INFANT EMOTION REGULATION STRATEGY MODERATES THE RELATION BETWEEN MATERNAL DEPRESSIVE SYMPTOMATOLOGY AND INFANT HPA-AXIS REGULATION

by

Jennifer E. Khoury B.Sc.,

University of Toronto, 2010

A Thesis presented to Ryerson University

In partial fulfillment of the requirements for the degree of

Master of Arts in the Program of Psychology

Toronto, Ontario, Canada, 2013

Author's Declaration

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

I authorize Ryerson University to lend this thesis to other institutions or individuals for the purpose of scholarly research

I further authorize Ryerson University to reproduce this thesis by photocopying or by other means, in total or in part, at the request of other institutions or individuals for the purpose of scholarly research.

I understand that my thesis may be made electronically available to the public.

Infant Emotion Regulation Strategy Moderates the Relation between Maternal Depressive Symptomatology and Infant HPA-Axis Regulation

Jennifer Khoury

Master of Arts in Psychology

Ryerson University

Abstract

Children of depressed mothers often have atypical cortisol levels. Child characteristics associated with emotion regulation difficulties moderate associations between maternal depression and child hypothalamic-pituitary-adrenal (HPA) activity. We hypothesize that infants of more depressed mothers who utilize more independent emotion regulation will have higher cortisol levels. Mother-infant dyads (N = 193) were recruited from the community. Maternal depression was assessed using the Beck Depression Inventory II, infant regulation strategies were coded during a Toy Frustration Task, and cortisol was collected at baseline, 20, and 40 minutes after two challenges (Toy Frustration and Strange Situation). Results indicate that infant emotion regulation moderates associations between maternal depressive symptoms and infant total cortisol output (AUC_G) and cortisol reactivity (AUC_I), during the Toy Frustration task. Infants who used more independent regulation had elevated cortisol secretion. Associations were not replicated during the Strange Situation procedure. Findings are discussed in terms of adaptive emotional and physiological regulation.

Acknowledgments

There are a number of people, without whom, this research would not have been possible. First and foremost, I would like to thank my supervisor Dr. Leslie Atkinson, who has guided me to become a more diligent and thoughtful researcher. Dr. Atkinson's intellectual rigor, attention to detail, and love for research has truly inspired me. This thesis would not have been possible without his continual guidance and relentless commitment. I would also like to thank my thesis committee members, Dr. Robert Levitan, Dr. Karen Milligan, and Dr. Todd Girard, for their insightful suggestions.

Thank you to all of the members of the Biopsychosocial Laboratory who contributed to data collection and preparation. A special thanks to Dr. Andrea Gonzalez, Jessica Pereira, Ashley Steinhauer, and Vanessa Villani for their guidance and support. Thank you to the families who participated in this research.

Lastly, I would like to thank my family and friends. Your continuous encouragement, support, and confidence in me has been inspirational. My favorite aspect of research (next to data analysis) is dissemination. Research wouldn't be nearly as rewarding or exciting if I wasn't able to share it with my family and friends.

To my mother, for consistently being my external source of regulation, then and now.

Table of Contents

CHAPTER I: Introduction

CHAPTER II: Methodology

CHAPTER III: Results

Lists of Tables

Lists of Figures

Infant Emotion Regulation Strategy Moderates the Relation between Maternal Depressive Symptomatology and Infant HPA-Axis Regulation

The hypothalamic-pituitary-adrenal (HPA) axis is programmed early in life and has enduring physical and mental health implications (Gunnar & Quevedo, 2007). Maternal mental health, in particular, maternal depressive symptomatology, has been associated with atypical HPA functioning in infants and children (e.g., Ashman, Dawson, Panagiotides, Yamada, & Wilkinson, 2002; Brennan et al., 2008). Maternal depressive symptoms may influence infant HPA functioning through the suboptimal parenting (Hatzinikolaou & Murray, 2010) and mother-infant interactions (Coyl, Roggman, & Newland, 2002; Martins & Gaffan, 2000) displayed by depressed mothers. Despite the established relation between maternal depression and infant HPA functioning, the specifics of this relation remain unclear. For instance, the degree and direction of this relation may depend on the stressor paradigm (e.g., Gunnar, Talge, & Herrera, 2009), the cortisol index (Azar et al., 2007; Feldman et al., 2009), and the measure of depression (Brennan et al., 2008). Furthermore, the extant research does not examine infant features that potentially moderate relations between maternal depressive symptoms and infant HPA functioning. Infant characteristics, such as temperament, interact with parental care to influence HPA functioning (Gunnar et al., 1990; Schmidt et al., 1997). Broadly, I argue that the ambiguous findings of the extant literature may indicate the need to assess potential moderators of this relation. In the current study, I examined infant emotion regulation strategy as a moderator of the relation between maternal depressive symptoms and infant HPA functioning.

Hypothalamic-Pituitary-Adrenal (HPA) Axis

The HPA axis is one of the most important regulatory systems in the human body (Kaltas & Chrousos, 2007). As organisms interact within the environment, the HPA system is activated

in response to stressors or novelty, resulting in the production of cortisol (Gunnar, 1994; Kirschbaum & Hellhammer, 1989, 1994). The HPA system, including its end product, cortisol, is central to physiological regulation under stressful circumstances, as well as adaptive coping on a day-to-day basis (Habib et al., 2000).

The stress system is designed to operate in a stable manner in order to maintain bodily homeostasis. Humans (and other mammals) are constantly interacting with the environment, exposing themselves to external and internal stressors. Organisms are able to detect external and internal changes and activate 'specialized adaptive responses' known as *allostatic processes,* in order to maintain stability when responding to stressors (Danese & McEwen, 2012). For some individuals, stressors contribute to atypical activation of the HPA axis and surrounding systems, which results in chronic activation of allostatic systems, known as *allostatic overload* (McEwen, 1998; McEwen & Wingfiel, 2003).

Atypical regulation of the HPA system is linked to almost all physical and psychological illness processes. The stress system influences an organism's growth and development, metabolism, immunity to disease, and reproductive capacity (Kaltsas & Chrousos, 2001). Most relevant to this thesis, atypical HPA regulation is associated with a number of psychological problems, including, anxiety, antisocial behaviour (Susman et al., 2007), and depression (Van den Bergh & Van Calster, 2009).

Organization of the HPA System

In response to threat, the stress system promotes an adaptive reaction encompassing the central nervous system (CNS), including the sympathetic and parasympathetic nervous systems (SNS and PNS, respectively), and the endocrine system. Under conditions of perceived threat/stress, various structures of the CNS, SNS, and endocrine system are activated to produce

a neurobiological response to stress (Figure 1). A central component of the CNS, the amygdala, influences both the SNS and endocrine system to activate a stress response (Figure 2). The amygdala is activated when a stressor is perceived. Activation of the amygdala is modulated by the hippocampus (through memory and learned processes) and the prefrontal cortex (through executive cognitive processes). The amygdala activates the locus coeruleus (LC) and noradrenaline systems (known as the LC-NE system; Kaltas & Chrousos, 2007). The activation of these systems produces the "flight or fight" response, and results in the stimulation of catecholamines. In addition, the amygdala activates the paraventricular nuclei (PVN) in the hypothalamus, which initiates the HPA response to stressors. The parvocellular neurons in the PVN release two hormones, corticotrophin-releasing hormone (CRH) and arginine vasopressin (AVP) (Ulrich-Lai & Herman, 2009). During stress, the portion of PVN that secrete these two peptides proliferate (Habib, Gold, & Chrousos, 2001). ACTH is transported to the adrenal cortex of the adrenal gland, where it interacts with adrenal cortex receptors to produce glucocorticoid hormones, one of which is cortisol (Gunnar & Quevedo, 2007). Cortisol is a pleiotropic modulator of cellular activity, meaning that it produces multiple effects, through glucocorticoid receptors (GR) (Guilliams & Edwards, 2010).

See Figure 1

See Figure 2

GRs play an important role in stress recovery. When cortisol production reaches an optimal level, cortisol binds to GRs to produce a negative feedback loop in the stress response. Cortisol activates pathways leading back to the hypothalamus (PVN) and anterior pituitary, which result in the halt of CRH and ACTH production (Figure 3; Sapolsky, Romero, & Munck, 2000). This negative feedback loop is designed to minimize the overproduction of cortisol, which could potentially have deleterious long-term effects (Guilliams & Edwards, 2010).

See Figure 3

Mediums for Cortisol Extraction

Circulating cortisol can either be bound or unbound to proteins. Approximately 90% of cortisol is bound to plasma proteins, and the remaining 10% is unbound and biologically active (Gunnar & White, 2001). There are multiple methods of sampling cortisol, including plasma, urine, and saliva sampling (Gunnar & Donzella, 2002). Plasma samples contain both bound and unbound cortisol. Urine and saliva samples only contain unbound cortisol. An advantage of collecting salivary cortisol is that it solely provides an index of active cortisol, thus it is a more accurate representation of cortisol reactivity levels. Also, plasma and urine sampling are especially difficult with children and infants, thus saliva is the predominant form of cortisol measurement with this demographic group. In particular, collecting saliva on absorbent material (e.g., salivette) is the most common method for children under the age of five (Bakermans-Kranenburg et al., 2008; Bevans, Cerbone, & Overstreet, 2008).

Cortisol Indices

Psychological studies typically measure cortisol activity in terms of diurnal patterns or in response to acute stressors. Diurnal patterns represent the natural circadian rhythm of cortisol secretion, and typically include the collection of saliva in the morning, shortly after awakening, in the afternoon, and in the evening. Here, I am particularly interested in acute stress responses, which are typically examined through baseline indicators, total cortisol output, cortisol reactivity in which cortisol levels become elevated following the onset of some stressor, cortisol recovery in which cortisol levels return to baseline levels, or cortisol trajectories across different time points and stressor paradigms (Laurent, Ablow, & Measelle, 2012).

Development of the HPA Axis

The characteristic diurnal pattern of cortisol as well as stress reactivity evolves throughout infancy and early childhood.

Diurnal rhythm. Cortisol levels follow a circadian pattern, showing a predictable variation from morning to night (Lovallo & Thomas, 2000). However, the typical diurnal rhythm of cortisol production changes throughout infancy, and by mid-childhood this pattern of production is similar to that of adults. Irrespective of the time of day, newborns show two peaks in cortisol, separated by 12 hours (Klug et al., 2000). By three months of age, infants no longer have two cortisol peaks; rather they display a single morning peak followed by a decline throughout the day (Matagos, Moustogiannis, & Vagenakis, 1998). However, diurnal cortisol changes, particularly from the mid-morning to mid-afternoon, do not stabilize until children stop taking daytime naps, approximately around the age of four. At this point children's diurnal rhythm of cortisol production mirrors that of adults (Gunnar & Quevedo, 2007).

The adult diurnal pattern of cortisol production differs greatly from patterns of infants and young children (i.e., children under the age of four). For adults the typical diurnal rhythm of

cortisol secretion involves increasing levels at the end of the sleep period, resulting in peak levels of both cortisol and ACTH approximately 30 minutes following awakening (Kaltas & Chrousos, 2007). This early morning rhythm is known as the cortisol awakening response (CAR). Thereafter, cortisol levels decline rapidly throughout the morning, and continue to decline, albeit more gradually, throughout the afternoon, resulting in lowest levels in the evening (Lovallo & Thomas, 2000).

Stress Response. Newborns have been shown to have physiological responses to a number of different stressors including weighing (Catelin et al., 2005), medical examinations (De Weerth & Buitelaar, 2007), and heelstick procedures (Gunnar et al., 1995). Over the first year of life, infants have a hyporesponsive HPA axis, resulting in a dampened cortisol response to stressors (Gunnar & Donzella, 2002). The hyporesponsivity of the HPA axis in infancy is related to developmentally adaptive physiological changes, that is, the improvement in HPA negative feedback loop, and reduced sensitivity to ACTH in the adrenal cortex (Gunnar & Quevedo, 2007). This hyporesponsiviity may also be adaptive because infants are not equipped to regulate arousal (through emotional and behavioral means) at such young age.

Healthy newborns display a cortisol response to stressors (e.g., physical exams), however this response diminishes when these stressors are repeated (Gunnar et al., 1989). When examined cross-sectionally, cortisol responses to physical exams diminish with age, and are no longer evident in infants 12 weeks of age or older (Larson, White, Cochran, Donzella, & Gunnar, 1998). Although, cortisol reactivity is not elicited, these infants continue to exhibit behavioural signs of distress. Toddlers and older children display a similar dampened response. For instance, 2-year-olds and 3-to 5-year-olds attending nursery school for the first time do not display elevated

cortisol (Gunnar et al., 1997). Even though at a group level infants and young children do not evince a cortisol response, there are individual differences in the level of cortisol responsivity.

In two recent systematic reviews of the literature, both Gunnar and colleagues (2009) and Jansen and colleagues (2010) provide evidence that many psychological stressors do not provoke infant cortisol elevations. Gunnar and colleague's (2009) review indicated that certain paradigms are more effective at evoking a stress response in children of certain ages. For instance, separation paradigms typically evoke cortisol elevations, but only in infants between six and nine months of age. In contrast, tasks designed to elicit negative emotions (e.g., fear, wariness, anger, or frustration) were typically unsuccessful in activating an HPA response, regardless of child age. In the systematic review conducted by Jansen et al (2010), infant separation paradigms only resulted in small cortisol increases, and the majority of the strange situation paradigms did not provoke cortisol reactivity. Both the Gunnar et al. and Jansen et al. reviews demonstrate that many paradigms designed to elicit a stress response do not evoke cortisol responses, with potency declining as infants age.

While group means do not indicate increased post-stressor cortisol, inter-individual variability is high (Gunnar et al., 1996b; Nachmias et al., 1996). Environmental influences greatly affect inter-individual variability of the stress response, particularly during infancy, a period of development in which there is high brain plasticity (Chrousos, 1998). Importantly, sensitive, responsive, and attentive caregiving is critical for maintenance of the hyporesponsive HPA axis during infancy (Gunnar & Donzella, 2002). The availability of supportive parental care can buffer both physiological and behavioural stress responses. For instance, the presence of a responsive attachment figure influences whether infants show increased cortisol in response to a stressor and whether their displays of behavioural distress correlate with this cortisol reactivity

(for review Gunnar & Donzella, 2002). Maternal depressive symptomatology, which is often associated with compromised caregiving, affects infant's HPA regulation (Laurent et al., 2011).

Maternal Depression

Maternal Depression vs. Depressive Symptoms

Depression has been measured in both clinically depressed patients and community samples. The former measurement typically signifies a categorical diagnosis according to DSM-IV requirements, whereas the latter represents a continuum of depressive symptoms. Individuals with high self-reported depressive symptoms do not necessarily meet diagnostic criteria for depression. Depressive symptomatology differs qualitatively and quantitatively from a clinical diagnosis of depression. Clinically depressed individuals typically experience long episodes of depression (between 6 to 9 months), often relapse, and experience dysfunction between episodes; whereas individuals experiencing depressive symptomology have less intense and less stable dysfunction (Coyne, 1994).

Although depressive symptomatology manifested in community samples is less severe, similar to clinically depressed mothers, the dysfunction associated with depressive symptoms deleteriously affects child outcomes (e.g., Silk, Shaw, Skuban, Oland, & Kovacs, 2006), one of which is abnormal HPA functioning (e.g., Luijk et al., 2010). As noted previously, it is unclear how the degree of maternal depressive symptom severity influences infant HPA functioning (Letourneau et al., 2011). The extant research on the relation between maternal depression and infant cortisol output is equivocal.

The Influence of Maternal Depression on Infant HPA Activity

Infants of mothers with depression display similar physiological profiles (e.g., lower dopamine and serotonin, and higher norepinephrine and cortisol) to adults with the disorder

(Field et al., 2004). These physiological associations may be a result of prenatal exposure. For instance, maternal neurotransmitter/neurohormone levels during pregnancy affect the newborn's neurohormone levels. In fact, newborns mirror the physiological profile of their depressed mothers; that is, they display heightened cortisol levels (Lundy et al., 1999). Women who experience chronic prenatal depression have higher cortisol levels than those with more acute depression (Field, Diego, Hernandez-Reif, 2010) and maternal cortisol crosses the placenta and can account for up to 40% of fetal cortisol levels (Gitau, Cameron, Fisk, & Glover, 1998).

Importantly, in addition to influencing infant cortisol prenatally, maternal depression and maternal cortisol levels (prenatal and postnatal) are associated with higher cortisol levels in infants, children and adolescents (Ashman et al., 2002; Diego et al., 2004; Essex, Klein, Cho, & Kalin, 2002; Halligan, Herbert, Goodyer, & Murray, 2004). The literature suggests that higher cortisol among children of depressed parents might persist through to adulthood, and contribute to the risk for subsequent depression (Halligan et al., 2004). The effect of maternal depression on infant cortisol is important, given that elevated cortisol secretion is a predisposing factor to depression (Bhagwagar, Hafizi, & Cowen, 2005; Goodyer et al., 2000). However, the association between maternal depression and children's physiological profile is complex. This relationship often differs depending on the type of maternal depression (e.g., prenatal, postnatal, current, past) and the cortisol measurement (e.g., diurnal, basal, reactivity).

Diurnal Cortisol. Under normal circumstances cortisol has a diurnal rhythm, which includes high levels at awakening, an increase shortly thereafter, followed by a decline throughout the day (Kirschbaum & Hellhammer, 1989; Watamura, Donzella, Kertes, & Gunnar, 2004). This pattern continues to stabilize throughout infancy and childhood (Watamura et al., 2004).

Although adults with depression do not display the normal diurnal decline in cortisol (Bhagwagar et al., 2005), there are contradictory findings for the diurnal cortisol responses of children with depressed mothers. For instance, Halligan and colleagues (2004) found that high-risk adolescents displayed elevated and more intra-individual variable cortisol levels in the morning, but not in the evening. Young and colleagues (2006) contradicted this finding by demonstrating that the largest effect of maternal depression was on children's bedtime cortisol levels. Other researchers have been unable to show any significant effect of maternal depression diagnosis group on the diurnal cortisol levels of children (Ashman et al., 2002). It is possible that the timing of maternal depression influences children's diurnal cortisol pattern. For instance, Essex and colleagues (2002) found that only children who were exposed to concurrent (i.e., during infancy and at 4.5 years-old) maternal stress (including depressive symptoms) showed increased afternoon/evening cortisol.

In addition, maternal variables that are closely related to depression have been shown to predict diurnal cortisol levels in infants. For instance, Letourneau and colleagues (2011) demonstrated that infants whose mothers displayed significantly below average socio-emotional growth fostering tendencies (e.g., gentle touch, eye contact, smiling) had higher overall cortisol levels. In addition, higher maternal responsivity to infant distress predicted greater decline in cortisol throughout the day (Letourneau et al., 2011). Therefore, the relationship between maternal depression and children's diurnal cortisol pattern is complex; it differs depending on the timing of maternal depression and other maternal variables related to depression.

Cortisol Reactivity. In addition to diurnal cortisol, studies have investigated the relationship between maternal depression and infant cortisol levels in response to stressors. As noted, there has been controversy in the literature as to whether laboratory stressor paradigms

elicit infant cortisol reactivity (Gunnar et al., 2009; Jansen et al., 2010). Although many laboratory stressors do not appear to elicit cortisol reactivity, there are inter-individual differences. Maternal depression is a potential moderating variable that might account for different cortisol responses to laboratory stressors. Previous research has demonstrated that children's cortisol reactivity to a variety of stressors is influenced by maternal depression.

Studies utilizing different stressor paradigms show that infants of depressed mothers have higher cortisol reactivity compared to infants of non-depressed mothers. For instance, in a study using an arm-restraint stressor, there was no significant change in cortisol for the entire group of infants; however infants with depressed mothers had lower cortisol baseline levels and elevated cortisol reactivity compared to infants of nondepressed mothers (Azar, Pawuette, Zoccolillo, Baltzwe, & Tremblay, 2007). In addition, Feldman et al. (2009) found that infants of depressed mothers had higher baseline cortisol and cortisol reactivity to a scary-mask procedure.

However, other researchers have demonstrated that various indices of maternal depression are differentially associated with various measures of child cortisol. For instance, Brennan and colleagues (2008) examined how different maternal depression classifications (lifetime history, peripartum, and current) were related to various indices of infant cortisol (baseline, mean, and reactivity). Infants were exposed to a separation stressor, a sound stressor, and arm restraint. Interestingly, lifetime maternal depression was significantly associated with baseline and mean infant cortisol; peripartum depression was only significantly associated with cortisol reactivity; and current depression was only marginally associated with cortisol reactivity (Brennan et al., 2008). Contrarily, using a dexamethasone suppressor test (DST), Young and colleagues (2006) found that children with parents who either currently have MDD or had a history of MDD showed higher baseline cortisol and higher cortisol reactivity after doses of

dexamethasone, a hypothalamic-pituitary-adrenal (HPA) axis suppressor.

In addition, using a fear-potentiated startle procedure, Ashman and colleagues (2002) found that not all children displayed a change in cortisol. In fact, the best predictor of cortisol reactivity was the presence of maternal depression before the child was 2 years-old. Interestingly, children's internalizing symptoms moderated the relation between maternal depression and cortisol reactivity. Only children with clinically elevated internalizing symptoms showed a significant increase in cortisol reactivity. Children with internalizing problems often experience more negative emotions (Eisenberg et al., 2001) and exert higher constraint over emotional expression (Robins, John, Caspi, Moffit, & Stouthamer-Loeber, 1996). Deficits in emotion regulation underlie child internalizing problems (Eisenberg et al., 2001), and are a risk for the development of later depression (e.g., Brockmeyer et al., 2012; Gross & Munoz, 1995). Therefore, the aforementioned findings may indicate that children of depressed mothers who have greater emotion regulation difficulties (as reflected in increased internalizing problems), also experience higher cortisol reactivity. Given that Ashman and colleagues did not directly examine emotion regulation strategies, further empirical investigation is warranted to understand the direct moderating effect of emotion regulation.

An additional study, conducted by Luijk and colleagues (2010), highlights how children's behaviour can moderate the relation between maternal depression and children's HPA activity. Luijk et al. employed the Strange Situation Procedure (SSP; Ainsworth et al., 1978) to test the association between maternal depressive symptoms and infant cortisol reactivity and diurnal cortisol. No main effects were found for maternal depressive symptoms on diurnal cortisol or cortisol reactivity. Interestingly, the relationship between maternal depressive symptoms and cortisol reactivity was moderated by infant attachment style. That is, infants classified as

resistant, whose mothers had higher levels of depressive symptoms, showed the highest cortisol reactivity. Infants who display insecure attachment patterns often utilize ineffective and maladaptive emotion regulation strategies (Cassidy, 1994). Insecure resistant infants display exaggerated distress signals to capture the attention of their often inconsistently responsive caregiver (Main, 1990). These exaggerated distress signals represent a maladaptive emotion regulation strategy (Cassidy, 1994). Utilizing ineffective emotion regulation strategies can contribute to greater distress and more reactivity during interactions with their parent. For instance, infants classified as insecure-resistant often display heightened fearfulness (Main & Hesse, 1990). Previous research has indicated that insecurity in the attachment relationship mirrors deficits in emotion regulation abilities (Cassidy, 1994). Therefore, infant emotion regulation strategies may underlie the interaction between infant attachment classification and maternal depression.

Given the ambiguous findings respecting maternal depression and infant HPA activity, it is imperative to investigate possible individual differences that may explain why some infants have higher reactivity compared to others. Although the specifics remain unclear, maternal depression has been shown to influence infant cortisol responses (both diurnal and reactive). In addition, child behaviours and interaction style that appear related to emotion regulation, interact with maternal depression to influence HPA activity (Ashman et al., 2002; Luijk et al., 2010). As noted by Jansen et al. (2010) awareness of the processes by which infants seek to control their stress reaction is vital for understanding infant development. Individual differences in infant HPA activity may reflect differences in emotional coping strategies, which can be affected by maternal depression. In particular, maternal depression adversely affects parenting abilities and mother-infant interactions, which in turn can lead to poor socioemotional development, including

the development of suboptimal emotion regulation skills.

The Influence of Maternal Depression on Parenting

Infants require caregivers to help modulate their behavioural and physiological responses to stress (Tronick, 1989; Gunnar & Quevedo, 2007). Thus maternal behaviour may influence infant HPA activity. Mothers who are postnatally depressed show less sensitive caregiving at both 8 and 18-months (Hatzinikolaou & Murray, 2010). Depressed mothers also have poorer communication with their children (e.g., use less supportive tone, attend less to their children's cues for help, and are more likely to ignore or verbally control their children; Cox, Puckering, Pound, & Mills, 1987), and are less emotionally responsive, presenting 'flat' affect and negative emotions in response to their infants (Righetti-Veltema, Conne-Perreard, Bousquet, & Manzano, 2002).

Features of depression likely influence parenting and children's attachment to their caregivers. Maternal depression is directly associated with negative parenting practices (e.g., spanking) and subsequent insecure infant attachment patterns (Coyl, Roggman, & Newland, 2002). Infants of depressed mothers are more likely to show avoidant (i.e., refusal to seek closeness or comfort from the caregiver) or disorganized (i.e., confused and inconsistent behaviour) styles of attachment, and less likely to show secure forms of attachment (Martins & Gaffan, 2000).

The Association between Parenting and Infant HPA Activity. Maternal behaviour is associated with infant HPA activity. For instance, five-and six-month-old infants with more responsive mothers showed faster HPA recovery to the still-face procedure than infants with less responsive mothers (Haley & Stansbury, 2003). Maternal sensitivity was related to lower baseline, higher peak and faster cortisol recovery in response to a task designed to elicit

negative-affect (Blair, Granger, Willoughby, & Kivlighan, 2006). There are numerous additional studies suggesting that maternal sensitivity is related to infant HPA activity, although the nature of this association can be ambiguous.

In a recent study by Atkinson and colleagues (under revision), the association between maternal sensitivity and both maternal and infant HPA regulation was examined, using two divergent stressor paradigms. In this study, the authors used the Toy Frustration Task (Braungart-Rieker, & Stifter, 1996) and Strange Situation Procedure (Ainsworth, Blehar, Waters, & Wall, 1978), which have been shown to differ in ability to elicit cortisol responses (e.g., Gunnar et al., 2009). Frustration paradigms (i.e., the Toy Frustration Task) are less potent stressors compared to separation paradigms (i.e., the Strange Situation Procedure). The authors demonstrated that more highly sensitive mothers and their infants had higher cortisol production, depending on the challenge, and had more variability in cortisol secretion depending on challenge demands (potency of the stressor). More specifically, infants of more sensitive mothers had greater declines in cortisol during the Toy Frustration task, compared to infants of less sensitive mothers. During the Strange Situation Procedure, infants of more sensitive mothers had higher cortisol elevations, compared to infants of less sensitive mothers. These findings suggest that maternal sensitivity is related to infant HPA functioning, including variability in cortisol secretion, depending on the potency of the stressor.

Maternal features, such as sensitivity, modulate infant behavioural and physiological stress responses. Although infants' physiological response to laboratory and environmental stressors (e.g., Larson et al., 1998) declines with age, they continue to display behavioural signs of distress. Infants who have a responsive caregiver convey their distress to their caregiver and in turn their caregiver responds in ways to help modulate their distress. This parental behaviour is

associated with infants showing lower physiological responsivity. Interestingly, the relation between physiological and behavioural responses to stressors is also often moderated by the attachment relationship. That is, when the mother and infant have an insecure (both avoidant and resistant) attachment relationship, behavioural markers of distress predict cortisol elevations (Gunnar & Donzella, 2002; Nachmias et al., 1996). Thus, both maternal features (e.g., sensitivity) and the parent-child relationship moderate the association between infant behavioural and physiological regulation. Maternal depression often influences both maternal sensitivity and the parent-child relationship, thereby influencing infants' physiological, emotional and behavioural regulation capacities.

Additional Implications of Maternal Depression

The effects of depression on children are apparent beginning in infancy, continue into adolescence and adulthood, and can possibly be transmitted to the next generation (Weissman et al., 2006). Children of depressed mothers have a heightened risk for developing major depression (Beardslee, et al., 1983; Nomura et al., 2002). Downey and Coyne (1990) found that children of depressed mothers, compared to children of nondepressed mothers, are 6 times more likely to develop depression.

The psychosocial and biological repercussions of maternal depression are important to children's physiological, emotional, cognitive, and psychosocial development. Newborn infants of depressed mothers display physiological and emotional profiles similar to their mothers; that is, lower dopamine and serotonin, and higher norepinephrine and cortisol (Field et al., 2004); as well as lower behavioural orientation, less excitability and more withdrawal (Lundy et al., 1999). Children with a depressed caregiver are at increased risk for socioemotional deficits (Campbell et al., 2004), such as internalizing (Weissman et al., 2006) and externalizing problems (Wright et

al., 2000). Maternal depression is also associated with delays in expressive language development (Cox, Puckering, Pound & Mills, 1987) and mild impairments in general cognitive ability in infants as young as one year of age (Cohn & Campbell, 1992; Petterson & Albers, 2001). Later in childhood, maternal depression is related to risk for poor academic performance (Kurstjens & Wolke, 2001).

However, "relative to our knowledge of the range of adverse outcomes for children of depressed mothers, we know little about the mechanisms that underlie the risk for these outcomes" (Goodman & Gotlib, 1999, p. 458). In addition to knowing little about the mechanisms to explain this association, we know very little about the potential moderators of this relation. Although it has been demonstrated that maternal depression is associated with infant HPA reactivity, which is a known risk factor for depression (e.g. Bhagwager et al., 2005; Goodyer et al., 2000), as noted above, there are inconsistencies in this literature.

The extant literature has demonstrated moderators of the relation between maternal depression and child cortisol levels. For instance, Luijk and colleagues (2010) showed that only resistantly attached infants of depressed mothers showed cortisol reactivity. In addition, Ashman and colleagues (2002) demonstrated that only children with internalizing problems, who had depressed mothers, demonstrated cortisol reactivity. One underlying factor that can account for both of these findings is child emotion regulation ability. Given that maternal depression contributes to suboptimal mother-infant interactions (e.g., Goodman & Gottlib, 1999), which can result in deficits in children's socioemotional abilities (e.g., Silk et al., 2006); it is important to investigate infant emotion regulation as a potential moderator of the relation between maternal depressive symptoms and infant HPA activity.

Child Emotion Regulation

Development of Emotion Regulation

Emotion regulation strategies involve the process of initiating, sustaining, and modulating emotional arousal in order to achieve one's goals and adapt to one's social environment (Thompson, 1994). Emotion regulation skills begin to develop in early infancy and continue to mature throughout life. Particularly during infancy, the development of emotion regulation skills is highly reliant on parental support, and typically by age three children have developed autonomous regulation strategies (Grolnick, Bridges, & Connell, 1996). The external regulation provided by caregivers early in life facilitates the later development of autonomous regulation abilities (Manian & Bornstein, 2009). It is during these formative years that individual differences in emotion regulation skills arise.

Given that infants are not equipped to regulate emotion independently (Kopp, 1989), they tend to use caregiver-reliant strategies to aid in emotion regulation. Beginning in early infancy, infants learn to respond to distress by engaging in strategies that signal their need for regulatory help from caregivers. These strategies include seeking physical comfort, attracting functional assistance, and focusing attention on a caregiver (Blandon et al., 2008; Calkins, Gill, Johnson, & Smith, 1999; Grolnick, Bridges, & Connell, 1996). When infants utilize maladaptive emotion regulation strategies, such as sustaining focus on frustrating stimuli, they experience increased anger and distress (Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002; Grolnick et al., 1996). During preschool years children are able to engage in more active and planned activities to independently regulate their emotions. For instance, young children are able to regulate their emotions adaptively by refocusing attention away from distressing stimuli toward nondistressing stimuli (Derryberry & Rothbart, 1988), and reevaluating frustrating situations in a positive

manner (Kalpidou, Power, Cherry, & Gottfried, 2004; Stansbury & Sigman, 2000); these strategies are effective in reducing distress (Buss & Goldsmith, 1998; Calkins & Johnson, 1998; Grolnick et al., 1996). The utilization of other-reliant versus autonomous regulation strategies evolves throughout development.

Stability and Consistency of Emotion Regulation Strategies. Throughout the first few years of development a child's emotion regulation repertoire develops extensively. For instance, regulation of emotional response to pain during medical examinations increases with age (Axia & Bonichini, 1998). Interestingly, in response to the same medication examination, the use of emotion regulation strategies were stable between 3 and 5 months, but differed between 8 and 11 months.

In addition to understanding the stability of emotion regulation strategies (across time), it is important to determine the consistency of emotion regulation strategies across different contexts. Based on definitions of emotion regulation proposed throughout the literature, it seems that emotion regulation strategies should differ depending on situational demands. For instance, emotion regulation has been defined as a continuous processing of emotions as they are associated with "contextual demands" (Cole, Martin, Dennis, 2004). Diener (1996) examined the use of emotion regulation strategies across a number of tasks designed to elicit different emotions (fear, frustration, positive affect). Interestingly, although infants were able to employ effective strategies across different tasks (i.e., consistency in effectiveness), the specific regulatory behaviours differed across tasks (i.e., inconsistency in strategies). Therefore, when examining emotion regulation strategies, not only is it important to take into account the developmental status, it is also crucial to consider the environmental context.

Influence of Parenting on the Development of Emotion Regulation Skills. Parenting exerts a strong influence on the development of adequate emotion regulation skills. There is extensive evidence that during the first few years of life external (i.e., parental) support is imperative for the development of emotion regulation skills (Feng et al., 2008; Kopp 1989). Particularly early on, infants are less equipped and more dependent on their parents to directly provide emotional regulation (McLennan & Offord, 2003). Parental socioemotional support influences the development of children's emotion regulation abilities (Zeman & Shipman, 1998). For instance, it has been suggested that parental displays of warmth enhance young children's attempts to self-regulate their emotions (Eisenberg et al., 2005). Parents who more frequently express positive emotions have children who utilize active emotion regulation skills and have higher levels of social competence (Denhan, Mitchell-Copeland, Strandberg, Auerbach, & Blair, 1997; Grolnick et al., 1996). Parental involvement also enhances infants' active emotion regulation strategies (Grolnick et al., 1996, 2006). Many of the parental qualities that enhance infants' emotion regulation development, such as high warmth (Cummings, Keller, & Davies, 2005) and low criticism (Rogosch, Cicchetti, & Toth, 2004), are lacking in depressed mothers, which likely contributes to poorer emotion regulation in their children.

Maternal Depression and Infant Emotion Regulation

Children with a depressed caregiver are at increased risk for a wide range of socioemotional deficits (Campbell et al., 2004), including less effective emotion regulation abilities (e.g., Hoffman, Crnic, & Baker, 2006). According to Hoffman and colleagues (2006), depressed mothers, compared to nondepressed mothers, are three times more likely to have children who display emotional dysregulation. Lower levels of maternal depressive symptoms predict improvement in children's emotion regulation strategies (measured by the Emotion

Regulation Checklist; Shields & Cicchetti, 1997, 2001) over a three-year period (from ages 4 to 7), whereas higher levels of depressive symptoms predict stable emotion regulation abilities over this time period (Blandon et al., 2008). The aforementioned emotion regulation abilities included emotional understanding and empathy, angry reactivity, emotional intensity, and dysregulation of positive emotions (Blandon et al., 2008).

One likely reason that children of depressed parents show less adaptive emotion regulation strategies is that these strategies are shaped through the parent-child relationship, which is often disrupted by maternal depression (Goodman & Gotlib, 1999). For instance, depressed mothers' effective scaffolding abilities predicted their children's emotion dysregulation one year later (Hoffman et al., 2006). Given that the development of emotion regulation is highly reliant on the social context (Eisenberg, Cumberland, & Spinrad, 1998), the suboptimal mother-child interactions associated with depression may influence children's emotion regulation abilities.

As children age they learn from their parents' guidance how to effectively deal with emotional states; yet depressed mothers often have suboptimal emotion regulation abilities themselves, thereby increasing their potential to model dysfunctional emotion regulation strategies (Silk et al., 2006; Feldman, 2007; Feng et al., 2008). Emotion regulation deficits are both a risk factor for the development of depression and a symptom of depression. Depressed adults endure extensive emotion regulation difficulties (Bradley, 2000). Mothers who are depressed model this dysregulation by displaying abnormal affective interactions with their children (Goodmand & Gotlib, 1999). In addition, they may not have the skill set required to teach, encourage or reward their child's adaptive emotion regulation (Silk et al., 2006).

Therefore, in addition to being less responsive to her infant's signals for regulatory help, depressed mothers may model maladaptive or ineffective emotion regulation strategies.

Given that depressed mothers are less responsive to their infants' signals for regulatory help (e.g., Feldman, 2003), their infants often engage in self-regulatory behaviours (e.g., self-soothing, focusing attention on objects; Manian & Bornstein, 2009). When infants attempt to engage their depressed caregiver, but she is unresponsive, the infant will often resort to self-directed regulation (Tronick & Gianino, 1986). At such a young age, infants are not equipped to self-regulate. Infants need help regulating their emotions; they rely heavily on their caregivers for successful emotion regulation and distress reduction. Relying on self-regulation, particularly at a young age when the independent emotion regulation repertoire is not entirely developed, can exacerbate infants' distress (Manian & Bornstein, 2009).

Present Study and Hypotheses

In the current study we sought to delineate the associations amongst maternal depression, infant emotion regulation strategy, and infant HPA axis activity. The literature supports an association between maternal depression and both emotional and physiological regulatory capabilities of offspring but it does not speak to the relations amongst all three simultaneously. More importantly, the association between maternal depression and infant cortisol is equivocal; often depending on the index of cortisol, measure of depression, and stressor paradigm. In order to better understand the mechanism underlying the association between maternal depression and offspring HPA axis functioning, I assessed the potential moderating role of infant emotion regulation capacities (particularly self-regulatory strategies). The literature demonstrates that a) infants of depressed mothers often, but not always, display higher cortisol reactivity to laboratory stressors, b) infants of depressed mothers are less able to rely on their caregivers for assistance in

regulating emotions, which results in a tendency to employ independent regulatory strategies, and c) child features related to emotion regulation difficulties (i.e., insecure attachment and internalizing symptoms) moderate the relation between maternal depression and child cortisol levels. Therefore, I hypothesized that infants with more depressed mothers would have significantly higher cortisol levels after a stressor task, but only if they utilize independent emotion regulation strategies.

The moderating role of infant emotion regulation strategies was examined across two different stressor paradigms (Toy Frustration Task and Strange Situation Procedure). However, emotion regulation strategies were only coded during the Toy Frustration Task. Given the previous research demonstrating contextual inconsistencies in emotion regulation strategies (Diener, 1996), our hypothesis is targeted primarily to the Toy Frustration Task context. The moderation hypothesis was tested during the Strange Situation Procedure simply for exploratory purposes.

Method and Material

Participants

The community sample of mother-infant dyads was recruited from postings and in-person visits at community centers as well as baby show conventions in the Greater Toronto Area (GTA). Inclusion criteria for participation were that infants were healthy with no major developmental disorder, pregnancy was over 32 weeks; mothers were 18 years or older at childbirth, had no known hormonal disorders, and were fluent in English. This sample is part of a larger longitudinal study, termed the Toronto Longitudinal Cohort (TLC), which consists of 314 mother-infant dyads. This study examines data collected during a home visit, where mother-infant dyads participated in a Toy Frustration Task (Braungart-Rieker & Stifter, 1996), and a

laboratory visit, where dyads participate in the Strange Situation Procedure (Ainsworth et al., 1978). Video recording was introduced part way through the study. Of the 314 participants in the larger study, 193 dyads participated in the video-recorded Toy Frustration task, and thus only this subset was included in the present analyses. The subset of infant-mother dyads included in the analysis did not differ significantly on dependent variables, independent variables, and many demographic variables (e.g, infant sex, maternal income, education, or smoking status; Table 1). Infants included in the analysis were significantly younger compared to infants in the larger sample, t (286) = 2.99, $p < .01$.

See Table 1

This sample included 100 (51.8%) female infants. At the time of the home and lab visits, infants were approximately 15-months-old (M = 15.41months; SD = 1.00) and 17-months-old (M = 16.69 months; SD = 1.08) respectively. Mothers ranged from 21 to 46 years (M = 33.03 years; SD = 4.67). The majority of the sample was Caucasian (72.3%), with a smaller proportion of Asian (10.1%), Afro- Canadian (3.9%) and 'other' ethnicities (13.6%). The majority of mothers were highly educated, with post-graduate (24.5%), undergraduate (45.3%), community college (22.2%), secondary school (7.1%) and primary school (0.9%) as their highest level of education. The greater part of the sample had high family income, >$200, 000 (23.7%), $150,000-200,000 (18.5%), $114,000-150,000 (22.2%), $92,000-114,000 (16.9%), $70,000-92,000 (10.1%), $35,000-70,000 (5.3%), and $20,000-35,000 (3.2%). This is a demographically low risk sample.

Procedure

During the home visit, two female research assistants observed mother-infant interactions during a Toy Frustration Procedure (Braungart-Rieker & Stifter, 1996; Atkinson et al, under revision). The Toy Frustration Procedure was videotaped and later coded for independent infant-regulatory behaviours. Coders were blind to all other aspects of the study. One month later, mother-infant dyads visited the laboratory, where they participated in the Strange Situation Procedure (Ainsworth et al., 1978). The Strange Situation Procedure was solely used for the purposes of a stressor; it was not used to code infant attachment style. Self-report inventories and saliva samples were collected during both the home and lab visits. All appointments occurred between 9 and 10 AM. Infant daytime, particularly afternoon, cortisol levels are highly variable due to daytime stressors, meals, and naps. Thus this study design enables greater sensitivity to infant reactivity given that morning cortisol is more stable for infants (Goldberg et al., 2003; Gunnar & White, 2001).

Challenges

Toy Frustration Procedure. At approximately 15 months, mother-infant dyads participated in the Toy Frustration Procedure (TFP; Braungart-Rieker & Stifter, 1996), which consisted of four episodes, each lasting 90-seconds in duration. 1) The mother engaged the infant with a toy, 2) the mother placed the toy in a clear container with the lid on (but not sealed), while depicting a "still-face" and not helping the infant regain possession of the toy, 3) the toy was removed from the container and the infant played with it, and 4) the mother placed the toy in the clear container with the lid sealed shut, while continuing to disengage. This procedure was discontinued if the infant cried continuously for 20 seconds. The fourth episode is the most frustrating and distressing for infants because the lid is fixed, and thus they are unable to retrieve

the toy; this episode also includes the cumulating stress from previous episodes. Thus, the fourth episode is the focus of subsequent investigation and analyses.

Strange Situation Procedure. At approximately 17-months dyads participated in the Strange Situation Procedure (SSP; Ainsworth et al., 1978). The SSP is a maternal separation procedure which consists of seven 3-minute episodes, designed to induce distress in the infant and evoke attachment-related behaviours (although for the purposes of this study, the SSP was only used as a stressor). During these episodes, 1) the infant and mother interact alone, 2) a female stranger joins and interacts with the infant and mother, 3) the mother leaves the room, and the female stranger and infant interact, 4) the mother returns and the female stranger leaves, 5) the mother leaves the infant alone in the room, 6) the stranger returns to the room, and 7) the mother returns and the stranger leaves the room. If the infant cried for more than 20 seconds, the procedure was terminated.

Saliva Collection

In order to avoid contamination, participants were asked to refrain from brushing their teeth, eating, and drinking one-hour before saliva was sampled (Kirschbaum & Hellhammer, 1989; Kirschbaum & Hellhammer, 1994). Saliva was collected from the dyad at 5 min pre-challenge and 20, 40, and 60 min after each challenge using Sorbettes (Salimetrics, State College PA) which were placed in the mother's and infant's mouth for 60 seconds. Post-stressor collection between 20 and 30 minutes is typical (Gunnar et al., 2009). Goldberg and colleagues (2003) found that some individuals peak at 20-mins and some peak at 40-min collections. Two Sorbettes were collected for each individual, and placed in a 2-mL cryovial and sealed.

Cortisol Assays

Saliva samples were centrifuged for 10-minutes at 3000 rpm to extract the saliva and then

stored in a freezer at -70 °C. Salivettes were thawed and centrifuged for 10-minutes at 3,000 rpm at 4°C. Saliva samples were assayed using a salivary cortisol enzyme immunoassay kit (Salimetrics, State College, PA). Samples from each dyad across both visits (15- and 17-months) were assayed together. All samples were assayed twice and average values were used in analyses. The interassay variability was 10.6%; the intraassay variation was 8.3%, for samples with low values, and 6.9 % for samples with high values.

Cortisol Indices

Using all three cortisol samples (i.e., baseline, 20-, and 40 minutes post challenge), two separate indices of cortisol were calculated. The first index represents the total magnitude of cortisol production, known as AUC_G (area under the curve with respect to ground). AUC_G is a measure of the total hormone output; it assesses both sensitivity (the difference between single measurement points) and intensity (the distance of single measurement points from the ground) (Fekedulegn et al., 2007). AUC_I (area under the curve with respect to increase) is a measure of cortisol change over time (Fekedulegn et al., 2007). AUC_I is related to HPA reactivity. An advantage of using AUC_I rather than a simple change index, is that the AUC_I index captures more than two measurements of cortisol. AUC_G and AUC_I are two of the most common indices of cortisol (Fekedulegn et al., 2007; Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). Although both AUC_I and AUC_G include cortisol levels from all three time points, AUC_G is effected by baseline levels, whereas AUC_I does not include the distance from zero to all data points in its measurement. In fact, AUC_G and AUC_I are often not related to one another, and are differently related to other indices of cortisol (e.g., reactivity, slope, peak etc.; Fekedulegn et al., 2007).

In the present study, we aimed to use AUC_G and AUC_I as independent measures of

cortisol. AUC_G and AUC_I were significantly correlated during both the Toy Frustration and Strange Situation Procedures (*rho* = -.17, $p < .05$, *rho* = .39, $p < .01$, respectively). Although these indices are not completely independent in the current sample, there is still unexplained variance between the two indices, suggesting they are measuring distinct aspects of the cortisol response.

Measures

Maternal Depression. Mothers completed the Beck Depression Inventory (BDI-II; Beck, Steer & Brown, 1996), a 21-item self-report questionnaire with a 4-point scale (ranging from 0 to 3). Each score on the 21-items of the BDI-II are summed to derive a total score of depressive symptoms (Beck et al., 2006). The BDI-II has good psychometric properties in diverse samples. The Cronbach's alphas in an outpatient and college sample were .92 and .93 respectively, with high internal consistency for both males and females (92 and .91 respectively; Dozois, Dobson, & Ahnberg, 1998). In addition, the BDI has been utilized repeatedly to assess depressive severity of mothers (e.g., Gelfand & Teti, 1990; Lovejoy, Graczyk, O'Hare & Newman, 2000; Murray & Cooper, 1997). In a sample of pregnant women and in the postpartum period, Cronbach's alphas were 0.88 and 0.89 respectively (Bos et al., 2009).

Infant Regulatory Behaviours. Given that infants of depressed mothers are more likely to utilize independent emotion regulatory strategies (Manian & Bornstein, 2009), infant self-regulatory behaviours were coded during the Toy Frustration Task. Infants self-regulate their emotions by avoiding or withdrawing from distressing stimuli (Rothbart & Derryberry, 1981), self-soothing (e.g., thumb sucking; Rothbart & Derryberry, 1981), and using attentional distraction strategies (e.g., averting gaze; Field, 1981; Stifter & Braungart, 1995). In order to capture the various forms of infant regulation, in the current study, we coded the duration infants

displayed a number of independent regulatory behaviours, including withdrawing from the task, distracting him/herself, wandering away from the task, orienting to another object, and scanning the environment (see Table 2 for definitions of each strategy). Each behaviour was coded in terms of the duration it was displayed by the infant. Researchers have argued that self-soothing behaviours are rarely exhibited by infants older than 10 months (Stifter & Braungart-Rieker, 1995), thus, it was not included in the independent regulation composite.

The Toy Frustration Task was coded by a research assistant. Videos were coded for the duration infants engaged in each behaviour. Twenty percent of the videos were coded by a second coder to ensure inter-rater reliability (r = .84).

See Table 2

Statistical Analyses

To account for missing data, multiple imputations were conducted, after which two separate multiple regression analyses (one predicting total cortisol output and one predicting cortisol reactivity) were conducted using the imputed data. Multiple imputations were only conducted for subjects who had a video recording of the Toy Frustration procedure (N = 193), data were not imputed for the entire sample (N = 314).

To account for potential error due to missing values, this procedure takes multiple random draws from the population and imputes values multiple times (Graham, 2009). In the present analysis, using SPSS, twenty imputations were conducted, which is well above the recommended amount (Graham, 2009; Collins, Shafer & Kam, 2001). As noted by Collins et al. (2001), in order to conduct an "inclusive" multiple imputation, in addition to the independent and

dependent variables, variables that are not part of the main model, but correlate highly with the variables in the model (i.e., the covariates), were included in the imputation model. Thus, the imputation model included maternal depression, the composite of infant independent emotion regulation strategies, infant cortisol indices (AUC_G and AUC_I), and infant breakfast time (the only significant covariate). The average of the twenty imputations for both the model significance and the pooled predictors are reported below.

During the toy frustration procedure (at 15 months), the percent of missing values ranged from 2.06 % for the composite of emotion regulation behaviours to 14.4% for BDI-II scores (10.31% due to drop out and 4.12% due to incomplete questionnaires). Values were imputed for all variables, regardless of the percent of missing data. To determine if the data is suitable for imputation a test must be conducted to demonstrate that the missing values are missing randomly, also known as MCAR (missing completely at random). Based on Little's MCAR test, χ^2 (9) = 8.18, p = .52, this data is missing at random and thus imputation is appropriate. During the strange situation procedure (at 17 months), the percent of missing values ranged from 2.06 % for the composite of emotion regulation behaviours and 25.8% for infant cortisol reactivity (AUC_I). Values were imputed for all variables, regardless of the percent of missing data. Based on Little's MCAR (missing completely at random) test, these data are *not* missing at random, χ^2 (13) = 31.59, p = .003. Because the data from the strange situation laboratory visit are not missing at random and thus *not* suitable for imputation, all analyses were completed *without* imputation.[1]

Results

[1] Although the analyses for the strange situation procedure were not suitable for multiple imputation, the results do not differ whether or not multiple imputation was used.

Descriptive Statistics

BDI total scores ranged from 0 to 36 (Median = 6.00, Interquartile Range = 7.00). The majority (94.5%) of the mothers did not exceed the BDI cut-off for clinical depression (Table 3). The duration of infant independent regulatory behaviours during the third episode ranged from 0 to 189.73 seconds (Median = 57.96, Interquartile Range = 68.23).

See Table 3

At 15 months, infant AUC_G scores ranged from 30.72 to 908.68 (Median = 148.28, Interquartile Range = 109.65) and AUC_I scores ranged from -662.28 to 254.00 (Median =-25.65, Interquartile Range = 62.15). At 17 months, AUC_G scores ranged from 35.60 to 5354.19 nmol/L (Median = 160.12, Interquartile Range = 144.84) and AUC_I scores ranged from -583.23 to 2338.25 nmol/L (Median = 14.45, Interquartile Range = 89.72). We log transformed both cortisol indices to minimize skew. After log transformations the AUC_G variable mirrored a normal distribution; however the AUC_I variable still had a leptokurtic distribution.

Preliminary Analyses

Demographics. Neither of the dependent variables, AUC_G or AUC_I, significantly correlated with sex of the baby, maternal education, maternal income, or maternal smoking status.

Wake and Feeding Times. Time of last infant feeding significantly correlated with AUC_G ($rho = -.17$, $p < .05$) at 15-months. We covaried infant breakfast time for subsequent analyses involving AUC_G. There were no other significant covariates.

Main Analyses

Exploratory Correlations. Table 4 displays the correlations amongst all study variables. There is a small negative correlation between infants cortisol reactivity (AUC_I) and total cortisol output (AUC_G) during the Toy Frustration Procedure, *rho* = -.17, $p < .05$. During the Strange Situation Procedure, infant AUC_G and AUC_I were positively correlated, *rho* = .39, $p < .01$. The opposing valences of the correlations between AUC_G and AUC_I during the Toy Frustration (negative correlation) and Strange Situation (positive correlation), are likely related to the cortisol trajectories during these tasks. More specifically, infant cortisol levels are declining during the Toy Frustration Task and increasing during the Strange Situation Procedure, thus AUC_G and AUC_I are negatively associated during the Toy Frustration Task and positively associated during the Strange Situation Procedure. There is also a positive correlation between infant AUC_G during the Toy Frustration and Strange Situation Procedures, *rho* = .27, $p < .01$. Maternal depression is positively correlated with the composite of infant independent regulatory behaviours, *rho* = .16, $p < .05$.

See Table 4

Toy Frustration Task. Two separate multiple regression analyses were conducted to examine whether maternal depression, infant regulatory behaviour, and their interaction predicted the infant's cortisol responses during the Toy Frustration Task at 15 months. In the first analysis, the model significantly predicted infants' AUC_G, F (4, 189) = 3.50, $p < .05$, adjusted R^2 = .069. The standardized regression coefficients shown in Table 5 indicate that only the interaction between maternal depression and infant regulatory strategy made a significant contribution to infant total cortisol output (AUC_G). The second analysis was conducted with

cortisol increase (AUC_I) as the dependent variable; the model was significant F (3, 190) = 3.42, p < .05, adjusted R^2 = .036. As shown in Table 5, the interaction between maternal depression and infant regulatory strategy significantly contributed to the prediction of AUC_I[2].

The interaction between maternal depression and infant independent regulation as they predict AUC_G and AUC_I are displayed in Figures 4 and 5 respectively. Although all variables are continuous, in order to depict associations, maternal depression score and infant emotion regulation strategy were dichotomized based on one standard deviation above and below the mean (Aiken & West, 1991). As shown in Figure 4, infants who had mothers with higher levels of depressive symptoms and utilized higher independent emotion regulation strategies, had the highest level of total cortisol output (AUC_G); whereas infants who had mothers with higher levels of depressive symptoms but utilized lower levels of independent emotion regulation strategies had lower AUC_G levels. As shown in Figure 5, infant cortisol reactivity (AUC_I) did not differ greatly depending on infant use of independent emotion regulation strategies, when mothers had lower levels of depressive symptoms; however, when mothers had higher levels of depressive symptoms, infants who used more independent regulation strategies had the highest AUC_I levels, and infants who used less independent regulation strategies had the lowest AUC_I levels.

These findings demonstrate that infants whose mothers endorse more depressive symptoms and who display higher levels of independent emotion regulation, experience higher total cortisol output (AUC_G) and cortisol reactivity (AUC_I). It is important to note that in the current analysis AUC_G and AUC_I have a small negative correlation (*rho* = -.17). This small correlation leaves a large amount of unexplained variance, suggesting that although not

[2] When conducted without multiple imputation, the moderation models predicting AUC_G (F (4, 128) = 2.739, p < .05, adjusted R^2 = .05) and AUC_I (F (3, 139) = 2.50, p < .05, adjusted R^2 = .053) remain significant.

completely independent, these indices are measuring different components of the cortisol response.

See Figure 4 and 5

See Table 5

Strange Situation Procedure. For the purposes of this thesis, exploratory secondary multiple regression analyses were conducted for the Strange Situation. The only difference in these analyses is that multiple imputation was not used for analyses of the Strange Situation procedure. Contrary to the Toy Frustration results, the overall regression models during the Strange Situation, AUC_G (F (3, 133) = .874, p = .46) and AUC_I (F (3, 127) = .177, p = .912), were not significant (Table 6).

See Table 6

Discussion

The present study investigated infant independent emotion regulation strategy as a potential moderator of the relation between maternal depressive symptoms and two distinct indices of infant cortisol, total cortisol output (AUC_G) and increase in cortisol (AUC_I), during two divergent stressor paradigms (Toy Frustration and Strange Situation procedures). The extant literature yields inconsistent results regarding the relations between maternal depression and infant cortisol, calling for the assessment of potential moderators. In previous research, maternal

depression predicted higher cortisol reactivity for infants classified as insecure-resistant, but not for infants classified as secure or avoidant (Luijk et al., 2010). In addition, children of depressed mothers who had higher levels of internalizing symptoms, evinced higher cortisol output (Ashman et al., 2002). Both insecure attachment patterns (Cassidy, 1994) and internalizing symptoms (Eisenberg et al., 2001) can be considered manifestations of emotion regulation difficulties. In fact, depressed mothers themselves display deficits in emotion regulation (e.g., Bradley, 2000), which has previously been shown to adversely affect mother-child interaction (e.g., Goodman & Gotlib, 1999) and hinder effective modeling of adaptive emotion regulation (Eisenber et al., 1998). In particular, we examined the moderating role of independent emotion regulation strategies because maternal depression is associated with less responsive caregiving, thereby contributing to infants depending less on caregiver-assisted strategies to regulate emotions (Manian & Bornstein, 2009). The current study is the first to directly investigate infant emotion regulation as a potential moderator of the relation between maternal depression and infant cortisol levels.

Overall Findings

The primary hypothesis pertained to the Toy Frustration Task. As hypothesized, infant emotion regulation strategy moderated the relation between the level of maternal depressive symptoms and two distinct indices of infant cortisol; infant total cortisol output (AUC_G) and increase in cortisol (AUC_I). Prior literature indicates that mothers who have more depressive symptoms are likely less responsive to their infants, and less able to assist their infants in emotion regulation. However, at this age, infants require their caregivers to aid in emotion regulation (Kopp, 1989). Therefore, infants who have more depressed mothers and rely less on their mothers for help regulating emotions (i.e., display more independent emotion regulation),

have higher total cortisol output and higher cortisol reactivity.

Moderation of Total Cortisol Output (AUC_G) and Cortisol Increase (AUC_I). Infants who had mothers with high levels of depression and who utilized high levels of independent regulation strategies had the highest total cortisol output (AUC_G). In comparison, infants with mothers with high levels of depression, but who utilized less independent emotion regulation strategies, had much lower AUC_G levels (Figure 4). The interaction between maternal depression and infant independent emotion regulation similarly predicted infant cortisol increase (AUC_I). For infants with mothers who had lower levels of depressive symptoms, their AUC_I levels did not differ greatly regardless of their level of independent emotion regulation. This relation differed for infants with mothers who had higher levels of depression. If these infants utilized higher levels of independent emotion regulation, they had high AUC_I levels; whereas if they utilized lower levels of independent emotion regulation they had low AUC_I levels (Figure 5).

Replication across these two indices of cortisol is important given that AUC_G and AUC_I capture different aspects of the cortisol response. AUC_G is an index of total cortisol output, it takes into account baseline levels, as well as the difference between subsequent points from one another and from baseline. In comparison, AUC_I is a measure of cortisol change; it does not have a reference to baseline levels. An advantage of both of these cortisol indices is the inclusion of multiple cortisol assessments (baseline, 20- minute, and 40-minute) in single indices. Given that previous research (e.g., Gunnar et al., 2009) has shown that frustration paradigms are not potent stressors, it is particularly important that this finding was replicated across two cortisol indices. Replication across AUC_G and AUC_I suggests that infants who have more depressed mothers and who utilize more independent emotion regulation strategies secrete more cortisol and have greater change in cortisol concentrations.

For exploratory purposes, the moderation model was also examined during the Strange Situation Procedure, which occurred during the follow-up laboratory visit. However, the findings from the Toy Frustration task were not replicated. During the Strange Situation Procedure, the multiple regression analyses predicting both AUC_G and AUC_I were not significant. These null findings are consistent with previous research demonstrating that emotion regulation strategies are inconsistent across childrens' age (Axia, & Bonichini, 1998) and situational task demands (Diener, 1996). In the current study, emotion regulation strategies were coded during the Toy Frustration task, and may not generalize across time or stressor paradigm. The independent emotion regulation strategies coded (i.e., duration of wandering off, withdrawal, distraction, orienting to another object, and scanning) are specifically relevant to the Toy Frustration task. The demands of a frustration procedure (Toy Frustration) are very different from the demands of a separation paradigm (Strange Situation), and thus likely evoke very different emotion regulation strategies. Extending these regulation strategies to a different stressor may not be viable for two reasons: 1) the regulation techniques employed likely differ depending on the situation, that is, the strategies used during one stressor may not be the same as those used in another; and 2) irrespective of stressor, the strategies used at one time point may not be used at a later time. The interaction between maternal depression and infant emotion regulation is likely to differ based on the timing of assessment and the stressor task. Therefore, the nonreplication during the Strange Situation Procedure can likely be attributed to the discontinuity in emotion regulation strategies across different situation task demands and time points.

Maternal Depression and Infant Cortisol

The current study helps explain the ambiguity in the extant literature, which investigates the relation between maternal depression and infant cortisol, by examining the moderating role

of infant emotion regulation strategy. Maternal depression is known to influence infant cortisol (Field et al., 2004; Lundy, 1999). Furthermore, the effects of maternal depression on child cortisol may continue into adulthood, thereby contributing to risk for depression (Young et al., 2006). However, the association between maternal depression and infant cortisol levels is, to say the least, complex. There are a number of factors that influence this relation and may contribute to the existing ambiguity.

The association between maternal depression and child cortisol levels in response to laboratory and everyday stressors is unclear. Some studies show that maternal depression is associated with higher cortisol reactivity (Azar et al., 2007), whereas others do not find an association with cortisol reactivity (Brennan et al., 2008). One possible explanation for these inconsistencies is the variety of stressor paradigms utilized. Given that different stressors have differential ability to elicit cortisol responses (Gunnar et al., 2009; Jansen et al., 2010), these inconsistencies may be due to the stressor employed. Additional inconsistencies have been found based on the index of cortisol. For instance, Brennan and colleagues (2008) showed that lifetime depression was related to baseline and mean infant cortisol, but not cortisol reactivity; whereas peripartum depression was only related to cortisol reactivity. The current study attempted to account for discrepant stressor paradigms and cortisol indices, by implementing two different stressor tasks and utilizing two cortisol indices.

In addition to the aforementioned factors that contribute to the ambiguity in the literature (e.g., stressor paradigm, cortisol index), the extant research supports mother and child variables as moderators of the association between maternal depression and child cortisol.

Potential Moderator: Infant Emotion Regulation

In previous research, mother-infant interactive behaviours, in particular maternal

responsivity to infant distress, moderated the relation between maternal depressive symptoms and infant cortisol (Letourneau et al., 2011). Different child variables have also previously been shown to moderate this relation. Ashman and colleagues (2002) found that not all children of depressed mothers showed reactivity in cortisol, only those with high levels of internalizing symptoms had elevated cortisol reactivity. Similarly, infants of depressed mothers who displayed resistant attachment had elevated cortisol responses (Luijk et al., 2010). An underlying factor that may explain both of the aforementioned moderations is child emotion regulation. Emotion regulation ability is related to both internalizing symptoms and insecure attachment styles. Although previous research has demonstrated that child variables moderate the relation between maternal depression and child cortisol, this study extends previous research by directly investigating infant emotion regulation as a moderator of this relation.

Maternal depression is known to affect parenting: depressed mothers are less responsive (Righetti-Veltema, Conne-Perreard, Bousquet, & Manzano, 2002), less sensitive (Hatzinikolaou & Murray, 2010) and less likely to respond to their children's cures for assistance (Cox et al., 1987). Therefore, infants of depressed mothers are more likely to utilize independent emotion regulation strategies because they are less able to rely on their mothers for regulation help. Given that young infants are not equipped to self-regulate (Kopp et al., 1989), utilization of these independent regulation strategies potentially contributes to increased dysregulation (emotional and physiological). This is consistent with the findings of the current investigation: infant independent emotion regulation strategy moderated the relation between maternal depressive symptoms and infant cortisol. In fact, this moderation was demonstrated across two different indices of physiological regulation (total cortisol output and increase in cortisol), indicating that infants who have mothers with higher levels of depression and who use more independent

regulation, secrete a larger amount of total cortisol and have higher increases in cortisol.

High Cortisol Output: Adaptive or Maladaptive?

Elevated cortisol, an indication of HPA activation, is often interpreted as an adaptive response. Although elevated cortisol in response to stressful conditions is adaptive, long-term over-activation of the system is maladaptive. Thus, optimal HPA responses are flexible, indicated by reactivity in stressful but not unstressful circumstances (Atkinson et al., under review). Given that the Toy Frustration Task is not a potent stressor, elevated cortisol responses in the current study are interpreted as maladaptive. Infants who are unable to rely on their depressed mothers for external regulation, employ independent emotion regulation strategies, which contribute to elevated cortisol levels.

An alternative interpretation of the current findings is that elevated cortisol responsivity is adaptive. There is theoretical and empirical support for the notion that activation of the HPA axis is adaptive. HPA activation stimulates physiological and behavioural responses, which allow individuals to respond effectively to stressors. The notion that higher cortisol responses are adaptive is consistent with other data from the TLC cohort. For instance, Atkinson et al. (under revision) demonstrated that infants of highly sensitive mothers displayed higher cortisol output and more flexible cortisol responses across varying challenges.

In accordance, it is possible that for infants of depressed mothers, employing independent emotion regulation strategies and mounting elevated cortisol response, is adaptive given the environmental demands. In fact, a body of literature demonstrates that blunted cortisol responsivity is associated with adverse outcomes (e.g., Luby et al., 2003; Ouellet-Morin, et al., 2011). Perhaps infants who are unable to disengage from their depressed mothers, and show a blunted cortisol response are at greater risk. Thus, an alternative explanation of the current

findings is that infants who employ independent regulation and elevated cortisol responses, are mounting adaptive emotional and physiological responses, given the environmental constrains associated with maternal depression. Ultimately, this in an empirical question, which warrants further investigation.

Strengths of Current Research

To date, the direct role of child emotion regulation strategy has not been assessed as a moderator of the relation between maternal depressive symptoms and child cortisol. Therefore, the findings of the current study help elucidate mechanisms pertaining to infant emotion regulation strategies as they moderate the impact of maternal depression on infant HPA regulatory capacities. In addition, various methodological considerations augment the present study's contribution to the literature. Numerous methodological factors potentially contribute to the ambiguous stance of the extant literature concerning the influence of maternal depression on child HPA activity. The current study addressed two of these methodological considerations: 1) the differential effects depending on the index of cortisol and 2) diverse stressor paradigms evoke differing emotion regulation strategies and have differential ability to evoke a stress response.

Cortisol Indices. The relation between maternal depression and child cortisol levels often differs depending on the index of cortisol. For instance, Feldman et al. (2009) found that maternal depression was related to elevated infant baseline cortisol and cortisol reactivity. Contrarily, Brennan et al. (2008) showed that lifetime major depression was associated with infant baseline and mean cortisol, but not cortisol reactivity (Brennan et al., 2008). The current study utilized two divergent indices of cortisol, total cortisol output (AUC_G) and cortisol increase (AUC_I), both of which encompassed all three time points of cortisol assessment (baseline, 20

minutes, 40 minutes). During the Toy Frustration procedure, the interaction between maternal depression and infant emotion regulation influenced the magnitude of infant cortisol production throughout the duration of the task (AUC_G), as well as the level of cortisol increase after the onset of the task (AUC_I).

Diverse Stressor Paradigms. An additional confound in the extant literature is the variety of stressor paradigms utilized. Various stressor paradigms are known to have differential abilities to elicit cortisol responses (Gunnar et al., 2009; Jansen et al., 2010). For exploratory purposes, the current study used two stressor paradigms known to differentially influence infant cortisol responses. Based on the reviews by Gunnar and colleagues, frustration paradigms (e.g., the Toy Frustration Task) are less efficient stressors compared to separation paradigms (e.g., Strange Situation Procedure). Given the known utility of each paradigm, the results of the current investigation may seem contradictory. However, given that emotion regulation strategies are context specific (Dierner, 1996), it is understandable that the model is significant only during the Toy Frustration Task. In addition to the applicability and effectiveness of emotion regulation strategies likely differing depending on the stressor paradigm, it is possible that the potency of the stressor paradigm influences infants' ability to utilize adaptive emotion regulation strategies.

Depressive Symptoms in a Community Sample. The current sample of mother-child dyads is considered low risk in many domains (e.g., socioeconomic status, education level, smoking status). Most importantly, the range of depressive symptoms in this community sample is positively skewed. The ability to demonstrate a significant moderation model in this low risk sample may attest to the strength of the effect. In addition there is high intrinsic value in understanding the nature of these processes in a low risk sample, so as to understand typical

developmental processes. In the future, it would be useful to replicate this finding in a sample of clinically depressed mothers and their children.

Limitations

In addition to its strengths, this study has a number of limitations. To begin, the maternal depressive symptoms were only assessed at one time point. Previous research (e.g., Brennan et al., 2008) has demonstrated that the timing of maternal depressive symptoms (e.g., current, past, lifetime) influences the relation between maternal depression and child cortisol levels. Thus, it is possible that assessing different durations of depression may influence the current findings. In addition, it is possible that the null findings during the Strange Situation procedure are a facet of the design of the study. In order to determine if child emotion regulation strategy moderates the relation between maternal depression and child cortisol, this moderation should be further explored using additional stressor paradigms, while coding emotion regulation during each respective procedure. It is also possible that the null findings during the Strange Situation Procedure are a result of missing data. Given that data was missing non-randomly during the Strange Situation Procedure, we were unable to account for this missing data using multiple imputation.

Directions for Future Research

This study is the first to examine infant emotion regulatory strategy as a moderator of the relation between maternal depression and infant physiological regulation. Additional research can be conducted to better elucidate the association between maternal depression and child HPA axis regulation. Given that depression is qualitatively different in community and clinical samples (Coyne, 1994), the proposed moderation model should be tested with a clinical sample

of depressed mothers and their children. Also, a diverse range of methodological considerations should be employed in an attempt to replicate the current findings. For instance, the interaction between maternal depression and child emotion regulation can be assessed as a predictor of diurnal cortisol. To add, emotion regulation can be assessed during different circumstances (i.e., diverse stressor paradigms), and using different instrumentation (e.g., a physiological measure, such as respiratory sinus arrhythmia (RSA)). In addition, other infant variables (e.g., temperament, emotional reactivity) that are related to maternal depression or HPA activity should be tested as moderators or mediators of this relation. Along those lines, maternal variables (e.g., emotion regulation, HPA activity) associated with depression or infant HPA activity should be examined in a similar way. Lastly, it would be interesting to examine the impact of the proposed model on the developmental trajectory of the HPA axis. Growth curve analyses could elucidate whether early emotional and HPA regulatory abilities influence later HPA activity.

Clinical Significance

Children of depressed mothers are at heightened risk for a myriad of problems. Maternal depression is also associated with physical health complications in infants (e.g., growth retardation) (Rahman, 2004). Children of depressed mothers are more likely to have internalizing and externalizing problems (Weissman et al., 2006). They are also more likely to experience delays in expressive language development (Cox, Puckering, Pound & Mills, 1987), mild impairments in general cognitive ability (Petterson & Albers, 2001), and poor academic performance (Kurstjens & Wolke, 2001). A mechanism underlying these risks is is the atypical HPA activity associated with depression. The existing literature demonstrates an ambiguous relation between maternal depression and infant HPA activity. This study shows that the relationship between maternal depression and infant HPA functioning is moderated by infant

emotion regulation. Further investigation of this moderation can be informative for developing appropriate interventions for at risk infants and children.

Summary

The present findings indicate that infant emotion regulation strategy moderates the relation between maternal depressive symptoms and infant cortisol levels. Infants with more depressed mothers, who utilized more independent emotion regulation strategies, showed higher cortisol reactivity and greater total cortisol output. This moderation was significant during the Toy Frustration Procedure, but not the Strange Situation procedure. These results suggest that maternal depression does influence infant HPA functioning, but this association is dependent on infants' emotion regulation strategies.

References

Aiken, L. S., & West, S. G. (1991). Multiple regression: Testing and interpreting interactions. Thousand Oaks, CA, US: Sage Publications.

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the Strange Situation*. Hillside, NJ: Erlbaum.

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

Atkinson, L., Paglia, A., Coolbear, J., Niccols, A., Parker, K.C.H. & Guger, S. (2000). Attachment security: A meta-analysis of maternal mental health correlates. *Clinical Psychology Reivew, 20*(8), 1019-1040.

Axia, G. & Bonichini, S. (1998). Regulation of emotion after acute pain from 3 to 18 months: A longitudinal study. *Early Development and Parenting, 7,* 203-210.

Azar, R., Pawuette, D., Zoccolillo, 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.

Bakermans-Kranenburg, M. J., Van Ijzendoorn, M. H., Mesman, J., Alink, L. R. A., & Juffer, F. (2008). Effects of an attachment-based intervention on daily cortisol moderated by dopamine receptor D4: A randomized control trial on 1- to 3-year-olds screened for externalizing behavior. *Development and Psychopathology, 20,* 805-820.

Beardslee, W. R., Bemporad, J., Keller, M. D., & Klerman, G. L. (1983). Children of parents with major affective disorder: A review. *American Journal of Psychiatry, 140,* 825–832.

Beck, C.T. (1999). Maternal depression and child behaviour problems: a meta-analysis. *Journal of Advanced Nursing, 29*(3), 623-629.

Beck, A.T., Steer, R.A., Brown, G.K. (1996). *Manual for the Beck Depression Inventory,* 2nd ed. San Antonio, TX: The Psychological Corporation.

Bevans, K., Cerbone, A., & Overstreet, S. (2008). Relations between recurrent trauma exposure and recent life stress and salivary cortisol among children. *Development and Psychopathology, 20,* 257-272.

Bhagwagar, Z., Hafizi, S., & Cowen, P. J. (2005). Increased salivary cortisol after waking in depression. *Psychopharmacology, 182*, 54 –57.

Blair, C., Granger, D., Willoughby, M., & Kivlighan, K. (2006). Maternal sensitivity is related to hypothalamic-pituitary-adrenal axis stress reactivity and regulation in response to emotion challenge in 6-month-old infants. *Annuals of the New York Academy of Sciences, 1094,* 263–267.

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.

Bos, S. C., Pereira, A. T., Marques, M., Maia, B., Soares, M. J., Valente, J., Gomes, A., Macedo, A., & Azevedo, M. H. (2009). The BDI-II factor structure in pregnancy and postpartum: Two or three factors? *European Psychiatry, 24,* 334-340.

Bradley, S. J. (2000). Affect regulation and the development of psychopathology. New York: Guilford Press.

Braungart, J., & Stifter, C.A. (1991). Regulation of negative reactivity during the strange situation: Temperament and attachment in 12-month old infants. *Infant Behaviour and Development, 14,* 349 – 364.

Braungart-Rieker, J. M. & Stifter, C. A. (1996). Infants' responses to frustrating situations: Continuity and change in reactivity and regulation. *Child Development, 67*, 1767-1779.

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

Brockmeyer, Holtforth, Pfeiffe, Backenstrass, Friederich, & Bents. (2012). Mood regulation expectancies and emotion avoidance in depression vulnerability. *Personality and Individual Differences, 53,* 351-354.

Buss, K.A., & Goldsmith, H.H. (1998). Fear and anger regulation in infancy: Effects on the temporal dynamics of affective expression. *Child Development, 69*, 359–374.

Calkins, S.D., Gill, K.L., Johnson, M.C., & Smith, C.L. (1999). Emotional reactivity and emotional regulation strategies as predictors of social behavior with peers during toddlerhood. *Social Development, 8*, 310–334.

Calkins, S. D., & Johnson, M. C. (1998). Toddler regulation of distress to frustrating events: Temperamental and maternal correlates. *Infant Behavior and Development, 21,* 379–395.

Campbell, S. B., Brownell, C. A., Hungerford, A., Spieker, S. J., Mohan, R. & Blessing, J. S. (2004). The course of maternal depressive symptoms and maternal sensitivity as predictors of attachment security at 36 months. *Development and Psychopathology 16,* 231-252.

Cassidy, J. (1994). Emotion regulation: Influences of attachment relationships. *Monographs of the Society for Research in Child Development, 59*(2), 228-249.

Catelin, C., Tordjman, S., Morin, V., Oger, E., Sizun, J., 2005. Clinical, physiologic, and biologic impact of environmental and behavioral interventions in neonates during a routine nursing procedure. *Journal of Pain, 6*, 791—797.

Chrousos, G. P. (1998). Stressors, stress, and neuroendocrine integration of the adaptive response. The 1997 Hans Selye Memorial Lecture. *Annuals of the New York Academy of Sciences, 851*, 311-335.

Cohn, J. F. & Campbell, S. B. (1992). Infiuence of maternal depression on infant affect regulation. In D. Cicchetti & S. Toth (Eds), *Rochester symposium on developmental psychopathology: Vol. 4. A developmental approach to affective disorders* (pp. 103-130). Rochester, NY: University of Rochester

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development, 75,* 317–333.

Collins, L. M., Schafer, J. L., Kam, C. M. (2001). A comparison of inclusive and restrictive strategies in modern missing data procedures. *Psychological Methods, 6*, 330–51.

Cox, A. D., Puckering, C , Pound, A. & Mills, M. (1987). The impact of maternal depression in young people. *Journal of Child Psychology and Psychiatry,* 28, 917-928.

Coyl, D. D., Roggman, L. A., & Newland, L. A. (2002). Stress, maternal depression, and negative mother-infant interactions in relation to infant attachment. *Infnt Mental Health Journal, 23*(1-2), 145–163.

Coyne, J. C. (1994). Self-reported distress: Analog or ersatz depression? *Psychological Bulletin,*

116, 29–45.

Crockenberg, S. A., & Leerkes, E. M. (2006). Infant and maternal behavior moderate reactivity to novelty to predict anxious behavior at 2.5 years. *Development and Psychopathology, 18*(1), 17-34.

Crockenberg, S. C., Leerkes, E. M., & Barrig, P. (2008). Predicting aggressive behavior in the third year infant reactivity and regulation as moderated by maternal behavior. *Development and Psychopathology, 20*(1), 37-54.

Cummings, E.M., & Cicchetti, D. (1990). Toward a transactional model of relations between attachment and depression. In M.T. Greenberg, D.Cicchetti, & E.M. Cummings (Eds.), *Attachment in the preschool years: Theory, research and intervention*. Chicago: University of Chicago.

Cummings, E.M. & Davies, P.T. (1994). Maternal Depression and Child Development. *Journal of Child Psychology and Psychiatry, 35*(1), 75-112.

Cummings, E. M., Keller, P. S., & Davies, P. T. (2005). Towards a family process model of maternal and paternal depressive symptoms: Exploring multiple relations with child and family functioning. *Journal of Child Psychology and Psychiatry, 46,* 479–489.

Danese, A. & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology and Behavior, 106*, 29-39.

Denhan, 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*(1), 65-86.

Derryberry, D. & Rothbart, M. K. (1988). Arousal, affect, and attention as components of temperament. *Journal of Personality and Social Psychology, 55*(6), 958-966.

De Weerth, C. & Buitelaar, J. K. (2007). Childbirth complications affect young infants' behavior. *European Child and Adolescent Psychiatry, 16*(6), 379-388.

Dickerson, S. S. & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*, 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*, 63–80.

Diener, M. L. (1996). Emotion regulation in toddlers: Developmental changes and individual differences. *ProQuest Dissertations and Theses*.

Doizois, 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.

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

Eisenberg, N., Cumberland, A., & Spinrad, T.L. (1998). Parental socialization of emotion. *Psychological Inquiry, 9*, 241–273.

Eisenberg, N., Cumberland, A., Spinrad, T.L., Fabes, R.A., Shepard, S.A., Reiser, M., et al. (2001). The relations of regulation and emotionality to children's externalizing and internalizing problem behavior. *Child Development, 72*, 1112–1134.

Eisenberg, N., Zhou, Q., Spinrad, T. L., Valiente, C., Fabes, R. A., & Liew, J. (2005). Relations among positive parenting, children's effortful control, and externalizing problems: A three-wave longitudinal study. *Child Development, 76,* 1055–1071.

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*, 776–784

Fekedulegn, D. B., Andrew, M. E., Burchfiel, C. M., Violanti, J. M., Hartly, T. A., Charles, L. E., & Miller, D. B. (2007). Area under the curve and other summary indicators of repeated waking cortisol measurements. *Psychosomatic Medicine, 69,* 651–659.

Feldman, R., & Eidelman, A.I. (2007). Maternal postpartum behavior and the emergence of infant–mother and infant–father synchrony in preterm and full-term infants: the role of neonatal vagal tone. *Developmental Psychobiology, 49,* 290–302.

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 American Academy of Child and Adolescent Psychiatry, 48*(9), 919-927.

Feng, X., Shaw, D. S., Kovacs, M., Lane, T., O'Rourke, F. E., & Alarcon, J. H. (2008). Emotion regulation in preschoolers: the roles of behavioral inhibition, maternal affective behavior, and maternal depression. *Journal of Child Psychology and Psychiatry, 49*(2), 132–141

Field, T. M. (1981). Infant arousal, attention, and affect during early interactions. In L.Lipsitt (Ed.), *Advances in infancy* (Vol. 1, pp. 57-100). New York: Ablex.

Field, T. M. (1987). Affective and interactive disturbances in infants. In J.D. Osofsky (Ed.), *Handbook of infant development* (2nd Edn. pp.972-1005). New York: John Wiley.

Field, T., Diego, M., Dieter, J., Hernandez-Reif, M., Schanberg, S., Kuhn, C., et al. (2004). Prenatal depression effects on the fetus and the newborn. *Infant Behavior and Development, 27*, 216–229

Field, T., Diego, M., & Hernandez-Reif, M. (2010). Prenatal depression effects and interventions: A review. *Infant Behavior and Development, 33,* 409-418.

Field, T., Sandberg, D., Garcia, R., Vega-Lahr, N., Goldstein, S. et al. (1985). Pregnancy problems, postpartum depression, and early mother-infant interactions. *Developmental Psychology, 21*(6), 1152-1156.

Francis, S. J., Walker, R. F., Riad-Fahamy, D., Hughes, D., Murphy, J. F., & Gray, O. P. (1987). Assessment of adrenocrotical activity in term newborn infants using salivary cortisol determinations. *Journal of Pediatrics, 111,* 129-133.

Gaynes, B.N., Gavin, N., Meltzer-Brody, S., Lohr, K.N., Swinson, T., Gartlehner, G., Brody, S., & Miller, W.C. (2005). Perinatal depression: Prevalence, screening accuracy, and screening outcomes. Evidence Report/ Technology Assessment No 119.

Gelfand, D. M. & Teti, D. M. (1990). The effects of maternal depression on children. *Clinical Psychology Review, 10*(3), 329-353.

Gilliom, M., Shaw, D., Beck, J., Schonberg, M., & Lukon, J. (2002). Anger regulation in disadvantaged preschool boys: Strategies, antecedents, and the development of self-control. *Developmental Psychology, 38*, 222–235.

Gitau, R., Cameron, A., Fisk, N. M., & Glover, V. (1998). Fetal exposure to maternal cortisol. *Lancet, 352*(9129), 707-708.

Goldberg, S., Levitan, R., Leung , E., Masellis, M., Basile, V., Nemeroff, C .B, & Atkinson L., (2003). Stability of baseline and stress cortisol concentrations in 12-18-month-old infants: Issues of time, location, and stressor. *Biological Psychiatry*, *54*, 719-726

Goodyer, I. M., Tamplin, A., Herbert, J., & Altham, P. M. E. (2000). Recent life events, cortisol, dehydroepiandrosterone and the onset of major depression in high-risk adolescents. *The*

British Journal of Psychiatry, 177, 499-504.

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*, 458–490.

Graham, J. W. (2009). Missing data analysis: Making it work in the real world. *Annual Review of Psychology, 60*, 549-576.

Grolnick, W.S., Bridges, L.J., & Connell, J.P. (1996). Emotion regulation in two-year-olds: Strategies and emotional expression in four contexts. *Child Development, 67*, 928–941.

Grolnick, W. S., Bridges, L. J., & Connell, J. P. (1996). Emotion regulation in two-year-olds: Strategies and emotional expression in four contexts. *Child Development, 67,* 928–941.

Guilliams & Edwards (2010). Chronic stress and the HPA Axis: Clinical assessment and therapeutic considerations. *The Standard 9*(2), 1-12.

Gunnar, M. R., Brodersen, L., Krueger, K., & Rigatuso, J. (1996b). Dampening of adrenocortical responses during infancy: Normative changes and individual differences. *Child Development, 67*(3), 877-889.

Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology, 27*(2), 199–220.

Gunnar, M. (1992). Reactivity of the hypothalamic-pituitary-adrenocortisol system to stressors in normal infants and children. *Pediatrics, 90*, 491-497.

Gunnar, M.R., Nelson, C.A., (1994). Event-related potentials in year-old infants: relations with emotionality and cortisol. *Child Development, 65*, 80–94.

Gunnar, M. & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Psychological Review, 58,* 145-173.

Gunnar, M.R., (1990). *The Psychobiology of Infant Temperament*. Erlbaum, Hillsdale, NJ.

Gunnar, M., Tout, K., de Haan, N,M Pierce, S., & Stansbury, K. (1997). Temperament, social competence, and adrenocortical activity in preschoolers. *Developmental Psychobiology, 31,* 65-85.

Gunnar, M. R. & White, B. P. (2001). Salivary cortisol measures in infant and child assessment. In Singer LT, Zeskind PS, editors. *Behavioral assessment of the infant*. New York: Guildford Press, pp 167-189.

Gunnar, M. R., Talge, N. M., & Herrera, A. (2009). Stressor paradigms in developmental studies: What does and does not work to produce mean increases in salivary cortisol. *Psychoneuroendocrinology, 34,* 953-967.

Habib, K. E. et al. (2000). Oral administration of a corticotropin-releasing hormone receptor antagonist significantly attenuates behavioral, neuroendocrine, and autonomic responses to stress in primates. *Proceedings of the National Academy of Sciences, 97*(11), 6079-6084.

Habib, K. E., Gold, P. W., & Chrousos, G. P. (2001). Neuroendocrinology of stress. *Endocrinology Metabolism Clinics of North America, 30*(3), 695-728.

Hadijan, A. J., Chedin, M., Cochet, C., & Chambaz, E. M. (1975). Cortisol binding to proteins in plasma in the human neonate and infant. *Pediatric Research, 9,* 40-45.

Haley, D. W., & Stansbury, K. (2003). Infant stress and parent responsiveness: Regulation of physiology and behavior during still-face and reunion. *Child Development, 74*, 1534–1546.

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

55(4), 376-381.

Hatzinikolaou, K. & Murray, L. (2010). Infant sensitivity to negative maternal emotional shifts: Effects of infant sex, maternal postnatal depression, and interactive style. *Infant Mental Healthy Journal, 31*(5), 591-610.

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.

Jansen, J., Biejers, R., Riksen-Walraven, M., & de Weerth, C. (2010). Cortisol reactivity in young infants. *Psychoneuroendocrinology*, *35*, 329-338.

Kalpidou, M. D., Power, T. G., Cherry, K. E., & Gottfried, N. W. (2004). Regulation of emotion and behavior among 3- and 5-year-olds. *The Journal of General Psychology, 13*(2), 159-178.

Kaltsas, G. A. & Chrousos, G. P. (2007). The neuroendocrinology of Stress. In Cacioppo, Tassinary, & Berntson (Eds.) Handbook of Psychophysiology. Cambridge University Press, New York.

Kirschbaum, C., & Hellhammer, D. H. (1989). Salivary cortisol in psychobiological research: an overview. *Neuropsychobiology, 22*, 150–169.

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

Kirschbaum, C. & Hellhammer, D. H. (1989). Salivary cortisol in psychobiological research: An overview. *Neuropsychobiology, 22*, 150-169.

Kirschbaum, C., & Hellhammer, D.H. (1994). Salivary cortisol in psychoneuroendocrine

research: Recent developments and applications. *Psychoneuroendocrinology, 19*, 313-333.

Klug, I., Dressendorfer, R. A., Strasburger, C., Kuhl, G. P., Reiter, A., et al. (2000). Cortisol and 17-hydroxyprogesterone in saliva of healthy neonates: normative data and relation to body mass index, arterial cord blood pH and time of sampling after birth. *Biology of the Neonate, 78*, 22-26.

Kopp, C.B. (1989). Regulation of distress and negative emotions: A developmental view. *Developmental Psychology, 25,* 343-354.

Kurstjens, S. & Wolke, D. (2001). Effects of Maternal Depression on Cognitive Development of Children Over the First 7 Years of Life. *Journal of Child Psychology and Psychiatry*, *42*(5), 623-636.

Larson, M., White, B. P., Cochran, A., Donzella, B., & Gunnar, M. R. (1998). Dampening of the cortisol response to handling at 3 months in human infants and its relation to sleep, circadian cortisol activity, and behavioral distress. *Developmental Psychobiology, 33,* 327-337.

Laurent, H. K., Ablow, J. C., & Measelle, J. (2011). Risky shifts: How the timing and course of mothers' depressive symptoms across the perinatal period shape their own and infant's stress response profiles. *Developmental Psychopathoogy*, *23*, 521-538

Letourneau, N., Watson, B., Duffett-Leger, L., Hegadoren, K., & Tryphonopoulos, P. (2011). Cortisol patterns of depressed mothers and their infants are related to maternal-infant interactive behaviors. *Journal of Reproductive and Infant Psychology, 29*(5), 439-459.

Lovallo, W. R. & Thomas, T. L. (2000). Stress hormones in psychophysiological research: Emotional, behavioral, and cognitive implications. In Cacioppo, John T. , Tassinary,

Louis G., Berntson, Gary G. (Ed). Handbook of psychophysiology (2nd ed.). Cambridge University Press, New York.

Lovejoy, M., 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.

Luby, J. L., Heffelfinger, A., Mrakotsky, C., Brown, K., Hessler, M., & Spitznagel, E. (2003). Alterations in stress cortisol reactivity in depressed preschoolers relative to psychiatric and no-disorder comparison groups. *Archives of General Psychiatry* 60, 1248 –1255.

Luijk, M. P. C. M., Saridjan, N., Tharner, A., van Ijzendoorn, M. H., Bakermans-Kranenburg, M. J., Jaddoe, V. W. V., Hofman, A., Verhulst, F. C., & Tiemeier, H. (2010). Attachment, depression, and cortisol: Deviant patterns in insecure-resistant and disorganized infants. *Developmental Psychobiology, 52,* 441-452.

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.

Main, M. (1990). Cross-cultural studies of attachment organization: Recent studies, changing methodologies, and the concept of conditional strategies. *Human Development, 33*(1), 48-61.

Manian, N. & Bornstein, M H. (2009). Dynamics of emotion regulation in infants of clinically depressed and nondepressed mothers. *Journal of Child Psychology and Psychiatry 50*(11), 1410–1418.

Matagos, S., Moustogiannis, A., & Vagenakis, A. G. (1998). Diurnal variation in plasma cortisol levels in infancy. *Journal of Pediatric Endocrine Metabolism, 11,* 549-553.

Martinez, A., Malphurs, J., Field, T., Pickens, J., Yando, R., Bendell, D., Valle, C., & Messinger,

D. (1996). Depressed mothers' and their infants' interactions with nondepressed partners. *Infant Mental Health Journal, 17*(1), 74-80.

Martins, C. & Gaffan, E. A. (2000). Effects of early maternal depression on patterns of infant-mother attachment: A meta-analytic Investigation. *Journal of Child Psychology and Psychiatry, 41*(6), 737-746.

Maughan, A., Cicchetti, D., Toth, S. L., & Rogosh, 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*(5), 685-703.

McEwen, B. S. (1998). Protective and damaging effects of stress mediator. *New England Journal of Medicine, 338*(3), 171-179.

McEwen, B. S. & Wingfield, J. C. (2003). The concepts of allostasis in biology and biomedicine. *Hormones and Behavior, 43*(1), 2-15.

McLennan, J., & Offord, D. R. (2003). Clinical and policy implications for prevention. *Journal of the American Academy of Child & Adolescent Psychiatry, 42*, 5-13.

Murray, L. & Cooper, P. J. (1997). Postpartum depression and child development. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences, 27*(2), 253-260.

Nachmias, M., Gunnar, M., Mangelsdorf, S., & Parritz, R. H. (1996). Behavioral inhibition and stress reactivity: The moderating role of attachment security. *Child Development, 67*(2), 508-522.

Nomura, Y., Wickramaratne, P. J., Warner, V., Mufson, K., & Weissman, A. M. (2002). Family Discord, Parental Depression, and Psychopathology in Offspring: Ten-Year Follow-up. *Journal of American Academy of Child and Adolescent Psychiatry, 41*(4), 402-409.

Ouellet-Morin, I., Odgers, C. L., Danese, A., Bowes, L., Shakoor, S., Papadopoulos, A. S., Caspi, A., Moffitt, T. E., & Arsenault, L. (2011). Blunted cortisol responses to stress signal social and behavioral problems among maltreated/bullied 12-year-old children. *Biological Psychiatry, 70,* 1016-1023.

Patterson, S.M. & Albers, A.B. (2001). Effects of Poverty and Maternal Depression on Early Child Development. *Child Development, 72*(6), 1794-1813.

Posner, M. I., & Rothbart, M. K. (2003). Developing mechanisms of self-regulation. In *Annual progress in child psychiatry and child development: 2000-2001,* by Posner, M. I., Rothbart, M.K., 21-42. New York, NY.

Rahman, A., Iqbal, Z., Bunn, J., Lovel, H., & Harrington, R. (2004). Impact of maternal depression on infant nutritional status and illness. *Archives of General Psychiatry,61*(9), 946-952.

Righetti-Veltema, M., Conne-Perreard, E., Bousquet, A., & Manzano, J. (2002). Postpartum depression and mother-infant relationship at 3 months old. *Journal of Affective Disorders, 70*(3), 291-306.

Rogosch, F. A., Cicchetti, D., & Toth, S. L. (2004). Expressed emotion in multiple subsystems of the families of toddlers with depressed mothers. *Development and Psychopathology, 16,* 689–709.

Rothbart, M., & Derryberry, D. (1981). Development of individual differences in infant temperament. In M. Lamb & A.Brown (Eds.), *Advances in developmental psychology* (Vol. 1, pp.37-87). Hillsdale, NJ: Erlbaum.

Sapolsky, R.M., Romero, L.M., & Munck, A.U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Review, 21*, 55–89.

Schmidt, L. A., Fox, N. A., Rubin, K. H., Sternberg, E. M., Gold, P. W., Smith, C. C., & Schulkin, J. (1997). Behavioral and neuroendocrine responses in shy children. *Developmental Psychobiology, 30*, 127–140.

Segerstrom, S. C. & Miller, G. E. (2004). Psychological Stress and the Human Immune System: A Meta-Analytic Study of 30 Years of Inquiry. *Psychological Bulletin, 130* (4), 601-630.

Shields, A. & Cicchetti, D. (1997). Emotion regulation among school-age children: The development and validation of a new criterion Q-Sort Scale. *Developmental Psychology, 33*(6), 906-916.

Shields, A. & Cicchetti, D. (2001). Parental Maltreatment and Emotion Dysregulation as Risk Factors for Bullying and Victimization in Middle Childhood. *Journal of Clinical Child & Adolescent Psychology, 30*(3), 349-363.

Siepak, K.J. (2008). Longitudinal influences of maternal sensitivity on infant temperamental reactivity and emotion regulation. *Dissertation Abstracts International Section B: The Sciences and Engineering, 69*(7-B), 4462.3

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

Stansbury, K. & Sigman, M. (2000). Responses of Preschoolers in two frustrating episodes: emergence of complex strategies for emotion regulation. *The Journal of Genetic Psychology: Research and Theory on Human Development*, *161*(2), 182-202.

Stein, A., Gath, D.H., Bucher, J., Bond, A., Day, A. & Cooper, P.J. (1991). The relationship between post-natal depression and mother-child interaction. *British Journal of Psychiatry, 158,* 46-52.

Stifter, C.A. & Braungart, J.M. (1995). The regulation of negative reactivity in infancy: Function and development. *Developmental Psychology, 31*(3), 448-455.

Streiner, D. L. (2003). Being inconsistent about consistency: when coefficient alpha does and doesn't matter. Journal of Personality Assessment, 80, 217–222.

Thompson, R.A. (1994). Emotion regulation: A theme in search of definition. In N.A. Fox (Ed.), *Monographs of the Society for Research in Child Development*, 59, (2–3, serial no. 240, pp. 25–52). Chicago: University of Chicago Press.

Tronick, E.Z. (1989). Emotions and emotional communication in infants. *American Psychologist, 44,* 112-119.

Tronick, E.Z. & Gianino, A.F. (1986). The transmission of maternal disturbance to the infant. In E. Z. Tronick & T. Field (Eds), *Maternal depression and infant disturbance* (pp. 5-11). San Francisco: Jossey-Bass.

Ulrich-Lai Y. M. & Herman, J. P. (2009). Neural regulation of endocrine and autonomic stress responses. *Nature Reviews Neuroscience,10*(6), 397–409.

Watamura, S. E., Donzella, B., Kertes, D. A., & Gunnar, M. R. (2004). Developmental changes in baseline cortisol activity in early childhood: Relations with napping and effortful control. *Developmental Psychobiology, 45,* 125-133.

Weissman, M.M, Pilowsky, D.J. & Wickramarante, P.J. (2006). Remissions in maternal depression and child psychopathology. *The Journal of the American Medical Association, 495*(12), 1389-1398.

Welsh-Allis, G. & Ye, W. (1988). Psychopathology in children of parents with recurrent depression. *Journal of Abnormal Child Psychology,* 16, 17-28.

Young, E.A., Vazquez, D., Jiang, H., & Pfeffer, C.R. (2006). Salivary cortisol and response to dexamethasone in children of depressed parents. *Biological Psychiatry, 60*, 831–836.

Zahn-Waxler, C , Denham, S., Iannotti, R. & Cummings, E. M. (1992). Peer relations in children with a depressed caregiver. In R. D. Parke & G. W. Ladd (Eds), *Family-peer relationships: Modes of linkage {pp*. 317-344). Hillsdale, NJ: Erlbaum.

Zeman, J. & Shipman, K. (1998). Influence of social context on children's affect regulation: A functionalist perspective. *Journal of Nonverbal Behavior, 22*(3), 141-165.

Table 1.

Comparison of subsample included in analyses and larger sample on relevant study variables.

	M	*SD*	*t*	*df*	*P*
Maternal BDI					
Included	7.67	6.73	.33	257	.74
Excluded	7.97	7.52			
15-month AUC_G					
Included	2.19	.26	.46	255	.65
Excluded	2.22	.33			
15-month AUC_I					
Included	3.99	.0047	1.52	82.36	.13
Excluded	4.00	.012			
16-month AUC_G					
Included	2.28	.31	1.42	233	.16
Excluded	2.22	.31			
16-month AUC_I					
Included	3.01	.085	.86	223	.40
Excluded	3.00	.052			
15-month wake time					
Included	7:23	00:59	-.422	269	.67
Excluded	7:19	1:03			
16-month wake time					
Included	7:12	0:45	-1.46	255	.146
Excluded	7:28	0:58			
Infant sex					
Included	1.51	.50	-1.38	246	.17
Excluded	1.43	.50			
Maternal education					
Included	3.85	.90	-1.59	330	.11
Excluded	3.69	.85			
Maternal Income					
Included	2.58	1.49	-.30	237	.76
Excluded	2.50	1.46			
Maternal Smoking					
Included	.047	.16	-.69	252	.52
Excluded	.024	.21			
Baby Age					
Included	15.79	1.73	2.99	286	.003
Excluded	16.28	1.02			

Table 2.

Infant Independent Emotion Regulation Strategies during the Toy Frustration Task.

Emotion Regulation Strategy	Definition
Withdrawal	The infant discontinues attempts to obtain the toy from the box. This behaviour may include the infant sitting or lying down, while no longer engaging with the task.
Wandering away	The infant walks or crawls away from the task.
Distraction	The infant focuses attention away from the task. This behaviour did not include attention focused on the mother or research assistant. This orientation behaviour must be present for a minimum of one second; otherwise this was coded as scanning behaviour.
Orienting to an object	The infant attended to an object, other than the primary toy of interest in the task.
Scanning	The infant is not focused on a specific object or person, but rather is engaging in visual exploration of the environment.

Table 3.

Distribution of maternal BDI-II scores.

	% of sample
None (score 1-10)	76.3
Mild mood disturbance (score 11-16)	14.4
Borderline clinical depression (score 17-20)	3.8
Moderate depression (score 21-30)	3.8
Severe depression (score 31-40)	1.9

Table 4.

Correlations amongst study variables.

	1	2	3	4	5	6	7
1. Maternal BDI							
2. Infant Independent Emotion Regulation	.16*						
3. Toy Frustration AUC_G	.092	.001					
4. Toy Frustration AUC_I	-.045	-.034	-.17*				
5. Breakfast time (T1)	.080	-.037	.146	-.11			
6. Strange situation AUC_G	.069	-.11	.27**	-.038	.009		
7. Strange situation AUC_I	.096	-.076	.027	.081	-.11	.39**	
8. Breakfast time (T2)	.055	.021	.017	-.035	.50**	.017	-.055

* $p < .05$, ** $p < .01$

Table 5.

Multiple regression results predicting 15-month infant cortisol (total output and reactivity).

	Total output (AUC_G)			Reactivity (AUC_I)		
	B	*Std. Error*	*Beta*	*β*	*Std. Error*	*Beta*
Infant breakfast time	.00001	.000	.09			
Maternal BDI	-.007	.005	-.23	.000	.000	-.26
Infant Independent (IR)	-.001	.001	-.24	-.00002	.000	-.20
BDI*IR	.000	.000	.46*	.000003	.000	.42**

** $p \leq .01$

Table 6.

Multiple regression results predicting 16-month infant cortisol (total output and reactivity).

	Total output (AUC_G)			Reactivity (AUC_I)		
	B	*Std. Error*	*Beta*	β	*Std. Error*	*Beta*
Maternal BDI	.002	.009	.043	.000	.002	-.011
Infant Independent (IR)	-.001	.001	-.149	.00001	.000	.003
BDI*IR	.0001	.000	.090	-.00001	.000	-.058

*p< .05, **p<.01

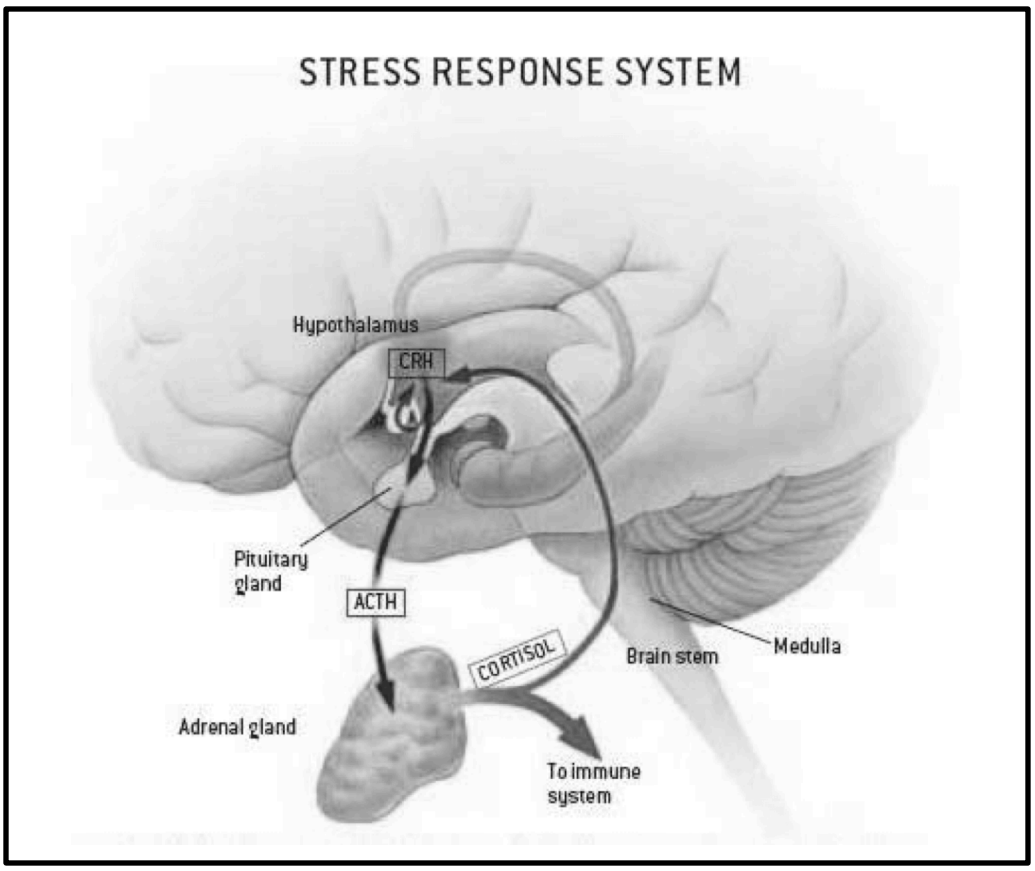

Figure 1. Neurological organization of the HPA system.

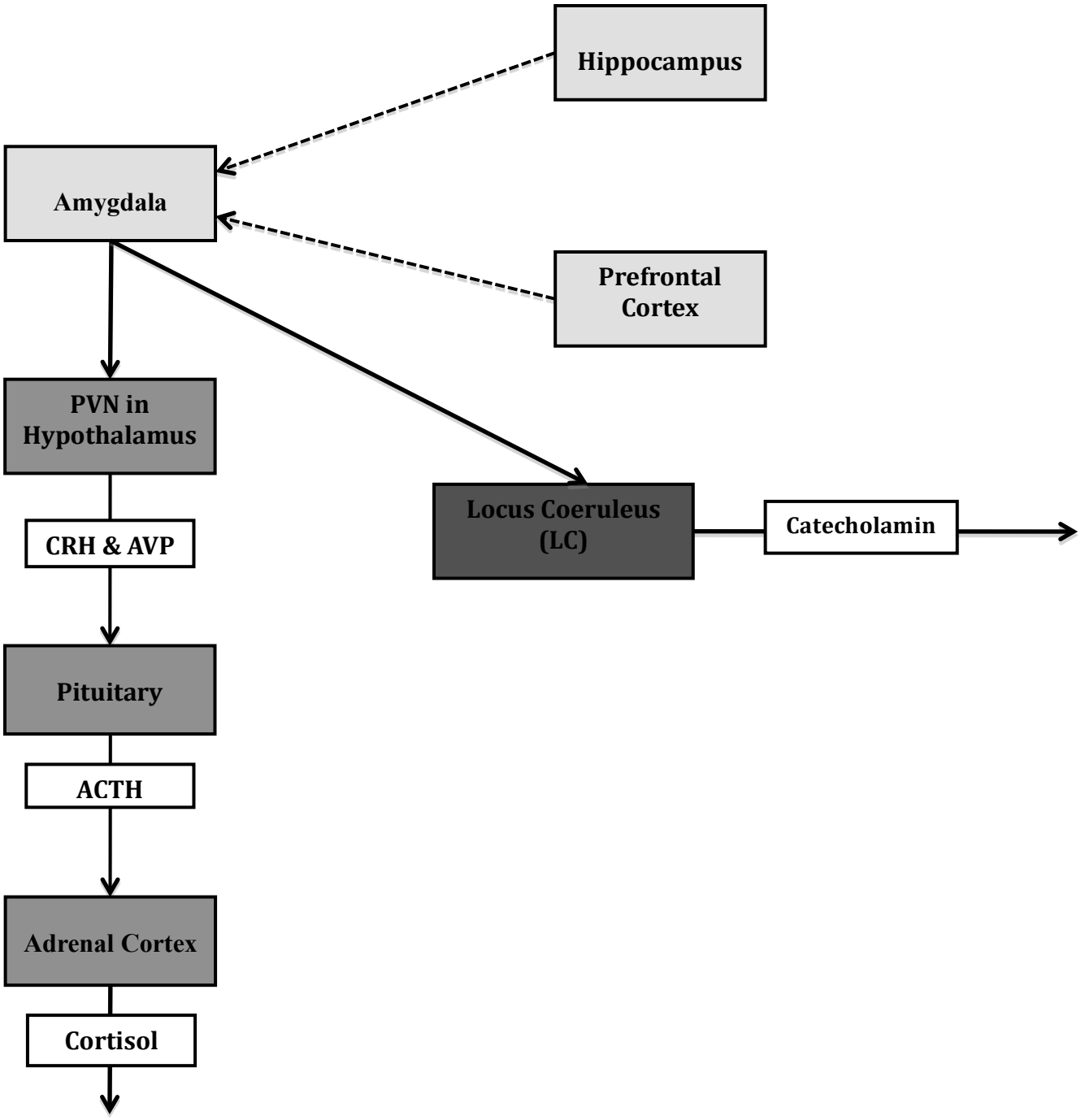

Figure 2. The relation between components of the Central Nervous System (CNS), Sympathetic Nervous System (SNS) and Hypothalamic-Pituitary-Adrenal (HPA) Axis.

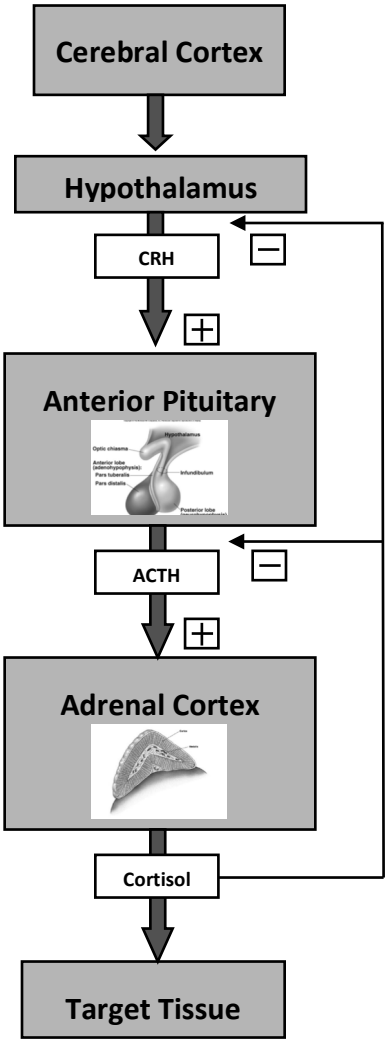

Figure 3. HPA-axis negative feedback loop.

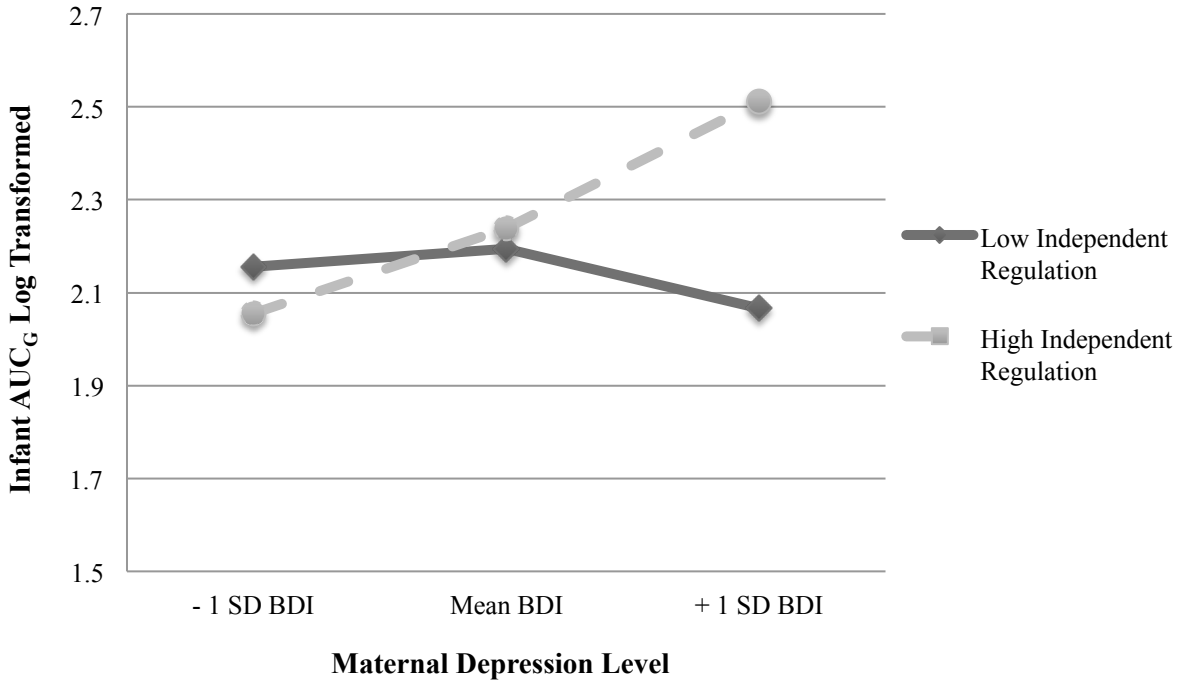

Figure 4. Infant independent emotion regulation strategy moderating the relation between maternal depressive symptoms and infant total cortisol output, indicated by area under the curve with respect to ground (AUC_G; nmol/L), during the toy frustration procedure (at 15-months).

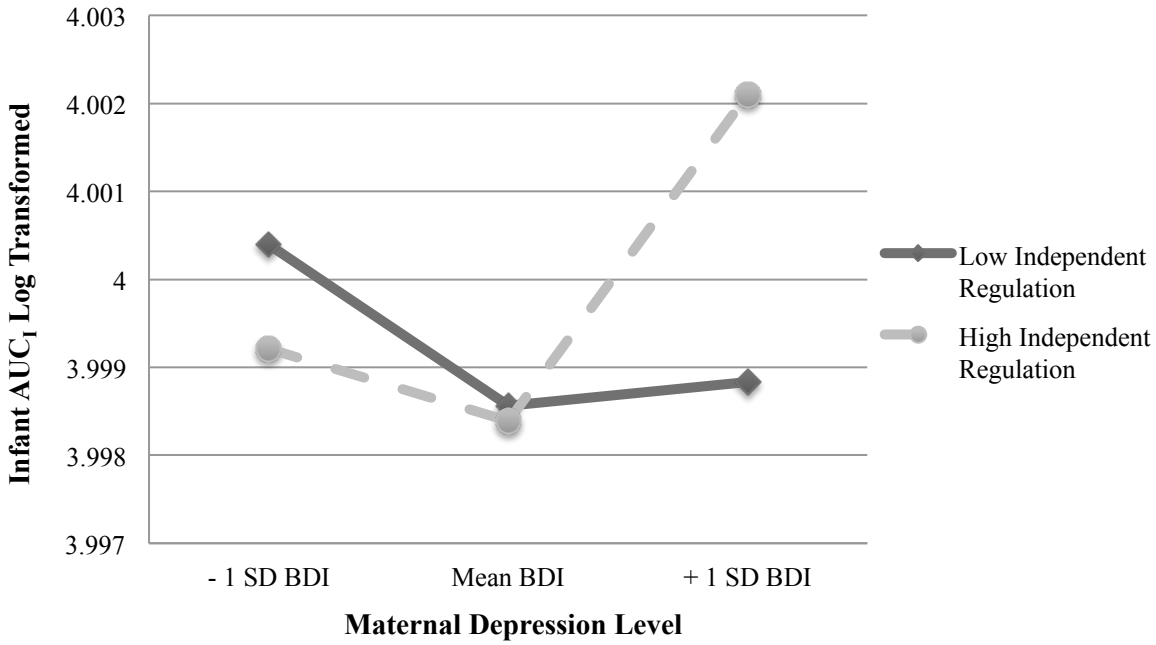

Figure 5. Infant independent emotion regulation strategy moderating the relation between maternal depressive symptoms and infant cortisol reactivity, indicated by area under the curve with respect to increase (AUC_I; nmol/L), during the toy frustration procedure (at 15-months).