

The Pennsylvania State University  
The Graduate School  
Human Development and Family Studies

**PATTERNS OF ADRENOCORTICAL REACTIVITY AND RECOVERY TO  
EMOTIONAL CHALLENGES IN EARLY CHILDHOOD: RELATIONS TO CHILD  
TEMPERAMENT AND PARENTING QUALITY**

A Thesis in  
Human Development and Family Studies

by

Christine K. Fortunato

© 2009 Christine K. Fortunato

Submitted in Partial Fulfillment  
of the Requirements  
for the Degree of  
Master of Science

May 2009

The thesis of Christine K. Fortunato was reviewed and approved\* by the following:

Douglas M. Teti  
Professor of Human Development and Family Studies  
Chair of the Graduate Department (Professor-In-Charge)  
Thesis Co-Advisor

Douglas A. Granger  
Professor of Biobehavioral Health  
Thesis Co-Advisor

Steven H. Zarit  
Professor of Human Development and Family Studies  
Head of Human Development and Family Studies Department

\*Signatures are on file in the Graduate School.

## ABSTRACT

Research suggests that different patterns of hypothalamus-pituitary-adrenal (HPA) reactivity across childhood and adolescence are associated with disparate developmental outcomes (e.g., Gunnar & Donzella, 2002; Granger et al., 1996). This longitudinal study examined how children's temperamental characteristics and maternal parenting qualities were associated with *stable* and *dampening* (i.e., cortisol reactivity to stressors in infancy but not toddlerhood) patterns in children's cortisol reactivity and recovery. The sample (n=547 mother-infant dyads) was ethnically diverse and included predominantly, low-income families from rural communities. Children's saliva was sampled before and after emotion eliciting, challenge tasks. The children's behavioral reactivity was coded and saliva was assayed for cortisol. Results revealed that children who exhibited frustration during infancy and fearfulness during toddlerhood were more likely to show *dampening* cortisol reactivity than no reactivity or hyporeactivity (*consistent NON-reactivity*) across early childhood. Alternatively, children who showed fearfulness during infancy *and* toddlerhood were more likely to demonstrate *consistent* cortisol reactivity than *dampening* reactivity and *consistent NON-reactivity*. Furthermore, infants who had highly engaged mothers had a greater tendency to exhibit *dampening* cortisol reactivity than *consistent* cortisol reactivity and *NON-reactivity*. Finally, children with positively engaging mothers at infancy and toddlerhood were more likely to demonstrate *consistent* HPA reactivity with *immediate* versus *delayed* cortisol recovery. In contrast, toddlers with negatively engaging mothers were more likely to have *delayed* versus *immediate* cortisol recovery. The findings are consistent with the theory of allostatic load (e.g., McEwen, 1998), and suggest that our understanding of how HPA reactivity and recovery influences child development may be advanced by differentiating inter- and intra-individual differences across time.

## TABLE OF CONTENTS

List of Tables.....	v
List of Figures.....	vi
Acknowledgements.....	ix
Introduction.....	1
Conceptual Issues and Empirical Findings .....	5
Temperament and Patterns of Cortisol Reactivity and Recovery.....	12
Quality of Parenting and Patterns of Cortisol Reactivity and Recovery.....	15
Interactions between Temperament and Quality of Parenting.....	19
Present Study: Research Questions and Hypotheses.....	20
Methods.....	22
Participants.....	22
Procedures and Measures.....	24
Analytical Strategy.....	33
Results.....	37
Discussion.....	46
Child Temperamental Characteristics and Patterns of Cortisol Reactivity.....	48
Maternal Parenting Qualities and Patterns of Cortisol Reactivity.....	53
Cortisol Recovery Patterns: Child Temperament and Parenting Qualities.....	55
Interactions with Child Temperament and Parenting Qualities.....	56
Limitations.....	59
Summary and Conclusions.....	60
Appendix: Figures and Tables.....	64
References.....	80

## LIST OF TABLES

Table 1: Cortisol Reactivity Groups: Demographic and Cortisol Characteristics.....	72
Table 2: Correlations of Predictors: Child Temperament and Parenting Quality.....	73
Table 3: Descriptive Statistics of Predictors.....	74
Table 4: Likelihood Ratio Tests of the Infancy and Toddlerhood Temperament Models.....	74
Table 5: Parameter Estimates for the Infancy Temperament Model.....	75
Table 6: Parameter Estimates for the Toddlerhood Temperament Model.....	75
Table 7: Likelihood Ratio Tests of the Parenting Quality in Infancy Model.....	76
Table 8: Parameter Estimates for the Parenting Quality in Infancy Model .....	76
Table 9: Cortisol Recovery Groups: Demographic and Cortisol Characteristics.....	78

## LIST OF FIGURES

Figure 1: Infant Temperament Model.....	64
Figure 2: Toddler Temperament Model.....	65
Figure 3: Parenting Quality of Infancy Model.....	66
Figure 4: Parenting Quality of Toddlerhood Model.....	67
Figure 5: Infant Temperament and Parenting Quality Interaction Model.....	68
Figure 6: Toddler Temperament and Parenting Quality Interaction Model.....	69
Figure 7: Parenting Quality and Infant Cortisol Recovery Model.....	70
Figure 8: Parenting Quality and Toddler Cortisol Recovery Model.....	71
Figure 9: Toddler Temperament and Parenting Quality Interaction.....	77

## ACKNOWLEDGEMENTS

This research was supported by a grant from the National Institute of Child Health and Human Development (PO1-HD-39667), Lynne-Vernon Feagans and Martha Cox, PIs, with co-funding from the National Institute on Drug Abuse. The Family Life Project Key Investigators include Lynne Vernon-Feagans, Martha Cox, Clancy Blair, Peg Burchinal, Linda Burton, Keith Crnic, Nan Crouter, Patricia Garrett-Peters, Douglas Granger, Mark Greenberg, Stephanie Lanza, Adele Miccio, Roger Mills-Koonce, Deborah Skinner, Cynthia Stifter, Lorraine Taylor, Emily Werner, and Mike Willoughby. I would like to thank Drs. Granger and Teti for their helpful suggestions on previous versions of this paper. In addition, I would like to thank my family and friends for all of their support through this process.

## *Introduction*

Among the most compelling research topics in developmental science is how early adversity and individual characteristics affect mental and physical well-being in children. After decades of exclusively studying social and behavioral responses to stress, theorists have increasingly called for a multi-level approach that incorporates biological correlates (e.g., Curtis & Cicchetti, 2003; Gottlieb, 1992). Contemporary conceptual models have focused on individual differences in biological sensitivity to context (Boyce & Ellis, 2005) with the brain as interpreter and responder to environmental challenges, as well as a target of those challenges (e.g., McEwen, 2000). Evidence in support of these general assumptions is accumulating rapidly. For instance, numerous studies demonstrate that individual differences in the regulation of one of the major components of the psychobiology of the stress response—the hypothalamic-pituitary-adrenal (HPA) axis—are associated with early care experiences including maternal sensitivity and negative parenting qualities (e.g., Gunnar & Donzella, 2002; Gunnar & Quevedo, 2008; Kauffman, Plotsky, Nemeroff, & Charney, 2000). To date, however, most studies have employed assessment strategies involving single time point measures of acute stress reactivity or the diurnal rhythm of HPA activity. Although these studies are valuable, they are limited because they fail to capture how patterns of HPA reactivity (i.e., the degree of elevation in cortisol in response to a stressor) and recovery (i.e., the degree of delay in returning to pre-stressor cortisol levels following a stressor) over time may influence developmental outcomes.

Advances in neuroscience reveal glucocorticoids, such as cortisol, secreted in response to the activation of the HPA axis, have protective effects over acute intervals but damaging effects if HPA activation is chronic or prolonged (McEwen, 1998, 2000; McEwen & Stellar, 1993). The process of adaptation to an acute stressor involves the appropriate release of stress hormones to



re-establish stability (i.e., homeostasis), followed by a deactivation of the HPA axis once the stressful situation has ceased. This process is referred to as allostasis (Sterling & Eyer, 1988). During allostasis, the major function of cortisol is to convert proteins and lipids into usable carbohydrates, allowing energy reserves to become readily available for the organisms use in the face of the current challenge. In addition, cortisol facilitates tissue repair, enhances memory formation, and regulates blood pressure (McEwen & Seeman, 1999; Thompson, 2000). Thus, cortisol has protective effects for an organism in the short term; however, researchers hypothesize that patterns of insufficient self-regulation of the HPA-axis (i.e., over- or under-activation) may lead to negative mental and/or physical outcomes (McEwen, 1998, 2000; McEwen & Stellar, 1993).

The over- or under-activation of the adrenocortical response system is called “allostatic load” (McEwen & Stellar, 1993). Allostatic load is the wear-and-tear on an organism’s physiology due to repeated physiological adaptation to adverse psychosocial or physical challenges. McEwen (1998) has proposed different types of allostatic load that represent exposure to an excessive amount of stress or the dysregulation of the adrenocortical response system. The stable patterns of allostatic load for the HPA system consist of 1) over-exposure to cortisol as a result of “appropriately” activating and deactivating the adrenocortical response system to multiple, novel events (i.e., chronic stress); 2) over-exposure to cortisol due to a failure to habituate to similar, repeated stressors; 3) over-exposure to cortisol due to a failure to turn off the stress response system “appropriately;” and 4) failure to produce an adequate adrenocortical response that meets the needs of an individual’s genotype (i.e., under-activation), which may result in excessive activities of other systems (e.g., immune system). The deleterious outcomes that have been associated with the various types of HPA dysregulation include higher levels of

internalizing behavior problems (e.g., Granger, Weisz, & Kauneckis, 1994; Granger, Weisz, McCracken, Ikeda, & Douglas, 1996), immune system suppression (e.g., Cacioppo et al., 1998), and increase risk for upper respiratory infections in children and adolescents (e.g., Cohen et al., 2002), along with chronic diseases including hypertension and diabetes in adults (Boomershine, Wang, & Zwilling, 2001; McEwen, 1998; Whitworth, Brown, Kelly, & Williamson, 1995).

Recently, researchers have discovered another pattern of cortisol reactivity that may occur across early childhood. In particular, studies have demonstrated that infants (on average) exhibit a gradual dampening in cortisol reactivity over the first 12 months of life to mild stressors such as inoculations (Gunnar, Brodersen, Krueger, & Rigatuso, 1996; Ramsay & Lewis, 1994), mock physical exams (Larson, White, Cochran, Donzella, & Gunnar, 1998), and separation from parents (Gunnar & Nelson, 1994; see review Gunnar and Donzella, 2002). A hypothesis has formed suggesting that this phenomenon in humans is analogous to the “normative” stress hyporesponsive period found in rodents (Rosenfield, Suchecki, & Levine, 1992; Sapolsky & Meaney, 1986). To expound, rat pups demonstrate vigorous cortisone reactivity to stressors (mild to severe) 0 to 4 days after birth, but it became almost impossible to elicit a cortisol response in rat pups from approximately 4 to 14 days after birth. Thus, researchers have speculated that the diminution of HPA reactivity found in the aforementioned studies on human infants may be a result of physiological changes (e.g., improved negative feedback regulation; Lashansky et al., 1991), as well as supportive caregivers acting as external regulators to buffer against HPA activation (see review, Gunnar & Quevedo, 2007).

Understanding the patterns in adrenocortical stress reactivity and recovery over time—both the *stable* patterns associated with allostatic load as well as the potential *dampening* patterns across infancy—appears central to distinguishing whether “dysregulation” of adrenocortical

mediators of stress confer risk or resilience in individuals. To comprehensively investigate these adrenocortical stress patterns, they must be viewed as physiological processes that are embedded in a complex, person-environment system (Cairns, Elder & Costello, 1996; Magnusson & Cairns, 1996; Bronfenbrenner & Morris, 1998), wherein development is bidirectional and interactions with the early environment have implications for later development (Goldhaber, 2000). Thus, to add to the emerging literature on the associations with patterns of cortisol reactivity and recovery in early childhood, this longitudinal study prospectively investigates the proximal (e.g., temperamental characteristics) and more distal (e.g., positive and negative parenting qualities) correlates of these *stable* and *dampening* cortisol reactivity and recovery patterns that may eventually result in negative physical and mental health outcomes.

This study utilizes a person-oriented approach, whereby children are divided into groups according to the *stable* or *dampening* cortisol reactivity and recovery patterns they exhibited during developmentally-appropriate, mild stressors at 7- and 15-months of age. In this study, developmentally-appropriate stressors were operationally defined as a series of challenges that were likely to elicit frustration (i.e., distress to limitations) and fear (i.e., distress to novelty) in 7- and 15-month-olds. Since the onset of fear to environmental stressors occurs, on average, between 7- and 9-months of age (e.g., Scarr & Salapatek, 1970), the current study anticipates that the display of frustration to the challenge tasks would be greater than that of fear in the 7-month-olds. However, the differences in the amount of fear versus frustration that would be exhibited by the 15-month-olds were unclear due to increased individual differences in behavioral distress during toddlerhood.

The cortisol reactivity groups included the 1) *consistent cortisol reactors*—children who exhibited cortisol reactivity at both 7- and 15-months-old, 2) *consistent cortisol NON-reactors*

group—children who did *not* demonstrate cortisol reactivity or exhibited a decrease in cortisol levels in response to the task at both 7- and 15-months-old; and 3) *dampening cortisol reactors*—children who exhibited cortisol reactivity at 7-months-old but *not* at 15-months of age. These groups were then examined for differences in temperamental characteristics of fear and frustration as well as quality of maternal parenting. Subsequently, groups that included cortisol reactivity, *consistent cortisol reactors* and *dampening cortisol reactors*, were subdivided into children who demonstrated cortisol recovery (i.e., *immediate* cortisol recoverers) and who did *not* exhibit cortisol recovery (i.e., *delayed* cortisol recoverers). Differences in temperamental characteristics and maternal behaviors between the recovery subgroups are examined.

#### *Conceptual Issues and Empirical Findings on Patterns of Cortisol Reactivity and Recovery*

Gottlieb's (1992) thoughts on individual development and the evolution of novel behavior suggest that certain physiological processes are activated only in conditions when or where components of the "behavioral surface" are unable to accommodate the challenge. By the behavioral surface, he implies that the first line of "defense" and most flexible and fluid mechanisms available to an individual to adjust to dynamic change in the environment involve coping resources, change in behavior, restructuring subjective experience held of the event, or all of the above. Only if these components of an individual's behavioral surface cannot "handle" the challenge they are faced with, then the fast acting components of the psychobiology of stress be activated. There are specific circumstances that this psychobiological sequence of stress activation is likely. In terms of the HPA axis, for example, the literature defines those events as characterized by novelty, pain, threat, and social evaluation. As mentioned above, this HPA activation to environmental stressors has strong evolutionary significance for survival because it

facilitates the availability of energy storage and physiological mechanisms required to re-establish homeostasis (McEwen & Seeman, 1999; Thompson, 2000).

If HPA activation promotes survival, what are the possible reasons that individual's may exhibit the various patterns of adrenocortical activation associated with allostatic load? First, if the individual was exposed to multiple, novel challenges (i.e., chronic stressors) that were dissimilar in nature, then the individual would have difficulty adapting to all the environmental challenges and form a pattern of recurring HPA reactivity and recovery that may lead to over-exposure of cortisol to the bodily systems (McEwen, 1998). Additionally, the activation of the physiological subsystems, such as the HPA axis, theoretically adjusts over repeated encounters (i.e., habituation) with similar situational stressors or set of circumstances. However, this diminution of HPA activation may not occur if there is a lack of exposure to environmental factors (e.g., temperamental characteristics and/or maternal behaviors) that facilitates the individual's behavioral surface to acquire the skills, resources, and knowledge to accommodate the threat, which may result in an over-exposure to cortisol and lead increased allostatic load (Gottlieb, 1992; McEwen, 1998; McEwen & Stellar, 1993). Similarly, HPA dysregulation due to prolonged cortisol activation—delayed cortisol recovery—may be associated with increased allostatic load as a result of a lack of skills, resources, and knowledge obtained by the behavioral surface to deal with the challenge. Finally, a consistent reduction of cortisol reactivity to stressors is referred to as hypocortisolism (Heim, Ehlert, & Hellhammer, 2000). Researchers have postulated that both trauma and prolonged, non-traumatic, chronic histories of exposure to stressors initially result in hyperactivity of the HPA axis that eventually causes the system to down-regulate over the course of time, ultimately producing blunted cortisol reactivity to challenges (Gold & Chrousos, 2002; Hellhammer & Wade, 1993; Susman, 2006). Patterns of

cortisol hyporeactivity to stress has been associated with asthma and chronic fatigue syndrome (Heim et al., 2000), as well as higher rates of externalizing behaviors in childhood and adolescence (Shirtcliff, Zahn-Waxler, & Klimes Dougan, 2005; van Goozen, Matthys, Cohen-Kettenis, Gispen-de Wied, Wiegant, & van Engeland, 1998).

The behavioral and environmental correlates of these *stable* patterns of cortisol reactivity and recovery to stressors have yet to be explored during childhood. However, emerging evidence suggests that examining these stable patterns may be worthwhile. For example, Granger, Stansbury, and Henker (1994) studied preschool-aged children with emerging behavior problems in small group activities that required them to adapt to unfamiliar teachers, peers, and social events. Saliva was collected before and after an activity session and again 2 weeks later in the same circumstance. Larger decreases in cortisol during the first session were associated with concurrent and subsequent undercontrolled, social behaviors. In contrast, overcontrolled behaviors and negative affect during the first *and* later activity sessions were associated with larger increases in cortisol during the later session. Moreover, Granger and colleagues (1996) studied adolescents and reported that consistent adrenocortical reactivity to parent-child conflict at intake *and* 6-months later predicted clinic-referred children's internalizing problem behaviors and anxiety disorders at follow-up. These studies highlight the importance of focusing on patterns within individuals over time when considering whether individual differences in stress-reactivity may lead to positive or deleterious outcomes during early childhood and later during adolescence.

Although there is a dearth of empirical studies that investigate how *stable* patterns of physiological stress-reactivity moderate and/or mediate links between exposure to contextual stressors in early childhood and subsequent outcomes, developmental scientists have begun to

formulate theoretical constructs in an attempt to define these underlying processes (e.g., Boyce & Ellis, 2005; Krantz & Manuck, 1984; Sroufe, 1979). For example, Boyce and Ellis (2005) have proposed an evolutionary-developmental perspective that emphasizes biological sensitivity to context (BSC), postulating that stable individual differences in stress reactivity among biological response systems develop over the course of early development due to both genetic and environmental factors. That is, there may be an adaptive patterning of phenotypic differences in biological stress reactivity that arises based on exposure to certain environmental factors in early childhood.

Specifically, Boyce and Ellis (2005) have hypothesized that early childhood experiences in either extremely supportive or severely stressful environments give rise to heightened reactivity in one or more of the primary response systems, such as HPA axis and locus coeruleus-norepinephrine (LC-NE). Due to the interactions between high reactivity phenotypes within each stress response system and different contexts, they suggest that bivalent effects may occur that are both harmful and protective. Specifically, children with highly reactive phenotypes in supportive and stable contexts will have lowered rates of physical and mental disorders; whereas, children that are highly reactive phenotypes in unsupportive and highly stressful environments will incur increased rates of deleterious physical and mental health outcomes. Moreover, Boyce and Ellis have proposed that stable, low levels of reactivity in children will be more prevalent in contexts that chronically include moderate (“normative”) levels of recurring, broad spectrum stressors. This low reactivity phenotype is believed to result from natural selection, and acts as a protective factor in children by allowing greater resilience under a large range of familial and ecological contexts.

An additional layer of complexity that has yet to be explicitly considered in theoretical frameworks addressing the associations between the adrenocortical stress system and context is that the brain is highly plastic throughout early childhood (e.g., Black, Jones, Nelson, & Greenough, 1998; Greenborough & Black, 1992; Huttenlocher, 2002). Animal studies have demonstrated that the HPA axis is not fully developed at birth and have revealed that the developing threshold of reactivity early social and environmental forces (e.g., Meaney et al., 1996). This suggests that human HPA functioning could change throughout early development, creating a pattern of cortisol reactivity that does not stabilize until later periods of children's development. Studies on the "normative" trajectories of cortisol reactivity from infancy to toddlerhood reveal that children develop an overall dampening of cortisol reactivity by the age of two years (Gunnar et al., 1996; Larson et al., 1998; Ramsay & Lewis, 1994). To elaborate, toddlers may demonstrate high levels of behavioral reactivity (e.g., crying, gaze aversion) to acute stressors as they did in infancy; yet, there is little to no HPA reactivity to that same acute stressor in toddlerhood. Unfortunately, with the exception of a few empirical studies using mock physical exams (Larson et al., 1998) and separation from parents (Gunnar & Nelson, 1994), the majority of these findings on patterns of adrenocortical reactivity in early development have been based on studies in which the same, pain-eliciting stressors (i.e., inoculations) was performed over the first 12 to 15 months of life. As mentioned previously, the present study hypothesizes that from infancy to toddlerhood a substantial portion of children will exhibit an inconsistent pattern of adrenocortical reactivity to psychosocial stress tasks, whereby the individual may demonstrate increased cortisol levels to a developmentally appropriate, acute stressor during infancy but not in later toddlerhood (see reviews by Gunnar & Donzella, 2002; Gunnar & Vazquez, 2007). Since this dampening pattern is considered normative, researchers



have speculated that this adrenocortical dampening would most likely occur in supportive environments such as familial contexts characterized by parental responsiveness and sensitivity.

Although such theories give researchers a framework for testing the process wherein stable patterns of reactivity may result in prolonged exposure to glucocorticoids and act as moderators and/or mediators to developmental outcomes, they overlook the potentially important role that recovery (or lack thereof) from HPA activation may play. For instance, a rapid and intense HPA response in conjunction with quick recovery may be adaptive in situations where an individual requires energy to diminish the goal threat (Linden, Earle, Gerin, & Christenfeld, 1997; Sapolsky, Ramero, & Munck, 2000). In contrast, a slow return to baseline levels post-stressor may result in increased exposure to glucocorticoids that have deleterious effects on mental and physical health, and is a potential indicator of underlying dysfunction in physiological stress response (McEwen, 2000). In fact, some researchers have speculated that examining patterns of HPA recovery post-stressors may actually be a better indicator than HPA reactivity for outcomes such as physical health (e.g., Burelson et al., 2003; Cohen & Hamrick, 2003; Dienstbier, 1989; Linden et al., 1997), whereby the likelihood of pathology is greater when the rate of recovery is slow (Sapolsky et al., 2000).

Furthermore, a recent meta-analytic study by Dickerson and Kemeny (2004) suggested that elucidating the factors associated with the recovery process could be a fruitful avenue of inquiry. Specifically, they discovered that uncontrollable, social-evaluative conditions influenced the recovery process, or the degree to which cortisol elevations persisted up to one hour after stressor termination (e.g., Linden et al., 1997). However, there remains a dearth of studies that examined how different contexts or individual differences predict cortisol recovery (cf. Dickerson and Kemeny, 2004; Earle, Linden, & Weinberg, 1999; Roy, Kirschbaum, & Steptoe,

2001). Therefore, in conjunction with investigating stress-reactivity throughout early childhood, this study examines how cortisol recovery from developmentally-appropriate stressors is related to individual and contextual correlates during early childhood.

In terms of correlates of cortisol reactivity and regulatory patterns, existing theoretical frameworks highlight the association of biological reactivity, childhood temperament, and parental behavior. However, no biological sensitivity theories could be found, to date, that speculate whether there is a specific relationship among temperamental characteristics, parental behavior, and cortisol recovery to stressors. In terms of biological reactivity, the BSC (Boyce & Ellis, 2005) is limited because it only includes postulations on the temperamental dimension of shyness and behavioral inhibition (i.e., distress to novel challenges as well as latency, or lack of approach, to novel objects and persons in social situations; Buss et al., 2003; Kagan, Snidman, & Arcus, 1998; Putnam & Stifter, 2005). Based on prior personality research (e.g., Bullock & Gilliland, 1993), Boyce and Ellis assume that temperamental characteristics of shyness or behavioral inhibition are associated with greater biological reactivity to moderate stressors, and therefore, are also a reflection of sensitivity to context. From this assumption, they went on to speculate that shy or behaviorally inhibited children exposed to harsh, insensitive, or inconsistent parenting may be more likely to develop high anxiety and neurotic personality dimensions; whereas, shy or behaviorally inhibited children that experience environments characterized by sensitive and responsive parenting may be more likely to develop low anxiety and emotionally stable personality dimensions. Unfortunately, the assumption that shyness and behavioral inhibition are always associated with greater biological reactivity is not always true. Many inconsistencies have been found in the literature regarding the association between higher order fearfulness such as shyness/behavioral inhibition and biological reactivity especially in relation

to adrenocortical reactivity (e.g., Gunnar, Larson, Hertzgaard, Harris, & Brodersen, 1992; Nachmias, Gunnar, Mangelsdorf, Parrity, & Buss, 1996). Hence, this study explores whether temperamental characteristics such as frustration and fear are correlated with consistent patterns of adrenocortical reactivity and recovery to challenge tasks in infancy and toddlerhood. Moreover, this study investigates how maternal behaviors (i.e., positive and negative parenting qualities), as well as the interaction between these maternal parenting qualities and children's temperamental characteristics, relate to various patterns of adrenocortical reactivity and recovery.

Increased knowledge of HPA reactivity and recovery to developmentally appropriate challenge tasks is warranted to determine the process(es) that contributes to increased resilience and risk in relation to diverse outcomes. As a potential first step, this prospective study aims to ascertain a more comprehensive understanding of the individual and environmental correlates of different patterns of cortisol reactivity and recovery to psychosocial stressors throughout early childhood in a low-income, rural population.

#### *Temperamental Characteristics and Patterns of Cortisol Reactivity and Recovery*

Among the individual characteristics of particular interest to developmental researchers is temperament, defined as constitutionally based individual differences in behavioral, response tendencies (Goldsmith et al., 1987). Considerable evidence indicates that individual differences in temperament are associated with disparities in brain and peripheral physiological functioning during early childhood development (e.g., Kagan, Reznick, & Snidman, 1988; Kirschbaum & Hellhammer, 1989, 1994). Furthermore, many researchers have suggested that physiological measures such as cortisol reactivity and recovery may be needed to identify the most extreme group of children that are likely to manifest into internalizing and externalizing disorders (e.g.,

Davidson, 2001; Kagan, Snidman, McManis, Woodward, & Hardway, 2002). Therefore, this study focuses on two characteristics of temperament that have demonstrated associations with cortisol reactivity during infancy in previous empirical research—frustration/distress to limitations and fear/distress to novelty.

Regarding the temperamental characteristic of frustration/distress to limitations, there is limited evidence as to its relation to cortisol reactivity during early childhood. Specifically, at 9-months of age, distress to limitations predicted cortisol reactivity to infant-parent separation (Gunnar et al., 1992). Additionally, in toddlers and pre-schoolers, anger proneness was positively related to cortisol reactivity (Davis, Donzella, Krueger, & Gunnar, 1999; Donzella, Gunnar, Krueger, & Alwin, 2000; Tout, de Haan, Kipp-Campbell, & Gunnar, 1998; van Bakel & Riksen-Walraven, 2004).

The majority of temperament research that investigated associations with physiological reactivity has focused on the construct of behavioral inhibition, defined as distress to novelty and a lack of approach to novel objects and persons in social situations (e.g., Buss et al., 2003; Kagan et al., 1987; Schmidt et al., 1997). Since behavioral inhibition encompasses multiple dimensions, it is sometimes difficult to discern which aspects of this temperamental construct—fear/distress to novelty or the latency/lack of approach to novelty—are associated to HPA reactivity. The few studies that primarily focused on fear/distress to novelty alone have revealed that children who possess an extreme temperamental characteristic of fearfulness to stressors such as inoculations, infant-parent separation, and encounters with novel stress are associated with increased cortisol levels in both infancy (Nachmias et al., 1996; Spangeler and Scheiche, 1998) and late childhood (Kagan et al., 1987; Schmidt et al., 1997).

Yet, the empirical evidence regarding the association between HPA reactivity and behavioral reactivity are inconsistent. For instance, during exposures to similar stressors, fear/distress to novelty was correlated with lower versus higher cortisol reactivity in infants (Gunnar et al., 1992) and school-aged children (de Haan, Gunnar, Tout, de Hart, & Stansbury, 1998; Gunnar, Tout, de Haan, Pierce, & Stansbury, 1997). These inconsistencies suggest that behavioral reactivity may not always correspond directly with physiological reactivity (Gottlieb, Johnson, & Scoville, 1982). This “loose coupling” between physiological and behavioral reactivity to stressors could allow for greater ability to adapt to diverse context (e.g., Quas, Hong, Alkon, & Boyce, 2000). For instance, habituation to similar stressors or a set of circumstances may result in a lack of HPA activation to an acute stressor in individuals, even though they are exhibiting behavioral distress in response to the stressor (e.g., Gunnar et al., 1996).

Despite the inconsistencies in findings, or “loose coupling,” between adrenocortical and behavioral reactivity, this study hypothesizes that behavioral reactivity will be associated primarily with adrenocortical reactivity. Thus, *consistent* and *dampening* cortisol reactors are expected to be associated with higher levels of frustration during the arm restraint task than *consistent NON*-reactors when children were infants, 7-months of age. During toddlerhood, children who exhibit greater frustration during the toy removal should be more likely to be *consistent* cortisol reactors than *dampening* cortisol reactors and *consistent NON*-reactors. Moreover, considering that most studies determined that cortisol reactivity was associated with fearfulness/distress to novelty, this study expects that children who demonstrated greater behavioral fearfulness to the mask task would have be more likely to be *consistent* cortisol reactors than *dampening* cortisol reactors and *consistent NON*-reactors. However, since the

average age of onset for fear is 7- to 9-months of age (e.g., Scarr & Salapatek, 1970), the study anticipates that these relationships would only hold during toddlerhood, at the 15-month assessment.

With regard to temperamental characteristics of frustration/distress to limitation and fear/distress to novelty, few studies to date have investigated their relationship to cortisol recovery. Recently, Keenan, Grace, and Gunthorpe (2003) did not find a significant relationship between behavioral distressed (average-high versus low) neonates to a heel stick and cortisol recovery. Moreover, Zimmerman and Stansbury (2004) discovered that pre-schoolers with immediate and prolonged cortisol recovery were associated with temperaments of behavioral inhibition and boldness to moderate stressors. Based on this research, this present study did not hypothesize a difference in the levels of fear and frustration between *immediate* and *delayed* cortisol recoverers during infancy and toddlerhood. Therefore, no analyses regarding this relationship were performed.

#### *Quality of Parenting in Relation to Patterns of Cortisol Reactivity and Recovery*

During early infancy and toddlerhood, children have limited abilities to self-regulate emotionally and physiologically. As a means of buffering children's emotional and HPA reactivity to stressors, caregivers may provide additional external regulation by sensitively responding to the needs of the child (Bowlby, 1969; Tronick, 1989; Repetti, Taylor, & Seeman, 2002). Theoretically, positive parenting qualities, such as sensitive and responsive caregiving, allow children to express emotional distress and elicit help, without triggering increased cortisol (e.g., Gunnar & Donzella, 2002; Repetti et al., 2002). Alternatively, if the caregiver demonstrates negative parenting qualities towards the child (i.e., inadequately responding to the child's needs and/or overwhelming the child with excessive stimulation), the parent-child

relationship may become an additional source of physiological stress. Based on these hypotheses, this study focuses on how children's exposure to positive and negative parenting qualities influences patterns of cortisol reactivity and recovery across early childhood.

Studies of animal models have demonstrated that maternal responsiveness and sensitivity, defined as maternal licking and grooming, during early childhood acts as a buffer against high levels of cortisol reactivity to stressors (Bayart, Havashi, Faull, Barchas, & Seymour, 1990; Levine & Weiner, 1988; Liu, 1997). In humans, researchers have hypothesized that secure attachment behaviors indicate a history of positive parenting qualities, including sensitive and responsive care, by the attachment figure (e.g., Ainsworth et al., 1978; Bowlby, 1969; Sroufe, 1983). Existing research on attachment quality has demonstrated that, when the attachment figure is present, securely-attached children elicit little to no response to stressors such as infant-parent separation and inoculations; whereas, children who are insecurely-attached demonstrate substantial elevation in cortisol (Gunnar et al., 1996; Nachmias et al., 1996; Spangler & Grossmann, 1993; Spangler & Schieche, 1998). Additionally, as previously mentioned, some researchers speculate that this adrenocortical dampening starting in the second year of life is associated with exposure to responsive and sensitive parenting (e.g., Gunnar & Donzella, 2002; Gunnar & Vazquez, 2007). Conversely, promising findings from the current study's sample revealed that maternal engagement was positively associated with cortisol reactivity to the challenge task during infancy (Blair et al., 2008). Moreover, children with engaged mothers during toddlerhood exhibited lower overall levels of cortisol during the stressor, but no differences in cortisol reactivity. Thus, the present study hypothesizes that children exposed to positive parenting qualities (e.g., maternal sensitivity, positive engagement) during the parent-

child interaction would be more likely to be *dampening* cortisol reactors than *consistent* cortisol reactors and *consistent NON*-reactors.

In contrast to positive parenting qualities, negative parenting qualities such as insensitivity (detachment or intrusiveness) may have a negative impact on the adrenocortical functioning in early childhood. The negative parenting quality of insensitivity can be defined as maternal behaviors that do not meet their children's needs and/or interrupt their children's activities (e.g., Ainsworth et al., 1978). Two major aspects of insensitive parenting include detachment and intrusiveness. Maternal detachment can be operationalized as a mothers' unresponsive or disengaged behaviors towards their children's needs and signals (e.g., Ainsworth et al., 1978; Isabella & Belsky, 1991); while, maternal intrusiveness can be defined as mothers' excessive verbal or physical stimulation or interruption meant to stop or change to course of their children's self-initiated activity (e.g., Carlson & Harwood, 2003; Egeland, Pianta, & O'Brien, 1993). Unfortunately, the empirical studies that specifically assess cortisol reactivity in relation to maternal insensitivity are very limited. In fact, after an extensive literature search, only one empirical study that focused on this topic was discovered. The study, by Spangler and colleagues (1994), demonstrated that insensitive parenting, including aspects of detachment and intrusiveness, was related to heightened cortisol reactivity in 3- and 6-month-old infants during free-play at home and in lab playroom. However, on average, when the infants were 9 months of age, cortisol reactivity was not significantly correlated with maternal intrusiveness and insensitivity. Overall, researchers speculate that insensitive parenting in the form of detachment deprives children of external regulation; whereas, intrusive parenting may constitute an additional source of stress for children (Adam, Kimes-Dougan, & Gunnar, 2007; Gunnar &



Donzella, 2002; Gunnar & Vazquez, 2007; Schore, 2001; Smeekens, Riksen-Walraven, & van Bakel, 2007; Teicher et al., 2003).

The reviewed literature suggests that negative parenting qualities such as detachment and intrusiveness may be associated with dysfunctional HPA activation to stressors. Therefore, this study expects that children exposed to greater maternal qualities of negative parenting (detachment and intrusiveness) would be more likely to exhibit stable patterns of cortisol reactivity that may be related to allostatic load (*consistent* cortisol reactors and *consistent NON*-reactors) than the *dampening* pattern of cortisol reactivity at 7- and 15-months. Furthermore, to determine whether qualities of negative as well as positive parenting at 7- and 15-months are related differentially to the categories of cortisol reactors (*consistent* cortisol reactors, *consistent NON*-reactors, and *dampening* cortisol reactors), children would display across early childhood, maternal parenting qualities at 7-months and 15-months will be tested separately.

Regarding associations between cortisol recovery and parenting quality, research is still in the nascent stage. Although many researchers have touted the importance of including cortisol recovery measures in empirical studies (e.g., Ramsay & Lewis, 2003; de Weerth & Buitelaar, 2005), there is a large dearth of investigations on HPA activity to stressors in early childhood that have incorporated such measures. Moreover, to date, there is only one study that has explicitly found that parenting quality is related to cortisol recovery during early childhood. The study revealed that infants exposed to higher quality, positive maternal caregiving were associated with more immediate recovery in cortisol levels after stress exposure than infants whose mothers elicited lower quality care (Albers, Riksen-Walraven, Sweep, & de Weerth, 2008). In other words, infants whose mothers were concurrently less sensitive and more intrusive sustained higher cortisol levels for longer periods of time after the mildly stressful event had ceased than

infants whose mothers were more sensitive and less intrusive. Therefore, in accordance with these findings and the theory that sensitive mothers act as an external regulator for children (e.g., Bowlby, 1969; Ruff & Rothbart, 1996; Posner & Rothbart, 2000), this study hypothesizes children whose mothers exhibited more positive parenting qualities (such as high levels sensitivity and responsivity) would have an increased likelihood of being *immediate* cortisol recoverers versus *delayed* cortisol recoverers in both the *consistent* and *dampening* cortisol reactor groups at 7- and 15-months of age. Additionally, this study anticipates that mothers of *delayed* cortisol recoverers would exhibit more negative parenting qualities than mothers of *immediate* cortisol recoverers.

#### *Interactions between Temperament and Maternal Behavior: Influence on Cortisol Patterns*

As noted earlier, developmental scientists have increasingly underscored the central role that reciprocal interactions between the individual characteristics of a person and factors in their environment play in influencing developmental trajectories (e.g., Belsky, 1984; Bronfenbrenner & Morris, 1997; Maccoby & Martin, 1983). Extending this logic to patterns of cortisol reactivity, researchers hypothesize that the dynamic interplay between children's temperament characteristics (e.g., fearfulness, frustration) and influences in the environmental factors such as maternal behaviors may shape adrenocortical stress responses, which affects subsequent developmental outcomes (e.g., Dettling et al., 2000; Gunnar & Donzella, 2002).

In particular, Gunnar and Donzella (2002) speculated that children with fearful or angry temperaments will be more likely to exhibit increased cortisol reactivity to stressors as the quality of care decreases. Alternatively, children with fearful or angry temperaments will be less likely to exhibit elevations in cortisol in response to a stressor as caregiving quality increases. Empirical support of this concept revealed that mothers' intrusive behaviors during an interactive

play were associated elevated cortisol reactivity, but only in 18-month olds who were high on behavioral inhibition (Nachmias et al., 1996). Therefore, these researchers hypothesized that intrusive maternal behaviors may have blocked behaviorally inhibited children's coping mechanisms during the stress task, resulting in increased cortisol reactivity. In contrast, children high on behavioral inhibition did not show increases in cortisol reactivity in the presence of a sensitive and responsive caregiver. Other findings have revealed that infants left with caregivers who were characterized as cold and distant exhibit elevated cortisol levels as temperamental characteristics of anger or fear increased (Gunnar, 1990; Spangler and Schieche, 1994). Based on this evidence, the present study predicts that children who are characterized with high fearfulness will be more likely to be *consistent* cortisol reactors, versus *consistent NON*-reactors and *dampening* cortisol reactors, as maternal intrusive behaviors increases at 7- and 15-months of age. In addition, this study expects that fearful children will be more likely to elicit the "normative," *dampening* pattern of cortisol reactivity, versus *consistent* cortisol reactivity and *NON*-reactivity, as maternal sensitivity increase at 7- and 15-months of age. Since children become increasingly able to regulate their emotions via attentional and inhibitory control systems that develop as part of ongoing neuropsychological maturation (Posner & Rothbart, 2000), the study predicts that the ability for interactions between temperament and maternal behaviors may be stronger at 7- versus 15-months of age.

### *Present Study*

To review, the current investigation explores the following hypotheses and questions using a sample of mother-infant dyads living in predominately low-income, non-metropolitan counties assessed when infants were 7- and 15-months old:

1. *Do temperamental characteristics of frustration or fear predict patterns of cortisol reactivity and recovery during infancy and toddlerhood?*
  - 1.1. During infancy, *consistent* and *dampening* cortisol reactors would be associated with higher levels of frustration/distress to limitation during the arm restraint task than *consistent NON-reactors* (see Figure 1). In addition, *consistent cortisol reactors* would be associated with greater fearfulness/distress to novelty during the mask task than *consistent NON-reactors* only.
  - 1.2. During toddlerhood, the current study expects that children who exhibited greater frustration during the toy removal would be more likely to be *consistent* cortisol reactors than *dampening* cortisol reactors and *consistent NON-reactors* (see Figure 2). Moreover, *consistent* cortisol reactors would reveal greater fearfulness/distress to novelty than *dampening* cortisol reactors and *consistent NON-reactors*.
2. *Does maternal quality of parenting predict patterns of cortisol reactivity and recovery during infancy and toddlerhood?*
  - 2.1. Children whose mothers demonstrated higher positive emotional engagement during the parent-child interactions would be more likely to be *dampening* cortisol reactors than *consistent* cortisol reactors and *consistent NON-reactors*. Moreover, children exposed to greater levels of negative maternal engagement would be more likely to be *consistent* cortisol reactors and *consistent NON-reactors* than *dampening* cortisol reactors when children are 7- and 15-months of age (see Figures 3 and 4).
  - 2.2. Children exposed more positive maternal engagement during the parent-child interaction would be more likely to be *immediate* cortisol recoverers than *delayed* cortisol recoverers in both the *consistent* and *dampening* cortisol reactor groups during infancy

and toddlerhood. Alternatively, this study anticipates that mothers of *delayed* cortisol recoverers would be associated with negative maternal engagement than mothers of *immediate* cortisol recoverers (see Figures 7 and 8).

3. *Do the interactions between temperament and maternal behaviors predict patterns of cortisol reactivity and recovery during infancy and toddlerhood?*

3.1. Children high on fearfulness would be more likely to elicit the “normative,” *dampening* pattern of cortisol reactivity than *consistent* cortisol reactivity and *NON*-reactivity, with higher levels of positive maternal engagement increases at 7- and 15-months of age. As previously mentioned, the strength of these predictions will be greater at 7- versus 15-months of age because children become increasingly able to regulate their emotions via attentional and inhibitory control systems that develop as part of ongoing neuropsychological maturation (Posner & Rothbart, 2000). See Figures 5 and 6.

3.2. Children who are characterized with high fearfulness or frustration would be more likely to be *consistent* cortisol reactors or *consistent NON*-reactors rather than *dampening* cortisol reactors, with higher levels of negative maternal engagement increases at 7- and 15-months of age (see Figures 5 and 6).

## Methods

### *Participants*

Participants for this study were selected from the Family Life Project (FLP), an ongoing, longitudinal examination of family functioning and child development in rural communities. In the larger study, a total of 1292 families were recruited via a complex sampling procedure from six counties with high a prevalence of rural poverty: Blair, Sampson, and Wilson counties in eastern North Carolina as well as Blair, Cambria, and Huntington counties in central

Pennsylvania. Low-income families were over-sampled (i.e., household income was < 200% below the national poverty threshold for 2002, use of social services requiring a similar income, or had less than a high school education) in both states. Families that identify themselves as black were over-sampled in North Carolina because the target communities in Pennsylvania were over 95% Caucasian. Approximately one-third of the sample is identified themselves as black, and the remaining two-thirds identified themselves as white. Further details on the Family Life Project sampling plan and recruitment procedures are available in Burchinal, Vernon-Feagans, Cox, and the Family Life Project Investigators (2008).

The current analysis concentrated on a subset of families ( $n=574$ ). The families that were selected only included those in which the mother was the primary caregiver (over 98% of the sample), and families who had complete baseline and 20-min post-task cortisol data at both the infant and toddler assessment (54.5% of the sample). In addition, given unavoidable variation in age at which children were assessed, the analyses were restricted to children between 5- and 9-months of age at the infancy assessment and 13- and 19- months at the toddler assessment. This resulted in the exclusion of 86 participants greater than 9 months of age in the analysis of the infancy data and the exclusion of 69 participants greater than 19 months of age in the analysis of the data from the toddler period, leaving a total sample of 547 children.

At the infant assessment, the children's ages ranged from 5.1 months to 8.9 months ( $M = 7.2$  months). At the toddler assessment (second home visit), the ages ranged from 14.1 months to 18.6 months ( $M = 15.4$  months). The average amount of time lapsed between visits was 8.21 months ( $SD = 1.22$ ).

Based on the mothers' ethnicity, the sample was 58% white and 42% black. Most (51.9%) of the mothers were not married at the time of the infant interview. The majority

(88.8%) of currently single mothers had never been married, and 66.6% had an income-to-needs ratio less than 200% of the poverty line, which paralleled the economic structure of the larger sample. Income-to-needs was calculated by dividing primary caregiver report of monthly household income by the federal poverty thresholds for 2004, adjusted for family size.

Statistical differences between the demographic characteristic of children included in the present study and those excluded from this analysis were tested. The results revealed that the children excluded from the study were not statistically different ( $p < .05$ ) than children on key demographic variables such as race, SES (income-to-needs ratio), age, gender, and mother's marital status.

### *Procedures and Measures*

#### *Overview*

When the child was approximately 7-months of age, two home visits were completed, in which each lasting over 2 to 3 hours. During one of the two visits, the primary caregiver filled out questionnaires regarding family demographics and income. Additionally, primary caregivers participated in a 10 minute, free-play interaction with their infant that was digitally recorded (Cox, Paley, Burchinal, & Payne, 1999; NICHD ECCRN, 1999). Subsequently, the caregiver-reported, health screening questionnaire regarding the child was completed, and pre-task saliva samples were collected. Immediately following pre-task saliva collection, all four challenge tasks (toy reach task, mask task, barrier task, and arm restraint) were performed to elicit emotional reactivity. The arm restraint was placed last in the sequence of task because it was hypothesized that this task would elicit peak arousal in most infants. The completion of the arm restraint task was speculated as taking place when the average infant experienced peak arousal, or 20 seconds of hard crying by the infant.

Twenty minutes after the arm restraint, or after 20 seconds of hard crying, a post-task saliva sample was collected. Approximately 40-minutes post-peak emotional arousal a third saliva sample was collected. These times for post cortisol samples were based on Dickerson and Kemeny's (2002) meta-analysis that found the average peak cortisol response occurs 20 to 40 min from the onset of peak arousal, and recovery (return of cortisol levels) occurs 40 to 60 min after the stressor has ended. Finally, post-visit questionnaires were completed concerning child and caregiver behaviors across the entire visit.

The same children were assessed again at 15-months of age. Unlike the infant assessment, the toddler visit only included a single visit to each of the family's households. All of the procedures described in the infant assessment were performed in the toddler assessment; however, some of the challenge tasks were slightly modified based on developmental appropriateness. The arm restraint task was replaced by the toy removal task, and the barrier task was no longer performed. Accordingly, pre-task saliva samples were collected, and challenge tasks were completed (toy reach, toy removal, and mask task). Peak arousal was defined as having occurred for the average infant at the completion of the mask task, or 20-seconds of hard crying. Therefore, twenty minutes after the mask task a second saliva sample was collected, and approximately 40-minutes after peak arousal a third saliva sample was collected.

### *Behavioral Assessments*

#### *Challenge Tasks*

Children were presented with three procedures designed to elicit emotional reactivity during the 7-month home visit and two procedures at 15-month home visit. The task procedures have been previously validated (e.g., Buss & Goldsmith, 1998; Kochanska, Tjebkes, & Forman,



1998; Stifter & Braungart, 1995). Tasks were digitally recorded and presented in the following order at the infant visit: (1) toy reach, (2) mask task, (3) barrier task, and (4) arm restraint task. For the mask task, mothers were seated beside their child, and for the last tasks, barrier and then arm restraint, mothers were out of the infant's sight. At the toddler visit, the order and task administered varied slightly. The barrier task was replaced with a toy removal challenge, followed by the mask presentation, and toy reach. The arm restraint task was not performed.

At the toddler visit, the order and tasks administered varied slightly. During infancy, the arm restraint task can be utilized to effectively frustrate children because they have developed the ability to control their arms (e.g., Braungart-Rieker & Stifter, 1996). However, by the latter half of the first year of life, infants begin to demonstrate a preference for exploring and manipulating toys and objects. Therefore, a powerful frustrator for older infants and toddlers is the toy removal task. Based on this reasoning, the barrier task was replaced with a toy removal challenge. The arm restraint task was not performed. As a result, the order at the 15-month assessment for the challenge tasks was (1) toy reach, (2) toy removal, and (3) mask task. Home visitors administered these challenge tasks while children were in a walker/infant seat.

The toy reach task designed to examine attention and latency to reach. The toy reach task involved alternating 1-minute presentations of stimulating toy sets (two presentations each) and was designed by Rothbart (1988) to assess approach/withdrawal tendencies. It should be noted that this study focused on emotional distress to the challenge tasks; therefore, the observations from the toy reach task were not included in the analyses. Alternatively, the remaining three tasks were intended to elicit distress. Specifically, the mask task was administered to elicit distress to novelty; whereas, the barrier, arm restraint, and toy removal task were both designed to elicit distress to frustration. During the mask task, children were presented with a succession

of four unusual masks for 10-second intervals. While wearing each of the masks, the home visitor leaned forward toward the seated child, turned his/her head from side to side and repeated the child's name out loud. For the barrier task at the infant home visit, the home visitor presented and encouraged the child to play with an attractive toy for 30 seconds. The toy was then placed behind a clear plastic barrier just out of the child's reach for 30 seconds. Finally, the toy was returned to the child and this procedure was performed three more times. During toy removal task at the toddler assessment, the child was encouraged to play with an attractive toy for 60 seconds. The mother of the child then removed the toy and began conversing with the home visitor. After two minutes, the mother gave the toy back to the child, while continuing to converse with the home visitor for one more minute. Lastly, for the arm restraint task, an experimenter crouched behind the infant and gently restrained the child's arms for 2 minutes or until 20-seconds of hard crying ensued. During the first two tasks, the mother remained seated beside her child, but was asked not to interact with the child during the task. For the last two tasks, mothers were out of the child's sight, but within hearing range. Mothers were asked not to intervene, but were told they could stop the tasks at any time. Following the challenge tasks, infants were allowed to self-soothe for 1 minute, and then mothers were told they could comfort their child as they would normally.

*Behavioral Coding of Challenge Tasks.* Teams of undergraduate coders were trained to assess behaviors related to negative reactivity and regulation during the "challenge tasks" (Braungart & Stifter, 1991; Stifter & Braungart, 1995; Stifter & Fox, 1990; Stifter & Spinrad, 2002). Interrater reliabilities were established using Cohen's Kappa, which compares second-by-second, microanalytic level data. Coders are trained to reliability (minimum kappa of .75)

and drift reliability was assessed on 15% of the DVDs (all kappas greater than .75) by task for each coding team.

*Negative behavioral reactivity during challenge tasks.* Negative vocalizations were assessed by raters on 4-point scale (0-3) where, 0 = “no reactivity”, 1 = “mild reactivity” (whimper or fuss), 2 = “moderate reactivity” (continuous crying), 3 = “high reactivity” (hysterical crying).

Based on this scale, for each task, proportions of time spent at each level of intensity of negative vocalization for each task were calculated by dividing the total amount of time in seconds at each level of intensity by the total time spent on the task (Braungart & Stifter, 1991; Stifter & Braungart, 1995). The mean intensity of negative reactivity during each of the three challenge tasks (masks, barrier, and arm restraint) was computed by multiplying the total number of seconds at each intensity level by the code used to reflect that intensity (0 for “no reactivity”, 1 for “mild reactivity”, 2 for “moderate reactivity”, and 3 for “high reactivity”). This score is then divided by the total number of seconds in the task. For example, the mean intensity score for a child that displayed 3 seconds at each intensity level during the arm restraint would be  $(3*0) + (3*1) + (3*2) + (3*3) = 18/12 = 1.5$  for that task.

Summary variables were also computed to reflect negative behavioral reactivity the entire task series. Once the mean intensity was computed for each task, these scores were summed across the three tasks to reflect total negative behavioral reactivity across the task series for each infant.

At the toddler assessment, separate coding was performed for the toy removal period and the interval of time after the toy is returned to the child and before the primary caregiver engages the child. Kappas were .91 for the toy removal episodes and .89 for the mask task.

## *Biological Assessments*

### *Saliva Collection*

To assess changes in cortisol in response to the emotion challenge tasks, three saliva samples were collected from the children: 1) a pre-task, baseline sample was collected prior to the task presentation, 2) 20-minutes after the children's peak arousal to the mask task, and 3) 40-minutes after peak arousal. Peak arousal was clearly outlined for home visitors in the protocol, and a substantial portion of the children reached peak arousal directly following the last emotional challenge tasks during both assessments. As mentioned above, the most developmentally arousing task was presented last at each age—the arm restraint which is meant to elicit distress to limitation/frustration during infancy, and the mask task which is meant to elicit distress to novelty/fear during toddlerhood.

Saliva samples were collected via a cotton dental rope, cotton and needleless syringe, or a micro sponge in which saliva is later expressed during centrifugation (Granger, Kivlighan, Fortunato et al., 2007). Studies have indicated that there are no differences in cortisol concentrations associated with these collection techniques (Granger, Kivlighan, Fortunato, Harmon, Hibel, Schwartz, & Whembolua, 2007; Harmon, Granger, Hibel, & Rummyantseva, 2007). Samples were placed on ice and temporarily stored in a -20°C freezer. Batches of samples were then shipped overnight to the Behavioral Endocrinology Laboratory at the Pennsylvania State University. Samples were stored in -80°C freezers until they were assayed.

Families were scheduled for in-home visits based on availability because of the special nature of this population (rural, single parent families, low SES), and the sizable length of each assessment (2 to 4 hours). To account for the time differential regarding emotional challenge task administration, all analyses that involved cortisol utilized time of day as a covariate.

All samples were assayed in duplicate for cortisol using a highly-sensitive, commercially available, enzyme immunoassay (510k) designed to measure adrenal function from saliva samples (Salimetrics, State College, PA). The immunoassay was run without modification to the manufacturer's recommended protocol. The tests utilized 25  $\mu\text{L}$  of saliva (for singlet determinations), had average intra- and inter-assay coefficients of variation less than 10 and 15%, and had a lower limit of sensitivity of .007  $\mu\text{g/dL}$  (range of sensitivity from .007 to 3.0  $\mu\text{g/dL}$ ). The average intra-and inter-assay coefficients of variation were less than 10% and 15%. The criterion for repeat testing of a subject's saliva sample was a variation between duplicates of greater than 20%. No samples were above the 20% error range; thus, repeat testing was not performed. Values from matched serum and saliva samples showed the expected strong, linear relationship,  $r(17) = .94, p < .0001$ .

*Group formation based on patterns of cortisol reactivity and recovery*

In order to address the research questions for the current study, groups were formed based on patterns of cortisol reactivity, and subgroups were created based 40-minute post recovery patterns during early childhood.

*Patterns of Cortisol Reactivity to Tasks.* To form different groups based on patterns of cortisol reactivity, the percent change in cortisol level was derived by subtracting the 20-min task cortisol values from the pre-task cortisol values, dividing by pre-task values, and multiplying by 100. Cortisol reactivity was subsequently defined as a greater than 10% change; this conservative estimate accounts for two times the margin of error (5%) that exists during the assaying procedure of cortisol (e.g., Granger et al., 1996). Children were assigned to groups based on whether or not cortisol reactivity occurred during the infant and toddler assessments. Specifically, children that displayed cortisol reactivity at *both* the infant and toddler assessment formed the group labeled *consistent*

cortisol reactors (N=179). Children who only displayed cortisol reactivity during the 7-month assessment but a decrease in cortisol reactivity (i.e., changes less than 10% over baseline) at 15-months formed the group named *dampening* cortisol reactors (N=155). Lastly, children who did *not* demonstrate cortisol reactivity or demonstrated a decrease in cortisol reactivity at *both* the infant and toddler assessments formed the group called *consistent NON-reactors* (N=213).

The means and standard deviations for the pre-task cortisol levels and the percent change in cortisol levels from pre-task to 20-minute post-task, controlling from pre-task levels, for each of the cortisol reactivity patterns (*consistent* cortisol reactors, *consistent NON-reactors* and *dampening* cortisol reactors) are included in Table 1. ANOVAs revealed that *consistent* cortisol reactors, *consistent NON-reactors*, and *dampening* cortisol reactors differed significantly regarding pre-task cortisol levels and percent changes in cortisol reactivity at 7- and 15-month assessments,  $F_s(2, 545)$  ranged from 19.36 to 191.18,  $ps < .001$ . Additionally, the absolute difference scores between the 20-minute post-task sample and the pre-task (baseline) sample for each of the cortisol reactivity pattern categories were calculated and appear in Table 1. Results revealed that the average difference scores (ug/dL) were at least 15 times greater than the lower limit of the cortisol assays sensitivity (.003 to .007 ug/dL).

*Subgroup Formation: Patterns of Cortisol Recovery Groups.* To differentiate the immediate recovery group of children from the delayed recovery group of children with the *consistent* and *dampening* cortisol reactors, percent changes from 20-min post-task to 40-min post task cortisol levels were calculated. That is, 40-min post-task cortisol values were subtracted from the 20-min post-task cortisol values, divided by 20-min values, and multiplied by 100. Cortisol recovery was subsequently defined as a less than -10% change (e.g., Granger et al., 1996). Thus, children who were *consistent* and *dampening* cortisol reactors were either placed into groups called *immediate*

cortisol recoverers (i.e., percent change in cortisol of less than -10%) or a *delayed* cortisol recoverers (i.e., percent change in cortisol of equal to or greater than -10%). The means and standard deviations for the percent change in cortisol levels from 20-minute post-task to 40-minute post-task, controlling for 20-minute post-task levels, for each of the cortisol recovery patterns (consistent *immediate* cortisol recoverers, consistent *delayed* cortisol recoverers, dampening *immediate* cortisol recoverers, and dampening *delayed* cortisol recoverers) are included in Table 9. Independent t-tests revealed statistically significant differences in percent changes in recovery between *immediate* cortisol recoverers and *delayed* cortisol recoverers for consistent and dampening cortisol reactors,  $t(67)=10.52$  to  $10.61$  and  $t(135)=13.63$ ,  $ps<.001$ , respectively.

#### *Mother-Child Interaction Assessment*

As part of the larger study, mothers and infants participated in a free-play episode (Vandell, 1979; NICHD Early Child Care Research Network, 1997; 1999; 2003) and were presented with three standard, age-appropriate toys. Mothers were instructed to play with their infants as they normally would, while seated on a baby blanket on the floor. Free-play episodes lasted about 10 minutes and were digitally recorded for later coding of infant and maternal social interaction behaviors.

*Quality of Maternal Parenting.* Coders were trained to assess maternal, infant, and dyadic interaction behaviors during the parent-child free-play episode (Cox, Paley, Burchinal, & Payne, 1999; NICHD ECCRN, 1999). Interrater reliabilities were established using Cronbach's alpha ( $\alpha = .81$  to  $.95$ ) on a set of practice tapes, as well as on the total sample in order to prevent coder drift. All interaction behaviors were globally coded on a 5-point scale (1-5), in which 1 = "not at all characteristic", 2 = "minimally characteristic", 3 = "somewhat characteristic", 4 = "moderately characteristic", and 5 = "highly characteristic". The following maternal interaction

behaviors were coded at the 7- and 15- assessments: mother's sensitivity, detachment, intrusiveness, positive regard, negative regard, and animation. Subsequently, these behaviors were factor analyzed with an oblique rotation, specifically Promax. Two factors were revealed, Positive Maternal Engagement ( $\alpha = .89$ ) and Negative Maternal Engagement ( $\alpha = .69$ ). Positive Maternal Engagement was comprised of the mean of mothers' scores on the following characteristics: detachment (reversed; degree of emotional disengagement), positive regard (expressed level of positive feelings towards the child), animation (energy level), and developmental stimulation (scaffolding activities with the child appropriately). Negative Maternal Engagement was defined as the mean of mothers' scores for three characteristics: sensitivity (reversed; level of responsiveness to the needs, gestures, and expressions of the child), intrusiveness (amount the mother imposed her own agenda on the interaction, and ignored the baby's needs and signals), and negative regard (degree of harsh, negative feelings expressed toward child). Reliability was calculated using intraclass correlations for 30% of mother-child interaction tapes that were rated made by two coders. Acceptable reliability was found for positive maternal engagement ( $r = .80$ ) and negative maternal engagement ( $r = .88$ ).

#### Data Management and Transformations

For all preliminary analyses, variables were examined for skewness and transformed if the skewness statistic was greater than 1 or less than -1. For salivary biomarkers, a log transformation for salivary cortisol to correct positively skewed distributions. There were no outliers greater than 3SD after the log transformations were performed.

#### Analytical Strategy

Preliminary analyses were conducted to identify situational (e.g., sample collection time<sup>1</sup>, medication use, sleep) and demographic factors (e.g., race, gender) associated with the three



cortisol reactivity groups (*consistent* cortisol reactors, *consistent* cortisol *NON*-reactors, and *dampening* cortisol reactors). Specifically, chi-square statistics and analysis of variances (ANOVAs) were conducted between these factors and the cortisol reactivity groups to determine the covariates that would have to be included in the multinomial regression models that had the cortisol reactivity groups as the dependent variables. Additionally, chi-square statistics and independent t-tests were run between the aforementioned situational and demographic factors to identify covariate to be included in the logistic regression models that had *immediate* and *delayed* cortisol recoverers as the dependent variables.

Multinomial logistic regressions (Hosmer & Lemeshow, 1989) were used to identify temperamental characteristics, maternal parenting qualities, and child temperament-maternal parenting quality interactions that were associated with variations of patterns of cortisol reactivity during infancy and toddlerhood. Multinomial logistic regressions were used because the dependent variable had greater than two, unordered categories (*consistent* cortisol reactors, *consistent* *NON*-reactors, and *dampening* cortisol reactors). After adjusting for the control variables in the model, the exponentiated regression coefficients signify the “risk” of having a particular cortisol reactivity pattern during early childhood relative to the comparison patterns (Hamilton & Seyfrit, 1994).

Unlike logistic regression, in multinomial logistic regression the dependent variable may be continuous or categorical. Since exploration of the distributions of the child temperament variables revealed a binomial distribution, whereby a substantial amount of infants did not behavioral react to the arm restraint and/or mask task, the variables were dichotomized and dummy coded. Moreover, these meaningful categorizations are consistent with the theoretical perspective that behavioral reactivity patterns may contain qualitative differences as a result of

their biological underpinning (Kagan, 1994), and have been utilized in analyses with prior observational data on children's behavioral reactivity (e.g., Loken, 2004).

For the 7-month assessment, infants who were in the highest quartile of negative behavioral reactivity were coded as 1 during the arm restraint ( $M=1.64$ ,  $SD=.23$ ) and mask task ( $M=.62$ ,  $SD=.54$ ); while, infants who were in the lower three quartiles were coded as 0 during the arm restraint ( $M=.40$ ,  $SD=.40$ ) and mask task ( $M=.02$ ,  $SD=.01$ ). For the 15-month assessment, infants who were in the highest quartile of negative behavioral reactivity were coded as 1 during the toy removal task ( $M=.68$ ,  $SD=.26$ ) and mask task ( $M=1.47$ ,  $SD=.34$ ); while, infants who were in the lower three quartiles were coded as 0 during the toy removal task ( $M=.14$ ,  $SD=.13$ ) and mask task ( $M=.29$ ,  $SD=.33$ ). It should be noted that no children significantly reacted to the barrier task, and there was little variation in their reactivity levels; therefore, the barrier task was not included in these analyses. After these variables were dummy coded, continuous predictors were standardized and all predictors were entered into the multinomial logistic regression at the same time. Additionally, demographic variables such as gender, race, and SES, along with sample collection time of day, were included in the multinomial logistic regression models if they explained a significant amount of the variance.

To determine if temperament predicted the three patterns of cortisol reactivity (*consistent* cortisol reactors, *consistent* cortisol non-reactors, and *dampening* cortisol reactors), two multinomial logistic regression models were analyzed. The first included the behaviorally coded variables of fear and frustration at 7-months (Infancy Temperament Model); whereas, the second included the behaviorally coded variables of fear and frustration at 15-months (Toddlerhood Temperament Model). Next, multinomial logistic regression models were analyzed, whereby the cortisol reactivity pattern variable was the dependent variable and the behaviorally coded

variables of maternal parenting qualities (Positive Maternal Engagement and Negative Maternal Engagement) were the independent variables during infancy (Parenting Quality at Infancy Model) and toddlerhood (Parenting Quality at Toddlerhood Model), respectively. The final multinomial logistic regression models that were run incorporated the interactions between temperamental characteristics and maternal parenting qualities as predictors. The interactions were formed by multiplying the dichotomized temperament variables (Frustration and Fearfulness) with the standardized maternal parenting quality variables (Positive Maternal Engagement and Negative Maternal Engagement) within each assessment, separately. The models were run separately for temperament and maternal parenting quality interactions assessed at 7- and 15-months (Infancy Temperament-Parenting Interaction Model and Toddlerhood Temperament-Parenting Interaction Model).

The three contrasts obtained from each of the multinomial logistic regressions examined how the aforementioned predictors were related to 1) *consistent* versus *dampening* cortisol reactors 2) *consistent* cortisol reactors versus *consistent NON*-reactors, and 3) *dampening* cortisol reactors versus *consistent NON*-reactor. The Wald chi-square reveals the effect of including that specific variable once all of the other variables are entered. Moreover, the odds ratio represents a probability ratio with respect to the reference group, whereby the closer the odds ratio is to 1.00, the lower the likelihood that variable can effectively predict category membership.

Subsequently, to explore how children's fear and frustration at 7- and 15-month relates to patterns of cortisol recovery (*immediate* and *delayed* cortisol recoverers), 2 X 2 contingency tables and chi-square tests were performed. The associations between maternal parenting qualities (positive maternal engagement and negative maternal engagement) and patterns of

cortisol recovery (*immediate* and *delayed* cortisol recoverers) were tested by binary logistic regression models.

## Results

### *Cortisol Reactivity Group Descriptives*

The means, standard deviations, and percentages for the different cortisol reactivity groups are provided in Table 1. Chi-square statistics and analysis of variances (ANOVAs) revealed that race was the only covariate that significantly explained variance in cortisol reactivity group membership (*consistent* cortisol reactors, *consistent* cortisol *NON*-reactors, and *dampening* cortisol reactors). Therefore, race was included as a covariate in the main analyses.

It should be noted, that all multinomial logistic regressions models were run with and without sampling time of day at 7- and 15-months as a covariate to ensure that the disparate sample collection time did not explain a significant amount of the variance predicting cortisol reactivity categories. Since the chi-square differences were not significant between models that were run with and without time of day as a covariate, only the models with race as a covariate are reported.

To determine how the child temperament characteristics and maternal parenting qualities were related to one another at each assessment and between assessments (7- and 15-month visits), the appropriate correlations coefficients were examined (Table 2). Based on the interpretations criteria of Cohen (1988), the temperamental qualities of fear and frustration are weakly and significantly correlated within and between the 7- and 15-month assessments. In terms of maternal parenting qualities, both positive and negative maternal engagement demonstrated strong, significant correlations within construct across 7- and 15-month

assessments. Furthermore, negative maternal engagement showed weak, negative correlations with positive maternal engagement.

### *Patterns of Cortisol Reactivity*

To answer the research questions regarding the prediction of pattern of cortisol reactivity, six multinomial logistic regression models were analyzed. In each of the models the categorical dependent variable (*consistent* cortisol reactors, *consistent* cortisol *NON*-reactors, and dampening cortisol reactors) was predicted from the control variables (race), child temperament, quality of parenting, as well as the interaction between temperament and parenting quality. Table 3 displays unadjusted descriptive statistics for predictor variables of child temperament and quality of parenting by cortisol reactor classifications at 7- and 15-months.

### *Do temperamental characteristics of frustration and/or fearfulness during infancy and toddlerhood predict cortisol reactivity patterns across early childhood?*

Two multinomial logistic regressions were utilized to examine the associations in child temperament during various challenge tasks among the three groups of cortisol reactors (*consistent* cortisol reactors, *consistent* cortisol *NON*-reactors, and dampening cortisol reactors), controlling for race. The Infancy Temperament Model related child temperamental characteristics of frustration and fearfulness to patterns of cortisol reactivity at infancy (7-month of age); while, Toddlerhood Temperament Model associated children's frustration and fearfulness to categories of cortisol reactivity patterns during toddlerhood (15-months of age).

*Infancy.* The Infancy Temperament Model of the multinomial logistic regressions predicted the categorical dependent variable of cortisol reactivity patterns from the control variable of race and two child temperament variables (frustration during the arm restraint and

fearfulness during the mask task) administered at the 7-month assessment. The multinomial logistic regression model was significant,  $\chi^2(6, 422) = 24.94, p < .001$ . Additionally, the likelihood ratio tests on the individual variables (see Infancy Temperament Model, Table 4) demonstrated that race, fearfulness (negative behavioral reactivity to the mask task), and frustration (negative behavioral reactivity to the arm restraint) were significant predictors of cortisol reactivity patterns.

The significant contrasts between each of the cortisol reactivity categories are determined by the parameter estimates included in Table 5. Based on the significant contrasts, infants who elicited high levels of fearfulness (within the top quartile) to the mask task were 2.15 and 2.01 times more likely to be classified as *consistent* cortisol reactors than *consistent NON*-reactors and *dampening* reactors, respectively. White infants were more likely to be classified as *consistent* and *dampening* cortisol reactors than *consistent NON*-reactors. Moreover, infants who demonstrated the highest levels of frustration to the arm restraint task (within the top quartile) were 1.99 times more likely to be categorized as *dampening* cortisol reactors than *consistent NON*-reactors.

*Toddlerhood.* The Toddlerhood Temperament Model of the multinomial logistic regressions predicted the categorical dependent variable of cortisol reactivity patterns (*consistent* cortisol reactors, *consistent* cortisol *NON*-reactors, and *dampening* cortisol reactors) from two child temperament variables (frustration during the toy removal and fearfulness during the mask task) administered at the 15-month assessment, controlling for race. The Toddlerhood Temperament Model was also significant,  $\chi^2(6, 371) = 23.08, p < .01$ , and the likelihood ratio tests on the individual variables (see Toddlerhood Temperament Model, Table 4) demonstrated

that race and fearfulness (negative behavioral reactivity to the mask task) were significant predictors of cortisol reactivity patterns.

The parameter estimate for the significant contrasts between each of the cortisol reactivity categories of the Toddlerhood Temperament Model are presented in Table 6. According to the contrasts of the Toddlerhood Temperament Model, similar to the 7-month assessment, toddlers who elicited high levels of fearfulness (within the top quartile) to the mask task were significantly more likely (2.5 times) to be classified as *consistent* cortisol reactors than *consistent NON*-reactors. However, unlike the Infancy Temperament Model, toddlers who elicited high levels of fearfulness to the mask task were 1.89 times more likely to be categorized as *dampening* cortisol reactors rather than *consistent NON*-reactors. Levels of frustration to toy removal during toddlerhood did not significantly differentiate between patterns of cortisol reactivity. Similar to the Infancy Temperament Model, white toddlers were more likely to be classified as *consistent* and *dampening* cortisol reactors than *consistent NON*-reactors.

*Does the quality of maternal parenting predict patterns during infancy and toddlerhood predict cortisol reactivity patterns across early childhood?*

To determine the relationship between different aspects of maternal parenting quality the three categories of cortisol reactors (*consistent* cortisol reactors, *consistent* cortisol *NON*-reactors, and dampening cortisol reactors) across early childhood, two additional multinomial logistic regression models were analyzed. Parenting Quality at Infancy Model explored how behaviors of positive maternal engagement and negative maternal engagement during the parent-child interaction were related to patterns of cortisol reactivity at infancy (7-month of age).

Parenting Quality at Toddlerhood Model associated those same parental qualities measured at the 15-month assessment to the categories of cortisol reactivity patterns across early childhood.

*Parenting Quality in Infancy.* Multinomial logistic regressions predicted the categorical dependent variable of cortisol reactivity patterns (*consistent* cortisol reactors, *consistent* cortisol *NON*-reactors, and dampening cortisol reactors) from the two maternal parenting qualities observed in infancy (positive maternal engagement and negative maternal engagement), controlling for race. The overall model was significant,  $\chi^2(6, 531) = 24.62, p < .001$ , and the likelihood ratio tests on the individual variables (Table 7) demonstrated that race and positive maternal engagement during infancy were significant predictors of cortisol reactivity patterns.

The parameter estimate for the contrasts between each of the cortisol reactivity categories of the Parenting Quality at Infancy Model are presented in Table 8. Contrasts revealed that children of mothers who exhibited more positive maternal engagement (low levels of detachment, as well as high levels of positive regard, animation, and developmental stimulation) during the parent-child interaction at the 7-month assessment were 1.39 (equivalent to .71 times less likely) and 1.40 times more likely to be *dampening* cortisol reactors than *consistent* cortisol reactors and *consistent NON*-reactors, respectively. Additionally, children of mothers who demonstrated positive maternal engagement were more likely (2.10 times) to be *consistent* cortisol reactors than *consistent NON*-reactors. Negative maternal engagement did not significantly predict categories of cortisol reactivity patterns across early childhood. Additionally, white infants were more likely to be classified as *consistent* and *dampening* cortisol reactors than *consistent NON*-reactors.

*Parenting Quality in Toddlerhood.* Multinomial logistic regressions predicted the categorical dependent variable of cortisol reactivity patterns (*consistent* cortisol reactors,



*consistent* cortisol *NON*-reactors, and *dampening* cortisol reactors) from the control variable of race and the same two maternal parenting qualities (positive maternal engagement and negative maternal engagement) administered at the 15-month assessment. The overall model was not significant,  $\chi^2(6, 478) = 16.27, p < .05$ . However, the likelihood ratio tests revealed that neither positive nor negative maternal engagement were significant predictors of cortisol reactivity patterns across early childhood. The significant likelihood ratio tests demonstrated that White infants were more likely to be classified as *consistent* and *dampening* cortisol reactors than *consistent NON*-reactors,  $ps < .01$ .

*Do interactions between child temperament and quality of maternal parenting during infancy and toddlerhood predict cortisol reactivity patterns across early childhood?*

In order to assess whether interactions between child temperament and quality of maternal parenting during infancy and toddlerhood predicted cortisol reactivity patterns, two multinomial logistic regression models were analyzed (Infancy Temperament-Parenting Interaction Model and Toddlerhood Temperament-Parenting Interaction Model).

Infancy Temperament-Parenting Interaction Model determined whether interactions between child temperament and maternal parenting qualities during the 7-month assessment predicted the three categories of cortisol reactors (*consistent* cortisol reactors, *consistent* cortisol *NON*-reactors, and *dampening* cortisol reactors). The four interactions that were examined included all of the combinations between temperamental characteristics of frustration and fearfulness with maternal parenting behaviors of positive and negative engagement. In the Infancy Temperament-Parenting Interaction Model, all main effects of child temperament and maternal parenting quality and interactions were added into the model simultaneously. Overall,

this model demonstrated that no interactions predicted the categories of cortisol reactivity patterns (*consistent* cortisol reactors, *consistent* cortisol *NON*-reactors, and *dampening* cortisol reactors) across early childhood.

For the toddlerhood assessment, a similar multinomial logistic model was analyzed. The Toddlerhood Temperament-Parenting Interaction Model included the main effects of frustration during the toy removal task, fearfulness during the mask task, the four variables of parenting quality, and the eight interactions between these effects as predictors. The Toddlerhood Temperament-Parenting Interaction Model was significant,  $\chi^2(30, 293) = 58.93, p < .01$ , and the likelihood ratio test revealed that only one interaction significantly predicted the categories of cortisol reactivity patterns (*consistent* cortisol reactors, *consistent* cortisol *NON*-reactors, and *dampening* cortisol reactors) across early childhood. Specifically, Fearfulness during the Mask Task X Negative Maternal Engagement at 15-months significantly predicted the classifications of cortisol reactivity patterns,  $\chi^2(2, 293) = 9.21, p < .01$ . Pairwise comparisons demonstrated that the interaction between Fearfulness during the Mask Task and Negative Maternal Engagement was due to differences in the prediction of *consistent* cortisol reactors versus *consistent NON*-reactors (Wald  $\chi^2 = 8.00, B = .77, SE = .27, OR = 2.15, CI = 1.42 - 3.91$ ). On the whole, toddlers that have an interaction between fearfulness and negative maternal engagement are 2.15 times more likely to be *consistent* cortisol reactors than *consistent NON*-reactors.

To further explore how the Fearfulness during the Mask Task X Negative Maternal Engagement predicted *consistent* cortisol reactors versus *consistent NON*-reactors, the plot of the interaction was created. The specifics of the plot were as follows: the probability of toddlers being in the *consistent* cortisol reactor group was the predicted variable, the dichotomized variable of high and low fearfulness to the mask task was the predictor, and high versus low

levels of negative maternal engagement was the moderating variable (see Figure 9). The probability that toddlers who demonstrated high fearfulness to the stress and are exposed to high levels of negative maternal engagement were *consistent* cortisol reactors was 68%; whereas, toddlers who are high on fearfulness exposed to low levels of negative maternal engagement only had a 40% of being *consistent* cortisol reactors. Whereas, the probability that toddlers who demonstrated low fearfulness to the stress and are exposed to high levels of negative maternal engagement were *consistent* cortisol reactors was 56%; whereas, toddlers who are low on fearfulness exposed to low levels of negative maternal engagement only had a 32% of being *consistent* cortisol reactors. Post hoc, multiple regression probing of this interaction effect (Aiken & West, 1991), with the probability of being a *consistent* cortisol reactor, revealed that toddlers of mothers who exhibited greater negative parenting towards their children ( $z \geq +1$ ) had an increased probability of being a *consistent* cortisol reactor as the amount of children's behavioral fearfulness increased,  $\beta = .68$ ,  $t(184) = 9.28$ ,  $p < .001$ . Additionally, multiple regression probing of this interaction effect revealed that mothers who displayed lower levels of negative parenting towards their children ( $z \leq -1$ ) had a decreased probability of being a *consistent* cortisol reactor as the amount of children's behavioral fearfulness increased,  $\beta = -.27$ ,  $t(184) = -3.07$ ,  $p < .01$ .

### *Patterns of Cortisol Recovery*

#### *Cortisol Recovery Group Descriptives*

The means, standard deviations, and percentages for *immediate* and *delayed* cortisol recoverers groups are presented in Table 9. Preliminary analyses were conducted to identify situational (e.g., sample collection time<sup>1</sup>, sleep habits) and demographic factors (e.g., race, gender) associated with *immediate* and *delayed* cortisol recoverers from the *consistent* cortisol as

well as the *dampening* cortisol reactors. Specifically, chi-square statistics and independent t-tests were conducted between these potential covariates and the *immediate* and *delayed* cortisol recoverers from the *consistent* cortisol and *dampening* cortisol reactors, separately. Results revealed that there were no significant covariates.

*Do maternal parenting qualities during infancy and toddlerhood predict patterns of immediate-versus delayed-cortisol-recoverers across early childhood?*

Two logistic regressions were performed to determine how positive maternal engagement and/or negative maternal engagement were related to categories of cortisol recoverers (*immediate* and *delayed* cortisol recoverers).

The first logistic regression models predicted *immediate* and *delayed* cortisol recoverers derived from the *dampening* reactor group using standardized maternal parenting qualities (positive maternal engagement and negative maternal engagement) ascertained at infancy. The second logistic regression model explored the relation between maternal parenting qualities collected during toddlerhood and the *dampening* reactor group's patterns of recovery. All predictor variables were entered into each of the models at the same time. Neither the 7- nor 15-month logistic regression model revealed significant relationships between any of the maternal parenting qualities and the categories of cortisol recovery from the *dampening* cortisol reactors.

In contrast, similar logistic regression models that explored the relationship between the same maternal parenting quality and categories of cortisol recovery (*immediate* and *delayed* cortisol recoverers) derived from the *consistent* cortisol reactors revealed significant results. The omnibus test of the logistic regression model with maternal parental quality at 7-months predicting the categories of *consistent* cortisol recoverers (*immediate* and *delayed* cortisol

recoverers) was significant,  $\chi^2(2, 62) = 9.82, p < .01$ . Parameter estimates revealed that mothers who engaged with their infants in a sensitive and responsive manner during infancy were 2.11 times more likely to be *immediate* than *delayed* cortisol recoverers, Wald  $\chi^2 = 7.53, \beta = .75, SE = .30, CI = 1.24 - 3.60, p < .01$ . In contrast, mothers who engaged with their infants in an intrusive and insensitive manner during infancy demonstrated a trend towards being 1.77 times *less* likely to be *immediate* than *delayed* cortisol recoverers, Wald  $\chi^2 = 2.97, \beta = -.58, SE = .30, CI = .92 - 3.45, p < .10$ . Similar significant findings were revealed association between positive maternal parenting qualities at the 15-months and the categories of *consistent* cortisol recoverers,  $\chi^2(2, 62) = 8.33, p < .05$ . Specifically, mothers who positively engaged with their toddlers during toddlerhood were 1.92 times more likely to be *immediate* than *delayed* cortisol recoverers, Wald  $\chi^2 = 4.72, \beta = .65, SE = .30, CI = 1.06 - 3.47, p < .05$ .

### *Discussion*

Overall, the current study provided evidence that children's temperamental characteristics of fearfulness and frustration exhibited during mild stressors, qualities of observed maternal parenting, as well as temperament and parent quality interactions at 7- and 15-months-old differentially and significantly predicted children's patterns of cortisol reactivity (*consistent* cortisol reactors, *consistent* cortisol *NON*-reactors, and dampening cortisol reactors). In particular, children who demonstrate frustration during infancy were more likely to be *dampening* cortisol reactors than *consistent NON*-reactors across early childhood. In contrast, children who showed fearfulness during infancy *and* toddlerhood were more likely to be *consistent* cortisol reactors than *consistent NON*-reactors.

Regarding maternal parenting qualities, *dampening* cortisol reactors demonstrated a straightforward relationship with positive parenting qualities in infancy. Specifically, infants exposed to positive maternal engagement (high emotional involvement, expression of positive feelings, and animation) during a parent-child interaction had an increased likelihood of being *dampening* cortisol reactors than *consistent* reactors and *consistent NON*-reactors. However, *consistent* cortisol reactors exhibited a more complex association with maternal parenting qualities. First, infants who were exposed to positive maternal engagement were more likely to be *consistent* cortisol reactors than *consistent NON*-reactors. Additionally, the interaction between child temperament and maternal parenting qualities revealed that both extremely fearful children exposed to high amounts of negative maternal engagement and children with low behavioral fearfulness exposed to low levels of negative maternal engagement significantly predicted membership in the *consistent* cortisol reactivity group versus the *consistent NON*-reactivity group.

Finally, concerning patterns of cortisol recovery, this investigation determined that positive maternal parenting qualities in infancy and toddlerhood were significant predictors of *immediate* versus *delayed* cortisol recovery across early childhood. To expound, children whose mothers were positively engaged during infancy and toddlerhood exhibited an increased likelihood of being *immediate* versus *delayed* cortisol recoverers in the consistent reactor group only. On the contrary, negative maternal engagement during infancy demonstrated a trend toward an increased likelihood that children would be *delayed* versus *immediate* cortisol recoverers in the *consistent* cortisol reactivity group.

In general, these findings underscore that both the proximal characteristics of child temperament and the more distal aspects of maternal parenting qualities are key factors

associated with cortisol reactivity patterns (*dampening* cortisol reactors, *consistent* cortisol reactors, and *consistent NON*-reactors) that develop across early childhood and may later confer risk or resilience. Moreover, the current analysis clearly demonstrates that positive and negative parenting qualities may be useful in predicting consistent patterns of *immediate* versus *delayed* cortisol recovery.

#### *Child Temperamental Characteristics and Patterns of Cortisol Reactivity*

The first goal of the present study was to examine whether child temperamental characteristics of frustration (distress to limitations) or fearfulness (distress to novelty) at 7- and 15-months of age predicted membership in the three groups of cortisol reactivity patterns (*dampening* cortisol reactors, *consistent* cortisol reactors, and *consistent NON*-reactors) across early childhood. In general, the challenge tasks (as predicted) demonstrated that children who displayed greater behaviors of frustration to the arm restraint task than fearfulness to the mask task during infancy, regardless of the pattern of cortisol reactivity they displayed across early childhood (Table 3). This is developmentally justifiable because the average age of onset for fear is 7- to 9-months of age (e.g., Scarr & Salapatek, 1970); hence, on average, children were more prone to demonstrating peak behavioral arousal during the frustration versus fear eliciting tasks.

The results also showed that children who demonstrated greater amounts of observed frustration and fearfulness to challenge tasks at 7-months of age were associated with the patterns of cortisol reactivity that demonstrated increased cortisol reactivity during infancy (i.e., *consistent* cortisol reactors and *dampening* cortisol reactors). Specifically, in accordance with the hypothesized relationship between behavioral frustration during the arm restraint and cortisol reactivity patterns, the findings revealed that infants were 1.99 times more likely to be

*dampening* cortisol reactors than *consistent NON*-reactors. However, contrary to the studies predictions, behavioral frustration during the arm restraint did not determine whether children would be more likely to be *consistent* cortisol reactors than *consistent NON*-reactors during infancy. Instead, a smaller contingency of infants who demonstrated fearfulness during the mask task were 2.01 to 2.15 times more likely to be *consistent* cortisol reactors than *dampening* and *consistent NON*-reactors, respectively. This finding suggests that those infants who had demonstrated the ability to experience fear earlier more likely to demonstrate *consistent* cortisol reactivity patterns across infancy and toddlerhood.

In infancy, these results indicated that behavioral reactions of frustration and fearfulness are coordinated with adrenocortical reactivity to stressors. That is, frustration to the arm restraint task was positively correlated to cortisol reactivity in *dampening* cortisol reactors during infancy, and fearfulness during the mask task was positively associated with cortisol reactivity in *consistent* cortisol reactors during infancy. A potential reason for these findings is that infants lack the experience and availability of effective self-regulatory behaviors, which may lead to both increased behavioral and adrenocortical reactivity. This interpretation of the findings is theoretically supported by Gottlieb's (1992) notion of the "behavioral surface," which speculated that individuals utilize coping behaviors and subjective experience restructuralization to adjust to the dynamic changes in the environment, and only after those have failed, are the physiological processes activated. Moreover, empirical research has found that antecedents of this self-regulatory mechanism such as sustained attention emerge between 6 and 12 months of age, and develop into a more concrete aspect of a child's temperament by the second year of life (e.g., Rothbart & Bates, 1998; Ruff & Rothbart, 1996). Thus, the lack of fully developed coping mechanisms in 7-month-old infants may increase the probability that both a behavioral and



adrenocortical response will occur during stress exposure. In sum, this association between increased behavioral frustration and cortisol levels to a stressor in *dampening* cortisol reactors, as well as increased behavioral fearfulness and cortisol levels to a stressor in *consistent* cortisol reactors, during infancy may be developmentally appropriate.

During toddlerhood, the challenge tasks demonstrated that children exhibited more behavioral fearfulness to the mask task than frustration to the toy removal task, regardless of their pattern of cortisol reactivity across early childhood (Table 3). As anticipated, children who demonstrated fearfulness during the mask task were 2.50 times more likely to be *consistent* cortisol reactors than *consistent NON*-reactors. Unexpectedly, however, toddlers who elicited higher levels of fearfulness during the mask task were not more likely to be *consistent* cortisol reactors than *dampening* cortisol reactors. Instead, the results revealed that toddlers who showed higher levels of fearfulness on tasks were 89% more likely to be *dampening* cortisol reactors than *consistent NON*-reactors.

Taken together, unlike the findings shown during infancy, toddlers who demonstrated behavioral fearfulness to challenge tasks was not coordinated necessarily with patterns of cortisol reactivity. In particular, *dampening* cortisol reactors had an increased likelihood of demonstrating behavioral fearfulness but not cortisol reactivity to the mask task during toddlerhood; while, *consistent* cortisol reactors had an increased likelihood of exhibiting both behavioral and cortisol reactivity to the mask task during toddlerhood. Researchers have speculated that the activation of each mechanism in response to a stress is dependant on prior experience with stressors and the availability of effective self-regulatory behaviors such as attention control (Gunnar et al., 1988; Gunnar et al., 1989; review, Egliston, McMahon, & Austin, 2007). To explicate, increased experience allows the HPA axis to habituate to similar

situational stressors or set of circumstances, resulting in diminution of HPA activation to comparable environmental challenge (e.g., McEwen, 1998). Furthermore, as mentioned above, the development of self-regulation skills allows children to behaviorally cope with stressors and decreases the probability that a physiological stress response will occur. Based on this hypotheses, these findings during toddlerhood indicate that *dampening* cortisol reactors may have more experience and/or availability of self-regulatory behaviors at 15-months of age than *consistent* cortisol reactors. Thus, although *consistent* cortisol reactivity had an earlier onset for developing the emotion of fear in infancy than *dampening* cortisol reactors, *consistent* cortisol reactors may not have the experience or behavioral self-regulation skills necessary to avoid the activation of the HPA system as compared to *dampening* cortisol reactors during toddlerhood.

In conjunction with the prior hypotheses that support the current study's findings, researchers have conjectured that physiological changes lead to different patterns of cortisol reactivity, but not necessarily behavioral reactivity, across early childhood (e.g., Hellhamer & Wade, 1993; see review, Adam, Klimes-Dougan, & Gunnar, 2007). Specifically, individuals who demonstrate decreased HPA activation across early childhood, but not behavioral reactivity, may have a greater propensity for negative feedback regulation of the HPA axis and/or decreased ATCH sensitivity in the adrenal cortex (Lashansky et al., 1991). In accordance with this theory, *dampening* cortisol reactors may undergo greater negative feedback regulation of the HPA axis, and/or their adrenal cortex may develop decreased sensitivity to the ATCH, than *consistent* cortisol reactors and *consistent NON*-reactors.

Lastly, the present study showed that the lowest levels of observed frustration or fearfulness to the challenge tasks were associated with *consistent NON*-reactors as compared to

*consistent* and *dampening* cortisol reactors. One potential explanation for the consistent lack of adrenocortical and behavioral reactivity to stressors in *consistent NON*-reactors is that they are genetically predisposed to developing individual differences in physiology that are associated with underarousal (Raine, 1996). For example, this pattern of hyporeactivity may be associated with dysfunctional serotonergic regulation of the HPA axis, which has been demonstrated in children (7 to 12 years old) and adolescents (Soloff, Lynch, & Moss, 2000; Snoek et al., 2002). Moreover, researchers have speculated that chronic exposure to stressors and/or trauma may eventually lead to the downregulation of the HPA system (Gold & Chrousos, 2002; Hellhamer & Wade, 1993; Susman, 2006). Therefore, the present studies may support the notion that *consistent NON*-reactors developed physiological differences in HPA activation to stressors over time because of a genetic predisposition that allows for a higher stress tolerance and/or chronic experience to stress and trauma. However, future analyses of this samples data and other studies that include genetic markers and extensive measures of environmental stress are warranted to determine the underlying mechanisms that result in patterns of *consistent* non- and hypo-reactivity across early childhood.

#### *Maternal Parenting Qualities and Patterns of Cortisol Reactivity*

The second goal of the present study was to investigate whether observed maternal parenting qualities (positive maternal engagement and negative maternal engagement) when children were 7- and 15-months of age predicted the likelihood of membership in the three categories of cortisol reactivity patterns (*dampening* cortisol reactors, *consistent* cortisol reactors, and *consistent NON*-reactors) across early childhood. Overall, positive maternal parenting qualities during infancy and toddlerhood were predictive of patterns of cortisol reactivity across

early childhood. However, the exact qualities of positive maternal parenting that were associated with the three categories of cortisol reactivity patterns (*dampening* cortisol reactors, *consistent* cortisol reactors, and *consistent NON*-reactors) differed when children were 7- and 15-months of age.

At the 7-month assessment, consistent with expectations and similar to Blair and colleagues' (2008) findings, results revealed that children whose mothers exhibited maternal engagement (low emotional disengagement, high positive regard, and high energy level) during a parent-child interaction were significantly more likely to be *dampening* cortisol reactors than *consistent* cortisol reactors and *consistent NON*-reactors. Thus, the theoretical implications of these results are that infants who are exposed to positive maternal qualities may form an internal working model that mothers will externally regulate their stress exposure (Ainsworth et al., 1979; Bowlby, 1969), and ultimately, buffer their children against HPA activation to challenges (Gunnar & Quevedo, 2007). These findings support the theory that children with positively engaging mothers, especially during the early months of infancy, may be associated with their infants' cortisol reactivity during the first year of life with an eventual diminution of HPA activation by the second year of life (e.g., Gunnar, 2003; Gunnar & Quevedo, 2007). This is similar to the research with rat pups, which revealed that greater maternal licking and grooming (positive maternal parenting qualities) is associated with vigorous glucocorticoid reactivity in rat pups for the first few days of life followed by enhanced development of glucocorticoid regulation, and the onset of a "normative" stress hyporesponsive period (Rosenfield et al., 1992; Sapolsky & Meaney, 1986). Moreover, high-grooming and licking rat pup mothers are associated with improved negative feedback regulation (Liu et al., 1997; Meaney & Szyf, 2005), and better cognitive capabilities concerning learning and memory

tasks (Liu et al., 2000). In general, these results suggest that positive maternal parenting qualities during infancy may assist children in developing a HPA stress response system that is more efficient, as shown in the *dampening* cortisol reactors of this sample.

At the 15-month assessment, contrary to the anticipated hypotheses, neither positive maternal engagement nor negative maternal engagement was associated significantly with any of the cortisol reactivity patterns. The fact that maternal engagement assessed via parent-child interactions is associated with cortisol reactivity patterns at 7-months but not at 15-months may suggest that the parental qualities encompassed in these larger constructs of maternal engagement have a greater influence on HPA functioning in infancy than in later in development. Alternatively, it may be that the qualities of maternal engagement that are assessed during the mother-child free play interactions specifically are not associated with toddler's cortisol reactivity patterns. Thus, future analyses should be performed to test whether maternal parenting qualities during toddlerhood that are assessed via alternative observational situations and self-report measures are related differentially to cortisol reactivity patterns across early childhood.

In total, the findings regarding parenting quality and patterns of cortisol reactivity demonstrated that positive maternal parenting during the sensitive period of infancy may assist in the development of efficient HPA stress response system. That is, children have a greater likelihood of exhibiting adrenocortical reactivity to a mild stressor during infancy, but not during toddlerhood (*dampening* cortisol reactors).

#### *Cortisol Recovery Patterns: Child Temperament and Parenting Qualities*

The third goal of the present study was to fill in the literature gap regarding the individual and environmental factors that may shape patterns of cortisol *recovery* across early childhood.

Specifically, this study examined whether maternal parenting qualities predicted *immediate* versus *delayed* cortisol *recovery* across early childhood in consistent cortisol reactors and dampening cortisol reactors, separately. Overall, maternal parenting qualities were not associated with patterns of cortisol recovery in *dampening* cortisol reactors during early childhood. Conversely, maternal parenting qualities at both 7- and 15-months of age influenced children's cortisol recovery patterns in *consistent* cortisol reactors.

In accordance with expectations, the data analysis revealed that high levels of maternal engagement during infancy and toddlerhood increased the likelihood that children would display *immediate* versus *delayed* patterns of recovery. In contrast, greater exposure to negative maternal engagement during infancy demonstrated a trend towards an increased likelihood that children would be *delayed* versus *immediate* cortisol recoverers in the *consistent* cortisol reactivity group. In general, these results support the theoretical notion that mothers who positively engage with their children act as an external regulator for stress during infancy and toddlerhood when effortful self-regulation has yet to be fully developed (e.g., Bowlby, 1969; Ruff & Rothbart, 1996; Posner & Rothbart, 2000). Empirically, the findings are consistent with the recent study that revealed the positive association between children's immediate cortisol recovery to a stressor and positive maternal caregiving during infancy (Albers et al., 2008). Lastly, these findings lend evidentiary support that infant's exposure to negative parenting qualities including high levels of intrusiveness, low levels of sensitivity, and high levels of negative regard during infancy may be associated with the stable pattern of allostatic load, in which there is a failure to "appropriately" turn off the stress response system.

In sum, the straightforward and consistent association between positive maternal engagement and *immediate* cortisol recovery across early childhood after reacting to a stressor

illuminates the importance of including cortisol recovery measures in empirical research designs that investigate HPA functioning during early childhood. Additionally, the finding that negative parenting qualities in infancy only increased the likelihood of *delayed* versus *immediate* cortisol recovery in consistent reactors may suggest that certain qualities of maternal parenting have a greater impact on HPA function at certain developmental time points.

#### *Interactions with Child Temperament and Parenting Qualities and Cortisol Reactivity Patterns*

The final goal of the present study was to investigate whether children's temperamental characteristics (frustration and fearfulness) interacted with maternal parenting qualities (positive maternal engagement and negative maternal engagement) during infancy and toddlerhood to predict membership in the three categories of cortisol reactivity patterns (*dampening* cortisol reactors, *consistent* cortisol reactors, and *consistent NON*-reactors). Contrary to expectations, only one interaction during toddlerhood was associated with patterns of reactivity across childhood, and the intricacies involved in this interaction was more complex than originally hypothesized.

The present study anticipated that children who are characterized with high fearfulness would be more likely to be *consistent* cortisol reactors rather than *dampening* cortisol reactors as the quality of negative parenting (negative maternal engagement) increased at 15-months of age. However, the results revealed that toddlers who demonstrated low fearfulness and were exposed to low levels of negative maternal engagement had a greater probability of being *consistent* cortisol reactors versus *consistent NON*-reactors (see Figure 9). Furthermore, toddlers who exhibited high fearfulness and had mothers who displayed high levels of negative maternal engagement had a greater probability of being a *consistent* cortisol reactor versus a *consistent NON*-reactor.

Similar to the bivalent relationship posed in the evolutionary-developmental perspective of biological sensitivity to context (BSC; Boyce and Ellis, 2005), this finding highlights that early childhood experiences in either supportive (exposure to low amounts of negative engagement) or severely stressful environments (high amounts of negative engagement) increases the likelihood of developing a pattern of heightened reactivity in the HPA axis across early childhood (*consistent* cortisol reactor). Boyce and Ellis describe these high cortisol reactors as context-sensitive individuals or orchid children, because much like the orchid, their developmental outcomes and survival is closely linked to the supportive or unsupportive characteristics of the environment. Therefore, children with highly reactive phenotypes in supportive and stable contexts will have lowered rates of physical and mental disorders. Conversely, children that are highly reactive phenotypes in unsupportive and highly stressful environments will incur increased rates of deleterious physical and mental health outcomes.

Boyce and Ellis (2005) also hypothesized that the possibility that supportive or aversive parent-child relationships may “constrain or modify the emergence of high [biological] reactivity-high [behavioral] inhibition phenotypes” (Boyce & Ellis, 2005, p. 285). Although the authors do not elaborate further, one could speculate that negative parenting qualities such as negative maternal engagement could exacerbate the emergence of high adrenocortical reactivity-high behaviorally fearful phenotypes. Multiple, cross-sectional studies have revealed similar findings, in which negative maternal qualities moderated the relationship between temperamental characteristics of fearfulness/behavioral inhibition and adrenocortical reactivity (Nachmias et al., 1996; Spangler & Schieche, 1994). These researchers have theorized that this phenomenon may occur because intrusive maternal behaviors have blocked toddlers from developing the self-regulation skills necessary for coping with changes in the environment and other stressors.



Conversely, supportive environments (low negative parenting qualities) may foster disassociation between the behavior and adrenocortical reactivity in these children. In particular, children with parents that are more sensitive, less intrusive, and elicit higher positive regard may encourage the development of high levels approach to novel environmental challenges. That is, children with mothers who are less intrusive may be more likely to engage with the novel stimuli; thus, eliciting a physiological stress response during toddlerhood but no negative or fearful behavioral response. In support of this interpretation, surgent preschoolers (fearless, impulsive, high approach, and high energy children) have exhibited elevated cortisol levels depending on the context in which HPA activity was measured (Davis, Donzella, Krueger, & Gunnar, 1999; Gunnar, Tout, de Haan, Pierce, & Stansbury, 1997; Zimmerman & Stansbury, 2004); therefore, it is not just children with fearful temperament that have demonstrated contextual HPA activation. However, it is still largely unknown how maternal parenting qualities may influence this type of relationship.

Taken together, the bivalent and complex manner in which the interaction between child temperament and maternal parenting qualities predicted the increased probability of exhibiting a pattern of *consistent* cortisol reactivity during early childhood, along with past empirical research, underscores the need for further investigation of this phenomenon. Particularly, future longitudinal studies should examine how the association among temperamental characteristics of fear, maternal parenting qualities, and patterns of *consistent* cortisol reactivity during early childhood may influence subsequent mental and physical health outcomes. Ultimately, these studies may add vital information needed to modify and/or verify theories such as the biological sensitivity to context hypothesis, as well as potentially create new theoretical frameworks.

### *Limitations*

The present study makes a unique contribution to the current literature because it not only highlights how children's temperamental characteristics and maternal parenting qualities during infancy and toddlerhood relate to patterns of cortisol reactivity across early childhood, but it also emphasizes that maternal parenting qualities at 7- and 15-months of age are primary predictors of cortisol recovery patterns. Nevertheless, there are certain limitations that must be addressed.

First, although grouping children into different patterns of cortisol reactivity was based on the theoretical premises underlying allostatic load and a proposed hyporesponsive period in humans, any grouping of continuous data inevitably results in a loss of information and power (e.g., Cohen, 1983; MacCallum, Zhang, Preacher, & Rucker, 2002). Thus, some significant relationships between child temperament, maternal parenting qualities, and patterns of reactivity and recovery across early childhood may have gone undetected.

Second, although this is among the first studies to investigate correlates of the *stable* patterns of cortisol reactivity and recovery during two crucial time points when HPA functioning is still developing (infancy and toddlerhood); the limited amount of time points does not allow the study to directly assess whether habituation to the particular stressors is occurring over time. This illuminates the need for future studies that focus at the stable patterns of cortisol reactivity over a greater number of time points that are placed closer together in time, in order to truly assess whether patterns associated with allostatic load are developing within certain children.

A third limitation of the current study is that there were variations in the time of day that children's cortisol samples were assessed during the home visits. Since cortisol has a pronounced diurnal rhythm, all logistic regression models were run controlling for time of day. The analyses revealed that models with and without the sample time of day as a covariate demonstrated no statically significant differences. Nonetheless, the disparate times of day may

have yielded variations in cortisol assessment that could have hindered the study's ability to find significant relationships between child temperament, maternal parenting qualities, and patterns of cortisol reactivity and recovery across early childhood.

Lastly, the sample for this analysis represents predominantly low-income families from rural communities. Although representative of a large subset of the United State nonurban and low income areas, the study did not examine the relationship between low-income risk and rurality. Thus, these results may not be generalizable to urban, low-income families.

### *Summary and Conclusions*

The current study provided evidence that both behavioral reactivity to a mild stressor (i.e., frustration during infancy and fearfulness during toddlerhood) and exposure to positive maternal engagement in infancy were associated with children eliciting “normative” dampening of cortisol reactivity across early childhood. These findings suggest that the coordination between behavioral and adrenocortical reactivity are likely during infancy. However, as children experience similar stressors and develop coping skills, there may be a disassociation between behavioral and adrenocortical reactivity, whereby children elicit a behavioral response but not an adrenocortical response (e.g., Gottlieb, 1992). Additionally, these findings provide evidence that positive maternal parenting during infancy may act as children's external buffers to stressors (e.g., Bowlby, 1969; Ruff & Rothbart, 1996; Posner & Rothbart, 2000), as well as foster the development of children's own self-regulation skills (e.g., Goldsmith & Harman, 1994; Kochanska, 1995). Ultimately, this analysis indicates that the pattern of *dampening* cortisol reactivity may be associated with positive outcomes in later childhood and adolescence. However, future research that directly assesses this hypothesized developmental pathway is warranted.

Additionally, the findings revealed that *consistent NON*-reactors across early childhood were associated with the lowest level of behavioral reactivity, and the least amount of positive maternal engagement. Taken together, the results showed that no cortisol reactivity or hyporeactivity across early childhood might be an indicator of increased allostatic load even in early childhood. The underactivation of the adrenocortical system that *consistent NON*-reactors exhibit may be a result of less maternal buffering of stress during environmental challenges, which increases children's exposure to chronic stress (e.g., Posner & Rothbart, 2000). These chronic experiences with stress and/or trauma such as excessively punitive and restrictive parenting could potentially result in the dysfunctional adrenocortical activation to stressors such as consistent hyporeactivity (Gold & Chrousos, 2002; Hellhamer & Wade, 1993; Soloff, Lynch, & Moss, 2000; Snoek et al., 2002; Susman, 2006). Since hyporeactivity of the HPA system has been associated with deleterious outcomes such as externalizing behaviors in childhood and adolescence (Shirtcliff, Zahn-Waxler, & Klimes Dougan, 2005; van Goozen, Matthys, Cohen-Kettenis, Gispen-de Wied, Wiegant, & van Engeland, 1998), the results from the current analyses offer a piece of information that may be vital in determining how the relationship between maternal parenting qualities and children's patterns of cortisol hyporeactivity may influence the subsequent development of negative outcomes.

In terms of the other patterns of *consistent* cortisol reactivity, the study revealed a straightforward relationship between children's temperamental characteristics and *consistent* cortisol reactors. That is, children who elicit fearfulness at 7- and 15-months are associated with *consistent* patterns of cortisol reactivity. However, whether to interpret these relationships as conferring later deleterious or positive outcomes remains unclear. Regarding *consistent* cortisol reactors, if these children have not been exposed to these types of stressors often, then HPA

activation to mild stressors during infancy and toddlerhood may be warranted and appropriate. In contrast, if the child has not habituated to similar stressors by toddlerhood, then this type of reactivity could be a potential risk factor for subsequent, deleterious outcomes (e.g., McEwen, 1998).

Alternatively, the manner in which maternal parenting qualities related to the stable pattern of *consistent* cortisol reactivity across early childhood was more complex, particularly regarding the child temperament-quality of parenting interaction. This study lends support to the hypothesis of biological sensitivity to context (Boyce & Ellis, 2005) because it demonstrated that both high levels of negative maternal engagement (negative parenting) and low levels of negative maternal engagement (positive parenting) may interact with children's fearfulness to increase their likelihood of being *consistent* cortisol reactors. Furthermore, this analysis indicates that *consistent* cortisol reactors could endure positive or negative outcomes in later childhood. Thus, the present study illuminates the need to move beyond the assessment of cortisol reactivity patterns alone, and highlights the importance of including patterns of cortisol recovery when investigating the underlying mechanisms of various outcomes.

In particular, these findings underscored the importance including measure of cortisol recovery to early childhood research by revealing that positive maternal parenting qualities during infancy and toddlerhood are linked to *immediate* cortisol recovery. Since sensitive and responsive mothers are thought to act as external regulators for children (e.g., Bowlby, 1969; Ruff & Rothbart, 1996; Posner & Rothbart, 2000), these results indicate that *consistent* cortisol reactivity across early childhood may be adaptive as long as there is *immediate* cortisol recovery. Additionally, the present study suggests that prolonged cortisol reactivity (*consistent* cortisol reactivity with *delayed* recovery) associated with allostatic load may confer risk in early

childhood. However, in order directly test this hypothesis, longitudinal studies must be performed that assesses the associations among maternal parenting qualities, patterns of cortisol recovery, and mental and physical outcomes such as internalizing behavior problems and number of acute illnesses across early childhood.

In conclusion, the present study is the first step in trying to determine which patterns of cortisol reactivity and recovery during early childhood may result in later positive or negative outcomes in predominantly low-income, rural families. Moreover, this study provides evidence that children's temperamental characteristics such as fear and frustration, as well as maternal parenting qualities (positive and negative), during infancy and toddlerhood are associated differentially with patterns of cortisol reactivity and recovery across early childhood. Lastly, the present study highlights the need for future, longitudinal investigations that will allow developmental science to determine how the interplay among individual characteristics, environmental factors, and patterns of cortisol reactivity and recovery results in individuals' positive or negative outcomes across childhood and into adulthood.

## Appendix: Figures and Tables

Figure 1

*Infant Temperament Model – Hypothesized manner that infants’*

*temperament at 7-months of age relates to categories of cortisol*

*patterns across early childhood.*

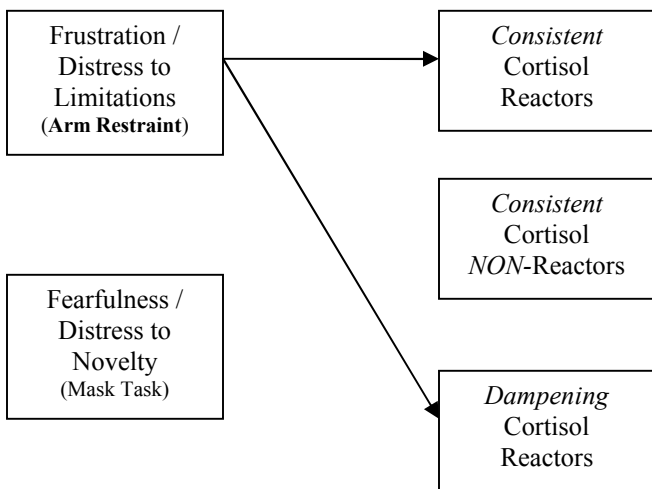


Figure 2  
*Toddler Temperament Model – Hypothesized manner that infants' temperament at 15-months of age relates to categories of cortisol patterns across early childhood.*

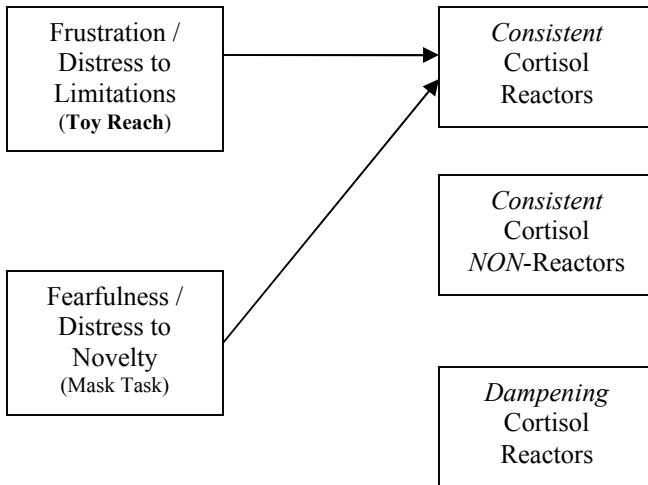




Figure 3

*Parenting Quality at Infancy Model – Hypothesized manner that mother's parenting qualities when infants are 7-months old relates to categories of cortisol patterns across early childhood.*

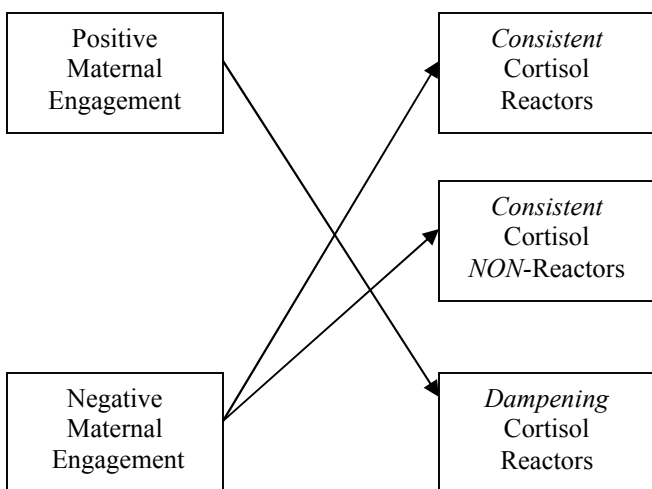


Figure 4  
*Parenting Quality at Toddlerhood Model – Hypothesized manner*  
*that mother's parenting qualities when infants are 15-months old*  
*relates to categories of cortisol patterns across early childhood.*

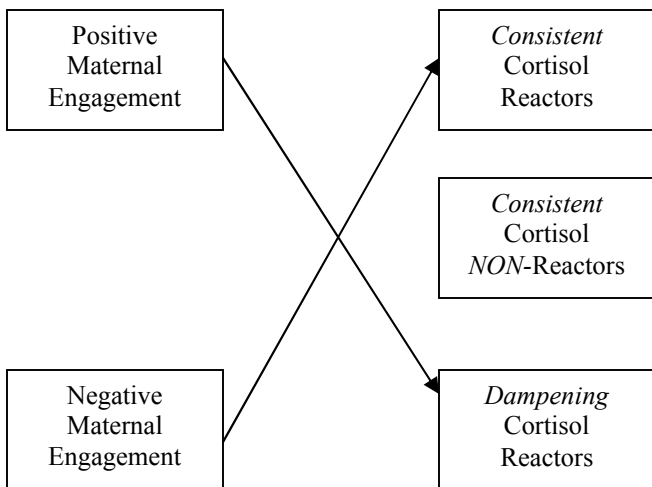


Figure 5  
*Infant Temperament and Parenting Quality Interaction Model – How the relationship between infants' temperament at 7-months of age and categories of cortisol patterns across early childhood is moderated by maternal parenting quality*

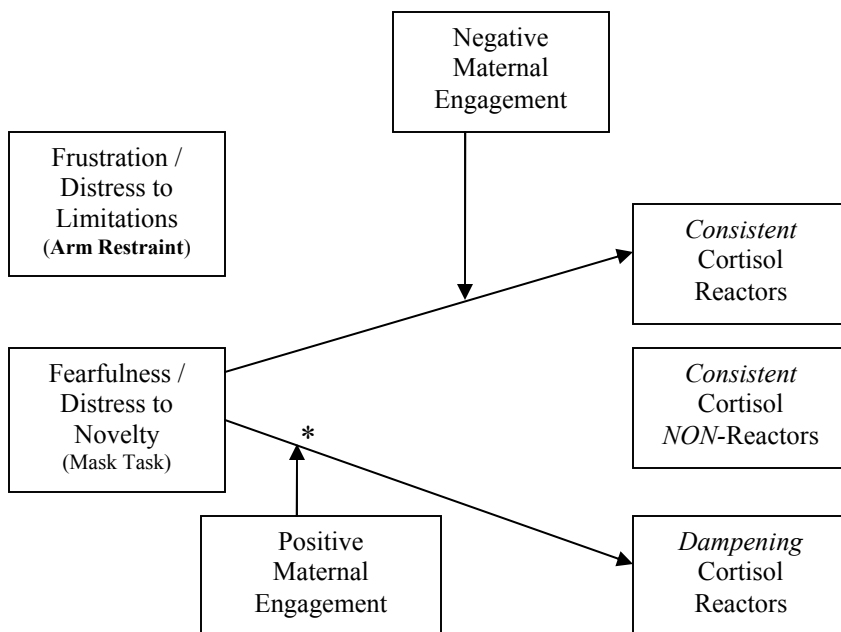


Figure 6  
*Toddler Temperament and Parenting Quality Interaction Model – How the relationship between infants' temperament at 15-months of age and categories of cortisol patterns across early childhood is moderated by maternal parenting quality*

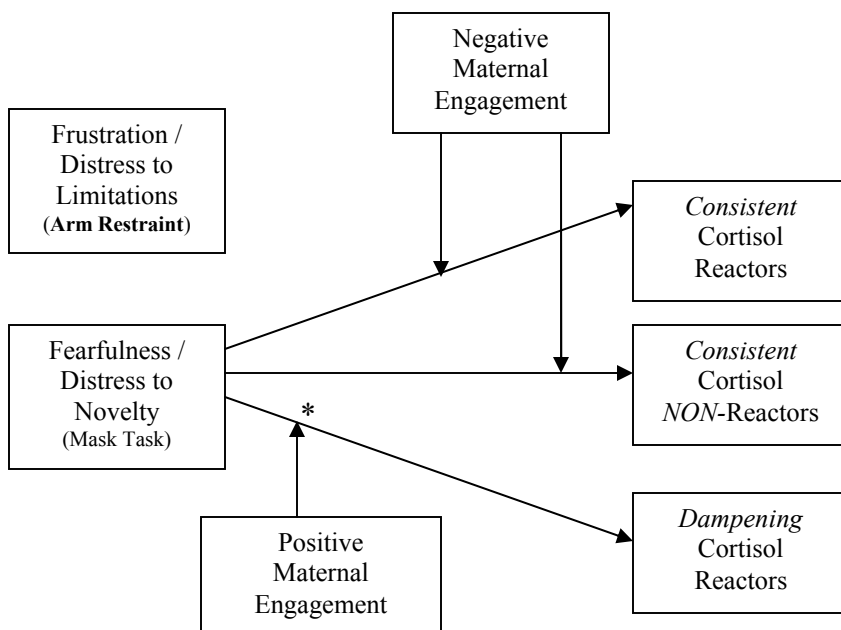


Figure 7

*During infancy, positive maternal parenting quality would be positively related to consistent and dampening immediate cortisol recoverers. Conversely, negative maternal parenting quality would be positively associated with consistent and dampening immediate cortisol recoverers.*

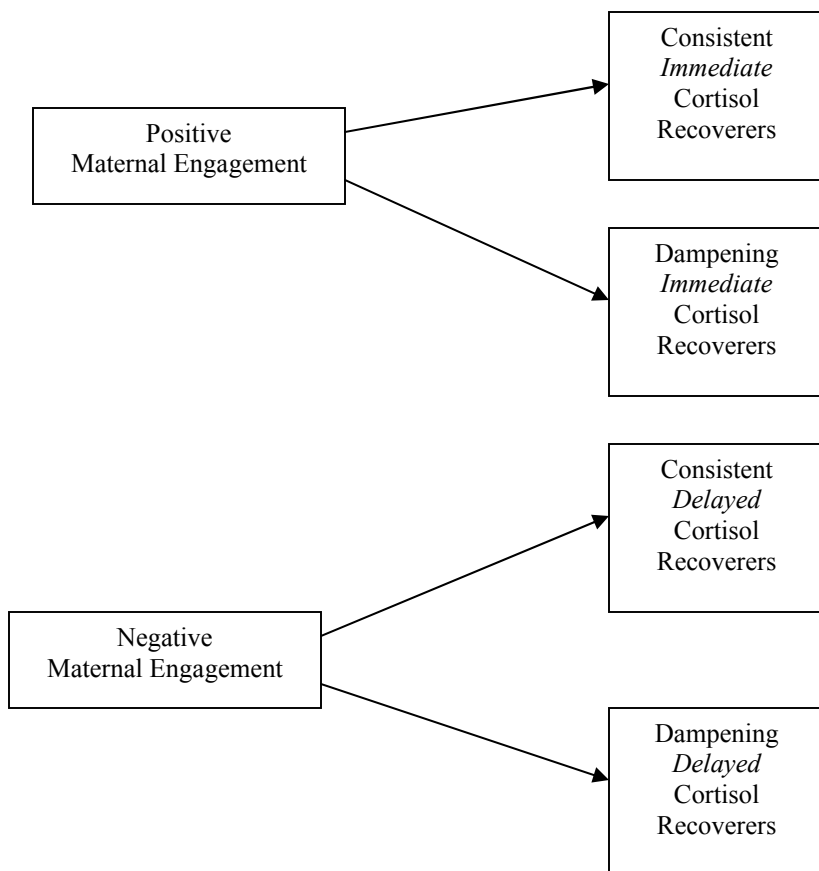


Figure 8

*During toddlerhood, positive maternal parenting quality would be positively related to consistent and dampening immediate cortisol recoverers.*

*Conversely, negative maternal parenting quality would be positively associated with consistent and dampening immediate cortisol recoverers.*

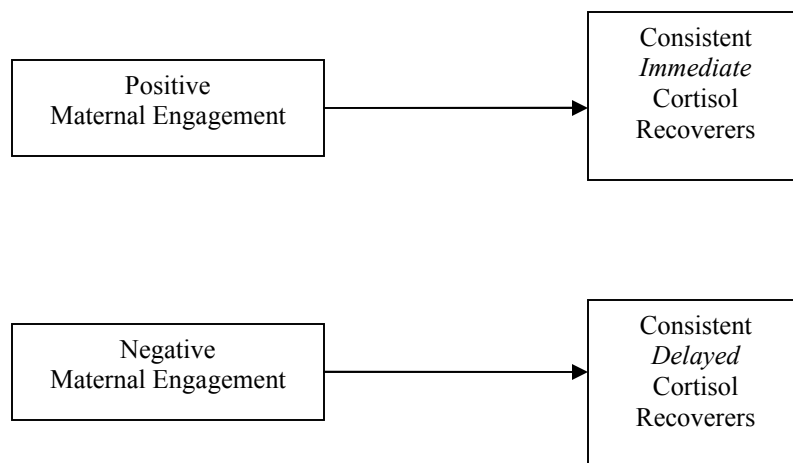


Table 1. Demographic, health-related, and cortisol characteristics of children by Cortisol Reactivity Group: Means (standard deviations) and percentages.

	Cortisol Reactivity Group across Infancy and Toddlerhood		
	Consistent Reactors (n= 179)	Consistent NON-Reactors (n=213)	Dampening Reactors (n=155)
White (%)	66.5	47.4**	62.6
Boys (%)	48.0	48.4	47.1
<b>Infant Visit</b>			
Age (months)	7.33 (1.21)	7.55 (1.15)	7.48 (1.19)
Low Income (%) <sup>a</sup>	63.0	68.2	58.7
Self- Reported Health <sup>b,c</sup>	1.59 (.82)	1.85 (1.00)	1.84 (1.00)
Body Temperature (°F)	98.4 (.62)	98.5 (.77)	98.3 (.80)
Hours Since Laying Down to Sleep <sup>c</sup>	5.24 (5.13)	6.51 (5.32)	5.69 (5.06)
Hours Since Eating <sup>c</sup>	1.56 (1.54)	1.39 (1.87)	1.63 (1.56)
Saliva Collection Time of Day	13:13 (2:37)	13:31 (3:05)	13:13 (2:49)
Pre-task Cortisol Levels (ug/dL)	.15 (.12)***	.27 (.25)***	.17 (.18)***
Cortisol Reactivity (% change) <sup>d</sup>	137.31 (177.45)***	-57.29 (22.75)***	142.52 (177.35)***
Absolute Differences in Cortisol <sup>e</sup>	.15 (.17)	.11 (.17)	.18 (.21)
<b>Toddler Visit</b>			
Age (months)	15.51 (1.10)	15.53 (1.04)	15.53 (1.04)
Low Income (%) <sup>a</sup>	63.0	72.3	62.5
Body Temperature (°F)	98.12 (.64)	98.32 (.70)	98.24 (.69)
Saliva Collection Time of Day	13:50 (2:48)	14:02 (3:06)	13:58 (3:01)
Pre-task Cortisol Levels (ug/dL)	.14 (.15)***	.21 (.20)***	.24 (.25)***
Cortisol Reactivity <sup>d</sup>	122.13 (140.64)***	-52.66 (21.01)***	-27.53 (21.30)***
Absolute Differences in Cortisol <sup>e</sup>	.15 (.18)	.07 (.11)	.09 (.21)Note: <sup>a</sup>

<sup>a</sup>Low Income defined by income-to-needs ratio less than 2.0. <sup>b</sup>Maternal reports of health were rated from 5 “poor” to 1 “excellent”. <sup>c</sup>Variables that were only collected at the Infant Visit, <sup>d</sup>Percent change from 20-min post-task to pre-task cortisol levels, controlling for pre-task levels [(20-min cortisol-baseline cortisol)/baseline cortisol]\*100], <sup>e</sup>Absolute difference scores from pre-task to 20-min post task. Two-tailed significance tests (independent Chi-square). \*\* p < .01; \*\*\*p<.001.

Table 2.

*Correlations among hypothesized predictors, child temperament and parenting, at 7- and 15-months.*

	Frustration (7-mos)	Fear (7-mos)	Positive Engage (7-mos)	Negative Engage (7-mos)	Frustration (15-mos)	Fear (15-mos)	Positive Engage (15-mos)	Negative Engage (15-mos)
<i>7-month assessment</i>								
Child Temperament								
Frustration (Arm Restraint)								
Fearfulness (Mask Task)	.18***							
Mother-child interactions								
Positive Engagement	.06	.06						
Negative Engagement	-.03	-.02	-.20***					
<i>15-month assessment</i>								
Child Temperament								
Frustration (Toy Reach)	.13*	.04	.06	.01				
Fearfulness (Mask Task)	-.01	.15**	-.07	.07	.05			
Mother-child interactions								
Positive Engagement	.06	.13**	.67***	-.19***	-.02	.09		
Negative Engagement	-.10*	-.05	-.17***	.39***	.07	.11*	-.28***	

\*\* p < .01; \*\*\*p<.001.



Table 3  
*Descriptive Statistics for Predictor Variables of Child Temperament and Quality of Maternal Parenting by Cortisol Reactivity Patterns at 7- and 15-months.*

Predictor	Cortisol Reactivity Patterns					
	Consistent Reactors		Dampening Reactors		Consistent NON-Reactors	
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
<i>7-month assessment</i>						
Child Temperament						
Frustration (Arm Restraint)	161	.74 (.67)	137	.80 (.68)	196	.61 (.61)
Fearfulness (Mask Task)	161	.22 (.46)	137	.16 (.37)	196	.11 (.30)
Mother-child interactions						
Positive Maternal Engagement	174	2.89 (.83)	150	3.09 (.71)	210	2.79 (.81)
Negative Maternal Engagement	174	2.33 (.74)	150	2.38 (.78)	210	2.41 (.79)
<i>15-month assessment</i>						
Child Temperament						
Frustration (Toy Removal)	145	.29 (.28)	113	.27 (.28)	166	.26 (.30)
Fearfulness (Mask Task)	145	.75 (.63)	113	.53 (.62)	166	.48 (.56)
Mother-child interactions						
Positive Maternal Engagement	157	2.84 (.81)	132	2.92 (.81)	191	2.67 (.80)
Negative Maternal Engagement	157	2.22 (.77)	132	2.22 (.69)	191	2.33 (.71)

Table 4  
*Likelihood Ratio Tests on Predictor Variables of Multinomial Logistic Regression Models Relating Child Temperament to Cortisol Reactivity Patterns at 7- and 15-months.*

Variable	$\chi^2$	<i>df</i>	<i>N</i>
<i>Infancy Temperament Model</i>			
Race (White vs. Black)	9.32**	2	422
Frustration during Arm Restraint	5.80*	2	422
Fearfulness during Mask Task	7.31*	2	422
<i>Toddlerhood Temperament Model</i>			
Race (White vs. Black)	14.08**	2	371
Frustration during Toy Removal	.77	2	371
Fearfulness during Mask Task	10.31**	2	371

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

Table 5  
*Parameter Estimates for the Infancy Temperament Model of the Multinomial Logistic Regression*  
*Associating Child Temperament to Cortisol Reactivity Patterns.*

Contrasts	Wald $\chi^2$	$\beta$	SE	Odds Ratio	Confidence Intervals
<i>Consistent vs Dampening Reactors</i>					
Race (White vs. Black)	<i>ns</i>				
Frustration during Arm Restraint	<i>ns</i>				
Fearfulness during Mask Task	5.21*	.77	.34	2.15	1.11 – 4.16
<i>Consistent vs Consistent NON-Reactors</i>					
Race (White vs. Black)	7.72**	.67	.24	1.96	1.22 – 3.15
Frustration during Arm Restraint	<i>ns</i>				
Fearfulness during Mask Task	5.16*	.70	.31	2.01	1.10 – 3.69
<i>Dampening vs Consistent NON-Reactors</i>					
Race (White vs. Black)	5.05*	.56	.25	1.74	1.07 -2.84
Frustration during Arm Restraint	5.67*	.69	.29	1.99	1.13 – 3.51
Fearfulness during Mask Task	<i>ns</i>				

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

Table 6  
*Parameter Estimates for the Toddlerhood Temperament Model of the Multinomial Logistic*  
*Regression Associating Child Temperament to Cortisol Reactivity Patterns.*

Contrasts	Wald $\chi^2$	$\beta$	SE	Odds Ratio	Confidence Intervals
<i>Consistent vs Dampening Reactors</i>					
Race (White vs. Black)	<i>ns</i>				
Frustration during Toy Removal Task	<i>ns</i>				
Fearfulness during Mask Task	<i>ns</i>				
<i>Consistent vs Consistent NON-Reactors</i>					
Race (White vs. Black)	9.92**	.82	.26	2.28	1.36 – 3.80
Frustration during Toy Removal Task	<i>ns</i>				
Fearfulness during Mask Task	9.78**	.92	.29	2.50	1.41 – 4.44
<i>Dampening vs Consistent NON-Reactors</i>					
Race (White vs. Black)	9.38**	.85	.28	2.34	1.36 – 4.06
Frustration during Toy Removal Task	<i>ns</i>				
Fearfulness during Mask Task	4.57*	.60	.32	1.89	.98 – 3.42

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

Table 7  
*Likelihood Ratio Tests on Predictor Variables of Multinomial Logistic Regression Models*  
*Relating Maternal Parenting Quality to Cortisol Reactivity Patterns at 7- and 15-months.*

Variable	$\chi^2$	df	N
<i>Parenting Quality in Infancy Model</i>			
Race (White vs. Black)	10.39**	2	531
Positive Maternal Engagement	9.84**	2	531
Negative Maternal Engagement	1.10	2	531

Table 8  
*Parameter Estimates for the Parenting Quality at Infancy Model of the Multinomial Logistic*  
*Regression Relating Quality of Maternal Parenting to Cortisol Reactivity Patterns.*

Contrasts	Wald $\chi^2$	$\beta$	SE	Odds Ratio	Confidence Intervals
<i>Consistent vs Dampening Reactors</i>					
Race (White vs. Black)	<i>ns</i>				
Positive Maternal Engagement	7.24**	-.33	.12	.72	.56 – .91
Negative Maternal Engagement	<i>ns</i>				
<i>Consistent vs Consistent NON-Reactors</i>					
Race (White vs. Black)	9.67**	.74	.26	2.10	1.32 – 3.35
Positive Maternal Engagement	<i>ns</i>				
Negative Maternal Engagement	<i>ns</i>				
<i>Dampening vs Consistent NON-Reactors</i>					
Race (White vs. Black)	3.94*	.49	.25	1.63	1.01 – 2.66
Positive Maternal Engagement	7.78**	.33	.12	1.40	1.10 – 1.80
Negative Maternal Engagement	<i>ns</i>				

Figure 9

*At the 15-month assessment, toddlers who are elicited high level of behavioral fearfulness during the mask task and have mother that demonstrate high levels of negative maternal engagement are more likely to be consistent reactors versus consistent non-reactors. Additionally, toddlers who demonstrate low levels of behavioral fearfulness and are exposed to low levels of harsh parenting also have a greater probability of being consistent reactors rather than consistent non-reactors.*

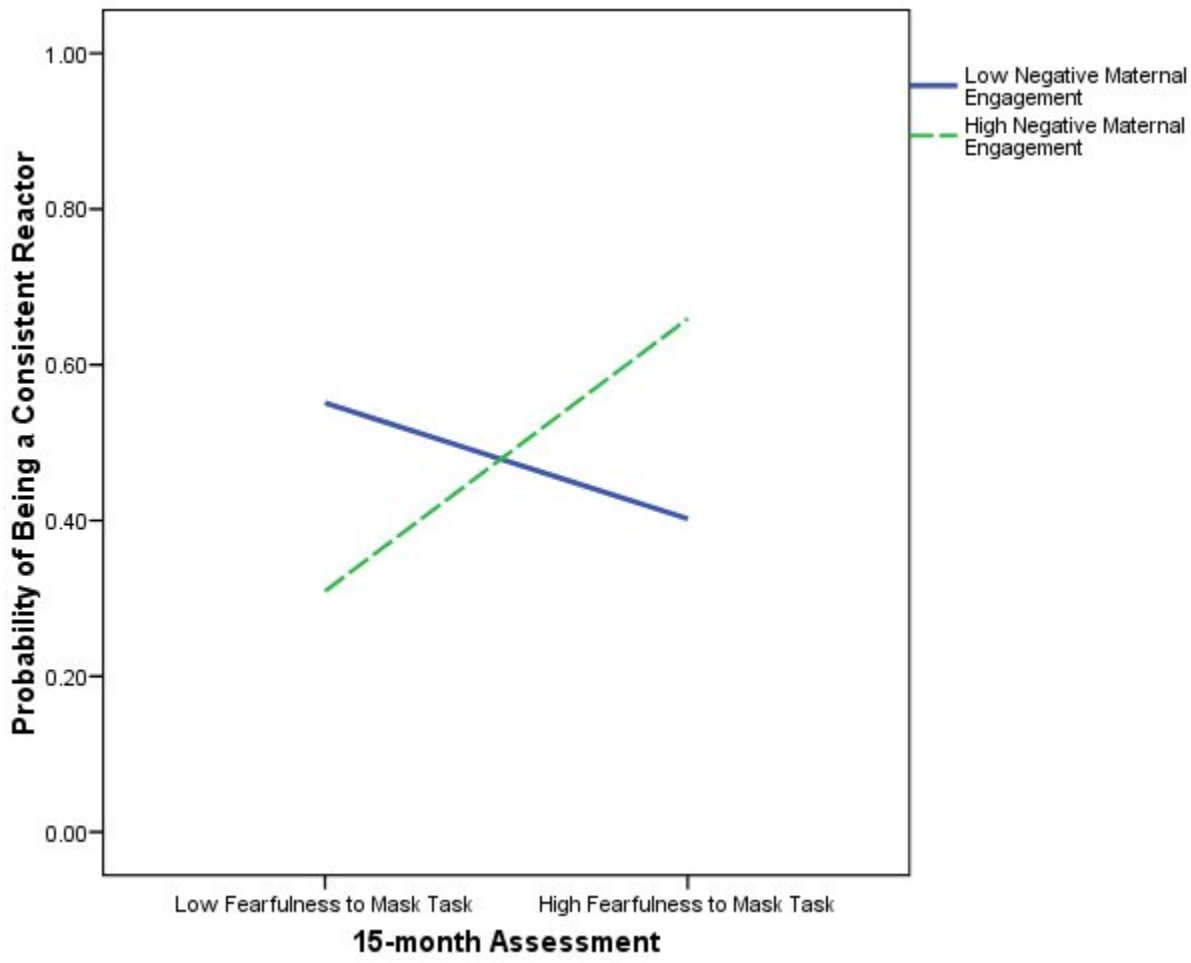


Table 9. Demographic characteristics by Cortisol Recoverer Group: Means (standard deviations) and percentages

	Cortisol Recovery Group across Infancy and Toddlerhood			
	Consistent <i>Immediate</i> Recovery (N=31)	Consistent <i>Delayed</i> Recovery (N=38)	Dampening <i>Immediate</i> Recovery (N=87)	Dampening <i>Delayed</i> Recovery (N=50)
White (%)	76.3	64.5	68.0	60.9
Boys (%)	52.6	51.6	43.7	44.0
<b>Infant Visit</b>				
Age (months)	6.99 (1.18)	7.00 (1.18)	7.43 (1.17)	7.48 (1.34)
Low Income (%) <sup>a</sup>	61.3	60.5	42.5	50.0
Self- Reported Health <sup>b,c</sup>	1.63 (.88)	1.67 (1.00)	1.76 (.98)	1.74 (.85)
Body Temperature (°F)	98.6 (.48)	98.4 (.65)	98.4 (.64)	98.4 (.78)
Hours Since Laying Down to Sleep <sup>c</sup>	7.04 (5.88)	7.30 (5.78)	5.88 (5.15)	5.64 (5.21)
Hours Since Eating <sup>c</sup>	2.11 (2.92)	1.73 (2.05)	1.65 (1.53)	1.42 (1.13)
Saliva Collection Time of Day	14:08 (2:45)	14:40 (3:21)	13:50 (2:55)	12:34 (2:39)
Cortisol Recovery (% change) <sup>d</sup>	-40.97 (18.49)**	27.36 (34.43)**	-34.37 (16.10)**	34.50 (62.01)**
<b>Toddler Visit</b>				
Age (months)	15.50 (1.05)	15.68 (1.12)	15.39 (0.96)	15.80 (1.08)
Low Income (%) <sup>a</sup>	67.4	65.2	67.1	61.9
Body Temperature (°F)	98.3 (.68)	98.5 (.74)	98.4 (.69)	98.6 (.54)
Saliva Collection Time of Day	12:56 (2:26)	13:16 (2:06)	13:44 (3:13)	14:08 (2:43)
Cortisol Recovery <sup>d</sup>	-33.61 (15.64)**	34.39 (35.55)**	----	----

Note: <sup>a</sup> Low Income defined by income-to-needs ratio less than 2.0. <sup>b</sup> Maternal reports of health were rated from 5 “poor” to 1 “excellent”. <sup>c</sup> Variables that were only collected at the Infant Visit, <sup>d</sup> Percent change from 40-min post-task to 20-min post-task cortisol levels, controlling for 20-min post-task levels:  $[(20\text{-min cortisol} - \text{baseline cortisol}) / \text{baseline cortisol}] * 100$ . \*\*p<.001.

Table 10  
*Descriptive Statistics for Predictor Variables of Child Temperament and Quality of Maternal Parenting by Cortisol Recovery Patterns at 7- and 15-months.*

Predictor	<i>Cortisol Recovery Patterns</i>							
	Consistent <i>Immediate</i> <u>Recoverers</u>		Consistent <i>Delayed</i> <u>Recoverers</u>		Dampening <i>Immediate</i> <u>Recoverers</u>		Dampening <i>Delayed</i> <u>Recoverers</u>	
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
<i>7-month assessment</i>								
Child Temperament								
Frustration (Arm Restraint)	27	.62 (.69)	35	.72 (.69)	78	.87 (.70)	41	.74 (.64)
Fearfulness (Mask Task)	27	.17 (.43)	35	.25 (.57)	78	.20 (.43)	41	.22 (.48)
Mother-child interactions								
Positive Engagement	30	2.54 (.96)	36	3.12 (.76)	82	3.13 (.72)	50	3.10 (.70)
Maternal Engagement	30	2.33 (.74)	36	2.48 (.87)	82	2.40 (.78)	50	2.37 (.80)
<i>15-month assessment</i>								
Child Temperament								
Frustration (Toy Removal)	29	.29 (.23)	31	.23 (.23)	61	.27 (.29)	38	.31 (.26)
Fearfulness (Mask Task)	29	.73 (.59)	31	.82 (.65)	61	.42 (.46)	30	.59 (.63)
Mother-child interactions								
Positive Engagement	27	2.63 (.85)	35	3.09 (.81)	76	2.97 (.81)	39	2.87 (.82)
Negative Engagement	27	2.25 (.68)	35	2.21 (.83)	76	2.17 (.66)	39	2.24 (.70)

#### Footnote

<sup>1</sup>Cortisol production follows a well-recognized, non-linear diurnal pattern, which begins to appear during the first year of life (de Weerth, Zijl, & Buitelaar, 2003). Levels rise early in the AM, decline rapidly before midday, and then decline slowly through the afternoon and evening. More recent work suggests that the diurnal rhythm continues to develop well into the preschool years, when afternoon naps become less common (Gunnar & Donzella, 2002). Specifically, the expected decreases in cortisol from late morning to late afternoon are not observed in children until around 4 years of age. Curve estimations were run to determine the relationship between sampling time of day and maternal and infant cortisol levels. As expected, in infants sampling time of day explained a relatively smaller portion of the variance [quadratic  $Rsq=.04$ , linear:  $Rsq=.02$ ] than for mothers [quadratic:  $Rsq=.15$ , linear:  $Rsq=.15$ ].

## References

- Adam, E. K., Klimes-Dougan, B., & Gunnar, M. R. (2007). Social regulation of the adrenocortical response to stress in infants, children and adolescents: Implications for psychopathology and education. In D. Coch, G. Dawson, & K. Fischer (Eds.), *Human behavior, learning, and the developing brain: Atypical development* (pp. 264–304). New York: Guilford Press.
- Ainsworth, M.S., Blehar, M.C., Waters, E., & Wall, S. (1978). Patterns of Attachment: A Psychological Study of the Strange Situation. XVIII, p 391. Lawrence Erlbaum: Oxford, England.
- Albers, E.M., Riksen-Walraven, J.M., Sweep, F., & de Weerth, C. (2008). Maternal behavior predicts infant cortisol recovery from a mild everyday stressor. *Journal of Child Psychology and Psychiatry*, 49, 97-103
- Ashman, S. B., Dawson, G., Panagiotides, H., Yamada, E., & Wilkins, C. W. (2002). Stress hormone levels of children of depressed mothers. *Developmental Psychopathology*, 14, 333 – 349.
- Bayart, F., Hayashi, K. T., Faull, K. F., Barchas, J. D., Levine, S., 1990. Influence of maternal proximity on behavioral and psychological responses to separation in infant rhesus monkeys (*Macaca mulatta*). *Behavioral Neuroscience*, 104, 98–107.
- Beckwith, L., Rozga, A., & Sigman, M. (2002). Maternal sensitivity and attachment in atypical groups. *Advances in child development and behavior*, Vol. 30. (pp. 231-274). San Diego, CA, US: Academic Press.
- Belsky, J. (1984). The determinants of parenting: A process model. *Child Development*, 55, 83-

96.

- Black, J., Jones, T.A., Nelson, C. A., & Greenough, W T. (1998). Neuronal plasticity and the developing brain. In N. E. Alessi, J. T. Coyle, S. I. Harrison, & S. Eth (Eds.), *Handbook of child and adolescent psychiatry* (pp. 31-53) New York: Guilford Press.
- Blair, C., Granger, D.A., Kivlighan, K.T., Willoughby, M., Greenberg, M.T., Hibel, L., Fortunato, C. & the Family Life Project Investigators (2008). Maternal and child contributions to cortisol response to emotional arousal in young children from low-income, rural communities. *Developmental Psychology*, 44, 1095-1109.
- Boomershine C. S., Wang T., & Zwilling B. S. (2001). Neuroendocrine regulation of macrophage and neutrophil function. In: Ader R, Felten DL, Cohen N, editors. *Psychoneuroimmunology*, 3rd ed. New York: Academic Press; 2001:289–300.
- Bowlby, J. (1969). *Attachment and loss: Vol. 1. Attachment*. New York: Basic Books.
- Boyce, W. T. & Ellis, B. J. (2005). Biological sensitivity to context: I. An evolutionary-developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology*, 17, 271-301.
- Braungart, J. & Stifter, C. A. (1991). Regulation of negative reactivity during the Strange Situation: Temperament and attachment in 12-month-old infants. *Infant Behavior and Development*, 14, 349-364.
- Braungart-Rieker, J., & Stifter, C. A. (1996). Infants' responses to frustrating situations: Continuity and change in reactivity and regulation. *Child Development*, 67, 1767–1769.
- Bronfenbrenner, U., & Morris, P. (1998). The ecology of developmental processes. In W. Damon (Series Ed.) & R. M. Lerner (Vol. Ed.), *Theoretical models of human development: Vol. 1. Handbook of child psychology* (5th ed., pp. 993–1028). New York:



Wiley.

- Bullock, W. A., & Gilliland, K. (1993). Eysenck's arousal theory of introversion-extroversion: A converging measures investigation. *Journal of Personality and Social Psychology, 64*, 113-123.
- Burleson, M. H., Poehlmann, K. M., Hawkley, L. C., Ernst, Berntson, G. G., Malarkey, W. B., Kiecolt-Glaser, J.K., Glaser, R., & Cacioppo, J.T. (2003). Neuroendocrine and cardiovascular reactivity to stress in mid-aged and older women: Long-term temporal consistency of individual differences. *Psychophysiology, 40*, 358–369.
- Buss, K. & Goldsmith, H. (1998). Fear and anger regulation in infancy: Effects on the temporal dynamics of affective expression. *Child Development, 69*, 359-374.
- Buss, K. A., Malmstadt Schumacher, J., Dolski, I., Kalin, N. H., Gold-smith, H. H., & Davidson, R. J. (2003). Right frontal brain activity, cortisol, and withdrawal behavior in 6-month-old infants. *Behavioral Neuroscience, 117*, 11–20.
- Cacioppo, J. T., Berntson, G. G., Malarkey, W. B., Kiecolt-Glaser, J. K., Sheridan, J. F., Poehlmann, K. M., Burleson, M. H., Ernst, J. M., Hawkley, L. C., & Glaser, R. (1998). Autonomic, neuroendocrine, and immune responses to psychological stress: The reactivity hypothesis. *Annals of the New York Academy of Sciences, 840*, 664-673.
- Cairns, R. B., Costello, E. J., & Elder, J. H., Jr. (1996). The making of developmental science. In R.B. Cairns, G. H. Elder, Jr., E. J. Costello (Eds). *Developmental science. Cambridge studies in social and emotional development* (pp. 223-234). New York, NY: Cambridge University Press.
- Caldwell, B. M., & Bradley, R. H. (1984). *Home observation for measurement of the environment* (Rev. ed.) Little Rock: University of Arkansas.

- Carlson, V. J., & Harwood, R. L. (2003). Attachment, culture, and the caregiving system: The cultural patterning of everyday experiences among Anglo and Puerto Rican mother – infant pairs. *Infant Mental Health Journal, 24*, 53–73.
- Cohen, J. (1983). The cost of dichotomization. *Applied Psychological Measurement, 7*, 249-253.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.
- Cohen, S., & Hamrick, N. (2003). Stable individual differences in physiological response to stressors: implications for stress-elicited changes in immune related health. *Brain, Behavior, & Immunology, 17*, 407-414.
- Cohen, S., Hamrick, N., Rodriguez, M. S., Feldman, P. J., Rabin, B. S., & Manuck, S. B. (2002). Reactivity and vulnerability to stress-associated risk for upper respiratory illness. *Psychosomatic Medicine, 64*, 302–310.
- Cox, M. J., Paley, B., Burchinal, M., & Payne, C. C. (1999). Marital perceptions and interactions across the transition to parenthood. *Journal of Marriage and the Family, 61*, 611–625.
- Cox, M. J., Paley, B., & Harter, K. (2001). Interparental conflict and parent-child relationships. In J. Grych & F. Fincham (Eds.), *Child development and interparental conflict* (pp. 249-272). New York: Cambridge University Press.
- Curtis, W. J. & Cicchetti, D. (2003). Moving research on resilience into the 21<sup>st</sup> century: Theoretical and methodological considerations in examining the biological contributors to resilience. *Development and Psychopathology, 15*, 773-810.
- Davidson, R. J. (2001). Toward a biology of personality and emotion. In A. R. Damasio, A. Harrington, J. Kagan, B. McEwen, H. Moss, & R. Shaikh (Eds.), *Annals of the New York*

- Academy of Sciences: Vol. 935. Unity of knowledge: The convergence of natural and human science* (pp. 191–207). New York: New York Academy of Sciences.
- Davis, E. P., Donzella, B., Krueger, W. K., & Gunnar M. R. (1999). The start of a new school year: individual differences in salivary cortisol response in relation to child temperament. *Developmental Psychobiology, 35*, 188–196.
- de Haan, M., Gunnar, M. R., Tout, K., Hart, J., & Stansbury, K. (1998). Familiar and novel contexts yield different associations between cortisol and behavior among 2-year-olds. *Developmental Psychobiology, 31*, 93–101.
- Derogatis, L. R. (1993). *Brief Symptom Inventory: Administration, Scoring & Procedures Manual*. Minneapolis: National Computer Systems.
- Derogatis, L. R. (1994). *SCL-90-R: Administration, Scoring and Procedures Manual*. Minneapolis: National Computer Systems, Inc.
- Derogatis, L. R. (2000). *Brief Symptom Inventory 18*. Minneapolis, MN: NCS Pearson, Inc.
- Dettling, A. C., Parker, S., Lane, S. K., Sebanc, A. M., & Gunnar, M. R., (2000). Quality of care and temperament determine whether cortisol levels rise over the day for children in full-day childcare. *Psychoneuroendocrinology, 25*, 819–836.
- deWeerth, C., & Buitelaar, J. K. (2005). Physiological stress reactivity in human pregnancy—a review. *Neuroscience & Biobehavioral Reviews, 29*, 293–310.
- 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.
- Dienstbier, R.A. (1989). Arousal and physiological toughness: implications for mental and physical health. *Psychological Review, 96*, 84–100.

- Donzella, B., Gunnar, M. K., Krueger, W. K., & Alwin, J. (2000). Cortisol and vagal tone responses to competitive challenge in preschoolers: Associations with temperament. *Developmental Psychobiology, 37*, 209–220.
- Earle, T. L., Linden, W., & Weinberg, J. (1999). Differential effects of harassment on cardiovascular and salivary cortisol stress reactivity and recovery in women and men. *Journal of Psychosomatic Research, 46*, 125-141.
- Egeland, B., Pianta, R., & O'Brien, M. A. (1993). Maternal intrusiveness in infancy and child maladaptation in early school years. *Development and Psychopathology, 5*, 359–370.
- Egliston, K.A., McMahon, C., Austin, M.P., 2007. Stress in pregnancy and infant HPA axis function: conceptual and methodological issues relating to the use of salivary cortisol as an outcome measure. *Psychoneuroendocrinology, 32*, 113.
- 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.
- Evans, G. W. (2003). A multimethodological analysis of cumulative risk and allostatic load among rural children. *Developmental Psychology, 39*, 924 –933.
- Evans, G. W. (2004). The environment of childhood poverty. *American Psychologist, 59*, 77-92.
- Field T (1994): The effects of mother's physical and emotional unavailability on emotion regulation. In: Fox NA, editor. *The Development of Emotion Regulation*. Chicago: University of Chicago Press, 208-227.
- Fisher, P.A., Burraston, B., & Pears, K.C. (2005). The early intervention foster care program: permanent placement outcomes from a randomized trial. *Child Maltreatment, 10*, 61–71.
- Friese, E., Hesse, J., Hellhammer, J., & Hellhammer, D. (2005). A new view on hypocortisolism.

- Psychoneuroendocrinology*, 30, 1010–1016.
- Gold, P. W., & Chrousos, G. P., (2002). Organization of the stress system and its dysregulation in melancholic and atypical depression: high vs low CRH/NE states. *Molecular Psychiatry*, 7, 254–275.
- Goldhaber, D. E. (1999). *Theories of human development: Integrative perspectives*. Mountain View, CA: Mayfield.
- Goldsmith, H., Buss, A., Plomin, R., Rothbart, M., Tomas, A., Chess, S., Hind, R., & McCall, R. (1987). Roundtable: What is temperament? Four approaches. *Child Development*, 58, 505-529.
- Goldsmith, H. H., & Harman, C. (1994). Temperament and attachment: Individuals and relationships. *Current Directions in Psychological Science*, 3, 53–57.
- Gottlieb, G. (1992). *Individual development and evolution*. The genesis of novel behavior. Oxford University Press.
- Gottlieb, G., Johnston, T. D., & Scoville, R. P. (1982). Conceptions of development and the evolution of behavior. *Behavioral and Brain Sciences*, 5, 284.
- Granger, D. A., Kivlighan, K. T., Fortunato, C. K., Harmon, A. G., Hibel, L. C., Schwartz, E. B., & Whembolua, G. L. (2007). Integration of salivary biomarkers into developmental and behaviorally oriented research: Problems and solutions for collecting specimens. *Physiology & Behavior*, 92, 583-590.
- Granger, D. A., Stansbury, K., & Henker, M. (1992, August). *Relations between behavioral style and behavioral and neuroendocrine responses to social challenge in preschoolers*. Presented at the XXIII Congress of the International Society of Psychoneuroendocrinology, Madison, WI.

- Granger, D., Weisz, J., & Kauneckis, D. (1994). Neuroendocrine reactivity, internalizing behavior problems, and control-related cognitions in clinic referred children and adolescents. *Journal of Abnormal Psychology, 103*, 267-276.
- Granger, D. A., Weisz, J. R., McCracken, J. T., Ikeda, S. C., & Douglas, P. (1996). Reciprocal influences among adrenocortical activation, psychosocial processes, and the behavioral adjustment of clinic-referred children. *Child Development, 67*, 3250-3262.
- Greenough, W., & Black, J. (1992). Induction of brain structure by experience: Substrates for cognitive development. In M. Gunnar & C. A. Nelson (Eds.) *Developmental and Behavioral Neuroscience*. New York: Oxford University Press.
- Gunnar, M.R., (1990). *The Psychobiology of Infant Temperament*. Erlbaum, Hillsdale, NJ.
- Gunnar, M. R., Brodersen, L., Krueger, K., & Rigatuso, J. (1996). Dampening of adrenocortical responses during infancy: Normative changes and individual differences. *Child Development, 67*, 877-889.
- Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology, 27*, 199-220.
- Gunnar M. R., Larson M. C., Hertsgaard, L., Harris M. L., & Brodersen, L. (1992). The stressfulness of separation among nine-month-old infants: effects of social context variables and infant temperament. *Child Development, 63*, 290–230.
- 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.R., Sebanc, A.M., Tout, K., Donzella, B., & van Dulmen, M. H. (2004). Peer rejection, temperament, and cortisol activity in preschoolers. *Developmental Psychobiology, 43*, 346 – 358.

- Gunnar, M. R., Tout, M., de Haan, Pierce, S., & Stansbury, K. (1997). Temperament, Social Competence, and Adrenocortical Activity in Preschoolers. *Development and Psychobiology, 3*, 65-85.
- Gunnar, M. R., & Quevedo, K., (2006). The neurobiology of stress and development. *Annual Review in Psychology, 58*, 145-174.
- Gunnar, M. R., & Vazquez, D. M. (2001). Low cortisol and a flattening of expected daytime rhythm: potential indices of risk in human development. *Development and Psychopathology, 13*, 515-538.
- Gunnar, M., & Vazquez, D. (2006). Stress neurobiology and developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Vol. 2. Developmental neuroscience* (2nd ed., pp. 533–577). Hoboken, NJ: Wiley.
- Hamilton, L.C. & Seyfrit, C.L. (1994). Resources and hopes in New Foundland. *Society and Natural Resources, 7*, 561-578.
- Harmon, A. G., Granger, D. A., Hibel., L C., & Rumyansteva, O. (2007). Measuring salivary cortisol in studies of child development: Watch out: What goes in my not come out of saliva collection devices. *Developmental Psychobiology, 49*, 495.
- Hibel, L. C., Granger, D. A., Kivlighan, K. T., Blair, C., & The Family Life Project Investigators (2006). Individual differences in salivary cortisol: Effects of common over the counter and prescription medications in infants and their mothers. *Hormones and Behavior, 50*, 293-300.
- Hellhammer, D.H., & Wade, S., (1993). Endocrine correlates of stress vulnerability. *Psychotherapy Psychosomatic, 60*, 8–17.
- Hessl, D., Dawson, G., Frey, K., Panagiotides, H., Self, H., Yamada, E., & Osterling, J. (1998). A longitudinal study of children of depressed mothers: Psychobiological findings related

- to stress. In: Hann DM, Huffman LC, Lederhendler KK, Minecke D, editors. *Advancing Research on Developmental Plasticity: Integrating the Behavioral Sciences and the Neurosciences of Mental Health*. Bethesda, MD: National Institutes of Mental Health.
- Heim, C., Ehlert, U., & Hellhammer, D. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology*, 25, 1–35.
- Hosmer, D. W., & Lemeshow, S. (1989). *Applied Regression*. New York: John Wiley & Sons.
- Huttenlocher, P. R. (2002). *Neural plasticity: The effects of experience on the development of the cerebral cortex*. Cambridge, MA: Harvard University Press.
- Isabella, R. A., & Belsky, J. (1991). Interactional synchrony and the origins of infant-mother attachment: A replication study. *Child Development*, 62, 373-384.
- Kagan, J. (1994). *Galen's Prophecy*. New York: Basic Books.
- Kagan, J., Reznick, J. S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. *Child Development*, 58, 1459–1473.
- Kagan, J., Snidman, N., & Arcus, D. (1998). Childhood derivatives of high and low reactivity in infancy. *Child Development*, 69, 1483-1493.
- Kagan, J., Snidman, N., McManis, M., Woodward, S., & Hardway, C. (2002). One measure, one meaning: Multiple measures, clearer meaning. *Development and Psychopathology*, 14, 463–475.
- Kaufman, J., Plotsky, P. M., Nemeroff, C. B., & Charney, D. S. (2000). Effects of early adverse experiences on brain structure and function: Clinical implications. *Biological Psychiatry*, 48, 788-790.
- Keenan, K., Grace, D., & Gunthorpe, D. (2003). Examining stress reactivity in neonates:



- Relations between cortisol and behavior. *Child Development*, 74, 1930 – 1942.
- Kirschbaum, C., Prussner, J.D., Stone, A.A., Federenko, I., Gaab, J., Lintz, D., Schommer, H.J., & Hellhammer, D.H. (1995). Persistent high cortisol responses to repeated psychology distress in a subpopulation of healthy men. *Psychosomatic Medicine*, 57, 469–474.
- Kirschbaum, C. & Hellhammer, D. H. (1989). Salivary cortisol in psychobiological research: an overview. *Neuropsychobiology*, 22, 150-169. Review.
- Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research: Recent developments and applications. *Psychoneuroendocrinology*, 19, 313–333.
- Kochanska, G. (1995). Children's temperament, mothers' discipline, and security of attachment: Multiple pathways to emerging internalization. *Child Development*, 66, 597-615.
- Kochanska, G., Tjebkes, T. & Forman, D. (1998). Children's emerging regulation of conduct: restraint, compliance, and internalization from infancy to the second year. *Child Development*, 69, 1378-1389.
- Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: a review and methodologic critique. *Psychological Bulletin*, 96, 435-464.
- 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.
- Lashansky, G., Saenger, P., Fishman, K., Gautier, T., Mayes, D., Berg, G., Di Martino-Nardi, J. & Reiter, E. (1991) Normative data for adrenal steroidogenesis in a healthy pediatric

- population. *Journal of Clinical Endocrinology and Metabolism*, 73, 674–86.
- Levine, S., & Wiener, S. G. (1988). Psychoendocrine aspects of mother-infant relationships in nonhuman primates. *Psychoneuroendocrinology*, 13, 143-154.
- Linden, W., Earle, T. L., Gerin, W., & Christenfeld, N. (1997). Physiological stress reactivity and recovery: conceptual siblings separated at birth? *Journal of Psychosomatic Research*, 42, 117-135.
- Liu, D. (1997). Maternal care, hippocampal glucocorticoid receptor gene expression and hypothalamic -pituitary-adrenal responses to stress. *Science*, 277, 1659-1662.
- Loken, E. (2004). Using latent class analysis to model temperament types. *Multivariate Behavioral Research*, 39,625 -652.
- MacCallum, R. C., Zhang, S., Preacher, K. J., & Rucker, D. D. (2002). On the practice of dichotomization of quantitative variables. *Psychological Methods*, 7, 19–40.
- Maccoby, E. E., & Martin, J. A. (1983). Socialization in the context of the family: Parent-child interaction. In P. H. Mussen (Series Ed.) & E. M. Hetherington (Vol. Ed.), *Handbook of child psychology: Vol. 4. Socialization, personality, and social development* (4th ed., pp. 1 -101). New York: Wiley.
- Magnusson, D., & Cairns, R. B. (1996). Developmental science: Toward a unified framework. In R. B. Cairns & G. H. Elder (Eds.), *Developmental science* (pp. 7–30). New York: Cambridge University Press.
- Makino, S., Gold, P. W., & Schulkin, J. (1994). Corticosterone effects on corticotropin-releasing hormone RNA in the central nucleus of the amygdala and the parvocellular region of the paraventricular nucleus of the hypothalamus. *Brain Research*, 640, 105–112.

- McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, 338, 171-179.
- McEwen, B. S. (2000). The neurobiology of stress: From serendipity to clinical relevance. *Brain Research*, 886, 172–189.
- McEwen, B. S., & Seeman, T. E. (1999). Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. In N. E. Adler, M. Marmot, B. S. McEwen, & J. Stewart (Eds.), *Socioeconomic status and health in industrial nations* (pp. 30–47). New York: New York Academy of Sciences.
- McEwen, B.S., & Stellar, E. (1993). Stress and the individual. Mechanisms leading to disease. *Archives of Internal Medicine*, 153, 2093-2101.
- Meaney, M. J., Diorio, J., Widdowson, J., LaPlante, P., Caldji, C., Seckl, J. R., & Plotsky, P. M. (1996). Early environmental regulation of forebrain glucocorticoid receptor gene expression: Implications for adrenocortical responses to stress. *Developmental Neuroscience*, 18, 49-72.
- Meaney, M. J., & Szyf, M. (2005). Maternal care as a model for experience-dependent chromatin plasticity? *Trends in Neurosciences*, 28, 456–463.
- Nachmias, M., Gunnar, M., Mangelsdorf, S., Parritz, R., & Buss, K. (1996). Behavioral inhibition and stress reactivity: Moderating role of attachment security. *Child Development*, 67, 508–522.
- NICHD Early Child Care Research Network. (1997). The effects of infant child care on infant-mother attachment security: Results of the NICHD Study of Early Child Care. *Child Development*, 68, 860-879.
- NICHD Early Child Care Research Network. (1999). Child care and mother-child interaction in

- the first three years of life. *Developmental Psychology*, 35, 1399-1413.
- NICHD Early Child Care Research Network. (2003). Early child care and mother-child interaction from 36 months through first grade. *Infant Behavior and Development*, 26, 345-370.
- Posner, M. I., & Rothbart, M. K. (2000). Developing mechanisms of self-regulation. *Development and Psychopathology*, 12, 427-441.
- Putnam, S. P., & Stifter, C.A. (2005). Behavioral approach-inhibition in toddlers: Prediction from infancy, positive and negative affective components, and relations with behavior problems. *Child Development*, 76, 212-226.
- Quas, J., Hong, M., Alkon, A., & Boyce, W. T. (2000). Dissociations between psychobiologic reactivity and emotional expression in children. *Developmental Psychobiology*, 37, 153-175.
- Raine, A. (1996). *Autonomic nervous system activity and violence*. In D. M. Stoff & R. B. Cairns (Eds.), *Aggression and violence. Genetic, neurobiological and biological perspectives* (pp. 145-168). Mahwah, NJ: Erlbaum.
- Ramsay, D. S., & Lewis, M. (1994). Developmental changes in infant cortisol and behavioral stress response to inoculation. *Child Development*, 65, 1491-1502.
- Ramsay, D., & Lewis, M. (2003). Reactivity and regulation in cortisol and behavioral responses to stress. *Child Development*, 74, 456 - 464.
- Repetti, R., Taylor, S., & Seeman, T. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychology Bulletin*, 128, 330-366.
- Rose, R. M. (1980). Endocrine responses to stressful psychological events. *Advanced Psychoneuroendocrinology: Psychiatry*, 3, 251-276.

- Rosenfield, P., Suchecki, D., & Levine, S., (1992). Multifactorial regulation of the hypothalamic–pituitary–adrenal axis during development. *Neuroscience Biobehavior Review*, *16*, 553–568.
- Rothbart, M. K. (1986). Longitudinal observation of infant temperament. *Developmental Psychology*, *22*, 356-365.
- Rothbart, M. K. (1988). Temperament and the development of inhibited approach. *Child Development*, *59*, 1241-1250.
- Roy, M. P., Kirschbaum, C., & Steptoe, A., (2001). Psychological cardiovascular and metabolic correlates of individual differences in cortisol stress recovery in young men. *Psychoneuroendocrinology*, *26*, 375–391.
- Ruff, H. A., & Rothbart, M. K. (1996). *Attention in early development: Themes and variations*. New York: Oxford University Press.
- Sapolsky, R. M. (1991). Testicular function, social rank and personality among wild baboons. *Psychoneuroendocrinology*, *16*, 281–293.
- Sapolsky, R. M., & Meaney, M. J. (1986): Maturation of the adrenocortical stress response: Neuroendocrine control mechanisms and the stress hyporesponsive period. *Brain Research*, *396*, 64 –76.
- Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews*, *21*, 55–89.
- Scarr, S., & Salapatek, P. (1970). Patterns of fear development during infancy. *Merrill-Palmer Quarterly*, *16*, 53-90.
- 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.
- Schore, A. N. (2001). Effects of a secure attachment relationship on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal, 22*, 7 – 66.
- Shirtcliff, E. A., Zahn-Waxler, C., & Klimes-Dougan, B. (April, 2005). Cortisol reactivity during social challenge is related to stress and coping strategies in at risk adolescents. *Society for Research in Child Development*, Atlanta, Georgia.
- Smeekens, S., Riksen-Walraven, J. M., & van Bakel, H. J. A. (2007). Multiple determinants of externalizing behavior in 5 year-olds: A longitudinal model. *Journal of Abnormal Child Psychology, 35*, 342-361.
- Snoek, H., Van Goozen, S. H. M., Matthys, W., Sigling, H. O., Koppeschaar, H. P. F., Westenberg, H. G. M., et al. (2002). Serotonergic functioning in children with oppositional defiant disorder: A sumatriptan challenge study. *Biological Psychiatry, 51*, 319–325.
- Soloff, P. H., Lynch, K. G., & Moss, H. B. (2000). Serotonin, impulsivity, and alcohol use disorders in the older adolescent: A psychobiological study. *Alcoholism, Clinical and Experimental Research, 24*, 1609–1619.
- Spangler, G., & Schieche, M. (1998). Emotional and adrenocortical responses of infants to the strange situation: The differential function of emotional expression. *International Journal of Behavioral Development, 22*, 681–706.
- Spangler, G. and Grossman, K. E. (1993) Biobehavioral organization in securely and insecurely attached infants. *Child Development, 64*, 1439–1450.
- Sroufe, L. A. (1979). Socioemotional development. In J. Osofsky (Ed.), *Handbook of infant*

- development* (pp. 462-516). New York: Wiley,
- Sterling, P., & Eyer, J. (1988). Allostasis: A new paradigm to explain arousal pathology, in: Fisher, S., Reason, J. (Eds.), *Handbook of Life Stress, Cognition, and Health*, Wiley, New York.
- Stifter, C. A., & Braungart, J. M. (1995). The regulation of negative reactivity in infancy: Function and development. *Developmental Psychology*, *31*, 448-455.
- Stifter, C. A., & Fox, N. A. (1990). Infant reactivity: Physiological correlates of newborn and five-month temperament. *Developmental Psychology*, *26*, 582-588.
- Stifter, C. A., & Jain, A. (1996). Psychophysiological correlates of infant temperament: Stability of behavior and autonomic patterning from 5 to 18 months. *Developmental Psychobiology*, *29*, 379-391.
- Stifter, C. A., & Spinrad, T. L. (2002). The effect of excessive crying on the development of emotion regulation. *Infancy*, *3*, 133 – 152.
- Susman, E. J. (2006). Psychobiology of persistent antisocial behavior: Stress, early vulnerabilities and the attenuation hypothesis. *Neuroscience and Biobehavioral Reviews*, *30*, 376–389.
- Teicher MN, Andersen SL, Polcari A, Anderson CM, Navalta CP, Kim DM (2003): The neurobiological consequences of early stress and childhood maltreatment. *Neuroscience and Biobehavioral Reviews*, *27*, 33-44.
- Thompson, R. F. (2000). *The brain: A neuroscience primer, 3rd ed.* New York, NY: Worth Publishers.
- Tout, K., de Haan, M., Kipp-Campbell, E., & Gunnar, M. R. (1998). Social behavior

- correlates of cortisol activity in child care: Gender differences and time-of-day effects. *Child Development*, *69*, 1247–1262.
- Tronick, E.Z. (1989). Emotions and emotional communication in infants. *American Psychologist*, *44*, 112–119.
- van Bakel, H. J. A., & Riksen-Walraven, J. M. (2004). Stress reactivity in 15-months-old infants: Links with infant temperament, cognitive competence, and attachment security. *Developmental Psychobiology*, *44*, 157–167.
- Vandell, D. (1979). Effects of a playgroup experience on mother – son and father – son interaction. *Developmental Psychology*, *15*, 379 – 385.
- van Goozen, S. H., Matthys, W., Cohen-Kettenis, P. T., Gispen-de Wied, C., Wiegant, V.M., & van Engeland, H. (1998). Salivary cortisol and cardiovascular activity during stress in oppositional defiant disordered boys and normal controls. *Biological Psychiatry*, *43*, 531-539.
- Whitworth, J. A., Brown, M. A., Kelly, J. J., and Williamson, P. M. (1995). Mechanisms of cortisol induced hypertension in humans. *Steroids*, *60*, 76–80.
- Zimmerman, L. K., & Stansbury, K. (2004). The influence of emotion regulation, level of shyness, and habituation on the neuroendocrine response of three-year-old children. *Psychoneuroendocrinology*, *29*, 973–982.