RISK FACTORS FOR DEPRESSION AND ANXIETY:
PARENTING, PERSONALITY AND COPING

by

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ABSTRACT

Two studies examined risk factors for depression and anxiety. Parenting attributes (care and protection), cortisol reactivity (in response to an experimental stressor, the Trier Social Stress Test), personality (self-efficacy and perceived social support), coping styles (task, emotion and avoidance) and stressors (life-events and daily hassles) were included as measures. Cortisol reactivity was applicable for Study 1 only.

Study 1 incorporated experimental and longitudinal components. There was a total of 18 participants for this study. Time 1 measures of self-efficacy, and of anxiety and depressive symptomatology failed to predict later symptomatology. Individual differences in cortisol reactivity were found in relation to differing parenting styles as measured by the Parental Bonding Instrument (PBI). Low care and high protection scores on the PBI were related to higher cortisol reactivity.

Study 2 examined the extent to which early parent-child relationships contribute to anxiety and depressive symptomatology through the development of personality and coping style. A total of 184 Acadia University students participated in this study. A proposed model with mediating and moderating relations among the variables was tested. The relations between parenting and coping styles were mediated by personality. Perceived social support was found to have both direct and indirect effects on mental health with task and emotion coping partially mediating their relation. Self-efficacy also had direct and indirect effects on mental health with emotion coping partially mediating their relation. Stress measures (life events and daily hassles) added to the prediction of anxiety and depressive symptomatology above coping and personality. Implications regarding findings and future directions are discussed.
LIST OF ABBREVIATIONS

ACTH........................Adrenocorticotropic Hormone
BDI-II........................Beck Depression Inventory-2nd Edition
C1..........................Cortisol Baseline (Time 1) Measure
C2..........................Cortisol Time 2 Measure
C3..........................Cortisol Time 3 Measure
C2 – C1........................Cortisol Time 2 minus Time 1 (Physiological Reactivity)
CISS........................Coping Inventory for Stressful Situations
CRH..........................Corticotrophin Releasing Hormone
DHS..........................Daily Hassles Scale
GSE..........................General Self-Efficacy scale
HPA..........................Hypothalamic-pituitary-adrenal
LES..........................Life-Experiences Survey
PBI-C........................Parental Bonding Instrument-Care
PBI-O........................Parental Bonding Instrument-Overprotection
SSS..........................Sense of Social Support
STAI-S........................State-Trait Anxiety Inventory-State subscale
STAI-T........................State-Trait Anxiety Inventory-Trait subscale
TSST..........................Trier Social Stress Test
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Risk Factors for Depression and Anxiety: Parenting, Personality and Coping

Due to the close relation and high comorbidity between depression and anxiety (Dobson, 1985; Hecht, von Zerssen, Krieg, Pössl, and Wittchen, 1989; Kendler, Neale, Kessler, Heath, & Eaves, 1992), it was decided to include both as measures of mental illness for the present study. It should be noted, however, that the primary focus of the present study is depression, and as such anxiety literature is primarily reviewed as it relates to depression.

Several risk factors for depression have been identified, including exposure to anomalous or pathogenic parenting (Bowlby, 1977; Holmes & Robins, 1988; Parker, 1979c), personality (Boyce, Parker, Barnett, Cooney, & Smith, 1991; Oliver & Paull, 1995; Parker, 1993), temperament (Kendler, Kessler, Neale, Heath, & Eaves, 1993), and stressful life events (Brown & Harris, 1978; Fleming, Baum, Gisriel, & Gatchel, 1985; Kendler et al., 1993; Richman & Flaherty, 1986). These risk factors were identified from decades of etiological research on mental illness from various theoretical perspectives. The search for internal and external factors (i.e., endogenous-exogenous dichotomy) that determine mental illness has, furthermore, resulted in much conflict over both the identification of symptom features, and nomenclature (Berrios, 1992; Glas, 1994).

Today's classification of depression stems from the mid-nineteenth century with the identifying features of "melancholia" leading to use of the term "mental-depression" and later, simply "depression" (Berrios, 1992). Early clinical observations of symptom patterns, influenced by theoretical background (e.g., medical or biological models and/or psychodynamic models), resulted in the nomenclature of distinct mental disorders.
Risk factors for depression and anxiety (Berrios, 1992; Glas, 1994) and eventually led to the current nosology of affective disorders appearing in the DSM-IV (American Psychiatric Association; APA, 1994).

The history of affective disorders and their many revisions based on differing etiological perspectives suggests a more integrated approach to research aimed at determining causal antecedents might be warranted. The intricate relations that exist among our perceptual and cognitive processes, situational cues, physiological responses and their developmental antecedents need extra consideration in etiological studies. Indeed, Glas’ (1994) overview of the history of depression and anxiety has pointed out the need for combined approaches (e.g., biological, developmental, and psychosocial) to keep our scientific interpretations closer to reality.

This history of mental disease shows that the explication of adult psychopathology without regard to biological, developmental and psychosocial research paradigms is not viable. Developmental psychopathology emerged from the integration of several such disciplines recognizing that biological processes interact with and are influenced by psychosocial variables within a developmental history to produce health or disease (Cicchetti, 1984).

Child-Development and Mental Health

Healthy parent-child relationships have long been accepted as an important determinant for adult adjustment. Psychoanalytic views originating from Freud (esp. Bowlby, 1966, 1977) led to the acceptance of the parent-child relationship as an underlying factor in the development of normal and problematic mental health (Blatt, Wein, Chevron, & Quinlan, 1979; Glas, 1994; Parker, 1992).
Bowlby (1969/1982) borrowed from evolutionary theory to build a model of development and explain individual variation according to genetic and environmental influences. Infant behaviour, which is initially biologically based according to basic needs for survival, becomes organized or regulated according to the infant’s relationship with his/her caregiver (especially in times of stress) thereby creating a bond. The nature of the attachment relationship develops in the first 18 months of life (Bowlby, 1969/1982). It is generally accepted that the quality of attachment organization is dependent on "the responsivity of the caregiver and the degree of reciprocity between the infant and the caregiver" (Carlson & Sroufe, 1995, p. 584).

While early studies of attachment focused on a causal link between parental loss and adult depression, Lloyd’s (1980a, 1980b) review of this literature found that only 20 to 40 percent of depressives had lost a parent before the age of 16. Although significant, other etiological factors had to account for the remaining 60 to 80 percent of depressives. Brown and Harris (1978) found that the loss of a parent alone may not be sufficient in predicting future depression. Furthermore, evidence pointed to a deficiency in the quality of parenting (especially care) as a more direct risk factor for adult depression than parental loss or separation (Brown & Harris, 1978; Parker, 1979b, 1992).

According to Bowlby (1977), a healthy parent-child relationship which fosters secure attachment is defined by parenting which encourages exploration of the environment while enhancing feelings of security. The fostering of independence is an important parental attribute (Bowlby, 1977) in that it provides a pathway to the development of a sense of mastery over the environment, considered essential for human adaptation (Carlson & Sroufe, 1995). The psychological integrity of a child is dependent
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on their own achievements and the support received from significant caregivers (Kopp, 1989).

A securely attached infant is, thus, identified by the infant’s readiness to explore his/her environment and subsequent comfort seeking from the caregiver when distressed. An insecurely attached infant can either engage in environmental exploration or be inhibited from exploration. Regardless, the insecure infant is unsure of the responsiveness of their caregiver, and as such is either ambivalent about their relationship (i.e., does not initiate contact with caregiver or is unresponsive to caregiver’s interaction). classified as “anxious, avoidant” attachment, or not easily comforted by their caregiver when distressed. classified as “anxious, resistant” attachment (Ainsworth, Blehar, Waters. & Wall, 1978; Carlson & Sroufe, 1995).

The healthy parent-child relationship aids in the development of a positive adult self-concept, thereby serving a protective function against the environmental stressors faced in later life. Sroufe, Carlson, and Schulman (1993) have demonstrated this stability of secure and insecure attachment. Infants classified as either securely attached or insecurely attached were followed up to ages 10 and 11. Securely attached individuals were found to have a greater sense of self-efficacy and social competence, while insecurely attached individuals showed signs of deviant behaviour such as isolation and aggression.

Other research has also provided support for the continuity of attachment behaviours with social-emotional functioning (Black & McCartney, 1998; Lieberman, Doyle & Markiewicz, 1999). These findings suggest that the experience of a secure attachment relationship during early childhood fosters a repertoire of healthy attachment
behaviours (evident in adulthood when an individual faces stress, illness, or fear) which persist throughout the lifespan and enable the adult to cope with the surrounding environment. The well-adjusted adult is "able to help himself and . . . [is] worthy of being helped should difficulties arise" (Bowlby, 1977, p. 206).

Bowlby (1969/1982) introduced the concept of "internal working models" based on early expectations of the caregiver and self. The caregiver's responsiveness to the care seeking behaviour of the infant is said to provide the "basis for self-regulation" whereby "confidence in the caregiver . . . becomes confidence in the self" (Carlson & Sroufe, 1995, p. 588). Emotional expression is used as a communicative tool to elicit a response capable of restoring a sense of security (Carlson & Sroufe, 1995).

Conversely, maladaptive patterns of functioning in adulthood are thought to be the result of pathogenic parenting (Bowlby, 1977). Parental attachment when developed in adverse conditions (e.g., when being ignored, or rejected), can result in faulty attachment behaviour as an adult, and subsequently lead to psychopathology. For example, during childhood, experiences of parent rejection are internalized so that the child feels unloved, and the parent is perceived as unloving and unreliable (Dozier, Stovall, & Albus. 1999). The child reconciles by developing alternative insecure coping strategies. Consequently, emotional expression becomes maladaptive or exaggerated (Carlson & Sroufe, 1995). Insecure, anxious individuals, characterized as being "over-dependent or immature," are more vulnerable to the effects of stress, and depression is a probable outcome (Bowlby, 1977). A recent review of the literature has also identified negative self-evaluation, depression, impaired social development, underachievement,
incompetence, and emotional instability as potential psychological sequelae to adverse parenting (Thompson & Kaplan, 1996).

Bowlby (1977) viewed pathogenic parenting as a persistent pattern of parental behaviour consisting of at least one of the following: unresponsiveness to a child's care-seeking behaviour; active rejection of a child; discontinuities of parenting (e.g., through separation); threats made by parents (e.g., not to love a child, abandonment, suicide) as a means of controlling or disciplining a child; or inducing feelings of guilt in a child (e.g., child's behaviour is or will be responsible for parent's illness, death). The proposed pattern observed by Bowlby has been supported by more recent research and has been variably termed adverse parenting, emotional abuse, emotional neglect, and psychological maltreatment. Consistent with Bowlby's view that persistent patterns were necessary for parenting to be pathogenic, Thompson and Kaplan's (1996) review of the literature found that the repeated exposure to adverse parenting, rather than isolated events, was important in determining whether negative sequelae would follow into adulthood.

Bowlby's (1977) attachment theory led to the identification of two specific and necessary attributes, "care" and "protection," required for optimal parenting and the development of a healthy parent-child bond. Parker, Tupling and Brown (1979) defined care and protection on two separate continuums. Affection, emotional warmth, empathy, and closeness are at one end of the care dimension, and emotional coldness, indifference and neglect are at the other. For the protection dimension, control, overprotection, intrusion, excessive contact, and prevention of independent behaviour are at one pole while allowance of independence and autonomy is at the other (Parker, et al., 1979). The
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cancept of good parental care subscribed by Bowlby (1966) included a mutually
satisfying relationship whereby warmth, intimacy, nurturance, and continuity are
exhibited toward an infant and child. Subsequent childhood deprivation studies also
suggested the importance of affection (constituted as nurturance, acceptance and concern)
and led to the demand for objective measurements of the parent-child relationship (e.g.,

Parenting Style and Depression

"Care" and "protection" came to be incorporated as the essential defining qualities
of any psychometrically sound measurement of parenting style (Gerlsma, Emmelkamp, &
Arrindell, 1990). Research escalated rapidly in the area after the development of the
Parental Bonding Instrument (PBI; Parker, et al., 1979). The PBI is a factor-analytically
derived retrospective questionnaire designed to measure dimensions of "care versus
indifference/rejection" and "overprotection/control versus autonomy and independence"
thereby distinguishing optimal parenting from anomalous parenting within Bowlby's
framework. The PBI identifies four possible parenting styles based on categorical
distinctions for the combined Care and Overprotection dimensions: "optimal parenting,"
defined by high care and low overprotection scores; "affectionate constraint,“ defined by
high care and high overprotection scores; "affectionless control,” defined by low care and
high overprotection scores; and "neglectful parenting,” defined by low care and low
protection scores (Parker et al., 1979). Care and Overprotection scores are negatively
correlated. which supports the contention that overprotection is not an indicator of care
(Parker & Gladstone, 1996). Overprotection. defined by the PBI as involving control and
intrusion, may prohibit or replace care (Parker, 1989).
Several studies looking at non-clinical samples provide consistent support linking low maternal care to adult depression and less consistent support linking the combination of low maternal care and high overprotection to adult depression (Lloyd & Miller, 1997; Mackinnon, Henderson & Andrews, 1993; Parker, 1979a; 1979c; 1982; Parker, Hadzi-Pavlovic, Greenwald, & Weissman, 1995; Richman & Flaherty, 1986). Gerlsma et al. (1990) reported a meta-analysis of the literature relating parenting style with depression and/or anxiety. Consistent results identified an "affectionless control" style (i.e., low care, high protection) of parenting with anxiety disorders but the literature held a less consistent trend for depressive disorders. Parker (1992) responded to these findings by stating that "any attenuated trend may well represent the extent to which 'melancholic' depressives have been included in some samples . . ." (p. 177), and he contends anomalous parenting is not related to the melancholic depressive type.

As indicated by the Parker (1992) quote above, depressive types have been differentiated in the literature relating anomalous parenting to depression. Bipolar disorder and endogenous depression or melancholia have been attributed to genetic and biochemical bases whereas neurotic or nonendogenous depression has been attributed to a psychosocial etiology, thus the assignment of anomalous parenting as a risk factor for neurotic depressives follows from this etiological distinction (Parker, Kiloh, & Hayward, 1987). Symptoms, such as age of onset, absence of environmental antecedents, and early morning awakenings, have been reported elsewhere to support the etiological features of endogenous depression or melancholia (see Dobson, 1985, for a review). The PBI classification "affectionless control" (i.e., low care, high protection) and the PBI Care subscale alone have been able to discriminate between neurotic depressives and controls.
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(Plantes, Prusoff, Brennan, & Parker, 1988) and between neurotic depressives and endogenous depressives (Parker et al., 1987). Neurotic depression is considered a less severe type of depression compared to endogenous depression in that it is linked to personality variables which are largely environmental in etiology (Mackinnon & Mitchell, 1994). This etiological distinction between depressive subtypes may, however, be risky if it narrows the scope of risk factors leading to depression.

Inconsistencies between European and North American nosology must also be kept in mind when looking at the more severe depressive types. Major depression is a North American term coinciding with the European term unipolar depression which is categorized as endogenous depression. It is this classification of severe depression that has been the focus of genetic and biological studies (Mackinnon & Mitchell, 1994).

Still others argue that genetic and epigenetic factors (e.g., stressful life events) interact from birth to puberty to increase vulnerability to depression under adverse conditions (Plotsky, Owens, & Nemeroff, 1998). There is a natural occurring plasticity to the development of neuroendocrine responses to stress which can be influenced by maternal behaviour (Liu et al., 1997; Meaney et al., 1996). This view discounts etiological distinctions based on depressive type.

Personality and Attachment Theory

Bowlby’s (1966) early work on attachment theory and findings of the relation between parental care and some forms of mental illness identified the need for additional research in the area of child-development and personality growth before prevention of these mental illnesses can be realized. Personality is considered especially important in
depression research given the many environmental influences associated with its development (Bowlby, 1966).

When a healthy childhood environment exists, there is increased potential for personality development to unfold in the manner prescribed by Bowlby (1966); that is, through “a process whereby we become less and less at the mercy of our immediate environment and of its impact upon us, and more and more able to pursue our own goals, often over long periods of time, and to select and create our own environment” (p. 52). How well adults adjust to their environment is dependent on how the aforementioned process of personality development is fostered. For example, personality development is noted to become disturbed during childhood when deprivation occurs (Bowlby, 1966). According to Bowlby, a child can experience deprivation either by living with a primary caregiver (e.g., mother or permanent mother-substitute) who is unresponsive toward the child’s needs (i.e., “pathogenic” parenting) or by being separated from their primary caregiver through death, illness, or desertion. Under deprived conditions, the process of assimilation into the surrounding social culture is disrupted and subsequently impedes personality development.

Research into personality variables such as self-esteem (Lloyd & Miller, 1997; Oliver, & Paull, 1995), self-efficacy (Oliver & Paull, 1995), autonomy-dependency and locus of control (Richman & Flaherty, 1986) has found these variables to be significantly correlated with parenting style. A study of adult coping resources using a medical student population supported a link between early parent-child relationships and depression and found that personality was a mediator between this relation (Richman & Flaherty, 1986). Lloyd and Miller (1997) found self-esteem to have a mediating effect on the parenting
style-depression relation for men but not for women. To test for mediation in their study, the partial correlation between parenting style and depression measures were calculated holding self-esteem constant. Mediation was considered established if parenting style was no longer significant.

Parker (1993) also tested the possibility of personality variables (e.g., low self-esteem and a dysfunctional cognitive style) mediating an anomalous parenting-depression relation, suggesting that anomalous parenting creates a personality vulnerability or diathesis to depression. He hypothesized that anomalous parenting might dispose an individual to a dysfunctional cognitive style, which would then make one vulnerable to depression. Using depressed patients and community depressives for his sample, he failed to find a significant correlation between depression scores and PBI scale scores, and therefore, could not test for mediation. Parker (1993) did, however, find significant correlations between PBI scores and both low self-esteem and dysfunctional cognitive style.

Self-efficacy, a major determinant of action, refers to “beliefs in one’s capabilities to organize and execute the courses of action required to produce given attainments” (Bandura, 1997, p. 3). Bandura (1997) proposes four processes at work which influence human functioning: cognitive, motivational, affective, and selective processes. Individuals who have confidence in their ability to act in response to environmental demands take more control over their psychosocial functioning. A positive perception of one’s capabilities fosters forethought and enables realistic goal setting behaviour. These ideas are consonant with the intricacies of attachment theory. The goals and ideas generated from interacting with the immediate environment are dependent on infancy and
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early childhood experiences whereby the mental processes of planning and thinking ahead were developed (Bowlby, 1966).

Considering the importance of personality development in the theoretical framework attributed to Bowlby, the self-efficacy construct appears to have a close link to attachment and mental health outcomes. In infancy, self-soothing (e.g., thumb-sucking or body rubbing) to alleviate mild distress is one of the first early experiences of efficacy or mastery (Kopp, 1989). As infants become more adept at exploring their environment, they discover new ways to distract themselves from boredom or mild distress (playing with toys). Furthermore, they become better able to signal to their caregivers (tugging on clothing or crawling to caregiver) when specific assistance or comfort is needed (Kopp, 1989).

Limited or unvaried responses to distressed infants by the caregiver can have detrimental ramifications to the behavioural repertoire of children (e.g., inability to develop their own calming strategies or modulate distress arousal) (Kopp, 1989). Early experiences with limited control over events create a psychological vulnerability to emotional disorder (see review by Chorpita & Barlow, 1998). The self is likely to be viewed as unworthy and incompetent (Carlson & Sroufe, 1995). Without perceived control to determine events, behaviour is inhibited and anxiety arousal ensues. In this sense, anxiety is in turn a risk factor for depression. The propensity to view events as uncontrollable and to presuppose negative outcomes evokes feelings of helplessness (anxiety) which eventually lead to hopelessness (depression) (Chorpita & Barlow, 1998). Anxiety and depression are both considered states consisting of cognitive, emotional, behavioural, and physiological components (Dobson, 1985). Cognitive models of anxiety
and depression distinguish the two constructs by relating to the time of threat. If the threat is perceived as something happening in the future, anxiety symptoms are likely. If a threat is certain to occur or has already occurred, however, depressive symptoms are likely (Dobson, 1985).

The relation between poor self-efficacy and extant depressive symptomatology has been established in the literature (Maciejewski, Prigerson & Mazure, 2000; Olioff, Bryson, & Wadden, 1989). For example, in a three-year longitudinal study by Maciejewski et al. (2000), high levels of self-efficacy were able to predict less depressive symptomatology at followup, and serious depressive symptomatology was able to predict lower levels of self-efficacy. Furthermore, self-efficacy was negatively affected by life-events, but only for those with a history of depression. According to Bandura (1997) “the perceived inability to fulfill stringent standards of self-worth and to secure things that bring satisfaction to one’s life creates depression” (p. 277). In contrast, high levels of self-efficacy serve to protect against the onset of depressive symptomatology (Maciejewski et al., 2000). Early research on parental loss and depression also showed that a lack of mastery and perceived inability to control one’s environment were associated with adult psychopathology (Brown & Harris, 1978). Furthermore, a lack of mastery and/or control have been found to provoke anxious arousal (Chorpita & Barlow, 1998). Self-efficacy and perceived control are both determining factors of action. The perceived ability to have an influence on one’s environment to produce desired outcomes creates a drive to take action. From a developmental perspective, early control experiences may foster self-efficacy for later experiences which require action.
Risk factors for depression and anxiety

Stress as a Risk Factor for Depression

An exploratory causal modeling study conducted by Kendler et al. (1993) found that “parental warmth” (measured by removing all the overprotection dimension items from the PBI) had an indirect effect as a predictor of depression while recent stressful life events were the most predictive risk factor for depression. Parental warmth’s effect on depression was mediated by lower levels of recent life difficulties (synonymous with daily hassles), fewer previous depressive episodes, lower levels of neuroticism, higher levels of social support, and fewer instances of lifetime traumas. In other studies which only looked at more serious life events as a provoking agent for the onset of depression, the assignment of unaccounted variance to the occurrence of more minor events or daily hassles has also been conjectured (Brown & Harris, 1978). The importance of examining acute stressors and chronic stressors when studying depression has been pointed out by others (Billings, Cronkite, & Moos, 1983; McLean, 1975; Musil, Haug, & Warner, 1998). In fact, Musil et al. (1998) note that the effects of one stressful life event (e.g., loss of spouse) may be further complicated through an increase in daily hassles related to the event (e.g., increased self-reliance).

There are multiple factors involved in the onset of any stress-related illness (Mikhail, 1985) making etiological studies difficult. The perception of a life event as stressful is dependent on situational and individual variables. For example, the psychological or cognitive processes involved when stress is encountered mediate its impact. Hormonal and cardiovascular functioning (as well as other bodily systems) are altered in response to stress. Behaviour is dependent on these mechanisms. Stress-related
diseases are conceptualized as functional disorders whereby the homeostasis of an organism is threatened.

Mikhail (1985) suggested that the definition of stress include both psychological and physiological components: “Stress is a state which arises from an actual or perceived demand-capability imbalance in the organism’s vital adjustment actions and which is partially manifested by a nonspecific response” (p. 37). Research on stress responses in animal models have found that psychological factors are able to modify physiological dysregulation resulting from stress (Chorpita & Barlow, 1998). It is agreed that the “demand-capability imbalance” must involve a cognitive appraisal of one’s ability or inability to cope with stress. Perceived coping self-efficacy has been identified as a critical cognitive mediator between life events and biological stress reactions (Bandura, 1988, 1997). Additionally, the internalized cognitive structures which shape our appraisals of perceived support, perceived control, and self-efficacy beliefs are noted to impact coping behaviours (Ptacek, 1996). The development of such an appraisal system can be explained in the context of attachment theory.

Kopp (1989) states that stress experienced in infancyh “serves as a catalyst to move the immature human to adaptive emotion regulation in the service of physiological and psychological well-being” (p. 343). Negative experiences can enable an infant to learn appropriate coping responses to environmental stresses. A secure parent-child relationship offers more opportunity to experience the environment and to develop a broader repertoire of coping behaviours (Ptacek, 1996). Information is obtained from new experiences and assimilated into existing cognitive structures. For example, a sense of agency is developed during the toddler period whereby toddlers have an awareness of the
cause of their distress and are able to alleviate or worsen their emotional state. The
toddlers’ choice in behaviour is based on their recall of similar experiences (Kopp, 1989).
Early experience is thus viewed as “disproportionately important in that it weights or
colors subsequent experience” (Chorpita & Barlow, 1998, p. 5). Cognitive structures and
processes developed from early parent-child relationships have been argued to be
relatively stable across time (Bowlby, 1988; Ptacek, 1996).

Social Support

Literature shows high levels of social support to be important in reducing
vulnerability to depression. Bandura (1997) has found evidence that social networks act
as a determinant of coping resources. Moreover, social dysfunction has been shown to be
a significant predictor of subsequent depression at follow-up in clinical samples of
depressed and anxious individuals (Hecht, et al., 1989). Social dysfunction was also
found to be more debilitating (described as coping deficits) for those with a comorbid
diagnosis of severe depression and severe anxiety, and the presence of social dysfunction
for those with an anxiety disorder increased their risk for future depression.

While life events present risk factors for developing depressive symptomatology,
social support can play a protective role to prevent the occurrence of such
symptomatology, especially if a trusting, intimate relationship exists within a social
network (Brown & Harris, 1978; Parker & Gladstone, 1996). When stress is encountered,
and social support is low, anxiety and depressive symptomatology are likely. Musil,
Haug, and Warner (1998) found marriage to act as a protector against depression in older
adults. Organizational research also gives support to the mediating effect of social
support between stress and depression (Bandura, 1997).
Bowlby (1966) also points out the basic need for positive human relations, and that "the outstanding disability of persons suffering from mental illness . . . is their inability to make and sustain confident, friendly, and co-operative relations with others" (p. 91). Brown and Harris' (1978) research supports this contention. They found that women who suffered the loss of their mother through either separation or death during early childhood (before the age of 11) and who could not sustain healthy personal relationships in adulthood were especially prone to depression. A poor quality of family life before loss or separation and inadequate care received afterward added to the propensity of depression in adulthood for these women (Brown & Harris, 1978). It has been speculated that a poor parent-child relationship may act to prevent individuals from making and keeping satisfactory social and intimate bonds in adulthood (Parker & Gladstone, 1996; Ptacek, 1996).

Perceived social support and social networks differ in that perceived social support refers to the extent to which individuals believe their needs for support, information, and feedback can be met, whereas social networks refer to actual social connections within the environment (Procidano & Heller, 1983). Perceived social support is also related to personality traits and changes in mood. Support perceptions are affected by early attachment relationships in that the resulting internalized cognitive structure or working model shapes one's beliefs of whether others can be trusted in stressful situations, as such it is part of an individual's appraisal of stress (Procidano & Heller, 1983). Perceived social support can, therefore, be considered "a characteristic of the individual" (Ptacek, 1996, p. 505) with a developmental history and temporal stability. Furthermore, evidence indicates that perceived support, rather than received support, has
greater predictive ability of adjustment to stressful life events (Fleming, Baum, Gisriel, & Gatchel, 1985; Wethington & Kessler, 1986). Research has also shown that depressives perceive they receive less support (Coyne, Aldwin, & Lazarus, 1981).

Adolescents and young adults identified as securely attached have a tendency for strong support perceptions, which result in an increased confidence to cope in times of stress (Carlson & Sroufe, 1995; Ptacek, 1996). Conversely, individuals identified as having experienced distorted parenting tend to have maladaptive defensive processes which impede the development of healthy social relationships (Carlson & Sroufe, 1995).

The results of one study on the mediating effects of perceived social support between stress and mental health provided partial confirmation for a “stress buffering” hypothesis (Fleming et al., 1985). Residents of Three Mile Island reporting moderate to high levels of social support exhibited less emotional distress following the environmental disaster on measures of anxiety and depression than those reporting low levels of social support. When stress was encountered, perceived social support improved the ability to cope, and there was less symptom reporting. In the absence of stress, however, symptom reporting among individuals with high and low perceived social support did not differ. Analysis of urinary catecholamines, however, did not support a stress buffering effect. In other words, perceived social support did not ameliorate that index of physical responses to stress (Fleming et al., 1985). Perhaps we still react physically to stress, but our internal resources (e.g., perceived social support) prevent these physical reactions from progressing into negative psychological sequelae. There are, obviously, factors other than perceived social support at play in determining individual differences in physiological responses to stress.
Physiological Stress Responses Integrated into a Developmental Perspective

Increased hypothalamic-pituitary-adrenal (HPA) activity occurs as a neuroendocrine response to stress (Ansseau, 1997; Kirschbaum & Hellhammer, 1994). Corticotrophin-releasing hormone (CRH) is secreted, followed by a release of adrenocorticotrophic hormone (ACTH) which in turn is followed by an increase in circulating levels of cortisol, a glucocorticoid hormone, released by the adrenal cortex (Ansseau, 1997; Kirschbaum & Hellhammer, 1994). The HPA system in which adrenal glucocorticoids are the end result is essential to the survival of mammalian organisms under stressful conditions (Meaney et al., 1996). After a threat to an organism is no longer present, an efficient HPA axis, through a glucocorticoid negative feedback system, is able to be turned off. An exaggerated HPA response (i.e., when glucocorticoid feedback regulation does not work well) can be threatening to the homeostasis of the organism, both physically and psychologically.

There are two co-occurring psychological variables at play when considering the stress response of the adrenal cortex, the anticipation of the event and the perception of the event as threatening (Kirschbaum & Hellhammer, 1989). Anticipation of an event increases HPA activity if the impending event is perceived as threatening. Conversely, the perception of an event as nonthreatening may preclude any increase in adrenal activity due to an anticipation factor. Anticipation of threat or harm as an antecedent to stress reactions has been well supported and is consonant to the concept of a cognitive appraisal in the coping literature.

Animal studies (using rat pups) have found early environmental influences on the development of the HPA response that can account for individual differences in
physiological responses to stress (Liu et al., 1997; Meaney et al., 1996). Higher basal levels of glucocorticoid hormones have been found in adult organisms which experience chronic stress (Chorpita & Barlow, 1998; Meaney et al., 1996). Further, animal models looking at control over environmental events and physiological responses have shown that both control over aversive stimuli and control over desirable stimuli affect glucocorticoid secretion (Chorpita & Barlow, 1998).

The inefficiency of inhibitory signals associated with the glucocorticoid negative feedback is thought to predispose individuals to pathology (Meaney et al., 1996). For example, HPA activity is found to be etiologically linked to psychopathology (Meaney et al., 1996), including anxiety and depression (Friedman et al., 1992). The elevated cortisol levels associated with anxiety and depression have been linked in recent research to the interplay of early childhood experiences, parental responsiveness, and temperament (Chorpita & Barlow, 1998).

Individual differences in physiological reactivity have been related to the quality of parental attachment in research involving infants and toddlers (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). Specifically, research has found that inhibited (behaviourally restrained), insecurely attached children had elevated levels of cortisol (Nachmias et al., 1996). Toddlers were classified according to attachment security using procedures consistent with Ainsworth's Strange Situation (Ainsworth, Blehar, Waters, & Wall, 1978), coping behaviour (defined as their ability to manage novel events), and behavioural inhibition (defined as their tendency to approach novel stimuli). Consistent with previous findings (Carlson & Sroufe, 1998), temperament (indexed by behavioural inhibition) and attachment security were independent constructs (Nachmias et al., 1996).
Elevated cortisol levels were found in toddlers classified as both inhibited and insecurely attached. The interaction of the two constructs (inhibition and attachment security) was significant in predicting elevated cortisol, but there were no main effects. Toddlers classified as inhibited did not differ from toddlers classified as uninhibited in their HPA response if they were securely attached. Nachmias et al. (1996) noted that the mothers of inhibited, insecurely attached toddlers appeared to interfere with infant coping efforts. Kagan, Reznick, and Snidman (1987) have also found that extremely behaviourally inhibited children tend to have elevated cortisol levels.

Chorpita and Barlow (1998) have hypothesized the following developmental account for these findings. When a parent reacts to their child’s communicating needs (even rudimentary communication), the child experiences control. During infancy, for example, behaviours (e.g., crying) communicate needs (physical or affectional). When these needs are met by the parent/caregiver the infant has experienced control. This is consistent with Kopp’s (1989) view that an individual’s first sense of mastery is experienced in infancy. Over time, with a secure base established, exploration of the environment creates a wide repertoire of behaviours which involve further control experiences. Without a secure base whereby the toddler achieves exploration and control of the environment, the child does not receive reinforcers, limited exploration takes place, behaviours are inhibited, and hence the opportunity for control experiences are few. These control experiences, which are related to attachment, are carried through into adulthood (Chorpita & Barlow, 1998) and may have an effect on HPA functioning. Burger (1992) has also found a statistical relation between early childhood control
experiences (parents encouraging independence) and a high desire for control in adulthood.

Research on early environment and cortisol reactivity has also been conducted with adolescent and adult populations. For example, maternal-attachment, social stress, and cortisol reactivity have been studied in relation to trait-anger in adolescents (Wright, Cameron, & Susman, 2000). Females classified with low maternal attachment and high trait-anger experienced decreased cortisol response to a laboratory stressor. Early stressors have also been studied in relation to subsequent depression in adulthood with particular emphasis on the long-term effects of stress on HPA functioning. HPA dysregulation has been found in individuals who have experienced early stressors (Weiss, Longhurst, & Mazure, 1999).

Coping Style and Depression

Coping can be defined as the efforts made by an individual when he/she approaches or confronts the demands of a stressful situation in order to reduce stress (Endler, 1997; Folkman & Lazarus, 1986). Whether an individual is successful in reducing stress is irrelevant to the definition of coping (Folkman, 1984). Theories of coping suggest that person variables interact with situation variables to evoke a response (Endler, 1997; Folkman, 1984; Parkes, 1986). The interaction hypothesis, although not widely tested, has been supported (Parkes, 1986).

A number of styles of coping have been identified, but they are invariably subsumed under problem-focused or emotion-focused coping (Folkman & Lazarus). A problem-focused or task-oriented style of coping occurs when efforts are made by the individual to solve a problem or change a stressful situation (Endler, 1997; Folkman &
Lazarus, 1986). Emotion-oriented coping involves self-oriented emotional reactions (Endler, 1997). An emotion-focused style can refer to an emotional discharge, distancing, avoiding, or support seeking in response to a stressful situation (Folkman & Lazarus, 1986) which all have the potential to increase rather than reduce stress (Endler, 1997).

The coping resources (internal and external) individuals employ affect psychological outcomes. Research has shown that depressed persons and non-depressed persons differ in the way they cope with stress (McLean, 1978). Depressed persons have been identified as more psychologically vulnerable to stressful encounters, finding it more difficult to regulate their emotions (Folkman & Lazarus, 1986). Research on coping in relation to depression has also found that depressives tend to need more information before acting, tend to seek more emotional support, engage in wishful thinking (Coyne, Aldwin, & Lazarus, 1981), and engage in more emotional discharge (Billings & Moos, 1984). Coyne et al. (1981) have also shown that depressives do not differ from a control group in the amount of problem-focused coping employed, though Billings and Moos (1984) have found that depressed persons made fewer problem-focused efforts to reduce stress than nondepressed persons.

Present Study

It has been argued above that there should be multiple variables involved in any etiological study. Research supports the need for etiological studies of depression to include environmental factors, such as stressful life events, and early-childhood experiences (Kendler et al., 1993). Research relating adult psychopathology and early environment, especially pathogenic parenting, has been especially constrained by simplified causal models. There has been a large number of studies linking pathogenic
parenting styles with adult depression (see Gerlsma, Emmelkamp, & Arrindell, 1990 for a review). Yet, there is a dearth of research on parenting style and depression incorporating personality variables, life stress, and physiological measurements. Furthermore, depression research is especially weak in bringing developmental processes to the fore in generating a probable causal chain among constructs.

Bowlby's work has provided a developmental framework in which to study individual adaptation and psychopathology. Conceptually relevant to attachment theory are the associations among parenting style, self-efficacy, perceived social support, life events, physiological stress responses and coping processes and their ability to predict anxiety and depressive symptomatology. There is a relationship between anomalous parenting and depression, though the nature of the relationship is not well understood. Certainly, not all individuals who experience anomalous parenting suffer depressive symptoms. The process by which anomalous parenting makes one vulnerable to depression is still questionable. We also know that behaviour in response to stress is multiply determined by relatively stable personality factors and cognitive processes which have a developmental history. Developmental antecedents and how they relate to depression and anxiety need to be explored.

The purpose of the present set of studies was to examine the effects of presumed stable personality variables (e.g., self-efficacy and perceived social support) on anxiety and depressive symptoms and to further examine the extent to which these personality variables determine coping styles employed to deal with stressors. The function of parenting style in determining personality variables, coping style used to deal with stressors, and mental health sequelae was explored. The effect of early environment (i.e.,
parenting style) on physiological stress responses was also explored. Finally, the role of physiological stress responses as an index of temperamental differences among participants was examined.

Part of the present set of studies builds upon another study (Hand, 2001) on psychosocial stress involving an experimental stressor and physiological measurements. Data collected in conjunction with that study adds an experimental component to the present correlational study. As such, there are two parts to the present research which will be referred to as Study 1 and Study 2. Study 1 includes data collected in collaboration with Denise Hand’s Master’s thesis on psychosocial stress (Hand, 2001) and will be referred to as Time 1 in Study 1. Time 1 data was collected between February 2000 and July 2000. Participants were asked to return for continuation in the present study to collect Time 2 data for Study 1 and for participation in Study 2.

STUDY 1
A Prospective Study Of Parenting, Personality, Physiological Reactivity, and Mental Health

For Study 1, the following hypotheses are relevant. 1) Low self-efficacy at Time 1 will be predictive of increases of depressive symptomatology at Time 2 in the event that stressors (negative life events and/or daily hassles) are encountered during the period between Time 1 and Time 2 (i.e., stress moderates the longitudinal relation between self-efficacy and depression). Chorpita and Barlow’s (1998) review of the developmental process of control perception and its relation with negative events to effect subsequent anxiety and depression has supported a moderating effect of cognitions (i.e., attributional style) for adult populations and a mediating effect of cognitions for child populations. As
cognitive frameworks become stable, events are moderated by interpretations or perceptions. This research is consistent with other cognitive models of depression (e.g., Abramson, Metalsky & Alloy, 1989). With the view that self-efficacy is a stable personality variable that is intricately intertwined with cognitions, a moderational effect with stress should hold true in its relation with adult depression. 2) The relation between physiological reactivity (measured at Time 1) and mental health (anxiety and/or depression) at Time 2 will be moderated by stressors. Individuals who have higher levels of physiological reactivity in response to a laboratory stressor will show higher scores on measures of anxiety and depression in the event of natural occurring stressors. 3) Statistically controlling for anxiety at Time 1, physiological reactivity will account for some of the variance in Time 2 anxiety scores over and above coping style. 4) Individuals who reported an anomalous parenting style ("affectionless control") will differ from those who reported any of the other three parenting styles in their physiological reactivity. Specifically, higher levels of physiological reactivity are expected for individuals reporting anomalous parenting. To explore the generalizability of Chorpita and Barlow’s (1998) review of animal findings to humans, the overprotection dimension of parenting will also be examined to see if behavioural inhibition alone can account for these differences in physiological reactivity.

Primarily, animals have been used to study the effects of early environment on stress responses, particularly the HPA response. It is recognized that using this body of research as the rationale for this hypothesis is a leap due the differences in complexity of the early environment of humans versus the rat pup in a controlled environment. However, in human research, the parent-child relationship (attachment quality) and
behavioural inhibition (temperament) have been related to individual differences in physiological reactivity (as measured by changes in cortisol) for children (Nachmias et al., 1996).

**Method**

**Participants**

Participants were recruited from Acadia University, Wolfville, Nova Scotia and consisted of psychology students and general population students as part of the psychosocial stress study (Hand, 2001). Prior to participation in the study, students were screened for factors known to influence normal cortisol secretion (see Hand, 2001). Students were excluded from the study if any of the following pertained to them: smoke more than five cigarettes per day; abnormal day/night sleep cycles; pregnant; on anti-depressant medication; or diagnosed with major depression, anorexia nervosa, endocrine disease or alcoholism.

Students eligible to participate in the study were asked to abstain from caffeine, large meals, smoking, alcohol, and strenuous exercise for one hour prior to their test session. Students read and signed an informed consent form prior to participation in the study (see Appendix A and B). During Hand's (2001) study, participants completed questionnaires for the present study which represent Time 1 measurements. Due to the sensitive nature of the questionnaires (i.e., measures of depression and anxiety), the contact number for the counselling centre on campus was included as part of the debriefing form (see Appendix C). Additionally, the depression questionnaires were screened, and at-risk participants were contacted to provide resource numbers directly. Participants from Time 1 (N = 79) who completed the battery of questionnaires and an
experimental stressor, and gave consent to be re-contacted for the present study (n = 70), were recruited by email for continuation in the present study.

Students who returned for continuation in the study had a chance to win a cash draw of $300 for their participation. Three participants were in psychology classes from which participants were being recruited for Study 2. These participants were given the option to received two course points or to enter the draw for $300. All three participants chose to receive course credit. The return rate for Study 1 was 23% yielding a sample of 18 participants [17 F / 1 M, age mean and (standard deviation) = 21.33 (2.97) years]. Analyses in which cortisol was being explored further reduced the sample size (n = 15) due to missing data.

**Experimental Stressor and Physiological Reactivity - Time 1**

Participants at Time 1 underwent the experimental stressor, the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) used in a previous Master’s thesis (Hand, 2001). The TSST has an anticipation period and a testing period where participants undergo a 10-minute public speaking and mental arithmetic task in front of a panel of two judges. A baseline measure of salivary cortisol was obtained before notification of the TSST. Salivary cortisol sampling is noninvasive and provides an easy assessment of cortisol levels in response to a psychological stressor (Kirschbaum & Hellhammer, 1994). Cortisol samples were also collected from participants at 10 and 20 minutes after completion of the TSST. For brevity, the three cortisol measurements will be referred to as C1 (baseline), C2 (10 minutes post TSST), and C3 (20 minutes post TSST). The TSST has been shown to significantly increase salivary cortisol (10 minutes after stress) from baseline (Kirschbaum, Pirke, & Hellhammer, 1993). There were
significant increases in cortisol (C2 and C3) from baseline measures (C1) for Hand's (2001) study participants. Although oral contraceptive users were to be excluded from analyses involving physiological reactivity due to its known effects on cortisol levels in response to psychological stress (Kirschbaum, Pirke, & Hellhammer, 1995), these differences were not found for the present sample (see Hand, 2001). Cortisol change scores (i.e., C2 - C1) were used in analyses where physiological reactivity was a factor. The change scores are positive, meaning higher numbers reflect a greater increase in cortisol. “Physiological reactivity” or “cortisol reactivity” will be used interchangeably to refer to the cortisol change scores.

**Time 1 Procedure**

Of relevance to the present study, Time 1 data collected in questionnaire format during Hand's (2001) study included depressive symptomatology, state and trait anxiety, and self-efficacy and will be described in detail in the Measurements section that follows. Additionally, participants who underwent the experimental stressor (the TSST) were videotaped, and their apparent stress was subsequently rated by a naive viewer of the videotapes (see Appendix D). A stressfulness rating of the experimental stressor was also obtained from the participants (see Appendix E). These two stress ratings (subject and observer rated) were measured according to a 14-point scale ranging from not stressful (1) to very stressful (14). Demographic information included participants' age, gender, degree program, and year of study. Further details of Time 1 procedure can be found in Hand (2001).
Time 2 Procedure

The time period between Time 1 and Time 2 data collection ranged between seven months and twelve months. Initially, participants recruited by e-mail were asked to sign up at specified time slots for continuation in the study. The response rate, however, was very poor (two participants), so a second e-mail was sent asking participants who would like to continue in the study to pick up their questionnaire packages from the Psychology secretary to complete and return at their convenience. The Psychology secretary was given the master list of names of Time 1 study participants with their numerical code. Questionnaire packages were assigned the numerical code corresponding to name at the time returning participants picked up their questionnaires. Two informed consent/debriefing forms were included and participants were asked to sign one and retain the other for their records (see Appendix F). Written instructions were provided on how to proceed and complete each of the questionnaires according to the guidelines of the instruments. The researcher's contact information was included to answer any questions. It was emphasized that the participants should complete the questionnaires honestly to ensure the validity and reliability of the study.

Measurements

Depression

The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) was completed at Time 1 and readministered at Time 2. The BDI-II is a 21-item self-report measure often used to detect depressive symptomatology in clinical and non-clinical populations. The BDI-II has satisfactory reliability and validity. The questionnaire was updated to better reflect DSM-IV criteria for major depression. Analyses of internal
consistency for the BDI-II revealed a coefficient alpha of .91 (Beck, Steer, Ball, & Ranieri, 1996).

Anxiety

The State-Trait Anxiety Inventory Form Y (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs. 1983). State and Trait subscales, was completed by participants at Time 1 and the Trait measure of the STAI was readministered at Time 2. Respondents described the extent to which they were currently experiencing anxiety symptoms (State anxiety - STAI-S) and the extent to which they generally experienced anxiety symptoms in response to stressful situations (Trait anxiety - STAI-T). Alpha reliabilities for the STAI range from .89 to .91 (Spielberger et al., 1983).

Parenting Style

The Parental Bonding Instrument (PBI; Parker, Tupling, & Brown, 1979) was completed at Time 2. It is a 25 item factorially derived retrospective questionnaire designed to measure two dimensions of parenting: care and protection. Twin studies have shown the PBI to be a valid measure of both actual and perceived parenting style (Mackinnon, Henderson, & Andrews, 1991; Parker, 1986). Another study supporting the validity of the PBI as a measure of actual parental characteristics used a sample of university students and their mothers (Parker, 1981). Significant correlations were found between the students’ ratings and the mothers’ ratings of themselves on the PBI’s Care and Overprotection scales (Parker, 1981). Parker (1981) also found that maternal care and overprotection correlated significantly with depression scores regardless of the rater (i.e., mother or student).
Participants are asked to rate their parents' behaviour based on their first 16 years. Maximum scores on the Care and Overprotection scales are 36 and 39, respectively. Total scores on both scales are in the positive direction. That is, a high score on Overprotection is indicative of a controlling parenting style, and a high score on the Care scale is indicative of a caring, nurturing parenting style.

The two PBI scales can be used together to compare types of parental bonding. As such, cutoff scores (based on the mean scores of four normative samples) for mothers and fathers were established by Parker (1979a). The suggested cutoff scores for mothers are a Care score of 27 and an Overprotection score of 13.5, and for fathers a Care score of 24 and a Overprotection score of 12.5. The four possible quadrants allow for the creation of categorical groups for comparison. For the present study, each participant completed only one PBI to assess overall parenting received from both mother and father. As such, cutoff scores were obtained by taking an average of the mother's and father's cutoff scores suggested by Parker, that is, 25.5 for the Care scale and 13 for the Overprotection scale. Research comparing mean maternal and paternal scores on the two subscales has found that differences are not significant (Parker, 1979c). The two scales also allow for quantitative analysis based on the two continuous dimensions. Parker et al. (1979) suggest, however, a correction (i.e., partialling out any contribution of one scale to the other) to create orthogonal dimensions for such regression based analysis.

**Self-Efficacy**

The General Self-Efficacy scale (GSE; Sherer et al., 1982) was completed at Time 1 and at Time 2. The Time 2 measurement of self-efficacy was only used for analysis which incorporated the augmented sample representing Study 2. Participant ratings on the
Risk factors for depression and anxiety

GSE measure are dependent on past experience. Three areas of foci are included in the 23-item scale: initiating a behaviour, sustaining effort in completing a behaviour, and persisting in aversive conditions. Varimax rotation has revealed two factors for the GSE, one unrelated to any specific behavioural domain and one related to social domains (Sherer et al., 1982). Cronbach alpha reliability coefficients for the two factors or subscales were .86 and .71 respectively. Validity studies support the GSE as a global measure of self-efficacy (Sherer et al., 1982). As the present study was not interested in specific self-efficacy, the less reliable and less relevant social domain items were omitted for Study 1 and Study 2 thereby providing a 17-item measure of "general" self-efficacy. Total scores on the GSE are such that high scores are indicative of high self-efficacy.

**Coping Style**

The Coping Inventory for Stressful Situations (CISS; Endler & Parker, 1990) is a 48-item factorially derived self-report measure of coping. There are three distinguishable styles of coping identified by the CISS: Task-Oriented, Emotion-Oriented, and Avoidance-Oriented. Avoidance-Oriented coping is further broken down into Distraction and Social Diversion subscales. Each coping style measured by the CISS includes a cognitive component. Respondents rate items on a 5-point frequency scale ranging from 1 (not at all) to 5 (very much). The CISS was completed at Time 2. Because analyses involving the CISS subscales are exploratory, all three main scales will be used for analyses involving coping style.

Norms have been collected on several groups, including university samples (Endler & Parker, 1990). Alpha coefficients for this population ranged from .81 to .90 on the three main CISS scales. Moderate to high test-retest reliabilities, ranging from .55 to .73,
have been obtained. Construct validity studies support the CISS as a measure of the three coping styles outlined above (Endler & Parker, 1990).

**Life Stressors**

The Life-Experiences Survey (LES; Sarason, Johnson, & Siegel, 1978) was completed at Time 2. The LES is a 60-item self-report measure of potentially stressful life events that may have occurred in the past year. Forty-seven of the items pertain to any general population and ten of the items are directly related to a student population’s academic experiences. Additionally, there are three blank spaces for any other stressful experiences not covered in the rest of the scale. Participant ratings for any events added were included for scoring. Respondents were to indicate those events experienced over the past year and to rate the perceived impact of the event on a 7-point scale, with -3 being extremely negative and +3 being extremely positive. Negative impact ratings are summed to derive a negative change score. Two test-retest reliability studies of the LES resulted in reliability coefficients of .56 and .88 for the negative change score (Sarason, Johnson, & Siegel, 1978).

The original Daily Hassles Scale (DHS; Kanner, Coyne, Schaefer & Lazarus, 1981) was also completed at Time 2. The DHS was shortened by removing 17 items that were unlikely to apply to a Canadian university student population. Additionally, four items were reworded to more closely pertain to a university student population (see Appendix G). The revised 96-item DHS was used to assess the occurrence and severity of minor stressful events. Respondents were to indicate hassles experienced over the past month and rate the severity of the hassle on a 3-point scale, with 1 being somewhat
severe and 3 being extremely severe. Cumulated severity scores were derived by summing the severity ratings.

Results

Data were screened using SPSS data analysis programs. One participant was identified as a univariate outlier based on an extreme z scores (i.e., > 3.29) on the BDI-II (Tabachnick & Fidell, 1996). The extreme score was replaced by one unit greater than the next highest unit on that measure, as recommended by Tabachnick and Fidell (1996). Due to missing cortisol data, analyses involving physiological reactivity reduced the number of participants to 15. Missing cortisol data was due to either experimenter error during collection of saliva samples or breakage during shipping. Means, standard deviations and coefficient alpha reliabilities for all measures (post screening) are presented in Table 1. All measures met good to excellent levels of reliability for the sample with alpha coefficients ranging from .81 to .92. An alpha level of .05 was used for all statistical analysis. Unless otherwise noted, all tests were two-tailed.

Table 2 presents the correlations between physiological reactivity (C2 – C1), mental health measures (depression and anxiety), stressors (life events and daily hassles), personality (self-efficacy), coping styles (task, emotion, and avoidance), and parenting style (care and overprotection) for Study 1 variables. Consistent with depression and anxiety literature (Dobson, 1985), BDI-II and STAI-T scores were significantly correlated at both Time 1 (r = .82) and Time 2 (r = .72). BDI-II and STAI-T scores at Time 1 and Time 2 were also significantly correlated with CISS-Emotion scores.
Table 1. Means, standard deviations, and Cronbach alpha reliabilities for Study 1 measures (N = 18).

<table>
<thead>
<tr>
<th>Measure</th>
<th>M</th>
<th>SD</th>
<th>Cronbach α</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortisol reactivity&lt;sup&gt;a&lt;/sup&gt; (C2 - C1 µg/dL)</td>
<td>.04</td>
<td>.14</td>
<td>-</td>
</tr>
<tr>
<td>Time 1 - BDI-II</td>
<td>7.17</td>
<td>5.09</td>
<td>.88</td>
</tr>
<tr>
<td>- STAI-S</td>
<td>38.50</td>
<td>8.42</td>
<td>.89</td>
</tr>
<tr>
<td>- STAI-T</td>
<td>37.78</td>
<td>10.00</td>
<td>.92</td>
</tr>
<tr>
<td>- GSE</td>
<td>172.50</td>
<td>22.16</td>
<td>.81</td>
</tr>
<tr>
<td>LES</td>
<td>7.94</td>
<td>8.70</td>
<td>-</td>
</tr>
<tr>
<td>DHS</td>
<td>42.00</td>
<td>27.59</td>
<td>-</td>
</tr>
<tr>
<td>PBI-Care</td>
<td>28.64</td>
<td>6.97</td>
<td>.90</td>
</tr>
<tr>
<td>PBI-Overprotection</td>
<td>13.22</td>
<td>7.17</td>
<td>.85</td>
</tr>
<tr>
<td>Time 2 - BDI-II</td>
<td>8.72</td>
<td>5.83</td>
<td>.92</td>
</tr>
<tr>
<td>- STAI-T</td>
<td>38.39</td>
<td>8.94</td>
<td>.88</td>
</tr>
<tr>
<td>- GSE</td>
<td>172.33</td>
<td>29.71</td>
<td>.90</td>
</tr>
<tr>
<td>CISS – Task</td>
<td>48.11</td>
<td>10.05</td>
<td>.85</td>
</tr>
<tr>
<td>CISS – Emotion</td>
<td>48.17</td>
<td>10.03</td>
<td>.86</td>
</tr>
<tr>
<td>CISS – Avoidance</td>
<td>48.56</td>
<td>10.85</td>
<td>.86</td>
</tr>
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</table>

<sup>a</sup>N = 15 due to missing data.
Table 2. Pearson correlations for Study 1 variables (N = 18).

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
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<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
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<tr>
<td>2  Time 1 - GSE</td>
<td>.29</td>
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<tr>
<td>3  - STAI-S</td>
<td>-.46</td>
<td>-.22</td>
<td>-</td>
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<tr>
<td>4  - STAI-T</td>
<td>-.03</td>
<td>-.63**</td>
<td>.55*</td>
<td>-</td>
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<tr>
<td>5  - BDI-II</td>
<td>-.23</td>
<td>-.42</td>
<td>.65**</td>
<td>.82***</td>
<td>-</td>
<td></td>
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<tr>
<td>6  DHS</td>
<td>-.11</td>
<td>.09</td>
<td>.35</td>
<td>.34</td>
<td>.32</td>
<td>-</td>
<td></td>
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</tr>
<tr>
<td>7  LES</td>
<td>-.23</td>
<td>-.24</td>
<td>.54*</td>
<td>.33</td>
<td>.41</td>
<td>.43</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>8  PBI-Care</td>
<td>-.14</td>
<td>.12</td>
<td>.15</td>
<td>.22</td>
<td>.21</td>
<td>-.66**</td>
<td>-.43</td>
<td>-</td>
<td></td>
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<tr>
<td>9  PBI-Overprotection</td>
<td>.28</td>
<td>.22</td>
<td>.41</td>
<td>.17</td>
<td>.19</td>
<td>.51*</td>
<td>.51*</td>
<td>-.56*</td>
<td>-</td>
<td></td>
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<tr>
<td>10 Time 2 - GSE</td>
<td>-.32</td>
<td>.57*</td>
<td>-.04</td>
<td>-.31</td>
<td>-.06</td>
<td>-.11</td>
<td>.05</td>
<td>.53*</td>
<td>-.24</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11 - STAI-T</td>
<td>.12</td>
<td>.21</td>
<td>.67**</td>
<td>.62**</td>
<td>.53*</td>
<td>.58*</td>
<td>.59**</td>
<td>-.47*</td>
<td>.76***</td>
<td>-.37</td>
<td>-</td>
<td></td>
<td></td>
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<tr>
<td>12 - BDI-II</td>
<td>.10</td>
<td>-.02</td>
<td>.54*</td>
<td>.32</td>
<td>.36</td>
<td>.47*</td>
<td>.65**</td>
<td>-.50*</td>
<td>.53*</td>
<td>-.32</td>
<td>.72***</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13 CISS-Task</td>
<td>.21</td>
<td>.56*</td>
<td>-.13</td>
<td>-.18</td>
<td>-.21</td>
<td>.12</td>
<td>-.02</td>
<td>.25</td>
<td>.01</td>
<td>.61**</td>
<td>-.01</td>
<td>-.07</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>14 CISS-Emotion</td>
<td>.02</td>
<td>-.23</td>
<td>.64**</td>
<td>.58*</td>
<td>.58*</td>
<td>.50*</td>
<td>.39</td>
<td>-.52*</td>
<td>.50*</td>
<td>-.38</td>
<td>.67**</td>
<td>.75***</td>
<td>-.18</td>
<td>-</td>
</tr>
<tr>
<td>15 CISS-Avoidance</td>
<td>.14</td>
<td>.20</td>
<td>.10</td>
<td>-.07</td>
<td>-.20</td>
<td>.06</td>
<td>.06</td>
<td>.04</td>
<td>-.02</td>
<td>.13</td>
<td>-.09</td>
<td>.13</td>
<td>.15</td>
<td>.21</td>
</tr>
</tbody>
</table>

Note. Cortisol reactivity = Cortisol Time 2 - Cortisol Time 1, STAI = State-Trait Anxiety Inventory, PBI = Parental Bonding Instrument, LES = Life Experiences Survey, DHS = Daily Hassles Scale, BDI-II = Beck Depression Inventory-II, SSS = Sense of Social Support, GSE = General Self-Efficacy Scale, CISS = Coping Inventory for Stressful Situations.

*n = 15 due to missing data.

*** p<.001; ** p<.01; * p<.05.
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(BDI-II Time 1: $r = .58$; BDI-II Time 2: $r = .75$; STAI-T Time 1: $r = .58$; and STAI-T Time 2: $r = .67$). GSE scores at Time 1 and Time 2 were the only measures significantly correlated with CISS-Task scores ($r = .56$ and $r = .61$, respectively).

**Stress Response**

A paired-samples t-test was used to test if C2 cortisol levels differed significantly from C1 baseline measures for the returning participants. The analysis revealed that C2 cortisol levels ($M = .35$, $SD = .31$) were higher from baseline measures ($M = .31$, $SD = .23$). however, the difference did not reach statistical significance. In contrast, the cortisol stress response did reach statistical significance for Hand’s (2001) study participants, likely due to the larger N.

**Stress Manipulation Check**

The average stress rating of the TSST for returning participants was $M = 10.39$, $SD = 1.91$, which was significantly higher than the midpoint of the 14-point scale, $t(17) = 6.40$, $p < .001$, two tailed. In contrast, the viewer’s stress rating was significantly lower than participants’ self-report stress rating ($M = 7.53$, $SD = 2.71$), $t(17) = -4.46$, $p < .001$, two tailed. Furthermore, the participants’ lowest stress rating was a 7, whereas, the viewer’s lowest rating was a 3. The participants’ perception of the stressor was higher than what was visibly apparent to the observer. The same pattern was found in Hand’s (2001) study with the complete sample.

**Hypothesis # 1: Stress Moderating the Personality-Depression Relation**

Testing for moderation is essentially testing the “differential effect of the independent variable on the dependent variable as a function of the moderator” (Baron & Kenny, 1986, p. 1174). In accordance with Baron and Kenny’s (1986) definition of a
moderator, for the present study stress (as measured by the LES and DHS) is considered a "condition" (p. 1174). That is stress evokes or triggers perceptions associated with self-efficacy and social support. Importantly moderator variables function at the same level as independent variables and can be either situation or person variables (Baron and Kenny, 1986).

To test the hypothesis that stress (life events or daily hassles) moderates the relation between self-efficacy (GSE) at Time 1 and depressive symptomatology (BDI-II) at Time 2, two hierarchical multiple regressions were used, one using negative life events (LES) and the other using daily hassles (DHS) as the measure of stress. In both sets of analyses, Time 1 BDI-II depression was entered at step 1 as a control variable, and the main effects, Time 1 GSE (IV) and stress (moderator), and the interaction term, GSE × stress (LES or DHS), were entered at step 2. The moderator hypothesis is supported if the interaction term is significant. Whether there are significant main effects is not relevant to testing for moderation (Baron & Kenny, 1986). The hypothesis was not supported for either of the models. With all IVs in the equation (LES), \( R^2 = .44 \) (adjusted \( R^2 = .26 \)), \( F(4, 13) = 2.53, p > .05 \), and (DHS) \( R^2 = .29 \) (adjusted \( R^2 = .07 \)), \( F(4, 13) = 1.34, p > .30 \).

With the low ratio of cases to IVs, these results need to be interpreted with caution. According to Tabachnick and Fidell (1996) a sample size of 82 would have been required for the analyses ran here, based on \( N \geq 50 + 8m \) (\( m \) being the number of IVs).

Hypothesis # 2: Stress Moderating the Physiological Reactivity-Mental Health Relation

Individual variance in physiological reactivity (\( C2 - C1 \)) was to be explored to test its predictive ability of Time 2 anxiety (STAI-T) and depressive symptomatology (BDI-II) in the event of natural occurring stressors (LES or DHS). With only 15 cases and 4
potential IVs, however, the regression analyses were not possible. Time 1 mental health measures were to be entered at step 1, and the interaction component, stress × physiological reactivity was to be entered with their main effect components at step 2 for each of the analyses.

Hypothesis #3: Physiological Reactivity and Coping Predicting Anxiety

Hierarchical multiple regression analyses to test whether physiological reactivity could explain anything over and above coping in the prediction of Time 2 anxiety were not run due to the low ratio of cases to IVs. Time 1 anxiety scores, stress, and coping were to be entered at Step 1, and physiological reactivity (C2 − C1) was to be entered at Step 2 to determine if physiological reactivity had any additional effect on anxiety.

Hypothesis #4: Anomalous Parenting and Physiological Reactivity

An independent t-test was used to test whether physiological reactivity in response to a laboratory stressor differed for those who reported an anomalous parenting style (i.e., affectionless control defined by low care and high overprotection scores based on cut-off scores on the PBI). As hypothesized, physiological reactivity was significantly higher for individuals who reported experiencing an affectionless control style of parenting (for n = 4, M = .17, SD = .18) than those who reported any of the other three parenting styles (for n = 11, M = -.01, SD = .10), t(13) = 2.45, p < .05, one-tailed.

Further analysis tested if physiological reactivity differed for individuals reporting high versus low overprotection scores on the PBI (i.e., based on cut-off score on PBI Overprotection subscale). As predicted, physiological reactivity was higher for individuals reporting high overprotection (for n = 7, M = .11, SD .16) than individuals
reporting low overprotection (for n = 8, M = -.02, SD = .10), t(13) = 1.77, p < .05, one tailed.

Testing for Sampling Bias

Due to the very low return rate for the present study (23%), further exploratory analyses were performed to determine if participants who returned for continuation in the study differed significantly from participants who did not on measures of self-efficacy, depression, and physiological reactivity. Independent t-tests were used to address this question. GSE scores (M = 172.50, SD = 22.16) for the sample of participants who returned for the study did not differ significantly from those who did not (M = 178.07, 26.85), t(77) = .80, p > .05, two tailed. Nor did BDI-II scores for returning participants (M = 7.17, SD = 5.09) differ significantly from those who did not return (M = 7.36, SD = 6.14), t(77) = .12, p > .05, two tailed. The difference between physiological reactivity for participants who did return (M = .04, SD = .14) and those who did not (M = .19, SD = .21) was significant, however, t(51) = 2.54, p < .05, two tailed. Those who returned for the present study experienced less physiological reactivity to the experimental stressor than those who did not, thereby providing a biased sample with respect to this variable.

Discussion

With a prospective design, one of the goals of the present study was to examine Time 1 measures of self-efficacy, anxiety and depressive symptomatology to predict later symptomatology and, thereby, obtain better evidence for the direction of causal relations than can be obtained for one-time correlational studies alone. Also of interest were individual physiological differences and whether these differences could differentially predict future anxiety and depressive symptomatology. Finally, whether individual
differences in physiological reactivity could be found in relation to differing parenting styles was explored. Due to a poor return rate from the original small sample (Hand, 2001), a number of the regression analyses to test certain proposed hypotheses were not reasonable. There will be no further discussion regarding hypotheses not tested.

**Stress Response for Returning Participants - A Biased Sample**

There was not a significant difference in baseline measures of cortisol and measures taken 10 minutes after completion of the TSST for returning participants. These results differ from those obtained by Hand (2001) where cortisol measures taken after completion of the TSST were significantly higher than baseline measures for the complete sample. To see if the returning sample experienced the TSST as stressful, responses to the self-report stress rating measure taken at Time 1 was analysed. All participants rated the TSST between moderately and very stressful. This finding is consistent with Hand's (2001).

Since it was possible that the returning participants represented a biased sample, additional analyses were warranted. No significant difference was found between the two groups' depressive symptomatology or self-efficacy. There was, however, a remarkable difference in their physiological reactivity to the TSST laboratory stressor, with those who did not return for participation exhibiting higher cortisol reactivity levels. This difference may also account for the discrepancy in findings for cortisol reactivity between the present study and Hand's (2001). Participants who returned for the present study did not have a significant increase in cortisol levels from baseline.

The TSST used at Time 1 of the study may have been so stressful that it lowered the return rate. Participants were assured, however, that there would be no experimental
component in the follow-up study. Any reason for such a low return rate is purely speculative. For example, the time of year (during final exams) when Time 2 data was being collected, or recruitment method may also have contributed significantly to the low return rate. Individuals who have higher physiological reactivity to stress may be less inclined to go through the hassle (i.e., taking the time out from exam study) to participate in a study, especially when participation points are not being offered. Future research using a prospective design should be cautious in obtaining a large enough sample to conduct the proposed regression analyses here, and sampling bias would still need to be tested. The use of a non-experimental stressor (e.g., collection of saliva samples during scheduled exam time) may also be considered.

**Stress as a Moderating Variable**

Contrary to similar research on cognitive (attributional) moderational models (see Chorpita & Barlow, 1998 for a review), there was no support found for stress (life events or daily hassles) moderating the relation between self-efficacy at Time 1 and depressive symptomatology at Time 2. The longitudinal component of the present study may have influenced these findings, however. The correlation between Time 1 self-efficacy and Time 2 depressive symptomatology was very low ($r = -.02$), and so failed to predict Time 2 symptomatology with such low statistical power. Furthermore, these results differ from previous longitudinal research on self-efficacy’s predictive ability for depression. Maciejewski et al. (2000), using path analysis, found a significant, indirect effect for a “baseline” measure of self-efficacy, mediated by self-efficacy at followup, on depressive symptomatology after a three year period. Again, the small sample used and low statistical power for the present study may account for the difference. Additionally, the
removal of social domain items from the GSE may have affected the sensitivity of the measure used for the present study.

**Anomalous Parenting and Stress Response**

Parker et al.'s (1979) construct “affectionless control” was used to test whether physiological reactivity differed as a result of early childhood experiences. To the researcher's knowledge, the PBI had not been previously used to study individual differences in physiological reactivity. The results of the exploratory analyses conducted here do, however, suggest that it may be a useful tool to examine individual differences in physiological reactivity in addition to other developmental processes and risk factors related to depression and anxiety.

Animal models have found that individual differences in physiological reactivity in response to stress is related to early experiences during the development of the organism (Meaney et al., 1996). Human studies have also found that early stressors result in HPA dysregulation in adulthood (Weiss, Longhurst, & Mazure, 1999). Research with toddlers classified according to parental attachment and behavioural inhibition has also found higher cortisol reactivity in those considered both insecurely attached and behaviourally inhibited (Nachmias et al., 1996).

To the extent that the two dimensions of parenting (care and the encouragement of independence) being measured by the PBI (Parker, 1983) are said to foster secure attachment, the present study using an adult population has established findings consistent with research on human infants and toddlers (Nachmias et al., 1996). Specifically, higher cortisol reactivity was found in individuals who reported both high
overprotection and low care on the two parenting dimensions (i.e., affectionless control). High overprotection alone was also related to higher physiological reactivity.

One possible interpretation of these findings is that parents who interfere with their children’s adaptation to their environment may impede their development of any sense of mastery or control (Carlson & Sroufe, 1995; Chorpita & Barlow, 1998; Kopp, 1989), which may in turn result in inefficient HPA functioning when stress is encountered. This would further support the view that biological and environmental influences interact to predispose individuals to depression (Liu et al., 1997; Meaney et al., 1996; Plotsky et al., 1998) and thus discount the position proposed by Parker and his colleagues (1987) that depressive types exist based on genetic and biochemical distinctions. Whether elevated cortisol reactivity found in the university population used for the present study predispose them to psychopathology is unknown.

These results need to be interpreted with caution as returning participants represented a biased sample. A significant difference was still found in cortisol reactivity based on reported parenting style without the higher cortisol reactivity participants returning, and one can only speculate as to whether these findings would hold true or be strengthened if all participants had completed the PBI. Unfortunately, parenting style was measured at Time 2 only. Due to the length of time required for each participant to complete Time 1 procedures (which included participation in Hand’s study, 2001), it was not feasible to have all measures completed at that time.
Risk factors for depression and anxiety

STUDY 2

Risk Factors For Depression and Anxiety: Developmental Antecedents

Study 2 examines whether an anomalous parenting style may have a direct effect on poor adult adjustment or whether anomalous parenting establishes a mediating vulnerability for poor adult adjustment. The extent to which early parent-child relationships might contribute to adult anxiety and depression through the development of stable personality features which determine coping mechanisms employed when stress is encountered will be explored using correlational data. Essentially, Study 2 explores the following questions: 1) Can we reliably predict anxiety/depressive symptomatology given knowledge of stressors (i.e., life events/daily hassles), coping style, personality variables, and parenting style? 2) What role might parenting style play in the development of anxiety and depressive symptomatology? 3) What are the roles of self-efficacy and perceived social support (personality variables) in determining anxiety and depressive symptomatology? More specifically, do these personality variables provide a protective role (through the development of an effective coping style) against the development of anxiety and depressive symptomatology following stressors?

Specific hypotheses for Study 2 consist of the following. 1) Parenting style (as assessed by the Care and Overprotection scales on the PBI) is related to personality (perceived social support and self-efficacy). Specifically, low care and high overprotection is related to low perceived social support and low self-efficacy. 2) The relation between parenting style and coping is mediated by personality (self-efficacy and perceived social support). 3) The relation between personality variables (self-efficacy and perceived support) and coping style is moderated by stressors (life events and daily
hassles). According to the process model of coping, a moderating effect should hold true. Studies of this transactional theory are limited (Parkes, 1986). 4) The relation between the personality × stress interaction and mental health is mediated by coping. The model proposed by these hypotheses is represented by Figure 1.

Method

Participants

The returning sample obtained for Study 1 was augmented with 166 additional psychology students for participation in Study 2. Psychology students received course credit (2 points) for their participation. Total recruitment for Study 2 was 184 participants. Of the 184 participants, 38 did not indicate their gender [126 F / 20 M / 38 missing], and 53 did not indicate their age [for N = 131, age mean and (standard deviation) = 20.47 (2.66) years] on the demographic questions. Age and gender analyses were not part of the set of hypotheses for Study 2 and, therefore, all participants were included in the analyses.

Procedure and Measurements

Participants were given questionnaires to complete in groups in classrooms at Acadia University. Prior to completing the questionnaires, an informed consent form was read and signed by each of the participants. Two copies of the consent form were included, one of which was to be retained by participants and acted as a debriefing form (see Appendix H). Written instructions were provided on how to complete each of the questionnaires according to the guidelines of the instruments. The administrator of the
Figure 1. Path diagram representing hypothesized relationships among variables for Study 2.

Constructs are shown in circles and their corresponding operational definitions (measures used to test the model) are in rectangles.
questionnaires remained available during their completion to answer any questions. It was emphasized that the participants should complete the questionnaires honestly to ensure the validity and reliability of the study.

Measurements taken included those obtained for Time 2 -Study 1 (BDI-II, STAI-T, PBI, GSE, CISS, LES, and DHS), described above. The following additional measure pertains to Study 2 only.

Perceived Social Support

The Sense of Support Scale (SSS; Dolbier & Steinhardt, 2000) is a measure of perceived social support. Perceptions of the quality and the quantity of social support are included in this 21-item self-report measure. Participants rate each statement on a 4-point Likert scale ranging from 0 (not at all true) to 3 (completely true). A total score is computed by adding all items, such that high scores are indicative of a high perception of social support. The internal reliability (Cronbach’s α) of the SSS was .84 for a university sample. Convergent and divergent validity have been established for the SSS (Dolbier & Steinhardt, 2000).

Results

Using SPSS data analysis programs, all variables were screened for accuracy of data entry, missing values, and evaluation of assumptions. Missing data on the BDI-II, PBI-Care and PBI-Overprotection, and SSS were replaced by the mean for all cases (Tabachnick & Fidell, 1996). There were three missing values on the BDI-II, two missing values on both of the PBI-Care and PBI-Overprotection, and one missing value on the SSS. One case was identified through Mahalanobis distance as a multivariate outlier with $p < .001$ (Tabachnick & Fidell, 1996) and was deleted leaving 183 cases for the
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remainder of analyses. Univariate outliers were detected through extreme z scores (i.e., > 3.29) (Tabachnick & Fidell, 1996). To reduce the impact of univariate outliers, scores were changed to either one unit larger or one unit smaller than the next extreme score for that variable (Tabachnick & Fidell, 1996). Two univariate outliers on each of the BDI-II and DHS, and three univariate outliers on the LES were detected and rescored.

Means, standard deviations and coefficient alpha reliabilities for all measures are presented in Table 3. All measures met acceptable levels of reliability with coefficient alphas ranging from .79 to .92. An alpha level of .05 was used for all statistical analysis in the present study.

Table 4 presents the correlations for all Study 2 variables. There were a large number of significant correlations between variables. As was the case in Study 1, BDI-II scores correlated significantly with STAI-T scores ($r = .71$). Both PBI-Care and PBI-Overprotection scores were significantly correlated with mental health measures in the expected direction (BDI-II: $r = -.28$ and $r = .33$, respectively, and STAI-T: $r = -.32$ and $r = .44$, respectively). That is, low PBI-Care scores were associated with high BDI-II and STAI-T scores, and high PBI-Overprotection scores were associated with high BDI-II and STAI-T scores. Additionally, PBI-Care and PBI-Overprotection scores have significant correlations with each of the two personality variables (SSS and GSE) in the expected direction. PBI-Care has significant positive correlations with SSS and GSE ($r = .52$, and $r = .22$, respectively), while PBI-Overprotection has significant negative correlations with SSS and GSE ($r = -.22$, and $r = -.23$, respectively). Also of interest are the significant correlations between coping styles and personality and mental health measures. CISS-Task coping has a significant positive correlation with GSE ($r = .57$),
Table 3. Means, standard deviations, and Cronbach alpha reliabilities for Study 2 measures ($N = 183$).

<table>
<thead>
<tr>
<th>Measure</th>
<th>M</th>
<th>SD</th>
<th>Cronbach $\alpha^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI-II</td>
<td>10.83</td>
<td>7.23</td>
<td>.88</td>
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<tr>
<td>STAI-T</td>
<td>41.70</td>
<td>10.31</td>
<td>.92</td>
</tr>
<tr>
<td>PBI (Care)</td>
<td>29.24</td>
<td>6.23</td>
<td>.90</td>
</tr>
<tr>
<td>PBI (Overprotection)</td>
<td>11.93</td>
<td>6.76</td>
<td>.85</td>
</tr>
<tr>
<td>GSE</td>
<td>165.40</td>
<td>31.53</td>
<td>.90</td>
</tr>
<tr>
<td>SSS</td>
<td>47.80</td>
<td>7.63</td>
<td>.79</td>
</tr>
<tr>
<td>LES</td>
<td>9.06</td>
<td>7.08</td>
<td>-</td>
</tr>
<tr>
<td>DHS</td>
<td>48.10</td>
<td>34.80</td>
<td>-</td>
</tr>
<tr>
<td>CISS – Task</td>
<td>45.90</td>
<td>10.99</td>
<td>.91</td>
</tr>
<tr>
<td>CISS – Emotion</td>
<td>50.69</td>
<td>10.31</td>
<td>.90</td>
</tr>
<tr>
<td>CISS – Avoidance</td>
<td>55.58</td>
<td>10.38</td>
<td>.85</td>
</tr>
</tbody>
</table>

$^a$For alpha reliabilities $N = 181$ for the BDI-II, $N = 182$ for the PBI-Care and PBI-Overprotection; and $N = 182$ for the SSS.

**Note.** BDI-II = Beck Depression Inventory II, STAI = State-Trait Anxiety Inventory-Trait subscale, PBI = Parental Bonding Instrument, GSE = General Self-Efficacy, SSS = Sense of Social Support, LES = Life Events Survey, DHS = Daily Hassles Scale, CISS = Coping Inventory for Stressful Situations
Table 4. Pearson correlations for Study 2 variables (N=183).

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
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<tbody>
<tr>
<td>1 STAI-T</td>
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</tr>
<tr>
<td>2 PBI-Care</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>3 PBI-Overprotection</td>
<td>.44***</td>
<td>-.30***</td>
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<td></td>
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<td>4 LES</td>
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<td>.25**</td>
<td></td>
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<td>5 DHS</td>
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<td>.25**</td>
<td>.48***</td>
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<tr>
<td>6 BDI-II</td>
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<td>.33***</td>
<td>.49***</td>
<td>.47***</td>
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<tr>
<td>7 SSS</td>
<td>-.41***</td>
<td>.52***</td>
<td>-.22**</td>
<td>-.18*</td>
<td>-.20**</td>
<td>-.38***</td>
<td></td>
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<td></td>
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<tr>
<td>8 GSE</td>
<td>-.62***</td>
<td>.22**</td>
<td>-.23**</td>
<td>-.22**</td>
<td>-.27***</td>
<td>-.57***</td>
<td>.42***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 CISS-Task</td>
<td>-.38***</td>
<td>.22**</td>
<td>-.22**</td>
<td>-.19**</td>
<td>-.08</td>
<td>-.36***</td>
<td>.25**</td>
<td>.57***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 CISS-Emotion</td>
<td>.75***</td>
<td>-.20**</td>
<td>.34***</td>
<td>.26***</td>
<td>.43***</td>
<td>.60***</td>
<td>-.29***</td>
<td>-.57***</td>
<td>-.24**</td>
<td></td>
</tr>
<tr>
<td>11 CISS-Avoidance</td>
<td>.00</td>
<td>.22**</td>
<td>-.07</td>
<td>-.03</td>
<td>.01</td>
<td>-.03</td>
<td>.18*</td>
<td>-.07</td>
<td>.13</td>
<td>.24**</td>
</tr>
</tbody>
</table>

*Note. STAI = State-Trait Anxiety Inventory-Trait subscale, PBI = Parental Bonding Instrument, LES = Life Experiences Survey, DHS = Daily Hassles Scale, BDI-II = Beck Depression Inventory-II, SSS = Sense of Social Support, GSE = General Self-Efficacy Scale, CISS = Coping Inventory for Stressful Situations.

*** p<.001; ** p<.01; * p<.05.
while CISS-Emotion coping has a significant negative correlation with GSE ($r = -.57$). Furthermore, CISS-Emotion coping has positive correlations with both STAI-Trait anxiety ($r = .75$) and BDI-II depression scores ($r = .60$).

**Hypotheses # 1 and # 2 - Personality Mediating the Parenting-Coping Relation**

To test the proposed model in Figure 1, which combines mediation and moderation components, methods proposed by Baron and Kenny (1986) were employed. According to Baron and Kenny (1986), the following conditions must hold for a variable to function as a mediator. In the first equation the independent variable must affect the mediator variable; in the second the independent variable must affect the dependent variable; and in the third, the mediator variable must affect the dependent variable. Mediation is established if the effect of the independent variable on the dependent variable is less in the third equation than in the second when the mediator’s effects are controlled (Baron & Kenny, 1986).

First, it was hypothesized that low PBI-Care scores and high PBI-Overprotection scores would predict personality (specifically, low perceived social support, and low self-efficacy). It was further hypothesized that the relation between parenting style and coping would be mediated by these personality variables (self-efficacy and perceived social support). As such, the first mediational model tested involved personality variables as potential mediators of the relation between parenting style and coping. In the first regression equation, parenting was predicting personality, in the second parenting was predicting coping style, and in the third parenting and personality were predicting coping. If the effect of parenting on coping was less in the third equation than in the second, there was support for personality mediating the relation (Baron & Kenny, 1986). These three
steps in testing the possible mediation role of personality subsequently will be referred to as equations 1-3 where the relation between the hypothesized IV and mediator is tested first, then the relation between the IV and DV is tested, and thirdly, the relative predictor values of the IV and hypothesized mediator are assessed using multiple regression with both variables entered together. Furthermore, Baron and Kenny (1986) distinguish between partial and complete mediation. If the independent variable(s) (parenting) in the third equation is no longer significant, there is support for complete mediation. If, however, the independent variable(s) remains significant but accounts for less variance in the third regression equation, there is support for partial mediation.

With three coping styles (task, emotion, and avoidance) and two personality measurements (self-efficacy and perceived social support), six separate sets of analyses were required to test the above hypotheses as operationally defined in this study. Each set of analyses is organized according to one of the three types of coping, and in each set, self-efficacy (GSE) is tested for mediation first followed by perceived social support (SSS). PBI-Care and PBI-Overprotection are entered together in the first equation of each set of analyses. Unstandardized regression coefficients (B), standardized regression coefficients (β), standard errors (SE), semipartial correlations (sr^2), and R^2 values are displayed in table format and organized according to coping style for each of the three regression equations. Additionally, Figure 2 represents the supported relations among these variables.¹

¹ The supported relations displayed in all figures are the results of independent tests of mediation and moderation analyses.
Figure 2. Path diagram representing supported relations: Personality mediating parenting style-coping relation. Dashed lines represent negative relations.

Note. PBI = Parental Bonding Instrument, GSE = General Self-Efficacy scale, SSS = Sense of Social Support, and CISS = Coping Inventory for Stressful Situations.
Predicting Task Coping

The results of analyses involving CISS-Task coping are presented in Table 5. In the first set of analyses, support was found for complete mediation of self-efficacy (GSE) between the parenting and task coping relation. In the first equation PBI-Care was related to GSE ($t = 2.137, p < .05, r^2 = .02$) and PBI-Overprotection was related to GSE ($t = -2.46, p < .05, r^2 = .03$). For the second, PBI-Care was related to CISS-Task coping ($t = 2.26, p < .05, r^2 = .03$) and PBI-Overprotection was related to CISS-Task coping ($t = -2.28, p < .05, r^2 = .03$). In the final regression equation, GSE was related to CISS-Task coping ($t = 8.56, p < .001, r^2 = .27$) and PBI-Care and PBI-Overprotection were no longer significant ($t = 1.29, ns, r^2 = .01$, and $t = -1.10, ns, r^2 = .00$, respectively), thereby supporting complete mediation. Furthermore, the standardized Beta coefficients are in the expected direction. As hypothesized, low PBI-Overprotection and high PBI-Care were predictive of high GSE scores. High GSE in turn significantly predicted CISS-Task coping. The parenting attributes, overprotection and care, were not associated with task coping when self-efficacy was statistically controlled. In the final model, with all three independent variables included, 34% of the variance in Task coping was accounted for.

Overprotection was not significant ($t = -1.06, ns, r^2 = .00$) in the first regression equation when perceived social support (SSS) was being tested as a mediator between parenting and CISS-Task coping, and was, therefore, not included in the second set of

---

2 The significance of the reduction in Beta coefficients for all meditational analyses in the present study was not tested statistically.
Table 5. Multiple regressions testing two measures of personality (GSE and SSS) as mediators between two measures of parenting (PBI-C and PBI-O) and task coping. Personality mediates the relation if the standardized Beta coefficient for parenting is less in the third equation and personality is significant.

<table>
<thead>
<tr>
<th>Eq #</th>
<th>Criterion Variable</th>
<th>Predictors</th>
<th>B</th>
<th>SE</th>
<th>β</th>
<th>sr²</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a</td>
<td>GSE</td>
<td>a) PBI-C</td>
<td>.81</td>
<td>.38</td>
<td>.16</td>
<td>.02*</td>
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<tr>
<td></td>
<td></td>
<td>a) PBI-O</td>
<td>-.86</td>
<td>.35</td>
<td>-.19</td>
<td>.03*</td>
<td>.08***</td>
</tr>
<tr>
<td>1b</td>
<td>SSS</td>
<td>b) PBI-C</td>
<td>.64</td>
<td>.08</td>
<td>.52</td>
<td>.27***</td>
<td>.27***</td>
</tr>
<tr>
<td>2a</td>
<td>CISS-Task</td>
<td>a) PBI-C</td>
<td>.30</td>
<td>.13</td>
<td>.17</td>
<td>.03*</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>a) PBI-O</td>
<td>-.28</td>
<td>.12</td>
<td>-.17</td>
<td>.03*</td>
<td>.08**</td>
</tr>
<tr>
<td>2b</td>
<td>CISS-Task</td>
<td>b) PBI-C</td>
<td>.39</td>
<td>.13</td>
<td>.22</td>
<td>.05**</td>
<td>.05**</td>
</tr>
<tr>
<td>3a</td>
<td>CISS-Task</td>
<td>a) GSE</td>
<td>.19</td>
<td>.02</td>
<td>.54</td>
<td>.27***</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>a) PBI-C</td>
<td>.15</td>
<td>.11</td>
<td>.08</td>
<td>.01</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>a) PBI-O</td>
<td>-.12</td>
<td>.11</td>
<td>-.07</td>
<td>.00</td>
<td>.34***</td>
</tr>
<tr>
<td>3b</td>
<td>CISS-Task</td>
<td>b) SSS</td>
<td>.27</td>
<td>.12</td>
<td>.19</td>
<td>.03*</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>b) PBI-C</td>
<td>.22</td>
<td>.15</td>
<td>.12</td>
<td>.01</td>
<td>.07**</td>
</tr>
</tbody>
</table>

Note. GSE = General Self-Efficacy scale, SSS = Sense of Social Support, PBI-C = Parental Bonding Instrument-Care, PBI-O = Parental Bonding Instrument-Overprotection, Eq # = equation number, CISS = Coping Inventory for Stressful Situations.

*p < .05. **p < .01. ***p < .001.
analyses. Care was significant, however ($t = 7.55, p < .001, \sigma^2 = .23$). When the analyses were redone with only PBI-Care, there was support for a complete mediating role of SSS between PBI-Care and CISS-Task coping. In the first regression equation PBI-Care was related to SSS, $t = 8.24, p < .001, \sigma^2 = .27$. In the second regression equation PBI-Care was related to CISS-Task coping, $t = 3.04, p < .01, \sigma^2 = .05$. In the third regression equation SSS was related to CISS-Task coping, $t = 2.24, p < .05, \sigma^2 = .03$, and PBI-Care was no longer significant, $t = 1.45, ns, \sigma^2 = .01$, thereby supporting complete mediation. High PBI-Care was predictive of high SSS, and a high SSS was in turn predictive of CISS-Task coping. In the final model, with both predictors in the equation, 7% of the variance in Task coping was accounted for.

**Predicting Emotion Coping**

The results of analyses involving CISS-Emotion coping are summarized in Table 6. When self-efficacy (GSE) was tested as a mediator between both PBI-Care and PBI-Overprotection and CISS-Emotion coping, PBI-Care was not related to CISS-Emotion coping in the second regression equation ($t = -1.48, ns, \sigma^2 = .01$), however, PBI-Overprotection was related ($t = 4.13, p < .001, \sigma^2 = .08$). The analyses were, therefore, redone with PBI-Overprotection only. The relation between PBI-Overprotection and CISS-Emotion Coping was found to be partially mediated by GSE. In the first regression equation PBI-Overprotection was related to GSE, $t = -3.22, p < .01, \sigma^2 = .05$. In the second PBI-Overprotection was related to CISS-Emotion coping ($t = 4.78, p < .001, \sigma^2 = .12$). In the final regression equation GSE was related to CISS-Emotion coping ($t = -8.61, p < .001, \sigma^2 = .26$) and PBI-Overprotection was also found to be significant ($t = 3.50, p < .01, \sigma^2 = .04$), with the magnitude much reduced ($\sigma^2 = .12$ to .04) thereby
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Table 6. Multiple regressions testing two measures of personality (GSE and SSS) as mediators between two measures of parenting (PBI-C and PBI-O) and emotion coping. Personality mediates the relation if the standardized Beta coefficient for parenting is less in the third equation and personality is significant.

<table>
<thead>
<tr>
<th>Eq #</th>
<th>Criterion Variable</th>
<th>Predictors</th>
<th>B</th>
<th>SE</th>
<th>β</th>
<th>( R^2 )</th>
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</thead>
<tbody>
<tr>
<td>1a</td>
<td>GSE</td>
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<td>-.23</td>
<td>.05**</td>
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<td>1b</td>
<td>SSS</td>
<td>b) PBI-C</td>
<td>.64</td>
<td>.08</td>
<td>.52</td>
<td>.27***</td>
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<tr>
<td>2a</td>
<td>CISS-Emotion</td>
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<td>.51</td>
<td>.11</td>
<td>.34</td>
<td>.12***</td>
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<td>2b</td>
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<td>.04**</td>
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<td>3b</td>
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<td>b) SSS</td>
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<td>.11</td>
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<td></td>
<td>b) PBI-C</td>
<td>-.10</td>
<td>.14</td>
<td>-.06</td>
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</table>

Note. GSE = General Self-Efficacy scale. SSS = Sense of Social Support, PBI-O = Parental Bonding Instrument-Overprotection, PBI-C = Parental Bonding Instrument-Care, Eq # = equation number, CISS = Coping Inventory for Stressful Situations.

**p < .01. ***p < .001.
supporting partial mediation. As hypothesized, high PBI-Overprotection was predictive of low self-efficacy. Furthermore, high PBI-Overprotection was predictive of CISS-Emotion coping, both directly and indirectly through low self-efficacy with the two variables accounting for 37% of the variance in the final model.

When perceived social support was being tested as a mediator between both PBI-Care and PBI-Overprotection and CISS-Emotion coping, PBI-Overprotection was not related to SSS in the first regression equation \( t = -1.06, \text{ns, } r^2 = .00 \), however, PBI-Care was \( t = 7.55, p < .001, r^2 = .23 \). The set of analyses was redone with PBI-Care only. In the first regression equation PBI-Care was related to SSS \( t = 8.24, p < .001, r^2 = .27 \). In the second regression equation PBI-Care was related to CISS-Emotion coping \( t = -2.73, p < .01, r^2 = -.04 \), and in the final equation SSS was related to CISS-Emotion coping \( t = -3.11, p < .01, r^2 = .05 \). PBI-Care was no longer significant \( t = -.76, \text{ns, } r^2 = .00 \), thereby supporting total mediation. As predicted, high PBI-Care was predictive of high SSS, which in turn was predictive of CISS-Emotion coping. With both predictors in the final equation, 9% of the variance in CISS-Emotion coping was accounted for.

**Predicting Avoidance Coping**

In the final set of analyses involving CISS-Avoidance coping, there was no support for the mediating role of self-efficacy (GSE) or perceived social support (SSS). PBI-Overprotection was not related to SSS in the first regression equation when SSS was being tested as a mediator \( t = -1.06, \text{ns, } r^2 = .00 \). When the analyses were redone with PBI-Care, the relation between PBI-Care and CISS-Avoidance coping was not found to be mediated by perceived social support (SSS). In the first regression equation PBI-Care was related to SSS \( t = 8.24, p < .001, r^2 = .27 \), and in the second PBI-Care was related
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to CISS-Avoidance coping (t = 3.00, p < .01, sr^2 = .05). In the third regression equation
SSS was not related to CISS-Avoidance coping (t = .98, ns, sr^2 = .00), effectively
demonstrating the lack of a mediational role for perceived social support. PBI-Care was
found to be significant (t = 2.05, p < .05, sr^2 = .02) in the third equation.

In the first regression equation, when GSE was being tested for mediation, both
PBI-Care and PBI-Overprotection were related to GSE (t = 2.14, p < .05, sr^2 = .02, and t
= -2.46, p < .05, sr^2 = .03, respectively). In the second regression equation PBI-Care was
related to CISS-Avoidance coping (t = 2.86, p < .01, sr^2 = .04), however, PBI-
Overprotection was not significantly related (t = -.01, ns, sr^2 = .00). Again, when the
analyses were redone with PBI-Care, the relation between PBI-Care and CISS-Avoidance
coping was not mediated by self-efficacy (GSE). GSE was no longer significant in the
third regression equation and the standardized coefficient (β) for PBI-Care was larger in
the third regression equation than in second equation. PBI-Care was related to GSE in the
first equation (t = 2.97, p < .01, sr^2 = .05) and related to CISS-Avoidance coping (t =
3.00, p < .01, sr^2 = .05) in the second regression equation. In the third equation, however,
GSE was not significant (t = -1.71, ns, sr^2 = -.01), and PBI-Care was significant (t = 3.32,
p < .01, sr^2 = .06), effectively demonstrating the lack of a mediational role for GSE. PBI-
Care had a direct effect on CISS-Avoidance coping.

Hypothesis # 3 - Stress as a Moderator of Personality-Coping Relation

It was predicted that the relation between personality variables (GSE and SSS)
and coping style would be moderated by stress (LES and DHS). Again, following Baron
and Kenny's (1986) recommended procedure, the dependent variable (coping) is
sequentially regressed on the independent variable (personality), the moderator variable
Risk factors for depression and anxiety

(stress), and their product (personality × stress interaction). If the interaction term is significant, there is support for the moderator hypothesis. With two personality variables, two stress variables, and three coping variables, twelve sets of analyses were required to fully test for moderation. Prior to running the moderation analyses total scores for each of the measures were converted to z scores and interaction terms (i.e., the product of the moderator and independent variable) were created using the standardized scores. The following sets of analyses are organized according to the type of coping (i.e., Task, Emotion, and Avoidance) and consist of four sets of analyses for each type of coping. The first two sets of analyses were done with self-efficacy (GSE) as the personality variable, first with LES and then with DHS as the stress variables. Perceived social support (SSS) was used as the personality variable for the next two sets of analyses, first with LES and then with DHS as the stress variables. No support was found for stress as a moderator of the personality-coping relation; the personality × stress interaction failed to predict coping in every set of analyses.

**Task Coping**

For the first set of analyses testing the GSE × stress interaction’s ability to predict CISS-Task coping, GSE was entered into the equation first (Step 1), LES was entered next (Step 2), and finally the GSE × LES interaction (Step 3). In the final model GSE had a main effect in predicting CISS-Task coping ($r^2 = .30, p < .001$). LES was not significant ($r^2 = .00, p > .20$), nor was the GSE × LES interaction ($r^2 = .00, p > .70$). For the second set of analyses using DHS as the measure of stress, GSE was entered into the first equation (Step 1), DHS was entered next (Step 2), and finally the GSE × DHS interaction was entered (Step 3). Again, in the final model GSE had a main effect in
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predicting CISS-Task coping ($r^2 = .34, p < .001$). Neither DHS nor the GSE × DHS interaction were significant ($r^2 = .00, p > .20$, and $r^2 = .00, p > .50$, respectively).

To test whether the SSS × stress interaction was able to predict CISS-Task coping above their main effects, SSS was entered into the equation first, followed by LES, and finally the SSS × LES interaction term. Main effects were found for both SSS and LES ($r^2 = .06, p < .01$, and $r^2 = .02, p < .05$, respectively). The SSS × LES interaction term was not significant ($r^2 = .00, p > .40$). With DHS in the set of analysis, SSS had a main effect in predicting CISS-Task coping ($r^2 = .07, p < .001$), however, neither DHS ($r^2 = .00, p > .70$) nor the SSS × DHS were significant ($r^2 = .00, p > .50$).

**Emotion Coping**

In the first set of analyses testing the GSE × stress interaction’s ability to predict CISS-Emotion coping, GSE was entered into the equation first, followed by LES, and finally the GSE × LES interaction. Both GSE and LES had main effects in predicting CISS-Emotion coping ($r^2 = .28, p < .001$, and $r^2 = .03, p < .05$, respectively). The GSE × LES interaction failed to predict CISS-Emotion coping ($r^2 = .00, p > .20$). Following the same procedure with DHS as the stress measure, GSE was entered first, followed by DHS, and then the GSE × DHS interaction. Again only GSE and DHS were found to have main effects in predicting CISS-Emotion coping ($r^2 = .23, p < .001$, and $r^2 = .08, p < .001$, respectively). The GSE × DHS interaction failed to predict CISS-Emotion coping ($r^2 = .00, p > .20$).

To test whether the SSS × stress interaction was able to predict CISS-Emotion coping, SSS was entered into the equation first, followed by LES, and finally the SSS ×
LES interaction term. Again, main effects were found for both SSS and LES ($\sigma^2 = .07, p < .001$, and $\sigma^2 = .05, p < .01$, respectively). The interaction term was not significant ($\sigma^2 = .00, p > .40$). With DHS as the stress variable both SSS and DHS were found to have main effects in predicting CISS-Emotion coping ($\sigma^2 = .05, p < .01$, and $\sigma^2 = .14$, respectively). The interaction term was not significant ($\sigma^2 = .00, p > .30$).

**Avoidance Coping**

With analyses following the same procedure above testing the GSE x stress interaction’s ability to predict CISS-Avoidance coping, neither GSE nor LES were found to have main effects ($\sigma^2 = .01, p > .20$, and $\sigma^2 = .00, p > .50$, respectively). The interaction term GSE x LES also failed to predict CISS-Avoidance coping ($\sigma^2 = .00, p > .50$). Likewise, with the second stress measure, GSE and DHS were not significant ($\sigma^2 = .00, p > .30$, and $\sigma^2 = .00, p > .70$, respectively) nor was the interaction term GSE x DHS ($\sigma^2 = .01, p > .20$).

In the final set of analyses testing for moderation with CISS-Avoidance coping, main effects were not found for either of the stress variables (LES or DHS). In the first set of analyses, following the same procedure above, testing the SSS x stress interaction’s ability to predict CISS-Avoidance coping, neither SSS nor LES were found to have main effects ($\sigma^2 = .02, p = .06$, and $\sigma^2 = .00, p > .90$, respectively). The interaction term SSS x LES also failed to predict CISS-Avoidance coping ($\sigma^2 = .00, p > .30$). In the second set of analyses with DHS as the stress measure there was a significant main effect found for SSS ($\sigma^2 = .03, p < .05$). Neither DHS ($\sigma^2 = .00, p > .40$) nor the interaction term GSE x DHS were found to be significant ($\sigma^2 = .00, p > .90$). The significant main effect for SSS was likely due to the Social Diversion subscale items which are part of the CISS-
Avoidance subscale. Individuals with high perceived social support are probably more apt to use social diversions to cope when stress is encountered.

**Hypothesis # 4 - Coping Mediating Personality-Mental Health Relation**

Finally, it was predicted that coping would mediate the relation between personality $\times$ stress and mental health. Because there was no support found for the personality $\times$ stress interaction predicting coping, it was not logical to test this particular mediation model. It was still possible, however, for coping to mediate the relation between personality (perceived social support and self-efficacy) and mental health (depression and anxiety).

To test this mediation model, Baron and Kenny's (1986) recommendations were again followed. Because self-efficacy (GSE) failed to have any predictive ability of CISS-Avoidance coping in the previous sets of analyses under hypothesis # 3, CISS-Avoidance coping was only included in analyses involving perceived social support (SSS) as a personality variable. Three regression equations were used for each set of analyses with personality predicting coping in the first, personality predicting mental health in the second, and both personality and coping predicting mental health in the third. Again, analyses were organized according to coping style. Unstandardized regression coefficients ($B$), standardized regression coefficients ($\beta$), standard errors ($SE$), semipartial correlations ($sr^2$), and $R^2$ values are displayed in Tables 7 and 8 for each of the three regression equations in the following sets of analyses. Table 7 depicts all analyses involving self-efficacy, and Table 8 depicts all analyses involving perceived social support.
Table 7. Multiple regressions testing coping style as mediator between self-efficacy and mental health relations. Two sets of analyses, corresponding to each coping style, are presented for each of the two mental health measures: a and b. A reduced Beta for personality in the third equation supports mediation only if coping is also significant in predicting mental health.

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Note. Eq = Equation, CISS = Coping Inventory for Stressful Situations, GSE = General Self-Efficacy scale, BDI-II = Beck Depression Inventory-II, and STAI-T = State-Trait Anxiety Inventory-Trait.

aThe first regression equation is the same for each of the two sets of analyses (personality predicting coping style).

bThe second regression equation is the same for each of the two sets of analyses (personality predicting mental health).

***p < .001.
Table 8. Multiple regressions testing coping style as mediator between perceived social support and mental health relations. Three sets of analyses, corresponding to each coping style, are presented for each of the two mental health measures: a, b, and c. A reduced Beta for personality in the third equation supports mediation only if coping is also significant in predicting mental health.

<table>
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**Note.** Eq = equation, CISS = Coping Inventory for Stressful Situations, SSS = Sense of Social Support, BDI-II = Beck Depression Inventory-II, STAI-T = State-Trait Anxiety Inventory-Trait.

*a*The first regression equation is the same for each set of analyses (personality predicting coping).

*b*The second regression equation is the same for each set of analyses (personality predicting mental health).

*p < .05. ***p < .001.*
Task Coping as Mediator

In the first set of analyses support was not found for CISS-Task coping as a mediator of the self-efficacy-depression (BDI-II) relation. In the first regression equation, for each of the two analyses with GSE as the personality variable, GSE was related to CISS-Task coping ($t = 9.43$, $p < .001$, $r^2 = .33$). In the second regression equation, with depression as the measure of mental health, GSE was related to BDI-II ($t = -9.21$, $p < .001$, $r^2 = .32$). CISS-Task coping failed to predict BDI-II in the third regression equation ($t = -.63$, $p > .50$, $r^2 = .00$). GSE was directly related to BDI-II ($t = -7.17$, $p < .001$, $r^2 = .19$)

Support was also not found for CISS-Task coping mediating the self-efficacy-anxiety (STAI-T) relation. In the second regression equation with anxiety as the measure of mental health, GSE was related to STAI-T ($t = 10.64$, $p < .001$, $r^2 = .38$). CISS-Task coping failed to predict STAI-T in the third regression equation ($t = -.46$, $p > .60$, $r^2 = .00$). GSE was directly related to STAI-T ($t = -8.43$, $p < .001$, $r^2 = .24$).

There was support for CISS-Task coping as a partial mediator between the perceived social support-mental health relation. In the first regression equation, SSS was related to CISS-Task coping ($t = 3.51$, $p = .001$, $r^2 = .06$). In the second regression equation SSS was related to BDI-II ($t = -5.55$, $p < .001$, $r^2 = .15$). In the final regression equation both SSS and CISS-Task coping were related to BDI-II ($t = -4.57$, $p < .001$, $r^2 = .09$, and $t = -4.07$, $p < .001$, $r^2 = .07$, respectively) with the magnitude of the SSS-BDI-II relation reduced ($r^2 = .15$ to .09) thereby supporting partial mediation.

Likewise, with STAI-T as the measure of mental health, there was support for CISS-Task coping as a partial mediator. In the first regression equation SSS was related
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To CISS-Task coping ($t = 3.51, p = .001, \text{sr}^2 = .06$). For the second, SSS was related to STAI-T ($t = -5.98, p < .001, \text{sr}^2 = .16$). In the third equation both SSS and CISS-Task coping were related to STAI-T ($t = -4.96, p < .001, \text{sr}^2 = .10$, and $t = -4.40, p < .001, \text{sr}^2 = .08$, respectively) with the magnitude of the SSS-STAI-T relation reduced ($\text{sr}^2 = .16$ to .10), again providing support for partial mediation.

**Emotion Coping as Mediator**

Support was found for CISS-Emotion coping as a partial mediator for the personality-mental health relation. In the first regression equation for each of the two analyses with GSE as the personality variable, GSE was related to CISS-Emotion coping ($t = -9.40, p < .001, \text{sr}^2 = .33$). In the second regression equation, with depression as the measure of mental health, GSE was related to BDI-II ($t = -9.21, p < .001, \text{sr}^2 = .32$). In the final equation both GSE and CISS-Emotion coping were related to BDI-II ($t = -4.78, p < .001, \text{sr}^2 = .07$, and $t = 6.10, p < .001, \text{sr}^2 = .12$, respectively) with the magnitude reduced ($\text{sr}^2 = .33$ to .07). In the second set of regression equations, with anxiety as the measure of mental health, GSE was related to STAI-T ($t = -10.64, p < .001, \text{sr}^2 = .38$). With both GSE and CISS-Emotion coping related to STAI-T ($t = -5.04, p < .001, \text{sr}^2 = .05$, and $t = 10.42, p < .001, \text{sr}^2 = .23$, respectively) the final regression equation provided support for partial mediation since the magnitude of the GSE-STAI-T relation was reduced ($\text{sr}^2 = .38$ to .05).

In the first regression equation for the sets of analyses with SSS as the personality variable, SSS was related to CISS-Emotion coping ($t = -4.12, p < .001, \text{sr}^2 = .09$). In the second equation with depression as the measure of mental health, SSS was related to BDI-II ($t = -5.55, p < .001, \text{sr}^2 = .15$). Providing support for partial mediation, in the final
equation both SSS and CISS-Emotion coping were related to BDI-II ($t = -3.74, p < .001$, $r^2 = .05$, and $t = 8.99, p < .001$, $r^2 = .27$, respectively) with the SSS-BDI-II relation reduced ($r^2 = .15$ to .05). With anxiety as the measure of mental health, in the second equation, SSS was related to STAI-T ($t = -5.98, p < .001, r^2 = .16$). Again, both SSS and CISS-Emotion coping were related to STAI-T in the final equation ($t = -4.14, p < .001, r^2 = .04$, and $t = 14.00, p < .001, r^2 = .44$, respectively) with the magnitude of the SSS-STAI-T relation reduced ($r^2 = .16$ to .04), thereby supporting partial mediation.

**Avoidance Coping as Mediator**

There was no support found for CISS-Avoidance coping as a mediator between perceived social support and mental health. Following the same procedure as the preceding sets of analyses, in the first regression equation SSS was related to CISS-Avoidance coping ($t = 2.39, p < .05, r^2 = .03$). In the second regression equation, with depression as the measure of mental health, SSS was related to BDI-II ($t = -5.55, p < .001, r^2 = .15$). SSS remained a significant predictor of BDI-II in the final regression equation ($t = -5.55, p < .001, r^2 = .15$). CISS-Avoidance was not related to BDI-II ($t = .55, p > .50, r^2 = .00$), thereby effectively concluding the lack of mediation. With anxiety as the measure of mental health, SSS was related to STAI-T in the second and final regression equations ($t = -5.98, p < .001, r^2 = .16$, and $t = -6.07, p < .001, r^2 = .17$, respectively). CISS-Avoidance was not related to STAI-T in the final equation ($t = 1.06, p > .20, r^2 = .01$) again ruling out mediation.

**Predictive Ability of Stress on Mental Health**

Hierarchical regression analyses were utilized to see if stress (life events and daily hassles) was able to add anything over and above coping and/or personality measures in
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predicting mental health (depression and anxiety) outcomes. There was only partial
support for the mediating role of coping, thus, these analyses excluded coping when there
was no support for its mediating role. That is, if coping did not mediate the personality-
mental health relation, and personality was directly related to mental health, the
hierarchical regression analysis was done with only personality measures entered in step
1. In step 2 of every set of analyses, both measures of stress (LES and DHS) were
entered. Stress added significantly to the prediction of mental health in every set of
analyses. The unstandardized regression coefficients (B), standardized regression
coefficients (β), standard error (SE), semipartial correlations (sr²), and R² values at each
step are displayed in table format for each of the following sets of analyses. Table 9
depicts hierarchical regressions predicting depression, and Table 10 depicts hierarchical
regressions predicting anxiety. Additionally, Figure 3 (self-efficacy) and Figure 4
(perceived social support) depict the supported relations among these variables.

Predicting Depression

The first set of analyses tested whether stress (LES and DHS) could improve
prediction of depression (BDI-II) over and above self-efficacy (GSE). After step 1 with
GSE in the equation, R² = .32, F (1, 181) = 84.85, p < .001. After step 2, with all IVs
(GSE, LES and DHS) in the equation, R² = .50, F (3, 179) = 58.81, p < .001. Adding
stress to the equation resulted in a significant increment in R², R²Δ = .18, Finc (2,179) =
31.211, p < .001.

The next set of analyses tested whether stress could improve the prediction of
depression over and above CISS-Task coping and perceived social support (SSS). After
step 1, with CISS-Task coping and SSS in the equation, R² = .22, F (2, 180) = 25.02, p <
Table 9. Hierarchical regressions with personality, coping and stress predicting depression (BDI-II). Three regression analyses are presented according to support found for personality and coping relations to depression.

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<td>.01</td>
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Note. GSE = General Self-Efficacy scale, SSS = Sense of Social Support, CISS = Coping Inventory for Stressful Situations, LES = Life-Experiences Survey, and DHS = Daily Hassles Scale.

*p < .05. **p < .01. ***p < .001.
Table 10. Hierarchical regressions with personality, coping and stress predicting anxiety (STAI-T). Three regression analyses are presented according to support found for personality and coping relations to anxiety.

<table>
<thead>
<tr>
<th>Step</th>
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<th>R²</th>
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</table>

**Note.** GSE = General Self-Efficacy scale, SSS = Sense of Social Support, CISS = Coping Inventory for Stressful Situations, LES = Life-Experiences Survey, and DHS = Daily Hassles Scale.

*p < .05. **p < .01. ***p < .001.
Figure 3. Path diagram representing supported relations: Self efficacy, coping, and stress predicting mental health outcomes. Dashed lines represent negative relations.

Note. GSE = General Self-Efficacy scale, CISS = Coping Inventory for Stressful Situations, BDI-II = Beck Depression Inventory-II, STAI-T = State Trait Anxiety Inventory-Trait, LES = Life-Experiences Survey, and DHS = Daily Hassles Scale.

* DHS is not related to depression when CISS-Emotion coping is entered in the statistical model.
Figure 4. Path diagram representing supported relations: Perceived social support, coping, and stress predicting mental health outcomes. Dashed lines represent negative relations.

Note. SSS = Sense of Social Support, CISS = Coping Inventory for Stressful Situations, BDI-II = Beck Depression Inventory-II, STAI-T = State Trait Anxiety Inventory-Trait, LES = Life-Experiences Survey, and DHS = Daily Hassles Scale.
With the addition of LES and DHS at step 2, \( R^2 = .43, F(4, 178) = 33.98, p < .001. \)

Adding stress to the equation resulted in a significant increment in \( R^2, R^2\Delta = .21, F_{\text{inc}}(2, 178) = 33.56, p < .001. \)

With SSS, GSE and CISS-Emotion coping entered first, stress was able to add significantly to the prediction of depression. After step 1, with personality variables and emotion coping in the equation, \( R^2 = .45, F(3, 179) = 48.17, p < .001. \) After step 2, with the addition of LES and DHS, \( R^2 = .56, F(5, 177) = 45.73, p < .001. \) DHS did not, however, contribute significantly to the final model. LES alone resulted in a significant increment in \( R^2, R^2\Delta = .11, F_{\text{inc}}(2, 177) = 21.90, p < .001. \)

**Predicting Anxiety**

The next set of analyses tested whether stress could improve the prediction of anxiety (STAI-T) beyond that afforded by self-efficacy. After step 1, with GSE in the equation, \( R^2 = .39, F(1, 181) = 113.11, p < .001. \) After the addition of LES and DHS at step 2, \( R^2 = .53, F(3, 179) = 66.25, p < .001. \) Again, the addition of stress to the equation significantly improved \( R^2, R^2\Delta = .14, F_{\text{inc}}(2, 179) = 26.85, p < .001. \)

The next set of analyses tested whether stress could improve the prediction of anxiety over and above CISS-Task coping and perceived social support (SSS). After step 1, with CISS-Task coping and SSS in the equation, \( R^2 = .25, F(2, 180) = 29.38, p < .001. \) After the addition of LES and DHS at step 2, \( R^2 = .44, F(4, 178) = 34.80, p < .001. \) The addition of stress significantly improved \( R^2, R^2\Delta = .19, F_{\text{inc}}(2, 178) = 30.64, p < .001. \)

With SSS, GSE, and CISS-Emotion coping entered first, LES and DHS were able to improve significantly the prediction of anxiety. After step 1, with personality and emotion coping in the equation, \( R^2 = .63, F(3, 179) = 102.65, p < .001. \) After step 2, with
the addition of LES and DHS, $R^2 = .69, F (5, 177) = 76.83, p < .001$. Adding stress to the
equation resulted in a significant increment in $R^2, R^2\Delta = .05, F_{\text{inc}} (2, 177) = 14.75, p < .001$.

**Accounting for Variance in Mental Health – Atheoretical Analyses**

Stepwise multiple regression analyses were used with all predictor variables in the
original model to test which best statistically predicted mental health (depression and
anxiety). With depression as the mental health measure the final model included CISS-
Emotion, LES, GSE, and CISS-Avoidance, and accounted for 56% of the variance in
BDI-II. Similar results were found with anxiety as the measure for mental health. In the
final model 71% of the variance in STAI-T was accounted for with CISS-Emotion, LES,
GSE, PBI-Overprotection, CISS-Avoidance, and DHS entered into the equation.

Interestingly, CISS-Avoidance was included in both of the final models despite its
zero, or close to zero, bivariate correlations with mental health (STAI-T: $r = .00; BDI-II:
$r = -.03$). With CISS-Avoidance being included in the final models, closer inspection of
these results was warranted. The standardized coefficients for CISS-Avoidance in both
models (STAI-T: $\beta = -.12$; and BDI-II: $\beta = -.13$) were considerably larger than their
simple correlations. CISS-Avoidance’s correlation with CISS-Emotion increases the
latter’s importance by reducing variance in CISS-Emotion that was not associated with
mental health (Tabachnick & Fidell, 1996) (as evidenced by the increased $\beta$ for CISS-
Emotion when CISS-Avoidance enters the equation at Step 4 for depression and Step 5
for anxiety, see Tables 11 and 12, respectively). That is, including CISS-Avoidance
suppresses irrelevant variance in CISS-Emotion purifying the latter’s relation with the
criterion variables.
Table 11. Stepwise regression analysis predicting depression.

<table>
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<th>Model</th>
<th>Predictors</th>
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<th>SE</th>
<th>β</th>
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<th>( R^2 )</th>
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<td>.36</td>
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</tr>
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<td>.35</td>
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<td></td>
</tr>
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<td></td>
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<tr>
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Note. CISS = Coping Inventory for Stressful Situations, LES = Life-Experiences Survey, and GSE = General Self-Efficacy scale.

*p < .05. ***p < .001.
Table 12. Stepwise regression analysis predicting anxiety.

<table>
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Note. CISS = Coping Inventory for Stressful Situations, LES = Life-Experiences Survey, GSE = General Self-Efficacy scale, PBI-O = Parental Bonding Instrument-Overprotection, and DHS = Daily Hassles Scale.

*p < .05. **p < .01. ***p < .001.
Risk factors for depression and anxiety

Discussion

This study looked at several of the risk factors for depression and anxiety in an integrated model to see if a theoretically based causal process could be supported within a developmental framework. Research on depression and anxiety supports the need for a developmental perspective recognizing that early environment, especially quality of care, is vital to psychological functioning (Carlson & Sroufe, 1995). The possible developmental course of personality and coping style and their role in the incidence of anxiety and depressive symptomatology were of particular interest. As expected, a complex set of relations among the variables was revealed (see Figure 5a and 5b).

Parenting Style and Personality Development

It was predicted that parenting attributes would be predictive of self-efficacy and perceived social support. Support was found for these relations. The care dimension of the PBI predicted both personality measures and their relations were in the expected direction. Specifically, those who rated their parents higher on the care scale scored higher on measures of perceived social support and self-efficacy. Overprotection was also a significant predictor of self-efficacy, such that low ratings on the overprotection scale were related to higher self-efficacy.

Personality Mediating Parenting and Coping

To the researcher’s knowledge, the PBI had not previously been used to predict coping styles. It was predicted that self-efficacy and perceived social support would mediate the relation between parenting style and coping. There was support for this mediating role. For every set of analyses involving task and emotion coping, the relations between parenting and these coping styles were mediated by personality. Only one set of
Figure 5. Path diagrams representing all supported relations with a) self-efficacy and b) perceived social support as measures of personality. Dashed lines represent negative relations.

* Relation does not exist when CISS-Emotion coping is in the model.

** Relation does not exist when PBI-Care is in the model.
analyses revealed partial mediation (parental overprotection was still related to emotion coping but to a lesser extent after self-efficacy entered the model), while the remainder of analyses found support for complete mediation. Parental care was directly related to avoidance coping and there was no support for personality mediating this relation.

The findings in the present study provide support for Parker's (1993) view that parental anomalies create a diathesis to psychopathology through personality, rather than dispose directly to psychopathology. The support found for a mediating role of personality when parenting is predicting coping style may in part explain the conflicting reports in the literature relating anomalous parenting directly to depression and anxiety (see review by Gerlsma et al., 1990). The avenue from anomalous parenting leading to psychopathology is obviously more complex than has generally been studied. The two dimensions of parenting have different relations to the personality measures and coping styles studied here.

**Personality-Stress Interaction**

The hypothesis that personality variables interact with stressors (measured quantitatively) to predict coping was not supported in the present study. This finding is in contrast to Parkes' (1986) study on similarly proposed interactions. Parkes (1986) found support for the following interactions in predicting coping: social support by importance of stressor; neuroticism by work demand (stressor); and extraversion by importance of stressor. However, Parkes (1986) used a specific environment in which to measure stress (work demands in a hospital setting), neuroticism and extraversion as personality variables, and social support as a characteristic of the work environment (as opposed to a personality factor). For Parkes' (1986) study, having a specific context in which to measure stress may
have increased the sensitivity of the study. The present study did not analyse coping responses in relation to specific stressors. Of course, the type of stressor and coping response are not independent, that is any coping response is “situation-specific” (Billings & Moos, 1981). For example, the death of a family member would be more likely to elicit an emotional response than a work-related stressor. Folkman, Lazarus, Gruen, and DeLongis (1986) found a high degree of variability in the coping process depending on the nature of the stressor, however, they found evidence for moderate stability for an emotion-focused style of coping.

It is also possible that the personality variables measured for the present study were not the most sensitive for testing an interaction or transactional model of coping. For example, self-efficacy may be considered a resource of the individual whereby a judgment (“cognitive appraisal”) is made about an individual’s capacity to problem-solve or resolve a stressful occurrence. With this conception of self-efficacy in mind, the present study supports the mediating effect of a cognitive appraisal suggested by Folkman, et al. (1986). Furthermore, the social domain items were removed from the GSE and it is possible that the removal of these items affected any potential interaction effects with the stress measures. The general problem-solving tendencies measured by the remaining items of the GSE may also have specific stressors (such as school or work related stress) with which they would interact if stress had been measured more situation-specifically. What's more, other personality variables, such as neuroticism, may be the result of low self-efficacy. Research has found neuroticism to be associated with mental health outcomes (Kendler et al., 1993; Parkes, 1986). Future research would benefit from including both self-efficacy and neuroticism measures of personality as well.
Personality, Coping and Stress Predicting Mental Health

Personality was found to have both direct and indirect effects on mental health. Emotion coping and task coping were found to partially mediate the relation between perceived social support and mental health. In contrast, only emotion coping was found to partially mediate the relation between self-efficacy and mental health.

Both social support and self-efficacy are deemed important factors in determining how individuals cope in times of stress (Bandura, 1997; Endler, 1997). Perceived social support’s negative relation to anxiety and depression found in the present study is consistent with the literature (Brown & Harris, 1978; Coyne et al., 1981). As expected, self-efficacy also held a negative relation with anxiety and depression (Bandura, 1997; Maciejewski et al., 2000; Olioff et al., 1989). These personality factors associated with less anxiety and depression are at least partially by virtue of the coping strategies employed by individuals with high self-efficacy and high perceived social support.

Stress was able to significantly improve the statistical models’ ability to predict mental health after entry of coping and/or personality. Between 43% and 56% of the variance in depression was accounted for with personality, coping, and stress in the model. Similarly, between 44% and 69% of the variance in anxiety was accounted for by these variables. Both life events and daily hassles were able to contribute significant independent variance in every model except where CISS-Emotion coping was included in the model for the prediction of depression, in which case life events was the only significant stressor. These findings support the need to include both measures of stress in anxiety and depression research. Indeed, stress research is abundant. However, the focus has been
Risk factors for depression and anxiety

primarily on life events and little research has included both types of stress measures (Musil et al., 1998).

A theoretical Analysis

The analyses discussed above were theoretically driven by a large body of research. To the researcher's knowledge, this is the only research to have included all of the variables used for the present study in the study of depression and anxiety. To provide a benchmark of the total amount of variance in mental health the corpus of variables measured could account for in the absence of preconceived causal pathways, stepwise regression analyses was performed with all variables included. Seventy-one percent of the variance in anxiety was accounted for, and 56% of the variance in depression was accounted for in these analyses.

Interestingly, although parenting is assumed to have only an indirect effect on adult mental health, the Overprotection dimension of the PBI remained significant in the final stepwise model for anxiety, but not for depression. This is not to say that parenting is not an important risk factor for the onset of depression. The causal pathway to mental health or mental illness is a complex one, whereby environmental risks during childhood are clearly at the beginning of the pathway (Zubrick, Silburn, Burton, & Blair, 2000). The importance of early antecedents may not be recognized through statistical analyses with the inclusion of more proximal variables. Abramson et al. (1989) also make note of the added weight given to proximal variables in determining the onset of depression. A reasonable explanation as to why overprotection was significant in the final model for anxiety is that University students may still be somewhat under their parents' influence. Thus, the findings for this population of young adults may not generalize to older populations.
Presumably, once individuals become independent of their parents’ influence, parental variables have a less direct effect on students’ mental health.

In the final model of the stepwise regression analyses, emotion coping and self-efficacy were most strongly related to anxiety, followed by life events, parental overprotection, and daily hassles. In contrast, emotion coping and negative life events were most strongly related to depression, followed by self-efficacy. As was the case in the hierarchical analyses, daily hassles were not related to depression in models where emotion coping was included. These findings suggest that daily hassles may cause anxious arousal for individuals employing an emotional coping style. Already being in an anxious state when a more significant stressor occurs may increase an individual’s vulnerability to depression. Kendler, Neale, Kessler, Heath, and Eaves (1992), using a large twin sample, have also found that environmental risk factors differ for major depression and generalized anxiety disorder, suggesting that some environmental experiences are unique to each disorder. The shared variance due to both genetic and environmental risk factors in their study weakened the possibility of determining whether anxiety increased the risk for depression. Kendler and his colleagues (1992) also looked at familial environmental risk factors and found no evidence for an etiological role in either of the disorders. It is unknown how Kendler and his colleagues assessed environmental risk factors (familial or otherwise) in their study. Presumably they did not measure these factors directly, but rather looked at twins in relation to their shared environmental background.

Limitations

Care must obviously be taken in interpreting these results due to the high degree of correlations among the predictor variables. Future research addressing hypotheses for the
model (i.e., Figure 1) under consideration in the present study would benefit from employing structural-equations modeling (Tabachnick & Fidell, 1996). The sample for the present study, however, was not large enough (Tabachnick & Fidell, 1996). In order to test the connecting paths which included potential mediating and moderating variables, acceptable methods of analysis proposed by Baron and Kenny (1986) were utilized.

With the use of the PBI, a retrospective questionnaire, as the measure of parenting style there are inherent methodological limitations in the present study. Research on the PBI using twin samples has demonstrated, however, that it is a valid measure of actual parenting (Mackinnon, Henderson, & Andrews, 1991; Parker, 1986). A review of the literature on retrospective reports of early experience has suggested that assertions regarding their unreliability are exaggerated (Brewin, Andrews, & Gotlib, 1993). Brewin and colleagues (1993) looked at mood states, psychiatric status, and normal memory deficits in their review to assess potential limitations of retrospective reports. Recall was found to be unaffected by mood state or psychiatric illness (depression or anxiety). Longitudinal designs over an extensive period (i.e., from infancy to adulthood) looking at the parent-child relationship as well as other variables related to personality development would be necessary to study their complex relations and how they dispose to psychopathology. This would of course be a worthwhile, albeit costly, endeavour.

Additionally, the simplified use of the PBI in the present study (i.e., having participants rate both of their parents' attributes as a unit) did not allow for separate analyses of maternal and paternal patterns of care and protection. The present study explored new potential relations with the PBI (e.g., PBI and coping) and future research would benefit from collecting maternal and paternal parenting data separately to see if the
findings here hold true for both parents, especially with regard to coping measures. The correlation between care and overprotection, although still significant, was weaker than that observed in other studies (Mackinnon et al. 1993) which may have resulted from measuring parents' attributes as a unit.

Parker's suggestion to partial out the overlapping variance between the two parenting dimensions (to create orthogonal scales) was not done for the present study. Parenting was being studied in relation to the development of personality and coping styles and there were no strong a priori hypotheses regarding the relative importance of the two parenting dimensions or of any interaction of the two dimensions in predicting these two measures.

A limited male population of psychology students at Acadia University prohibited the analysis of gender differences in the present study. Gender differences have, however, been found in the depression, social support, and coping literature. Social support for example, has been found to be more important for women (Billings & Moos, 1984). Future research might benefit from obtaining a large enough male sample for such gender-specific analyses.

Concluding Remarks for Study 1 and Study 2

Depression and Anxiety

Examining the results of both studies presented indicates that a developmental perspective is viable in detecting a causal chain to adult depression and anxiety. Although depression was the main mental health outcome variable of concern here, the two studies add to the literature base which purports the importance of including both depression and anxiety when researching either. Twin studies have found evidence of a common genetic
risk factor for major depression and generalized anxiety disorder, wherein Kendler et al. (1992) concluded that the expression of genes through physiological pathways creates a diathesis that is common to both disorders. If there is indeed a degree of plasticity in early neurological development (Liu et al., 1997; Meaney et al., 1996), the individual differences in physiological reactivity to stress based on anomalous parenting styles (low care, high overprotection) found in Study 1 may signify an accessible pathway to prevent the onset of anxiety and depression.

Consistent with other research (Brown, Chorpita, & Barlow, 1998; Dobson, 1985; Kendler et al., 1992), anxiety and depression were highly correlated in the present study. Yet, the findings suggest that the two measures of mental health differ (albeit minimally) in their proximal antecedents to predict onset. The practical importance of this finding raises the importance of proper diagnoses in order to implement an appropriate treatment plan and the importance of identification of antecedents to facilitate prevention initiatives. Whether the two disorders share a common diathesis or anxiety acts as a risk factor for depression still needs to be determined. Their relation to coping and personality constructs suggest, however, that interventions aimed at developing social supports and self-efficacy as well as adaptive coping (increased task coping and decreased emotion coping) may be viable interventions for both disorders. Bandura (1988) has found strong evidence supporting the use of self-efficacy enhancement (especially aimed at coping efficacy in phobics) to regulate anxious arousal at both psychological and physiological levels. Bandura (1988) aptly uses a Chinese proverb to describe the relation of cognitive control and anxious arousal: "You cannot prevent the birds of worry and care from flying over your head. But you can stop them from building a nest in your head" (p. 89).
Future Directions

The potential for the PBI to help understand symptoms of depression and anxiety has been shown. The PBI’s observed relations with personality, coping and physiological reactivity support the need for additional research on the developmental pathways proposed here. Research agrees that early environmental experiences are linked to adult psychopathology. The physiological and psychological mechanisms that interact to determine mental health outcomes need further exploration within a developmental framework.

Research which can support a developmental causal pathway that leads to depression and anxiety may provide the needed support for research on prevention of psychopathology during early childhood with the hope of coming closer to policy changes involving prevention initiatives for mental health disorders. Indeed, Zubrick et al. (2000) point out that risk factors “may no longer be current by the time the patient sees a mental health practitioner and their control (if still current) may be irrelevant to treatment” (p. 573). In other words, what was done to bring on mental illness is not always important to its treatment. Once a person has reached the doors of the mental health practitioner, “prevention has failed” (Zubrick et al., 2000, p. 573). Reducing the prevalence of mental illness is the only measure of prevention.
References


Risk factors for depression and anxiety


**British Journal of Psychiatry.** 134, 138-147.


**Australian and New Zealand Journal of Psychiatry.** 13, 51-56.


Appendix A: Informed Consent for Introductory Psychology Students for Study 1

Acadia University - Department of Psychology
Psychosocial Stress Study
Investigators: Denise Hand B.Sc., Marilyn Roberts B.A., and Peter McLeod Ph.D.

We invite you to participate in an experimental study investigating physiological responsiveness and stress as part of the Masters theses of Denise Hand and Marilyn Roberts.

During this study, you will be asked to fill out several survey questionnaires and personality inventories, give blood pressure and heart-rate readings, and provide saliva samples for the purpose of hormonal analysis. This study also involves a psychosocial stressor that may be somewhat anxiety-producing and has been approved by the Acadia Research Ethics Board.

The duration of this study is approximately one hour and you will receive introductory psychology course credit for your participation. All information provided for the study will be kept confidential.

Finally, please note that participation in this experiment is entirely voluntary and you can discontinue participation in this study at any time without prejudice or penalty.

If any questions arise during or after participation in the study, they can be addressed to Marilyn Roberts or Denise Hand at 585-1490 and Peter McLeod at 585-1589.

Signature
I have read the explanation about this study. I have been given the opportunity to discuss it and my questions have been answered to my satisfaction. I hereby consent to take part in this study.

_________________________  _______________________
Signature of Participant    Date Signed

I further consent to be contacted in the fall of 2000 to be asked if I would like to volunteer for a follow-up study that will involve completing additional questionnaires and will take approximately thirty minutes. I understand that my signature does not oblige me in any way to participate in this follow-up.

_________________________  _______________________
Signature of Participant    Date Signed

If yes, please provide contact information (e.g., e-mail) below.
Appendix B: Informed Consent for Non-Introductory Psychology Students for Study 1

Acadia University - Department of Psychology
Psychosocial Stress Study
Investigators: Denise Hand B.Sc., Marilyn Roberts B.A., and Peter McLeod Ph.D.

We invite you to participate in an experimental study investigating physiological responsiveness and stress as part of the Masters theses of Denise Hand and Marilyn Roberts.

During this study, you will be asked to fill out several survey questionnaires and personality inventories, give blood pressure and heart-rate readings, and provide saliva samples for the purpose of hormonal analysis. This study also involves a psychosocial stressor that may be somewhat anxiety-producing and has been approved by the Acadia Research Ethics Board.

The duration of this study is approximately one hour and you will receive a payment of $10.00 for your participation. All information provided for the study will be kept confidential.

Finally, please note that participation in this experiment is entirely voluntary and you can discontinue participation in this study at any time without prejudice or penalty.

If any questions arise during or after participation in the study, they can be addressed to Marilyn Roberts or Denise Hand at 585-1490 and Peter McLeod at 585-1589.

Signature
I have read the explanation about this study. I have been given the opportunity to discuss it and my questions have been answered to my satisfaction. I hereby consent to take part in this study.

Signature of Participant Date Signed

I further consent to be contacted in the fall of 2000 to be asked if I would like to volunteer for a follow-up study that will involve completing additional questionnaires and will take approximately thirty minutes. I understand that my signature does not oblige me in any way to participate in this follow-up.

Signature of Participant Date Signed

If yes, please provide contact information (e.g., e-mail) below.
Appendix C: Debriefing Form for Study 1

The Influence of Control and Self-Efficacy on the Cardiovascular and Adrenocortical Response to a Psychosocial Stressor
Denise Hand, B.Sc., Marilyn Roberts, B.A., Peter McLeod, Ph.D.

Thank you for your participation in this study. The purpose of the study was to investigate the emotional and physiological response to psychosocial stress. The speech and arithmetic task you participated in was designed to be mildly anxiety-producing to elicit the body's natural stress response. One component of this response is an increase in certain stress hormones. The saliva samples you provided allow us to measure cortisol, one of these stress hormones. We also measured the cardiovascular stress response, through blood pressure and heart-rate readings. The videotape of your speech and mental arithmetic task will be erased after viewing and rating is complete.

During the study you completed several paper and pencil questionnaires about such topics as stress, depression, self-efficacy and anxiety. If in answering the questionnaires you have some concerns about yourself, please contact the free on-campus Counseling Centre located at Godfrey House, #3 Horton Avenue or phone 585-1246. We will be investigating how these variables are related to each other and to physiological reactivity to stress. Some of these variables, such as a well-developed sense of self-efficacy or self-competence, are associated with successful coping in stressful situations (Gattuso, Litt and Fitzgerald, 1992; Wiedenfeld et al., 1990).

Another purpose of this study was to assess the impact of control over the stressful situation. Participants were randomly assigned to a control or no-control condition. Participants in the control condition chose their speech topics, which provided a degree of control over the stressfulness of the task. Those in the no-control condition were assigned one of the same topics to speak about, and could not exercise this form of control. In previous research, groups given control over stress generally show a decreased stress response, and we hope to replicate this finding in the present study.

Finally, during the study you were asked several questions about such topics as medications, diet, previous illness and oral-contraceptive use. All of the variables we inquired about have been shown to alter either cardiovascular or hormonal stress reactivity. In order to control for these factors it was necessary to screen for them prior to participation.

Because of the nature of this research, it is very difficult to recruit participants who fit the many requirements. We would ask that you do not disclose the information about this study to other potential participants as this seriously jeopardizes the results of the study.

If you have any questions about your participation in the study or if you would like to know more about this topic please contact Denise Hand or Marilyn Roberts at 585-1490 or Peter McLeod at 585-1589.
Appendix D: Subjective Stress Rating Scale

How stressed does participant appear?

(Please circle one.)

1  2  3  4  5  6  7  8  9  10  11  12  13  14

does not look  looks somewhat  looks very
stressed        stressed        stressed
Appendix E: Global Rating of Subjective Stress

How stressful did you find the public speaking and mental arithmetic task?

(Please circle one.)

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<tr>
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<tr>
<td>stressful</td>
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Appendix F: Informed Consent/Debriefing for Returning Participants from Study 1:

Continuation in Study 1 (Time 2 Data Collection) and Participation in Study 2

Consent/Debriefing Form

ACADIA UNIVERSITY
DEPARTMENT OF PSYCHOLOGY
Stress, Coping and Mental Health Study
Investigators: Marilyn Roberts Del Re, BA (Graduate Student, MSc, Clinical Psychology) and Dr. Peter McLeod, PhD (Supervisor)

Approved by Acadia Research Ethics Board - Date: ______________

PARTICIPATION FOR NON-INTRODUCTORY PSYCHOLOGY STUDENTS
We invite you to take part in a research study at Acadia University. Taking part in this study is voluntary. Participating in the study might not benefit you, but we might learn things that will benefit others. You may withdraw from the study at any time without penalty. You will receive a chance to win $300 for your participation which you do not forfeit if you decide to withdraw at any time during the study. The study is described below. You should discuss any questions you have about this study with the people who explain it to you.

PURPOSE OF THE STUDY:
The purpose of the study is to explore factors that may determine coping styles for dealing with stress and their relations to emotional states.

WHO CAN PARTICIPATE IN THIS STUDY:
Participants in this study include students in attendance at Acadia University in Wolfville, NS.

DESCRIPTION OF THE STUDY:
The procedure of the study will consist of the completion of a set of paper-and-pencil questionnaires in a classroom at Horton Hall, Acadia University, Wolfville, Nova Scotia. The questionnaires will be administered in group format. You will be answering questions pertaining to stress you may have experienced, the way you cope with stress, the attitudes and behaviours of your parents/guardians as you remember them, your personality, and any feelings of anxiety or depression. This study is in no way experimental, therefore, there will be no manipulation with respect to the participants involved. The completion of questionnaires is expected to take approximately one hour, however, times will vary according to participant’s pace of responses to the questions.
Appendix F: Informed Consent/Debriefing for Returning Participants from Study 1:

Continuation in Study 1 (Time 2 Data Collection) and Participation in Study 2

POTENTIAL HARM:
During the completion of questionnaires you will be telling us of recent stressful experiences and current feelings of anxiety and/or depression. If you are sensitive to answering questions related to such experiences and feelings, you may find this upsetting as you may feel as though you are reliving a stressful event, and you may have concerns about yourself. Please inform me if you are experiencing any discomfort as you may discontinue at any time without penalty.

POSSIBLE BENEFITS:
We anticipate that your being able to tell us of your stressful life experiences may benefit your well being. We also anticipate that by you telling us of any stressful life experiences and any associated feelings, you may have a better understanding of yourself. Counseling is available if you feel the need to discuss any of your concerns. Please contact the free on-campus Counseling Centre located at Godfrey House, # 3 Horton Avenue or phone 585-1246.

CONFIDENTIALITY:
You will not be identified as a study participant in any reports or publications of this research. All identifying demographic information obtained from the questionnaires will be removed. Your records will be kept in a locked file cabinet. Only individuals directly involved in the research study will see them.

QUESTIONS OR PROBLEMS:
If you have any questions about the study, please contact: Marilyn Roberts Del Re - Phone: (902) 585-1490 or (902) 835-5514 or Peter McLeod - Phone: (902) 585-1589.

OTHER PERTINENT INFORMATION:
• You will be told about any new information which might affect your decision about being in this research study.
• Please retain a copy of this consent form for your own records.

CONSENT:
I have read the explanation about this study. I have been given the opportunity to discuss it and my questions have been answered to my satisfaction. I hereby consent to take part in this study.

__________________________________________
Signature of Participant

__________________________________________
Date Signed

Thank you for your participation.
Appendix G: Revisions to the Daily Hassles Scale

Items removed

1. Financial responsibility for someone who doesn’t live with you
2. Decisions about having children
3. Home maintenance (inside)
4. Concerns about retirement
5. Not enough money for health care
6. Difficulties with getting pregnant
7. Sexual problems other than those resulting from physical problems
8. Neighborhood deterioration
9. Financing children’s education
10. Problems with employees
11. Declining physical abilities
12. Problems with your children
13. Problems with persons younger than yourself
14. Difficulties seeing or hearing
15. Problems with divorce or separation
16. Property, investments or taxes
17. Yardwork or outside home maintenance
Appendix G: Revisions to the Daily Hassles Scale

<table>
<thead>
<tr>
<th>Items Reworded</th>
<th>Original Wording</th>
<th>New Wording</th>
</tr>
</thead>
<tbody>
<tr>
<td>Customers or clients give you a hard time</td>
<td>People giving you a hard time</td>
<td></td>
</tr>
<tr>
<td>Laid-off or out of work</td>
<td>Trouble finding employment</td>
<td></td>
</tr>
<tr>
<td>Concerns about bodily functions</td>
<td>Concerns about your body</td>
<td></td>
</tr>
<tr>
<td>Hassles from boss or supervisor</td>
<td>Hassles from boss, supervisor or professor</td>
<td></td>
</tr>
</tbody>
</table>
Appendix H: Informed Consent/Debriefing for Study 2: New Participants

Consent/Debriefing Form

ACADIA UNIVERSITY
DEPARTMENT OF PSYCHOLOGY
Stress, Coping and Mental Health Study
Investigators: Marilyn Roberts Del Re, B.A.(Graduate Student, MSc, Clinical Psychology) and Dr. Peter McLeod, Ph.D. (Supervisor)

Approved by Acadia Research Ethics Board - Date: ______________________

PARTICIPATION FOR PSYCHOLOGY STUDENTS
We invite you to take part in a research study at Acadia University. Taking part in this study is voluntary. Participating in the study might not benefit you, but we might learn things that will benefit others. You may withdraw from the study at any time without penalty. You will receive two course points for your participation, and you do not forfeit points if you decide to withdraw at any time during the study. The study is described below. You should discuss any questions you have about this study with the people who explain it to you.

PURPOSE OF THE STUDY:
The purpose of the study is to explore factors that may determine coping styles for dealing with stress and their relations to emotional states.

WHO CAN PARTICIPATE IN THIS STUDY:
Participants in this study include students in attendance at Acadia University in Wolfville, NS.

DESCRIPTION OF THE STUDY:
The procedure of the study will consist of the completion of a set of paper-and-pencil questionnaires in a classroom at Horton Hall, Acadia University, Wolfville, Nova Scotia. The questionnaires will be administered in group format. You will be answering questions pertaining to stress you may have experienced, the way you cope with stress, the attitudes and behaviours of your parents/guardians as you remember them, your personality, and any feelings of anxiety or depression. This study is in no way experimental, therefore, there will be no manipulation with respect to the participants involved. The completion of questionnaires is expected to take approximately one hour, however, times will vary according to participant’s pace of responses to the questions.

POTENTIAL HARM:
During the completion of questionnaires you will be telling us of recent stressful experiences and current feeling of anxiety and/or depression. If you are sensitive to answering questions related to such experiences and feelings, you may find this upsetting...
Appendix H: Informed Consent/Debriefing for Study 2: New Participants

as you may feel as though you are reliving a stressful event, and you may have concerns about yourself. Please inform me if you are experiencing any discomfort as you may discontinue at any time without penalty.

POSSIBLE BENEFITS:
We anticipate that your being able to tell us of your stressful life experiences may benefit your well being. We also anticipate that by you telling us of any stressful life experiences and any associated feelings, you may have a better understanding of yourself. Counseling is available if you feel the need to discuss any of your concerns. Please contact the free on-campus Counseling Centre located at Godfrey House, #3 Horton Avenue or phone 585-1246.

CONFIDENTIALITY:
You will not be identified as a study participant in any reports or publications of this research. All identifying demographic information obtained from the questionnaires will be removed. Your records will be kept in a locked file cabinet. Only the staff involved in the research study will see them.

QUESTIONS OR PROBLEMS:
If you have any questions about the study, please contact: Marilyn Roberts Del Re - Phone: (902) 585-1490 or (902) 835-5514 or Peter McLeod - Phone: (902) 585-1589.

OTHER PERTINENT INFORMATION:
- You will be told about any new information which might affect your decision about being in this research study.
- Please retain a copy of this consent form for your own records

CONSENT:
I have read the explanation about this study. I have been given the opportunity to discuss it and my questions have been answered to my satisfaction. I hereby consent to take part in this study.

________________________________________  ________________________________
Signature of Participant                        Date Signed