Emotion Regulation and Stress Reactivity in the Adolescent Daughters of Depressed Mothers

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Thesis submitted to the

Faculty of Graduate and Postdoctoral Studies

In partial fulfillment of the requirements

For the Ph.D. in Clinical Psychology

School of Psychology

Faculty of Social Sciences

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Abstract

The daughters of women with a history of depression are at heightened risk for a range of mental health problems. The present study investigated emotion regulation, cortisol reactivity to stress, and interpersonal competence as potential indicators of risk in adolescent girls at high versus low risk for depression. Participants were a community sample of 47 girls and their mothers (27 high risk and 20 low risk). Mothers and daughters had been interviewed to assess diagnostic history as part of a previous longitudinal study. In the current study, daughters completed the Trier Social Stress Test for Children (TSST-C) and cortisol samples were collected before and after exposure to this psychosocial stressor. Both mothers and daughters completed self-report questionnaires and daughters were re-assessed using the Depressive Disorders module of the Kiddie Schedule for Affective Disorders and Schizophrenia. High risk mothers were also interviewed to assess the timing and chronicity of their depressive episodes during their daughters' lifetime. High and low risk girls had equivalent ratings of self-reported stress following the TSST-C, but different physiological responses. Girls at high risk for depression showed a blunted cortisol response to the TSST-C whereas low risk girls showed a normal cortisol response. High risk status for depression predicted a blunted cortisol response to stress, which predicted difficulties with emotion regulation; difficulties with emotion regulation in turn predicted a greater number of self-reported depressive symptoms. These results suggest that maternal depression may act as a stressor that compromises stress-response system functioning in daughters and produces related difficulties with emotion regulation.

Acknowledgments

I would like to express my sincere thanks to my thesis supervisor, Dr. Valerie Whiffen, for the opportunity to be involved in her longitudinal research project and allowing me to recruit from this valuable sample of girls and their mothers. I would also like to thank her for her support and guidance in the completion of my thesis and throughout my years as a graduate student at the University of Ottawa. I also very much appreciated her willingness to continue to supervise my work from afar after moving from Ottawa to Vancouver.

I would also like to thank each of the other members of my thesis committee: Dr. Diana Koszycki for her encouragement and support while I was an undergraduate student, as well as for her helpful comments and suggestions throughout the completion of my thesis; Dr. Zul Merali for analyzing the cortisol samples obtained in this study and his assistance in discussing ideas for interpreting the results; and Dr. Elisa Romano for her support and helpful comments and ideas. I would also like to acknowledge the thoughtful review provided by my external examiner, Dr. Lisa Serbin from Concordia University.

There are several other individuals who contributed to this project that I would like to acknowledge. Sincere thanks to Dwayne Schindler for so generously being available to provide helpful consultative support regarding the statistical analyses for this project. Thanks also to the laboratory of Dr. Zul Merali, particularly Jon James, for analyzing the saliva samples and responding to my questions. I am grateful to each of the individuals who volunteered their time to assist with research sessions; Gillian Tibbetts in particular was invaluable in helping things to run smoothly. Dr. Alexandra Fiocco also deserves acknowledgment and thanks for her helpful consultations regarding the cortisol aspect of this project.

Finally, I would like thank my husband, Ian, and my family, especially my parents and brother, for their constant support and encouragement, and for always believing in me.

Emotion Regulation and Stress Reactivity in the Adolescent Daughters of Depressed Mothers

The children of depressed mothers evidence compromised functioning from the earliest stages of development through to adolescence (Goodman & Gotlib, 1999). Parental affective illness is associated with significantly increased rates of childhood-onset depression, anxiety disorder, and conduct disorder, as well as early-adult onset depression (Downey & Coyne, 1990; Tompson et al., 2010; Wickramaratne & Weissman, 1998). It is during adolescence that girls, regardless of parental diagnosis, exhibit a significant increase in depressive disorders compared to boys. By age 14, the gender gap in depression emerges, and by age 16 and 17, girls are more than twice as likely to have experienced an episode of depression compared to boys (Wade, Cairney, Pevalin, 2002). The rate of depression in adolescent daughters with one or more depressed parents is particularly striking: 43% of these girls become clinically depressed by the age of 19, compared to 16% of the adolescent sons (Wickramaratne & Weissman, 1998). A 20-year follow-up of this sample continued to show a significantly greater incidence of depression in the offspring of depressed compared to healthy parents, with peak age of first onset for girls being during adolesence (Weissman et al., 2006).

Depression in children and adolescents is associated with both short and long-term functional impairment, illness, and death (Neal, 2003). Longitudinal studies have repeatedly shown that depression in youth is predictive of depressive disorders in adulthood along with significant psychosocial impairment (Essau, Conradt, & Petermann, 1999). Women in particular are significantly and negatively affected by early-onset chronic major depressive disorder in terms of reduced educational achievement and lifetime earnings (Berndt, Koran,

Finkelstein, Gelenberg, Kornstein, Miller, et al., 2000). Therefore, focussing on early adolescent girls who are known to be at risk for depression is particularly important in order to determine how best to direct prevention and treatment efforts for this group.

The association between maternal depression and risk for adverse outcomes in offspring is well documented, and researchers have started to focus on the mediators and moderators underlying this risk (Goodman & Gotlib, 1999). Several mechanisms of risk have been proposed, including genetic predisposition, dysfunctional neuro-regulatory mechanisms, exposure to maladaptive cognitions, behaviours and affect from the depressed parent, and exposure to high levels of stress (Goodman & Gotlib, 1999). As noted by Whiffen (2005), the mediator with the most empirical support is the parent-child relationship. Moderators of risk also have been proposed, with the most frequently studied being the psychological health and involvement of the child's father (Whiffen, 2005). Other proposed moderators include the timing and chronicity of the mother's depression, and characteristics of the child such as temperament, gender, cognitive ability, and social skills (Goodman & Gotlib, 1999). These mediating and moderating variables often have been studied in isolation. Consequently, the present study aimed to take an integrative approach focussed on the interrelations among emotional, psychobiological, and interpersonal factors. Specifically, emotion regulation, stress reactivity, and interpersonal competence were investigated and compared in adolescent girls of mothers with and without a history of depression. The emphasis was be placed on how each of these factors relate to each other and how difficulties in these areas may increase risk for adverse outcomes in the adolescent daughters of mothers with a history of depression. The timing and chronicity of the mothers' depression, an important but understudied moderator of risk, was also examined in

relation to daughters' emotion regulation, stress reactivity, and interpersonal competence.

Emotion Regulation

Although there is no single definition of emotion regulation, it is often conceived as the ability to flexibly manage or modulate emotional arousal in order to accomplish goals and to control the expression of emotion in ways that are socially adaptive (Bradley, 2000; Cichetti, Ganiban, & Barnett, 1991; Calkins, 1994; Cole, Michel, & Teti, 1994; Thompson, 1994). The development of emotion regulation is an important task in early life and has far-reaching implications in terms of social competence with peers and one's ability to handle stress (Bell & Calkins, 2000; Eisenberg, Fabes, & Losoya, 1997; Gross, 2002; Martin & Dahlen, 2005; McCarthy et al, 2006): two factors that may be protective for girls at risk for depression. Several studies have shown that the ability to regulate emotion is linked with better social functioning in samples ranging in age from the toddler years through to adulthood.

Calkins, Gill, Johnson, and Smith (1999) showed that toddlers who became distressed in response to a frustrating laboratory task and who were observed to engage in less effective emotion regulation were more likely to have conflicted interactions with their peers. Longitudinal studies with children of preschool age have found that poor emotion regulation is linked with peer rejection over time (Maszk, Eisenberg, & Guthrie, 1999) and to both current and kindergarten social competence (Denham et al., 2003). The emotion regulation abilities of school-aged children also has been linked to social competence with peers (e.g., McDowell, Kim, O'Neil, & Parke, 2002; Rydell, Thorell, & Bohrin, 2007), with some studies showing that this association is particularly strong for children high on negative emotionality (Contreras, Kerns, Weimer, Gentzler, & Tomich, 2000). Similar results have

been found with adolescent populations, with dysfunctional emotion regulation linked with greater problems with peers (Phillips & Power, 2007).

Studies of young adults have repeatedly shown an association between emotion regulation and social competence. Gross and John (2003) studied two commonly used emotion regulation strategies: cognitive reappraisal, defined as viewing a situation in a way that alters its emotional impact, and expressive suppression, defined as suppressing the expression of one's emotions. In their sample of undergraduate participants, Gross and John (2003) found that reappraisers were rated by their peers as having closer relationships and as better liked. Emotion regulation via reappraisal also was associated with greater social sharing of emotions. In contrast, individuals who attempted to regulate their emotions via suppression reported greater discomfort with closeness and sharing in close relationships, and their peers rated them has having less close relationships. Although they were not disliked by others, they reported experiencing lower levels of social support. Not surprisingly, suppression also was associated with less sharing of both positive and negative emotions with others (Gross & John, 2003).

Other researchers have found that the use of effective emotion regulation strategies is linked with more positive interpersonal relationships, less negative interactions, more reciprocal friendship nominations, and more positive peer nominations in samples of young adults (Lopes, Salovey, Strauss, 2003; Lopes et al., 2004; Lopes, Salovey, Cote, & Beers, 2005). Emotion regulation abilities also have been found to be correlated with both self- and peer-reports of interpersonal sensitivity and prosocial behaviour (Lopes, Salovely, Cote, & Beers (2005), and with peer reports of skill at providing emotional support (Lopes et al., 2004). In a recent report, Rivers, Brackett, Katulak, and Salovey (2007) looked specifically

at the regulation of two emotions, anger and sadness, in a sample of young adults. The effective regulation of both emotions was found to be associated with good social functioning. Specifically, the effective management of anger was related to a positive conflict resolution style that was likely to help maintain relationships, whereas the ability to effectively regulate sadness was associated with the presence of fulfilling relationships characterized by trust (Rivers et al., 2007).

The ability to successfully regulate one's emotions has implications not only for interpersonal competence but also for general well-being and coping with stress. In large samples of young adults, mood regulation expectancies (i.e., the belief that one can lessen a negative mood state by engaging in certain behaviours or thought processes) are negatively associated with indicators of self-reported stress and stress-related emotions such as hopelessness (McCarthy, Lambert, & Moller, 2006). Martin and Dahlen (2005) assessed the use of cognitive emotion regulation strategies among young adults and found that less effective strategies such as rumination, low positive appraisal, and self blame were associated with greater self-reported stress levels. The use of ineffective strategies also was associated with greater emotional distress, including higher levels of depressive symptoms, anxiety, and anger (Martin & Dahlen, 2005).

Poor emotion regulation capabilities may play a role in the development of psychopathology (Bradley, 2000). Difficulties regulating negative affect and maintaining positive affect have been implicated in depressive disorders specifically (Gross & Munoz, 1995). Individuals with greater depressive symptoms have been shown to have less confidence in their ability to effectively regulate their negative mood states (Flett, Bankstein, & Obertynski, 1996). Recent cross-sectional research with a community sample of

adolescents supported the link between the ineffective regulation of negative emotions and self-reported depressive symptomatology (Silk, Steinberg, & Sheffield Morris, 2003). In addition, studies investigating cognitive emotion regulation have shown that adolescents with greater depressive symptoms are more likely to endorse the use of less effective cognitive emotion regulation strategies (Garnefski, Kraaij, & Spinhoven, 2001; Garnefski et al., 2002; Garnefski, Boon & Kraaij, 2003; Kraiij et al., 2003). Depressive symptoms in adolescents also are linked with greater use of expressive suppression and the less frequent use of cognitive reappraisal (Betts et al., 2009). Consistent with the use of less effective emotion regulation strategies, clinically depressed adolescents as well as those with high levels of self-reported depressive symptoms have more difficulty moving out of negative affective states than their non-depressed counterparts (Reijntjes et al., 2009; Sheeber, Allen, Davis, & Sorensen, 2000)

Parenting factors have been investigated in relation to children's and adolescents' emotion regulation abilities. Yap, Allen, & Ladouceur (2008) studied mother-adolescent pairs during an event planning and a problem-solving interaction task. Adolescents whose positive affect was invalidated or dampened by their mothers displayed more emotionally dysregulated behaviour and reported greater use of less effective emotion regulation strategies. In addition, they reported higher levels of depressive symptoms. Interestingly, adolescents' use of ineffective emotion regulation strategies mediated the association between maternal invalidation of positive affect and adolescents' depressive symptoms (Yap, Allen, and Ladouceur, 2008). The effects of parenting factors on offspring emotion regulation also have been demonstrated in a recent longitudinal study. Feng et al. (2009) examined mothers and daughters in a conflict resolution task. Daughters who expressed less

positive emotion at age 9 tended to have greater depressive symptoms at age 10, but higher levels of parental psychological control strengthened this association. In addition, maternal reports about their daughters' sadness regulation were correlated with age 10 depressive symptoms, such that lower levels of sadness regulation were linked with greater subsequent depressive symptoms when the girl perceived her parent as more rejecting. These results suggest that problems with emotion regulation precede the development of later depressive symptoms, and that parenting factors may moderate this link (Feng et al., 2009).

If difficulties establishing healthy emotion regulation represent an important vulnerability factor for later psychopathology among children of depressed parents, the interactions between parents and their children likely are the most important mechanism for potential disruptions in emotion regulation abilities (Silk, Shaw, Skuban, Oland, and Kovacs, 2006; Yap, 2007). Parents use modeling and coaching to facilitate the socialization of emotional expression and regulation in their children (e.g., Denham et al., 1997; Denham & Grout, 1993; Gottman, 2001). In a longitudinal study using a community sample, Gottman, Katz, and Hooven (1996) examined parenting behaviour in relation to children's emotion regulation abilities and developmental outcomes, beginning when the children were age 5 and ending when they were 8 years old. The researchers found that parents' validation of and sensitivity to their children's negative emotions as well as their involvement in coaching their child to recognize and cope with these emotions was linked to children's improved emotion regulation capabilities, social competence with peers, and greater academic achievement and physical health over time. In contrast to these "emotion-coaching" parents (Gottman, Katz, & Hooven, 1996, p. 244), parents with a history of depression are likely to experience difficulty in helping their children to recognize and manage negative emotions

such as sadness and anger.

Depressed individuals are characterized by difficulties modulating their own negative affect, and become overwhelmed by negative thoughts and sad mood (Gross & Munoz, 1995). Recovered depressed individuals also report greater difficulty regulating negative affect, and endorse the use of more maladaptive strategies such as rumination and catastrophizing, and less frequent use of the more effective strategy of "putting things into perspective" (Ehring, Fischer, Schnulle, Bosterling, & Tuschen-Caffier, 2008). Interestingly, recovered depressed individuals also report a greater lack of acceptance of negative emotions (Ehring et al., 2008). Consistent with this finding, research indicates that depressed mothers may have difficulty providing a healthy model of emotion regulation and expression (e.g., Cohn & Tronick, 1989; Field, 1995; Field 1994). In fact, depressed mothers' interactions with their children tend to be characterized by hostility, negativity, and diminished emotional communication (Lovejoy, Graczyk, O'Hare, & George, 2000; Cichhetti, Ganiban, & Barnett, 1991) which may present a significant challenge to the healthy development of children's ability to regulate their emotions (e.g., Ashman & Dawson, 2002; Tronick & Gianino, 1986).

In response to their children's negative emotions, depressed mothers respond with less supportive and problem-solving behaviour, and with higher levels of directiveness (Garber, Braafladt, & Zeman, 1991). Based on self-report and observational data, Shaw et al. (2008) showed that mothers with childhood-onset depression (i.e., depression with first onset during the mothers' childhood) are more likely to neglect their children's expressions of sadness or fear and to respond with less support to the expression of these emotions. These results remained even after accounting for current levels of maternal depressive

symptoms (Shaw et al., 2008). In a study of preschool-aged children and their mothers, Hoffman, Crnic, and Baker (2006) found that maternal depressive symptoms were associated with less effective emotional scaffolding, which was defined as the mother's ability to be sensitive to their child's emotional state, to accept and value their child's attempts at challenging and undesirable tasks (i.e., problem solving and putting away toys, respectively – both considered to increase the child's need to regulate their emotion) and to make comments that contribute toward the child's sense of self-efficacy. Less effective scaffolding was in turn related to children's greater emotional and behavioural dysregulation (Hoffman, Crnic, & Baker, 2006). Structure and guidance in emotional situations fosters children's emotion regulation capabilities (Southam-Gerow & Kendall, 2002; Gottman, Katz, & Hooven, 1996; Gottman, 2001). Thus, the high levels of negative emotion expressed by depressed parents combined with their low levels of supportive and problem-solving behaviour are likely to result in their children developing ineffective emotion regulation strategies. However, few studies have directly investigated the emotion regulation skills of children of clinically depressed parents, and most have focused on very young children.

Maughan, Cicchetti, Toth, and Togosch (2007) found that early occurring maternal depression (i.e., depression that occurred during the first 21 months of their child's life) was associated with children's dysregulated emotion patterns at age 4. Garber, Braafladt, and Zeman (1991) found that the 8- to 13-year old children of clinically depressed mothers generated fewer strategies for regulating their own and another person's negative affect, and they reported having less confidence in the efficacy of their proposed strategies. Silk et al. (2006a) examined the emotion regulation strategies used by the 4- to 7-year old offspring of

mothers with childhood-onset depression and the children of never-depressed mothers. Consistent with Maughan et al. (2007) and Garber et al. (1991), the children of childhood-onset depressed mothers exhibited difficulty in the cognitive and behavioural domains of emotion regulation. Specifically, during a laboratory delay task, the children of the childhood-onset depressed mothers engaged in more passive and less active strategies for regulating their emotions compared to children of never-depressed mothers. In a related study using the same delay paradigm, Silk, Shaw, Forbes, Lane, and Kovacs (2006b) found that the child's ability to generate positive affect in the face of frustration moderated the effects of maternal childhood-onset depression on children's internalizing problems, particularly when mothers had currently elevated levels of depressive symptoms.

Using a partially overlapping sample from Silk et al. (2006 a,b), Santucci et al. (2008) examined vagal tone and two dimensions of temperament in relation to emotion regulation in the 4- to 7-year old offspring of mothers with and without a history of childhood onset depression. Although children's lower vagal recovery and higher levels of negative affect were associated with maladaptive emotion regulation in response to frustration, there were no group differences based on maternal depression status. Blandon, Calkins, Keane, and O'Brien (2008) assessed mothers and their children across time from the ages of 4 to 7 and found that maternal depressive symptoms were associated with compromised emotion regulation development, and in contrast to Santucci et al. (2008), they found that children's capacity for physiological regulation acted as a buffer against some of the negative effects associated with maternal depressive symptoms.

In summary, emotion regulation is related to interpersonal competence and one's ability to cope with stress. Difficulty with emotion regulation may be a risk factor for the

development of psychopathology, and depression specifically. Poor emotion regulation appears to precede the development of depressive symptoms in children, and parenting factors appear to play a role in this association. Research suggests that mothers with a history of depression may have difficulty helping their children to develop healthy emotion regulation capabilities. Depressed and recovered depressed individuals struggle with regulating their own negative affect and have difficulty accepting their negative emotions. Not surprisingly, they also respond to their children's negative affect with less supportive and fewer problem solving behaviours. Consistent with this result, research suggests that the children of depressed mothers are more likely than the children of healthy parents to engage in ineffective emotion regulation strategies.

The HPA Axis & Cortisol

A physiological system closely linked to emotion regulation is the hypothalamic-pituitary-adrenal (HPA) axis (Stansbury & Gunnar, 1994). The HPA axis is comprised of the hypothalamus, pituitary gland, and the adrenal glands, and it plays a central role in an organism's reaction to stress (Anisman & Merali, 1999). Following the perception or experience of threat (i.e., stress), the hypothalamus releases a 41-amino acid peptide called corticotropin-releasing factor (CRF) along with the neuropeptide arginine vasopressin (AVP). CRF and AVP act together to enhance the release of adrenocorticotropic hormone (ACTH) from the pituitary gland (Claes, 2004; Michelson, Licinio, & Gold, 1995). ACTH travels through the bloodstream to the adrenal glands where it activates the release of glucocorticoids. Glucocorticoids, such as cortisol, function as negative feedback regulators of the HPA axis. Their presence signals the hypothalamus and pituitary to reduce CRF and ACTH secretion, which in turn prevents the over-activation of the stress response. Under

conditions of stress, this negative feedback is accomplished primarily through glucocorticoid receptors in the hippocampus, hypothalamus, and pituitary (Anisman & Merali, 1999; Claes 2004; McEwen & Schmeck, 1994). Cortisol is the main glucocorticoid in humans (King & Hegadoren, 2002), and therefore plays a critical role in concluding the stress response and protecting the body and brain from excessive exposure to stress hormones (Tsigos & Chrousos, 1998).

Dysregulation of the HPA axis and its primary hormonal product, cortisol, has been implicated in the depressive disorders. A substantial proportion of depressed adults have been shown to exhibit basal hypercortisolism (Ehlert, Gaab, & Heinrichs, 2001; Plotsky, Owens, & Nemeroff, 1998). Blunted cortisol reactivity to psychological stress, combined with impaired recovery from stress, also has been shown in some patients (Burke, Davis, Otte, & Mohr, 2005). The observation of disturbed functioning of the stress response system among individuals with depression has lead to a focus on biological factors as potential mechanisms of risk in vulnerable populations (Meyer, Chrousos, & Gold, 2001). Two longitudinal studies, one with adults (Harris et al., 2000) and one with adolescents (Goodyer, Herbert, Tamplin, & Altham, 2000), demonstrated that elevated levels of morning cortisol were predictive of subsequent depression in those at risk for depression, with risk defined by psychosocial factors. In the Harris et al. (2000) study, psychosocial risk factors for depression included low self-esteem, negative interactions with a significant other, and chronic subclinical levels of depression or anxiety. Parental psychopathology, the presence of recent stressful life events, exposure to current marital problems or past breakdown, at least two significant loss events, and high emotionality defined the high risk group in the Goodyer, Herbert, Tamplin, & Altham (2000) study. The former authors proposed that

exposure to persistently high cortisol levels may heighten risk for depression by making the individual more susceptible to the adverse effects of stress.

Family studies have suggested that those at genetic risk for depression show altered HPA axis functioning (Holsboer Lauer, Schreiber, & Krieg, 1995; Mannie, Harmer, & Cowen, 20007; Wichers et al. 2008), although not in all reports (Young, Aggen, Prescott, & Kendler, 2000). In a small sample of monozygotic twin pairs, no differences in morning and evening cortisol levels were found between individuals with and without a depressed co-twin (Young et al., 2000). However, in a considerably larger sample, Wichers et al. (2008) showed that individuals whose co-twins have a history of depression displayed different diurnal cortisol profiles. Specifically, these individuals had higher cortisol levels at several time points during the day compared to individuals whose co-twins had no history of depression. Furthermore, Mannie et al. (2007) reported that young people whose parents had a history of depression exhibited higher waking salivary cortisol levels than those whose parents had no history of depression. Similarly, Holsboer et al. (1995) found that the first degree relatives of depressed individuals had baseline cortisol levels and cortisol responses to a combined dexamethasone/corticotropin releasing hormone test that fell between those of a control group and those of depressed patients. This pattern was replicated in a four year follow-up study of the same sample (Modell et al., 1998). Collectively, these results suggest that HPA axis dysfunction is a potential risk factor for depression (Holsboer et al., 1995).

Early adverse experiences also have been implicated in dysregulation of the HPA axis. Research with animals has shown that exposure to a stressful environment during early life, a time of neuronal plasticity and critical developmental processes, can lead to significant and long lasting behavioural abnormalities and HPA axis dysregulation (Penza, Heim, &

Nemeroff, 2003). In rats, prolonged separation from the mother during the first few weeks of life results in increases in corticosterone levels, which is the rodent equivalent to cortisol, sensitization of the HPA axis to subsequent stressors, and to concomitant changes in related neurocircuits involved in the stress response (Penza, Heim, & Nemeroff, 2003). For example, McCormick, Kehoe, and Kovacs (1998) found that rat pups that experienced one hour of maternal deprivation per day from the second to eighth day following birth exhibited increased corticosterone response to further separation on postnatal day nine compared to control rats. Thus, HPA axis sensitization can occur immediately following exposure to early life stress. Other researchers have demonstrated that these sensitization effects can last into adulthood. Rat pups exposed to maternal separation for three hours daily during the postnatal period show greater corticosterone responses to stressors during adulthood compared to control rats (e.g., Aisa, Todera, Lahseras, Del Rio, & Ramirez, 2007; Parfitt et al., 2004; Plotsky & Meaney, 1993). Similar findings have been reported for adult rats whose mothers naturally provided lower levels of maternal care during their early life (Liu et al., 1997).

The increased sensitivity of the stress response that occurs following exposure to early life stress is accompanied by a number of related changes, such as elevated levels of hypothalamic CRF mRNA (Aisa et al., 2007; Plotsky & Meaney, 1993) and lower glucocorticoid receptor density in the hippocampus (Aisa et al., 2007). A review by Heim, Plotsky, and Nemeroff (2004) described additional central nervous system changes underlying the heightened responses to stress in offspring exposed to early life stress: sensitization of CRF neurons in the hypothalamic and limbic areas, increased mineralocorticoid receptors in the hippocampus, reduced mossy fibre development and

neurogenesis in the hippocampus, increased norepinephrine activity in the locus coeruleus, reduced central benzodiazepine receptor binding, decreased oxytocin receptor binding in females, and lower concentrations of neuropeptide Y in certain areas of the brain (Heim, Plotsky, & Nemeroff, 2004). Overall, the research in this area has suggested that maternal behaviour in early life can "program" basic, biological responses to stress in offspring and lead to changes in various neurocircuits involved in the stress response (Liu et al., 1997, p. 1661; Heim, Plotsky, & Nemeroff, 2004; Lupien, McEwan, Gunnar, & Heim, 2009). High levels of maternal care can regulate offspring physiology and reduce their response to stress in later life, while neglect or low levels of care can increase stress reactivity and have consequences extending into adulthood (Liu et al., 1997; Meaney, 2001; Weaver et al., 2004).

Interestingly, the long-term behavioural effects of prolonged maternal separation are consistent with depressive and anxiety disorder symptoms (Nemeroff, 2004). Following prolonged maternal separation during the postnatal period, depressive-like behaviour has been noted in rats and mice on the forced swim test in adolescence (Macri & Laviola, 2004) and adulthood (Aisa et al., 2007). In some cases, this effect was seen only for those rats that experienced additional chronic stress later in life (Marais, van Rensburg, van Zyl, Stein, & Daniels, 2008). Reduced social motivation also has been observed in adolescence and adulthood (Macri & Laviola, 2004; Reudi-Bettschen et al., 2006). Other researchers have noted signs of anhedonia, such as reduced intake of sweetened fluids and lower motivation to obtain a gustatory reward (unpublished data cited in Nemeroff, 2004; Reudi-Bettschen et al., 2006). Cognitive deficits and greater anxiety-like behaviour in adulthood also have been observed (Aisa et al., 2007; Daniel, Pieterson, Carstens, & Setin, 2004; Kalinichev et al.,

2002). Glucocorticoids have been shown both to disrupt HPA axis functioning and to increase depression-like behaviour in rats in a dose-dependent manner (Johnson, Fournier, & Kalynchuk, 2006), which suggests that high levels of cortisol may play a role in the development of depressive symptoms in humans (Johnson, Fournier, & Kalynchuk, 2006).

Findings are less consistent for non-human primates exposed to early life stress, with some studies showing higher and others showing lower basal cortisol levels (Sanchez et al 2001). Of those reporting on stress reactivity, there is some evidence that a blunted cortisol response to later stress follows a period of repeated maternal separations (Sanchez et al., 2001). Both hypo- and hyper-responsivity of the HPA axis are believed to have serious repercussions for one's health and development (Sanchez et al., 2001).

Human studies of exposure to early adversity have yielded generally comparable findings, with inadequate parenting being the primary early life stressor linked to risk for future depression (Goodman, 2002). However, limited data exist on stress responses among the offspring of depressed mothers, with the majority of studies having focussed on infants and young children. Newborn children of prenatally depressed mothers exhibit higher cortisol levels than infants of well mothers (Lundy et al., 1999; Field et al., 2004; Diego et al., 2004). Maternal depression during pregnancy also is associated with significantly greater reactivity (Davis et al., 2007) and negative affect (Huot et al. 2004) in their infants. These temperamental features are linked to greater cortisol responses to mild stress (Huot et al., 2004). In contrast, Azar et al. (2007) did not find a correlation between prenatal depression and infant cortisol levels. However, their sample of prenatally depressed women was quite small, and they did find that mothers with a lifetime history of depression had infants who responded with greater cortisol reactivity to a mild laboratory stressor (Azar et

al. 2007).

Dawson and Ashman (2000) noted that exposure to maternal depression early in life may represent a significant source of stress for the infant. Specifically, they observed that depressed mothers' interaction styles are characterized by irritability and insensitivity. The depressed mother may be unable to assist her child in regulating or soothing distress and her behaviour may be somewhat unpredictable as a result of the episodic nature of depressed mood. When maternal depression occurs early in the child's life, it may in turn have implications for the development of the child's HPA axis (Dawson & Ashman, 2000). Consistent with this hypothesis, in a longitudinal study of the offspring of depressed mothers, maternal depression during the child's first two years of life was the best predictor of elevated baseline cortisol levels at age 7 (Ashman, Dawson, Panagiotides, Yamada, & Wilkinson, 2002). However, while children with internalizing symptoms showed a greater cortisol response to a mild laboratory stressor, a maternal history of depression did not predict stress reactivity (Ashman et al., 2002).

In a sample of four-and-a half-year-old children of currently depressed mothers, baseline cortisol levels were found to be elevated only among those children who also were exposed to maternal depression during infancy (Essex, Klein, Cho, & Kalin, 2002). Other studies have shown that exposure to maternal depressive symptoms early in life is associated with higher baseline cortisol in toddlers (Bugental, Martorell, & Barraza, 2003). In a more recent study, Brennan et al. (2008) found that lifetime maternal depression predicted baseline and mean cortisol levels in 6-month old infants, but not cortisol reactivity to stress. In contrast, maternal depression in the peripartum period (i.e., prenatal or postnatal) predicted higher stress reactivity in infants, as did maternal depression comorbid with an anxiety

disorder. Feldman et al. (2009) also showed that the 9-month old infants of mothers with postpartum depression had higher baseline cortisol levels as well as greater cortisol reactivity to a laboratory fear paradigm compared to controls. However, the results were similar among the offspring of mothers with an anxiety disorder, which suggests that elevated baseline cortisol levels and reactivity are not specific to maternal depression. Interestingly, this study also found that depressed mothers were observed to be less sensitive and more withdrawn when interacting with their infants than both mothers with anxiety disorders and control mothers.

Few studies have considered the impact of maternal depression on HPA axis functioning in school-aged children. Lupien et al. (2000) reported that current maternal depressive symptoms were associated with significantly higher basal cortisol levels in offspring ranging in age from 6 to 10 years. Two studies assessed cortisol responses to challenge in children of depressed parents. Young, Vazquez, Jiang, and Pfeffer (2006) examined basal cortisol and cortisol response to a dexamethasone suppression test in offspring of parents with a history of depression compared to the offspring of healthy parents. The majority of parents with lifetime and current depression were mothers. Young et al. (2006) found that the offspring of parents with a history of depression had higher cortisol levels than the offspring of well parents, both basally and after administration of dexamethasone. However, these findings were driven largely by children whose parents were currently experiencing depression (Young et al., 2006). Gotlib, Joorman, Minor, and Hallmayer (2008) exposed girls of depressed versus healthy mothers to a psychosocial stressor and assessed cortisol levels, but the high and low risk groups were not compared; rather girls were grouped and compared by genotype. This study showed that girls who are

homozygous for the short allele in the promoter region of the serotonin transporter gene, a characteristic linked with higher rates of depression, tended to have greater cortisol reactivity to a stressor. However, the authors did not specifically address the impact of maternal depression on daughters' stress reactivity. More recently, Gump et al. (2009) showed that school-aged children of mothers reporting chronically elevated depressive symptoms over the child's lifetime had lower baseline cortisol levels, as assessed via a single measurement prior to a laboratory stressor. Although no association was found between maternal depressive symptoms and cortisol reactivity to the stressor, the task did not elicit a significant rise in cortisol for the sample as a whole (Gump et al., 2009).

Very few studies have examined cortisol levels in the adolescent offspring of depressed mothers. In a longitudinal study of children of postnatally depressed and well mothers, Halligan, Herbert, Goodyer, and Murray (2004) found that maternal postnatal depression was associated with higher morning cortisol levels in 13-year-old adolescent offspring. A follow-up study of this sample showed that higher morning cortisol levels predicted depressive symptoms in the adolescents 3 years later, and explained the association between maternal postnatal depression and adolescent depressive symptoms at age 16 (Halligan et al., 2007). Findings of disturbed cortisol levels were not replicated in one longitudinal study (Ronsaville et al., 2006). However, those offspring whose mothers also had a comorbid diagnosis of avoidant personality disorder had higher ACTH levels but similar cortisol responses to a corticotropin-releasing hormone (CRH) stimulation test, which the authors suggested may reflect hypersensitivity of the pituitary to CRH and hypo-responsivity of the adrenal.

In summary, most of the research investigating maternal depression and HPA axis

dysregulation in offspring has focused on infants and young children. This research has repeatedly shown that newborns of prenatally depressed mothers exhibit higher cortisol levels and more negative affect than newborns of well mothers. Support also has been found for the hypothesis that exposure to maternal depression early in life is associated with higher baseline cortisol levels in children. At least one study, however, has demonstrated lower baseline cortisol levels in offspring of chronically depressed mothers. Results have been less consistent with regard to cortisol reactivity to stress in children of depressed parents, with two studies showing no association and three showing greater cortisol reactivity to stress among children of depressed parents. Of those studies showing an association between parental depression and stress reactivity in offspring, the timing of parental depression may be important. While Young et al. (2006) and Azar et al. (2007) found that lifetime depression in parents was associated with children's greater stress reactivity, Brennan et al. (2008) found that only prenatally and/or postnatally depressed mothers had children with greater stress reactivity.

It is unclear why these discrepancies exist, but methodological differences may play a role, such as differences in the ages of the participants and the stress paradigm used. The two studies that evaluated the adolescent children of depressed mothers produced conflicting results. Given the limited and inconsistent findings to date, further research is needed to examine the stress response systems of adolescent offspring of depressed mothers. In light of the evidence from the animal literature on the effects of stress in early life, the timing of the mother's depression may account for some of the discrepant findings.

Interpersonal Relationship Functioning

Depressed individuals experience significant disturbances in their relationships

(Hammen, 2006; Joiner & Coyne, 1999), which in turn can have deleterious consequences for securing social support and managing stress (Segrin & Abramson, 1994). Depressed individuals behave in ways that tend to elicit negative reactions from significant others over time (Coyne, 1976; Joiner, 1999; Marcus & Nardone, 1992). Among adults, depression is consistently linked with disturbances in marital and family relationships (Joiner & Coyne, 1999). Adolescents with depressive symptoms also are more likely to have interpersonal problems, including less social participation, difficulties in close friendships and romantic relationships, lower perceived social competence, and higher levels of interpersonal stress (Cole, 1990; Daley & Hammen, 2002; Davila, Hammen, Burge, Paley, & Daley, 1995; La Greca & Moore Harrison, 2005; Mufson, Weissman, Moreau, & Garfinkel, 1999). Adolescents with early onset, recurrent depression have particularly poor social functioning (Hammen, Brennan, Kennan-Miller, & Herr, 2008). Negative interpersonal characteristics (i.e., low interpersonal mastery, a domineering interpersonal style, social isolation, and greater attachment fears) also adversely affect the speed of recovery from major depression among individuals participating in psychotherapy (Comninos & Grenyer, 2007).

Studies also have shown that interpersonal competence can predict depressive symptoms. For example, longitudinal studies of children and adolescents have shown that those with lower self-reported social competence tend to experience increases in depressive symptoms over time (Cole, Jacquez, & Maaschman, 2001; Wierzbicki & McCabe, 1988). Adolescent girls with lower self-reported interpersonal competence and problem-solving skills experience higher levels of chronic interpersonal stress up to one year later (Davila, Hammen, Burge, Paley, & Daley, 1995; Herzberg, Hammen, Burge, Daley, Davila, & Lindberg, 1998). Herzberg et al. (1998) found that difficulty providing emotional support to

others specifically was the strongest interpersonal predictor of later interpersonal stress. They speculated that individuals who lack the ability to listen empathically and to offer helpful advice may experience heightened interpersonal stress by increasing the likelihood of conflict with those who seek support as well as the withdrawal of those who might have otherwise provided support (Herzberg et al., 1998).

Individuals with difficulties in the area of interpersonal competence may experience more frequent interpersonal challenges and handle them poorly, which in turn may increase their risk for emotional difficulties (Van Orden, Wingate, Gordon, & Joiner, 2005). Poor social skills in fact have been shown to interact with stressful life events to predict increases in depression among adolescents (Segrin & Flora, 2000). In a sample of young women, Eberhart and Hammen (2006) showed that interpersonal problems contributed to the development of depression in those women with no prior history of mood disturbance. Specifically, lower family relationship quality and anxious attachment were found to predict the onset of depressive episodes over a 2-year period. Several other interpersonal factors, including poor peer and family relationships, discomfort with closeness, and difficulty depending on others, predicted depressive symptoms over a 6-month period (Eberhart & Hammen, 2006).

The depressogenic effects of interpersonal difficulties may be particularly relevant to adolescent girls. From adolescence onwards, the confiding and supportive aspects of friendships take on greater importance and appear to be especially meaningful to girls (Berndt, 1982). As shown by Buhrmester (1990), increasing intimacy in adolescent friendships tends to be coupled with an increase in the importance of intimate friendships to emotional adjustment. According to self- and friend- reports, adolescents whose friendships

were rated as companionate, disclosing, and satisfying reported better adjustment, including lower levels of depression, anxiety, and hostility as well as greater interpersonal competence and sociability (Burhmester, 1990). The interpersonal competencies required for adolescent peer relationships begin to resemble those required in adult relationships, including knowledge of when and how to disclose personal information, skill in appropriately providing emotional support to friends, as well as the ability to honestly express opinions and manage interpersonal conflict (Buhrmester, 1990). Problems in the area of interpersonal functioning with peers are expected to be related to the development of psychopathology (Bukowski & Adams, 2005). Interestingly, the increased importance that girls place on interpersonal relationships during adolescence has been proposed as one possible reason for the emergence of the greater incidence of depression among girls at this stage in comparison to boys (Cyranowski, Frank, Young, & Shear, 2000; Rudolph, 2002). Consistent with this hypothesis, Shih, Eberhart, Hammen and Brennan (2006) found that the higher rates of depression in adolescent girls compared to boys were explained by their exposure to greater amounts of stress that was interpersonal in nature, while girls with positive perceptions of their own interpersonal competence are less likely to develop depression (Eberhart, Shih, Hammen, & Brennan, 2006).

Social dysfunction exists among remitted depressed individuals (Hammen & Brennan, 2002; Herr, Hammen, and Brennan, 2007), and is also present prior to the onset of depression (e.g., Eberhart & Hammen, 2006), thus representing a potentially important risk factor for daughters of depressed parents. In a community sample of women, Hammen and Brennan (2002) found that formerly depressed women showed significantly more problematic interpersonal functioning than never depressed women across a wide range of

interpersonal variables. Specifically, they had more negative and conflicted marital relationships, more difficulties in their relationships with their children, friends, and extended family members, as well as involvement in more stressful life events with interpersonal conflict themes (Hammen & Brennan, 2002).

Thus, the evidence links depression with ongoing interpersonal difficulties, even during periods of remission, which has implications for the transmission of depression to the children of depressed parents (Hammen & Brennan, 2002). The offspring of parents with a history of depression may be exposed to problematic interpersonal behaviour and relationships from early in life, which may in turn contribute to their vulnerability to affective illness (Hammen, Shih, Altman, & Brennan, 2003). The development of interpersonal competence may be compromised by repeatedly observing their mothers engage with others in maladaptive and unsuccessful ways. Furthermore, the relationships between depressed mothers and their children tend to be characterized by maladaptive interaction patterns (Cummings & Davies, 1994; Downey & Coyne, 1990, Goodman & Gotlib, 1999), thus providing an additional mechanism by which the children of depressed mothers may learn dysfunctional interpersonal skills and cognitions (Hammen & Brennan, 2001).

Research has demonstrated the presence of interpersonal disturbance among the offspring of depressed mothers. Studies have shown that the children of depressed mothers from both longitudinal clinical (e.g., Adrian & Hammen, 1993) and cross-sectional community samples (e.g., Goodman, Brogan, Lynch, & Fielding, 1993) tend to be less popular among their classmates and to experience more conflicted peer interactions than do children of non-depressed mothers. Ashman, Dawson, and Panagiotides (2008) found that

the children of chronically depressed mothers, assessed over a 7 year period, have reduced social competence. Self-reported maternal depressive symptoms also have been linked with poor social skills in children (Koblinsky, Kuvalanka, & Randoph, 2006). Similar to the phenomenon in which depressed women have been found to generate high levels of interpersonal stress (Hammen, 1991), their children also have been shown to experience high levels of interpersonal difficulties in which their own behaviour likely plays a role (Adrian & Hammen, 1993).

Hammen, Shih, and Brennan (2004) tested an interpersonal transmission model proposing that the harmful effects of maternal depression on adolescents' risk for affective disorder are due in part to the interpersonally stressful nature of the family environment. They argued that this stressful interpersonal context, particularly the mother's stressful interpersonal relationships and parent-child relationship quality, has detrimental effects on adolescent interpersonal competence. The adolescent's compromised interpersonal competence and related involvement in stressful interpersonal events is suggested in turn to increase their risk for depression. The authors found support for this model in a large community sample of mothers with and without a history of depression and their 15-year-old children. Hammen, Shih, and Brennan (2004) found specifically that the transmission of depression from mothers to their adolescent offspring was fully mediated by maternal stress, parenting quality, and the youth's social competence. These results are indicative of the salience of interpersonal factors in the transmission of depression from parent to offspring. Maternal depression is linked with interpersonal difficulties and stress, which in turn are linked with lower social competence and depression in adolescents (Hammen, Shih, & Brennan, 2004).

Recent research by Hammen and her colleagues has demonstrated the relevance of interpersonal difficulties among the adolescent offspring of depressed mothers in relation to their own experiences with depression. In a large community sample of depressed adolescents, those with depressed mothers exhibited greater impairment in social functioning than did those with healthy mothers (Hammen & Brennan, 2001; Hammen, Shih, Altman, & Brennan, 2003). Those whose mothers had a history of depression had more impaired functioning across their relationships. They also reported elevated rates of negative interpersonal events and conflict, as well as more negative self-concepts with regard to their ability to establish close friendships (Hammen and Brennan, 2001). In a subsequent study of the same sample, Hammen, Shih, Altman, and Brennan (2003) showed that depression in children of depressed mothers was more strongly linked to chronic social dysfunction than was depression in children of healthy mothers, suggesting heightened depressogenic reactions to ongoing interpersonal problems in the high-risk group (Hammen, Shih, Altman, & Brennan, 2003).

A substantial subset of the Hammen et al. sample was followed-up five years later when the youth were age 20 (Hammen, Brennan, & Keenan-Miller, 2008). Maternal depression was a significant predictor of depression occurring between the ages of 15 and 20. In addition, those offspring with early onset-recurrent depression had significantly poorer interpersonal functioning than those with non-recurrent early onset depression, later onset depression, and those who never became depressed. When youth gender was controlled, early onset depression and interpersonal dysfunction accounted for the relationship between maternal depression and late adolescent depression. However, significantly different patterns were observed when gender was considered. For men, early

onset depression mediated the link between maternal depression and late adolescent major depression. This was not the case for women, however. For women, interpersonal difficulties accounted for the effect of maternal depression on the development of late adolescent depression. These findings underscore the salience of interpersonal difficulties in the development of depression in daughters at risk (Hammen, Brennan, & Keenan-Miller, 2008).

In summary, depression has been repeatedly associated with interpersonal difficulties, and research suggests that these difficulties may be present prior to the onset of depression and to persist during periods of remission. The offspring of mothers with a history of depression may be at particular risk for the development of poor interpersonal competence and its associated depressogenic effects given that they are exposed to their mother's problematic interpersonal behaviours and relationships from early in life. In comparison to offspring of healthy mothers, children of mothers with a history of depression have greater interpersonal difficulties. One study showed that, for girls, interpersonal difficulties mediate the relationship between maternal depression and the development of depression in adolescence.

Parameters of Maternal Depression & Risk for Children

Factors that may moderate the association between maternal depression and the risk for adverse outcomes in offspring are also important. The parameters of maternal depression are one set of possible moderators that has received little empirical attention. Children may be affected in different ways depending on: (1) at what developmental stage they are exposed to their parents' depression (timing); (2) the frequency and duration of their exposure to parental depression (chronicity); (3) how much impairment is experienced by

the affected parent (severity); and (4) whether or not the depressive illness exists alone or in conjunction with additional lifetime diagnoses (comorbidity). The parameters to be considered in the present study include the timing and chronicity of maternal depression.

Timing of Maternal Depression. Studies investigating the effects of the timing of maternal depression on children have focused on infant behaviour and physiology, maternal parenting behaviour, children's' interpersonal and emotion regulation skills, as well as their internalizing and externalizing symptoms.

Research with infants suggests that the timing of maternal depression can have differing effects on various infant behaviours and cortisol reactivity to stress. Diego, Field, and Hernandez (2005) showed that infants born to pre-partum depressed mothers cried and fussed more and appeared more stressed than infants of mothers who were depressed only during the postpartum period or not at all. While higher infant baseline cortisol levels have been linked to a lifetime history of maternal depression, only maternal depression during the pre- and/or post-partum period has been linked with higher infant cortisol reactivity (Brennan et al., 2008). These results suggest that the timing of maternal depression differentially affects stress-related behaviours and physiological responses of infants.

The association between the timing of maternal depression and parenting behaviours was examined in a meta-analytic review of 46 observational studies (Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Negative affect and hostile, coercive maternal behaviour was associated with both current and lifetime maternal depression with moderate and small effect sizes, respectively. In contrast, the timing of depression did not moderate the link between maternal depression and either positive parenting behaviours or maternal disengagement, where the effect sizes for current and lifetime depression were both small in the case of

positive behaviour and moderate with respect to disengagement. As noted by the authors, this suggests that there are likely enduring features of depression that exist outside of episodes that are linked with mothers' degree of involvement with and nurturance towards their children (Lovejoy, Graczyk, O'Hare, & Neuman, 2000).

Few studies have considered the impact of the timing of maternal depression on children's interpersonal competence and emotion regulation skills. In a longitudinal study of close to 5000 children and their mothers, Brennan et al. (2000) obtained self-reports of depressive symptoms from mothers during pregnancy, immediately postpartum, as well as when their child was 6 months and 5 years of age. This study showed that the 5-year old children of postpartum depressed mothers were less responsive to friendly invitations to play from their peers, but there was no such association with recent maternal depression. Maughan et al. (2007) also highlighted the impact of exposure to maternal depression early in life in a longitudinal study of mothers with a history of depression occurring within the first 21 months of their child's birth and a comparison group of well mothers. Consistent with the results of Brennan et al. (2000), children's self-reported social competence at age 5 was associated with early-occurring maternal depression (i.e., during the first 21 months of their child's life), but was not significantly related to maternal depression occurring at child age 3, 4, or 5, or to recurrent episodes. Similarly, early-occurring but not later or recurrent maternal depression was related to poorer emotion regulation in four-year old children (Maughan et al., 2007).

A number of studies have considered the timing of maternal depression in relation to offspring internalizing and externalizing symptoms. In a prospective community study, Essex, Klein, Miech, and Smider (2001) showed that children first exposed to maternal

depression in infancy were more likely to be rated by their kindergarten teachers as high in internalizing symptoms, whereas girls whose initial exposure occurred during the toddler/preschool years were at greater risk for externalizing symptoms. In the Brennan et al. (2000) study, the timing of maternal depressive symptoms was related to child behaviour problems at age 5, such that more depressive symptoms at six months or 5 years were more strongly linked with child behavioural problems than maternal depressive symptoms during pregnancy or immediately post-partum. Murray et al. (1999) conducted a follow-up investigation of post-partum depressed and well mothers when their children were age 5, and in contrast to Brennan et al. (2000), showed that depression in the post-partum period but not recent depression (i.e., within the past 12 months) was associated with child behaviour problems and reduced responsiveness in interactions with the mother.

While most existing studies on the impact of the timing of maternal depression have considered very young children, research by Hammen and colleagues has focussed on adolescents and their risk for depression. A cross-sectional, community-based study by Hammen and Brennan (2003) revealed that exposure to maternal depression any time from birth to age ten resulted in equivalent risk for adolescent depression. This finding was considered preliminary, however, given that the youth were last assessed at age 15 and therefore had not yet passed through the age of highest risk, which is generally during late adolescence through early adulthood (Hammen & Brennan, 2003). Subsequent research by Hammen, Brennan, and Keenan-Miller (2008) showed that maternal depression does not predict first onset of major depression in adolescent offspring after age 15, suggesting that the negative effects of maternal depression occur before children reach 15 years of age.

Chronicity of Maternal Depression. The effect of chronicity of maternal depression

on children has been investigated in a small number of studies with a focus on child behaviour and physiology, parenting behaviour, and various social and psychological difficulties experienced by children.

Neuro-behavioural and physiological differences have been noted among infants and young children exposed to maternal depression that is chronic in nature. Specifically, infants of more chronically depressed mothers (i.e., those depressed during both the pre- and post-partum periods) performed more poorly on neuro-behavioural assessments than did other infants (Diego, Field, & Hernandez, 2005). Chronic maternal depression has also been found to be associated with decreased frontal (Ashman, Dawson, and Panagiotides, 2008; Dawson et al., 2003) and parietal brain activation in children (Dawson et al., 2003). When maternal depression is chronic, it has also been associated with greater parasympathetic withdrawal in response to emotion-eliciting film clips, which has been suggested as a possible indicator of general dysregulation (Ashman, Dawson, and Panagiotides, 2008).

Parenting behaviour has also been shown to be influenced by the chronicity of maternal depression. For example, results from a large longitudinal study of 1215 women and their infants showed that women with chronic depressive symptoms were observed to be the least sensitive when playing with their children compared to women who never or sometimes reported symptoms of depression (NICHD Early Child Care Research Network, 1999). Consistent with this, chronically depressed women have been demonstrated to be more withdrawn when interacting with their children, to have less positive interactions with their children, and provide less structure in the form of set rules and procedures in the home environment (Campbell, Cohn, & Meyers, 1995; Dawson et al., 2003; Ewell Foster et al., 2008).

With regard to difficulties experienced by children of chronically depressed mothers, research has focussed on social skills, cognitive development, and externalizing and internalizing symptoms. The chronicity of maternal depression has been associated with social skills as early as the toddler years, with more chronically depressed mothers having 26-month old toddlers that are less skilled in asserting themselves with an examiner in a research setting (Dietz, Jennings, and Abrew, 2005). Chronic maternal depression also has been associated with decreased social competence in school aged children (Ashman, Dawson, and Panagiotides, 2008).

Cognitive development also appears to be affected by the chronicity of maternal depression. Specifically, more severe and more chronic maternal depressive symptoms have been associated with lower vocabulary scores in children at age 5 (Brennan et al., 2000). This is consistent with reports of lower scores on measures of expressive language skills among children of chronically depressed mothers (NICHD Early Child Care Research Network, 1999) and reviews suggesting generally poorer cognitive and language development in children when maternal depression has been chronic in nature (Sohr-Preston & Scaramarella, 2006).

A small number of studies have evaluated children's internalizing and externalizing symptoms in relation to the chronicity of maternal depression, with somewhat inconsistent results. Chronically depressed mothers were found to rate their children as less cooperative and more problematic behaviourally in one study (NICHD Early Child Care Research Network), while others have shown that the 3-year old children of chronically and remitted depressed mothers had equivalent rates of internalizing symptoms and behavioural problems (Dawson et al., 2003). This latter study, which included fathers' ratings of children's

symptoms to validate mothers' reports, suggests that past episodes of maternal depression may have enduring effects on the functioning of offspring (Dawson et al., 2003).

In research with early school-aged children, Alpern and Lyons-Ruth (1993) found that the 4- to 6-year old children of mothers reporting depressive symptoms during both the infancy and preschool period showed higher rates of hostile behaviour problems. The children of recently depressed mothers were more hyperactive and demanding, while children of mothers who were previously but not currently depressed showed significantly more anxious and withdrawn behaviour (Alpern & Lyons-Ruth, 1993). Similarly, Brennan et al. (2000) found that more severe and more chronic maternal depressive symptoms were associated with greater behaviour problems at age 5. These results are consistent with those found in a study by Ewell Foster et al. (2008) of older children ranging in age from 7 to 17, where depressed mothers with longer current depressive episodes had children who were more likely to have internalizing and externalizing symptoms. However, neither depression severity nor the number of maternal depressive episodes was related to symptoms in offspring. The authors suggested that the absence of an association between child adjustment and these maternal depression parameters may have been due to the limited variability across participants given that the majority reported symptoms falling in the severe range (Ewell Foster et al., 2008). While fewer studies have examined the occurrence of depression in adolescents as a function of the chronicity of their mother's depression, at least one cross-sectional, community-based study by Hammen and Brennan (2003) revealed that exposure to brief but severe maternal depression as well as longer term, mild maternal depression can lead to increased risk of depression for adolescent offspring.

To summarize, the timing and chronicity of maternal depression may be important

moderators of the association between a maternal history of depression and increased risk for adverse outcomes in children. However, the bulk of the research considering these aspects of maternal depression has been carried out with infants and young children. The few existing studies considering infant stress-related behaviour and cortisol reactivity suggest that earlier exposure to maternal depression is associated with worse outcomes. While current depression is associated with greater negative parental behaviour than lifetime depression, a history of depression regardless of its timing is associated with more disengaged maternal behaviour and less positive parenting behaviour. Earlier exposure to maternal depression appears to be associated with poorer interpersonal and emotion regulation skills in young children. Results are less consistent among studies investigating the effect of timing of maternal depression on children's internalizing and externalizing symptoms, with one study showing that more recent maternal depression (rather than depression during pregnancy or immediately post-partum) is associated with increased behavioural problems, while a second study found the opposite. With regard to risk for adolescent depression, one study found that exposure to maternal depression from birth to age 10 resulted in an equivalent degree of risk. Later research showed that the negative effects of maternal depression are manifested before children reach age 15.

More chronic maternal depression has been consistently associated with less desirable parenting behaviour and parent-child interactions, as well as poorer social and cognitive skills in offspring. While some studies have suggested that chronic maternal depression is associated with worse behavioural outcomes in children, at least one has shown that chronic and remitted depressed mothers have children with equal rates of internalizing and externalizing symptoms. Furthermore, it has been demonstrated that brief, severe

maternal depression may be just as detrimental to adolescent offspring as chronic maternal depression. Additional research with older children and adolescents is needed to shed further light on the longer term impact of the timing and chronicity of maternal depression.

The Present Study

Existing research shows that emotion regulation and interpersonal competence are compromised in the children of depressed mothers and that difficulties in these areas appear to precede the development of depression. There is also evidence suggesting that the stress response systems of children whose mothers have a history of depression may differ from those of the children of well mothers, but most studies have considered cortisol levels at baseline. Among those studies examining cortisol reactivity to stress, the results have been inconsistent and may vary depending on the timing of maternal depression. While the timing and chronicity of maternal depression are potentially important moderators of risk, few studies have examined these variables in relation to children's emotion regulation, interpersonal competence, and stress reactivity. There is some evidence to suggest that earlier exposure to maternal depression is linked with more problems in the areas of emotion regulation and interpersonal competence, while risk for depression appears to be equivalent regardless of when the child is exposed to the mother's depression during the first 10 years of life. While more chronic depression has been associated with poorer social skills, both chronic and brief but severe maternal depression are linked with increased risk for depression in offspring.

Thus, the literature to date suggests that difficulties in the areas of emotion regulation, interpersonal competence, and stress reactivity may help to explain the increased risk for depression in children of depressed mothers. However, these variables have

typically been studied in isolation and samples have consisted primarily of infants and young children. Further research is needed to examine the effects of maternal depression on older children and adolescents with regard to these variables of interest, as well as to consider the possible longer term effects of the timing and chronicity of mothers' depression on offspring.

The present study differs from past research in that it takes an integrative approach and considers the relationships among emotion regulation, interpersonal competence, and stress reactivity to determine how they might work together to increase the risk for depression in girls at high risk for the disorder. The present study focused on a community sample of adolescent girls of mothers with and without a history of depression prior to their adolescent years. The study is cross-sectional in design and the primary variables of interest include emotion regulation, cortisol reactivity to stress, and interpersonal competence. These variables are compared in girls at high and low risk for depression with an emphasis on the relationships among these factors and how they may serve to increase the likelihood of adverse outcomes in daughters. In this study, adverse outcomes are defined as daughters' self-reported depressive symptoms and the occurrence of major depressive episodes, as assessed with structured diagnostic interviews. The timing and chronicity of the mothers' depression are considered as potential moderators of risk in relation to daughters' emotion regulation, stress reactivity, interpersonal competence, and depressive symptoms.

Hypotheses

1. Compared to the daughters of healthy mothers, the daughters of mothers with a history of depression will show greater difficulty with emotion regulation, atypical cortisol levels at baseline and in response to a stressor, and poorer interpersonal

competence.

2. Poorer interpersonal competence will be linked with difficulties in emotion regulation, and both of these variables are expected to be linked with atypical cortisol levels.
3. Compromised interpersonal competence, emotion regulation, and stress reactivity, will play mediating roles in the link between maternal history of depression and depressive symptoms in adolescent offspring.
4. The timing of maternal depression will moderate the relationship between a maternal history of depression and emotion regulation and cortisol levels, such that earlier versus later exposure to a mothers' depression will be associated with poorer emotion regulation and less typical cortisol levels. Daughters' interpersonal competence also will be affected by chronicity, but not by age of first exposure to maternal depression.

Figure 1 illustrates the proposed relations among the variables described above.

Method

Participants

Participants were recruited from an existing sample of girls, ranging in age from 12 to 17, and their mothers. The girls are divided into two groups based on their risk for depression. Girls at high risk for depression have a mother with a history of depression prior to the daughter's adolescence, while low risk girls have mothers with no history of depression prior to adolescence. Participants were originally recruited when they were between the ages of 9 and 13 as part of a larger, 4-year longitudinal study called The Adolescent Girls Development Project (AGDP). The AGDP was a two-site study that took

place in Ottawa, Ontario and Halifax, Nova Scotia. The current study involved the Ottawa participants only. Only participants from the AGDP were used to address the issues of the current study given the confirmed diagnostic status of mothers and daughters.

A diversity of methods was used for AGDP participant recruitment. Posters and pamphlets were placed in community health centres as well as in the offices of local family physicians. Additional advertisements were placed in community newspapers. Recruitment through schools was the most successful method. In the Ottawa and surrounding areas, 80 schools (67 elementary and 13 intermediate) were contacted and 38 agreed to allow the distribution of information about the study. Fifty-five percent of the participants were recruited through schools, while 41% learned of the study through newspaper advertisements and only 3% through family physicians and community health centres. There is no record of how the remaining 1% of participants was recruited. Interested parents telephoned the research coordinator, who administered a brief screening interview over the phone, which included a self-reported depression history item. The parent who phoned was asked to report on their own history of depression as well as that of their daughter's other biological parent. In Ottawa, a total of 129 girls and their parents were recruited for the AGDP; 13 were ineligible and 14 withdrew at some point after the first assessment. Forty-eight percent of families included both the mother and father as participants and 52% included only mothers.

To be included in the AGDP, all participants had to understand written and spoken English. Daughters were required to be between the ages of 9 and 13 at the time of entry into the study. They were also required to be free from current and past psychopathology at the time of entry into the study. All girls were screened using the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS; Kaufman et al., 1997), a semi-structured

interview schedule used to assess current and past DSM-IV Axis I diagnoses in school-aged children and adolescents. The participating parents had to be the biological parent of the participating daughter. All participating parents were screened using the Structured Clinical Interview for DSM-IV. Parents in the high risk group met current or lifetime criteria for a major depressive episode, major depressive disorder or dysthymia, but not bipolar disorder. Parents in the low risk group did not meet current or lifetime criteria for depression at the time of entry into the study. Parents were re-assessed on an annual basis and daughters were assessed every six months. At termination of the AGDP, a total of 155 parents and 102 daughters were involved at the Ottawa site. The high risk group included 32 fathers, 62 mothers and 64 daughters, and the low risk group included 28 fathers, 33 mothers, and 38 daughters.

Recruitment for the present study followed termination of the AGDP, and consisted of calling former AGDP participants and advising them of this related study. Girls who had been classified as high risk because of paternal depression (n=9) were not contacted for participation in the present study. Of the 93 remaining AGDP participants, 21 (13 high risk, 8 low risk) daughters were not interested, 10 (9 high risk, 1 low risk) could not be reached, and 8 (5 high risk, 3 low risk) could not be scheduled after multiple attempts. There is no record of contact regarding the remaining two mother-daughter pairs (1 high, 1 low risk). In total, 52 daughters and 51 mothers agreed to participate in the present study. Two mother-daughter pairs were excluded because in each case the mother met criteria for a mood disorder due to a general medication condition, and as a result was not clearly appropriate for either the high or low risk group. Women with an episode of depression resulting from a medical condition or a reaction to medication may not be representative of mothers with a

history of depression and the associated interpersonal, emotional, and biological risk factors. One mother-daughter pair from the low risk group was excluded because the mother reported experiencing depressive symptoms in the moderately depressed range at the time of participation in the present study. Finally, two mother-daughter pairs from the low risk group were excluded because the daughters were only nine and ten years of age, respectively, at the time of their mother's last diagnostic assessment in the AGDP. Therefore, it could not be confirmed that their mothers had not experienced an episode of depression prior to their adolescence. As a result, the final sample consisted of 47 daughters (20 low risk and 27 high risk) and 46 mothers (19 low risk and 27 high risk).

Differences between AGDP participants who agreed versus declined to take part in the present study were explored. Of the eligible participants, 64% of the low risk group and 49% of the high risk group agreed to participate. However, this difference was not statistically significant, $\chi^2(1, N=86) = 1.74$, $p=19$. In addition, there were no significant differences between participating and non-participating participants in marital status, mother's level of education, or family income. These results are presented separately for high and low risk groups in tables 1 and 2.

Participating and non-participating groups were also compared on their average level of depressive symptoms during the course of their involvement in the AGDP. Specifically, the Beck Depression Inventory-II was administered to all mothers and daughters at regular intervals during the course of the AGDP and an average score was created for each participant. No significant differences were found between mothers or daughters who agreed versus declined to participate in the present study on self-reported depressive symptoms in either the high or low risk group. These results are presented in table 3. Thus,

non-participation in the study was not linked to key demographic variables or to the severity of depression experienced by mothers or their non-participating daughters.

Of the 47 daughters participating in the present study, diagnostic information collected during the AGDP was missing for 4 girls. Of the remaining 43 daughters, three low risk and five high risk girls met criteria for a DSM-IV disorder during their involvement in the AGDP. One low risk participant met criteria for panic disorder and separation anxiety disorder, another met criteria for social phobia, and one met criteria for adjustment disorder with depressed mood. Diagnoses in the high risk group included one case of major depressive disorder, two cases of social phobia, one case of anorexia nervosa, and one case of adjustment disorder with depressed mood

The daughters participating in the present study were, on average, 14.8 years of age and mothers were on average 47.15 years of age. The majority of the mothers were married and over 60% had annual family incomes of $75K or greater. Over 80% of the mothers had a college diploma, university degree, or some post-secondary education. All participants were Caucasian. The demographic information is summarized in Table 4. Daughters were given $40 for their participation in the present study and mothers were provided with $10 to cover parking costs.

Procedure

In the present study, maternal risk status was based on the SCID results obtained in the AGDP. On average, the time between the last SCID assessment and participation in the present study was 18 months. Daughters were, on average, 13 years of age at the time their mothers were last assessed, with a range of 10 to 15 years in the high risk group and 12 to 15 years of age in the low risk group. Low risk girls whose mothers were last assessed before

the daughter turned 12 were not included in the present study because I wished to confirm that the low risk daughters had not experienced maternal depression prior to adolescence. Thus, daughters considered high risk are those whose mothers experienced one or more episodes of depression prior to the daughter's adolescence. Daughters considered low risk had mothers who did not have a history of depression prior to the participant's adolescence, nor did these mothers report significant depressive symptoms at the time of participation in the present study. The majority of low risk mothers were free of other forms of psychopathology as well. Four met criteria for specific phobia but were retained in the low risk group given the prevalence of this diagnosis in the general population. Two had diagnosable disorders other than specific phobia: one an Adjustment Disorder NOS and the other Post-Traumatic Stress Disorder. Given that these diagnoses involve responses to specific life stressors, their daughters were considered low risk for the purpose of the present study. However, data analyses were conducted both with and without these participants, as reported in the Results section. I also determined whether mothers were currently depressed using the Beck Depression Inventory-II, described below.

Experimental Protocol. Upon arrival at the laboratory, daughters were asked to complete a package of questionnaires, described below. Subsequently, they underwent a modified version of the Trier Social Stress Test for Children (TSST-C; Buske-Kirschbaum, Jobst, Wustmans, Kirschbaum, Raugh, & Hellhammer, 1997).

Collection of salivary cortisol samples were done at 25, 10, and 0 minutes before the TSST-C as well as at 0, 5, 15, 30, and 45 minutes after the TSST-C. Three baseline samples were collected to obtain a more accurate estimate of participants' baseline cortisol levels because initial samples may be influenced by the stress associated with arriving at the

laboratory (A. Fiocco, personal communication, December 4, 2006). At the end of a relaxation period, following completion of the TSST-C, the daughters were administered the Depressive Disorders Section of the Kiddie Schedule for Affective Disorders (K-SADS) to assess episodes of depression since the time of their last assessment in the AGDP. Previously, the K-SADS was administered every 6 months to the girls during their involvement in the AGDP.

Experimental sessions were conducted in the afternoon, starting between 2:00 and 3:30 PM in order to control for the circadian variation in cortisol levels (Kudielka, Hellhammer, & Wüst, 2010). Participants were asked to avoid eating, drinking, or consuming caffeine for one hour before the session. They were also asked to avoid strenuous exercise the day before and the day of the session and none smoked within a half-hour of the session.

All mothers completed a self-report measure of current depressive symptoms. Mothers with a history of depression also were interviewed about the timing and chronicity of their depressive episodes.

Cortisol Sampling. Cortisol samples were collected using Sarstedt Salivettes® and stored in a freezer at -80 degrees Celsius until they were transferred on dry ice to the uOttawa Institute of Mental Health Research. There they were stored at the same temperature until assayed in duplicate in the laboratory of Dr. Zulfiquar Merali. The technician analyzing the cortisol samples had no knowledge of the purpose of the study or its hypotheses. Assay kits were purchased from MP Biomedicals and had an intra-assay variation of 5.3 to 8.9% and inter-assay variation of 7.5 to 9.3%. The minimum detectable level was 0.02 µg/dL.

*Measure*s

Lab Visit Questionnaire (Appendix A.1). Prior to completing the TSST-C, daughters were asked to complete a questionnaire designed to assess factors that are known to influence salivary cortisol levels, such as physical exercise, medication use, cigarette smoking, alcohol and drug use, time of last food intake, time of awakening on the day of the study, and phase of menstrual cycle (Dickerson Kemeny, 2004; Hanrahan, McCarthy, Kleiber, Lutgendorft, & Tsalikaian, 2006). The Lab Visit Questionnaire is an adaptation of the Admissions Questionnaire developed by McCardle (2004).

Stress Test (Appendix B). The Trier Social Stress Test for Children (TSST-C; Buske-Kirschbaum, Jobst, Wustmans, Kirschbaum, Raugh, & Hellhammer, 1997) is a psychosocial stress protocol based on the standardized stress paradigm developed and evaluated for laboratory studies with adult participants (Kirschbaum, Pirke, & Hellhammer, 1993). The TSST-C has been evaluated in children 8 to 14 years old (Buske-Kirscbaum et al. 1997) and others have used it with children up to 16 years old (e.g., Dorn et al., 2003). It has been shown that this protocol leads to a significant increase in free cortisol in children and adolescents (Buske-Kirscbaum et al. 1997), and a recent meta-analysis by Dickerson and Kemeny (2004) showed that the TSST is associated with greater cortisol responses than other types of stressors when used with adult participants.

The TSST-C involves public speaking and arithmetic tasks performed in front of an audience of two confederates. In the present study, the confederates sat behind a conference table facing the participant. A video camera also was in the room and directed towards the participant. Although it did not record the girl's performance, the girls were lead to believe that it was recording to ensure that the stress test was sufficiently anxiety provoking. Next,

one of the confederates read the beginning of a story and participants were asked to develop an ending to the story. Specifically, they were told that after 5 minutes of preparation in another room, they would be asked to return to finish telling the story in front of the two confederates. Participants were to finish the story in a speech lasting five minutes in duration. As per the TSST-C protocol (Buske-Kirscbaum et al. 1997), they were encouraged to try to perform better than all the other participants to increase the likelihood that the stress test would be sufficiently challenging. If they finished telling the story in less than five minutes, they were asked by the confederates to continue; this was done in a supportive and friendly manner.

In the Buske-Kirschbaum et al. (1997) protocol, the unfinished story appeared to be geared towards younger children rather than adolescents. Therefore, the present study employed the following more age-appropriate story, which was adapted from the novel "The Face on the Milk Carton" by C.B. Coonie (1991):

"My best friend Sarah and I were sitting in the school cafeteria talking and eating lunch when I glanced down at the face on my milk carton. No one ever really paid close attention to the faces of missing children on the back of milk cartons, but as I looked at the face of the ordinary little girl with her hair in tight pigtails – a three-year-old who had been kidnapped 12 years before from a shopping mall in New York, I was overcome with shock. I recognized that little girl -- it was me."

Following the speech task, the confederates asked the participant to serially subtract the number 13 from 1023 as quickly and as accurately as possible for five minutes. On every failure, one member of the committee would intervene by saying "Stop, please start again" (Buske-Kirschbaum et al., 1997). Following the stressor, all participants were told by

the committee that they had performed as well as other participants (Buske-Kirschbaum et al., 1997).

Manipulation Check (Appendix A.2). Anxiety levels before and after exposure to the TSST-C were assessed using a 100 mm Visual Analog Scale. The scale ranged from "not at all anxious" to "extremely anxious" and participants were instructed to indicate their current anxiety level by making a vertical mark along the scale. Scales were completed prior to the TSST-C, immediately afterwards, and again at the end of the relaxation phase following collection of the last cortisol sample.

Emotion Regulation (Appendix A.3). The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) is a 36-item measure completed by the daughters, which was designed to provide a comprehensive assessment of problems with emotion regulation. Items represent difficulties in the following areas of emotion regulation: (1) awareness and understanding of emotions; (2) acceptance of emotions; (3) the ability to engage in goal-directed behaviour and to refrain from impulsive behaviour when experiencing negative emotions; and (4) access to emotion regulation strategies perceived as effective. The DERS is comprised of six factors: (1) Nonacceptance of emotions (e.g., When I'm upset, I become angry with myself for feeling that way); (2) Difficulties Engaging in Goal-Directed Behaviour (e.g., When I'm upset, I have difficulty focusing on other things); (3) Impulse Control Difficulties (e.g., When I'm upset, I have difficulty controlling my behaviours); (4) Lack of Emotional Awareness (e.g., When I'm upset, I believe my feelings are valid and important; item reverse scored); (5) Limited Access to Emotion Regulation Strategies (e.g., When I'm upset, I believe that there is nothing I can do to make myself feel better); (6) Lack of Emotional Clarity (e.g., I have difficulty making sense out of my feelings). Respondents

are asked to indicate how often the items apply to themselves, with responses rated on a 5-point scale ranging from 1 ("almost never") to 5 ("almost always"). The DERS has high internal consistency (alpha = 0.93), adequate construct and predictive validity, and good test-retest reliability over a period ranging from four to eight weeks (Gratz & Roemer, 2004).

Interpersonal Competence (Appendix A.4). The Interpersonal Competence Questionnaire, Revised (ICQ-R; Burhmester, 2002) is a 40-item questionnaire completed by the daughters, which was designed to assess five domains of competence that are important in close relationships. The ICQ-R is a revised version of the Adolescent Interpersonal Competence Questionnaire (AICQ), a measure designed to assess the interpersonal skills of adolescents in their close friendships (Buhrmester, 1990). The ICQ-R has been used with children as young as 11 years of age, as well as with older adolescents and adults (Burhmester, 2002). Low scores are linked with loneliness and symptoms of depression while high scores are associated with closeness in best friendships and the number of friends that participants report having. Interpersonal competency ratings can be made by self-report and/or by the report of a friend and/or parents (Buhrmester, 2002). Only self-perceptions were evaluated in the present study.

The ICQ-R assesses five domains of competence considered to be important in close relationships: self-disclosure (e.g., "How good are you at opening up and letting someone get to know everything about you"); providing emotional support (e.g., "How good are you at making someone feel better when they are unhappy or sad?"); conflict resolution (e.g., How good are you at resolving disagreements in ways that make things better instead of worse?"); asserting influence ("e.g., How good are you at voicing your desires and opinions?"); and initiating relationships (e.g., "How good are you at calling new people on

the phone to set up a time to do things together?"). Items are rated on a 5-point scale to indicate how competent and comfortable the adolescent is in handling each situation described, where 1 = poor at this; would be so uncomfortable and unable to handle this situation that it would be avoided if possible and 5 = extremely good at this; would be very comfortable and could handle this situation very well. Internal consistency has been reported for the earlier version of this scale, the AICQ, with alphas ranging from .92 for adolescents to .93 for preadolescents (Buhrmester, 1990). Modest but significant correlations have been reported between self- and friend-ICQ-R ratings in both preadolescents (r = .26) and adolescents (r = .31 to .32), thus supporting the convergent validity of the measure (Burhmester, 2002).

Current Depressive Symptoms (Appendix A.5). Symptoms of depression in both mothers and daughters were assessed by the second edition of the Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a 21-item self-report measure of the presence and severity of depressive symptoms in adolescents and adults. This commonly-used instrument has been shown to have high internal consistency in samples of adolescents, with alphas ranging from .91 to .92 (Krefetz, Steer, Gulab, & Beck, 2002; Steer, Kumar, Ranieri, & Beck, 1998). High internal consistency has also been reported for adults, with alphas ranging from .91 to .93 for undergraduate student samples (Beck, Steer, & Brown, 1996; Dozois, Dobson, and Ahnber, 1998) and .92 in adult outpatient samples (Beck, Steer, & Brown, 1996).

Daughters' Diagnostic Status. The Schedule for Affective Disorders and Schizophrenia – Present and Lifetime (K-SADS-PL; Kaufman et al. 1997) was used to assess the presence of psychopathology in the daughters. The K-SADS-PL (Kaufman et al.,

1997) is a semi-structured interview that assesses current and past DSM-IV Axis I diagnoses in school-aged children and adolescents. The interview can range from 45 to 90 minutes in length (Hoge, 1999). Diagnoses are scored as definite, probable (i.e., at least 75% of criteria met for a particular disorder), or not present.

Psychometric data support the reliability and validity of diagnoses obtained using the K-SADS (Kaufman et al., 1997). Specifically, inter-rater agreement in scoring ranges from 93 to 100%. Test-retest reliability is in the excellent range for present and/or lifetime diagnoses of major depression, bipolar disorder, generalized anxiety disorder, conduct disorder, and oppositional defiant disorder, with correlations ranging from .77 to 1.00 (Kaufman et al. 1997). During the AGDP, both the SCID and the K-SADS were administered by doctoral level students in clinical psychology who had been trained by the principal investigator (PI), Dr. Valerie Whiffen, or by senior doctoral students experienced with the administration of the interview. Any uncertainty regarding participant diagnoses was resolved through discussion with the PI.

Mothers' Diagnostic Status. The Structured Clinical Interview for DSM-IV – Nonpatient Version (SCID-I/NP; First, Spitzer, Gibbons, & William, 1994) is the most widely used semi-structured interview for diagnosing psychiatric disorders according to the DSM-IV. It was used in the AGDP to assess parental diagnoses, both lifetime and current, on an annual basis during the AGDP. Psychometric data suggest acceptable inter-rater reliability (kappa >.70) for disorders such as major depression and the anxiety disorders. Validity studies have demonstrated high correspondence between SCID diagnoses and ratings of symptoms obtained with other standardized measures (Summerfeldt & Antony, 2002).

Timing & Chronicity of Maternal Depression. To establish the timing and chronicity of mothers' episodes of depression, their onset and course was established vis a vis the age of the child in the study. Interviews were conducted with the mothers to gather this information using an adapted version of Lyketsos et al. (1994) Life Chart Interview (Appendix C). First, important landmarks/life events such as births, deaths, job status, changes in marital status, and moves were inquired about to trigger the participant's memories for each year of their daughters' life. Next, participants were asked if they had experienced depression during each year. Threshold for a past episode of major depression included recollection of a minimum two-week period of depressed mood along with at least one other DSM-IV symptom of depression for the same time period. Threshold for a past episode of dysthymia included recollection of a minimum two-year period of depressed mood along with at least one other DSM-IV symptom of dysthymia during the same time period. Each reported depressive episode was coded for timing (i.e., age of daughter) and chronicity (i.e., number of depressive episodes and number of weeks or months duration).

The test-retest reliability of retrospective reports of depression tends to be modest (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1993). With regard to validity, however, the use of memory cues such as life events enhances the accuracy of recall, particularly when memories are accessed chronologically (Lyketsos et al., 1994). In a review of the research, Lyketsos et al. (1994) concluded that the recollection of autobiographical events and the dating of such events is potentially accessible and accurate for up to 20 years.

Results

The results are presented in three main sections: (1) The comparison of high and low risk groups on the study variables; (2) Regression based analyses to examine associations

among the variables and to test the proposed path model; (3) The examination of mother interview data to determine the impact of age at first exposure to maternal depression as well as the number and duration of maternal depressive episodes on daughter outcomes.

Data screening and cleaning was carried out according to the recommendations of Tabachnick and Fidell (2001). Relevant variables were examined for the accuracy of data entry and the presence of missing values. The fit between variable distributions and the assumptions of multivariate analysis were also examined. Scores with a z value of +/- 2.58 were considered univariate outliers and dealt with by changing their value to that of the next most extreme case. Multivariate outliers were considered significant at $p < .001$. Transformations were performed as needed to improve the normality of the variables. Potential confounding variables are considered and controlled for as required.

One individual questionnaire item was missing for each of three participants. Specifically, one participant was missing a response for one item on the ICQ-R, and two were missing responses for a single item on the BDI-II. Given the small amount of missing data, the mean for all cases was used to replace these missing item values and was used in calculating each participant's total score for the relevant variable.

Based on recommendations by Tabachnick and Fidell (2001), further screening and cleaning was conducted separately for group based analyses and regression-based analyses, as described below. For analyses involving group comparisons, cleaning and screening was conducted separately for each group being considered. In contrast, all participants were considered together for the screening and cleaning conducted prior to the regression based analyses.

Consideration of potential confounds

A number of potential confounding variables were considered. Tables 5 and 6 display the results for participants in the high and low risk groups on these variables. Although mothers in the high risk group reported lower family income, this variable was not correlated with any of the daughter variables. Mothers in the high and low risk groups did not differ on any other demographic variables. Therefore, none were controlled for in the analyses.

High risk girls had significantly more contact with the mental health system than daughters in the low risk group, and a history of treatment was linked with poorer conflict resolution skills, more difficulty providing emotional support to others, and blunted stress reactivity. Therefore, instead of masking potential differences between the groups, a history of treatment appeared to be another indicator of problems among participants in the high risk group. Consequently, this variable was not controlled in the analyses.

Mothers with a history of depression reported significantly more severe current depressive symptoms. Mothers' current depressive symptoms were significantly related to their daughters' emotion regulation, stress reactivity and recovery, as well as daughters' ratings on the final Visual Analog Scale. As a consequence, results of the analyses are presented both with and without current maternal depressive symptoms controlled.

A number of potential confounds specifically relevant to the analyses involving cortisol also were considered, including physical exercise before the session, cigarette smoking, eating and drinking in the one hour prior to the session, number of hours slept the night before the session, use of over-the-counter and prescription medication in the preceding two weeks, oral contraceptive use, and alcohol use the night before the session.

However, there were no significant differences between the high and low risk groups on any of these variables.

None of the participants met criteria for an episode of current major depression. However, one participant met criteria for a past episode of major depression and seven others met criteria for various other disorders during their participation in the AGDP. However, the presence of past diagnoses did not correlate with any of the cortisol variables. Nevertheless, the main cortisol analysis will be conducted with and without these participants because doing so is normative in published cortisol studies.

Comparison of high and low risk groups on the study variables

The first objective of this study was to compare the adolescent daughters of mothers with and without a history of depression on the following variables: emotion regulation, cortisol response to the TSST-C, interpersonal competence, and depressive symptoms. Daughters were also compared on their self-reported anxiety-level before and after exposure to the psychosocial stressor.

Screening and cleaning. High and low risk groups were considered separately for screening and cleaning procedures prior to conducting group comparisons. A single univariate outlier was found in the low risk group on each of the following variables: emotion regulation, two subscales of the Interpersonal Competence Questionnaire and on the Visual Analog Scale administered at baseline. In the high risk group, there was a single univariate outlier on the Visual Analog Scale administered at the end of the recovery period. There were also single univariate outliers on cortisol measurements taken at 25, 10, and zero minutes before the TSST-C as well as at 30 and 45 minutes post-stressor. Each of these univariate outliers was reduced to the next most extreme score on the variable of interest.

Such transformation of outliers is considered a valid way to deal with univariate outliers according to Tabachnick and Fidell (2001). This approach was selected over other options, such as deletion of outliers, because of the small sample size of the present study. No multivariate outliers were present in either the high or low risk group. The assumption of homogeneity of variance was met as assessed by Levene's test in all cases with the exception of the cortisol variables where the variances were significantly different across groups at each measurement time point. Inspection of minimum and maximum values across groups indicates that the low risk group tends to have higher minimum values than the high risk group and a much greater range, such that their maximum values tend to be higher than the high risk group as well. These differences in the variances across groups likely contribute to the results, presented below.

Analyses. Means and standard deviations on the study variables are presented in Tables 7 and 8. A t-test showed that daughters at high vs. low risk for depression did not differ significantly on the total score for emotion regulation. However, there was a trend for the high risk group to show more difficulty with emotion regulation than the low risk group (t = -1.92, p = .06). When the mother's current depressive symptoms were controlled, the difference between the high and low risk group was no longer even marginally significant, F (1, 44) = 1.07, p = .31, which suggests that group differences were due to current levels of depression rather than a maternal history of depression.

A MANOVA was used to assess group differences on the five subscales of the Interpersonal Competence Questionnaire. The composite dependent variate was not significantly related to risk status, Wilks' criterion, F (5, 41) = 1.05, n.s. Thus, the self-reported interpersonal competence of the high- and low-risk girls did not differ significantly.

High and low risk daughters' self-reported anxiety levels on the three Visual Analog Scales (VAS) were compared using GLM repeated measures analysis. The main effect for time was statistically significant, F (1.57, 70.82) = 37.85, $p < .001$, $\eta^2 = .46$. Pairwise comparisons showed that the VAS administered at baseline ($M = 18.16$, $SD = 17.67$) differed significantly from the VAS administered immediately following the TSST-C ($M = 41.90$, $SD = 28.44$). This result indicates that the psychosocial stress protocol was effective. The VAS administered at the end of the recovery period ($M = 13.99$, $SD = 13.49$) did not differ significantly from the VAS administered at baseline. Thus, participants' self-reported anxiety returned to baseline by the end of the recovery period (see Figure 2). The interaction between risk status and time was not significant ($F < 1.0$), which indicates that the girls in the high and low risk groups reported comparable levels of anxiety across time.

Given that maternal depressive symptoms were significantly correlated with the daughters' final VAS scores, this analysis was re-run with mothers' current depressive symptoms as a covariate. However, the results remained unchanged.

GLM repeated measures analysis was used to examine participants' cortisol levels across the eight measurement points. The main effect for risk status was significant, F (1, 45) = 5.53, $p < .05$, $\eta^2 = .11$, as was the main effect for time, F (3.12, 139.88) = 8.25, $p < .001$, $\eta^2 = .16$. However, these main effects were modified by a significant interaction between risk status and time, F (3.12, 139.88) = 5.73, $p = .001$. As shown in Figure 3, pairwise comparisons showed that the girls in the high risk group had significantly lower cortisol levels than those in the low risk group at 5 ($p < .01$), 15 ($p = .001$), and 30 minutes ($p < .01$). When the results were graphed (see Figure 3), the low risk group showed a normal cortisol response to the stressor in comparison to participants in the high risk group, who

showed a flat or blunted response.

This analysis was re-run with mothers' current depressive symptoms as a covariate. The main effect for risk status was no longer statistically significant, F (1, 44) = 2.78, p = .10. However, the main effect for time was still significant, F (3.16, 139.13) = 7.30, p = .001, as was the interaction between risk status and time, F (3.16, 139.13) = 3.70, p = .01. Even when maternal depressive symptoms were controlled, the general pattern remained, with the low risk group showing a normal stress response and the high risk group showing a blunted response. These results are illustrated in Figure 4.

When the GLM repeated measures analysis was conducted without the two low risk participants whose mothers were positive for a history of psychopathology (Adjustment Disorder and PTSD, respectively), the main effect for risk status was reduced to a trend, F (1,43) = 3.61, p = .064. The main effect for time, however, was still significant, F (3.23, 138.99), = 8.28, p < .001, as was the interaction between risk status and time F (3.23, 138.99), = 6.61, p < .001. Similarly, when daughters with a positive diagnostic history (n=8) were excluded, the main effect for risk status was no longer significant, F (1, 37) = 2.36, p = .13. However, the main effect for time was still significant, F (3.21, 119.10), = 5.97, p = .001, as was the interaction between risk status and time F (3.21, 119.10), = 5.18, p < .01. In both cases, the general pattern remained with the low risk group showing a rise and fall in cortisol levels over time and the high risk group showing a tendency toward a more blunted response.

In addition, the area under the curve with respect to ground (AUCg) was calculated using the trapezoid formula proposed by Pruessner, Kirschbaum, Meinlschmid, and Hellhammer (2003). AUCg provides a measure of total hormonal output (Fekedulegn et al.,

2007). When the groups were compared using a t-test, the low risk group was found to have significantly greater cortisol output compared to the high risk group, $t = 2.22$, $p < .05$. This finding is consistent with the repeated measures analysis discussed above.

Regression Based Analyses

A second objective of the present study was to assess the relationships among the main study variables, including possible mediating and moderating relationships. For this purpose, variables were created to represent cortisol reactivity to the TSST-C and cortisol recovery from the TSST-C. Stress reactivity was defined as a percent change score, calculated by the following formula: [(cortisol at +15 minutes – the average of three baseline cortisol samples)] / the average of the three baseline samples] x 100. The cortisol values obtained at 15 minutes post-stressor were used in this calculation because they represented the highest mean peak in cortisol values for the sample as a whole. Positive values indicate an increase in cortisol from pre- to post-stressor and negative values indicate a decrease in cortisol from pre- to post-stressor. Stress recovery was also defined as a percent change score, calculated by the following formula: [(cortisol at +45 minutes – cortisol at +15 minutes)] / cortisol at +15 minutes] x 100. Positive values indicate an increase in cortisol from post-stressor to the end of the recovery period whereas negative values indicate a decrease in cortisol from post-stressor to the end of the recovery period.

Screening and cleaning. All participants were considered for screening and cleaning procedures prior to conducting bivariate correlations and path analysis. Initial frequencies on the relevant variables were obtained. Univariate outliers were detected on variables representing daughters' stress reactivity and stress recovery. Univariate outliers were also detected on the mothers' BDI-II. Daughters' stress reactivity and BDI-II were significantly

positively skewed. Daughers' stress recovery and mothers' BDI-II were both positively skewed and kurtotic.

Square root transformations improved the normality of daughers' BDI-II and also reduced the extremeness of univariate outliers for stress reactivity, stress recovery, and mothers' BDI-II. Following these transformations, the variables stress reactivity and stress recovery continued to have one univariate outlier each, while all other variables were free of univariate outliers. Mahalanobis distances were obtained and no multivariate outliers were detected at $p < .001$. Given the small number of univariate outliers, value transformations were performed. Specifically, the single univariate outlier on each of Stress Reactivity and Stress Recovery was reduced to the next most extreme value in the sample. The shape of these variables was further improved by these transformations and a re-examination of Mahalanobis distances continued to reveal no multivariate outliers beyond $p<.001$. The assumptions of linearity and homoscedasticity were met as assessed by visual inspection of residuals scatterplots using the transformed variables. These assumptions were assessed for the variables in the regression analyses, and a reasonable number of variables were assessed for the analysis involving correlations among the main study variables.

Correlations among the study variables. In order to evaluate the path model illustrated in Figure 1, zero-order correlations among the study variables were examined (see Table 9).

Daughters' depressive symptoms were associated with greater difficulties with emotion regulation and with more problems with interpersonal competence, specifically, with asserting influence and resolving conflicts. Daughters' current depressive symptoms were not significantly associated with their stress reactivity and recovery in response to the

TSST-C. Greater difficulties with emotion regulation were significantly linked with reduced interpersonal competence across all subscales of the Interpersonal Competence Questionnaire as well as with reduced stress reactivity in response to the TSST-C. All of the interpersonal competence subscales significantly and positively correlated with one another. The conflict resolution subscale was significantly associated with stress reactivity, such that greater self-reported conflict resolution skills was linked with greater stress reactivity in response to the TSST-C.

Given that a maternal history of depression was not significantly associated with daughters' current depressive symptoms, the path model was modified to assess the possible indirect influence of a maternal history of depression on the daughter's depressive symptoms. Daughter's stress reactivity and emotion regulation were retained. However, of the interpersonal competence subscales only conflict resolution skills were retained as a result of the association between this variable and both stress reactivity and the daughter's depressive symptoms.

Path Analysis. A series of multiple regression equations were constructed to test the proposed path model. This model should be considered exploratory given the small sample size in the present study. A maternal history of depression was hypothesized to be associated with daughter's depressive symptoms through an effect on daughter's stress reactivity, emotion regulation, and conflict resolution skills. Subsequently, difficulties with emotional regulation and poorer conflict resolution were expected to be linked with more depressive symptoms. The order of the variables was determined by the proposed mediation model in conjunction with the correlation analysis presented above. Alternative models were not tested.

The first equation in the path model included the effects of maternal history of depression, daughter emotion regulation, and daughter conflict resolution skills on daughter depressive symptoms. The results of this equation yielded a significant R^2 of .443, F (3, 43) = 11.38, $p < .001$. However, only the path from daughter emotion regulation to daughter depressive symptoms was significant, which suggested that daughters' greater difficulties with emotion regulation are associated with higher levels of depressive symptoms ($t = 4.09$, $p < .001$).

The second equation assessed the effects of daughters' stress reactivity and conflict resolution skills on daughters' emotion regulation. The squared multiple correlation for this equation was significant at .343, F (2, 44) = 13.01, $p < .001$. Conflict resolution skills were negatively correlated with emotion regulation. Stress reactivity did not have a significant association with daughter emotion regulation ($t = -.97$) when considered together with daughter conflict resolution skills.

A third equation assessed the effects of daughters' stress reactivity and emotion regulation on daughters' conflict resolution skills. The results yielded a significant R^2 of .383, F (2, 44) = 13.66, $p < .001$. Only the path from conflict resolution skills was significantly associated with daughter emotion regulation.

A fourth regression equation assessed the effect of maternal history of depression on daughter stress reactivity. The results were significant, with R^2 of .151, F (1, 45) = 8.00, $p < .05$.

The integrated results of these analyses are illustrated in Figure 5. Given that the paths from daughter conflict resolution skills and maternal history of depression to daughter depressive symptoms failed to achieve statistical significance, a second model was proposed

that deleted these paths. The results for the trimmed model are illustrated in Figure 6. Daughter's emotion regulation explained 43.1% of the variance in daughter depressive symptoms, which was statistically significant with an F (1, 45) of 34.06, p <.001. The trimmed model also showed that daughter's stress reactivity was significantly associated with her emotion regulation, with an R^2 of .095, F (1, 45) of 4.74, p < .05. Finally, a maternal history of depression was significantly associated with daughter's stress reactivity, with an R^2 of .151, F (1, 45) of 8.00, p = .05.

Overall, the results of this path analysis suggest that a maternal history of depression influences daughter depressive symptoms indirectly. Specifically, a maternal history of depression was related to a blunted cortisol response to a stressor, which in turn was linked with poorer self-reported emotion regulation skills. Poorer emotion regulation skills were related to daughters' higher levels of current depressive symptom.

In order to determine whether the results of the trimmed path model were changed by controlling for mothers' current depressive symptoms, the effect of mothers' current depressive symptoms were controlled in the stress reactivity variable. The effect of mothers' current depressive symptoms were not removed from daughters' depressive symptoms or difficulty with emotion regulation due to nonsignificant zero order correlations. When the analysis was repeated, the results were no longer statistically significant. Specifically, daughter emotion regulation continued to explain 43.1% of the variance in daughter depressive symptoms, which was statistically significant with an F (1, 45) = 34.06, p <.001. However, daughter's stress reactivity was no longer significantly associated with daughter's self-reported emotion regulation, F (1, 45) = 2.58, p =.12. In addition, daughter's stress reactivity was associated with maternal depression history at the level of a trend, F (1, 45) =

2.98, $p < .10$. The mother's current level of depressive symptoms appears to be a more significant factor in her daughter's stress reactivity and self-reported emotion regulation than is her history of depression.

As previously discussed, the highest mean peak was used in calculating the stress reactivity variable used in the above regression analyses because this approach was used in previous research (e.g., Fiocco et al., 2007). However, calculating stress reactivity using individual peaks is also a valid approach (D. Koszycki, personal communication, April 6, 2011). Therefore, the full path model was also evaluated using individual peaks to determine possible differences in the results. One outlier was detected on stress reactivity calculated using individual peaks; this score was reduced to the next most extreme score on this variable.

The first equation in the path model assessed the effects of maternal history of depression, daughter emotion regulation, and daughter conflict resolution skills on daughter depressive symptoms and was not repeated given that it did not include the newly created stress reactivity variable.

The second equation assessed the effects of daughters' stress reactivity and conflict resolution skills on daughters' emotion regulation. The squared multiple correlation for this equation remained significant at .359, F (2, 44) = 12.33, $p < .001$. Stress reactivity did not have a significant association with daughter emotion regulation ($t = .23$) when considered together with daughter conflict resolution skills.

A third equation assessed the effects of daughters' stress reactivity and emotion regulation on daughters' conflict resolution skills. The results yielded a significant R^2 of .368, F (2, 44) = 14.42, $p < .001$. The path from emotion regulation was significantly

associated with daughter conflict resolution skills, while the path from stress reactivity failed to reach significance (t=1.65, p =.11).

A fourth regression equation assessed the effect of maternal history of depression on daughter stress reactivity. The results were marginally significant, with R^2 of .08, F (1, 45) = 3.73, p =.06.

Given that the path from daughter stress reactivity to daughter emotion regulation was no longer marginally significant, an alternative model was proposed that deleted this path. Daughter's conflict resolution skills explained 19.4% of the variance in daughter depressive symptoms, which was statistically significant with an F (1, 45) of 10.83, p <.01. This trimmed model also showed that daughter's stress reactivity was associated with her conflict resolution skills at the level of a trend, with an R^2 of .08, F (1, 45) of 3.67, p = .06. Finally, a maternal history of depression was associated with daughter's stress reactivity, also at the level of a trend, with an R^2 of .08, F (1, 45) of 3.73, p = .06.

These results suggest that when individual peaks are used to calculate stress reactivity, different results are obtained. Specifically, maternal history of depression predicted lower stress reactivity at the level of a trend, and lower stress reactivity predicted poorer conflict resolution skills, also at the level of a trend. Finally, poorer conflict resolution skills were significantly associated with more current depressive symptoms in daughters. In contrast, when mean peaks were used to calculate stress reactivity, maternal history of depression was significantly associated with reduced cortisol reactivity, which in turn was associated with more difficulty regulating emotion. Greater difficulty regulating emotion predicted more current depressive symptoms in daughters. Thus, lower stress reactivity calculated using individual peaks was associated with difficulty with conflict

resolution while lower stress reactivity calculated using means peaks was linked with more difficulty regulating emotion.

Interview Data

Age at First Exposure. Of the 24 high risk mothers interviewed, 18 (75%) reported at least one episode of depression during their daughters pre-school years (i.e., prior to age five) and six (25%) reported depression only when their daughter was age five or older. The mothers in the low risk group (N=20) were never depressed prior to their daughters' adolescent years.

Prior to analysis, the data were screened. One univariate outlier was detected on emotion regulation and two subscales of the Interpersonal Competence Questionnaire within the low risk group. Within the group of girls first exposed to their mothers' depression prior to age five, one univariate outlier was found on stress recovery and one on baseline cortisol. No univariate outliers were identified in the group of girls first exposed to their mothers' depression at age five or older. All univariate outliers were reduced to the next most extreme score within their group on the variable of interest. No multivariate outliers were present in the low risk group or either of the two high risk groups. The assumption of homogeneity of variances was met as assessed by Levene's test with the exception of the variable representing baseline cortisol. Inspection of minimum and maximum values across the three groups indicates that the low risk group and the high risk group exposed to their mothers' depression prior to age five have a greater range than the high risk group exposed to their mothers' depression at age five or older. This is likely as a result of the small number of daughters in the latter of the two high risk groups (n=6). All variables were approximately normally distributed.

Daughters' age at first exposure to maternal depression was used to categorize daughters: (1) never exposed; (2) first exposure prior to age five; (3) first exposure at age five or older. This age was selected because infancy and early childhood have been argued to be important foundational periods for the development of various individual differences, including emotion regulation (Gullone et al., 2010). Furthermore, this developmental period is marked by greater dependence on and contact with parents, particularly the mother, compared to middle or later childhood. Cole et al. (1994) noted that the preschool period is a time when children develop characteristic patterns of emotion regulation that may be difficult to influence in later childhood.

Univariate ANOVAs with Bonferroni correction were conducted to assess group differences on each of the daughter variables: emotion regulation, the five interpersonal competence subscales, depressive symptoms, baseline cortisol levels, stress reactivity, and stress recovery. Given the number of dependent variables, a Bonferroni corrected alpha (.05/10) of .005 was used to assess significance. There were no significant differences between the three groups on any of the variables using this corrected alpha level. Thus, age at first exposure to maternal depression (i.e., before age five or at age five and older) did not influence daughter outcomes in this sample. These results are presented in Table 10.

Chronicity of Maternal Depression. Chronicity of maternal depression was defined as the number of depressive or dysthymic episodes and the total number of months depressed or dysthymic during the daughter's childhood.

The number of episodes reported by the mothers ranged from 0 to 7, with a mean of 1.61, *SD* = 1.85. Within the high risk group, the number of episodes ranged from one to seven, with a mean of 2.96, *SD* = 1.49. The total time spent depressed or dysthymic prior to

the daughters' adolescence ranged from 0 to 96 months, with a mean of 13.16 months and a standard deviation of 20.29. Within the high risk group, the total number of months spent depressed or dysthymic ranged from 2 to 96 months with a mean of 24.13, $SD = 22.21$.

A hierarchical regression was planned to test for moderation. However, this analysis was determined to be inappropriate following preparation of the data for analysis. After the proposed moderators were centered to reduce the likelihood of multicollinearity in the data, correlations between the predictor variable (maternal history of depression) and the proposed moderators remained extremely high for both the number of months of maternal depression ($r = .90$, $p < .001$) and the number of episodes ($r = .94$, $p < .001$). Consequently, testing for moderation was not possible as a result of multicollinearity in the data. The effect of chronicity of maternal depression on the daughter variables was instead assessed using a correlational analysis within the high risk group only.

The data were cleaned and screened prior to examining correlations between the chronicity variables and the daughter variables. One univariate outlier was detected on each of the two chronicity variables as well as the variables representing baseline cortisol and stress recovery. The two chronicity variables and stress recovery were also all positively skewed. Square root transformations improved the shape of their distributions and univariate outliers were no longer present in either of the chronicity variables. However, one univariate outlier remained on stress recovery and baseline cortisol, both of which were reduced to the next most extreme score in the distribution. No multivariate outliers were identified through Mahalanobis distance. The assumptions of linearity and homoscedasticity were met as assessed by visual inspection of residuals scatterplots.

Correlations are presented in Table 11. The number of months of maternal

depression and the number of episodes of maternal depression during the daughters' childhood did not correlate significantly with any of the daughter variables. However, the correlation between total time depressed and daughters' baseline cortisol levels showed a trend towards significance (r = .41, p = .05), suggesting that daughters of more chronically depressed mothers tended to have higher baseline cortisol levels.

Discussion

The primary goal of this study was to compare the adolescent daughters of mothers with and without a history of depression on their emotion regulation skills, interpersonal competence, cortisol reactivity and recovery from stress. We know that the daughters of depressed mothers are at elevated risk for depression. The relationships among the study variables were considered jointly with the aim of determining how difficulties in emotion regulation, interpersonal competence, and stress reactivity may increase this risk. In addition, the timing and chronicity of the mother's depression were examined as potential moderators of this risk. Previous research has generally considered these variables in isolation. Thus, the present study aimed to take an integrative approach.

Baseline cortisol and cortisol reactivity to stress

The daughters of mothers with a history of depression were hypothesized to show atypical cortisol levels at baseline and in response to a stressor. Baseline cortisol levels were assessed prior to introducing the girls to the psychosocial stressor. Contrary to expectation, no differences were found between groups on baseline cortisol levels. This result differs from those of several previous studies showing higher baseline cortisol levels in children of depressed mothers (Ashman et al., 2002; Brennan et al., 2008; Bugental et al., 2003; Essex et al., 2002; Feldman et al., 2009; Lupien et al., 2000; Young et al., 2006). Four of these

previous seven studies found that maternal depression occurring during the child's infancy predicted elevated baseline cortisol (Ashman et al., 2002; Bugental et al., 2003; Essex et al., 2002; Feldman et al., 2009), while two others showed that current depression affected offspring baseline cortisol levels (Lupien et al., 2000; Young et al., 2006) and only one demonstrated this effect with lifetime maternal depression (Brennan et al., 2008). The current study did not assess the specific impact of exposure in infancy versus other stages of development and this may account in part for the discrepant findings. It should be noted, however, that the current study found a correlation between chronicity of maternal depression and baseline cortisol levels, such that more chronically depressed mothers tended to have daughters with higher baseline cortisol levels. This is in contrast to Ashman et al. (2002) and Gump et al. (2009) but appears to be consistent with Essex et al. (2002) who found that the young children of mothers reporting significant depressive symptoms currently as well as during their child's infancy had higher baseline cortisol levels than children of mothers reporting only current symptoms or no history of symptoms during the period covered. Given these varied results, further research is needed to determine the impact of chronicity of maternal depression on offspring cortisol levels at baseline.

Other findings identified important differences between the daughters of mothers with and without a history of depression in their cortisol response to a psychosocial stressor. Specifically, low risk girls showed a normal cortisol response to the stressor characterized by a rise in cortisol levels post-stressor and a subsequent decrease to baseline levels. In contrast, the high risk group showed a flat or blunted response to the stressor. This general pattern of results remained even when mothers' current depressive symptoms were controlled. These results are particularly interesting in light of the fact that both the high-

and low risk girls reported significant increases in anxiety from pre- to post-stressor, with no difference in the intensity of self-reported anxiety observed between the two groups. Thus, the girls perceived themselves as equally stressed by the experimental task. This suggests that high and low risk girls have differing physiological responses to challenge despite having similar levels of perceived stress in response to the TSST-C.

These findings are in contrast to earlier studies demonstrating higher cortisol reactivity to stress in the young offspring of mothers with a history of depression (Azar et al., 2007; Brennan et al., 2008; Feldman et al., 2009) as well as to studies showing no association between maternal depression and cortisol reactivity (Ashman et al., 2002; Gump et al., 2009). However, the present study differs in its use of an older, adolescent sample. Previous research on both baseline cortisol and cortisol reactivity to stress has focussed on infants and toddlers (Brennan et al., 2008; Bugental, Martorell, & Barraza, 2003), preschoolers (Essex, Klein, Cho, & Kalin, 2002), or pre-adolescent school age children (Ashman et al., 2002; Lupien et al., 2000; Young et al., 2006), with few studying adolescents (Halligan, Herbert, Goodyer & Murray, 2004).

Overall, the cortisol results suggest that a maternal history of depression may compromise stress response system functioning in adolescent daughters at a biological level. The relatively quick activation and inhibition of cortisol observed in the low risk group reflects a normal, adaptive response to stress, while the blunted response in the high risk group appears to reflect a maladaptive one (McEwen, 2000). The difference between the high- and low risk groups may result, in part, from inherited differences in stress response system functioning as research has suggested some degree of heritability in cortisol levels. For example, based on an analysis of five twin studies, Bartels, Van den Burg, Sluyter,

Boomsa, and de Geus (2003) concluded that approximately 62% of the variance in basal cortisol levels is due to genetic factors. More recently, Steptoe et al. (2009) studied the heritability of afternoon cortisol levels and cortisol reactivity using a twin design and a sample of 11-year old children. They found that cortisol levels at baseline and in response to a stressor are influenced at least in part by genetic factors. Cortisol reactivity specifically was noted to have a heritability value of 44% (Steptoe et al., 2009).

Interestingly, Ouellet-Morin et al. (2008, 2009) found differing degrees of genetic contribution to cortisol levels in infants from families rated as high versus low in adversity. In a sample of 6-month old twins, those from families high in adversity had a greater genetic component to their morning cortisol samples than did those from lower risk families (Ouellet-Morin et al., 2009). In an earlier study, these authors looked at cortisol reactivity to social novelty in 19-month old twins (Ouellet-Morin et al. (2008). They found that, in lower risk families, both genetic and environmental factors explained differences in cortisol reactivity, while in families characterized by high adversity, only environmental factors explained the variance in cortisol reactivity. Given that the environments shared by twins, rather than their shared genetics, accounted for the similarity in their cortisol reactivity, the authors suggested that adverse environmental conditions may in fact program the development of the HPA axis over and above the effect of genetic factors (Ouellet-Morin et al., 2008). While it is unclear what degree of environmental adversity may be required for this effect to occur, these results suggest that the pattern of cortisol reactivity in the present study may be the result of exposure to a mother with a history of depression and the associated stress that accompanies this disorder. The differing contributions of environmental and genetic factors across the two previous studies may reflect the fact that

one considered cortisol reactivity to social novelty (Ouellet-Morin et al., 2008), while the other focussed on awakening and morning cortisol levels (Ouellet-Morin et al., 2009).

Parental care may function as a "social regulator" of the HPA axis early in life, such that less sensitive or less responsive care adversely affects the regulation of this system (Gunnar & Quevedo, 2008, p.138). Studies with rodents indicate that good quality maternal care can regulate rat pup physiology and reduce stress reactivity later in life. The opposite also is true, whereby poorer quality maternal care increases corticosterone reactivity to stress in offspring (Liu et al., 1997, Meaney et al., 2001; Weaver et al., 2004). The differing effects of maternal care on offspring's stress reactivity have been shown to be transmitted across the generations, not as a result of genetics but rather due to the quality of maternal caregiving specifically (Francis et al., 1999). While the current study did not assess maternal care-giving, previous studies have repeatedly demonstrated that depression significantly affects parenting behaviour. A meta-analysis by Lovejoy, Graczyk, O'Hare, and Neuman (2000) showed that mothers with current as well as lifetime depression are more likely to engage in negative interactions with their children, including showing more irritability and higher levels of hostile and coercive behaviour. More recent reviews have also demonstrated that maternal depressed mood is associated with lower parenting confidence and competence, fewer positive interactions with their children, as well as more anger and greater use of harsh discipline (Whiffen, 2005).

Interestingly, studies have shown associations between parenting behaviour and offspring HPA axis functioning. For example, Ellenbogen and Hodgins (2009) demonstrated that when parents provide low levels of structure during middle childhood, their adolescent children show greater cortisol reactivity to a laboratory stressor as well as

increased cortisol response to awakening during adolescence. The sample used in the Ellenbogen and Hodgins (2009) study was comprised of offspring of parents with bipolar disorder as well as offspring of parents with no history of psychiatric disorder. While their findings did not differ according to risk group, exploratory analyses suggested that the association between parental lack of structure and cortisol levels was stronger in the high risk group. More recently, Murray, Halligan, Goodyer, and Herbert (2010) demonstrated that the withdrawn parenting behaviour of mothers with postnatal depression is associated with higher baseline cortisol levels in 13-year old offspring as well as higher maximum morning cortisol levels.

Research with nonhuman primates also has highlighted the importance of maternal care on the developing HPA system (Sanchez et al., 2001; Sanchez, 2006). This research is particularly important given that the brain maturation and parent-offspring interactions of nonhuman primates are more similar to those of humans than are those of other animals (Lupien et al., 2009; Sanchez, 2006). In contrast to rodent studies, findings have not consistently pointed to increased cortisol reactivity as a result of early life stress in non-human primates (Gunnar & Quevedo, 2008; Heim, Plotsky, & Nemeroff, 2004). For instance, squirrel monkeys that underwent repeated social separations were found to have reduced cortisol responses to subsequent separation stress (Lyons, 1999), as well as greater glucocorticoid feedback sensitivity in early adulthood (Lyons 2000). Sanchez et al. (2005) showed that repeated maternal separation in rhesus monkeys between three to six months of age was associated with a flattened diurnal cortisol rhythm at 12 months combined with an increased startle response. It is interesting to note that greater cortisol reactivity to the separation paradigm in early life was linked with lower basal cortisol levels at one year of

age, suggesting that a chronically activated HPA axis may be followed by a reduction in cortisol levels over time. Similarly, Dettling et al. (2002) exposed marmoset monkeys to maternal separation from postnatal days (PND) 2 to 28, which resulted in a significant increase in cortisol post-separation. However, by PND 28, basal morning cortisol levels were significantly reduced compared to control subjects. Thus, stress early in life that causes repeated activation of cortisol may lead to decreased rather than increased cortisol secretion in later life (Sanchez, 2006).

It is interesting to note that studies addressing the impact of other early life stressors have yielded similar findings. Specifically, a number of studies have shown diminished cortisol responses among adults with a history of early life stress. Heim et al. (2001) found that women with a history of childhood abuse, but without major depressive disorder, had lower baseline cortisol levels and responded with reduced cortisol reactivity to an $ACTH_{1-24}$ stimulation test compared to three other groups of women: those with a history of childhood abuse and major depression, those with major depression and no history of abuse, and comparison participants. In a more recent study also employing an endocrine challenge, Carpenter et al. (2009) found a diminished cortisol response to a DEX/CRH test among individuals with a history of childhood emotional abuse but without current Axis I psychopathology. The finding of diminished cortisol reactivity was increasingly prominent with age and independent of other types of abuse, past diagnoses, and current symptoms. Thus, the authors hypothesized that childhood emotional abuse may lead to suppressed biological reactivity in a cumulative fashion over time (Carpenter et al., 2009). These results are important as they demonstrate that early life stress is associated with reduced cortisol reactivity in adulthood, in the absence of major depression. Although some studies have

found elevated cortisol reactivity to endocrine challenge tests in samples with early life stress exposure (Heim et al., 2000; Heim et al., 2008), these results were found only among those individuals with current major depression.

A number of recent studies employing standardized laboratory stressors have also found reduced cortisol responses among individuals who have experienced significant stress in childhood. Carpenter et al. (2007) showed that healthy adults with a history of childhood maltreatment had diminished cortisol levels in response to a psychosocial stressor compared to control participants with no history of abuse. Consistent with this finding, Elzinga et al. (2008) found that adults without a history of psychiatric disorder but with high levels of exposure to adverse events in childhood had a blunted cortisol response to a psychosocial stressor, compared to participants with no exposure to early adverse life events. This finding occurred primarily among the men in the sample. Similar to the present study, there were no differences in baseline cortisol levels between the two groups with a history of high versus low exposure to adversity in childhood (Elzinga et al., 2008).

MacMillan et al. (2009) conducted a similar study with maltreated adolescent girls and control participants ranging in age from 12 to 16 years of age. Maltreated participants were recruited from child protection agencies and control girls were matched on age and postal code. Maltreated girls exhibited a blunted cortisol response to the psychosocial laboratory stressor despite a lack of difference in heart rates between the two groups. These results remained after controlling for current symptoms of depression and post-traumatic stress. The authors argued that responding to stress by briefly increasing cortisol levels is critical for survival and facilitates one's ability to effectively manage stress. For example, cortisol is important for mobilizing energy required for action, it plays a role in regulating

other systems that are sensitive to stress including the immune system, and it also can impact memory, learning and emotion through its action in the brain (Stansbury & Gunnar, 1994; Vazquez, 1998). MacMillan et al. (2009) argued, therefore, that the inability to elevate cortisol may be a risk factor for mental and physical health problems. They also hypothesized that chronic exposure to stressful events may be associated with diminished physiological responsiveness to subsequent stressors over time (MacMillan et al., 2009).

A mother with a history of depression may be a stressor that results in initially higher levels of cortisol, as reported in some studies of young children of depressed mothers (Ashman et al., 2002; Brennan et al., 2008; Bugental, Martorell, & Barraza, 2003; Essex, Klein, Cho, & Kalin, 2002; Halligan, Herbert, Goodyer, and Murray, 2004; Lupien et al., 2000; Young et al. 2006), but which over time leads to the development of attenuated cortisol reactivity to stress as demonstrated in the present study. This hypothesis is consistent with the above research demonstrating diminished cortisol levels in adolescents and adults exposed to stressful circumstances or abuse during childhood.

Stressful events are a central aspect of the lives of women with a history of depression and their families (Hammen, 2006). Stress levels across a variety of roles are elevated among depressed women. Chronically high levels of stress have an effect on children, separate from mothers' history of depression (Hammen et al., 1987). Furthermore, depressed mothers are particularly likely to experience stressful events to which they have contributed, such as interpersonal conflict, and this effect is present even when these women are not in an episode of depression. Hammen's (2006) stress generation model proposes that women with a history of depression create stressful family, marital, and child-rearing contexts, likely as a result of their personality traits, thinking patterns, and ways of behaving

that exist beyond the presence of depressive symptoms. Therefore, children of mothers with a history of depression are likely exposed to stressful circumstances that may be chronic in nature, persisting both during and outside of depressive episodes.

A number of theories exist to explain how exposure to stress early in life may lead to hypocortisolism, which has been defined, in part, as reduced adrenal cortical reactivity (Heim et al., 2000b). Hellmammer and Wade (1993) suggested that diminished HPA axis reactivity may result from long-term exposure to stress along with a hyper-reactive HPA axis and excessive release of glucocorticoids. Over time, the body may adjust such that it shifts from hyper- to hypo-reactivity. The body's ability to adjust in this way is considered an adaptive mechanism to protect against the harmful side effects of prolonged exposure to high levels of cortisol. However, if the HPA axis does not respond sufficiently to subsequent stress, over correction may have occurred (Fries, Hesse, Hellhammer, & Hellhammer, 2005).

McEwen (1998) characterized reduced cortisol responsiveness as one form of allostatic load, defined as "...the strain on the body produced by repeated ups and downs of physiologic response, as well as by the elevated activity of the physiological systems under challenge, and the changes in metabolism and the impact of wear and tear on a number of organs and tissues" (McEwen & Stellar, p. 2094, 1993). In other words, allostatic load is the effect of the physiological response to stress over the long-term, and the state of allostatic load can increase the risk for developing illness or disease (McEwen, 1998). From this perspective, reduced cortisol responsiveness may have the potential to increase the activity level of other systems, such as the immune system, and thus have consequences for one's health. When inadequate cortisol secretion occurs in response to a challenge, the secretion of inflammatory cytokines increases, which in turn can have negative consequences, such as

an increased risk for autoimmune and inflammatory problems, as demonstrated in animal studies (McEwen, 1998). Fries et al. (2005) noted that while a reduction in HPA activity has the advantage of protecting the body and brain from excessive exposure to glucocorticoids, it does so at the cost of increased sensitivity to stress, pain, and fatigue.

Dienstbier (1989) introduced the concept of "physiological toughness" and suggested that blunted HPA responses to stress may result from early life stress exposure combined with successful coping. Elzinga et al. (2008) also hypothesized that blunted cortisol reactivity may reflect resilience to stress. In the present study, it is not clear whether diminished cortisol reactivity in the high risk group reflects resiliency because the girls had not yet entered the high risk period for the development of psychopathology.

De Bellis (2001) posited a developmental traumatology model of PTSD which suggests that childhood trauma can lead to changes in biological stress systems, including reduced cortisol levels. Specifically, he argued that, under traumatic conditions, some vulnerable children respond with hypersecretion of cortisol that, over time, may have a neurotoxic effect on the developing brain. High levels of cortisol are suggested to have a potentially disruptive effect on the HPA axis and to lead to enhanced negative feedback inhibition and lower basal cortisol levels over time. The mechanism by which hypo-cortisolism develops is not clear, but Fries et al. (2005) suggested that the mechanisms could include down-regulation of certain receptors at different points on the HPA axis, depletion in CRF, ACTH, and/or cortisol, and/or increased negative feedback sensitivity to glucocorticoids. Heim et al. (2000b) noted that the process may be quite complex and involve changes at various levels of the HPA axis. In addition, the development of hypo-cortisolism could be influenced not only by early life stress exposure but by other factors as

well, such as genetics and gender (Heim et al., 2000b).

Interestingly, research has shown differences in the HPA axis functioning of children with parents with other forms of psychopathology, although the direction of the difference varies across studies and types of parental psychopathology. For example, Ellenbogen et al. have shown higher baseline cortisol levels in the adolescent offspring of parents with bipolar disorder compared to offspring of well parents (Ellenbogen, Hodgins, & Walker, 2004; Ellenbogen, Hodgins, Walker, Couture, & Adam, 2006, Ellenbogen, Santo, Linnen, Walker, & Hodgins, 2010), although no differences were found in cortisol reactivity to a psychosocial laboratory stressor (Ellenbogen et al., 2006). Similarly, higher cortisol levels have been reported in some studies of children of mothers with panic disorder compared to children of healthy mothers (e.g., Warren et al., 2003) but not in all reports (Battaglia et al., 1997; Terleph et al., 2006).

More consistent results have been found for the offspring of parents with PTSD. In a series of studies by Yehuda et al. (2000, 2002, 2007), the adult children of Holocaust survivors with parental PTSD have been repeatedly shown to have lower 24-hour urinary cortisol levels compared to the offspring of well parents. Lower cortisol levels in these offspring are related to the severity of parental PTSD as well as the severity of participants' own PTSD symptoms (Yehuda, Halligan, & Bierer, 2002). However, parental PTSD also has been associated with lower cortisol in offspring without a personal history of PTSD (Yehuda, Halligan, & Bierer, 2000; Yehuda et al., 2007), with one study showing that lower cortisol levels were related specifically to maternal rather than paternal PTSD (Yehuda et al., 2007).

In summary, the results of the present study suggest that exposure to a mother with a

history of depression may be sufficiently stressful to lead to changes in the HPA axis over time. Interestingly, differences in HPA axis functioning do not appear to be specific to offspring with a maternal history of depression. Studies of children of parents with other mood and anxiety disorders also show signs of HPA axis dysregulation. However, the differences in cortisol levels in these high risk offspring vary, with studies of youth at risk for bipolar disorder showing elevated baseline levels, PTSD offspring showing lower baseline cortisol levels, and mixed findings with regard to the offspring of parents with panic disorder. Fewer studies exist with regard to cortisol reactivity to stress in high risk offspring. Further research is needed to determine how other forms of parental psychopathology affect children's HPA axis functioning in response to stress.

Relationships among study variables

The second major goal of the study was to understand the links among interpersonal competence, emotion regulation, and stress reactivity. I hypothesized that these variables would mediate the link between a maternal history of depression and depressive symptomatology in adolescent offspring. Contrary to this hypothesis, a maternal history of depression was not found to correlate significantly with the daughter's self-reported depressive symptoms. Nevertheless, a mediation model was tested because the association between maternal depression and daughter depressive symptoms is well established in the literature (e.g., Connell & Goodman, 2002; Downey & Coyne, 1990; Wickramaratne & Weissman, 1998). Maternal depression was found to influence daughters' depressive symptoms indirectly via an association with daughters' cortisol levels and emotion regulation. The interpersonal competence subscale, conflict resolution, was not included in the final model because, when considered together with emotion regulation difficulties, it

was not significantly associated with daughters' depressive symptoms. Together the results indicate that, in the present sample, a maternal history of depression was linked with an atypical, blunted cortisol response to stress which in turn was associated with greater difficulties with emotion regulation. Not surprisingly, difficulties with emotion regulation were in turn linked with higher levels of daughters' depressive symptoms.

Mothers' current depressive symptoms played a role in these results. As noted earlier, the pattern of a blunted cortisol response in the high risk group remained significant when current maternal depressive symptoms were controlled in the repeated measures analysis. However, when mothers' current depressive symptoms were controlled in the path analysis, the link between a maternal history of depression and daughters' stress reactivity was reduced to a statistical trend, and the association between stress reactivity and daughter emotion regulation became nonsignificant. It should be noted that stress reactivity in the path analysis was defined as the percentage change in cortisol from pre- to post-stressor and does not reflect the full pattern of cortisol levels over the eight measurement time points, as was considered in the repeated measures analysis. However, the results of the controlled path analysis suggest that the mother's current depressive symptoms had some effect on the daughter's cortisol reactivity to stress as well as a significant impact on her self-reported ability to regulate emotion.

Girls of mothers with a history of depression appear to have an atypical physiological response to stress, and this atypical physiological response is associated with difficulty effectively regulating their emotions. Additionally, mothers' current depressive symptoms play a role in these relationships such that greater current maternal depressive symptoms are associated with reduced stress reactivity. The daughters of depressed mothers are likely

exposed to chronically stressful contexts and relationships which, over time, may lead to changes in their physiological reactivity to stress as discussed above. For these girls, the effective regulation of emotion is likely to be particularly important, but also extremely challenging. Many of these girls would find themselves ill equipped to manage such stress. Research has suggested that mothers with a history of depression have greater difficulty modeling and teaching healthy emotion regulation skills to their children (e.g., Garber, Braafladt, & Zeman, 1991; Hoffman, Crnic, & Baker, 2006; Shaw et al., 2006). Consequently, their children are more likely to engage in ineffective emotion regulation strategies than are the children of healthy parents (Blandon, Calkins, Keane, & O'Brien, 2008; Garber, Braafladt, and Zeman, 1991; Maughan, Cicchetti, Toth, & Togosch, 2007; Silk et al., 2006a; Silk et al., 2006b). It is unclear how a reduced physiological response to stress may influence emotion regulation. However, difficulty with emotion regulation has been linked with the development of depressive symptoms (Flett, Bankstein, & Obertynski, 1996; Silk, Steinberg, & Sheffield Morris, 2003; Garnefski, Kraaij, & Spinhoven, 2001; Garnefski et al., 2002; Garnefski, Boon & Kraaij, 2003; Kraiij et al., 2003). The findings of the present study are consistent with this previous literature.

These results are based on an analysis using stress reactivity calculated with a common mean peak in cortisol for the sample as a whole. This analysis provides information about participants' cortisol levels at a time that most would be experiencing their peak response. However, as previously noted, calculating stress reactivity using individual peaks is also a valid approach and provides an indication of each participant's highest cortisol level post stressor relative to their baseline level of cortisol. When the latter approach was used, the results of the regression analyses differed in important ways. First,

although a maternal history of depression continued to be linked with lower stress reactivity, this association was reduced to a statistical trend. In addition, the association between emotion regulation and stress reactivity was no longer significant. However, stress reactivity was linked with poorer conflict resolution skills at the level of a trend. Conflict resolution in turn predicted depressive symptoms in the daughters. The association between poorer conflict resolution skills and reduced cortisol reactivity is consistent with previous research (Fehm-Wolfsdorf, Groth, Kaiser, & Hahlweg, 1999).

Overall, these results suggest that reduced cortisol reactivity at a time when most participants are experiencing a peak in cortisol is associated with difficulty regulating emotion, which in turn is linked with depressive symptoms. In contrast, lower overall cortisol increase relative to one's own baseline is related to difficulty in conflict resolution, which also predicts elevated depressive symptoms. Thus, the two approaches to calculating stress reactivity yield results that differ in important ways. Given the small sample size of the present study, however, it should be emphasized that these path models are to be considered exploratory only and need to be confirmed by replication with a larger sample.

For the sample as a whole, poorer interpersonal competence was hypothesized to be linked with difficulties in emotion regulation, and both of these variables were expected to be linked with atypical cortisol levels. As expected, difficulty with emotion regulation was associated with cortisol reactivity to stress. Previous research has also shown an association between emotion regulation and HPA activity. Zimmerman and Stansbury (2004) showed that better emotion regulation in three-year old children, as assessed by behavioural observations, was associated with lower cortisol elevations during a stranger approach situation compared to children with less effective emotion regulation skills. In contrast, the

present study showed that difficulties with emotion regulation were associated with a reduced cortisol response to stress. The inconsistency in the two sets of results may be related to differences in the link between emotion regulation and stress reactivity in toddlers compared to adolescents. Furthermore, the HPA axis of adolescents may have had time to adapt to the nature of their environment and, in the case of girls of depressed mothers in the present study, their HPA axis may have adjusted to a stressful environment that over time resulted in reduced cortisol reactivity. Based on the results of the current study, it appears that adolescent girls with less effective emotion regulation capabilities are less likely to respond to stress with a normal physiological response characterized by a marked rise and fall in cortisol levels.

Interpersonal competence was also expected to be associated with an atypical cortisol response to stress given that the ability to interact with others in positive ways is dependent at least in part on effective self-regulation. For example, Schmidt et al. (1999) found that 7-year old children with higher levels of perceived social competence evidenced a greater decrease in cortisol during recovery from a performance stressor compared to children with lower levels of perceived social competence. The authors speculated that this may reflect better regulation of both negative emotion and the adrenocortical system. However, the present study generally did not find an association between cortisol and interpersonal competence. Specifically, four of the five interpersonal competence subscales were not associated with stress reactivity when the sample was considered as a whole. However, greater difficulty with one aspect of interpersonal competence, conflict resolution, was associated with lower cortisol reactivity in response to the psychosocial stressor in the present study. This finding is consistent with those of Hart, Gunnar, and Cicchetti (1995)

who demonstrated that reduced cortisol reactivity in maltreated children was associated with poorer social competence (Hart, Gunnar, & Cicchetti, 1995). Interestingly, couples observed to interact negatively with one another during a conflict failed to respond with a rise in cortisol compared to couples who interacted more positively (Fehm-Wolfsdorf, Groth, Kaiser, & Hahlweg, 1999). The authors hypothesized that a dampened stress response may develop over time as a result of chronic exposure to marital stress. Given the importance of conflict resolution for successful interpersonal relationships, difficulty in this area is likely to become a source of chronic stress for the daughters in the present study.

As expected, fewer difficulties with emotion regulation were associated with better interpersonal competence in the present study, for the sample as a whole, including the ability to initiate relationships, to provide emotional support, to assertively express oneself, to openly confide in friends, and to resolve conflicts. These associations are consistent with previous research demonstrating the importance of emotion regulation to interpersonal competence as early as the toddler and preschool years (Calkins, Gill, Johnson, & Smith, 1999; Maszk, Eisenburg, & Guthrie, 1999; Denham et al., 2003) through to adolescence (Phillips & Power, 2007) and adulthood (Gross & John, 2003; Lopes, Salovey, Strauss, 2003; Lopes et al., 2004; Lopes, Salovey, Cote, & Beers, 2005; Rivers et al., 2009). Collectively, previous research has demonstrated that emotion regulation problems or the use of less effective emotion regulation strategies is associated with more conflicted interactions with peers, less close and confiding relationships, and lower levels of social support, all of which likely put these individuals at risk for psychopathology, including depression.

Null differences between the high and low risk girls

Contrary to expectations, the daughters at high risk for depression by virtue of having a mother with a history of depression did not differ significantly from the daughters in the low risk group on a number of variables. For instance, they did not report significantly greater difficulties with emotion regulation than did the low risk daughters in the present study. Although this result is consistent with Santucci et al. (2008), it differs from those of the majority of previous studies in this area (Blandon, Calkins, Keane, & O'Brien, 2008; Garber, Braafladt, & Zeman, 1991; Maughan, Cicchetti, Toth, & Togosch, 2007; Silk et al., 2006a; Silk et al., 2006b). Past research has generally shown that young children exposed to maternal depression show dysregulated emotion patterns and that the older children of depressed mothers report fewer strategies for regulating their own and another person's negative affect, as well as less confidence in their proposed strategies.

Also contrary to expectations, high and low risk daughters did not differ significantly on self-reported interpersonal competence. In addition, there was no significant relationship between mothers' current depressive symptoms and daughters' self-reported interpersonal competence. These results are surprising given the existing research suggesting that the children of mothers with a history of depression are more likely to have interpersonal difficulties in comparison to children of healthy mothers. It has been previously demonstrated that children of depressed mothers are less popular with their peers, have reduced social competence, and more conflicted interactions with peers compared to the children of mothers without a history of depression (Adrian & Hammen, 1993; Ashman, Dawson, & Panagiotides, 2008; Goodman, Brogan, Lynch, & Fielding, 1993; Hammen & Brennan, 2001; Hammen, Shih, Altman, & Brennan, 2003; Hammen, Brennan, & Keenan-

Miller, 2008; Koblinsky, Kuvalanka, & Randolph, 2006). Interpersonal factors have been demonstrated to play a role in the transmission of depression from parent to offspring (Hammen, Shih, & Brennan, 2004), and greater social dysfunction has been reported among the depressed adolescent children of mothers with a history of depression compared to the depressed children of healthy mothers (Hammen & Brennan, 2001; Hammen, Shih, Altman, & Brennan, 2003), although this finding was shown particularly among those youth with early onset-recurrent depression (Hammen, Brennan, & Keenan-Miller, 2008).

The absence of differences between the high and low risk daughters on emotion regulation and interpersonal competence in the present study is surprising and may reflect methodological differences between the current study and previous research. With regard to emotion regulation, the majority of previous studies used samples of very young children whereas the current study used an older, adolescent sample. Difficulty with emotion regulation may be more pronounced among younger rather than older children of mothers with a history of depression. Differing definitions of maternal depression also may have contributed to the discrepant findings. Most previous studies focussed on current maternal depression or depression that originated during the mother's childhood, while the current study differs in its focus on mothers with a lifetime history of depression.

In addition, the present study used self-report measures of emotion regulation and interpersonal competence. Most previous studies of emotion regulation relied on observational methods. Adolescent girls' awareness of their own difficulties with effectively regulating their emotions could be limited, particularly if they are attempting to manage emotion by shutting it down or suppressing it, as may be suggested by the correlation between poorer emotion regulation and reduced cortisol reactivity to stress. It would have

been interesting to obtain parent, teacher, and/or peer perspectives on the daughters' use of emotion regulation strategies. The majority of previous studies of interpersonal competence used interview measures, parent and/or teacher ratings, or self-report questionnaires in combination with all of the above. Goodman, Brogan, Lynch, and Fielding (1993) found that across multiple measures of social competence, children of depressed mothers and well fathers were rated as less popular by their teachers but did not differ from low risk children on measures of social problem solving or their ability to accurately detect their peers' intentions. It may be that the use of single self-report measures of interpersonal competence and emotion regulation in the present study was insufficient to detect the presence of significant differences. Finally, the small sample size may have played a role in the non-significant findings, particularly with regard to emotion regulation, because the means were in the predicted direction. Despite the absence of significant differences between the high and low risk groups on these variables, a maternal history of depression did appear to affect daughters' emotion regulation indirectly through its impact on their cortisol reactivity to stress, described above.

Timing and chronicity of maternal depression

The timing of maternal depression was hypothesized to moderate the relationship between a history of maternal depression and the daughter's emotion regulation and cortisol levels. Specifically, earlier versus later exposure to a mother's depression was hypothesized to be linked with poorer emotion regulation skills and atypical cortisol levels. Furthermore, daughters' interpersonal competence was hypothesized to be affected by chronicity but not by age at first exposure to maternal depression.

Contrary to expectations, the daughter's age at first exposure to maternal depression

did not differentially affect her self-reported ability to regulate emotions or her cortisol levels at baseline or in response to stress. The absence of an effect of timing of maternal depression on emotion regulation in the present study differs from Maughan et al. (2007). This previous study found that maternal depression occurring early in the child's life (i.e., within their first 21 months of age) but not later maternal depression was related to poorer emotion regulation in children. However, it should be noted that, in contrast to the present study, Maughan et al. (2007) focused on four-year old children and assessed emotion regulation via observational methods.

The absence of an effect of age at first exposure to maternal depression on cortisol reactivity is consistent with the results of Ashman et al. (2002), who also did not find any effect of the timing of maternal depression on cortisol reactivity to stress in 7- to 8- year old children. Although Brennan et al. (2008) showed that depression in the pre- or post-partum period predicts greater stress reactivity in offspring, their study differs from the present research in their focus on infants rather than adolescents. Other studies showing an effect of timing of maternal depression on offspring cortisol levels have focused on baseline cortisol rather than cortisol reactivity to stress (Essex et al., 2002). Where both were considered, a significant effect for timing of maternal depression was found for baseline cortisol levels only (Ashman et al. 2002). While the present study also assessed baseline cortisol prior to the introduction of the stress task, no differences were found between daughters exposed to their mothers' depression before or after age 5.

The absence of significant effects in the present study also could be a result of the small sample size. In addition, there was little variability with regard to the different ages of first exposure, with the majority of the high risk girls (75%) being first exposed to their

mothers' depression before the age of five. Some of the existing research has found differences in outcomes when children were exposed to their mother's depression in infancy versus during the toddler and preschool years, but such differences would not have been detected in the present study. Future research with larger samples and greater variability in age of first exposure are important to provide a better test of the effects of timing of maternal depression on these outcomes in adolescents.

Unfortunately, the chronicity of maternal depression could not be assessed in the present study as a possible moderator because of multicollinearity in the data. However, correlations showed that the chronicity of maternal depression was related to higher baseline cortisol in offspring at the level of a trend, as discussed above, but was not significantly associated with any of the daughter outcome variables, including interpersonal competence. The majority of previous studies used samples of very young children. Two studies demonstrated greater problems with interpersonal competence when maternal depression is chronic (Dietz, Jennings, & Abrew, 2005; Ashman, Dawson, & Pangiotides, 2008), while the third did not find a significant effect (Maughan et al., 2007). Thus, the present study tends to confirm the results of Maughan et al. (2007). However, it should be noted that these previous studies used samples of young children, ranging in age from 26 months to 6.5 years, whereas the current study focussed on adolescents.

The interviews used to establish age of first exposure to maternal depression and chronicity of maternal depression were retrospective in nature. To minimize the limitations associated with retrospective reports of depressive episodes, a life chart was created in collaboration with the interviewee which included important life events such as births, deaths, job status, changes in marital status, and moves. Mothers and daughters ages were

also used as landmarks on the chart in an effort to trigger the participant's memories for each year of their daughters' life. Use of memory cues has been reported to enhance the accuracy of recall (Lyketsos et al., 1994).

Limitations and future directions

The present study has several limitations. First, the sample size was small which, as previously discussed, resulted in reduced power to detect effects. The small sample size is a result of the fact that a significant proportion of former AGDP participants declined to participate in the current study. Although non-participating and participating individuals did not differ significantly on demographic characteristics or depressive symptoms, it is unlikely that drop-out was random. Those who chose not to participate may have differed from the current sample in important ways that were not assessed or not evaluated by this researcher. Thus, null findings should be regarded with caution and the conclusions should be considered suggestive until the results have been extended and/or replicated.

A second limitation is that risk status was defined as a maternal history of depression before the daughters' adolescent years. The present study assessed maternal depression prior to the daughters' adolescence, not after this period. It is possible that some low risk mothers experienced an episode of depression once their daughters reached adolescence, which would attenuate the observed differences between the high and low risk groups. However, this outcome is unlikely given that first episodes of depression most frequently occur in adolescence or early adulthood. Low risk mothers in the present study were 47 years of age on average, with a range of 42 to 57 years. It should be noted, however, that these mothers fall within the age range of the perimenopause, which is a time of increased vulnerability for the development of depression. Nevertheless, the incidence of first onsets of depression is

relatively low and tends to be predicted by a history of anxiety disorder (Bromberger et al. 2009), which would have been detected by the lifetime SCID. Thus, these women are considered unlikely to have experienced a first episode after termination of their involvement with the AGDP. The absence of significant depressive symptoms reported by the low risk mothers on the BDI-II at the time of the study further increases our confidence that they are low risk for the disorder.

An additional limitation related to the definition of risk status is the fact that fathers' mental health and its potential impact on daughters was not considered. Although depression in mothers has been shown to be more closely linked to children's internalizing problems than is depression in fathers, it is important to consider psychopathology in both mothers and fathers when examining mental health outcomes in offspring (Connell & Goodman, 2002). Maternal psychopathology has been shown to be more closely related to emotional and behavioural problems in younger children, while paternal psychopathology has a greater association with children's emotional and behavioural problems later in their development (Connell & Goodman, 2002). Other researchers have shown that fathers' mental health status can influence whether maternal depression is associated with offspring psychopathology during both the toddler years (Dietz, Jennings, Kelley, & Marshal, 2009) as well as adolescence (Brennan, Hammen, Katz, & Le Broque, 2002). Therefore, it would have been useful to consider fathers' psychopathology to determine to what extent it may have influenced the results of the current study.

The retrospective nature of the SCID interviews used to establish a history of psychopathology is a third limitation of the present study. Although it is considered unlikely, it is possible that some women in the low-risk group actually did experience

clinically significant episodes of depression that they did not recall in the SCID interview. However, this is a common design in this area of research and the SCID has been shown to have good test-rest reliability for major depression (kappa = .73) and dysthymia (kappa = .60) (Zanarini & Frakenburg, 2001).

A fourth limitation is the use of retrospective life chart interviews to establish daughters' age of first exposure to maternal depression as well as the chronicity of maternal depression. Although efforts were made to minimize the limitations associated with this methodology, a longitudinal design including full diagnostic interviews with mothers would increase accuracy.

A fifth limitation includes the use of a self-report questionnaire to assess mothers' current depressive symptoms. Ideally, diagnostic interviews would have been used to establish mothers' current psychopathology. In addition, diagnostic interviews with the daughters assessed for depression only. Ideally, the full range of psychopathology would have been assessed in order to examine the potential impact of comorbid disorders on the variables of interest. Limited resources prevented more in-depth diagnostic interviewing.

As previously noted, emotion regulation and interpersonal competence were assessed with self-report measures only, which represents the sixth limitation of the present study. It would have been interesting to have multi-informant data for both of these variables. Parents', teachers', and/or peers' observations about the girls' use of emotion regulation strategies and interpersonal competence would have been useful.

A seventh limitation of the present study concerns the stress task and the collection of salivary cortisol. Numerous factors can influence cortisol secretion and the only variable controlled for in the present study was time of day the research sessions were held (i.e.,

afternoon). Although certain variables were inquired about via participant self-report, and no significant differences were found between the high and low risk groups, other important factors were not addressed. Specifically, menstrual cycle phase can affect cortisol response to stress (e.g., Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; Kudeilka, Hellhammer, & Wust, 2009) and was not controlled in the present study. In addition, self-reported anxiety level was used to assess participants' perceived level of anxiety before and after the TSST-C. As suggested by Stroud et al. (2009), including behavioural observations of participants' level of anxiety may also be useful in objectively assessing participant anxiety level in response to a laboratory stress task. Finally, the results of the present study are limited in terms of their generalizability. The sample consisted of mothers who were well educated and had high annual family incomes. Consequently, the results may not be applicable to families of lower socioeconomic status.

Future research with larger samples will be necessary to replicate the results of the present study. If replicated, additional research will be necessary to determine how and why daughters at high risk for depression show blunted cortisol reactivity to stress and whether this pattern is associated with greater risk for depression. Ideally, future studies should use a longitudinal research design and document exposure to maternal depression over the course of the child's life. A longitudinal design would allow for other important variables to be assessed, including the effects of the timing and chronicity of maternal depression to determine whether there are sensitive developmental periods during which exposure to maternal depression leads to changes in offspring's cortisol reactivity to stress. Future studies should consider including measures of parenting quality given the research showing an association between HPA axis functioning and maternal care giving.

Table 1. *Comparison of eligible AGDP low risk participants who agreed vs. declined to participate in the present study on demographic characteristics.*

Variable	Agreed	Declined	χ^2
Mothers Marital Status	*n*=21	n=12	1.34
Married (%)	55	33	
Widowed (%)	3	0	
Divorced (%)	3	3	
Separated (%)	3	0	
Never Married (%)	0	0	
Mothers' Level of Education	*n*=21	n=11	.47
Less than high school (%)	0	0	
High school (%)	3	6	
Some college (%)	6	6	
College diploma (%)	6	0	
University degree (%)	25	6	
Some graduate or professional school (%)	6	6	
Completed graduate or professional school (%)	19	9	
Family Income	*n*=21	n=11	.50
Under $15K (%)	0	0	
$15-$30K (%)	0	3	
$30-45K (%)	6	0	
$45-60K (%)	0	3	
$60-75K (%)	13	6	
$75-90K (%)	9	6	
> $90K (%)	38	16	

Notes: Percentages of mothers across educational levels does not equal 100% due to rounding; *$p < .05$; **$p < .01$; 3% of mothers were missing education and income data.

Table 2. *Comparison of eligible AGDP high risk participants who agreed vs. declined to participate in the present study on demographic characteristics.*

Variable	Agreed	Declined	χ^2
Mothers Marital Status	n=26	n=23	.13
Married (%)	37	33	
Widowed (%)	0	0	
Divorced (%)	8	8	
Separated (%)	6	4	
Never Married (%)	2	2	
Mothers' Level of Education	n=26	n=23	.08
Less than high school (%)	2	0	
High school (%)	4	0	
Some college (%)	4	12	
College diploma (%)	12	12	
University degree (%)	20	10	
Some graduate or professional school (%)	2	2	
Completed graduate or professional school (%)	8	10	
Family Income	n=25	n=21	.10
Under $15K (%)	0	2	
$15-$30K (%)	0	4	
$30-45K (%)	11	4	
$45-60K (%)	11	7	
$60-75K (%)	4	4	
$75-90K (%)	4	2	
> $90K (%)	24	22	

Notes: Percentages of mothers across education and income levels does not equal 100% due to rounding; *$p < .05$; **$p < .01$; 7.5% of mothers were missing marital status and education data; 13.2% were missing income data.

Table 3. *Comparison of eligible AGDP participants by risk status on average BDI-II scores for those who agreed vs. declined to participate in the present study.*

Low Risk Group	Agreed	Declined		
	n=20 mothers *n*=20 daughters	*n*=11 mothers *n*=11 daughters	*t*	*p*
Mothers' Mean BDI-II	5.52	3.20	-1.02	.32
Daughters Mean BDI-II	6.90	6.06	-.56	.58
High Risk Group	Agreed	Declined		
	n=23 daughters *n*=23 daughters	*n*=23 daughters *n*=23 daughters	*t*	*p*
Mothers' Mean BDI-II	11.87	13.42	.51	.62
Daughters Mean BDI-II	8.33	11.21	1.50	.14

Note: 6.1% of low risk mother-daughter pairs and 13.2% of high risk mother-daughter pairs were missing BDI-II data from all waves of the AGDP.

Table 4. *Demographic information.*

Variable *n* = 47 girls; *n* = 46 mothers	*M*	*SD*	*Range*
Daughters Age	14.80	1.20	12-17
Mothers Age	47.15	4.00	32-57
Mothers' Marital Status:			
Married (%)	77		
Separated or Divorced (%)	19		
Widowed (%)	2		
Never Married (%)	2		
Annual Family Income:			
$30-45K (%)	15		
$45-60K (%)	11		
$60-75K (%)	13		
$75-90K (%)	11		
> $90K (%)	50		
Mothers' Level of Education:			
Less than high school (%)	2		
High school (%)	6		
Some college (%)	9		
College diploma or university degree (%)	55		
Some graduate or professional school (%)	6		
Completed graduate or professional school (%)	21		

Table 5. *Comparison of high and low risk groups on study variables.*

Variable	*Low Risk* *n*=20 girls *n*=19 mothers		High Risk *n*=27 girls *n*=27 mothers		
T-Test/Chi-Square Comparisons:	*M*	*SD*	*M*	*SD*	t / χ^2
Daughters Age	14.80	1.20	14.74	1.35	.16
Mothers Age	47.15	4.00	45.78	5.38	.96
Daughters' Depressive Symptoms	6.00	7.03	8.52	5.85	-1.34
Mothers' Depressive Symptoms	3.25	3.74	11.89	11.11	-3.48**
Family Income					6.23*
$30-45K (%)	5%		22%		
$45-60K (%)	0%		19%		
$60-75K (%)	15%		11%		
$75-90K (%)	15%		11%		
> $90K (%)	65%		37%		
Mothers' Level of Education					1.57
Less than high school (%)	0%		4%		
High school (%)	5%		7%		
Some college (%)	15%		4%		
College diploma or university degree (%)	40%		67%		
Some graduate or professional school (%)	10%		4%		
Completed graduate or professional school (%)	30%		15%		
% of Mothers Married	85%		70%		1.37

Notes:
Percentage of high risk mothers across educational levels does not equal 100% due to rounding.
*$p < .05$
**$p < .01$

Table 6. *Comparison of high and low risk girls on relevant control variables.*

Variable	Low Risk $n = 20$		High Risk $n = 27$		
T-Test/Chi-Square Comparisons:	*M*	*SD*	*M*	*SD*	t / χ^2
Number of hours slept night before	9.78	1.86	9.28	1.94	.88
% Taking any medication	55%		44%		.51
% Smoking 10+ cigarettes per day	0%		7%		1.55
% Eating/Drinking within one hour of session	5%		4%		.05
% Consuming Caffeine within one hour of session	5%		0%		1.38
% Using Oral Contraceptives	0%		11%		2.37
% Using Alcohol the night before session	0%		7%		1.55
% Engaging in Strenuous exercise before session	25%		30%		.12
% of Daughters with History of Treatment	15%		44%		4.58*

Notes:
*$p < .05$

Table 7. *Means and standard deviations of main self-report variables.*

Variable	Low Risk *n*=20 Girls; *n*=19 Mothers			High Risk *n*=27 Girls; *n*=27 Mothers			
	M	*SD*	*Range*	*M*	*SD*	*Range*	*t*
Difficulty with Emotion Regulation	64.82	12.40	40-88	72.59	14.55	44-98	-1.92*
Interpersonal Competence:							
Initiating Relationships	3.62	.82	2.13-5	3.22	.75	2.13-4.88	1.74*
Providing Emotional Support	3.90	.48	3-4.75	3.85	.64	2.38-4.88	.31
Asserting Influence	3.61	.70	2.13-4.75	3.44	.55	2.50-4.50	.94
Self-Disclosure	3.28	.90	1.50-4.88	3.11	.76	1.63-4.50	.68
Conflict Resolution	3.55	.54	2.63-4.50	3.27	.74	1.75-4.88	1.40
Daughters' Depressive Symptoms	6.00	7.03	0-24	8.52	5.85	0-22	-1.34
Mothers' Depressive Symptoms	3.25	3.54	0-11	11.89	10.64	0-38	-3.94**

Notes: **p<.10; **<.01*

Table 8. *Means and standard deviations of TSST-C variables.*

Variable	Low Risk *n*=20 Girls; *n*=19 Mothers			High Risk *n*=27 Girls; *n*=27 Mothers			
	M	*SD*	*Range*	*M*	*SD*	*Range*	*t*
Cortisol at 25 minutes Pre Stress	1.13	.70	.28-2.50	.87	.37	.38-1.69	1.34
Cortisol at 10 minutes Pre Stress	1.11	.67	.43-2.68	.82	.34	.33-1.55	1.42
Cortisol at 0 minutes Pre Stress	1.00	.67	.28-2.60	.87	.34	.21-1.55	.57
Cortisol at 0 minutes Post Stress	1.05	.69	.27-2.49	.90	.36	.19-1.56	.75
Cortisol at 5 minutes Post Stress	1.46	.84	.33-3.33	.93	.39	.25-1.76	2.63*
Cortisol at 15 minutes Post Stress	1.53	.82	.43-3.54	.92	.41	.16-1.67	3.10**
Cortisol at 30 minutes Post Stress	1.31	.70	.41-2.79	.83	.32	.20-1.34	2.65*
Cortisol at 45 minutes Post Stress	1.12	.64	.25-2.60	.87	.37	.25-1.57	1.38
Visual Analog Scale 1	16.98	15.74	0-52	19.04	19.22	0-67	-.39
Visual Analog Scale 2	37.75	29.48	0-100	44.96	27.80	0-100	-.86
Visual Analog Scale 3	13.50	13.24	0-44	14.35	13.92	0-44	-.21

Notes: *p <.05; **p <.01

Table 9. *Correlations among the main (transformed) study variables.*

Variables	1	2	3	4	5	6	7	8	9	10	11
1 Risk Status		.25	.47**	.24	-.25	-.02	-.14	-.10	-.16	-.39**	.39**
2 Daughters Depressive Symptoms			.28	.66**	-.12	-.14	-.44**	-.18	-.44**	-.20	.10
3 Mothers Depressive Symptoms				.27	-.10	-.11	-.02	-.15	-.02	-.33*	-.32*
4 Total Emotion Regulation Difficulties					-.29*	-.31*	-.52**	-.31*	-.60**	-.31*	.25
5 Initiating Relationships (ICQ-R)						.64**	.44**	.64**	.52**	.16	-.16
6 Providing Emo. Support (ICQ-R)							.41**	.50**	.58**	.27	-.14
7 Asserting Influence (ICQ-R)								.34*	.46**	-.06	-.04
8 Self-Disclosure (ICQ-R)									.34*	.09	-.13
9 Conflict Resolution (ICQ-R)										.33*	-.06
10 Stress Reactivity (Percent Change)											-.62**
11 Stress Recovery (Percent Change)											

Notes:
Low Risk Group: n=20 girls and 19 mothers
High Risk Group: n=27 girls and 27 mothers
Risk Status: 0 = low risk; 1 = high risk
*p <.05; **p <.01

Table 10. *One-way ANOVA results examining age at first exposure to maternal depression.*

Dependent Variable	Low Risk Group *n* = 20 *M (SD)*	Before Age 5 *n* = 18 *M (SD)*	Age 5+ *n* = 6 *M (SD)*	*F*
Total Emotion Regulation Difficulties	64.82 (12.40)	69.89 (15.19)	75.33 (13.37)	1.55
Initiating Relationships (ICQ-R)	3.62 (.82)	3.36 (.70)	3.07 (.98)	1.29
Providing Emo. Support (ICQ-R)	3.90 (.48)	3.86 (.67)	3.73 (.73)	.19
Asserting Influence (ICQ-R)	3.61 (.70)	3.52 (.58)	3.11 (.44)	1.51
Self-Disclosure (ICQ-R)	3.28 (.90)	3.20 (.88)	3.00 (.53)	.24
Conflict Resolution (ICQ-R)	3.55 (.54)	3.86 (.67)	3.73 (.73)	1.25
Daughters Depressive Symptoms	6.00 (7.03)	7.06 (5.95)	10.67 (4.68)	1.25
Baseline Cortisol	1.08 (.66)	.85 (.27)	.74 (.36)	1.53
Stress Reactivity (Percent Change)	52.20 (60.92)	16.00 (41.97)	-14.09 (30.04)	4.87*
Stress Recovery (Percent Change)	-22.32 (27.50)	4.51 (39.83)	8.83 (29.93)	3.84*

Note:
*$p < .05$
All analyses were evaluated with $df = 2, 41$.

Table 11. *Correlations between chronicity of maternal depression and daughter variables.*

n = 24 mothers *n* = 24 daughters										
Variables	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Number of Maternal Depressive Episodes	.12	.27	-.23	-.16	.08	-.35	.08	-.11	.00	.02
Number of Months of Maternal Depression	.04	.10	.16	-.04	-.05	.05	-.13	.41*	-.18	.01

* $p = .05$

Note: 1=Daughters' Depressive Symptoms
2=Daughters Difficult with Emotion Regulation
3=Initiating Relationship (ICQ-R)
4=Providing Emotion Support (ICQ-R)
5=Asserting Influence (ICQ-R)
6=Self-Disclosure (ICQ-R)
7=Conflict Resolution (ICQ-R)
8=Baseline Cortisol
9=Stress Reactivity (Percent Change)
10=Stress Recovery (Percent Change)

Figure 1. Hypothesized mediating and moderating relationships.

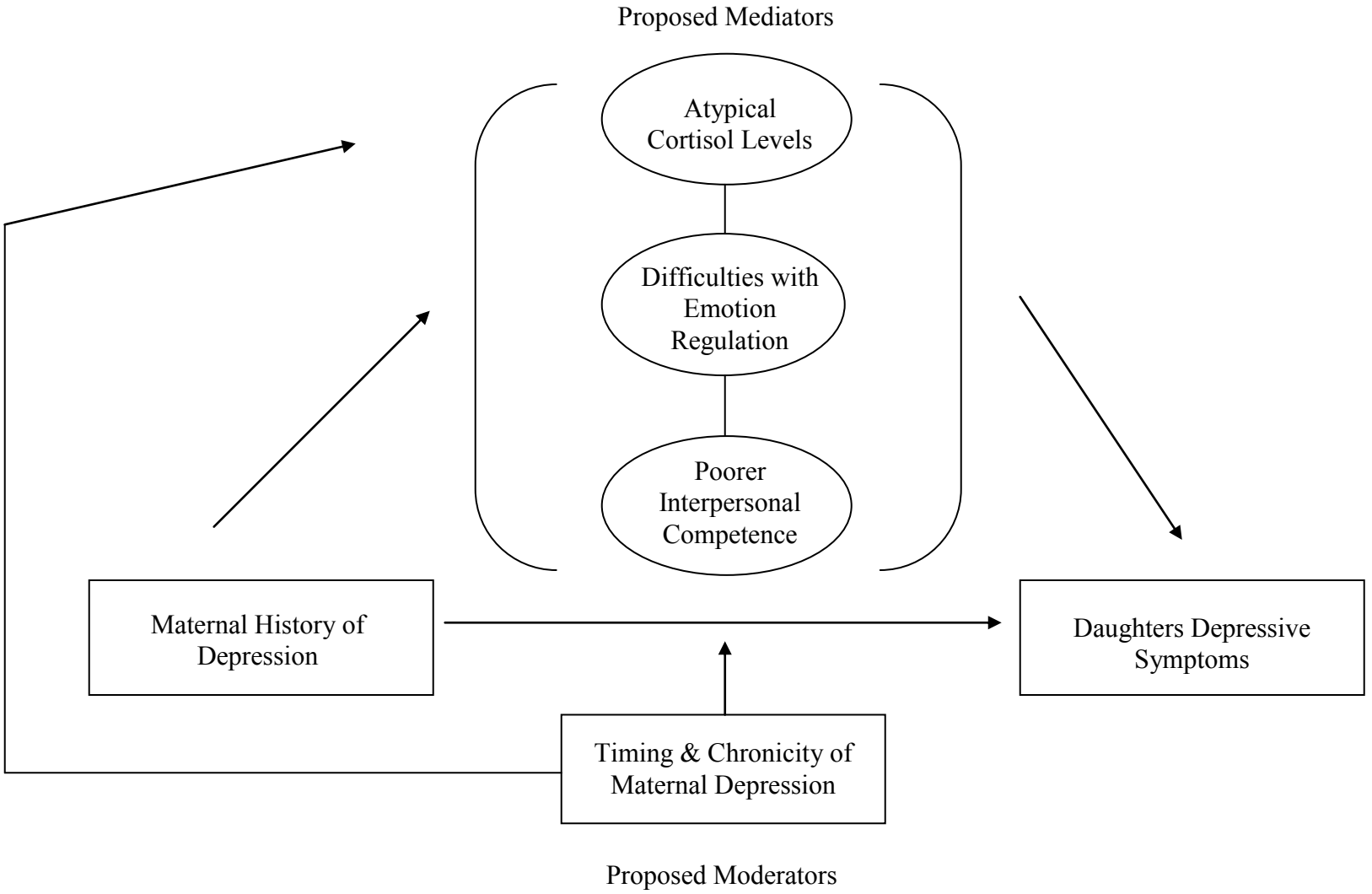


Figure 2. Visual Analog Scale (VAS) scores over time.

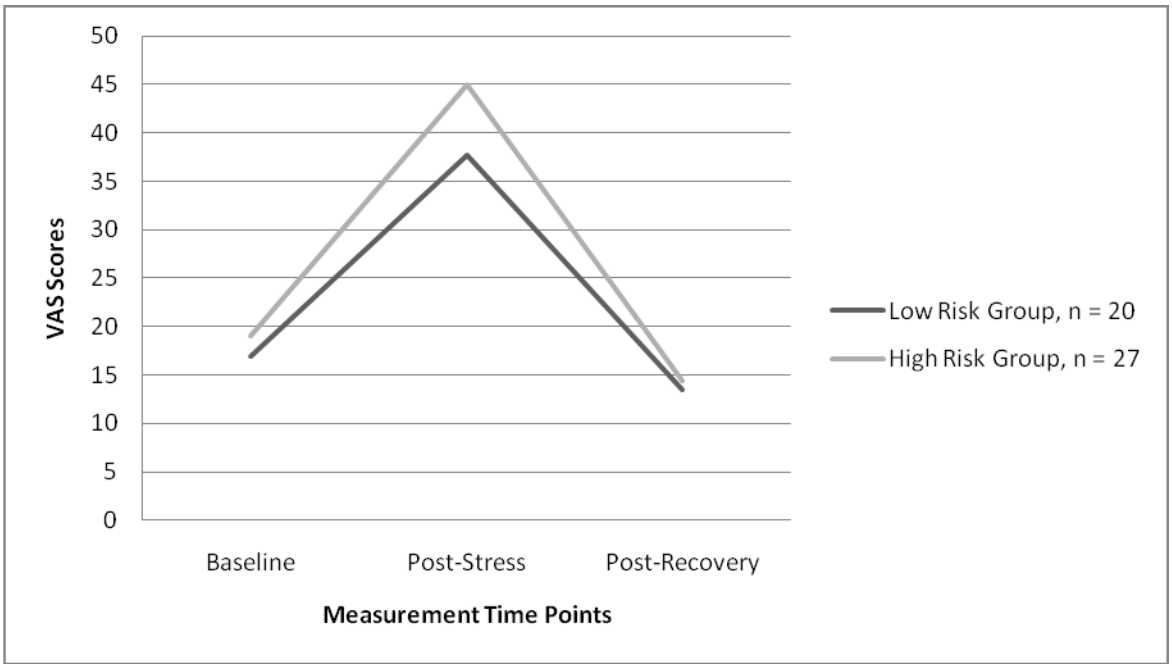


Figure 3. Cortisol levels pre and post TSST-C.

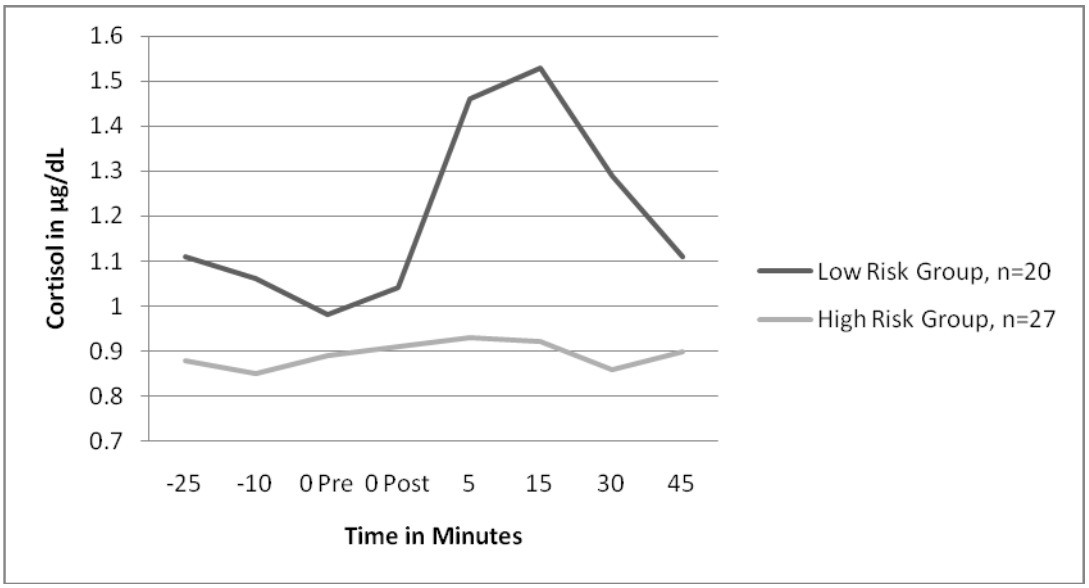


Figure 4. Cortisol levels pre and post TSST-C, controlling for mothers' current depressive symptoms.

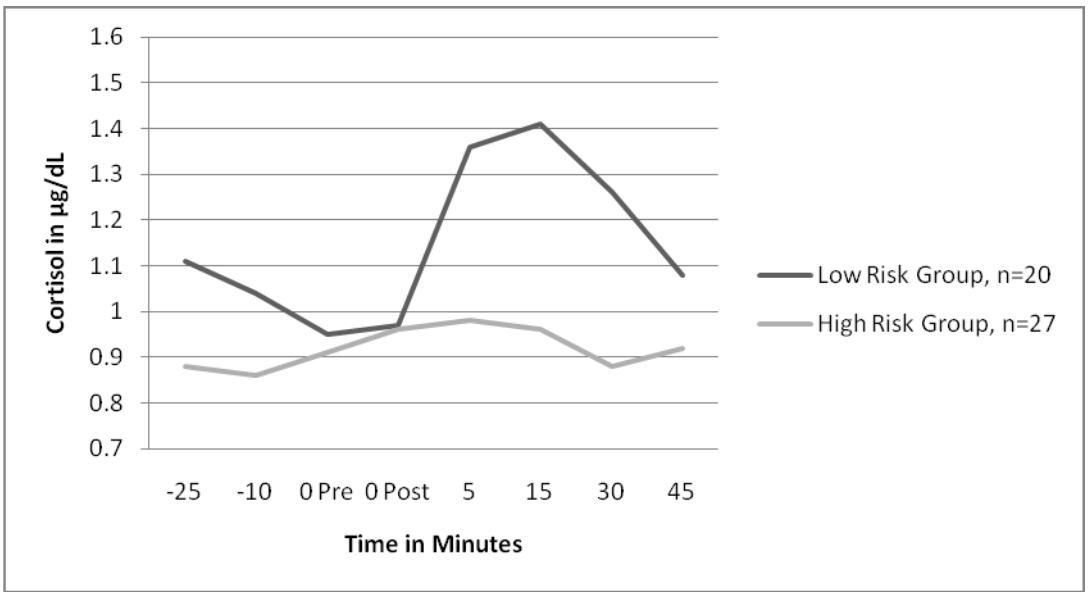
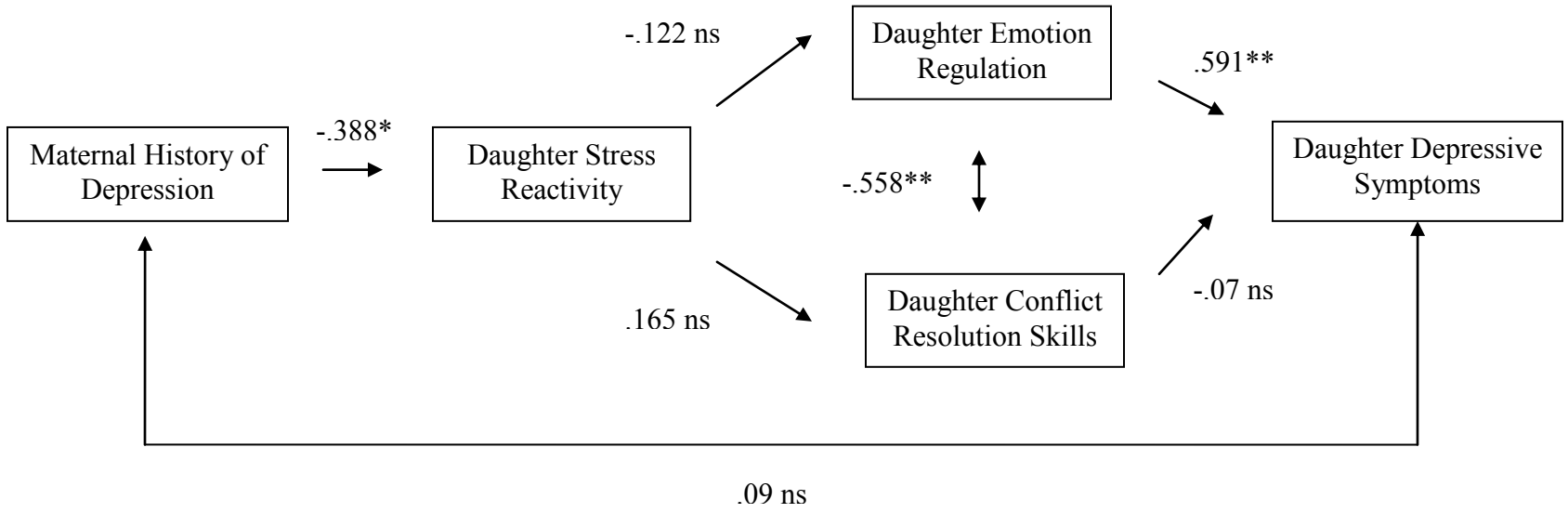
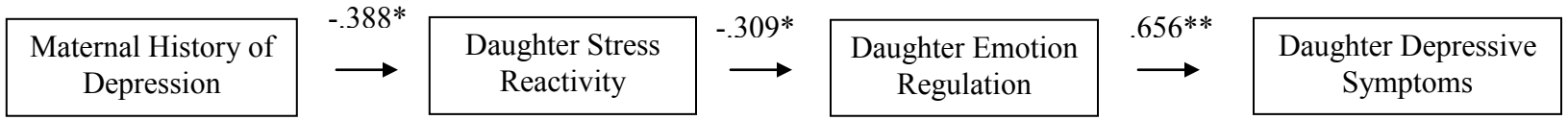


Figure 5. Full mediation model.



Notes:
Higher scores on the Emotion Regulation (ER) measure represent more difficulties with ER.
*p <.01
**p <.001

Figure 6. Trimmed model showing indirect effect of maternal history of depression on daughter current depressive symptoms.



Notes:
Higher scores on the Emotion Regulation (ER) measure represent more difficulties with ER.
*p <.05
**p <.001

References

Adrian, C. & Hammen, C. (1993). Stress exposure and stress generation in children of depressed mothers. *Journal of Consulting and Clinical Psychology, 61 (2)*, 354-359.

Aisa, B., Tordera, R., Lasheras, B., Del Rio, J., Ramirez, M.J. (2007). Cognitive impairment associated to HPA axis hyperactivity after maternal separation in rats. *Psychoneuroendocrinology, 32 (3)*, 256-266.

Alpern, L. & Lyons-Ruth, K. (1993). Preschool children at social risk: Chronicity and timing of maternal depressive symptoms and child behavior problems at school and at home. *Development and Psychopathology, 5 (3)*, 371-387.

Anisman, H. & Merali, Z. (1999). Understanding stress: Characteristics and caveats. *Alcohol Research and Health, 23 (4)*, 241-249.

Ashman, S.B. & Dawson, G. (2002). Maternal depression, infant psychobiological development, and risk for depression. In S.H. Goodman & I.H. Gotlib (Eds.), *Children of depressed parents: Mechanisms of risk and implications for treatment* (pp.37-58). Washington, DC: American Psychological Association.

Ashman, S.B., Dawson, G., Panagiotides, H., Yamada, E., & Wilkinson, C.W. (2002). Stress hormone levels of children of depressed parents. *Development and Psychopathology, 14*, 333-349.

Ashman, S.B., Dawson, G., & Panagiotides, H. (2008). Trajectories of maternal depression over 7 years: Relations with child psychophysiology and behavior and role of contextual risks. *Development and Psychopathology, 20*, 55-77.

Azar, R., Paquette, D., Zoccollilo, M., Baltzer, F., & Tremblay, R.E. (2007). The association of major depression, conduct disorder, and maternal overcontrol with a

failure to show a cortisol buffered response in 4-month old infants of teenage mothers. *Biological Psychiatry, 62*, 573-579.

Bartels, M., Van den Burg, M., Sluyter F., Boomsa, D.I. & de Geus, E.J.C. (2003). Heritability of cortisol levels: Review and simultaneous analysis of twin studies. *Psychoeneuroendocrinology, 28*, 121-137.

Battaglia, M, Bajo, S., Ferini Strambi, L., Brambilla, F., Castronovo, C., Vanni, G., & Belodi, L. (1997). Physiological and behavioural responses to minor stressors in offspring of patients with panic disorder. *Journal of Psychiatry Research, 31 (3),* 365-376.

Beck, A.T., Steer, R.A., & Brown, G.K. (1996). *Manual for the Beck Depression Inventory-II*. San Antonia, Texas: Psychological Corporation.

Bell, K.L., & Calkins, S.D. (2000). Relationships as inputs and outputs of emotion regulation. *Psychological Inquiry, 11 (3)*, 160-209.

Berndt, E.R., Koran, L.M., Finkelstein, S.N. Gelenberg, A.J., Kornstein, S.G., Miller, I.M., Thase, M.E., Trapp, G.A., Keller, M.B. (2000). Lost human capital from early onset chronic depression. *American Journal of Psychiatry, 157*, 940-947.

Berndt, T.J. (1982). The features and effects of friendships in early adolescence. *Child Development, 53*, 1447-1460.

Betts, J., Gullone, E., Allen, J.S. (2009). An examination of emotion regulation, temperament, and parenting style as potential predictors of adolescent depression risk status: A correlational study. *British Journal of Developmental Psychology, 27*, 473-485.

Blandon, A.Y., Calkins, S.D., Keane, S.P., & O'Brien, M. (2008). Individual differences in

trajectories of emotion regulation processes: The effects of maternal depressive symptomatology and children's physiological regulation. *Developmental Psychology, 44 (4)*, 1110-1123.

Bradley, S.J. (2000). *Affect Regulation and the Development of Psychopathology*. New York: Guilford Press.

Brennan, P.A., Pargas, R., Walker, E.F., Green, P., Newport, D.J., & Stowe, Z. (2008). Maternal depression and infant cortisol: Influences of timing, comorbidity, and treatment. The *Journal of Child Psychology and Psychiatry, 49 (10)*, 1099-1107.

Brennan, P.A., Hammen, C., Anderson, M.J., Bor, W., Najman, J.M., & Williams, G.M. (2000). Chronicity, severity, and timing of maternal depressive symptoms: Relationships with child outcomes at age 5. *Developmental Psychology, 36 (6)*, 759-766.

Brennan, P.A., Hammen, C., Katz, A.R., & Le Brocque, R.M. (2002). Maternal depression, paternal psychopathology, and adolescent diagnostic outcomes. *Journal of Consulting and Clinical Psychology, 70 (5)*, 1075-1085.

Bromberger, J.T., Kravitz, H.M., Matthews, K., Youk, A., Brown, C., & Feng, W. (2009). Predictors of first lifetime episodes of major depression in midlife women. *Psychological Medicine: A Journal of Research in Psychology and Allied Sciences, 39 (1)*, 55-64.

Bugental, D.B., Martorell, G.A., Barraza, V. (2003). The hormonal costs of subtle forms of infant maltreatment. *Hormones and Behavior, 43*, 237-244.

Buhrmester, D. (1990). Intimacy of friendship, interpersonal competence, and adjustment during preadolescence and adolescence. *Child Development, 61*, 1101-1111.

Buhrmester, D. (2002, April). *The development of interpersonal competence during adolescence: A six-year longitudinal study*. Poster session presented at the Society for Research on Adolescence Conference.

Bukowski, W.M. & Adams, R. (2005). Peer relationships and psychopathology: Markers, moderators, mediators, mechanisms, and meanings. *Journal of Clinical Child and Adolescent Psychology, 34 (1)*, 3-10.

Burke, H.M., Davis, M.C., Otte, C., & Mohr, D.C. (2005). Depression and cortisol responses to psychological stress: A meta-analysis. *Psychoneuroendocrinology, 30*, 846-856.

Buske-Kirschbaum, A., Jobst, S., Wustmans, A., Kirschbaum, C., Raugh, W., & Hellhammer, D. (1997). Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. *Psychosomatic Medicine, 59 (4)*, 419-426.

Calkins, S.D. (1994). Origins and outcomes of individual differences in emotion regulation. In N.A. Fox (Ed.), The development of emotion regulation: Biological and behavioural considerations (pp.53-72). *Monographs for the Research on Child Development, 59 (Nos. 2-3, Serial No. 240).*

Calkins, S.D., Gill, K.L., Johnson, M.C., & Smith, C.L. (1999). *Social Development, 8 (3)*, 310-334.

Campbell, S.B., Cohn, J.F., & Meyers, T. (1995). Depression in first-time mothers: mother-infant interaction and depression chronicity. *Developmental Psychology, 31 (3)*, 349-357.

Carpenter, L.L., Carvalho, J.P., Tyrka, A.R., Wier, L.M., Mello, A.F., Mello, M.F. et al., (2007). Decreased adrenocorticotropic hormone and cortisol responses to stress in

healthy adults reporting significant childhood maltreatment. *Biological Psychiatry, 62*, 1080-1087.

Carpenter, L.L., Tyrka, A.R., Ross, N.S, Khoury, L., Anderson, G.M., &Price, L.H. (2009). Effect of childhood emotional abuse and age on cortisol responsivity in adulthood. *Biological Psychiatry, 66*, 69-75.

Cichetti, D., Ganiban, J., & Barnett, D. (1991). Contributions from the study of high risk populations to understanding the development of emotion regulation. In J. Garber & K.A. Dodge (Eds.), *The development of emotion regulation and dysregulation* (pp.15-48). New York: Cambridge University Press.

Claes, S.J. (2004). Corticotropin-releasing hormone (CRH) in psychiatry: from stress to psychopathology. *Annals of Medicine, 36,* 50-61.

Cohn, J.F & Tronick, T. (1989). Specificity of infants' response to mothers' affective behaviour. *Journal of the American Academy of Child and Adolescent Psychiatry, 28 (2)*, 242-248.

Cole, D.A. (1990). Relation of social and academic competence to depressive symptoms in childhood. *Journal of Abnormal Psychology, 99 (4)*, 422-429.

Cole, P.M., Michel, M.K., & Teti, L.O. (1994). The development of emotion regulation and dysregulation: A clinical perspective. In N.A. Fox (Ed.), The development of emotion regulation: Biological and behavioural considerations (pp.73-100). *Monographs for the Research on Child Development, 59 (Nos. 2-3, Serial No. 240).*

Cole, D.A., Jacquez, F.M., & Maaschman, T.L. (2001). Social origins of depressive cognitions: A longitudinal study of self-perceived competence in children. *Cognitive Therapy and Research, 25 (4)*, 377-395.

Comninos, A. & Grenyer, B.F.S. (2007). The influence of interpersonal factors on the speed of recovery from major depression. *Psychotherapy Research, 17 (2)*, 230-239.

Connell, A.M. & Goodman, S.H. (2002). The association between psychopathology in fathers versus mothers and children's internalizing and externalizing behaviour problems: A meta analysis. *Psychological Bulletin, 128 (5)*, 746-773.

Contreras, J.M., Kerns, K.A., Weimer, B.L., Gentzler, A.L., & Tomich, P.L. (2000). Emotion regulation as a mediator of associations between mother-child attachment and peer relationships in middle childhood. *Journal of Family Psychology, 14 (1)*, 111-124.

Coonie, C.B. (1991). *The Face on the Milk Carton*. New York: Bantam Doubleday Dell Publishing Group, Inc.

Coyne, J.C. (1976). *Toward an interactional description of depression*. Psychiatry, 39, 28-40.

Cummings, M.E. & Davies, P.T. (1994). Maternal depression and child development. *Journal of Child Psychology and Psychiatry, 35 (1)*, 73-112.

Cyranowski, J.M., Frank, E. Young, E. & Shear, M.K. (2000). Adolescent onset of the gender difference in lifetime rates of major depression. Archives of General Psychiatry, 57, 21-27.

Daley, S.E. & Hammen, C. (2002). Depressive symptoms and close relationships during the transition to adulthood: Perspective from dysphoric women, their best friends, and their romantic partners. *Journal of Consulting and Clinical Psychology, 70 (1)*, 129-141.

Daniels, W.M.U., Pietersen, C.Y., Carstens, M.E., & Stein, D.J. (2004). Maternal separation

in rats leads to anxiety-like behavior and a blunted ACTH response and altered neurotransmitter levels in response to a subsequent stressor. *Metabolic Brain Disease, 19 (1/2)*, 3-14.

Davila, J., Hammen, C., Burge, D., Paley, B., & Daley, S.E. (1995). Poor interpersonal problem solving as a mechanism of stress generation in depression among adolescent women. *Journal of Abnormal Psychology, 104 (4)*, 592-600.

Davis, E.P., Glynn, L.M., Schetter, C.D., Hobel, C., Chicz-Demet, A., & Sandman, C.A. (2007). Prenatal exposure to maternal depression and cortisol influences infant temperament. *Journal of the American Academy of Child and Adolescent Psychiatry, 46(6)*, 737-746.

Dawson, G. & Ashman, S.B. (2000). On the origins of a vulnerability to depression: The influence of the early social environment on the development of psychobiological systems related to risk for affective disorder. In C.A. Nelson (Ed.), The effects of early adversity on neurobehavioral development. *The Minnesota Symposia on Child Psychology, 31*, 245-279.

Dawson, G., Ashman, S.B., Panagiotides, H., Hessl, D., Self, J., Yamada, E., & Embry, L. (2003). Preschool outcomes of children of depressed mothers: Role of maternal behavior, contextual risk, and children's brain activity. *Child Development, 74 (4)*, 1158-1175.

DeBellis, M.D. & Putnam, F. (1994). The psychobiology of childhood maltreatment. *Child and Adolescent Psychiatric Clinics of North America, 3*, 1-16.

De Bellis, M.D. (2001). Developmental traumatology: The psychobiological development of maltreated children and its implications for research, treatment, and policy.

Development and Psychopathology, 13, 539-564.

Denham, S.A & Grout, L. (1993). Socialization of emotion: Pathway to preschoolers' emotional and social competence. *Journal of Nonverbal Behaviour, 17 (3)*, 205-227.

Denham, S.A., Mitchell-Copeland J., Strandberg, K., Auerbach, S., & Blair, K. (1997). Parental contributions to preschoolers' emotional competence: Direct and Indirect effects. *Motivation and Emotion, 21*, 65-86.

Denham, S.A., Blair, K.A., DeMulder, E., Levitas J., Sawyer, K., Aurbach-Major, S., & Queenan, P. (2003). Preschool emotional competence: Pathway to social competence? *Child Development, 74 (1)*, 238-256.

Dettling, A.C., Feldon, J., & Pryce, C.R. (2002). Repeated parental deprivation in the infant common marmoset (Callithrix Jacchus, Primates) and analysis of its effects on early development. *Biological Psychiatry, 52*, 1037-1046.

Dickerson, S.S. & Kemeny, M.E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130 (1)*, 355-391.

Diego, M.A., Field, T., Hernandez-Reif, M., Cullen, C., Schanberg, S. & Kuhn, C. (2004). Prepartum, postpartum, and chronic depression effects on newborns. *Psychiatry, 67 (1)*, 63-80.

Diego, M.A., Field, T., & Hernandez-Reif, M. (2005). Prepartum, postpartum, and chronic depression effects on neonatal behavior. *Infant Behavior and Development, 28*, 155-164.

Dienstbier, R.A. (1989). Arousal and physiological toughness: Implications for medical and physical health. *Psychological Review, 96 (1)*, 84-100.

Dietz, L.J., Jennings, K.D. & Abrew, A.J. (2005). Social skill in self-assertive strategies of toddlers with depressed and nondepressed mothers. *The Journal of Genetic Psychology, 166 (1)*, 94-116.

Dietz, L.J., Jennings, K.D., Kelley, S.A., & Marshal, M. (2009). Maternal depression, paternal psychopathology, and toddlers' behavior problems. *Journal of Clinical Child and Adolescent Psychology, 38 (1)*, 48-61.

Dorn, L.D., Campo, J.C., Thato, S., Dahl, R.E., Lewin, D., Chandra, R., & Di Lorenzo, C. (2003). Psychological comorbidity and stress reactivity in children and adolescents with abdominal pain and anxiety disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 42 (1)*, 66-75.

Downey, G. & Coyne, J.C. (1990). Children of depressed parents: An integrative review. *Psychological Bulletin, 108 (1)*: 50-76.

Dozois, D.J. A., Dobson, K.S., & Ahnberg, J.L. (1998). A psychometric evaluation of the Beck Depression Inventory-II. *Psychological Assessment, 10 (2)*, 83-89.

Eberhart, N.K. & Hammen, C.L. (2006). Interpersonal predictors of onset of depression during the transition to adulthood. *Personal Relationships, 13*, 195-206.

Eberhart, N.K., Shih, J.H., Hammen, C.L., & Brennan, P.A. (2006). Understanding the sex difference in vulnerability to adolescent depression: An examination of child and parent characteristics. *Journal of Abnormal Child Psychology, 34 (4)*, 495-508.

Ehlert, U., Gaab, J., & Heinrichs, M. (2001). Psychoneuroendocrinological contributions to the etiology of depression, post-traumatic stress disorder, and stress-related bodily disorders: The role of the hypothalamus-pituitary-adrenal axis. *Biological Psychology, 57 (1-3)*, 141-152.

Ehring, T., Fischer, S., Schnulle, J., Bosterling, A., & Tuschen-Caffier, B. (2008). Characteristics of emotion regulation in recovered depressed versus never depressed individuals. *Personality and Individual Differences, 44*, 1574-1584.

Eisenberg, N., Fabes, R.A., & Losoya, S. (1997). Emotional responding: Regulation, social correlates, and socialization. In P. Salovey & D.J. Sluyter (Eds.), *Emotional Development and Emotional Intelligence: Educational Implications* (pp.129-167). New York: Basic Books.

Ellenbogen, M.A., Hodgins, S., & Walker, C.-D. (2004). High levels of cortisol among adolescent offspring of parents with bipolar disorder: A pilot study. *Psychoneuroendocrinology, 29*, 99-106.

Ellenbogen, M.A., Hodgins, S., Walker, C.-D., Couture, S., & Adam, S. (2006). Daytime cortisol and stress reactivity in the offspring of parents with bipolar disorder. *Psychoneuroendocrinology, 31*, 1164-1180.

Ellenbogen, M.A. & Hodgins, S. (2009). Structure provided by parents in middle childhood predicts cortisol reactivity in adolescence among the offspring of parents with bipolar disorder and controls. *Psychoneuroendocrinology, 34*, 773-785.

Ellenbogen, M.A., Santo, J.B., Linnen, A.-M, Walker, C.-D., & Hodgins, S. High cortisol levels in the offspring of parents with bipolar disorder during two weeks of daily sampling. *Bipolar Disorders, 12*, 77-86.

Elzinga, B.M., Roelofs, K., Tollenaar, M.S., Bakvis, P., van Pelt, J., & Spinhoven, P. (2008). Diminished cortisol responses to psychosocial stress associated with lifetime adverse events: A study among healthy young subjects. *Psychoneuroendocrinology, 33*, 227-237.

Essau, C.A., Conradt, J., Petermann, F. (2000). Course and outcome of depressive disorders. In: C.A. Essau & F. Petermann (Eds.), *Depressive disorders in children and adolescents: Epidemiology, risk factors, and treatment* (pp.105-135). New Jersey: Jason Aronson, Inc.

Essex, M.J., Klein, M.H., Miech, R., & Smider, N.A. (2001). Timing of initial exposure to maternal major depression and children's mental health symptoms in kindergarten. *British Journal of Psychiatry, 179*, 151-156.

Essex, M.J., Klein, M.H., Cho, E., & Kalin, N.H. (2002). Maternal stress beginning in infancy may sensitize children to later stress exposure: Effects on cortisol and behavior. *Biological Psychiatry, 52 (8)*, 776-784.

Ewell Foster, C., Webster, M.C., Weissman, M.M., Pilowsky, D.J., Wickramaratne, P.J., Rush, A.J. et al. (2008). Course and severity of maternal depression: Associations with family functioning and child adjustment. *Journal of Youth and Adolescence, 37*, 906-916.

Fehm-Wolfsdorf, G., Groth, T., Kaiser, A., Hahlweg, K. (1999). Cortisol responses to marital conflict depend on marital interaction quality. *International Journal of Behavioral Medicine, 6 (3)*, 207-227.

Feldman, R., Granat, A., Pariente, C., Kanety, H., Kuint, J., & Gilboa-Schechtman, E. (2009). Maternal depression and anxiety across the postpartum year and infant social engagement, fear regulation, and stress reactivity. *Journal of the American Academy of Child and Adolescent Psychiatry, 48 (9)*, 919-927.

Feng, X., Keenan, K., Hipwell, A.E., Henneberger, A.K., Rischall, M.S., Butch, J., Coyne, C., Boeldt, D., Hinze, A.K., & Babinski, D.E. (2009). Longitudinal associations

between emotion regulation and depression in preadolescent girls: Moderation by the caregiving environment. *Developmental Psychology, 45 (3)*, 798-808.

Field, T. (1994). The effects of mothers physical and emotional unavailability on emotion regulation. *Monographs of the Society for Research in Child Development, 59 (2/3)*, 208-227.

Field, T. (1995). Infants of depressed mothers. *Infant Behavior and Development, 18 (1),* 1-13.

Field, T., Diego, M., Hernandez-Reif, M., Vera, Y., Gil, K., Schanberg, S., & Kuhn, C. (2004). Prenatal maternal biochemistry predicts neonatal biochemistry. *International Journal of Neuroscience, 114*, 933-945.

Fiocco, A.J., Joober, R., & Lupien, S. J. (2007). Education modulates cortisol reactivity to the Trier Social Stress Test in middle-aged adults. *Psychoneuroendocrinology, 32*, 1158-1163.

First, M., Spitzer, R. L., Gibbon, M., & Williams, J. B. (1994). *Structured Clinical Interview for DSM-IV--Non Patient*. New York: New York State Psychiatric Institute, Biometrics Research Department.

Flett, G.L., Bankstein, K.R., & Obertynski, M. (1996). Affect intensity, coping styles, mood regulation expectancies, and depressive symptoms. *Personality and Individual Differences, 20 (2)*, 221-228.

Francis, D., Diorio, J., Liu, D., & Meaney, M.J. (1999). Nongenomic transmission across generations of maternal behavior and stress responses in the rat. *Science, 286*, 1155-1158.

Fries, E., Hesse, J., Hellhammer, J., & Hellhammer, D.H. (2005). A new view on

hypocortisolism. *Psychoneuroendocrinology, 30*, 1010-1016.

Garber, J., Braafladt, N., & Zeman, J. (1991). The regulation of sad affect: An information processing perspective. In J. Garber & K.A. Dodge (Eds.), *The development of emotion regulation and dysregulation* (pp.208-240). New York: Cambridge University Press.

Garnefski, N., Kraaij, V., & Spinhoven, P. (2001). Negative life events, cognitive emotion regulation, and emotional problems. *Personality and Individual Differences, 30*, 1311-1327.

Garnefski, N., Van Den Kommer, T., Kraaij, V., Teerds, J., Legerstee, J., Onstein, E. (2002). The relationship between cognitive emotion regulation and emotional problems: Comparison between a clinical and nonclinical sample. European Journal of Personality, 16, 403-420.

Garnefski, N., Boon, S., & Kraaij, V. (2003). Relationships between cognitive strategies of adolescents and depressive symptomatology across different types of life event. *Journal of Youth and Adolescence, 32 (6)*, 401-408.

Goodman, S.H. & Gotlib, I.H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review, 106 (3)*, 458-490.

Goodman, S. H. (2002). Depression and early adverse experiences. In I.H. Gotlib & C.L. Hammen (Eds.), *Handbook of Depression* (pp. 245-267). New York: Guilford Press.

Goodman, S.H., Brogan, D., Lynch, E., & Fielding, B. (1993). Social and emotional competence in children of depressed mothers. *Child Development, 64*, 516-531.

Goodyer, I.M., Herbert, J., Tamplin, A., & Altham, P.M.E. (2000). Recent life events,

cortisol, dehydroepiandrosterone and the onset of major depression in high-risk adolescents. *British Journal of Psychiatry, 177*, 499-504.

Gotlib, I.H., Joorman, J., Minor, K.L., & Hallmayer, J. (2008). HPA axis reactivity: A mechanism underlying the associations among 5-HTTLPR, stress, and depression. *Biological Psychiatry, 63*, 847-851.

Gottman, J. (2001). Meta-emotion, children's emotional intelligence, and buffering children from marital conflict. In C.D. Ryff & B.H. Singer (Eds.), *Emotion, social relationships, and health* (pp. 23-40). New York: Oxford University Press

Gottman, J.M., Fainsilber Katz, L., & Hooven, C. (1996). Parental meta-emotion philosophy and the emotional life of families: Theoretical models and preliminary data. *Journal of Family Psychology, 10 (3)*, 243-268.

Gratz, K.L. & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation. Development, factor structure, and initial validation of the Difficulties in Emotion Regulation Scale. *Journal of Psychopathology and Behavioral Assessment, 26 (1)*, 41-54.

Gross, J.J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*, 281-291.

Gross, J.J. & John, O.P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85 (2)*, 348-362.

Gross, J.J. & Munoz, R.F. (1995). Emotion regulation and mental health. *Clinical Psychology: Science and Practice, 2 (2)*, 151-164.

Gump, B.B., Reihman, J., Stewart, P., Lonky, E., Darvill, T., Granger, D.A., & Matthew,

K.A. (2009). Trajectories of maternal depressive symptoms over her child's life span: Relation to adrenocortical, cardiovascular, and emotional functioning in children. *Development and Psychopathology, 21*, 207-225.

Gunnar, M.R. & Quevedo, K.M. (2008). Early care experiences and HPA axis regulation in children: A mechanism for later trauma vulnerability. *Progress in Brain Research, 167*, 137-147.

Halligan, S.L., Herbert, J., Goodyer, I.M., & Murray, L. (2004). Exposure to postnatal depression predicts elevated cortisol in adolescent offspring. *Biological Psychiatry, 55*, 376-381.

Halligan, S.L., Herbert, J., Goodyer, I.M., & Murray, L. (2007). Disturbances in morning cortisol secretion in association with maternal postnatal depression predict subsequent depressive symptomatology in adolescents. *Biological Psychiatry, 62*, 40-46.

Hammen, C. (2006). Stress generation in depression: Reflections on origins, research, and future directions. *Journal of Clinical Psychology, 62 (9)*, 1062-1082.

Hammen, C. & Brennan, P.A. (2002). Interpersonal dysfunction in depressed women: Impairments independent of depressive symptoms. *Journal of Affective Disorders, 72 (2)*, 145-156.

Hammen, C. & Brennan, P.A. (2001). Depressed adolescents of depressed and nondepressed mothers: Tests of an interpersonal impairment hypothesis. *Journal of Consulting and Clinical Psychology, 69 (2)*, 284-294.

Hammen, C. (1991). Generation of stress in the course of unipolar depression. *Journal of Abnormal Psychology, 100 (4)*, 555-561.

Hammen, C., Shih, J., Altman, T., & Brennan, P.A. (2003). Interpersonal impairment and the prediction of depressive symptoms in adolescent children of depressed and nondepressed mothers. *Journal of the American Academy of Adolescent Psychiatry, 42 (5)*, 571-577.

Hammen, C. & Brennan, P.A. (2003). Severity, chronicity, and timing of maternal depression and risk for adolescent offspring diagnoses in a community sample. *Archives of General Psychiatry, 60 (3)*, 253-258.

Hammen, C., Shih, J.H., & Brennan, P.A. (2004). Intergenerational transmission of depression: Test of an interpersonal stress model in a community sample. *Journal of Consulting and Clinical Psychology, 72 (3)*, 511-522.

Hammen, C., Brennan, P.A., & Keenan-Miller, D. (2008). Patterns of adolescent depression to age 20: The role of maternal depression and youth interpersonal dysfunction. *Journal of Abnormal Child Psychology, 36*, 1189-1198.

Hammen, C., Brennan, P.A., Kennan-Miller, D., & Herr, N.R. (2008). Early onset recurrent subtype of adolescent depression: Clinical and psychosocial correlates. *Journal of Child Psychology and Psychiatry, 49 (4),* 433-440.

Hammen, C., Gordon, D., Burge, D., Adrian, C., Jaenicke, C., & Hiroto, D. (1987). Maternal affective disorders, illness, and stress: Risk for children's psychopathology. *American Journal of Psychiatry, 144*, 736-741.

Hanrahan, K., McCarthy, A.M., Kleiber, C., Lutgendorf, S., & Tsalikian, E. (2006). Strategies for salivary cortisol collection and analysis in research with children. *Applied Nursing Research, 19*, 95-101.

Harris, T.O., Borsyani, S. Messari, K., Cleary, S.E., Shiers, H.M., Brown, G.W., & Herbert,

J. (2000). Morning cortisol as a risk factor for subsequent major depressive disorder in adult women. *British Journal of Psychiatry, 177*, 505-510.

Hart, J., Gunnar, M., Cicchetti, D. (1995). Salivary cortisol in maltreated children: Evidene of relations between neuroendocrine activity and social competence. *Development and Psychopathology, 7*, 11-26.

Heim, C., Newport, D.J., Heit, S., Graham, Y.P., Wilcox, M., Bonsall, R., et al. (2000a). Pituitary adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *Journal of the American Medical Association, 284*, 592-597.

Heim, C., Ehlert, U., & Hellhammer, D.H. (2000b). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology, 25*, 1-35.

Heim, C., Newport, D.J., Bonsall, R., Miller, A.H., & Nemeroff, C.B. (2001). Altered pituitary-adrenal axis responses to provocative challenge tests in adult survivors of childhood abuse. *American Journal of Psychiatry, 158*, 575-581.

Heim, C., Plotsky, P.M., & Nemeroff, C.B. (2004). Importance of studying the contributions of early adverse experience to neurobiological findings in depression. *Neuropsychopharmacology, 24*, 641-648.

Heim, C., Mletzko, T., Purselle, D., Musselman, D.L., Nemeroff, C.B. (2008). The dexamethasone/corticotropin releasing factor test in men with major depression: Role of childhood trauma. *Biological Psychiatry, 63*, 398-405.

Hellhammer D.H. & Wade, S. (1993). Endocrine correlates of stress vulnerability. *Psychotherapy and Psychosomatics, 60*, 8-17.

Herzberg, D.S., Hammen, C., Burge, D., Daley, S.E., Davila, J., & Lindberg, N. (1998). Social competence as a predictor of chronic interpersonal stress. *Personal Relationships, 5*, 207-218.

Hoffman, C., Crnic, K.A., & Baker, J.K. (2006). Maternal depression and parenting: Implications for children's emergent emotion regulation and behavioral functioning. *Parenting: Science and Practice, 6 (4)*, 271-295.

Hoge, R.D. (1999). *Assessing adolescents in educational, counselling, and other settings*. London: Lawrence Earlbaum Associates

Holsboer, F., Lauer, C.J., Schreiber, W., Kreig, J.-C. (1995). Altered hypothalamic-pituitary-adrenocortical regulation in healthy subjects at high familial risk for affective disorder. *Neuroendocrinology, 62*, 340-347.

Huot, R.L., Brennan, P.A., Stowe, Z.N., Plotsky, P.M., & Walker, E.F. (2004). Negative affect in offspring of depressed mothers is predicted by infant cortisol levels at 6 months and maternal depression during pregnancy, but not postpartum. *Annals of the New York Academy of Science, 1032*, 234-236.

Johnson, S.A., Fournier, N.M., & Kalynchuk, L.E. (2006). Effect of different doses of corticosterone on depression-like behaviour and HPA axis responses to a novel stressor. *Behavioural Brain Research, 168*, 280-288.

Joiner, T.E. (1999). A test of interpersonal theory of depression in youth psychiatric inpatients. *Journal of Abnormal Child Psychology, 27 (1)*, 77-85.

Joiner, T. & Coyne, J.C. (1999). *The interactional nature of depression*. Washington: American Psychological Association.

Kalinichev, M., Easterling, K.W., Plotsky, P.M., & Holtzman, S.G. (2002). Longlasting

changes in corticosterone response and anxiety-like behaviours as a consequence of neonatal maternal separation in Long-Evans rats. *Pharmacology, Biochemistry, and Behavior, 73 (1)*, 131-140.

Kaufman, J., Birmaher, B., Brent, D., Roa, U., Flynn, C., Moreci, P., et al. (1997). Schedule for affective disorders and schizophrenia for school-age children – present and lifetime version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child and Adolescent Psychiatry, 36 (7)*, 980-988.

Kendler, K.S, Neale, M.C., Kessler, R.C., Heath, A.C., Eaves, L.J. (1993). The lifetime history of major depression in women: reliability of diagnosis and heritability. *Archives of General Psychiatry*, 50 (11), 863-870.

Kendler, K.S., Gardner, C.O., Prescott, C.A. (1999). Clinical characteristics of major depression that predict risk of depression in relatives. *Archives of General Psychiatry,* 56, 322-327.

King, S.L. & Hegadoren, K.M. (2002). Stress hormones: How do they measure up? *Biological Research for Nursing, 4 (2)*, 92-103.

Kirschbaum, C. & Hellhammer, D.H. (1994). Salivary cortisol in psychoneuroendocrine research: Recent developments and applications. *Psychoneuroendocrinology, 19 (4)*, 313-333.

Kirschbaum, C., Pirke, K-M. & Hellhammer, D.H. (1993). The "Trier Social Stress Test": A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28 (1-2)*, 76-81.

Koblinsky, S.A., Kuvalanka, K.A., & Randoph, S.J. (2006). Social skills and behavior problems of urban, African American preschoolers: Role of parenting practice,

family conflict, and maternal depression. *American Journal of Orthopsychiatry, 76 (4)*, 554-563.

Kraaij, V., Garnefski, N., Jan de Wilde, E., Dijkstra, A., Gebhardt, W., Maes, S., & ter Doest, L. (2003). Negative life events and depressive symptoms in late adolescence: Bonding and cognitive coping as vulnerability factors? *Journal of Youth and Adolescence, 32 (3)*, 185-193.

Krefetz, D.G., Steer, R.A. Gulab, N.A., & Beck, A.T. (2002). Convergent validity of the Beck Depression Inventory-II with the Reynolds Adolescent Depression Scale in psychiatric inpatients. *Journal of Personality Assessment, 78 (3)*, 451-460.

Kudielka, B.M., Hellhammer, D.H., & Wüst, S. (2009). Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology, 34*, 2-18.

La Greca, A.M. & Moore Harrison, H. (2005). Adolescent peer relationships, friendships, and romantic relationships: Do they predict social anxiety and depression? *Journal of Clinical Child and Adolescent Psychology, 34 (1)*, 49-61.

Liu, D., Diorio, J., Tannenbaum, B., Caldji, C., Francis, D., Freedman, A., Sharma, S., Pearson, D., Plotsky, P.M., & Meaney, M.J. (1997). Maternal care, hippocampal glucocorticoid receptors, and hypothalamic-pituitary adrenal responses to stress. *Science, 277*, 1659-1962.

Lopes, P.N., Brackett, M.A., Nezlek, J.B., Schutz, A., Sellin, I., & Salovey, P. (2004). Emotional intelligence and social interaction. *Personality and Social Psychology Bulletin, 30 (8)*, 1018-1034.

Lopes, P.N., Salovey, P., Coté, S. & Beers, M. (2005). Emotion regulation abilities and the

quality of social interaction. *Emotion, 5 (1)*, 113-118.

Lopes, P.N., Salovey, P., & Straus, R. (2003). Emotional intelligence, personality, and the perceived quality of social relationships. *Personality and Individual Differences, 35 (3)*, 641-658.

Lovejoy, M.C., Graczyk, P.A., O'Hare, E., & Neuman, G. (2000). Maternal depression and parenting behavior: A meta-analytic review. *Clinical Psychology Review, 20 (5)*, 561-592.

Lundy, B.L., Jones, N.A., Field, T., Nearing, G., Davalos, M., Pietro, P.A., Schanberg, S., & Kuhn, C. (1999). Prenatal depression effects on neonates. *Infant Behavior and Development, 22 (1)*, 119-129.

Lupien, S.J., King, S, Meaney, M.J., & McEwen, B. (2000). Child's stress hormone levels correlate with mother's socioeconomic status and depressive state. *Biological Psychiatry, 48*, 976-980.

Lupien, S.J., McEwen, B.S., Gunnar, M.R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour, and cognition. *Nature Reviews Neuroscience, 10*, 434-445.

Lyketsos, C.G., Nestadt, G., Cwi, J., Heithoff, K., & Eaton, W.W. (1994). The life chart interview: A standardized method to describe the course of psychopathology. *International Journal of Methods in Psychiatric Research, 4*, 143-155.

Lyons, D.M., Martel, F.L., Levine, S., Risch, N.J., Schatzberg, A.F. (1999). Postnatal experiences and genetic effects on squirrel monkeys social affinities and emotional distress. *Hormones and Behavior, 36*, 266-275.

Lyons, D.M., Yang, C., Mobley, B.W., Nickerson, J.T., & Schatzberg, A.F. (2000). Early

environmental regulation of glucocorticoid feedback sensitivity in young adult monkeys. *Journal of Neuroendocrinology, 12 (8)*, 723-728.

MacMillan, H.L., Georgiades, K., Duku, E.K., Shea, A., Steiner, M., Niec, A., et al., (2009). Cortisol response to stress in female youths exposed to childhood maltreatment: Results of the youth mood project. *Biological Psychiatry, 66*, 62-68.

Macri, S. & Laviola, G. (2004). Single episode of maternal deprivation and adult depressive profile in mice: Interaction with cannabinoid exposure during adolescence. *Behavioural Brain Research, 154 (1)*, 231-238.

Mannie, Z.N., Harmer, C.J., & Cowen, P.J. (2007). Increased waking salivary cortisol levels in young people at familial risk for depression. *American Journal of Psychiatry, 164*, 617-621.

Marais, L., van Rensburg S.J., van Zyl, J.M., Stein, D.J., & Daniels, W.M.U. (2008). Maternal separation of rat pups increases the risk of developing depressive-like behaviour after subsequent chronic stress by altering corticosterone and neurotrophin levels in the hippocampus. *Neuroscience Research, 61*, 106-112.

Marcus, D.K. & Nardone, M.E. (1992). Depression and interpersonal rejection. *Clinical Psychology Review, 12*, 433-449.

Martin, R.C. & Dahlen, E.R. (2005). Cognitive emotion regulation in the prediction of depression, anxiety, stress, and anger. *Personality and Individual Differences, 39*, 1249-1260.

Maszk, P., Eisenberg, N., & Guthrie, I.K. (1999). Relations of children's social status to their emotionality and regulation: A short-term longitudinal study. *Merrill-Palmer Quarterly, 45 (3)*, 468-492.

Maughan, A., Cicchetti, D., Toth, S.L., & Togosch, F.A. (2007). Early occurring maternal depression and maternal negativity in predicting young children's emotion regulation and socioemotional difficulties. *Journal of Abnormal Child Psychology, 35*, 685-703.

McCardle, E.T. (2004). HPA-axis reactivity to interpersonal stress in young adults who self-injure. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 64 (10-B).* (UMI No. 3110531).

McCarthy, C.J., Lambert, R.G., & Moller, N.P. (2006). Preventative resources and emotion regulation expectancies as mediators between attachment and college students' stress outcomes. *International Journal of Stress Management, 13 (1)*, 1-22.

McCormick, C.M., Kehoe, P., & Kovacs, S. (1998). Corticosterone release in response to repeated, short episodes neonatal isolation: Evidence of sensitization. *International Journal of Developmental Neuroscience, 16 (3/4)*, 175-185.

McDowell, D.J., Kim, M., O'Neil, R., & Parke, R.D. (2002). Children's emotion regulation and social competence in middle childhood: The role of maternal and parternal interactive style. *Marriage and Family Review, 34 (3)*, 345-364.

McEwen, B.S. (1998). Protective and damaging effects of stress mediators. *Seminars in Medicine of the Beth Israel Deaconness Medical Centre, 338 (3)*, 771-779.

McEwen, B.S. (2000). Allostasis and allostatic load: Implications for neuropsychopharmacology. *Neuropsychopharmacology, 22*, 108-124.

McEwen, S. & Schmeck, H.M. (1994). *The Hostage Brain*. Japan: Rockefellar University Press.

McEwen, B.S. & Stellar, E. (1993). Stress and the individual: Mechanisms leading to

disease. *Archives of Internal Medicine, 153*, 2093-2101.

Meaney, M.J. (2001). Maternal care, gene transmission, and the transmission of individual differences in stress reactivity across generations. *Annual Review of Neuroscience, 24*, 1161-1192.

Meyer, S.E., Chrousos, G.P., & Gold, P.W. (2001). Major depression and the stress system: A life span perspective. *Development and Psychopathology, 13*, 565-580.

Michelson, D., Licinio, J., & Gold, P.W. (1995). Mediation of the stress response by the hypothalamic-pituitary adrenal axis. In M.J. Friedman, D.S. Charney, & A.Y Dutch (Eds.), *Neurobiological and Clinical Consequences of Stress.* Philadelphia: Lippincott-Raven Publishers.

Miller, G.E., Chen, E., & Zhou, E.S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenal axis in humans. *Psychological Bulletin, 133 (1)*, 25-45.

Modell, S., Lauer, C.J., Schreiber, W., Huber, J., Krieg, J.-C., & Holsboer, F. (1998). Hormonal response pattern in the combined DEX-CRH test is stable over time in subjects at high familial risk for affective disorders. *Neuropsychopharmacology, 18 (4)*, 253-262.

Mufson, L., Weissman, M.M., Moreau, D., & Garfinkel, R. (1999). Efficacy of interpersonal psychotherapy for depressed adolescents. *Archives of General Psychiatry, 56 (6)*, 573-579.

Murray, L, Sinclair, D., Cooper, P., Ducournau, P., & Turner, P. (1999). The socioemotional development of 5-year old children of postnatally depressed mothers. *Journal of Child Psychology and Psychiatry, 40 (8)*, 1259-1271.

Murray, L., Halligan, S.L., Goodyer, I., & Herbert, J. (2010). Disturbances in early parenting of depressed mothers and cortisol secretion in offspring: A preliminary study. *Journal of Affective Disorders, 122*, 218-223.

National Institute of Child Health and Human Development (NICHD). (1999). Chronicity of maternal depressive symptoms, maternal sensitivity, and child functioning at 36 months. *Developmental Psychology, 35 (5)*, 1297-1310.

Neal, R. (2003). Child and adolescent depression: Short-term treatment effectiveness and long-term opportunities. *International Journal of Methods in Psychiatric Research, 12 (1)*, 44-53.

Nemeroff, C.B. (2004). Neurobiological consequences of childhood trauma. *Journal of Clinical Psychiatry, 65 (suppl 1)*, 18-28.

Ouellet-Morin, I., Boivin, M., Dionne, G., Lupien, S.J., Arsenault, L., Barr, R.G., Perusse, D. et al., (2008). Variations in heritability of cortisol reactivity to stress as a function of early familial adversity among 19-month old twins. *Archives of General Psychiatry, 65 (2)*, 211-218.

Ouellet-Morin, I., Dionne, G., Perusse, D., Lupien, S.J., Arsenault, L., Barr, R.G., et al., (2009). Daytime cortisol secretion in 6-month old twins: Genetic and environmental contributions as a function of early familial adversity. *Biological Psychiatry, 65*, 409-416.

Parfitt, D.B., Levin, J.K., Saltstein, K.P., Klayman, A.S., Greer, L.M., & Helmreich, D.L. (2004). Differential early rearing environments can accentuate or attenuate the responses to stress in male C57BL/6 mice. *Brain Research, 1016 (1)*, 111-118.

Penza, K.M, Heim, C., & Nemeroff, C.B. (2003). Neurobiological effects of childhood

abuse: implications for the pathophysiology of depression and anxiety. *Archives of Women's Mental Health, 6*, 15-22.

Phillips, K.F.V. & Power, M.J. (2007). A new self-report measure of emotion regulation in adolescents: The regulation of emotions questionnaire. *Clinical Psychology and Psychotherapy, 14*, 145-156.

Plotsky, P.M. & Meaney, M.J. (1993). Early, postnatal experience alters hypothalamic corticotropin-releasing factor (CRF) mRNA, median eminence CRF content and stress-induced release in adult rats. *Brain Research, 18 (3)*, 195-200.

Plotsky, P.M., Owens, M.J., & Nemeroff, C.B. (1998). Psychoneuroendocrinology of depression: Hypothalamic-pituitary-adrenal axis. *Psychiatric Clinics of North America, 21 (2)*, 293-307.

Reijntjes, A., Dekovic, M., Vermande, M., & Telch, M.J. (2009). Role of depressive symptoms in early adolescents' online emotional responding to a peer evaluation challenge. *Depression and Anxiety, 26*, 135-146.

Rivers, S.E., Brackett, M.A., Katulak, N.A., & Salovey, P. (2007). Regulating anger and sadness: An exploration of discrete emotions in emotion regulation. *Journal of Happiness Studies, 8*, 393-427.

Ronsaville, D.S., Municchi, G., Laney, C., Cizza, G., Meyer, S.E., Haim, A., et al. (2006). Maternal and environmental factors influence the hypothalamic-pituitary-adrenal axis response to the corticotropic-releasing hormone infusion in offspring of mothers with or without mood disorders. *Development and Psychopathology, 18*, 173-194.

Rudolph, K.D. (2002). Gender differences in emotional responses to interpersonal stress during adolescence. *Journal of Adolescent Health, 30S*, 30-13.

Rüedi-Bettschen, D., Zhang, W., Russig, H., Ferger, B., Weston, A., Pederson, E.-M., Feldon, J., & Pryce, C.R. (2006). Early deprivation leads to altered behavioural, autonomic, and endocrine responses to environmental challenge in adult Fischer rats. *European Journal of Neuroscience, 24*, 2879-2893.

Rydell, A.-M., Thorell, L.B., & Bohrin, G. (2007). Emotion regulation in relation to social functioning: An investigation of child self-reports. *European Journal of Developmental Psychology, 4 (3)*, 293-313.

Sanchez, M.M., Ladd, C.O., & Plotsky, P.M. (2001). Early adverse experience as a developmental risk factor for later psychopathology: Evidence from rodent and primate models. *Development and Psychopathology, 13*, 419-449.

Sanchez, M.M., Noble, P.M., Lyon, C.K., Plotsky, P.M., Davis, M., Nemeroff, C.B., & Winslow, J.T., (2005). Alterations in diurnal cortisol rhythm and acoustic startle response in nonhuman primates with adverse rearing. *Biological Psychiatry, 57*, 373-381.

Sanchez, M.M. (2006). The impact of early adverse care on the development of the HPA axis: Nonhuman primate models. *Hormones and Behavior, 50*, 623-631.

Santucci, A.K, Silk, J.S., Shaw, D.S., Gentzler, A., Fox, N.A., Kovacs, M. (2008). Vagal tone and temperament as predictors of emotion regulation strategies in young children. *Developmental Psychobiology, 50*, 205-216.

Schmidt, L.A., Fox, N.A., Sternberg, E.M., Gold, P.W., Smith, C.C., & Schulkin, J. (1999). Adrenocortical reactivity and social competence in seven-year olds. *Personality and Individual Differences, 26 (6)*, 977-985.

Segrin, C. & Abramson, L.Y (1994). Negative reactions to depressive behaviors: A

communication theories analysis. *Journal of Abnormal Psychology, 103 (4)*, 655-668.

Segrin, C. & Flora, J. (2000). Poor social skills are a vulnerability factor in the development of psychosocial problems. *Human Communication Research, 26 (3)*, 489-514.

Shaw, D.S., Schonberg, M., Sherrill, J., Huffman, D., Lukon, J., Obrosky, D., & Kovacs, M. (2006). Responsivity to offspring's expression of emotion among childhood-onset depressed mothers. *Journal of Clinical Child and Adolescent Psychology, 35 (4)*, 490-503.

Sheeber, L., Allen, N., Davis, B., & Sorensen, E. (2000). Regulation of negative affect during mother-child problem-solving interactions: Adolescent depressive status and family processes. *Journal of Abnormal Child Psychology, 28 (5)*, 467-479.

Shih, J.H., Eberhart, N.K., Hammen, C.L., & Brennan, P.A. (2006). Differential exposure and reactivity to interpersonal stress predict sex differences in adolescent depression. *Journal of Clinical Child and Adolescent Psychology, 35 (1)*, 103-115.

Silk, J.S., Steinberg, L., & Sheffield Morris, A. (2003). Adolescents' emotion regulation in daily life: links to depressive symptoms and problem behavior. *Child Development, 74 (6)*, 1869-1880.

Silk, J.S., Shaw, D.S., Skuban, E.M., Oland, A.A., & Kovacs, M. (2006a). Emotion regulation strategies in offspring of childhood-onset depressed mothers. *Journal of Child Psychology and Psychiatry, 47 (1)*, 69-78.

Silk, J.S., Shaw, D.S., Forbes, E.E., Lane, T.L., & Kovacs, M. (2006b). Maternal depression and child internalizing: The moderating role of child emotion regulation. *Journal of Clinical Child and Adolescent Psychology, 35 (1)*, 116-126.

Sohr-Preston, S.L. & Scaramarella, L.V. (2006). Implications of timing of maternal depressive symptoms for early cognitive and language development. *Clinical Child and Family Psychology Review, 9 (1)*, 65-83.

Sokolov, S. & Kutcher, S. (2001). Adolescent depression: Neuroendocrine aspects. In I.M. Goodyer (Ed.), *The depressed child and adolescent* (2nd Ed.). New York: Cambridge University Press.

Southam-Gerow, M.A. & Kendall, P.C. (2002). Emotion regulation and understanding implications for child psychopathology and therapy. *Clinical Psychology Review, 22*, 189-122.

Stansbury, K. & Gunnar, M.R. (1994). Adrenocortical activity and emotion regulation. In N.A. Fox (Ed.), The development of emotion regulation: Biological and behavioural considerations (pp.108-134). *Monographs for the Research on Child Development, 59 (Nos. 2-3, Serial No. 240).*

Steer, R.A., Kumar, G., Ranieri, W.F., & Beck, A.T. (1998). Use of the Beck Depression Inventory-II with adolescent psychiatric outpatients. *Journal of psychopathology and behavioural assessment, 20 (2)*, 127-137.

Steptoe, A., van Jaarsveld, C.H.M., Semmler, C., Plomin, R., &Wardle, J. (2009). Heritability of daytime cortisol levels and cortisol reactivity in children. *Psychoneuroendocrinology, 42 (2)*, 273-280.

Summerfeldt, L.J. & Antony, M.M. (2002). Structured and semi-structured diagnostic interviews. In M.M. Antony & D.H. Barlow (Eds.), *Handbook of assessment and treatment planning for psychological disorders*. New York: The Guilford Press.

Tabachnick, B.G. & Fidell, L.S. (2001). *Using Multivariate Statistics* (3rd ed.). Boston:

Allyn and Bacon.

Tarullo, A.R. & Gunnar, M.R. (2006). Child maltreatment and the developing HPA axis. *Hormones and Behaviour, 50*, 632-639.

Terleph, T.A., Klein, R.G., Roberson-Nay, R., Mannuzza, S., Moulton, J.L., Woldehawariat, G. et al. (2006). Stress responsivity and HPA axis activity in juveniles: Results from a home based CO_2 inhalation study. *American Journal of Psychiatry, 163 (4)*, 738-740.

Thompson, R.A. (1994). Emotion regulation: A theme in search of definition. In N.A. Fox (Ed.), The development of emotion regulation: Biological and behavioural considerations (pp.25-52). *Monographs for the Research on Child Development, 59 (Nos. 2-3, Serial No. 240).*

Tompson, M.C., Pierre, C.B., Dingman Boger, K., McKowen, J.W., Chan, P.T., & Freed, R.D. (2010). Maternal depression, maternal expressed emotion, and youth psychopathology. *Journal of Abnormal Child Psychology, 38*, 105-117.

Tops, M., Reese, H., Oldehinkel, A.J., Rijsdijk, F.V., & Ormel, J. (2008). Rejection sensitivity relates to hypocortisolism and depressed mood state in young women. *Psychoneuroendocrionology, 33*, 551-559.

Tronick, E.Z. & Gianino, A.F. (1986). The transmission of maternal disturbance to the infant. *New Directions for Child Development, 34*, 5-11.

Tsigos, C. & Chrousos, G.P. (2002). Hypothalamic-pituitary-adrenal axis, neuroendocrine factors, and stress. *Journal of Psychosomatic Research, 53*, 865-871

Van Orden, K., Wingate, L.R., Gordon, K.H., & Joiner, T.E. (2005). Interpersonal factors as

vulnerability to psychopathology over the life course. In B.L. Hankin & J.R.Z. Abela (Eds.), *Development of psychopathology: A vulnerability-stress perspective* (pp.136-160). Thousand Oaks: Sage Publications.

Wade, T.J., Cairney, J., & Pevalin, D.J. (2002). Emergence of gender differences in depression during adolescence: National panel of results from three countries. *Journal of the American Academy of Child and Adolescent Psychiatry, 41 (2)*, 190-198.

Warren, S.L., Gunnar, M.R., Kagan, J., Anders, T.F., Simmens, S.J., Rones, M. et al. (2003). Maternal panic disorder: Infant temperament, neurophysiology, and parenting behaviours. *Journal of the American Academy of Child and Adolescent Psychiatry, 42 (7)*, 814-825.

Weaver, I.C.G., Cervoni, N., Champagne, F.A., D'Alessio, A.C., Sharma S., Seckl, J.R., Dymov, S., Szyf, M., & Meaney, M.J. (2004). Epigenetic programming by maternal behavior. *Nature Neuroscience, 7 (8)*, 847-854.

Whiffen, V.E. (2005). Disentangling causality in the associations between couple and family processes and depression. In W.M Pinsof & J.L. Lebow (Eds)., *Family psychology: The art of the science*. New York: Oxford University Press

Wierzbicki, M. & McCabe, M. (1988). Social skills and subsequent depressive symptomatology in children. *Journal of Clinical Child Psychology, 17 (3)*, 203-208.

Wichers, M.C., Myers-Germeys, I., Jacobs, N., Kenis, G., Derom, C., Vlietinck, R., Delespaul, P., Mengelers, R., Peters, F., Nicolson, N., & Van Os, J. (2008). Susceptibility to depression expressed as alterations in cortisol day curve: A cross-

twin, cross-trait study. *Psychosomatic Medicine, 70*, 314-318.

Wickramaratne, P.J. & Weissman, M.M. (1998). Onset of psychopathology in offspring by developmental phase and parental depression. *Journal of the American Academy of Child and Adolescent Psychiatry, 37 (9),* 933-942.

Wierzbicki, M. & McCabe, M. (1988). Social skills and subsequent depressive symptomatology in children. *Journal of Clinical Child Psychology, 17 (3)*, 203-208.

Yap, M.B.H., Allen, N.B., & Ladouceur, C.D. (2008). Maternal socialization of positive affect: The impact of invalidation on adolescent emotion regulation and depressive symptomatology. *Child Development, 79 (5)*, 1415-1431.

Yap, M.B.H., Allen, N.B., & Sheeber, L. (2007). Using an emotion regulation framework to understand the role of temperament and family processes in risk for adolescent depressive disorders. *Clinical Child and Family Psychology, 10 (2)*, 180-196.

Yehuda, R., Bierer, L.M., Schmeidler, J., Aferiat, D.H., Breslau, I., & Dolan, S. (2000). Low cortisol and risk for PTSD in adult offspring of Holocaust survivors. *American Journal of Psychiatry, 157 (8)*, 1252-1259.

Yehuda, R., Halligan, S.L., & Grossman, R. (2001). Childhood trauma and risk for PTSD: Relationship to intergenerational effects trauma, parental PTSD, and cortisol excretion. *Development and Psychopathology, 13*, 733-753.

Yehuda, R., Halligan, S.L., & Bierer, L.M. (2002). Cortisol levels in adult offspring of Holocaust survivors: Relation to PTSD symptom severity in the parent and child. *Psychoneuroendocrinology, 27*, 171-180.

Yehuda, R., Teicher, M.H., Seckl, J.R., Grossman, R.A., Morris, A., & Bierer, L.M. (2007).

Parental posttraumatic stress disorder as a vulnerability factor for low cortisol trait in offspring of Holocaust survivors. *Archives of General Psychiatry, 64 (9)*, 1040-1048.

Zanarini, M.C. & Frankenburg, F.R. (2001). Attainment and maintenance of reliability of Axis I and Axis II disorders over the course of a longitudinal study. *Comprehensive Psychiatry, 42 (5)*, 369-374.

Zimmerman, L.K. & Stansbury, K. (2004). The influence of emotion regulation, level of shyness, and habituation on the neuroendocrine response of three-year old children. *Psychoneuroendocrinology, 29*, 973-982.

Appendix A

Self Report Measures

Appendix A.1

Lab Visit Questionnaire

Instructions: Please answer the following questions as honestly and as accurately as possible. There are no right or wrong answers. All information provided on this questionnaire will be kept strictly confidential and anonymous.

1. What time did you fall asleep last night? _____
2. What time did you wake up today? _____
3. How many hours of sleep have you gotten in the past 24 hours?
   _____
4. When did you last eat or drink? _____ a.m./p.m.
5. In the past hour have you had any dairy products?
   a. Yes
   b. No
6. In the past hour have you had any caffeine products?
   a. Yes
   b. No
7. In the past hour have you had any acidic products?
   a. Yes
   b. No
8. Did you brush your teeth in the last three hours?
   a. Yes
   b. No
9. Have you had dental work in the past 24 hours?
   a. Yes
   b. No
10. What medication (including birth control) did you take today?
    _____

11. Have you been taking any medications (including birth control) for the past two weeks, but did not take it today? If yes, what medications?
    _____

12. In the past 24 hours, have you drunk any alcohol?

    a. Yes
    b. No

13. Did you drink alcohol or take any non-prescription drugs last night?

    a. Yes (explain) _____
    b. No

14. Did you use any other drugs (e.g., marijuana) today?

    a. Yes (explain) _____
    b. No

15. Did you smoke any cigarettes today?

    a. Yes. If yes, how long ago? _____ am/pm
    b. No

16. Indicate day and length of last period, if currently, mark when began:
_____

17. Did you engage in any exercise yesterday or today?

- ❑ Yes (explain) _____
- ❑ No

Appendix A.2

Date of Visit:_____

Participant ID: _____

VISUAL ANALOGUE SCALE

VAS # ____
Time done: _____

We are interested in how you perceive your anxiety level. Please mark by drawing a vertical line on this scale how you feel right now, that is, at this moment.

_____

Not anxious Extremely anxious

Appendix A.3

The DERS

Please indicate how often the following statements apply to you by writing the appropriate number from the scale below on the line beside each item:

1	2	3	4	5
almost never (0-10%)	sometimes (11-35%)	about half the time (36-65%)	most of the time (66-90%)	almost always (91-100%)

_____ 1) I am clear about my feelings.

_____ 2) I pay attention to how I feel.

_____ 3) I experience my emotions as overwhelming and out of control.

_____ 4) I have no idea how I am feeling.

_____ 5) I have difficulty making sense out of my feelings.

_____ 6) I am attentive to my feelings.

_____ 7) I know exactly how I am feeling.

_____ 8) I care about what I am feeling.

_____ 9) I am confused about how I feel.

_____ 10) When I'm upset, I acknowledge my emotions.

_____ 11) When I'm upset, I become angry with myself for feeling that way.

_____ 12) When I'm upset, I become embarrassed for feeling that way.

_____ 13) When I'm upset, I have difficulty getting work done.

_____ 14) When I'm upset, I become out of control.

_____ 15) When I'm upset, I believe that I will remain that way for a long time.

_____ 16) When I'm upset, I believe that I'll end up feeling very depressed.

_____ 17) When I'm upset, I believe that my feelings are valid and important.

_____ 18) When I'm upset, I have difficulty focusing on other things.

_____ 19) When I'm upset, I feel out of control.

1----------2----------3----------4----------5

almost never (0-10%)	sometimes (11-35%)	about half the time (36-65%)	most of the time almost always (66-90%) (91-100%)

_____ 20) When I'm upset, I can still get things done.

_____ 21) When I'm upset, I feel ashamed with myself for feeling that way.

_____ 22) When I'm upset, I know that I can find a way to eventually feel better.

_____ 23) When I'm upset, I feel like I am weak.

_____ 24) When I'm upset, I feel like I can remain in control of my behaviors.

_____ 25) When I'm upset, I feel guilty for feeling that way.

_____ 26) When I'm upset, I have difficulty concentrating.

_____ 27) When I'm upset, I have difficulty controlling my behaviors.

_____ 28) When I'm upset, I believe that there is nothing I can do to make myself feel better.

_____ 29) When I'm upset, I become irritated with myself for feeling that way.

_____ 30) When I'm upset, I start to feel very bad about myself.

_____ 31) When I'm upset, I believe that wallowing in it is all I can do.

_____ 32) When I'm upset, I lose control over my behaviors.

_____ 33) When I'm upset, I have difficulty thinking about anything else.

_____ 34) When I'm upset, I take time to figure out what I'm really feeling.

_____ 35) When I'm upset, it takes me a long time to feel better.

_____ 36) When I'm upset, my emotions feel overwhelming.

Appendix A.4

ICQ-R

Instruction: Circle the number which best describes you. See bottom of page for what each number means.

1. How good are you at asking someone new to do things together, like go to a ball game or a movie?	1	2	3	4	5
2. How good are you at making someone feel better when they are unhappy or sad?	1	2	3	4	5
3. How good are you at getting people to go along with what you want?	1	2	3	4	5
4. How good are you at telling people private things about yourself?	1	2	3	4	5
5. How good are you at resolving disagreements in ways that make things better instead of worse?	1	2	3	4	5
6. How good are you at going out of your way to start up new relationships?	1	2	3	4	5
7. How good are you at being able to make others feel like their problems are understood?	1	2	3	4	5
8. How good are you at taking charge?	1	2	3	4	5
9. How good are you at letting someone see your sensitive side?	1	2	3	4	5
10. How good are you at dealing with disagreements in ways that make both people happy in the long run?	1	2	3	4	5

1 = **Poor at this;** would be so uncomfortable and unable to handle this situation that it would be avoided at possible.
2 = **Fair at this;** would feel uncomfortable and would have some difficulty handling this situation.
3 = **O.K. at this;** would feel somewhat uncomfortable and have a little difficulty handling this situation.
4 = **Good at this;** would feel very comfortable and could handle this situation very well.
5 = **EXREMELY good at this;** would feel very comfortable and could handle this situation very well.

11. How good are you at carrying on conversations with new people that you would like to know better?	1	2	3	4	5
12. How good are you at helping people work through their thoughts and feelings about important decisions?	1	2	3	4	5
13. How good are you at sticking up for yourself?	1	2	3	4	5
14. How good are you at telling someone embarrassing things about yourself?	1	2	3	4	5
15. How good are you at resolving disagreements in ways so neither person feels hurt or resentful?	1	2	3	4	5
16. How good are you at introducing yourself to people for the first time?	1	2	3	4	5
17. How good are you at helping people handle pressure or upsetting events?	1	2	3	4	5
18. How good are you at getting someone to agree with your point of view?	1	2	3	4	5
19. How good are you at opening up and letting someone get to know everything about you?	1	2	3	4	5
20. How good are you at dealing with disagreements in ways so that one person does not always come out the loser?	1	2	3	4	5
21. How good are you at calling new people on the phone to set up a time to get together to do things?	1	2	3	4	5

1 = **Poor at this;** would be so uncomfortable and unable to handle this situation that it would be avoided at possible.
2 = **Fair at this;** would feel uncomfortable and would have some difficulty handling this situation.
3 = **O.K. at this;** would feel somewhat uncomfortable and have a little difficulty handling this situation.
4 = **Good at this;** would feel very comfortable and could handle this situation very well.
5 = **EXREMELY good at this;** would feel very comfortable and could handle this situation very well.

22. How good are you at showing that you really care when someone talks about problems?	1	2	3	4	5
23. How good are you at deciding what should be done?	1	2	3	4	5
24. How good are you at sharing personal thoughts and feelings with others?	1	2	3	4	5
25. How good are you at dealing with disagreements in ways that don't lead to big arguments?	1	2	3	4	5
26. How good are you at going places where there are unfamiliar people in order to get to know new people?	1	2	3	4	5
27. How good are you at helping others understand Your problems better?	1	2	3	4	5
28. How good are you at voicing your desires and opinions?	1	2	3	4	5
29. How good are you at telling someone things that you do not want everyone to know?	1	2	3	4	5
30. How good are you at getting over disagreements quickly?	1	2	3	4	5
31. How good are you at making good first impressions when getting to know new people?	1	2	3	4	5
32. How good are you at giving suggestions and advice in ways that are received well by others?	1	2	3	4	5
33. How good are you at getting your own way with others?	1	2	3	4	5

1 = **Poor at this;** would be so uncomfortable and unable to handle this situation that it would be avoided at possible.
2 = **Fair at this;** would feel uncomfortable and would have some difficulty handling this situation.
3 = **O.K. at this;** would feel somewhat uncomfortable and have a little difficulty handling this situation.
4 = **Good at this;** would feel very comfortable and could handle this situation very well.
5 = **EXREMELY good at this;** would feel very comfortable and could handle this situation very well.

34. How good are you at telling someone your true feelings about other people?	1	2	3	4	5
35. How good are you at controlling your temper when having a conflict with someone?	1	2	3	4	5
36. How good are you at being an interesting and fun person to be with when first getting to know people?	1	2	3	4	5
37. How good are you at listening while others "let off steam" about problems they are going through?	1	2	3	4	5
38. How good are you at making decisions about where to go or what to do?	1	2	3	4	5
39. How good are you at telling someone what you personally think about important issues?	1	2	3	4	5
40. How good are you at backing down in a disagreement once it becomes clear that he is wrong?	1	2	3	4	5

1 = **Poor at this;** would be so uncomfortable and unable to handle this situation that it would be avoided at possible.
2 = **Fair at this;** would feel uncomfortable and would have some difficulty handling this situation.
3 = **O.K. at this;** would feel somewhat uncomfortable and have a little difficulty handling this situation.
4 = **Good at this;** would feel very comfortable and could handle this situation very well.
5 = **EXREMELY good at this;** would feel very comfortable and could handle this situation very well.

Appendix A.5

BDI-II

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past 2 weeks, including today.** Place a check mark beside the statement you have picked. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness
- __ I do not feel sad.
- __ I feel sad much of the time.
- __ I am sad all the time.
- __ I am so sad or unhappy that I can't stand it.

2. Pessimism
- __ I am not discouraged about my future.
- __ I feel more discouraged about my future than I used to be.
- __ I do not expect things to work out for me.
- __ I feel my future is hopeless and will only get worse.

3. Past Failure
- __ I do not feel like a failure.
- __ I have failed more than I should have.
- __ As I look back, I see a lot of failures.
- __ I feel I am a total failure as a person.

4. Loss of Pleasure
- __ I get as much pleasure as I ever did from the things I enjoy.
- __ I don't enjoy things as much as I used to.
- __ I get very little pleasure from the things I used to enjoy.
- __ I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings
- __ I don't feel particularly guilty.
- __ I feel guilty over many things I have done or should have done.
- __ I feel quite guilty most of the time.
- __ I feel guilty all of the time.

6. Punishment Feelings
- __ I don't feel I am being punished.
- __ I feel I may be punished.
- __ I expect to be punished.
- __ I feel I am being punished.

7. Self-Dislike

__ I feel the same about myself as ever.
__ I have lost confidence in myself.
__ I am disappointed in myself.
__ I dislike myself.

8. Self-Criticalness

__ I don't criticize or blame myself more than usual.
__ I am more critical of myself than I used to be.
__ I criticize myself for all of my faults.
__ I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

__ I don't have any thoughts of killing myself.
__ I have thoughts of killing myself, but I would not carry them out.
__ I would like to kill myself.
__ I would kill myself if I had the chance.

10. Crying

__ I don't cry anymore than I used to.
__ I cry more than I used to.
__ I cry over every little thing.
__ I feel like crying, but I can't.

11. Agitation

__ I am no more restless or wound up than usual.
__ I feel more restless or wound up than usual.
__ I am so restless or agitated that it's hard to stay still.
__ I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

__ I have not lost interest in other people or activities.
__ I am less interested in other people or things than before.
__ I have lost most of my interest in other people or things.
__ It's hard to get interested in anything.

13. Indecisiveness

__ I make decisions about as well as ever.
__ I find it more difficult to make decisions than usual.
__ I have much greater difficulty in making decisions than I used to.
__ I have trouble making any decisions.

14. Worthlessness

__ I do not feel I am worthless.
__ I don't consider myself as worthwhile and useful as I used to.
__ I feel more worthless as compared to other people.
__ I feel utterly worthless.

15. Loss of Energy

__ I have as much energy as ever.
__ I have less energy than I used to have.
__ I don't have enough energy to do very much.
__ I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

__ I have not experienced any change in my sleeping pattern.
__ I sleep somewhat more than usual.
__ I sleep somewhat less than usual.
__ I sleep a lot more than usual
__ I sleep a lot less than usual.
__ I sleep most of the day.
__ I wake up 1-2 hours early and can't get back to sleep.

17. Irritability

__ I am no more irritable than usual.
__ I am more irritable than usual.
__ I am much more irritable than usual.
__ I am irritable all the time.

18. Changes in Appetite

__ I have not experienced any change in my appetite.
__ My appetite is somewhat less than usual.
__ My appetite is somewhat greater than usual.
__ My appetite is much less than before.
__ My appetite is much greater than usual.
__ I have no appetite at all.
__ I crave food all the time.

19. Concentration Difficulty

__ I can concentrate as well as ever.
__ I can't concentrate as well as usual.
__ It's hard to keep my mind on anything for very long.
__ I find I can't concentrate on anything.

20. Tiredness or Fatigue

__ I am no more tired or fatigued than usual.
__ I get more tired or fatigued more easily than usual.
__ I am too tired or fatigued to do a lot of the things I used to do.
__ I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

__ I have not noticed any recent change in my interest in sex.
__ I am less interested in sex than I used to be.
__ I am much less interested in sex now.
__ I have lost interest in sex completely.

Appendix B

Stress Test Protocol

Appendix B.1

How to Introduce the TSST-C

The next part of the session involves doing some activities similar to what you might to do in school. There are two activities: the first involves coming up with an ending to a story and the next part involves solving some problems. These activities are administered by two people in the next room who are affiliated with our study. Let's go over there now.

Accompany the participant to the classroom. Briefly introduce her to the confederates. The confederates will then begin explaining the activities. Remain in the room while this is being done and accompany the participant back to the office for five minutes while they prepare the unfinished story.

After exactly five minutes, say:

Five minutes is up. Let's go back to the classroom now.

Walk them back to the classroom and let them in (door code: 3-12-5) and wait outside the door while they are meeting with the confederates for 10 minutes. When they come out, go directly to the office and begin collecting cortisol samples and administering the remainder of the questionnaires.

Appendix B.2

TSST-C: SPEECH TASK

Prep Time: 5 minutes

Speech Time: 5 minutes for speech

We are going to give you the beginning of a story and we would like you to finish telling the story in a speech lasting five minutes long. You will have five minutes to prepare in another room.

This is the beginning of the story:

"My best friend Sarah and I were sitting in the school cafeteria talking and eating lunch when I glanced down at the face on my milk carton. No one ever really paid close attention to the faces of missing children on the back of milk cartons, but as I looked at the face of the ordinary little girl with her hair in tight pigtails – a three-year-old who had been kidnapped 12 years before from a shopping mall in Toronto, I was overcome with shock. I recognized that little girl -- it was me."

We would like to you try your best – try to perform better than all the other participants.

You can take the beginning of the story with you to help you prepare [Give participant the handout with the story]. See you again in five minutes.

Participant returns to give speech:

Set timer to 5 minutes.

Please begin.

Note: Whenever a participant finishes telling the story in less than five minutes, ask them to continue. Do this in a supportive and friendly manner.

When the five minutes is up:

Thank you very much. We have one more task for you to complete…

TSST-C: ARITHMETIC TASK

Time: 5 minutes

The next task involves some mental math. We would like you to serially subtract the number 13 from the number 1023 as fast and as accurately as you can for the next five minutes. We will let you know when the five minutes is up.

Note: Each time the participant makes a mistake, say the following:

Stop, please start again. (i.e., they must start again from the beginning)

At the end of the arithmetic task:

Thank you for completing the speech and mental math task. You performed just as well as all of our other participants.

Mental Math Answers:

(1) 1023 - 13 = 1010	(25) 711 - 13 = 698	(49) 399 - 13 = 386	(73) 87 - 13 = 74
(2) 1010 - 13 = 997	(26) 698 - 13 = 685	(50) 386 - 13 = 373	(74) 74 - 13 = 61
(3) 997 - 13 = 984	(27) 685 - 13 = 672	(51) 373 - 13 = 360	(75) 61 - 13 = 48
(4) 984 - 13 = 971	(28) 672 - 13 = 659	(52) 360 - 13 = 347	(76) 48 - 13 = 35
(5) 971 - 13 = 958	(29) 659 - 13 = 646	(53) 347 - 13 = 334	(77) 35 - 13 = 22
(6) 958 - 13 = 945	(30) 646 - 13 = 633	(54) 334 - 13 = 321	(78) 22 - 13 = 9
(7) 945 - 13 = 932	(31) 633 - 13 = 620	(55) 321 - 13 = 308	(79) 9 - 13 = -4
(8) 932 - 13 = 919	(32) 620 - 13 = 607	(56) 308 - 13 = 295	
(9) 919 - 13 = 906	(33) 607 - 13 = 594	(57) 295 - 13 = 282	
(10) 906 - 13 = 893	(34) 594 - 13 = 581	(58) 282 - 13 = 269	
(11) 893 - 13 = 880	(35) 581 - 13 = 568	(59) 269 - 13 = 256	
(12) 880 - 13 = 867	(36) 568 - 13 = 555	(60) 256 - 13 = 243	
(13) 867 - 13 = 854	(37) 555 - 13 = 542	(61) 243 - 13 = 230	
(14) 854 - 13 = 841	(38) 542 - 13 = 529	(62) 230 - 13 = 217	
(15) 841 - 13 = 828	(39) 529 - 13 = 516	(63) 217 - 13 = 204	
(16) 828 - 13 = 815	(40) 516 - 13 = 503	(64) 204 - 13 = 191	
(17) 815 - 13 = 802	(41) 503 - 13 = 490	(65) 191 - 13 = 178	
(18) 802 - 13 = 789	(42) 490 - 13 = 477	(66) 178 - 13 = 165	
(19) 789 - 13 = 776	(43) 477 - 13 = 464	(67) 165 - 13 = 152	
(20) 776 - 13 = 763	(44) 464 - 13 = 451	(68) 152 - 13 = 139	
(21) 763 - 13 = 750	(45) 451 - 13 = 438	(69) 139 - 13 = 126	
(22) 750 - 13 = 737	(46) 438 - 13 = 425	(70) 126 - 13 = 113	
(23) 737 - 13 = 724	(47) 425 - 13 = 412	(71) 113 - 13 = 100	
(24) 724 - 13 = 711	(48) 412 - 13 = 399	(72) 100 - 13 = 87	

Appendix C

THE LIFE CHART CALENDAR

Participant ID: _____

Date of Interview: _____

M's Age	D's Age	Year	Landmarks	Residence	Marital Status	Occupational	Depression	
							Epi	Dur
		2008						
		2007						
		2006						
		2005						
		2004						
		2003						
		2002						
		2001						
		2000						
		1999						
		1998						
		1997						
		1996						
		1995						
		1994						
		1993						
		1992						

Appendix D

DEBRIEFING TEXT

After the final cortisol sample has been provided, participants will be read the following debriefing text:

Thank you very much for participating in this study. At this time we would like to clarify something with you about your participation:

We told you that your performance during the speech and arithmetic tasks was being videotaped. However, we did not actually record your performance during these tasks. We pretended to be using a videotape so that we could understand how people feel when they believe they are being recorded.

Do you have any questions about this? Do you understand?

The researcher will address and respond to any questions or concerns presented by the participant.

Appendix E

RESOURCES FOR PARTICIPANTS

Call Centres:

Ottawa Distress Centre – [Telephone Number]

Kids Help Phone – [Telephone Number]

Individual Therapy:

Centre for Psychological Services – [Telephone Number]

Appendix F

Telephone Recruitment Script –Low Risk Families

Hello, this is Meredith Foot calling from the Adolescent Girls Development Project (AGDP) at the University of Ottawa. Is this a good time to talk?

I was an interviewer with the AGDP and I'm a Ph.D. student working with Dr. Valerie Whiffen, who as you know is the researcher and psychologist who lead the AGDP.

I am calling to ask if you would be interested in hearing about a study I am conducting for my doctoral thesis, under Dr. Whiffen's supervision.

We are inviting girls and their mothers who were participating in the AGDP to take part in a study looking at risk and protective factors for depression. We would like to learn more about how girls of parents with and without a history of depression manage their emotions and how they feel about their peer relationships. We would also like to measure their stress hormone levels by obtaining some saliva samples. Participation in the project is completely voluntary.

For the girls, participation would involve coming to the University of Ottawa for about 2 hours to complete a brief interview, some questionnaires, and to do activities similar to what they might do in school, like solving problems and coming up with an ending to an unfinished story. A number of times before and after these activities, we would be asking them to chew on a cotton swab to obtain saliva samples, which would allow us to measure their stress hormone levels.

For the mothers, participation involves filling out a brief questionnaire.

Participation in the study would result in no cost to you. Parking costs at the University will be reimbursed and the girls receive $40 for their participation. Also, the girls have the option of entering a raffle where they have the chance to win a $100 gift certificate and the mothers have the option of entering a raffle where they have the chance to win a $50 gift certificate.

Would you be interested in having your daughter participate in this study?

<u>If interested</u>: Thank you for your interest in participating. We ask that the girls avoid strenuous exercise they day before and the day of the session, and avoid drinking or eating anything for one hour before the session. These are things that could influence the stress hormone levels we are interested in.

Schedule Session. Confirm location. Give phone number.
We will give you a call the day before the session to remind you of your appointment.

<u>If not interested</u>: Thanks anyway.

Telephone Recruitment Script – High Risk Families

Hello, this is Meredith Foot calling from the Adolescent Girls Development Project (AGDP) at the University of Ottawa. Is this a good time to talk?

I was an interviewer with the AGDP and I'm a Ph.D. student working with Dr. Valerie Whiffen, who as you know is the researcher and psychologist who lead the AGDP.

I am calling to ask if you would be interested in hearing about a study I am conducting for my doctoral thesis, under Dr. Whiffen's supervision.

We are inviting girls and their mothers who participated in the AGDP to take part in a study looking at risk and protective factors for depression. We would like to learn more about how girls of parents with a history of depression manage their emotions and how they feel about their peer relationships. We would also like to measure their stress hormone levels by obtaining some saliva samples. Participation in the project is completely voluntary.

For the girls, participation would involve coming to the University of Ottawa for about 2 hours to complete a brief interview, some questionnaires and to do activities similar to what they might do in school, like solving problems and coming up with an ending to an unfinished story. A number of times before and after these activities, we would be asking them to chew on a cotton swab to obtain saliva samples, which would allow us to measure their stress hormone levels.

For you, participation would involve coming to the University to fill out a brief questionnaire and for an interview. In the interview, you would be asked questions about when and how often you have experienced times of depression since you were pregnant for your daughter and up until the present time. This would take about one hour.

Participation in the study would result in no cost to you. Parking costs at the University will be reimbursed and the girls receive $40 for their participation. Also, the girls have the option of entering a raffle where they have the chance to win a $100 gift certificate. The mothers who participate have the option of entering a separate raffle where they have the chance to win a $50 gift certificate.

Would you be interested in participating in this study?

<u>If interested</u>: Thank you for your interest in participating. We ask that the girls avoid strenuous exercise they day before and the day of the session, and avoid drinking or eating anything for one hour before the session. These are things that could influence the stress hormone levels we are interested in.

Schedule Session. Confirm location. Give phone number.

We will give you a call the day before the session to remind you of your appointment.
<u>If not interested</u>: Thanks anyway.

Appendix G

Assent and Consent Forms

Appendix G.1

ASSENT FORM (GIRLS)

INTRODUCTION

We are inviting you to take part in a study. This invitation is being extended to all of the girls that participated in the Adolescent Girls Development Project at the University of Ottawa. There are two groups of girls in the study. In one group, the girls' parents have had problems in the past that have made them feel sad or depressed. In the other group, the girls' parents may have had problems as well, but their problems did not lead them to experience periods of depression. A doctoral student from the School of Psychology at the University of Ottawa, Meredith Foot, is conducting the study with her supervisor, Dr. Valerie Whiffen. Dr. Whiffen is a psychologist and a Professor at the University of Ottawa. At the end of this form, you will find their contact information if you have any questions or concerns about the study.

It is important to understand why we are doing the study and what being in the study means for you before you decide that you want to be part of it. **Taking part in the study is your choice.** If you have any questions, please ask us.

WHY ARE WE DOING THE STUDY?

When parents have been depressed or sad, their daughters may feel sad a lot too when they become teenagers, and they may have problems with managing their feelings, with friends, and with stress. Now that you are a teenager, we would like to see how you are doing in these different areas.

PARTICIPATION – WHAT YOU ARE ASKED TO DO

We would like you to come to the University of Ottawa for about two hours. During this time, we would like you to complete some questionnaires that ask you about your mood, your emotions, and how you are doing in your close friendships. We will also ask you about your mood in a brief interview. In addition, we will ask you to do some activities that are similar to what you might be asked to do in school, such as solving problems and making up an ending to an unfinished story. During your visit to the University, we will be asking you at different times to chew on a cotton swab for 60 seconds. This allows us to get a sample of your saliva, which will allow us to measure a hormone that we are interested in called cortisol.

For participating in this study, you will receive $40 and you will have the option of being entered into a raffle with a chance to win a $100 gift certificate for Bayshore Shopping Centre.

POTENTIAL HARM & DISCOMFORT

Some girls may get stressed or upset when participating in the study. If you don't want to answer certain questions on a questionnaire or complete the activities, you don't have to.

VOLUNTARY PARTICIPATION

You do not have to be in the study if you don't want to, and you can stop at anytime. You can also ask us not to use any of the information that you gave us.

YOUR PRIVACY

All of the information learned about you will be kept confidential. We will not put your name on any of the tests. If we tell other people about our study, we won't tell your name or repeat anything that you said. Your name will be kept in a locked drawer at the University, and only the people working on the study will be able to see it.

There are only a few times that we would have to tell someone other than your parents what you told us. This would happen if you tell us that you are being hurt or that you are thinking of hurting yourself or another person. If we have to repeat what you told us, we will let you know this right away.

QUESTIONS OR CONCERNS?

If you have any questions or concerns about the study, you can talk to Meredith Foot and/or Dr. Whiffen. The following is their contact information:

Meredith Foot
[Contact Information Provided Here]

Dr. Valerie Whiffen
[Contact Information Provided Here]

You can also talk to a person at the University whose job it is to make sure that people don't get upset about the studies that are done here. You can reach this person by calling [Telephone Number] or by email at [Email Address]

ACCEPTANCE & SIGNATURE

There are two copies of this form and one is for you to keep. If you don't have any more questions and agree to be in the study, print your name then write your signature on the lines below.

Name (print) _____
Signature _____
Date _____
Research Coordinator's
Signature _____
Date _____

Appendix G.2

INFORMED CONSENT (MOTHERS - 1)

INTRODUCTION

You and your daughter are invited to take part in a study comparing girls who have at least one parent with a history of depression and those whose parents have no history of emotional difficulties. This invitation is being extended to mothers and daughters who participated in the Adolescent Girls Development Project (AGDP). A doctoral student from the School of Psychology at the University of Ottawa, Meredith Foot, is conducting the study with her supervisor, Dr. Valerie Whiffen. As you know, Dr. Whiffen led the AGDP and she is a clinical psychologist and Professor in the School of Psychology at the University of Ottawa. At the end of this form, you will find their contact information if you have any questions or concerns about the study.

It is important to understand the purpose of the study, what your participation involves, and what the risks and benefits to you and your daughter may be before you decide that you want to take part. Taking part is entirely your choice. If you have any questions, we would be happy to answer them.

PURPOSE OF THIS STUDY

Research suggests that the adolescent daughters of parents who have a history of depression may be at greater risk for problems regulating their emotions and for experiencing difficulties in their peer relationships. There is also research suggesting that there may be differences in the way their body reacts to stress. We would like to see how your daughter is doing in these different areas. We would also like to see how mothers' difficulties are related to problems experienced by their daughters.

WHAT YOU ARE ASKED TO DO

Girls. Your daughter is asked to come to the University of Ottawa for about two hours. We will ask her to complete questionnaires asking about her mood, how she manages her emotions and interpersonal relationships. We will ask her about her mood in a brief interview as well. We also ask her to do some activities similar to what she might be asked to do at school, such as solving problems and making up an ending to an unfinished story. A number of times during this visit, we will ask her to chew on a cotton swab for 60 seconds. This will allow us to measure a hormone we are interested in called cortisol.

Mothers. Mothers with a history of depression are also asked to come to the University of Ottawa. We would like you to fill out a brief questionnaire and interview you about when and how often you experienced periods of depression since you were pregnant for your daughter and up until the present time. This should take about 1 hour.

POTENTIAL HARM & DISCOMFORT

There are no known risks for long-term harm associated with our study. Some people may be upset by talking about their past experiences with depression and some girls may feel upset or stressed when completing the questionnaires or activities. It is important to remember that you and your daughter can decide not to answer any questions that you don't want to or do anything that you don't want to do.

VOLUNTARY PARTICIPATION

You do not have to take part in this study and you may withdraw from the study at any time. You are also free to withdraw any data you provided previously if you choose to withdraw from the study.

ANONYMITY & CONFIDENTIALITY

All of the information learned about you and/or your daughter will be kept confidential. Only the individuals involved in conducting the study will know your names. Your daughter's name will not appear on the questionnaires or cortisol samples nor will names or identifying information appear in any publication that may result from this research. All information is stored securely in locked filing cabinets at the University of Ottawa. Cortisol samples will be stored in a freezer in a secure laboratory and later destroyed following guidelines for the disposal of such material.

COSTS & REIMBURSEMENTS

Participation will not result in any expense to you. We will reimburse you for the cost of parking at the University of Ottawa. Daughters will receive $40 for their participation in addition to having the option of being entered into a raffle for a $100 gift certificate for Bayshore Shopping Centre. Mothers will have the option of being entered into a raffle for a $50 gift certificate, also for Bayshore Shopping Centre.

SIGNATURE

Your signature on this form shows that you have understood to your satisfaction the information provided about the research study, and that you agree to participate. You will be given a copy of this form to keep. Should you have any questions or concerns about the study at any time, you may contact Meredith Foot and/or Dr. Whiffen. Their contact information is as follows:

Meredith Foot
[Contact Information Provided Here]

Dr. Valerie Whiffen
[Contact Information Provided Here]

You may also contact the Research Ethics Protocol Officer as follows:

Protocol Officer for Ethics in Research
[Contact Information Provided Here]

I agree to have my daughter participate in the study as described in this consent form.

Name (print) _____

Signature _____

Date _____

Research Coordinator's
Signature _____

Date _____

I agree to participate in the study as described in this consent form.

Name (print) _____

Signature _____

Date _____

Research Coordinator's
Signature _____

Date _____

Appendix G.3

INFORMED CONSENT (MOTHERS - 2)

INTRODUCTION

You and your daughter are invited to take part in a study comparing girls who have at least one parent with a history of depression and those whose parents have no history of emotional difficulties. This invitation is being extended to mothers and daughters who participated in the Adolescent Girls Development Project (AGDP). A doctoral student from the School of Psychology at the University of Ottawa, Meredith Foot, is conducting the study with her supervisor, Dr. Valerie Whiffen. As you know, Dr. Whiffen led the AGDP and she is a clinical psychologist and Professor in the School of Psychology at the University of Ottawa. At the end of this form, you will find their contact information if you have any questions or concerns about the study.

It is important to understand the purpose of the study, what your participation involves, and what the risks and benefits to your daughter may be before you decide that you want to take part. Taking part is entirely your choice. If you have any questions, we would be happy to answer them.

PURPOSE OF THIS STUDY

Research suggests that the adolescent daughters of parents who have a history of depression may be at greater risk for problems regulating their emotions and for experiencing difficulties in their peer relationships. There is also research suggesting that there may be differences in the way their body reacts to stress. We would like to see how your daughter is doing in these different areas.

WHAT YOU ARE ASKED TO DO

Girls. Your daughter is asked to come to the University of Ottawa for about two hours. We will ask her to complete questionnaires asking about her mood, how she manages her emotions and interpersonal relationships. We will ask her about her mood in a brief interview as well. We will also ask her to do some activities similar to what she might be asked to do at school, such as solving problems and making up an ending to an unfinished story. A number of times during this visit, we will ask her to chew on a cotton swab for 60 seconds. This will allow us to measure a hormone we are interested in called cortisol.

Mothers. Mothers are also asked to come to the University of Ottawa. We would like you to fill out a brief questionnaire that asks about depressive symptoms.

POTENTIAL HARM & DISCOMFORT

There are no known risks for long-term harm associated with our study. It is important to remember that you and your daughter can decide not to answer any questions that you don't want

to or do anything that you don't want to do.

VOLUNTARY PARTICIPATION

You and your daughter do not have to take part in this study and you may withdraw from the study at any time. You are also free to withdraw any data you provided if you choose to withdraw from the study.

ANONYMITY & CONFIDENTIALITY

All of the information learned about you and your daughter will be kept confidential. Only the individuals involved in conducting the study will know your names. Your names will not appear on the questionnaires and your daughter's name will not appear on the cortisol samples nor will names or identifying information appear in any publication that may result from this research. All information is stored securely in locked filing cabinets at the University of Ottawa. Cortisol samples will be stored in a freezer in a secure laboratory and later destroyed following guidelines for the disposal of such material.

COSTS & REIMBURSEMENTS

Participation will not result in any expense to you. We will reimburse you for the cost of parking at the University of Ottawa. Daughters will receive $40 for their participation in addition to having the option of being entered into a raffle for a chance to win a $100 gift certificate for Bayshore Shopping Centre and mothers will have the option of being entered into a raffle for a $50 gift certificate, also for Bayshore Shopping Centre.

SIGNATURE

Your signature on this form shows that you have understood to your satisfaction the information provided about the research study, and that you agree to participate. You will be given a copy of this form to keep. Should you have any questions or concerns about the study at any time, you may contact Meredith Foot and/or Dr. Whiffen. Their contact information is as follows:

Meredith Foot
[Contact Information Provided Here]

Dr. Valerie Whiffen
[Contact Information Provided Here]

You may also contact the Research Ethics Protocol Officer as follows:

Protocol Officer for Ethics in Research
[Contact Information Provided Here]

I agree to have my daughter participate in the study as described in this consent form.

Name (print) _____

Signature _____

Date _____

Research Coordinator's
Signature _____

Date _____

I agree to participate in the study as described in this consent form.

Name (print) _____

Signature _____

Date _____

Research Coordinator's
Signature _____

Date _____

Appendix H

CONFEDERATE CONFIDENTIALITY AGREEMENT

ADOLESCENT GIRLS AT RISK FOR DEPRESSION: THE MEDIATING ROLE OF EMOTION REGULATION

As a confederate in the research project, *Adolescent Girls at Risk for Depression: The Mediating Role of Emotion Regulation*, I understand that the identity of participants and information about the study is privileged information and must be kept confidential. I will not voluntarily disclose any of this information.

I agree to comply with the requirements as noted above.

_____	_____	_____
Name (please print)	Signature of Confederate	Date

_____	_____	_____
Witness (please print)	Signature	Date