al., 2000; Kellner et al., 1997; Mason et al., 1986; Yehuda et al., 1993; Yehuda et al., 1996, 1998; Wang, Mason, Charney et al., 1997), long-term arousal of these adaptive mechanisms by

sufficiently intense or protracted trauma, at least one sufficiently potent to provoke PTSD symptoms, may have deleterious consequences (e.g. physical and psychological disturbances) (McEwen, 2000), ultimately culminating in down regulation of adrenal functioning (McEwen, 1998; Yehuda, et al., 1996).

In light of these earlier findings, it was expected that reminder cues of previous traumatic experiences might influence neuroendocrine reactivity, but such an outcome would be related to the intensity or frequency of such experiences. Specifically, it was expected that cortisol levels would increase in response to reminder cues associated with a small number or intensity of adverse experiences, whereas a blunted response would be evident among individuals that had experienced frequent and/or severe trauma. In line with this proposition, participants that reported more traumatic experiences exhibited lower cortisol levels in response to the reminder stimuli, compared to those individuals that had experienced less traumatic experiences. It is uncertain, however, whether or not the reduced cortisol was due to the traumatic experiences. While some researchers suggest that it is the experience, rather than the presence of PTSD, that is the fundamental contributor to this outcome (Seedat, LeRoux & Stein, 2003), research conducted in our laboratory indicated that cortisol changes also varied as a function of whether past trauma provoked PTSD symptoms in response to this same challenge (i.e., the TLEQ). In effect, recall of past traumas provoked an inverted U-shaped function in relation to IES-R scores, irrespective of the type of trauma experienced.

As predicted, early adverse events also seemed to influence the cortisol responses elicited by the experimental challenge. Indeed, participants that reported adverse early life events tended to exhibit higher cortisol concentrations than did the participants

reporting no such event. This is consistent with previous studies showing that stressful events, including early life trauma, may result in the sensitization of neurochemical processes, so that later introduction of stressors elicit an exaggerated response (Anisman et al., 2003; Heim and Nemeroff, 2001; Nemeroff, 1996; Post, 1992), depending on the nature of the challenge (Heim and Nemeroff, 2001; Heim, Newport, Bonsall et al. 2001; Heim, Newport, Heit et al., 2000). This sensitized neuroendocrine response could potentially account for the increased risk of depression and PTSD associated with previous stressful experiences.

Conclusion

In summary, poor parental bonding seems to facilitate the acquisition of ineffective coping skills, and hence increase the vulnerability to depressive symptoms. Indeed, poor parental bonding was related to the endorsement of particular coping strategies and this coping profile consistently mediated the relations between bonding and depressive symptoms. As previously reported (Billings & Moos, 1981; Carver et al., 1989; Matheson & Anisman, 2003), depressive symptoms were associated with increased endorsement of emotion-focused strategies and diminished use of problem-focused coping. Interestingly, the pathways by which poor maternal and paternal bonding lead to depressive symptoms appears to be different. While poor paternal bonding seems to be related to a competition-based self-worth and this may promote the use of emotion-focused strategies, the greater propensity to use emotion-focused strategies associated with poor maternal bonding was unrelated to competition-based self-worth.

Limited data are available concerning the influence of bonding on stressor-provoked neuroendocrine or brain changes, although Preussner et al (2004) reported that

bonding was related to brain changes when participants were challenged within a laboratory setting. In contrast, there are several studies that indicated that abuse encountered early in life resulted in the sensitization of neuroendocrine and brain systems, thereby influencing the subsequent response to stressors encountered during adulthood. What is not known is whether a passive form of abuse (i.e., neglect) would have a similar effect. Absence of bonding may well represent a form or precursor of neglect and might thus be expected to have profound effects on neuroendocrine functioning. While the present findings are certainly consistent with such a position, it must again be underscored that this conclusion is based primarily on retrospective findings. Despite the studies suggesting that the individual's perceptions of past events are accurate, retrospective data must be considered cautiously.

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Appendix A

Informed Consent

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 : Stressor Appraisal and ambiguous events: relation to psychological well-being, previous trauma and neuroendocrine function – Phase 1

Study Personnel: Dr. Kim Matheson (Faculty Investigator, 520-2648)
Dr. Hymie Anisman (Faculty Investigator, 520-2699)
Kathy Michaud (Researcher, 520-2600 ext. 2683)
Owen Kelly (Researcher, 520-2600 ext. 2683)

If you have any ethical concerns about how this study please contact Dr. M. Gick (Chair of the Carleton University Research Ethics Committee for Psychological Research, 520-2600, ext. 2664) or John Logan, Chair of the Department of Psychology, Carleton University, 520-2600, ext. 2648

Purpose, Task Requirements, and Time for Participation: This study is comprised of two phases. The goal of the first phase (which you will be completing today) is to examine the relationship between a number of variables including the experience of previous traumatic events, self-esteem and family history, and how they may relate to psychological well-being. Essentially, this involves completing a number of questionnaires regarding psychological health, family history, how you cope with stressful events, as well as a variety of other psychological factors. During the study, you will be given a series of questions that asks about some potentially traumatic events you may have experiences (e.g. war, assault, or loss of a love one) that may have affected how you deal with other events in your life. This first phase of the study will take about 1hr to complete.

You may also be asked for permission to contact you for your participation in the second phase of the study, as well as a follow-up study later in the year. You will receive **1 credit** toward your 49.101/102 experimental credits for your participation in this phase of the study.

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 that require focusing on the distress associated with past events.

Anonymity/Confidentiality: The data collected in this study will be kept confidential. Your informed consent form will be separated from your saliva samples and questionnaires and kept in a separate and secured file by one of the research investigators who will keep this information confidential. It will be associated with a code, and only this code will identify your questionnaires in both phases of the study (should you wish to participate in both).

Right to Withdraw: Your participation in this study is entirely voluntary. At any point during the study you have the right to withdraw entirely. You also have the right to refuse to answer specific questions.

I have read the above description of the study concerning appraisal of ambiguous events in relation to psychological well-being, early-life experiences and neuroendocrine activation. The data collected will be used in research publications and/or for teaching purposes. Participants may request a summary of results for the study when it is completed; however, scores of individual participants cannot be provided. 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 Signature:_____
Date:_____
Researcher Signature : _____
Date :_____

Appendix B
BACKGROUND

Age _____

Sex_____

Ethnic/racial background_____

Current GPA _____

Year in program_____

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 C

BECK DEPRESSION INVENTORY (BDI)

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

9. ___ 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 c= 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

Appendix D
PARENTAL BONDING INDEX (PBI)

This questionnaire lists various attitudes and behaviors of parents (e.g. Mother and Father) that we are interested in. If you feel that any of these questions do not apply to your specific situation (Example: you were raised in a single parent household by your mother and had no contact with you father) please fill in only those sections that you feel apply to you. If you have chosen not to fill in a section, you may indicate your reason for not doing so in the space below.

_____
_____
_____
_____

First, as you remember your **Mother** in the **first 16 years of your life**, please place a check in the space that indicates the most appropriate response to each statement.

	Very *True*	**Rather** *True*	**Rather** *Untrue*	**Very** *Untrue*
1. Spoke to me with a warm and friendly voice.	( )	( )	( )	( )
2. Did not help me as much as I needed.	( )	( )	( )	( )
3. Let me do those things that I liked doing.	( )	( )	( )	( )
4. Seemed emotionally cold to me.	( )	( )	( )	( )
5. Appeared to understand my problems and worries.	( )	( )	( )	( )
6. Was affectionate to me.	( )	( )	( )	( )
7. Wanted me to make my own decisions.	( )	( )	( )	( )
8. Did not want me to grow up.	( )	( )	( )	( )
9. Tried to control everything I did.	( )	( )	( )	( )
10. Invaded my privacy.	( )	( )	( )	( )
11. Enjoyed talking things over with me.	( )	( )	( )	( )
12. Frequently smiled at me.	( )	( )	( )	( )
13. Tended to baby me.	( )	( )	( )	( )
14. Did not seem to understand what I needed or wanted.	( )	( )	( )	( )
15. Let me decide things for myself.	( )	( )	( )	( )
16. Made me feel I wasn't wanted.	( )	( )	( )	( )
17. Could make me feel better	( )	( )	( )	( )

when I was upset.				
18. Did not talk to me very much.	( )	( )	( )	( )
19. Tried to make me dependent on her.	( )	( )	( )	( )
20. Felt I could not look after myself unless she was around.	( )	( )	( )	( )
21. Gave me as much freedom as I wanted.	( )	( )	( )	( )
22. Was overprotective of me.	( )	( )	( )	( )
23. Did not praise me.	( )	( )	( )	( )
24. Let me dress in any way I wanted.	( )	( )	( )	( )

As you remember your **Father** in the **first 16 years of your life**, please place a check in the space that indicates the most appropriate response to each statement.

	True	*True*	**Very** *Untrue*	**Rather** *Untrue*	**Rather**	**Very**
1. Spoke to me with a warm and friendly voice.			( )	( )	( )	( )
2. Did not help me as much as I needed.			( )	( )	( )	( )
3. Let me do those things that I liked doing.			( )	( )	( )	( )
4. Seemed emotionally cold to me.			( )	( )	( )	( )
5. Appeared to understand my problems and worries.			( )	( )	( )	( )
6. Was affectionate to me.			( )	( )	( )	( )
7. Wanted me to make my own decisions.			( )	( )	( )	( )
8. Did not want me to grow up.			( )	( )	( )	( )
9. Tried to control everything I did.			( )	( )	( )	( )
10. Invaded my privacy.			( )	( )	( )	( )
11. Enjoyed talking things over with me.			( )	( )	( )	( )
12. Frequently smiled at me.			( )	( )	( )	( )
13. Tended to baby me.			( )	( )	( )	( )
14. Did not seem to understand what I needed or wanted.			( )	( )	( )	( )

15. Let me decide things for myself.	( )	( )	( )	( )
16. Made me feel I wasn't wanted.	( )	( )	( )	( )
17. Could make me feel better when I was upset.	( )	( )	( )	( )
18. Did not talk to me very much.	( )	( )	( )	( )
19. Tried to make me dependent on her.	( )	( )	( )	( )
20. Felt I could not look after myself unless she was around.	( )	( )	( )	( )
21. Gave me as much freedom as I wanted.	( )	( )	( )	( )
22. Was overprotective of me.	( )	( )	( )	( )
23. Did not praise me.	( )	( )	( )	( )
24. Let me dress in any way I wanted.	( )	( )	( )	( )

Appendix E

SURVEY OF COPING ENDORSEMENT (SCOPE)

The purpose of this questionnaire is to find out how people deal with their problems or stresses in their lives. The following are activities that you may have done. After each activity, please indicate the extent to which you would use this as a way of dealing with problems or stresses in the recent weeks.

Ordinarily, in recent weeks have you	Never	Seldom	Sometimes	Often	Almost always
1. accept that there is nothing you an do to change your situation?	0	1	2	3	4
2. try to just take whatever comes your way?	0	1	2	3	4
3. talk with friends or relatives about your problems?	0	1	2	3	4
4. try to do things which you typically enjoy?	0	1	2	3	4
5. sought out information that would help you resolve your problems?	0	1	2	3	4
6. blame others for creating your stress or making it worse?	0	1	2	3	4
7. sought the advice of others to resolve your problems?	0	1	2	3	4
8. blamed yourself for your problems?	0	1	2	3	4
9. exercised?	0	1	2	3	4
10. fantasized or thought of unreal things (e.g. the perfect revenge, or winning a million dollars) to feel better?	0	1	2	3	4
11. gone over your problems in your mind over and over again?	0	1	2	3	4
13. asked others for help?	0	1	2	3	4
14. thought about your problems a lot?	0	1	2	3	4
15. became involved in recreation or pleasure activities?	0	1	2	3	4
16. worried about your problems a lot?	0	1	2	3	4
17. tried to keep your mind off things that are upsetting you?	0	1	2	3	4
18. tried to distract yourself from your troubles?	0	1	2	3	4
19. avoided thinking about it?	0	1	2	3	4
20. made plans to overcome your problems?	0	1	2	3	4
21. told jokes about your situation?	0	1	2	3	4
22. thought a lot about who is responsible for your problems (besides yourself)?	0	1	2	3	4
23. shared humorous stories etc. to cheer yourself and others up?	0	1	2	3	4
24. told yourself that other people have dealt with problems such as yours?	0	1	2	3	4
25. thought a lot about how you have brought					

your problems on yourself?	0	1	2	3	4
26. decided to wait and see how things turn out?	0	1	2	3	4
27. wished the situation would go away or be over with?	0	1	2	3	4
28. decided that your current problems are a result of your own past actions?	0	1	2		4
29. go shopping?	0	1	2	3	4
30. asserted yourself and taken positive action on problems that are getting you down?	0	1	2	3	4
31. sought reassurance and moral support from others?	0	1	2	3	4
32. resigned yourself to your problems?	0	1	2	3	4
33. thought about how your problems have been caused by other people?	0	1	2	3	4
34. daydreamed about how things may turn out?	0	1	2	3	4
35. been very emotional in how you react, even to little things?	0	1	2	3	4
36. decided that you can grow and learn through your problems?	0	1	2	3	4
37. told yourself that other people have problems like your own?	0	1	2	3	4
38. wished you were a stronger person or better at dealing with your problems?	0	1	2	3	4
39. looked for how you can learn something out of your bad situation?	0	1	2	3	4
40. asked for God's guidance?	0	1	2	3	4
41. kept your feelings bottled up inside?	0	1	2	3	4
42. found yourself crying more than usual?	0	1	2	3	4
43. tried to act as if you were not upset?	0	1	2	3	4
44. prayed for help?	0	1	2	3	4
45. gone out?	0	1	2	3	4
46. held in your feelings?	0	1	2	3	4
47. tried to act as if you weren't feeling bad?	0	1	2	3	4
48. taken steps to overcome your problems?	0	1	2	3	4
49. made humorous comments or wise cracks?	0	1	2	3	4
50. told others that you were depressed or emotionally upset?	0	1	2	3	4
51. gone gambling?	0	1	2	3	4
52. eaten more than usual?	0	1	2	3	4

Appendix F
CONTINGENCIES OF SELF-WORTH SCALE (CSW)

1	2	3	4	5	6	7
Strongly disagree	**Disagree**	**Disagree somewhat**	**Neutral**	**Agree somewhat**	**Agree**	**Strongly agree**

____ 1. When I think I look attractive, I feel good about myself.

____ 2. My self-worth is based on God's love.

____ 3. I feel worthwhile when I perform better than others on a task or skill.

____ 4. My self-esteem is unrelated to how I feel about the way my body looks.

____ 5. Doing something I know is wrong makes me lose my self-respect.

____ 6. I don't care if other people have a negative opinion about me.

____ 7. Knowing that my family members love me makes me feel good about myself.

____ 8. I feel worthwhile when I have God's love.

____ 9. I can't respect myself if others don't respect me.

____ 10. My self-worth is not influenced by the quality of my relationships with my family members.

____ 11. Whenever I follow my moral principles, my sense of self-respect gets a boost.

____ 12. Knowing that I am better than others on a task raises my self-esteem.

____ 13. My opinion about myself isn't tied to how well I do at work or school.

____ 14. I couldn't respect myself if I didn't live up to a moral code.

____ 15. I don't care what other people think of me.

____ 16. When my family members are proud of me, my sense of self-worth increases.

____ 17. My self-esteem is influenced by how attractive I think my face or facial features are.

____ 18. My self-esteem would suffer if I didn't have God's love.

____ 19. Doing well in school gives me a sense of self-respect.

____ 20. Doing better than others gives me a sense of self-respect.

____ 21. My sense of self-worth suffers whenever I think I don't look good.

____ 22. I feel better about myself when I know I'm doing well academically or at work.

____ 23. What others think of me has no effect on what I think about myself.

____ 24. When I don't feel loved by my family, my self-esteem goes down.

____ 25. My self-worth is affected by how well I do when I am competing with others.

____ 26. My self-esteem goes up when I feel that God loves me.

____ 27. My self-esteem is influenced by my performance either at work or school.

____ 28. My self-esteem would suffer if I did something unethical.

____ 29. It is important to my self-respect that I have a family that cares about me.

____ 30. My self-esteem does not depend on whether or not I feel attractive.

____ 31. When I think that I'm disobeying God, I feel bad about myself.

____ 32. My self-worth is influenced by how well I do on competitive tasks.

____ 33. I feel bad about myself whenever my academic or work performance is lacking.

____ 34. My self-esteem depends on whether or not I follow my moral/ethical principles.

____ 35. My self-esteem depends on the opinions others hold of me.

Appendix G

Debriefing

While most individuals have extensive experience in dealing with everyday stressors, exposure to traumatic events (e.g. combat, car accident, unexpected death of a loved one, natural disaster) can be challenging for many individuals. Not surprisingly, traumatic events frequently cause intense feelings of loss or hopelessness, which may lead to a decrease in psychological well-being. Some of the factors that can lead a decrease in psychological well-being are the threat posed by the event, as well as the individual ability to cope effectively with the trauma.

Interestingly, exposure to negative events early in life may also render some people more vulnerable to stress, and stressor related illness as adults. Indeed, there is a large body of research to suggest that the experience of negative early life events in childhood (e.g. poor relationship between child and parents) is related to major depression in adults. As such, in the current investigation we are interested in identifying those processes by which early life events and other variables (e.g. the coping methods people employ in the face of stressful events, an individual self-esteem) may affect psychological well-being in adulthood.

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)
Owen Kelly (Researcher, 520-2600 ext. 7513)

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

Dr. M. Gick, Chair of the Carleton University Research Ethics Committee for Psychological Research, 520-2600, ext. 2664

John Logan, Chair of the Department of Psychology, Carleton University, 520-2600, ext. 2648

If you are experiencing distress, or if you feel unhappy or depressed, then it is advisable that you contact your family physician, or Health and Counselling Services on campus at **520-6674**. 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.

Appendix H

Informed Consent – Phase 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 : Stressor Appraisal and ambiguous events: relation to psychological well-being, previous trauma and neuroendocrine function – Phase 1

Study Personnel: Dr. Kim Matheson (Faculty Investigator, 520-2648)
Dr. Hymie Anisman (Faculty Investigator, 520-2699)
Kathy Michaud (Researcher, 520-2600 ext. 2683)
Owen Kelly (Researcher, 520-2600 ext. 2683)

If you have any ethical concerns about how this study please contact Dr. M. Gick (Chair of the Carleton University Research Ethics Committee for Psychological Research, 520-2600, ext. 2664) or John Logan, Chair of the Department of Psychology, Carleton University, 520-2600, ext. 2648

Purpose, Task Requirements, and Time for Participation: The general purpose of this second phase of the study is to assess physiological indices of stress by obtaining a number of saliva samples to measure levels of stress hormones. Specifically, you will be given 5 salivettes in order to collect saliva samples at home which may be indicative of stress you are experiencing. In brief, on a date to be determined by both yourself and the interviewer, you will be asked to provide saliva samples upon awakening, 30 minutes later, and then 1, 4, and 6 hours after later for a total of 5 samples. Collecting a saliva sample involves placing a piece of dental cotton in your cheek for a 2 min period. Once the cotton is thoroughly wet, it is placed in a tube, and kept in your fridge until the samples are collected and brought to the lab for analysis. As well, we will be asking you to write a brief account of the events that occurred the day prior to, and the morning of the day on which the saliva samples were obtained. On the same day that you return the samples, we will be asking you to fill out some additional questionnaires, as well as provide additional saliva samples. This portion of the study takes will take about 1 hour.

During the study, 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 vents in your life. As well, we will also be asking you to indicate how you perceive a variety of potentially stressful situations. You may also be asked for permission to contact you for your participation in a follow-up study later in the year. You will receive 1 credit toward your 49.101/102experiemental credit for participating in this second phase of the study, your name will be also entered in a lottery for several weekly prizes (e.g. movie tickets, tickets for a sporting event, dinner coupon, etc).

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 that require focusing on the distress associated with past events.

Anonymity/Confidentiality: The data collected in this study will be kept confidential. Your informed consent form will be separated from your saliva samples and questionnaires and kept in a separate and secured file by one of the research investigators who will keep this information confidential. It will be associated with a code, and only this code will identify your questionnaires in both phases of the study (should you wish to participate in both).

Right to Withdraw: Your participation in this study is entirely voluntary. At any point during the study you have the right to withdraw entirely. You also have the right to refuse to answer specific questions.

I have read the above description of the study concerning appraisal of ambiguous events in relation to psychological well-being, early-life experiences and neuroendocrine activation. The data collected will be used in research publications and/or for teaching purposes. Participants may request a summary of results for the study when it is completed; however, scores of individual participants cannot be provided. 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 Signature:_____
Date:_____
Researcher Signature : _____
Date :_____

Appendix I
TAKE-HOME BOOKLET FOR SALIVA COLLECTION

Code Number:_____

To assess stress levels, we would like you to provide us with saliva samples on :

_____ (date).

These give us an index of your hormonal stress response. We are asking you to provide 5 samples throughout the morning and afternoon of that day..

Sample 1: Provide this sample as soon as you awaken.
Sample 2: Provide this sample 30 minutes after awakening, preferably before you eat.
Sample 3: Provide this sample 1 hour after awakening.
Sample 4: Provide this sample 4 hours after awakening.
Sample 5: Provide this sample 6 hours after awakening.

To do this, place the cotton from inside the tube into your cheek for about 2 minutes. Once the cotton is fully wet (this usually takes 2-3 minutes), place it into the plastic tube. Please try to get as much saliva into the cotton as you can so that it is quite wet.

When you have completed these 5 samples, please put them in the baggie provided along with this form, and keep them in your fridge (when possible). We will collect these from you on the day of your last experimental session.

Thank you very much for your cooperation!

Date of samples: _____

Time of Sample 1 (as soon as you awaken):_____

Time of Sample 2 (30 minutes after awakening): _____

Time of Sample 3 (1 hour after awakening): _____

Time of Sample 4 (4 hours after awakening): _____

Time of Sample 5 (6 hours after awakening): _____

IMPORTANT: Please put the exact time at which each saliva sample was obtained. If you are late collecting a sample, take it anyway, but if at all possible collect the next sample at the appropriate time. You will not be penalized in any way for missing, forgetting or having skip a sample; however, it is absolutely essential that we have accurate information concerning the time at which the saliva samples were obtained.

If you have any questions or concerns about this, please call us at 520-2600, ext. 7513. If you leave a message, we will get back to you shortly.

Please complete the following questions on the same morning that you provide your saliva samples.

1. Did anything happen to you yesterday that irritated or stressed you? If so, please describe.

_____

_____

_____

_____

Please rate how stressful you would describe that event (or if more than one, rate the most distressing event).

1	2	3	4	5
Hardly Stressful at all				Extremely stressful

At approximately what time did this event occur yesterday?_____

2. Did anything happen to you This morning that irritated or stressed you? If so, please describe.

_____

_____

_____

_____

Please rate how stressful you would describe that event (or if more than one, rate the most distressing event).

1	2	3	4	5
Hardly Stressful at all				Extremely stressful

At what time did this event occur this morning?_____

Appendix J
TRAUMATIC LIFE EVENT QUESTIONNAIRE (TLEQ)

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
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

19. Has anyone stalked you – in other words: followed you or kept track of your activities – causing you to feel intimidated or concerned for your safety?

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
Was the person a stranger? yes / no **friend or acquaintance?** yes / no
relative? yes / no **other relative?** yes / no
Did you experience fear, helplessness, or horror when it happened? yes / no

20. Have you ever had a miscarriage?

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

21. Have you ever had an abortion?

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

22. Have you ever had something happened to you that you believe represented an experience of discrimination (e.g., religious, racial, sex)?

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

Was the source of the discrimination a stranger? yes / no
friend or acquaintance? yes / no
someone in your workplace/school? yes / no
an organization/institution yes / no

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

Appendix K
Debriefing (Phase 2)

Ordinarily, levels of the body's stress hormone, cortisol, vary throughout the day in a predictable manner. Specifically, cortisol levels tend to be the greatest about a half-hour following awakening, after which levels of the hormone decline quickly. While most individuals demonstrate similar levels of cortisol upon awakening, the experience of recent or on-going stressful vents may cause certain individuals to exhibit an increase in cortisol secretion over the first half-hour following awakening, in comparison to individuals who have not experienced similar events.

We are also interested in identifying those processes by which early life events may affect later perception of potentially stressful vents in adulthood. For instance, there is a large body of research to suggest that negative early life events (e.g. poor relationship between child and parents) may affect self-esteem and the way we appraise stressors, which may in turn, affect the types of coping strategies people choose when dealing with stressors.

We also wish to assess whether an individual appraisal of events influences the body's response to stress and psychological well-being (e.g. depression). This is done by examining the physiological indices, in this case the lvels of the stress hormone cortisol, which appear in saliva. While stress hormones normally aid the body to deal with stress, having high levels of stress hormones for extended periods of time have harmful effects on the body.

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)
Owen Kelly (Researcher, 520-2600 ext. 7513)

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

Dr. M. Gick, Chair of the Carleton University Research Ethics Committee for Psychological Research, 520-2600, ext. 2664

John Logan, Chair of the Department of Psychology, Carleton University, 520-2600, ext. 2648

If you are experiencing distress, or if you feel unhappy or depressed, then it is advisable that you contact your family physician, or Health and Counselling Services on campus at **520-6674**. 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.