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INTER-PARENTAL CONFLICT AND EARLY CHILDHOOD ADRENOCORTICAL ACTIVITY: A BIOSOCIAL FAMILY SYSTEMS APPROACH

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Abstract

It is universally accepted that family conflict exposure is a significant stressor in the lives of children and a driving force in the development of maladjustment. Unfortunately, little progress has been made in determining the mechanisms by which this occurs. Theoretical models of risky families highlight the potential for exposure to conflict and violence to dysregulate stress physiology, thus precipitating the development of mental and physical health problems. While past studies have found variations in intensity, and frequency of conflict, to be implicated in the development of pathology, recent mechanistic studies of stress physiology have failed to incorporate this level of detail in their examination of conflicted families. Furthermore, maternal behaviors have been shown to be critical in both facilitating the development of stress responsive physiological systems and mitigating the repercussions of conflict and violence exposure. The analyses presented in the following chapters will attempted to integrate a family systems approach along with a biosocial model of atypical development. Study 1 will examine the unique contributions of verbal and physical aggression on infant adrenocortical levels, reactivity, and recovery. Study 2 will examine the implications of chronic exposure to violence across the first two years of life on toddler adrenocortical levels, reactivity, and recovery. Data come from the Family Life Project, a multi-site study of child development in the context of non-urban poverty. At enrollment the sample (n = 1,292 mother-infant dyads; 49.4 % male) was racially diverse (41% African American) and from predominantly low-income (78% of the families were more than 200% below the poverty line), rural communities. During a home visit, the child's saliva was sampled before, 20-minutes, and 40-minutes after standardized tasks designed to elicit the child's emotional arousal and later assayed for cortisol. Mothers completed self-report measures of her and her partner's verbal and physical aggression. Parenting behaviors were assessed via

structured interview and mother-child interactions. Study 1 focused on the later infancy assessment when the child was 15 months old and Study 2 focused on data up to and including the toddler assessment when the child was 24 months old. Results from Study 1 found the confluence of inter-parental verbal aggression and maternal insensitivity to be related to heightened cortisol levels. Similarly, the combination of physical violence and low levels of maternal negative-intrusion was related to higher overall cortisol levels. Analyses for Study 2 revealed chronic exposure to inter-parental violence to be related to heightened cortisol reactivity. However, early maternal sensitivity was found to completely buffer children from heightened cortisol reactivity. In other words, children exposed to maternal sensitivity during early infancy (at approximately 7-months) did not exhibit high cortisol reactivity even when exposed to chronic inter-parental violence. Findings are discussed within the framework of current biopsychosocial and risky family theories.

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CHAPTER 1

Introduction

Overview of the Problem

It is universally accepted that family conflict exposure is a significant stressor in the lives of children and a driving force in the development of maladjustment. Unfortunately, little progress has been made in determining the mechanisms by which this occurs. A multitude of theoretical models (e.g., emotional security hypothesis, social learning theory, trauma theory) have been proposed giving researchers a framework to guide *why* children develop behavioral problems, yet there is a dearth of studies exploring *how* these family relationship patterns lead to maladjustment. Thus, there is a need to move beyond "first generation" research establishing family conflict and violence as a risk factor for maladaptive outcomes, and begin a thorough "second generation" examination of the mechanisms underlying this link. The urgency to uncover explanatory processes is underscored by the surprisingly high prevalence and the long term repercussions of children's exposure to family conflict (National Institutes of Health, 2003).

Exposure to inter-parental conflict and violence: a risk factor

Many psychologists argue witnessing aggression between parents is a form of emotional abuse. Therefore, exposure to extreme family conflict and violence is a traumatic experience akin to child maltreatment (Wolfe & McGee, 1994). Children consistently elicit high levels of distress immediately surrounding angry or aggressive interactions between parents. This distress is demonstrated by changes in behaviors (e.g., freezing, crying, covering ears, requests to leave),

and verbalizing anxiety, discomfort, or concern, and self-reported negative emotions (Cummings, 1987; Cummings, Iannotti, & Zahn-Waxler, 1985; Cummings, Zahn-Waxler, Radke-Yarrow, 1981; Lewis, Siegal, & Lewis, 1984). In addition, children exposed to violence show behavioral and emotional changes consistent with traumatic experiences. Specifically, violence exposed children have intrusive, re-experiencing, and/or traumatic arousal symptoms (Graham-Bermann & Levendosky, 1998) and simulate post-traumatic play involving family members (Davies, 1992).

Even when inter-parental anger occupies only a small amount of the overall emotional environment, these interactions influence the child's ability to regulate emotions and behavior (Davies & Cummings, 1994). Children clearly discriminate angry emotions during interactions, evoking changes in emotional arousal (Cummings et al., 1981). When children are asked how they feel during and following conflict between adults, they often indicate sadness and anger, with young children expressing fear (Cummings, 1987; Cummings, Vogel, Cummings, & El-Sheikh, 1989; Cummings, Ballard, & El-Sheikh, 1991). When the content of the argument involves the child's behavior, children often describe feeling guilty or ashamed (Grych & Fincham, 1993). These negative emotions sensitize the child setting the stage for more extreme emotional reactivity (e.g., extreme fear, distress, and/or anger) to subsequent conflict (Davies & Cummings, 1998; Davies, Myers, Cummings, & Heindel, 1999).

Exposure to high levels of anger and negative interactions between parents has also been associated with long-term behavioral problems. Specifically, inter-parental conflict is directly related to negative child outcomes such as internalizing behaviors, externalizing behaviors, attention seeking, and deficient social and cognitive competence, (see Grych & Fincham, 1990 for review). Children surrounded by family violence have more conflict with their peers

(McCloskey & Stuewig, 2001) and use aggression, manipulation, and coercion as strategies during inter-peer conflict (Cummings et al., 1985; Dodge, Bates, & Pettit, 1990; Jouriles, Murphy, & O'Leary, 1989; Jouriles & Norwood, 1995). As teenagers, family violence exposed children are more likely to become violent offenders, resulting in incarceration (Spaccarelli, Coatsworth, & Bowden, 1995). Finally, as adults these children have higher rates of severe mood disorders, depression, substance abuse, and chronic diseases such as cardiovascular disease (Lu, Mueser, Rosenberg, & Jankowski, 2008; Felitti et al., 1998).

The majority of research examining the negative consequences of family violence has focused on school-aged children. However, recent studies indicate exposure at even a young age can be detrimental. Specifically, violence exposure in infancy is associated with the expression of trauma symptoms, disturbances in eating and sleeping, and greater distress in the face of conflict (Bogat, DeJonghe, Levendosky, Davidson, & Von Eye, 2006; DeJonghe, Bogat, Levendosky, Von Eye, & Davidson, 2005; Layzer, Goodson, & Delange, 1986). Maternal report of emotional abuse by her partner has been associated with aggressive peer interactions (e.g., bullying and insulting) in children as young as three years old (Graham-Bermann & Levendosky, 1998). In addition, research indicates parent-parent interactions to most profoundly influence parenting behavior toward an infant as opposed to parental behavior toward a preschooler (Crnic & Greenberg, 1984). In sum, beginning at a very early age, exposure to conflict and violence sets a child on a trajectory to poor mental and physical health.

The psychobiology of the stress response: a potential mechanism?

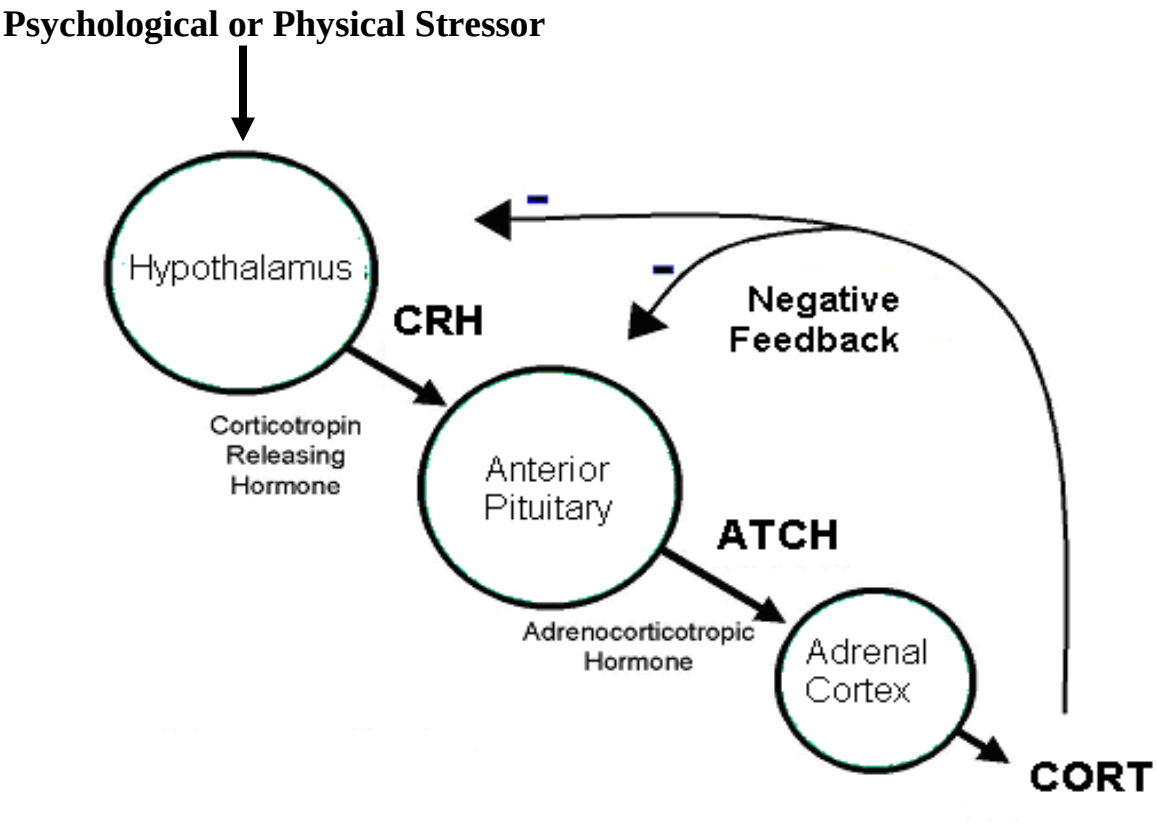
The stress response is an integrated multi-system reaction of physiological and behavioral systems (Chrousos & Gold 1992; Selye, 1976). The stress response is evolutionarily adaptive

(Canon, 1932), functioning to maintaining internal physiological balance in the face of physical or psychological challenge. This response system is beneficial in the short term, facilitating cognitive enhancement, mobilization of energy, increased immune function, and suppressing unnecessary behaviors such as eating and sleeping (Sternberg, Chrousos, Wilder, & Gold, 1992; Sapolsky, Romero, & Munck, 2000; McEwen, 2004). However, if threat is chronic, occurring at an unpredictable frequency, or high intensity, constant attempts at maintaining balance comes with a cost. Specifically, over exposure to the physiological substrates of stress responsive systems can damage hippocampal neurons, increase cardiovascular activity, depress immune function, and heighten fat deposition (Karlamangala, Singer, McEwen, Rowe, & Seeman, 2002; McEwen, 1998; Seeman, McEwen, Rowe, & Singer, 2001; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997; Sapolsky, 1992). In the long-term, heightened stress physiology has been associated with chronic diseases such as cardiovascular disease, metabolic syndrome, and glucose intolerance (see Walker, 2007 for review).

When faced with an internal or external stressor the body responds by producing a cascade of neuroendocrine transmitters with the main goal of preparing the body to successfully overcome the challenge. The two distinct (but closely related) physiological systems that respond to stressors are the hypothalamic pituitary adrenocortical (HPA) axis and the sympathetic adreno-medulary (SAM) axis. These chapters will specifically focus on the HPA axis (see Figure 1.1). Activation of this system begins with the secretion of both corticotrophin releasing hormone (CRH) and arginine vasopressin (AVP) from the paraventricular nuclei of the hypothalamus. These hormones travel through the bloodstream to the pituitary gland, stimulating the release of adrenocorticotropic hormone (ACTH). ACTH in turn travels down to the adrenal glands, located on top of the kidneys, resulting in the production of cortisol in humans (see

Gunnar & Quevedo, 2007 for review). Cortisol, a glucocorticoid, binds to receptors found on cells through the entire body and is therefore responsible for a wide array of responses ranging from regulating blood pressure and glucose metabolism to regulating immune function (Stratakis & Chrousos, 1995). Once inside the cell, glucocorticoids such as cortisol regulate the transcription of genes resulting in changes that take minutes to hours to be detected, and have the potential to continue exerting effects on both physiology and behavior for extended periods of time. Finally, circulating glucocorticoids also play the important role of counter regulating the neuroendocrine stress response by feeding back on the hypothalamus and pituitary gland inhibiting the further release of CRH and ACTH.

Figure 1.1. Schematic showing the structural components and neuroendocrine substrates of the hypothalamic pituitary adrenocortical axis.



There is reason to believe stress physiology plays a critical role in the development of mental and physical health problems in children exposed to inter-parental conflict and violence. Initial psychobiological studies revealed the autonomic nervous system to be highly sensitive to background anger and exposure to conflict. Specifically, children expressing anger in response to laboratory inter-adult conflict have reductions in heart rate, while children showing signs of concern or distress show increases in heart rate (El-Sheikh, Cummings, & Goetsch, 1989). Similar responses have been revealed in measures of skin conductance and systolic blood

pressure (El-Sheikh & Cummings, 1992). Furthermore, sympathetic nervous system reactivity (El- Sheikh, 2005; El- Sheikh, Keller, & Erath, 2007), parasympathetic nervous system reactivity (El-Sheikh & Whitson, 2006) and the coordination of these two systems (El-Sheikh, Kouros, Erath, Cummings, Keller, & Staton, 2009) have been shown to be related to the long-term development of internalizing and externalizing behavior problems in children exposed to high marital conflict. The recent incorporation of adrenocortical activation in studies of inter-parental conflict exposure promises to further illuminate the connection to pathology. As expected, children exhibiting high levels of distress to simulated marital conflict also elicited high adrenocortical responses (Davies, Sturge-Apple, Cicchetti, & Cummings, 2008). Furthermore, in a separate population, it was found that children exhibiting a dampening in cortisol activity in response to a simulated marital conflict had the highest level of externalizing symptoms two years later (Davies, Sturge-Apple, Cicchetti, & Cummings, 2007). Thus, there seems to be evidence that adrenocortical activity may be related to both the immediate and the long-term repercussions of inter-parental conflict exposure.

Integrating a family systems approach with a biosocial model

Conflict between romantic partners is ubiquitous across families; however, there is great variability in the frequency and style of conflict both within and between couples. Past developmental psychopathology studies have found children exhibit differential oucomes based on the style of conflict (i.e., verbal aggression compared to physical aggression), and the duration of exposure (i.e., chronic exposure compared to acute exposure). However, while an extensive literature has examined these nuances in relation to the development of mental and physical health problems (see Grych & Ficham, 1994 for review) no study to date has examined different

aspects of family conflict in relation to stress physiology. Primarily, all studies to date examining the physiological repercussions of exposure have aggregated conflict and violence (Davies et al., 2007; 2008; El-Sheikh et al., 2007), have only examined verbal conflict (Gottman & Katz, 1989; El Sheikh et al., 2009; Porter, Wouden-Miller, Shizuko Silva, & Porter, 2003) or physical conflict (Saltzman, Holden, & Holahan, 2005); and none, to the best of my knowledge, have prospectively accounted for duration of exposure. If these dynamics prove important in the development of psychopathology, clearly they should be integrated into studies containing physiology. Unfortunately, in an attempt to incorporate specificity at the response level, these second generation studies have compromised the assessment of complex family conflict dynamics.

This level of examination might be particularly important in furthering our understanding of individual differences in risk and resilience in conflict exposed children. Not exposed children develop adjustment problems and developmental psychopathologists suggest these differences in risk and resilience are due to individual differences in stress physiology (Cicchetti & Blender, 2006). However, family level differences in conflict dynamics *and* child level individual differences in stress physiology have the potential to interact or combine to lead to the development of children's psychopathology. The analyses presented in the following chapters will directly address how 1) the style of conflict (Study 1), and 2) the duration of violence exposure (Study 2), relate to infant and toddler adrenocortical levels, reactivity, and recovery during an emotion eliciting challenge. Given past research emphasizing maternal caregiving and parent-child relationships as moderators of inter-parental conflict and violence (e.g., El-Sheikh & Elmore Staton, 2004; Frosch & Mangelsdorf, 2001), positive and negative caregiving behaviors

will be assessed as attenuators and potentiators of the link between conflict exposure and cortisol levels, reactivity, and recovery.

Data will come from the Family Life Project, a longitudinal study of child development in a large ethnically diverse sample living within the context of rural poverty. As part of the larger study, children underwent a series of emotionally arousing challenge tasks aimed at eliciting anger and frustration. While the tasks themselves were novel to the infants and toddlers, they mimic common everyday challenges faced by children. Saliva samples were collected before, and 20- and 40 minutes after peak behavioral arousal to the tasks. During the home interview, mothers reported on her own and her partner's verbal and physical aggression over the past year. Maternal sensitivity was assessed during a semi-structured mother-child interaction.

Data from the Family Life Project will provide unique insight into the two questions addressed. Foremost, inter-parental conflict and violence is particularly prevalent in this rural and impoverished sample, providing ample power to assess the impact of these family conflict dynamics on adrenocortical reactivity in children. The data from the Family Life Project are a potentially stronger indicator of the individual differences in adrenocortical pathways to mental or physical pathology, than less ecologically valid laboratory paradigms (e.g., cold pressor, Trier Social Stress Test). The developmentally appropriate emotion eliciting tasks provide a naturalistic examination of the regulatory abilities of the children during typical, day-to-day challenges. Physiological stress responsive systems exhibit response specificity. In other words, individuals tend to consistently respond to a host of different stressors with a similar hierarchy of physiological stress responses (Engel, 1972). While the bulk of this work examined the autonomic nervous system, there is reason to believe the HPA axis exhibits the same specificity with respect to the degree of the response (Cohen et al., 2000; Hawkley et al., 2001; Kirschbaum

et al., 1995; see Lundberg et al., 1990 for opposing view). Thus, adrenocortical reactivity during the emotion eliciting challenge used in the Family Life Project may be indicative of normative adrenocortical reactions for that child in response to common emotionally arousing stressors. Lastly, the multi-method (e.g., self-report questionnaires, semi-structured interviews, parent-child interaction tasks, and physiological markers) and longitudinal approach will provide greater breadth and depth to our understanding of both the children and the families involved.

In sum, the present investigation examines a variety of family conflict dynamics in families with young children in order to explore the adrenocortical implications during an emotional stressor. Both chapters are grounded in family systems theory (Cox & Paley, 1997) and therefore the focus of the present analyses is at the marital, parental, and child level. This investigation is a first step toward a more complete understanding of family interactions on individual differences in the development of pathology.

CHAPTER 2

Unique contributions of inter-parental verbal and physical aggression to infant adrenocortical levels, reactivity, and recovery

Abstract

Inter-parental conflict style is thought to be a major source of the heterogeneity in child functioning in the context of inter-parental aggression. However, few studies have examined this level of conflict specificity when examining the underlying physiological processes associated with conflict exposure. Therefore, this study aimed to examine the relationship of inter-parental verbal and physical aggression to infant adrenocortical levels, reactivity and recovery in response to an emotion eliciting task. The sample (n = 1,169 mother-infant dyads; 49.4 % male) was racially diverse (43.3% African American) and from predominantly low-income (43.3% of the families were more than 200% below the poverty line), rural communities. During a home visit, the dyad's saliva was sampled before, 20-minutes, and 40-minutes after standardized tasks designed to elicit the infant's emotional arousal and later assayed for cortisol. Mothers completed self-report measures of her and her partner's verbal and physical aggression. Parenting behaviors were assessed via structured interview and mother-child interactions. Infants exposed to the confluence of high verbal aggression and low maternal sensitivity exhibited higher overall cortisol levels. Similarly, infants exposed to the combination of physical violence and low maternal intrusion also had higher overall cortisol levels. As expected, both of these risky family profiles were independently and uniquely related to infant cortisol. Findings are discussed within a risky family and a biosocial family systems perspective.

Introduction

The family has been described as both the most loving and supportive, and the most violent human institution (Coleman & Strauss, 1980). Recent studies estimate that between 10 and 20% of all children within the United States live in homes where they are exposed to violence (e.g., Moore, Probst, Tompkins, Cuffe, & Martin, 2007) with 75% of children physically present at the time of the altercation (Ernst, Weiss, & Enright-Smith, 2006). Even when inter-parental anger consists of only a small amount of the overall emotional environment, these interactions influence the child's ability to regulate emotions and behavior (Davies & Cummings, 1994), and as expected, exposure to inter-parental conflict and violence has been related to a myriad of socio-emotional and cognitive problems (see Grych & Fincham, 1990 for review). A leading hypothesis posits the relationship between inter-parental conflict and violence exposure, and behavioral maladjustment is at least in part due to individual differences in the regulation of stress-linked physiological processes (see Katz, 2001 for review). Studies of child development and family processes highlight marital verbal and physical aggression to be distinct in their antecedents and developmental outcomes (see Jouriles, Norwood, McDonald & Peters, 2001 for review; Straus & Gelles, 1986). Yet surprisingly little empirical attention has addressed the possible unique implications of exposure to verbal, compared to physical aggression, on stress physiology. Previous studies have focused solely on childhood exposure to conflict either in the form of verbal aggression (Davies, Sturge-Apple, Cicchetti & Cummings, 2007; El Sheikh, 2005), or physical aggression (Saltzman, Holden, & Holahan, 2005); or else did not differentiate between the two (Davies, Sturge-Apple, Cicchetti & Cummings, 2007). Therefore, prior research has not been able to disentangle the potentially distinct mechanisms by which

verbal and physical conflict exposure contribute to the development of pathology. The present set of analyses will attempt to address this important knowledge gap.

Inter-parental conflict and violence

Conflict can be broadly defined as any difference in opinion, ranging from minor to major, and from primarily positive to extremely negative interactions. Similarly, marital conflict includes displays of both positive and negative emotions and constructive (e.g., problem-solving) and destructive (e.g., physical assault) tactics. However, *marital aggression* is at the far most negative end of the marital conflict continuum and is characterized by physical and/or psychological abuse (Cummings, 1998). *Marital verbal aggression* refers to threats, insults, and throwing objects, whereas *marital physical violence* is characterized by physical assaults on a partner's body (Jouriles, Norwood, & McDonald, 1996) and can range from hitting and slapping to fatal assaults with guns and knives (Jaffe, Wolfe, Wilson, 1990).

National surveys revealing the pervasiveness of family aggression have prompted an extensive empirical investigation characterizing the antecedents, concomitants, and outcomes associated with these familial relationship patterns (e.g., Coleman & Straus, 1980; Gelles & Straus, 1979; Hotaling & Straus, 1980). Findings reveal couples with high verbal aggression have lower consensus regarding who holds the power, poorer communication quality, less marital happiness, and are less likely to facilitate problem solving than couples reporting lower levels of conflict (e.g., Gottman, Markman, & Notarius, 1977; Markman & Hahlweg, 1993). Marital conflict tends to revolve around family responsibilities; and changes in finances, employment, pregnancy, and other family stressors precipitate or attenuate the intensity of the conflict (e.g., Fox, Benson, DeMaris, & Van Wyk, 2002; Gelles, 1974). As the rate of verbal

aggression between romantic partners in a family increases, so does the incidence of minor violence with verbal conflict almost always preceding violence (O'Leary, Malone, Tyree, 1994). However, while conflict is thought to be ubiquitous in romantic relationships, violence is not and the majority of high conflict families do not exhibit physical violence (Coleman & Strauss, 1980). Violent couples are more likely to have been abused or witness abuse as a child, have high consumption of alcohol, and are more likely to be abusive toward their children than couples with high conflict alone (Edleson, 1999; Telch & Lindquist, 1984). Taking all of these factors into consideration, violence should not be considered merely an extreme form of verbal aggression (Rossman & Rosenberg, 1992) and may be indicative of more pervasive family problems.

Children reared in 'risky families' characterized by recurrent episodes of anger and aggression exhibit deficits in the control and expression of emotions, and in social competence (Repetti, Talyor, & Seeman, 2002). While physical aggression is a more potent risk factor for child adjustment problems than verbal aggression alone, both modes of conflict have been found to have independent effects on child outcomes (Fantuzzo, et al., 1991; Jouriles, Murphy, & O'Leary, 1989). Exposure to physical aggression between parents has been directly related to a wide range of mental and physical health difficulties in children, such as anxiety, sleep difficulties, aggression, difficulty in school, and somatic complaints (see Jouriles, et al., 2001 for review). Similarly, verbal marital conflict is also related to a range of negative child outcomes including internalizing and externalizing behavior problems, attention seeking, decreased social and cognitive competence, and low grade point averages (see Grych & Fincham, 1990 for review). Furthermore, exposure to verbal aggression predicts unique variance in child adjustment, after accounting for the effects of marital violence exposure (Jouriles et al., 1996).

Given the independent contributions of both physical and verbal aggression to child adjustment, it is tempting to speculate independent physiological processes are at play.

Developmental researchers have attempted to understand the mechanisms facilitating both adverse and resilient outcomes in children exposed to high levels of parental conflict, with much of the theorizing focused on parent-child relationships (see Cox, Paley, & Harter, 2001, for review). Maternal caretaking behaviors help to appropriately structure early experience and aid in the child's development of self-regulation (Feldman, Greenbaum, & Yirmiya, 1999; Kopp, 1982). When faced with a stressor, young children turn to their caregivers to make sense of the problem, and for help in coping with the crisis. Sensitive and responsive maternal behavior, thus buffers the child from high levels of negative reactivity, engendering arousal levels that are within the limits of the child's regulatory capabilities (Crockenberg & Leerkes, 2004; Jahromi, Putnam, & Stifter, 2004). Positive and supportive parenting, therefore, has the potential to result in long-term mitigation of the negative effects of adverse early environments on child adjustment (e.g., Pettit, Bates, & Dodge, 1997). Furthermore, while parent-child relationships are related to the quality of parent-parent relationships (Easterbrooks & Emde, 1988; Levendosky & Graham-Bergman, 2001), parent-child relationships prove to be a stronger moderator than mediator of the association between marital behavior and child outcomes (e.g., El-Sheikh & Elmore Staton, 2004; Frosch & Mangelsdorf, 2001). Thus, the present analyses will examine the moderating role of maternal behavior on the link between inter-parental conflict and violence, and infant adrenocortical levels, reactivity, and recovery.

Hypothalamic-pituitary-adrenal axis and early experiences

A major component of the psychobiology of the stress response involves activation of the HPA axis (e.g., Chrousos & Gold, 1992). Physical or psychological stressors inducing behavioral withdrawal, avoidance, or loss of control facilitate HPA activation (Dickerson & Kemeny, 2004; Gunner, Talge, & Herrera, 2009). Secretion of corticotrophin releasing hormone (CRH) from the hypothalamus initiates the release of adrenocorticotropic hormone (ACTH) into the bloodstream from the pituitary. ACTH stimulates cortisol, a glucocorticoid, to be secreted from the adrenal glands. This response is considered adaptive in the short-run as glucocorticoids facilitate mobilization of resources to accommodate changing or novel environmental demands (Sapolsky, 1996). As repeated experiences become familiar, the HPA axis habituates, such that the magnitude of its reactivity to those events is dampened (e.g., McEwen, 1998). However, if the events are extreme in intensity or duration, or they occur on an unpredictable schedule, the result may be frequent and prolonged HPA activation.

Children reared in "risky families" are thought to be particularly vulnerable to the development of mental and physical pathologies. These detrimental outcomes are thought to arise via the influence of aggressive interaction styles on children's biological responses to stress (Repetti et al., 2002). Children living with conflicted families are chronically exposed to stressors resulting in hyper emotional and physiological arousal (e.g., Cummings, 1987; Cummings, Vogel, Cummings, & El-Sheikh, 1989; Cummings, Ballard, & El-Sheikh, 1991; Davies et al., 2007; El-Sheikh, 2005). Constant conflict does not allow the child to sufficiently recover or recuperate from the heightened arousal, sensitizing them to subsequent conflict, and taxing emotional and physiological resources (Davies & Cummings, 1998; Davies, Myers, Cummings, & Heindel, 1999). Furthermore, neglectful and cold parental behaviors do not

provide the structure and support necessary for the development of appropriate self-regulatory behaviors or socio-emotional competencies (e.g., van Den Boom, 1994) to deal with these stressors. Exposure to family conflict thus jeopardizes the overall ability to cope both within the context of family conflict but also in the face of subsequent challenge (Davies & Cummings, 1994). Thus, the combination of excessive anger and unsupportive parenting make risky families an integrated risk profile (Repetti et al., 2002) leading to deficient emotional and physiological regulation.

Recent biosocial research has provided ample support for these perspectives connecting family dynamics (i.e., parent-parent and parent-child interactions) and child physiological development. Individual differences in children's adrenocortical activity has been shown to be related to parental conflict exposure at home (Davies et al., 2007), marital functioning (Pendry & Adam, 2007), and behavioral distress during the conflict (Davies et al., 2008). Similarly, evidence from both human and animal models points to early caregiving interactions determining the set-point or threshold of adrenocortical reactivity (Gunnar & Donzella, 2002; Gunnar & Vasquez, 2007; Meaney & Francis, 2001). Studies of early child development indicate harsh and insensitive or unresponsive caregiving behaviors are related to heightened stress reactivity (Gunnar, Larson, Hertsgaard, Harris, & Brodersen, 1992; Lisonbee, Mize, Payne, & Granger, 2008; Spangler, Schieche, Ilg, Maier, & Ackerman, 1994) and compromised development of self-regulation (e.g., Egeland, Pianta, & O'Brien, 1993). Sensitive caregiving and higher maternal caregiving quality, on the other hand, are associated with overall lower levels of adrenocortical arousal (Blair, et al., 2008; Kaplan, Evans, & Monk, 2007; Pendry & Adam, 2007) and reactivity (Morelius, Nelson, & Gustafsson, 2007; Spangler, et al., 1994) and greater amounts of recovery (Albers, Riksen-Walraven, Sweep, & de Weerth, 2008; Fortunato et al., in

review). In addition, studies have shown that while mothers are able to buffer their children from high HPA reactivity when in securely attached relationships, mothers in insecure relationships with their children may be unable to successfully regulate their child's stress response (Ahnert, Gunnar, Lamb, & Barthel, 2004; Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996). Understandably, family systems theorists (e.g., Cox & Paley, 1997) highlight the potential for parent-parent relationships and parent-child relationships to interact to determine behavioral and physiological processes (Repetti, Taylor, & Seeman, 2000; Hibel, Granger, Blair, Cox, & the FLP investigators, in press).

However, few studies have examined how the family system interacts to facilitate the development of regulation by combining multiple levels of familial influence on child physiology. This is particularly surprising considering the vast number of studies supporting the need for this level of family systems detail when examining child *behavioral* regulation (e.g., El-Sheikh & Elmore Staton, 2004; Frosch & Mangelsdorf, 2001; Owen & Cox, 1997; Peterson & Zill, 1986). To address this gap, the following analyses will incorporate a family systems perspective, and draw on the theoretical framework of risky families to examine the moderating effect of parent-child interactions on the link between family conflict and violence, and infant physiological regulation to an emotional challenge.

Hypotheses

The current set of analyses has three main goals. First, the relationships between verbal aggression and infant adrenocortical levels, reactivity, and recovery to a developmentally appropriate, emotion inducing event will be examined. To test this, three cortisol values surrounding a challenge task (pre-task, post-task1, and post-task2) designed to induce fear and

frustration will be examined in relation to verbal aggression. Adrenocortical levels will be operationalized as the mean cortisol values across the three collection points, reactivity will be operationally defined as the change in cortisol from pre-task to the first post-task. Recovery is defined as the change in cortisol from the first post-task to the second post-task. It is anticipated that the chaotic and fear inducing interactions in homes characterized by aggression will result in a state of constant and heightened readiness to respond to ever-present threat and challenge. Thus, verbal aggression will set the stage for elevated adrenocortical levels, exaggerated HPA stress-reactivity, and a reduced ability to recover. However, positive maternal behaviors will buffer the infant, such that infants exposed to inter-parental conflict with high positive caregiving will not show elevated HPA levels or reactivity. Similarly, infants exposed to verbal aggression and treated with negative or harsh caregiving will show more extreme elevations in HPA levels and reactivity, and reduced recovery.

Second, the relationships between inter-parental physical aggression and infant adrenocortical levels, reactivity, and recovery to the challenge will be examined. As with verbal aggression, infants living in homes characterized by violence will exhibit hyper-vigilance to fearful stimuli resulting in elevated adrenocortical arousal, exaggerated HPA stress-reactivity and a reduced ability to recover. However, unlike verbal aggression, given the extreme nature of violence, positive maternal behaviors will not buffer the infant, such that exposure to violence will determine cortisol levels, reactivity, and recovery, even in families with high maternal sensitivity. Conversely, negative or harsh caregiving will potentiate the repercussions of violence and infants exposed to both will show more extreme elevations in HPA levels and reactivity, and reduced recovery.

Third, the unique contributions of verbal and physical aggression on infant cortisol levels, reactivity, and recovery will be assessed. It is expected that both verbal aggression and physical aggression will independently predict these adrenocortical responses, that verbal aggression will explain unique variance in adrenocrtical levels, reactivity, and recovery above after controlling for physical aggression.

The challenge tasks and age of the infants used for this project were specifically selected to examine individual differences in behavioral and physiological responses to fear. The capacity to experience and express fearful emotions is developed in the second half of the first year of life, on average, between 7- and 9-months of age (e.g., Scarr & Salapatek, 1970). Thus, the analyses examine 15-month old infants, ensuring all infants have developed this capacity. Furthermore, around 12- to 15-months of age, infants exhibit a heightened fear response and therefore the challenge tasks presented to the 15-month old infant were implemented to capture this time of heightened fear reactivity.

Method

Participants

The Family Life Project (NICHD, P01HD39667) was designed to study families in two areas of high child poverty: the Rural South and Northern Appalachia (see Burchinal, Vernon-Feagans, Cox, & Key FLP Investigators, 2008 for description). Data for this analysis came from the larger project's home interview and assessments, when the infant was approximately 15-months old (range 13.5 – 22.3 months, M = 15.7). Interviews were conducted by home visitors.

From the total of 1,292 mother-infant dyads eligible to participate in the study, 1,169 were seen at the 15-month home visit. Based on the mother's race and income-to-needs ratio,

this sub-sample was 56.7% White and 43.3% African American, with 67.8% of the dyads 200% below the poverty line. Many (45.9%) of the mothers were not married, and the majority (88.8%) of the single mothers had never been married.

Procedures

Overview. As part of the larger project's 2-3 hour home visit, the Family Life Project assessments included self-report measures, semi-structured interviews, and a mother-child interaction task. Self-report questionnaires include those assessing intimate partner violence and demographic information. For the mother-infant interaction, mothers were given a set of toys and were instructed as follows: "*We would like for you to play with [infant's name] as you normally would if you had a little free time during the day"* (Cox, Paley, Burchinal, & Payne, 1999). Infants underwent "challenge tasks" (taken from the Laboratory Temperament Assessment Battery: Goldsmith & Rothbart, 1988) designed to elicit fear and frustration, including a toy removal task and a mask presentation challenge. Three saliva samples were collected surrounding the challenge tasks and were later assayed for cortisol (Hellhammer, Wust, & Kudielka, 2009).

Behavioral assessments

Inter-parental conflict and violence. To assess conflict and violence between partners, the *Conflict Tactics Scale- Couple form revised* (CTS-CF-R) was administered to all mothers, regardless of whether or not the mother's spouse/partner lived in the household with the infant. Mothers completed the scale twice reporting on both her and her partner's conflict style. The CTS-CF-R (Straus, Hamby, Boney-McCoy, & Sugarman, 1996) consists of 19 items exploring

conflict and violence between the partner and the mother in the past 12 months. The CTS-CF-R contains three subscales: verbal discussion, verbal aggression, and physical aggression. Only the verbal aggression and physical aggression scales were used in the analyses. Subscales from maternal report of her conflict style was summed with maternal report of her partner's style, resulting in one scale representing the overall levels of mother-to-partner and partner-to-mother verbal aggression and one scale representing the overall mother-to-partner and partner-to-mother physical aggression.

Observed parenting. Mother–child interactions during the free-play task were video recorded and later coded to assess levels of parental sensitivity, detachment, intrusiveness, stimulation, positive regard, negative regard, and animation in interacting with the child (see NICHD ECCRN, 1999). Ratings for each code were made on a scale ranging from 1 (*not at all characteristic*) to 5 (*highly characteristic*). On the basis of the results of factor analyses conducted with an oblique rotation (i.e., Promax), two broad-based parenting factors emerged: sensitivity and negative-intrusiveness. Sensitivity included five parental ratings: sensitivity (level of responsiveness to child's needs, gestures, and expressions), detachment (emotional unavailability, reverse coded), positive regard (positive feelings expressed toward child), animation (level of energy), and stimulation of development (appropriate level of scaffolding of activities with child). The factor loadings for maternal sensitivity were .83, -.89, .82, .85, and .81 and displayed adequate internal consistency (α = .87). Negative-intrusiveness included two parental characteristics: intrusiveness (the level at which the parent's agenda dominated that of the child) and negative regard (the level of harsh, negative feelings expressed toward child). The factor loadings for maternal negative-intrusiveness were .84 and .83 and displayed adequate internal consistency (α = .70). Reliability was determined by calculating the intraclass

correlation coefficients for ratings made by pairs of trained coders. A minimum of 30% of all observations were double coded; any discrepancies in codings were resolved by conferencing. All coding pairs maintain reliability estimates at above $r = .80$ for both sensitivity and negative-intrusiveness.

The HOME Inventory (Bradley & Caldwell, 1984) was employed to assess restrictive and punitive parenting styles. This measure examines the quantity and quality of support given to a child in the home environment. The HOME revealed 93% inter-rater agreement using raw percent agreement with high internal consistency ($\alpha = .89$) for the total scale. Items were completed by the home visitors based on conversations with the mother and observations of maternal behavior throughout the interview. Items included "caregiver shouts at the infant," "caregiver is hostile to the infant," "caregiver used more than 1 physical punishment in the last week," "caregiver scolds the infant during the visit," "caregiver physically restricts the infant," and "caregiver slaps or spanks the infant during the visit."

Laboratory Temperament Assessment Battery (Lab-TAB). Two "challenge tasks" designed to elicit emotional reactivity were administered to the child. The task procedures have been previously validated (e.g., Buss & Goldsmith, 1998; Kochanska, Tjebkes, & Forman, 1998; Stifter & Braungart, 1995), and are only briefly mentioned here. The tasks are from the Laboratory Temperament Assessment Battery (Goldsmith & Rothbart, 1988). For the first task, the toy removal task, mothers were asked to show their child a standardized toy provided by the home visitor. The mother demonstrated the toy and played with the toy and the child. Mothers were then asked to place the toy in a clear container, tighten the lid and then cease interactions with the child for two minutes. After 2 minutes mothers returned the toy to the child and played with, and soothed him/her as necessary for one minute. For the second task, the mask

presentation, the child was presented with 4 unusual masks, one at a time. The experimenter wore each mask for 10 seconds while calling the child's name and moving from side to side in front of the child. Mothers were asked not to intervene, but were told they could stop the task at any time. After the completion of the tasks, children were given 1 minute to self-soothe, after which the mother was allowed to soothe the child.

Behavioral Coding of Challenge Tasks. Teams of undergraduate coders were trained to assess behaviors related to negative reactivity during the "challenge tasks" (Stifter & Braungart, 1995; Stifter & Spinrad, 2002). Inter-rater 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 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 two challenge tasks 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. Kappas were .91 for the toy removal episodes and .89 for the mask task. Total behavioral reactivity was computed by summing the mean reactivity for each of the two challenge tasks.

Psychobiology of the stress response. To assess changes in cortisol indicative of the child's adrenocortical response to the emotion challenge tasks, 3 saliva samples were collected: a pre-task sample collected prior to administration of the challenge tasks, a sample 20-minutes after the final mask was shown (or prior, if the child reached "peak emotional arousal"), and then a final sample 20-minutes later. The child was considered to have reached peak arousal if he or she produced 20-seconds of hard crying at which point the tasks were terminated. The second and third saliva samples were then collected 20- and 40-minutes post peak behavioral reactivity to the task and represent post-task1 and post-task2 samples, respectively. Unstimulated whole saliva was collected using hydrocellulose absorbent material and expressed into storage 2 ml cryogenic storage vials by centrifugation. After collection, samples were immediately placed on ice, then stored frozen (-20 ^{0}C) until shipped on dry-ice overnight to the Behavioral Endocrinology Laboratory at the Pennsylvania State University. Samples were then stored frozen at – 80 ^{0} C until assayed. All samples were assayed for salivary cortisol using a highly-sensitive enzyme immunoassay (Salimetrics, State College, PA). The test used 25 μl of saliva, had a range of sensitivity from .007 to 3.0 μg/dl, and average intra-and inter-assay coefficients of variation less than 5%. All samples were assayed in duplicate and the average of the duplicates was used in all analyses. Cortisol levels were natural log transformed to correct for skewed distributions. All analyses used transformed values; for ease of interpretation, raw levels are reported.

Analytical Strategy

First, descriptive statistics are provided to overview the changes in cortisol levels across the task period. Then, partial correlations or ANCOVAs (controlling for sampling time of day) are computed to identify demographic (age, sex, race), infant physical health, maternal mental

health (depression, anxiety) and cognitive factors associated with pre-task cortisol levels, and cortisol reactivity and recovery to the challenge tasks to be included in the main analyses as covariates. Preliminary GLMM ANOVAs are used to determine the most appropriate error structure of the repeated cortisol measures. Next, the incidence and demographic characteristics associated with verbal and physical aggression are examined. Finally, the main analyses employed a series of general linear mixed model (GLMM) repeated measures ANOVA to determine the relationship between inter parental conflict and violence, and infant physiology. SAS PROC MIXED and PROC GLM (SAS v9.1.3, SAS Institute, Cary, N.C.) are used for all main analyses.

Results

Preliminary and Descriptive Analyses

Percent missing data ranged from 0 to 30%. To avoid bias in estimates associated with listwise deletion, full-information maximum likelihood or multiple imputation (Schafer & Graham, 2002) was used to replace missing values. Five data sets were imputed using the 'impute module' of Imputation and Variance Estimation Software, (IVEware: Raghunathan, Lepkowski, Van Hoewyk, & Solenberger, 2001) launched from SAS.

Infants with single mothers (M = .27, SD = .42) F (1065) = 5.53, $p < .05$ had higher overall cortisol levels than infants with non single mothers (M = .22, SD = .35). Maternal singlehood status and sampling time of day F (1, 1065) = 52.73, $p < .0001$ were included as covariates in all analyses. Preliminary GLMM revealed an unstructured error pattern to result in the lowest AIC and BIC model fit parameters. Confirming Blair et al., (2008), cortisol levels increased from pre-task to post-task1 F (1, 1047) = 47.18, $p < .0001$ but had no change from

post-task1 to post-task2 F (1, 1009) = 1.47, p = ns. Table 2.1 provides the raw means, standard deviations, and correlations of both infant cortisol levels before, and in response to the challenge tasks along with verbal and physical aggression and maternal caregiving behaviors.

As expected, all families exhibited some form of verbal aggression, with verbal aggression being used on average 3 to 5 times a year. Families with lower income to needs ratios had higher levels of verbal aggression $r(1163) = -.10$, $p < .01$. Mothers reporting higher verbal aggression were younger $r(1163) = -.15$, $p < .001$, less educated $r(1163) = -.12$, $p < .001$, more anxious $r(1163) = .41$, $p < .001$, and more depressed $r(1163) = -51$, $p < .001$. African American families (M = 3.1, SD = 2.3) reported more verbal aggression than white families (M = 2.8, SD = 2.2). Higher frequencies of verbal aggression were also associated with more physical aggression $r(1163) = .57$, $p < .001$, however, the majority of families (60.9%) did not report the use of violence. Families reporting violence (N = 455), used on average three different types of violence between 6 and 10 times in the past year. The most common violence used was pushing, grabbing, and shoving (42%), followed by hitting with an object (20%). Families using violence had mothers that were more likely to be African American χ^2 (1) = 106.0, $p < .0001$, unmarried χ^2 (1) = 123.3, $p < .0001$, younger t (1161) = 7.8, $p < .0001$, less educated $t(1161) = 9.7$, $p < .0001$ and more likely to have an income to needs ratio 200% below the poverty line χ^2 (1) = 80.3, $p < .0001$ than families without violence. Mothers in a violent relationship were also more depressed t (595.3) = 12.1, $p < .0001$ and more anxious t (595.4) = 8.3, $p < .0001$ than mothers in families without violence.

Table 2.1 Means, Standard Deviations, and Inter-correlations of cortisol (μg/dL), verbal and physical aggression and maternal behavior.

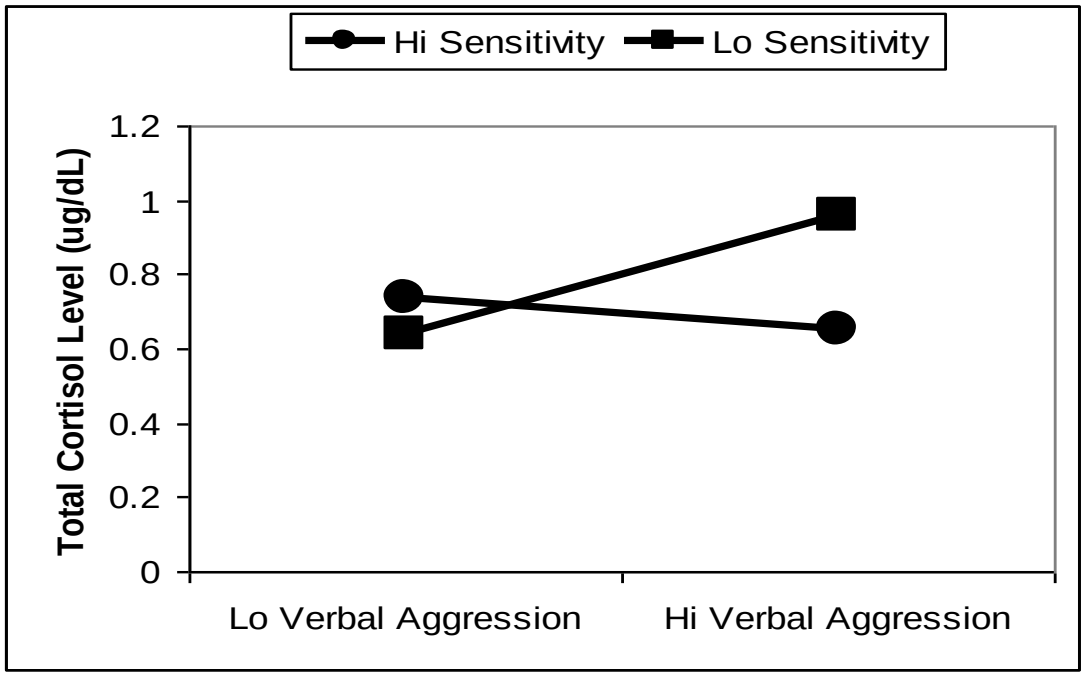
Variable	M	SD	1	2	3	4	5	6	7	8
1. Child Pre-task cortisol	.23	.42	__							
2. Child Post-task1 cortisol	.26	.37	.54***	__						
3. Child Post-task2 cortisol	.25	.39	.63***	.69***	__					
4. Total Verbal Aggression	3.0	2.2	.06*	.04	.03	__				
5. Total Physical Aggression	.35	.77	.03	.02	.01	.57***	__			
6. Maternal Sensitivity	13.9	3.9	-.03	-.04	-.02	-.09**	-.21***	__		
7. Maternal Intrusion	4.5	1.3	-.01	-.04	-.01	.16***	.16***	.24***	__	
8. Maternal Harshness	.71	.99	-.03	-.03	-.04	.13***	.21***	-.23***	.24***	__

Note: * $p < .05$, ** $p < .01$, *** $p < .001$

Main Analyses

Verbal aggression and infant cortisol. The total verbal aggression reported in the household was used to predict cortisol levels, reactivity, and recovery across the emotion eliciting task. Verbal aggression was not related to overall cortisol levels F (1, 1062) = 0.08, p = ns, or reactivity or recovery to the challenge tasks F (2, 1061) = 0.02, p = ns. Maternal caregiving behaviors (maternal sensitivity, maternal negative-intrusion, and restrictive and punitive parenting) were entered in as moderators of verbal aggression. A main effect was found for restrictive and punitive parenting F (1, 1062) = 6.78, $p < .01$, revealing higher levels of restrictive and punitive behaviors to be associated with higher overall cortisol levels. A significant interaction between total verbal aggression and maternal sensitivity was found F (1, 1061) = 4.70, $p < .05$. The interaction was probed using criteria outlined by Aiken and West (1991) with high and low groups specified as one standard deviation above and below the mean, respectively (Figure 2.1). This interaction revealed that in dyads with high maternal sensitivity, verbal aggression was not related to child cortisol levels F (1, 200) = 0.00, $p = ns$. However, when the mother exhibited lower levels of sensitivity, high verbal aggression was associated with higher levels of cortisol F (1, 191) = 4.47, $p < .05$. No main or interactive effects were found for negative-intrusive maternal behavior.

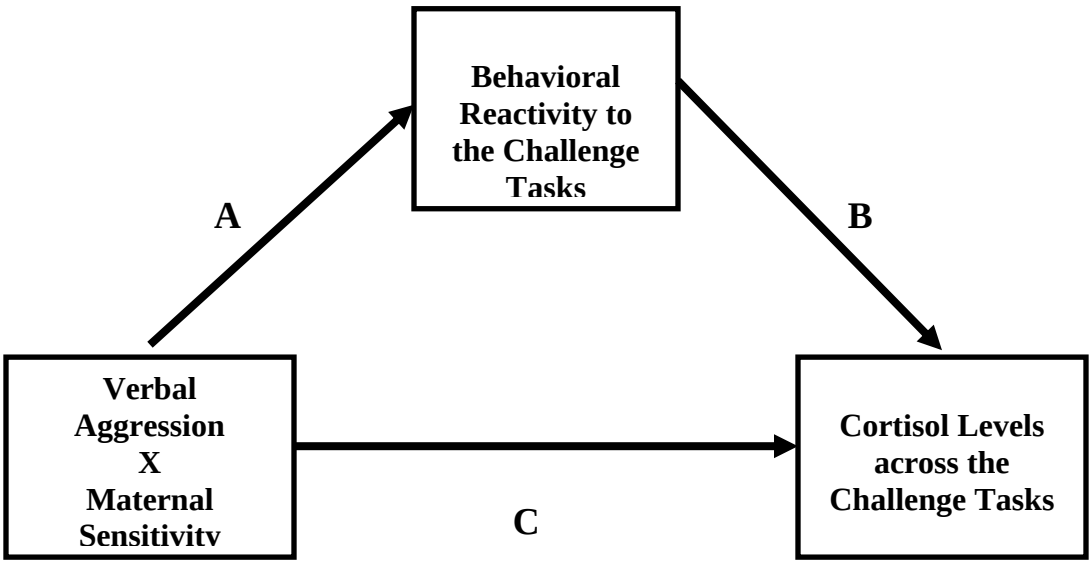
Figure 2.1. Infants exposed to the confluence of high levels of verbal aggression and low maternal sensitivity exhibit higher cortisol levels.



Follow-up analyses. Behavioral reactivity during the task was then examined as a mediator of the relationship between verbal aggression X maternal sensitivity on infant cortisol. Baron and Kenny (1986) define four steps that must be met to determine mediation (Figure 2.2). Within the scope of these analyses the four steps are 1) verbal aggression X maternal sensitivity is correlated with infant cortisol levels (Pathway C), 2) verbal aggression X maternal sensitivity is correlated with behavioral reactivity (Pathway A), 3) Behavioral reactivity is correlated with infant cortisol levels, 4) When controlling for behavioral reactivity, the correlation between

verbal aggression X maternal sensitivity and infant cortisol levels is significantly (partial mediation) or completely (full mediation) reduced. Controlling for infant age and race, verbal aggression X maternal sensitivity was not associated with infant behavioral reactivity to the challenge tasks F (1, 1090) = 1.71, *p* = *ns* (Pathway A). Therefore, a mediation model was not supported and no further pathways were tested.

Figure 2.2 Behavioral reactivity to the emotion eliciting challenge task did not mediate the association between the combination of high verbal aggression and low maternal sensitivity, and infant cortisol levels across the task. In other words, the combination of verbal aggression and maternal sensitivity was not related to infant cortisol levels, through their association with infant behavior.



Physical aggression and infant cortisol. The total physical aggression reported in the household was used to predict cortisol levels, reactivity, and recovery across the emotion eliciting task. No main effect F (1, 1062) = 0.37, p = ns or interaction with cortisol sample collection point F (2, 1061) = 0.12, p = ns was found for physical aggression. Maternal caregiving behaviors were entered in as moderators of physical aggression. As reported above, the main effect for restrictive and punitive parenting F (1, 1062) = 8.35, $p < .01$, continued to reveal higher levels of restrictive and punitive behaviors to be associated with higher overall cortisol levels. A significant interaction between total physical aggression and maternal negative-intrusion was found F (1, 1062) = 5.24, $p < .05$. The interaction was probed using criteria outlined by Aiken and West (1991) with high and low groups specified as one standard deviation above and below the mean, respectively (Figure 2.3)[1]. This interaction revealed that in dyads with high maternal negative-intrusion, physical aggression was not related to child cortisol levels F (1, 207) = 0.08, p = *ns*. However, when the mother exhibited lower levels of negative-intrusion, high physical aggression was associated with higher levels of cortisol F (1, 238) = 4.84, $p < .05$. No main or interactive effects were found for restrictive and punitive behaviors or maternal sensitivity.

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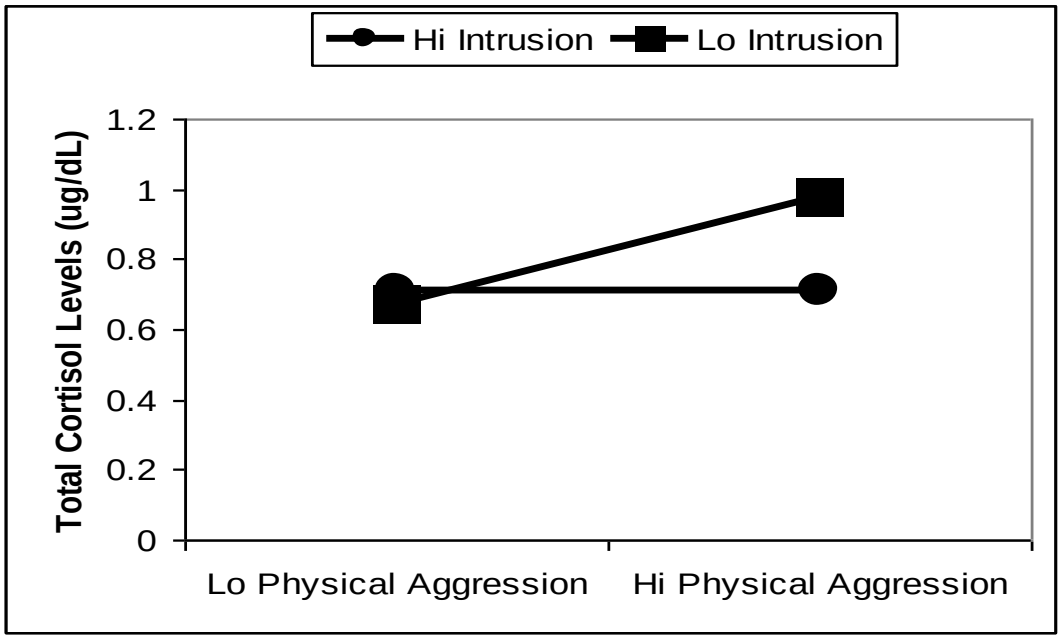
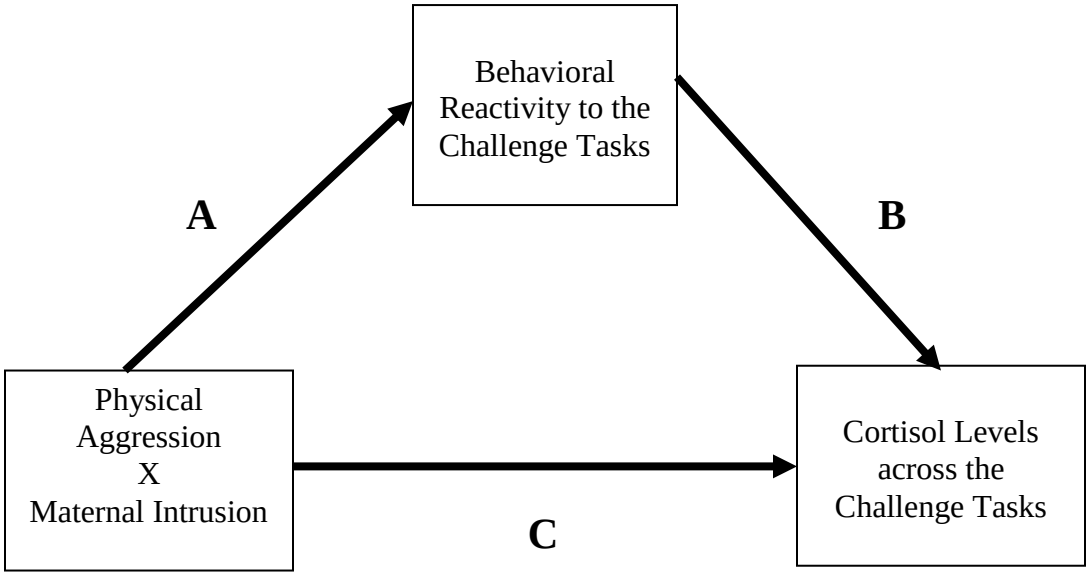
[1] The nature of the distribution of violence resulted in the "low violence group" to be families with no reported violence.

Figure 2.3. Infants exposed to the confluence of high levels of violence and low maternal intrusion exhibit higher cortisol levels.



Follow-up analyses. Behavioral reactivity during the task was then examined as a mediator of the relationship between physical aggression and maternal negative-intrusion on infant cortisol. Mediation was tested following guidelines set forth by Baron and Kenny (1986). Thus, pathways A, B, and C on figure 2.4 were assessed. Controlling for infant age and race, physical aggression X maternal intrusion was not associated with infant behavioral reactivity to the challenge tasks F (1, 1090) = 0.73, $p = ns$ (Pathway A). Therefore, a mediation model was not supported and no further pathways were examined.

Figure 2.4 Behavioral reactivity to the emotion eliciting challenge task did not mediate the association between the combination of high physical aggression and low maternal negative-intrusion, and infant cortisol levels across the task. In other words, the combination of physical aggression and maternal negative-intrusion was not related to infant cortisol levels through its association with infant behavior.



Verbal and physical aggression, and infant cortisol. Physical aggression along with their interactions with maternal sensitivity and maternal negative-intrusion, were run in the same model to determine the unique prediction of cortisol levels, reactivity, and recovery. Both the interaction between verbal aggression and maternal sensitivity F (1, 1059) = 6.42, $p < .01$, and physical aggression and maternal negative-intrusion F (1, 1059) = 6.23, $p = .01$, remained significant, suggesting unique prediction of cortisol levels from both verbal and physical aggression, and maternal caregiving behaviors.

Comments/Conclusions

The analyses presented examined the contributions of inter-parental verbal and physical aggression on adrenocortical levels, reactivity, and recovery to an emotion eliciting task during infancy. Maternal behaviors were examined as moderators, and behavioral reactivity was examined as a mediator. No main effects were found for either conflict style, however, consistent with prior research (e.g., Frosch & Mangelsdorf, 2001) interactions between parenting behaviors and conflict were found for both inter-parental verbal and physical aggression. These moderator models revealed infants exposed to the confluence of high verbal aggression and low maternal sensitivity, or high physical violence and low maternal negative-intrusion had higher overall cortisol levels. Neither of these relationships was mediated by behavioral reactivity to the task. Supporting past research on the development of behavioral maladjustment (see Jouriles et al., 2000 for review), both risky family profiles (i.e., verbal aggression and maternal insensitivity, and physical aggression and maternal low negative-intrusion) were uniquely related to cortisol levels. Findings are discussed within a risky family perspective.

Supporting the family systems characterization of 'risky families' as a combination of both aggressive parent-parent interactions along with cold parent-child processes (Repetti et al., 2002), family conflict and violence was found to influence infant physiology only within the context of certain maternal behaviors. Specifically, the confluence of both frequent familial verbal aggressions along with little maternal sensitivity was found to create a stressful and unpredictable environment for the infant as measured by heightened cortisol levels. Infants exposed to high verbal conflict but with sensitive mothers were physiologically indistinguishable from those exposed to low levels of conflict. While neither verbal aggression nor maternal insensitivity alone predicted adrenocortical dysregulation, cumulative exposure to both risk

factors was associated with heightened cortisol levels. Studies of contextual risk rarely find single risk factors to explain individual differences in the development of atypical behaviors. However, a child's exposure to multiple risks has proven to be a strong indicator of negative mental and physical heath outcomes (Rutter, 1983; Sameroff, Seifer, Baldwin, & Baldwin, 1993). Adversities have potentiating effects on the development of disorder and combinations of stressors result in more profound repercussions than two risks considered separately (Rutter, 1983). This may be particularly relevant when insensitive mothers fail to facilitate the regulatory abilities necessary to deal with the chronic stress associated with inter-parental conflict.

Interestingly, infants exposed to inter-parental violence and low levels of negative-intrusive behaviors, also exhibited high physiological activation. Following the work of Ainsworth and colleagues (1978) negative-intrusive behavior was defined as parental behavior that dominates the child, forcing the parent's agenda on the child, paired with harshness, disapproval, or punishing behaviors. Following the attachment literature, one would expect the stress of negative and intrusive maternal behaviors to be associated with heightened adrenocortical activity, however the opposite was found. Why would low negative-intrusion "intensify" exposure to violence? Potentially, negative-intrusive maternal behavior represents an engaged and active parenting style. While sensitive caregiving is considered ideal in facilitating the child's socio-emotional development (e.g., Sroufe, 2000; Bowlby; 1979), intrusive mothers are at least providing a source of external regulation, albeit excessive amounts. Within the unpredictable and potentially physically threatening context of living in a violent home, where infants and toddlers may be especially vulnerable to harm during these violent interactions, maternal behavioral and physiological over-control may be more beneficial than no control at all.

Thus, to "over ride" the influence of family violence exposure, maternal behaviors may need to be dominant and oppressive.

Evolutionary ecologist and epigenetic theorists view maternal behavior as a key mechanism in programming the offspring's lifelong behavioral and physiological responses (e.g., see Mousseau & Fox, 1998 for review). Maternal behavior is thought to portray information about the surrounding environment, serving as a signal of the environment's quality. Thus, the maternal behaviors *program* the offspring's adrenocortical responses to be uniquely sensitive to the specific contextual and environmental demands in which the offspring is raised (Francis, Caldji, Champagne, Plotsky, & Meaney, 1999). In other words, unpredictable environments elicit harsh maternal behavior which in turn facilitates the highly reactive physiological profiles necessary to respond to increased environmental demands. In support of these general ideas, recent studies have shown prolonged cortisol reactivity in infants with insensitive and intrusive mothers (Albers, Riksen-Walraven, Sweep, & de Weerth, 2008) and heightened reactivity in cocaine exposed infants with high caregiver instability (Eiden, Veira, & Granger, 2009). Similarly, Nachmias and colleagues (1996) reported that intrusive maternal behaviors during interactive play were associated with elevated cortisol reactivity in infants with high behavioral inhibition. Fitting within this framework, the combination of verbal aggression and low sensitivity was found to be related to higher cortisol levels. However, seemingly counter to these past studies, it was the confluence of inter-parental violence and *low* negative-intrusive maternal behaviors that resulted in heightened cortisol levels.

Family systems theorists underscore the importance of viewing phenomena within a web of hierarchical relationships, and understanding and interpreting processes within these interconnections (e.g., Cox & Paley, 1997). In other words, mother-child relationships must be

viewed within the context of their immediate and surrounding environments. Thus, the backdrop of inter-parental violence may be critical in interpreting mother-child dynamics and the meaning of negative and intrusive maternal behaviors. Cross-cultural studies do not consistently find relationships between intrusive maternal behavior and negative child outcomes, with some finding intrusive maternal behaviors to be associated with positive outcomes (e.g., Carlson & Harwood, 2003; Ispa et al., 2004; Fracasso, Busch-Rossnagel, & Fisher, 1994). In certain contexts, therefore, intrusive parenting may represent beneficial, effective parenting (Grusec, Rudy, & Martini, 1997). Despite a recent call to incorporate multiple familial systems in determining risk and resilience to inter-parental conflict (El-Sheikh & Elmore-Staton, 2004), few studies have concurrently assessed marital, parental, and child behavioral and physiological measures. However, there is reason to believe that variations in contextual and marital processes may influence the nature of parent-child relationships and subsequent child development (McLoyd, 1990; Levendosky & Graham-Bergman, 2000; 2001). While it is clearly premature to assume that negative-intrusive behaviors are beneficial, more research needs to be carried out to understand the implications of individual differences in caregiving styles in the context violence with respect to child outcomes.

Much research has been devoted to exploring the links between maternal psychopathology, maternal caregiving behavior, and child outcomes. At the present time, maternal depression has been fairly conclusively linked to negative child outcomes, placing children of all ages at high risk for psychopathology (e.g., Cummings & Davies, 1994; Field 1992). During infancy and toddlerhood, children of depressed mothers exhibit deficits in the ability to regulate emotions (Zahn-Waxler, Cummings, Iannotti, & Radke-Yarrow, 1984). Furthermore, these children are less responsive, and show more gaze aversion and distress during

mother-child interactions than children with non-depressed mothers (Field, 1984). In the present analyses, neither maternal depression nor anxiety was related to child cortisol levels, reactivity, or recovery, however, both were moderately correlated with verbal and physical aggression. A larger model examining a broader range of maternal characteristics may have found maternal depression or anxiety to play a role in child physiology. More specifically, maternal psychopathology may help clarify the role of maternal negative-intrusion in the relationship between family violence and infant cortisol levels. The presence or absence of maternal depression may explain why negative-intrusion seems to play a protective role in the context of family violence.

Behavioral and cognitive responses to environmental threats are considered the first line of defense. These immediate responses are thought to mediate the impact of a stressor on slightly slower acting physiological responses. Thus, only if changes in the "behavioral surface" (i.e., behavior or subjective experience) are unable to accommodate the challenge, do psychobiological components of the stress response activate (Gottlieb, 1992). Therefore, heightened adrenocortical activity exhibited by the infants in risky families could represent an inability to successfully respond behaviorally. However, behavioral reactivity to the challenge task did not mediate the link between negative familial interactions and cortisol over activity. In fact, neither familial risk profile (i.e., conflict or violence) was related to behavioral reactivity during the task. Why did the behavioral responses to the fear and frustration inducing challenge tasks not mediate the link between family risk and infant physiology? Infants exposed to family negativity did not exhibit heightened adrenocortical *reactivity* to the challenge, but heightened overall cortisol *levels*. The increased physiology could represent a constant state of physiological over activity independent of the emotion challenge tasks, or the behavioral responses they elicit.

Developmental theorists highlight the emotional and cognitive responses surrounding conflict as possible mechanisms accounting for the association between inter-parental conflict and child psychosocial adjustment (Crockenberg & Langrock, 2001; Grych & Fincham, 1994; Rhoades, 2008). Specifically, repeated fearful or angry responses to inter-parental conflict are thought to lead to behavioral maladjustment (Crockenberg & Langrock, 2001). Constant adaptations to negative interactions, such as those found in response to inter-parental conflict, are thought to result in chronic physiological dysregulation (Fries, Shirtcliff, & Pollak, 2008; McEwen & Seeman, 1998; Repetti et al., 2002; Seeman et al., 1997). Thus, repeated adaptations to threatening and stressful family environments may have facilitated biological dysregulations resulting in chronically high cortisol levels, completely independent of the acute stressor (i.e., the challenge tasks). While this study was not specifically designed to assess basal adrenocortical functioning, this could explain why family risk was related to heightened cortisol levels, independent of the acute behavioral responses.

Three physiological profiles are highlighted as enhancing vulnerability to chronic disease and early mortality in adulthood; 1) frequent, recurrent reactivity, 2) failed physiologic shut down, and 3) inadequate physiologic response (McEwen, 2004). In the present analyses, infants exposed to either risky family profile (i.e., verbal and physical aggression) had higher overall cortisol levels than those not living in conflicted and unsupportive homes. Infants within these risky homes seem to exhibit a failure to shut down adrenocortical activity, exhibiting chronically high cortisol levels. McEwen describes this “chronic activity” as an inability of the physiological system to down-regulate and recover. This physiological profile has been associated with neuronal death, dendritic atrophy, obesity, and acceleration of Type II diabetes (McEwen, 2004; Lundberg, Granquist, Hansson, Magnsson, & Wallin, 1989) and in the long term metabolic

syndrome, glucose intolerance, and cardiovascular disease (see Walker, 2006 for review). The scope of the present analysis does not allow for definitive statements predicting the development of these diseases and disorders. However, given the increased morbidity and mortality of individuals exposed to family conflict and violence, it is tempting to speculate the high circulating cortisol found in these analyses may be a causal mechanism, or at the very least, a risk factor for the development of future mental and/or physical pathology.

Implications for theory and application

The past one hundred years has been marked by a distinct epidemiological shift from acute illnesses (e.g., influenza and pneumonia) to chronic diseases (e.g., cardiovascular disease) contributing to the majority of morbidity and mortality in developed countries. While biomedical research provided huge advances in eliminating and treating pathogen related diseases, pharmaceutical interventions have been less successful at addressing the needs of individuals plagued by chronic illnesses. Unfortunately, laboratory bench procedures and paradigms fail to capture the complexities of the human experience and huge individual differences in exposure and response to environmental influences. Thus, the field of health psychology emerged showing physiological, behavioral, psychological, and social factors to be critical in the etiology and progression of these "diseases of lifestyle" (see Taylor et al., 1997 for review). Developmental psychologists have long attributed the development of pathology to early adverse social environments, however, recently a movement has been made exploring the physiological mechanisms that potentiate or attenuate the development of disorder (Cicchetti & Blender, 2006). Both separately and together, these two fields, health psychology and developmental

psychology, have implicated dysregulations in stress physiology as a mechanism in the developmental of mental and physical health outcomes.

In response to the hypothesis that dysregulated adrenocortical activation may, in part, be responsible for the relationship between negative contextual factors and the development of pathology (Calkins & Howse, 2004; Cicchetti, 2002; Repetti et al., 2002), advances have been made in incorporating physiological markers into studies of risk and resilience. The current analyses, therefore, fit within a broader network of studies exhibiting a consistent pattern of physiological dysregulation in the context of risk. Specifically, studies have revealed risk factors such as maternal depression (Lupien, King, Meaney, & McEwen, 2001), low socio-economic status (Essex, Klein, Cho, & Kalin, 2002), severe maternal deprivation (Gunnar, Morison, Chisholm, & Schuder, 2001), exposure to community violence (Suglia, Staudenmayer, Cohen, & Wright, 2009), and prenatal cigarette, lead, and cocaine exposure (Eiden, Veira, Granger, 2008; Gump et al., 2008; Schuetze, Lopez, Granger, & Eiden, 2008) are related to heightened cortisol levels or reactivity during early childhood. As expected, these physiological dysregulations have been associated with the development of psychological problems such as externalizing and internalizing behavior problems (Kestler & Lewis, 2009; Freitag, Hanig, Palmason, Meyer, Wust, & Seitz, 2009; Lopez-Duran, Olson, Hajal, Felt, & Vazquez, 2009; Shirtcliff & Essex, 2008), and somatic complications such as metabolic syndrome, and type II diabetes in obese children (Sen, Aygun, Yilmaz, & Ayar, 2008; Soros, Zadik, & Chalew, 2008). Together, these studies underscore the potential for disease pathways to begin during early childhood, and exposure to adversity to be a precipitator of these processes.

Unlike acute infectious illnesses, chronic diseases are characterized by a cascade of physiological changes set in motion long before the obvious physical manifestations of disease

are present. The previously mentioned studies linking early adversity, physiological dysregulation, and pathology raise the possibility that the "pre-disease pathways" (Committee on Future Directions for Behavioral and Social Sciences Research at the National Institutes of Health, 2001) preceding morbidity and mortality may be beginning as early as infancy and early childhood. In addition, disease processes previously thought to be confined to adulthood (e.g., diabetes, obesity) have become an increasing concern in children (Bloomgarden, 2004; Ebbeling, Pawlak, & Ludwig, 2002). The confluence of dysregulated stress physiology starting during childhood and the current trend of "adult diseases" emerging during childhood highlights the need to redirect resources toward early developmental periods. The knowledge flowing from this redirected research will be critical in increasing the understanding of associations between exposure to familial risk and child outcomes, and thus is instrumental for the creation of effective preventative interventions.

Most exciting in the field of health psychology and developmental psychopathology is the theoretical understanding that biology is not deterministic (Rutter & Sroufe, 2000). According to the biopsychosocial model (e.g., Engel, 1977; Lerner, 1984), socio-contextual and psychological factors, and biological processes interact to influence health outcomes. Biological changes as a function of contextual influences are neither rigid nor permanent. Thus, the next step in this interdisciplinary field of study is to incorporate physiological markers into intervention studies, to 1) better identifying individuals in need, 2) determine the short- and long-term efficacy of interventions aimed at ameliorating mental and physical health risk, and 3) further promote resilience (Cicchetti & Gunnar, 2008). Clearly in its nascent stage, biological markers are beginning to be integrated into the design and evaluation of preventive interventions, with promising results. For instance, interdisciplinary interventions aimed at children in foster

care (Dozier, Peloso, Lewis, Laurenceau, & Levine, 2008; Fisher & Stoolmiller, 2008) and low-income families (Fernald & Gunnar, 2009) have rendered diurnal or basal cortisol levels to be significantly different than those not receiving the intervention, and importantly, indistinguishable from children not at risk.

The present analyses may aid in the effective incorporation of measures of psychobiology into preventative interventions. In the present analyses, behavioral reactivity to the challenge task was not associated with the risky family profiles, despite past literature consistently indicating exposure to this risk to be linked with maladaptive behaviors (see Jouriles et al., 1989 for review). However, given the current analyses were conducted in infants, the physiological dysregulations seen in this sample have the potential to be precursors signaling the eventual development of maladaptive behaviors. Typical preventions must wait until the time when maladaptive behavioral patterns emerge in order to determine efficacy. Incorporating physiological markers into interventions targeting this young population has potential to determine efficacy (i.e., normative physiology) before maladaptive behavioral changes have occurred. Most important, are the complimentary findings implicating maternal behavior in potentiating the physiological outcomes associated with conflict and violence. Thus, the present analyses reiterate the importance of intervening both at the child and at the maternal level (Hofer, 2006). Teaching a child more positive coping styles may be futile if these responses to stress are ineffective in helping the child deal with aversive family situations. Only if the mother is involved in the intervention and taught more positive parent-child interaction strategies will intervention efforts be successful (e.g., Field, 1998).

The Family Life Project

Being born into a poor and rural population represents profound risk, jeopardizing the mental and physical development of children. Unfortunately, this population has been under studied, and therefore the characteristics of the communities, families, and individuals that shape child development in the poor rural context, and potentially serve as factors of risk and resilience, are unknown. Data for the present analyses come from the Family Life Project, an epidemiological sample representative of children living in rural Appalachia and the Black South. The epidemiological sample allows for greater generalizability to individuals living in these conditions. For this reason, there is increased importance in integrate findings across reports from this multi-site program project in an effort to reveal how these findings collectively advance our understanding of the concomitants and sequelae of early individual differences in HPA reactivity and recovery.

Our first report (Blair et al., 2008) revealed engaged maternal behaviors are associated with increased cortisol reactivity at 7-months and with overall lower levels of cortisol at 15-months. The next paper in our series (Towe-Goodman et al, under review) explored self-regulatory strategies and parenting behaviors associated with profiles of associated and disassociated emotional and adrenocortical reactivity at the 7-month assessment. Similar to the Blair et al analyses, Towe-Goodman et al found evidence that infants with less engaged mothers were more likely to show reduced adrenocortical reactivity but high behavioral reactivity. Another paper coming out of this project (Fortunato et al., under review) found early engaged maternal behavior to result in consistent rapid adrenocortical recovery to the challenge tasks at both the 7- and 15- month assessments. Together, these papers highlight the importance of early maternal caregiving in structuring reactivity of the HPA axis, recovery from activation, and the

coordination of bio-behavioral responses. The present findings extend these observations in important ways. Primarily, the analyses present here were the first to examine parent-parent interaction styles in relation to infant adrenocortical levels and reactivity, and thus incorporate a more holistic view of family interactions. Secondly, the present analyses reveal the influence of maternal behavior to be dependent on these parent-parent conflict styles, and to be important moderator of inter-parental conflict style and infant adrenocortical activity. While past analyses on these data have underscored the importance of maternal behavior, the present analyses provide a family systems framework for interpreting those influences. The combination of parental behavior and parent-parent interactions seems to play a large role in the development of the HPA axis in this population and as such may also influence the patterns of behavioral-adrenocortical reactivity and consistency of adrenocortical reactivity across infancy, as examined in previous Family Life Project papers.

During early childhood (i.e., infancy and toddlerhood) the world that a child directly interacts with is limited. Larger socioeconomic and demographic pressures may not have an immediate influence on the child's development. However, previous research has shown these socioeconomic and demographic factors to be a significant source of family stress and therefore may indirectly influence the developing child through changes in parent-parent and parent-child interactions (Conger et al., 1994). As such, the Family Life Project is attempting to systematically examine these levels of influence on child development in children growing up in non-urban and impoverished areas.

Limitations and Future Directions

This study had several limitations. First, researchers have emphasized the importance of using multiple measures to assess the psychobiology of stress (e.g. Bauer, Quas, & Boyce, 2002). The stress response is a multi-system response that is characterized by both an autonomic nervous system response [comprised of the sympathetic (SNS) and parasympathetic nervous systems (PNS)] and an HPA axis response. These physiological systems are in a constant process of coordinated adjustment and fine-tuning, meeting the individual's needs in responding to an ever-changing environment. Examining measures of SNS and PNS arousal would give further insight into the physiological processes associated with verbal and physical aggression exposure. On a further note, past studies have found outcomes associated with exposure to conflict and violence to be moderated by activation of the SNS, the PNS, and the interaction of these two systems (see El-Sheikh et al., 2009 for review). Given the HPA axis activation may also play an important role in the link between conflict exposure and pathology, future studies should assess both the autonomic nervous system and the HPA axis to determine if each individual system or the coordination of the two systems facilitates risk or resilience. Second, despite controlling for sampling time of day in all analyses, the large variation in sampling time may have contributed to a portion of the unexplained variance. This may have interfered with attempts to estimate the relation of family and child characteristics to cortisol levels and cortisol reactivity, or recovery. Similarly, the stress tasks varied in length. The post task saliva sample was taken 20 minutes after peak behavioral arousal. In some individuals, peak arousal occurred shortly after initiating the task while, in others this may not have occurred until the completion of the tasks. Consequently, the time in between samples varied from infant to infant, resulting in unexplained variance into the model. Third, this set of analyses is preliminary in determining the influence of

conflict and violence exposure on individual differences in pathology. Clearly, the next step is to examine the mediating or moderating role of stress physiology in the development of mental and physical health problems. This may prove more difficult than first appears. While the link between conflict and violence exposure and pathology is well established, there are individual difference in the timing and development of disorders. Prospective longitudinal studies are needed to track when physiological and behavioral changes first come on line. Longitudinal studies are especially important given the dynamic nature of conflicted families (e.g., frequent changes in cohabitation, waves of increase and decrease in conflict and violence). While the present analyses indicate there may be differences in adrenocortical activation based on risky family exposure, it is not known if these changes are lasting, independent of continued exposure or if future experiences might buffer or potentiate these differences. Furthermore, families are dynamic, thus, more research needs to be done examining intra-familial differences in interactions to determine which particular conflict styles present the largest physiological risk for the children exposed. Fourth, the sample for this analysis is an epidemiologically valid representation of children and families living in the areas from which it is drawn. As such, it is representative of a substantial segment of the low-income population in the United States in non-urban areas. While this is a major strength in allowing findings to be generalizable to a large portion of the rural United States, this limits its generalizability to urban populations. Future studies should be aimed at other populations to increase our understanding of how these processes operate across contexts.

Lastly, the most appropriate approach for determining chronically elevated cortisol levels would be to assess physiology across the day. Specifically, basal or diurnal levels should be sampled at multiple time points from wakening to bed time. While the present analyses indicate

children in risky families have higher cortisol levels, it is not know if these levels are higher across the entire day or if these children are "reacting" to the home visitor in their house. The children in these risky homes may be more sensitive to an "intruder" or novel person and thus have higher cortisol levels in response to the home visit, but may not exhibit chronic elevation. While the physiological profile found in the current analyses still represents an atypical pattern, future studies are needed to adequately contextualize this pattern.

Conclusions

Developmental researchers have made great strides in determining familial characteristics that present risk to the children exposed. The present analyses fit within a larger frameworks of studies attempting to better understand the pathways and mechanisms by which family dysfunction are translated into developmental disorders and pathologies. The diversity of interaction styles and family compositions highlights the need to assess multiple levels of family systems (e.g., marital and parental) and interactions across subsystems. It is only through a systematic examination of how adversity facilitates mental and physical health problems will more efficacious interventions be developed.

CHAPTER 3

Chronic violence exposure over the first two years of life and toddler adrenocotical levels, reactivity, and recovery

Abstract

Family violence in childhood is widely recognized as a significant public health problem, both in terms of the implications for health and development and the surprisingly high prevalence in the general population. The first two to three years of life are characterized by rapid neurological reorganization and development, and thus, early exposure to violence may permanently alter physiological functioning. This study examined the relationship between inter-parental physical aggression exposure from birth to age two on toddler adrenocortical levels, reactivity, and recovery to an emotion eliciting task. The sample (n = 1,102 mother-infant dyads; 49.2 % male) was racially diverse (40.3% African American) and from predominantly low-income (54% were more than 200% below the poverty line), rural communities. During a home visit, the dyad's saliva was sampled before, 20-minutes, and 40-minutes after standardized tasks designed to elicit the infant's emotional arousal and later assayed for cortisol. Mothers completed self-report measures of her and her partner's physical aggression at three time points across early childhood (child age 7-, 15-, and 24 months). Parenting behaviors were also assessed via structured interview and mother-child interactions at the three visits. Toddlers cumulatively exposed to high levels of violence across all three assessments exhibited heightened adrenocortical reactivity. However, early maternal sensitivity (within the first 7 months) completely buffered this relationship. Thus, toddlers exposed to high levels of violence but also high maternal sensitivity exhibited normative patterns of adrenocortical activation. Findings are discussed within cumulative risk, biological sensitivity to context, and evolutionary frameworks.

Introduction

The identification of "pre-disease pathways" that describe the biological and psychosocial influences preceding morbidity and mortality has become a top public health initiative (Committee on Future Directions for Behavioral and Social Sciences Research at the National Institutes of Health, 2001). Specifically, a call has been made to assess the cumulative physiological risk of individuals exposed to challenge at vulnerable times (e.g., during early stages of postnatal development). Health psychology theories implicate early dysregulations in stress physiology as a causal mechanism in the development of negative health outcomes and chronic diseases (e.g., Boyce & Ellis, 2005; Repetti, Taylor, & Seeman, 2002). Exposure to inter-parental violence during early childhood is a well known risk factor for the development of adjustment difficulties that to persist into adulthood (Furgusson & Horwood, 1999; Jouriles, Norwood, McDonald & Peters, 2001). Surprisingly, little empirical attention has addressed the possible influence of domestic violence exposure on stress responsive physiological systems in children. Furthermore, studies that have examined the physiological repercussions of conflict or violence exposure have not focused on exposed infants (see Porter, Wouden-Miller, Shizuko Silva, & Porter, 2003; and Moore, 2009 for exceptions), nor have they assessed the cumulative risk (i.e., chronic exposure) over early childhood (e.g., Davies et al., 2007; 2008; El-Sheikh, 2005). Conflicted families are dynamic with changes in the frequency and intensity of conflict along with shifts in household composition (e.g., Fox, Benson, DeMaris, & Van Wyk, 2002; Gelles, 1974; c). Thus, family violence is not a single event occurrence and past cross-sectional studies, while informative, fail to capture this dynamic process. These analyses will examine cumulative exposure to family violence from birth to age two and the association with levels, reactivity, and recovery of stress physiology.

Family violence and early childhood

Family violence in childhood is widely recognized as a significant public health problem, both in terms of the implications for health and development and the surprisingly high prevalence in the general population (National Institutes of Health, 2003). Recent studies estimate that between 10 and 20% of all children within the United States live in homes where they are exposed to violence (e.g., Moore, Probst, Tompkins, Cuffe, & Martin, 2007) disproportionate number of children under the age of 5 years (Fantuzzo et al., 1991). Reports reveal approximately 75% of children in violent homes are physically present at the time of a violent interaction (Ernst, Weiss, & Enright-Smith, 2006) witnessing a range of behaviors from insults and hitting to fatal assaults with guns and knives (Jaffe, Wolfe, & Wilson, 1990). Importantly, violence is not a single event occurrence in these risky families and children are likely to be exposed to frequent intense conflict (Repetti, Taylor, & Seeman, 2002) and chronic back ground anger (Cummings, Ballard, & El-Sheikh, 1991).

Children from violent families are four times more likely to exhibit clinical levels of problem behavior than comparison children (Holden, 1998). Specifically, exposure to marital violence has been related to symptoms of trauma, fear and anxiety, aggression and defiance, and delinquent behavior (see Jouriles, et al., 2001 for review). Furthermore, violence exposed children are more likely to have cognitive deficits along with somatic complaints such as headaches and difficulty sleeping (Rossman, 1997; Bailey, Delaney-Black, Hannigan, Ager, Sokol, & Covingtion, 2005). Children surrounded by family violence have more conflict with their peers (McCloskey & Stuewig, 2001) and use aggression, manipulation and coercion as

strategies during inter-peer conflict (Cummings, Iannotti, & Zahn-Waxler, 1985; Dodge, Bates, & Pettit, 1990; Jouriles, Murphy, & O'Leary, 1989; Jouriles & Norwood, 1995).

Family violence researchers and developmental psychopathologist emphasize the importance of taking a developmental perspective when examining the repercussions of family violence exposure (Cicchetti, 1993; Pynoos, 1993). Specifically, the child's experience of violence is, in part, determined by the child's ability to appraise, understand, respond to, and cope with danger (Finkelhor & Kendall-Tackett, 1997). These capacities are directly related to the child's physical, cognitive, socio-emotional, and physiological development. Furthermore, the child's stage of development determines the different developmental milestones potentially disrupted by abuse and violence. For example, exposure to violence during infancy can interfere with the development trust, jeopardizing secure attachment relationships (Owen & Cox, 1997). Thus, exposure to violence during infancy may result in unique behavioral and physiological implications that studies of middle childhood have failed to capture.

However, previous inter-parental violence research has largely ignored infants, assuming them to be at least partially protected from the psychological distress associated with violence exposure, due to their limited comprehension (Osofsky, 1995). Contrary to this view, recent research has shown changes in irritability, sleep disturbances, emotional distress, somatic complaints, fears of being alone, and regression in toileting behavior and language in infants and young children exposed to violence (DeJonghe, Bogat, Levendosky, von Eye, & Davidson, 2005; Osofsky & Scheeringa 1997, Zeanah, Boris, & Scheeringa 1997). In fact, infants and toddlers may be at a heightened risk of sustaining negative repercussions from violence exposure due to their increased dependence on caregivers. Specifically, when faced with a stressor, infants and young toddlers rely on their immediate caregivers to make sense of the problem, protect

them from danger, and help them to cope with the stressor (e.g., Hofer, 2006). However, the mother's ability to serve as an external regulator of the child's emotional and physiological states may be compromised in families with violence (Stevenson-Hinde, 1990; Sturge-Apple, Davies & Cummings, 2006) potentially indicating infancy to be a time of unique sensitivity to the stress associated with violence exposure.

In most families, the mother initially assists the young child in regulating physiological and emotional reactivity through sensitive and responsive behaviors (e.g., Jahromi, Putnam, & Stifter, 2004). Mother-infant co-regulation then sets the stage for child self-regulation in the face of subsequent challenge (e.g., Hofer, 2006; Schore, 2000). Positive and supportive parenting, therefore, has the potential to result in long-term mitigation of the negative effects of adverse early environments on child adjustment (e.g., Pettit, Bates, & Dodge, 1997). Similarly, within the context of negative marital interactions, competent, caring, and positive parent-child relationships have been shown to buffer children from maladaptive behaviors (e.g., El-Sheikh & Elmore Staton, 2004; Frosch & Mangelsdorf, 2001; Osofsky, 1999). Thus, exposure to positive caregiving specifically during early childhood may also serve as an important buffer of the *physiological* consequences of exposure to violence.

The hypothalamic-pituitary-adrenal axis

A major component of the psychobiology of the stress response involves activation of the hypothalamic-pituitary-adrenal (HPA) axis (e.g., Chrousos & Gold, 1992). Physical or psychological stressors inducing behavioral withdrawal, avoidance, or loss of control activate the HPA axis (Dickerson & Kemeny, 2004; Henry, 1992). This begins with the secretion of corticotrophin releasing hormone (CRH) from the hypothalamus. CRH then initiates the release

of adrenocorticotropic hormone (ACTH) into the bloodstream from the pituitary which in turn stimulates cortisol, a glucocorticoid, to be secreted from the adrenal glands. Cortisol has protective effects over acute intervals and is essential for adaptation, maintenance of homeostasis/allostasis, and survival (e.g., McEwen & Stellar, 1993; Sapolsky, 1996). However, if stressors are extreme in intensity or duration, or they occur on an unpredictable schedule, the result may be frequent and/or prolonged HPA activation. Repeated physiological adaptations to adverse psychosocial or physical challenges, has been linked to wear and tear on the body via glucocorticoid over-exposure (McEwen, 1998; Sterling & Eyer, 1988).

HPA activity has been hypothesized to be a mediating link between negative familial experiences and interactions (e.g., family violence) and the development of mental health problems (e.g., Heim & Nemeroff, 2001; Repetti et al., 2002; Susman, 2006). Repetti and colleagues propose a theoretical framework Families characterized by violence and hostility Children living with conflicted families are chronically exposed to stressors resulting in hyper emotional and physiological arousal (e.g., Cummings, 1987; Cummings, Vogel, Cummings, & El-Sheikh, 1989; Cummings, Ballard, & El-Sheikh, 1991; Davies et al., 2007; El-Sheikh, 2005). Constant conflict does not allow the child to sufficiently recover or recuperate from the heightened arousal, sensitizing them to subsequent conflict, and taxing emotional and physiological resources (Davies & Cummings, 1998; Davies, Myers, Cummings, & Heindel, 1999). Furthermore, neglectful and cold parental behaviors do not provide the structure and support necessary for the development of appropriate self-regulatory behaviors or socio-emotional competencies (e.g., van Den Boom, 1994) to deal with these stressors. Exposure to family conflict thus jeopardizes the overall ability to cope both within the context of family conflict but also in the face of subsequent challenge (Davies & Cummings, 1994). Thus, the

combination of excessive anger and unsupportive parenting make risky families an integrated risk profile (Repetti et al., 2002) leading to deficient emotional and physiological regulation. Supporting this framework, individual differences in adrenocortical reactivity have recently been related to behavioral distress during inter-parental conflict (Davies et al., 2008) and externalizing symptoms in conflict exposed children (Davies et al., 2007).

Developmental models of psychopathology highlight early experiences as key in understanding sensitivity and resistance to stress throughout life (e.g., Levine, 2005). Experiences during neurobiological sensitive periods (marked by rapid development) can have lasting effects on biological systems and behavioral processes (Dawson et al., 2000; Nachmais et al., 1996). While much of the HPA axis develops during the prenatal period, integration and maturation of these circuits occurs during infancy (Vazquez, 1998) and evidence from both human and animal models points to early caregiving interactions determining the set-point or threshold of adrenocortical reactivity (Gunnar & Donzella, 2002; Gunnar & Vasquez, 2007; Meaney & Szyf, 2005). Specifically, sensitive and high quality maternal caregiving, is associated with overall lower levels of adrenocortical arousal (Blair et al., 2008; Kaplan, Evans, & Monk, 2007; Pendry & Adam, 2007) and reactivity (Morelius, Nelson, & Gustafsson, 2007; Spangler, Schieche, Ilg, Maier, & Ackerman, 1994), and greater amounts of recovery (Albers, Riksen-Walraven, Sweep, & de Weerth, 2008; Fortunato et al., in review). In addition, studies have shown that mothers are able to buffer their children from high HPA reactivity when in securely attached relationships, while mothers in insecure relationships cannot (Ahnert, Gunnar, Lamb, & Barthel, 2004; Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996). Thus, early sensitive caregiving, even in the context of family violence, may facilitate normative patterns of physiological development.

The majority of studies associating early adversity with HPA system activity and reactivity are retrospective or cross-sectional. This comes as a surprise given it is *repeated* physiological adaptations to psychosocial or physical challenges that are responsible for bodily wear-and-tear (McEwen, 1998). In support of this theoretical framework, recent studies suggest the duration or chronicity of exposure to adversity may be important in determining health outcomes (DeBellis, 2001; Gunnar, Morison, Chisholm, & Schuder, 2001; Rutter, O'Connor, & the ERA study team, 2004). Chronicity may be particularly important when studying family violence, given the dynamic and ever-changing structure of conflicted families. Primarily, high conflict families are more likely to go through residence changes such as divorce and separation (e.g., Rogge & Bradford, 1999). While the majority of women in violent relationships reporting previous violent relationships (Kemp, Green, Hovanitz, & Rawlings, 1995), changes in cohabitation underscores the lack of stability in the nature, frequency, and intensity of conflict. Even when family composition is stable, changes in finances, employment, pregnancy, and other family stressors precipitate or attenuate conflict (e.g., Fox, Benson, DeMaris, & Van Wyk, 2004; Gelles, 1974). Therefore, only long-term prospective studies can accurately assess the cumulative family violence that occurs within a household, and reliably capture the familial milieu in which children are raised.

Present Study

Data come from a prospective longitudinal study assessing early childhood development in a non-urban, low income, epidemiological sample. The current set of analyses has two main goals. First, cumulative exposure to family violence over the first two years of life will be examined in relation to adrenocortical levels, reactivity, and recovery to an emotional challenge

when the child is 24 months old. To test this, three cortisol values surrounding a challenge task (pre-task, post-task1, and post-task2) designed to induce fear and frustration will be examined in relation to cumulative violence. Adrenocortical levels will be operationalized as the mean cortisol values across the three collection points, reactivity will be operationally defined as the change in cortisol from pre-task to the first post-task. Recovery is defined as the change in cortisol from the first post-task to the second post-task. It is expected that the unpredictable nature and high intensity of conflict in homes characterized by family violence will result in a state of constant and heightened readiness to respond to ever-present threat and challenge. Thus, I hypothesize infants cumulatively exposed to higher amounts of intimate partner violence will exhibit higher cortisol levels, heightened adrenocortical reactivity, and reduced recovery. This cumulative effect will be over and above reports of violence at any single time point within the study.

Second, sensitive maternal caregiving behaviors across early childhood will be assessed as moderators of the relationship between family violence exposure and physiological outcomes. In line with prior research, including previous analysis of these data (Blair et al., 2008) it is expected that maternal behavior will exert a greater influence on physiological regulation during infancy as opposed to toddlerhood. During the infant's first year of life, emotion is predominantly regulated by the mother; however, by the end of the child's first year, the responsibility of emotion regulation shifts towards the child (Feldman, Greenbaum, & Yirmiya, 1999; Kopp, 1982). Therefore it is hypothesized that sensitive and responsive maternal behavior during infancy, will set a trajectory toward an increased ability for self regulation on the part of the child, even in the face of adversity. Therefore, *early* maternal sensitivity will be the strongest buffer of the risk associated with cumulative violence exposure.

Method

Participants

The Family Life Project (NICHD, P01HD39667) was designed to study families in two areas of high child poverty: the Rural South and Northern Appalachia. Complex sampling procedures were used to recruit a representative sample of 1,292 families at the time that mothers gave birth to a child, over-sampling low-income families in both states, and African-American families in NC. African-American families were not over-sampled in PA, as the target communities were 95+% Caucasian. Further details on the FLP sampling plan and recruitment procedures are available in Burchinal, Cox, Vernon-Feagans and the FLP Investigators (2008).

Data for this analysis came from the larger project's home interview and assessments at three time points across early childhood, when the child was approximately 7-months (range 5.0 - 13.4 months, M = 7.7 months), 15-months (range 13.5 – 22.3 months, M = 15.7), and 24-months of age (range 22.2 – 35.4 months, M = 24.9). From the total of 1,292 dyads eligible, 1,102 participated in all three home visits and are thus included in the analyses. Based on the mother's race and income-to-needs ratio, this sub-sample was 59.5% White and 40.3% African-American, with 54% of the dyads 200% below the poverty line at all three assessments Many (40.6%) of the mothers were single at all three assessments.

Procedures

Overview. Home visits lasted approximately 2-3 hours, and included self-report measures, semi-structured interviews, and mother-child interaction tasks. Self-report questionnaires include those assessing family conflict and violence, and demographic information. For the mother-child interactions, mothers were given a set of toys and were

instructed as follows: "*We would like for you to play with [child's name] as you normally would if you had a little free time during the day"* (Cox, Paley, Burchinal, & Payne, 1999). Data for this analysis come from the questionnaires and mother-child interaction tasks conducted at 7-, 15-, and 24-month visits, while the physiological data come from the 24-month assessment. Physiological data from the toddler was collected surrounding "challenge tasks" (taken from the Laboratory Temperament Assessment Battery: Goldsmith & Rothbart, 1988) designed to elicit frustration and fear in the toddler, including a toy removal task and a mask presentation challenge (Hellhammer, Wust, & Kudielka, 2009).

Behavioral assessments at the 7-, 15-, and 24-month visits

Inter-parental violence. To assess conflict and violence between partners, the *Conflict Tactics Scale- Couple form revised* (CTS-CF-R) was administered to all mothers, regardless of whether or not the mother's spouse/partner lived in the household with the infant. Mothers completed the scale twice reporting on both, their and their partner's conflict style. The CTS-CF-R (Straus, Hamby, Boney-McCoy, & Sugarman, 1996) consists of 19 items exploring conflict and violence between the partner and the mother in the past 12 months. The CTS-CF-R contains three subscales: verbal discussion, verbal aggression and physical aggression. Following Jouriles and colleagues, only items pertaining to physical or threatened physical aggression, were used to determine inter-parental violence (see Jouriles, Norwood, MacDonald, & Peters, 2001 for review). Subscales from maternal report of her violence toward her partner was summed with maternal report of her partner's violence toward her, resulting in one overall scale of mother-to-partner and partner-to-mother physical aggression at each of the three assessments. Physical

aggression was summed across the three assessments creating a scale representing the total amount of physical violence in the household from birth to age 24 months.

Parenting Behaviors. Mother–child interactions in the free play interactionwere video recorded and later coded to assess levels of parent's sensitivity, detachment, intrusiveness, stimulation, positive regard, negative regard, and animation in interacting with the child (see NICHD ECCRN, 1999). Ratings for each code were made on a scale ranging from 1 (*not at all characteristic*) to 5 (*highly characteristic*) at the infancy assessment and ranging from 1 to 7 at the toddlerhood assessment (scores from the toddlerhood observations were rescaled to a 1-5 range for the current analyses). On the basis of the results of factor analyses conducted with an oblique rotation (i.e., Promax), two broad-based parenting factors emerged for both mothers and fathers: sensitivity and negative-intrusiveness. Sensitivity included five parental ratings: sensitivity (level of responsiveness to child's needs, gestures, and expressions), detachment (emotional unavailability), positive regard (e.g., positive feelings expressed toward child), animation (level of energy), and stimulation of development (appropriate level of scaffolding of activities with child). The factor loadings for maternal sensitivity were .78, -.88, .86, .87, .78 (at early infancy; 7 months), .83, -.89, .82, .85, .81 (at later infancy; 15 months) and .82, -.82, .84, .81, .77 (at toddlerhood; 24 months) and displayed adequate internal consistency at each time point (α = .89 at early infancy; α = .90 at later infancy; α = .87 at toddlerhood). Negative-intrusiveness included two parental characteristics: intrusiveness (the level at which the parent's agenda dominated that of the child) and negative regard (the level of harsh, negative feelings expressed toward child). The factor loadings for maternal negative-intrusiveness were .83 and .88 (at infancy), .83 and .84 (at later infancy), and .88 and .87 (at toddlerhood) and displayed adequate internal consistency at each time point (α = .89 at early infancy; α = .90 at later infancy;

α = .87 at toddlerhood). Reliability was determined by calculating the intra-class correlation coefficients for ratings made by pairs of trained coders. A minimum of 30% of all observations were double coded; any discrepant codings were resolved by conferencing. All coding pairs maintain reliability estimates at above r = .80 for both sensitivity and negative intrusiveness for observations of mothers.

Challenge tasks and biological assessments at the 24-month visit

Laboratory Temperament Assessment Battery (Lab-TAB). Two "challenge tasks" designed to elicit emotional reactivity were administered to the child. The task procedures have been previously validated (e.g., Buss & Goldsmith, 1998; Kochanska, Tjebkes, & Forman, 1998; Stifter & Braungart, 1995), and are only briefly mentioned here. The tasks are from the Laboratory Temperament Assessment Battery (Goldsmith & Rothbart, 1988). For the first task, the toy removal task, mothers were asked to show their child a standardized toy provided by the home visitor. The mother demonstrated the toy and played with the toy and the child. Mothers were then asked to place the toy in a clear container, tighten the lid and then cease interacting with the child for two minutes. After 2 minutes mothers returned the toy to the child and played with, and soothed him/her as necessary for one minute. For the second task, the mask presentation, the child was presented with 4 unusual masks, one at a time. The experimenter wore each mask for 10 seconds while calling the child's name and moving from side to side in front of the child. Mothers were asked not to intervene, but were told they could stop the task at any time. After the completion of the tasks, children were given 1 minute to self-soothe, after which the mother was allowed to soothe the child.

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 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 two challenge tasks 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. The total behavioral reactivity to the task was computed by summing the mean intensities from each of the two challenge tasks. Kappas were .91 for the toy removal episodes and .89 for the mask task. Total behavioral reactivity was computed by summing the mean reactivity for each of the two challenge tasks.

Psychobiological stress reactivity to the challenge tasks. To assess changes in cortisol indicative of the child's adrenocortical response to the emotion challenge tasks, 3 saliva samples were collected: a pre-task sample collected prior to administration of the challenge tasks, a

sample 20-minutes after the final mask was shown (or prior, if the child reached "peak emotional arousal"), and then a final sample 20-minutes later. The child was considered to have reached peak arousal if he or she produced 20-seconds of hard crying at which point the tasks were terminated. Unstimulated whole saliva was collected using hydrocellulose absorbent material and expressing sample into storage 2 ml cryogenic storage vials by centrifugation. After collection, samples were immediately placed on ice, then stored frozen (-20 ^{0}C) until shipped on dry-ice overnight to the Behavioral Endocrinology Laboratory at the Pennsylvania State University. Samples were then stored frozen at – 80 ^{0} C until assayed. All samples were assayed for salivary cortisol using a highly-sensitive enzyme immunoassay (Salimetrics, State College, PA). The test used 25 μl of saliva, had a range of sensitivity from .007 to 3.0 μg/dl, and average intra-and inter-assay coefficients of variation less than 10% and 15%. All samples were assayed in duplicate and the average of the duplicates was used in all analyses. Cortisol levels were natural log transformed to correct for skewed distributions. All analyses used transformed values; for ease of interpretation, raw levels are reported.

Analytical Strategy

First, descriptive statistics are provided to overview the changes in cortisol levels across the task period. Then, partial correlations or ANCOVAs (controlling for sampling time of day) are computed to identify demographic (age, sex, race), infant physical health, maternal mental health (maternal depression, anxiety), and cognitive factors associated with pre-task cortisol levels, and reactivity and recovery to the challenge tasks to be included in the main analyses as covariates. Preliminary GLMM ANOVAs are used to determine the most appropriate error structure of the repeated cortisol measures. Next, incidence rates and demographic characteristics

associated with verbal and physical aggression are presented. Finally, the main analyses employed a series of general linear mixed model (GLMM) repeated measures ANOVA to determine the relationship between IPV exposure and infant physiology. SAS PROC MIXED and SAS PROC GLM (SAS v9.1.3, SAS Institute, Cary, N.C.) are used for all main analyses.

Results

Preliminary and Descriptive Analyses

Percent missing data ranged from 0 to 30%. To avoid bias in estimates associated with listwise deletion, full-information maximum likelihood or multiple imputation (Schafer & Graham, 2002) was used to replace missing values. Five data sets were imputed using the 'impute module' of Imputation and Variance Estimation Software, (IVEware: Raghunathan, Lepkowski, Van Hoewyk, & Solenberger, 2001) launched from SAS.

African-American race, maternal singlehood status, and income level were all positively related to child cortisol levels. However, when all were entered into the model, maternal singlehood status and income level were no longer significant and were trimmed. African-American infants (M = .22, SD = .33) F (982) = 25.96, $p < .0001$ had higher overall cortisol levels than white infants (M = .17, SD = .27). African-American race along with sampling time of day $F(982) = 96.51$, $p < .0001$ were included as covariates in all analyses. Preliminary GLMM revealed an unstructured error pattern to result in the lowest AIC and BIC model fit parameters. Overall, cortisol levels were constant from pre-task to post-task1 $F(969) = 0.05$, p = ns and decreased from post-task1 to post-task2 $F(958) = 7.13$, $p < .01$. Table 3.1 provides the raw means, standard deviations, and correlations of infant cortisol levels before, and in response to

the challenge tasks, along with acute and chronic indices of physical aggression and early maternal sensitivity.

Of the families seen at all three visits, 595 reported at least one instance of violence, with 233 families reporting violence at all three visits. Levels of violence decreased from early infancy (7-months) to later infancy (15-months) $t(1095) = 4.11$, $p < .0001$ but stayed constant from later infancy to toddlerhood (24-months) $t(1095) = 1.39$, $p = ns$. Families exhibiting high levels of cumulative violence had mothers that were more likely to be African American, $t(689.1) = 9.34$, $p < .0001$, single $t(676.3) = 8.22$, $p < .0001$ and have an income to needs ratio 200% below the poverty line $t(1066.4) = 6.34$, $p < .0001$ at all three assessments. Cumulative violence was also related to maternal report of depression r's > .21, p's < .0001 and anxiety r's > .21, p's < .0001 at all three visits. Cumulative violence exposure was negatively related to maternal age $r(1096) = -.22$, $p < .0001$ and maternal education $r(1096) = -.23$, $p < .0001$. Cumulative violence was highly correlated with acute maternal reports of violence at all thee assessments (see Table 1).

Table 3.1 Means, Standard Deviations, and Inter-correlations of cortisol (μg/dL), violence at 7-, 15-, and 24-months and maternal sensitivity at 7-months.

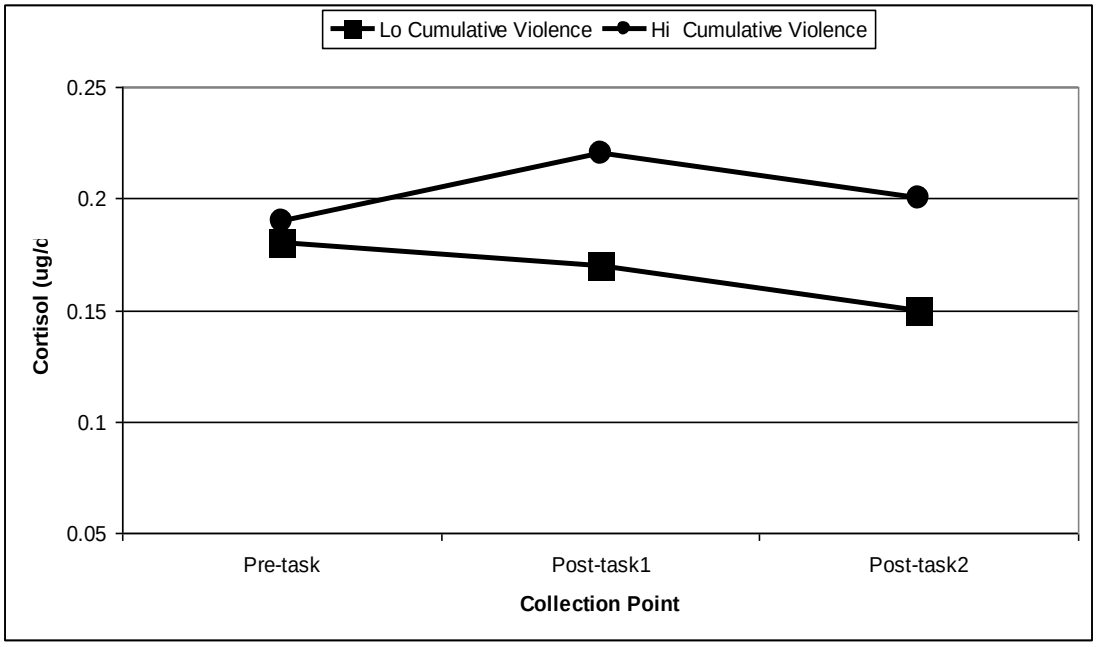
Variable	M	SD	1	2	3	4	5	6	7	8
1. Child Pre-task cortisol	.19	.30	__							
2. Child Post-task1 cortisol	.19	.30	.57***	__						
3. Child Post-task2 cortisol	.18	.27	.79***	.50***	__					
4. 7-month Violence	.40	.80	.05	.12***	.11***	__				
5. 15-month Violence	.32	.78	.02	.09**	.08*	.67***	__			
6. 24-month Violence	.29	.68	.07*	.16***	.13***	.54***	.55****	__		
7. Cumulative Violence	1.02	1.93	.05	.14***	.13***	.88***	.88***	.80***	__	
8. Maternal Sensitivity	14.52	3.90	-.13***	-.14***	-.08*	-.26***	-.25***	-.26***	-.30***	__

Note: * $p < .05$, ** $p < .01$, *** $p < .001$

Main Analyses

Cumulative violence exposure and infant cortisol. The cumulative physical aggression reported in the household across the child's first two years of life was used to predict cortisol levels, reactivity, and recovery across the emotion eliciting task when the child was 24-months old. Cumulative violence exposure interacted with cortisol across the task F (2, 979) = 4.71, $p <$ .01. The interaction was probed using criteria outlined by Aiken and West (1991) with high and low cumulative violence groups specified as one standard deviation above and below the mean, respectively (Figure 3.1). This interaction revealed children exposed to higher levels of physical violence had higher cortisol reactivity from pre-task to post-task1. Specifically, GLMM within the high violence exposure group revealed significant reactivity from pre-task (M = .19, SD = .19) to post-task1 (M = .22, SD = .18) F (104) = 8.58, $p < .01$ but no change from post-task1 to post-task2 (M = .20, SD = .15) F (104) = 2.92, $p < .01$. Children in the low violence exposure group, on the other hand, did not show any change across the task Fs (431, 435) = 0.44 & 1.68, *ps* = ns.

Figure 3.1. Toddlers that are chronically exposed to high levels of violence across their first two years of life exhibit heightened cortisol reactivity.



Maternal reports of physical violence at each of the three assessments were then entered into the model to determine if cumulative violence exposure predicted cortisol levels above and beyond acute exposure. As determined by comparisons of the AIC and BIC with smaller numbers indicating better fit, the model with cumulative violence alone revealed the best fitting model (Table 3.2). Cumulative violence exposure, therefore, is the greatest predictor of cortisol reactivity across the task.

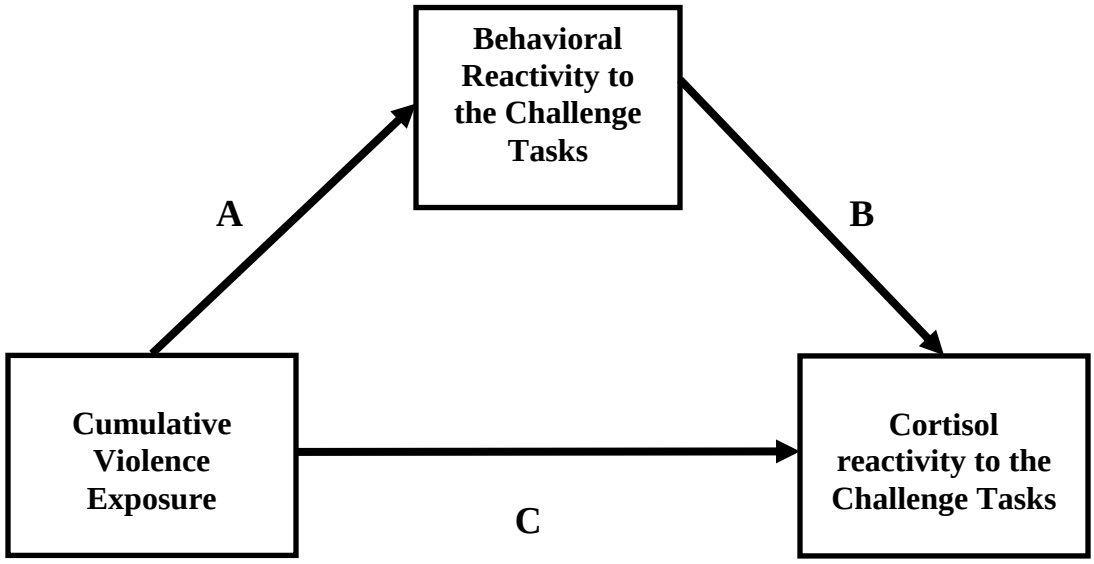
Table 3.2. Model fit parameters and inferential statistics for the Violence x change in cortisol across the task GLMM repeated measures ANOVA. Cumulative violence exposure was the best fitting model, compared to violence at any other time point.

	Model Fit		Inferential Statistics	
Model	AIC	BIC	F	p
7-month Violence	5396.7	5426.0	3.41	.01
15-month Violence	5399.7	5429.0	2.80	.06
24-month Violence	5398.6	5428.0	4.45	.01
Cumulative Violence	5389.2	5418.0	4.71	.01

Follow-up analyses. Behavioral reactivity during the task was then examined as a mediator of the relationship between cumulative violence and toddler cortisol reactivity. Baron and Kenny (1986) define four steps that must be met to determine mediation (Figure 3.2). Within the scope of these analyses the four steps are 1) the cumulative violence is correlated with toddler cortisol reactivity (Pathway C), 2) cumulative violence is correlated with behavioral reactivity (Pathway A), 3) behavioral reactivity is correlated with toddler cortisol reactivity, 4) when controlling for behavioral reactivity, the correlation between cumulative violence and toddler cortisol reactivity is significantly (partial mediation) or completely (full mediation) reduced. Controlling for toddler age and race, Pathway A revealed cumulative violence exposure to be associated with toddler behavioral reactivity to the challenge tasks $F\ (1, 1091) = 15.64$, $p < .001$. Examination of Pathway B revealed behavioral reactivity was related to toddler cortisol

reactivity (1, 1090) = 37.62, $p < .05$. Behavioral reactivity to the challenge task and cumulative violence exposure (pathways B and C) were then entered into the model predicting cortisol reactivity. This final model revealed no change in the relationship between cumulative violence and toddler cortisol reactivity F (1, 1089) = 5.06, $p < .05$. Thus, there is no evidence the relationship between violence exposure and cortisol reactivity is mediated by behavioral reactivity to the challenge tasks.

Figure 3.2 Exposure to higher levels of cumulative violence is related to higher levels of behavioral reactivity to the emotion eliciting challenge task. However, behavioral reactivity did not mediate the association between cumulative violence exposure and toddler cortisol reactivity to the challenge task.



Maternal caregiving behavior as a moderator. Sensitive maternal caregiving behaviors across early childhood were then assessed as moderators of the relationship between violence

exposure and adrenocortical activity. Models with sensitivity at each time point, along with cumulative sensitivity were compared with regards to AIC and BIC, revealing early sensitivity (sensitivity at the 7- month assessment) and its interaction with cumulative risk produced the best fitting model (Table 3.3). Sensitivity at the 7-month visit significantly interacted with cumulative violence exposure and cortisol across the challenge task F (2, 978) = 3.60, p = .01. The three-way interaction was probed using criteria outlined by Aiken and West (1991) with high and low cumulative violence exposure and high and low sensitivity groups specified as one standard deviation above and below the mean, respectively (Figure 3.3). This interaction revealed early sensitive caregiving behaviors buffered children exposed to high levels of physical violence, such that they did not exhibit the high cortisol reactivity seen in children exposed to high levels of violence but without sensitive caregiving. Specifically, GLMM within the high violence/ low sensitivity group revealed significant reactivity from pre-task (M = .17, SD = .14) to post-task1 (M = .21, SD = .16) F (35) = 5.04, $p < .05$ and a decrease post-task1 to post-task2 (M = .17, SD = .11) F (33) = 4.92, $p < .05$. Children in all other groups (high violence/high sensitivity[1], low violence/high sensitivity, and low violence/low sensitivity), on the other hand, did not show any change across the task Fs (23-107) = 0.33 – 1.33, *ps* = ns.

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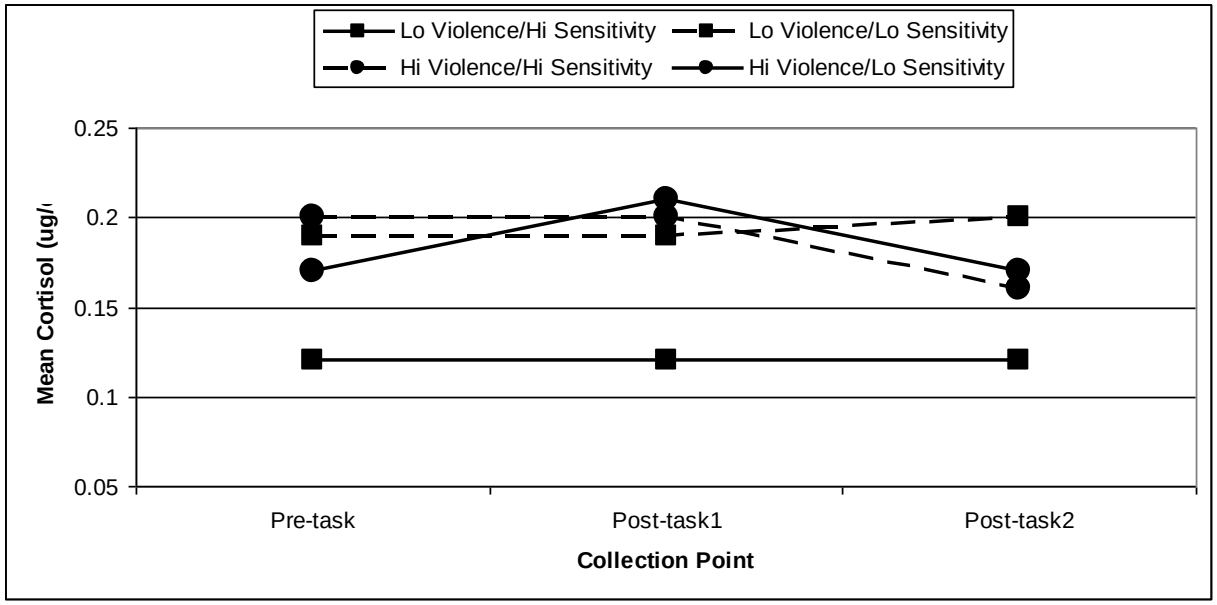
[1] Only four families had cumulative violence exposure and sensitive maternal caregiving that was one standard deviation above the mean on both of these parameters. Therefore, in order to run the GLMM, families with violence one standard deviation above the mean and with sensitive caregiving above the mean were included. This resulted in the inclusion of 30 families in the analysis.

Table 3.3. Model fit parameters and inferential statistics for the Cumulative Violence x Sensitivity GLMM repeated measures ANOVA. Sensitivity at 7 months produced the best fitting model.

	Model Fit		Inferential Statistics	
Model	AIC	BIC	F	p
7-month Sensitivity	5423	5452.4	3.62	.01
15-month Sensitivity	5429.6	5458.9	2.17	.09
24-month Sensitivity	5426.4	5455.8	2.57	.05
Cumulative Sensitivity	5404.6	5433.9	1.55	.21

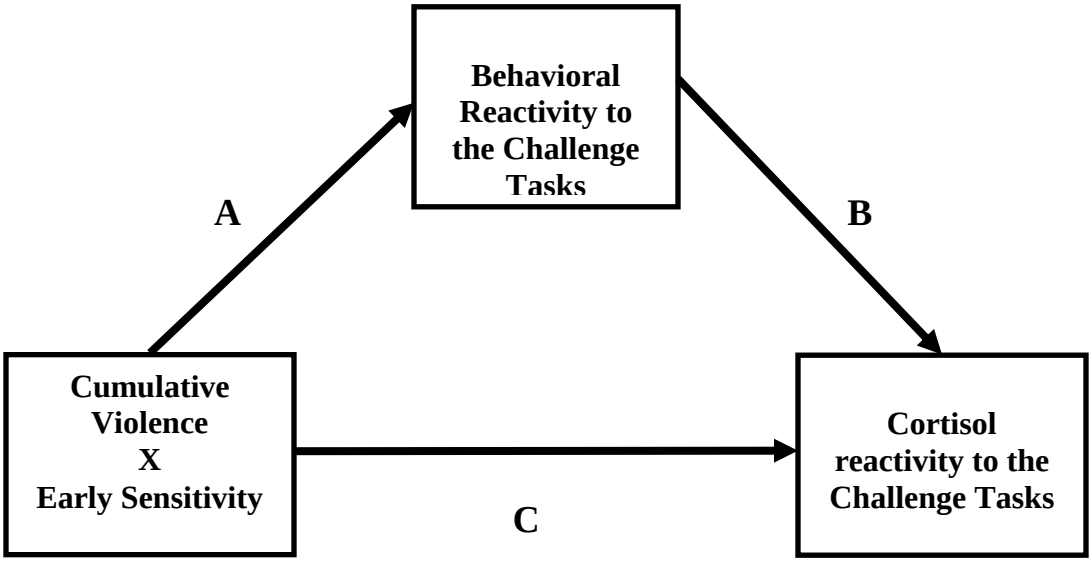
Figure 3.3. Only toddlers with low early sensitive caregiving and chronically exposed to high levels of violence across their first two years of life exhibit heightened cortisol reactivity. Toddlers exposed to higher levels of cumulative violence but with early sensitive caregiving did not exhibit high cortisol reactivity.



Follow-up analyses. Behavioral reactivity during the task was then examined as a mediator of the relationship between cumulative violence X early maternal sensitivity and toddler cortisol reactivity. Baron and Kenny (1986) steps were again followed to determine mediation (Figure 3.4). Within the scope of these analyses the four steps are 1) the cumulative violence X early maternal sensitivity is correlated with toddler cortisol reactivity (Pathway C), 2) cumulative violence X early maternal sensitivity is correlated with behavioral reactivity (Pathway A), 3) behavioral reactivity is correlated with toddler cortisol reactivity, 4) when controlling for behavioral reactivity, the correlation between cumulative violence X early

maternal sensitivity and toddler cortisol reactivity is significantly (partial mediation) or completely (full mediation) reduced. Controlling for toddler age and race, cumulative violence exposure X early maternal sensitivity was not associated with toddler behavioral reactivity to the challenge tasks F (1, 1089) = 0.73, *p* = *ns* (Pathway A). Thus, there is no evidence the relationship between violence exposure X early sensitivity and cortisol reactivity is mediated by behavioral reactivity, and no other pathways were tested.

Figure 3.4 Higher levels of early maternal sensitivity did not moderate the relationship between high cumulative violence exposure and high behavioral reactivity. Therefore, behavioral reactivity is not a mediator of the relationship between cumulative violence and maternal sensitivity, and cortisol reactivity.



Comments/Conclusions

The analyses presented examined the contributions of cumulative inter-parental physical aggression on adrenocortical levels, reactivity, and recovery to an emotion eliciting challenge task during toddlerhood. Behavioral reactivity was assessed as a mediator and sensitive maternal behaviors were examined as moderators. Toddlers exposed to high levels of cumulative violence over the first two years of life had exaggerated cortisol reactivity to the emotion eliciting challenge task. Cumulative violence exposure was a stronger predictor of heightened adrenocortical reactivity than maternal reports of exposure at any single time point across infancy and toddlerhood. Sensitive maternal caretaking behaviors during early infancy protected children from these adrenocortical repercussions. Behavioral reactivity did not mediate these processes. Findings are discussed within cumulative risk, allostatic load, and evolutionary perspectives.

Studies of contextual risk rarely find single risk factors to explain individual differences in the development of atypical behaviors. However, a child's cumulative exposure to risk has proven to be a strong indicator of negative mental and physical heath outcomes (Rutter, 1983). Adversities have potentiating effects on the development of disorder and combinations of stressors result in more profound repercussions than two risks considered separately (Rutter, 1980; Seifer, 1995). Demanding and stressful environments require the body to call upon a higher level of physiological reserves to mobilize enough resources for an adequate response. The resulting wear and tear on the body from ongoing physiological activations and attempts at recovery are hypothesized to be the mechanism by which chronic environmental stressors influence mental and physical health outcomes (Heim, Owens, Plotsky, & Nemeroff, 1997; McEwen & Stellar, 1993; McEwen, 2004).

Conflict and violence within a family is not constant, and fluctuations in family discord can occur in response to family situations and changes (e.g., Fox, Benson, DeMaris, & Van Wyk, 2004; Gelles, 1974; Rogge & Bradford, 1999). Furthermore, changes in family composition may mean the violent family member or conflict instigator may no longer be present. Thus, children raised in "violent families" may or may not be chronically exposed to these highly stressful and disturbing events. This means acute accounts of conflict and violence may not be indicative of the repercussions of overall exposure. In support of a cumulative risk model (e.g., Rutter, 1980), chronic exposure to family violence was associated with heightened adrenocortical reactivity to an emotional stressor, above and beyond any acute report of violence. That is not to say that acute reports of violence in the home did not explain variance in adrenocortical reactivity, it is that high cumulative exposure was the strongest indicator of subsequent adrenocortical functioning.

Despite exposure to risk, some children emerge largely unscathed. Thus, developmental researchers continue to strive to understand the individual, familial, and extra-familial characteristics or conditions that suppress or neutralize the detrimental outcomes associated with risk exposure (e.g., Richman & Fraser, 2001). Caregivers have been shown to be critical in facilitating resilience by in structuring the infant's early environment in such a way that appropriate socio-emotional skills are developed (e.g., Bowlby; 1979; Sroufe, 2000; Jahromi et al., 2004). Particularly important may be the caregiver's role in programming the degree to which the HPA axis responds to stressors, and how efficiently this response is deactivated (Szyf, Weaver, & Meaney, 2007; Gunnar, Morison, Chisholm, & Schuder, 2001) thus buffering the physiological burden of exposure to cumulative risk. In support of these ideas, early (within the first year of life) sensitive maternal behaviors completely ameliorated the heightened reactivity

exhibited by toddlers cumulatively exposed to high levels of violence across their first two years of life. Despite multiple exposures to high levels of violence, toddlers raised with early sensitive maternal caregiving were physiologically indistinguishable from those not exposed to violence. This profound difference was only found for early sensitivity, such that high maternal sensitivity during later infancy or toddlerhood, did not buffer the toddler from adrenocortical over reactivity. Thus, sensitive caretaking behaviors particularly during periods such as early infancy may be critical in fostering the regulatory abilities needed to successfully cope with chronic stress.

The examination of sensitive and critical periods within the field of child development has waxed and waned (Knudsen, 2004). During the early part of the 1900s this area of research generated a great deal of interest; however, subsequent developmental studies revealed exposure to adversity at anytime could result in profound behavioral and physiological changes (Rutter, 2004). Recent quantitative genetic and neurobiological advances have once again highlighted certain sensitive periods to be crucial in programming lifelong behavioral and physiological responses (see Mousseau & Fox, 1998 for review). Animal models have validated the epigenetic transmission of stress responsive profiles (Meaney & Szyf, 2005) revealing maternal behaviors directly influence the overall adrenocortical activation in response to a stressor and the level of negative feedback (i.e., recovery from HPA reactivity). The present analyses may suggest similar processes in humans. Sensitive caregiving during the first year of life may be fundamental in setting a normative threshold of adrenocortical reactivity to stress. Specifically, the results indicated the influence of maternal caregiving on adrenocortical reactivity was strongest during early infancy than any other time point. In addition, the influence of positive maternal caregiving was strong enough to promote resilience even in the face of continued exposure to violence, by

producing cortisol reactivity that was indistinguishable from children not exposed to violence. These findings fit within a growing literature linking early caregiving and long-term physiological activity (Gunnar et al., 2001; Rutter, 2004; Fries, Shirtcliff, & Pollak, 2008).

Past literature examining stress physiology highlighted *hyper* reactivity to as a potent risk factor facilitating the development of mental and physical health problems ranging from cardiovascular disease to depression (McEwen, 2002). Recently, it has become apparent that being either overly responsive or having under arousal (the inability to mount an effective physiological response) in the face of a stressor may be indicative of future maladjustment (Fries, Hesse, Hellhammer, & Hellhammer, 2005; McEwen & Seeman, 1998; Susman, 2006). Attenuation of the stress response may be an adaptive physiological response thereby reducing the allostatic load and subsequent wear and tear on the body associated with repeated physiological fluctuations. Despite being adaptive in the short run, these physiological alterations may still be associated with problem behavior in the long run (Susman, 2006). Thus, even though toddlers exposed to violence but with sensitive early caregiving environments exhibited seemingly normative *adrenocortical* patterns, these children may still not develop typical *behavioral* patterns. The analyses partially illustrate this point by revealing early sensitivity did not buffer the increased behavioral reactivity related to violence exposure. In other words, children exposed to violence but with early sensitivity still exhibited higher behavioral reactivity than those not exposed to violence, despite showing similar cortisol reactivity as non-exposed children. To date, other than a few noted exceptions (Gunnar, Borderson, Nachmias, Buss, & Rigatuso, 1996; Quas, Hong, Alkon, & Boyce, 2000; Towe-Goodman et al., under review), few studies have examined how exposure risk relates to the dissociation of behavioral and adrenocortical reactivity (i.e., high levels of behavioral reactivity with normative adrenocortical

reactivity) and how these patterns facilitate or attenuate negative mental and physical health outcomes. These ideas will be discussed more fully in the following paragraphs.

Theoretical Implications

Stress responsive physiological systems activate to prepare the individual to successfully respond to, and overcome a challenge. However, physiological pathways are only activated in conditions where the "behavioral surface" is unable to accommodate the challenge (Gottlieb, 1992). Within this conceptualization, only if changes in behavior or subjective experience do not alleviate the stressor, do psychobiological components activate. Therefore, heightened reactivity represents an inability to successfully respond behaviorally. Overall, in the present analyses the toddlers did not show an adrenocortical response to the task and therefore physiological resources were not necessary in coping with the challenge. However, toddlers cumulatively exposed to high levels of violence did exhibit adrenocortical reactivity along with increased behavioral reactivity to the emotion eliciting challenge tasks. Interestingly, these two pathways seem to be independent, such that cumulative violence exposure directly impacts both behavioral and adrenocortical reactivity to an emotional challenge. Thus, the heightened behavioral reactivity did not explain the relationship between violence exposure and cortisol reactivity. Confirming the 'loose coupling' between behavioral and physiological responses (Gunnar & Davis, 2003), the current analyses found that despite behavioral and adrenocortical reactivity being significantly related, adversity may independently dysregulate these responses to challenge.

Recognition of the loose coupling between behavioral and physiological responses presents new opportunities to explore how these processes lead to negative mental and physical

health outcomes. An organism's response to a stressor is multi-faceted comprised of multiple behavioral, cognitive, and physiological responses. Recently, developmental researchers have emphasized the importance of using multiple biological measures to assess the psychobiology of stress (e.g. Bauer, Quas, & Boyce, 2002) highlighting the potential for physiological systems to interact. However, an individual's behavioral response should also be considered as part of this response profile, with the potential to interact with, or moderate (as oppose to mediate) the physiological response (Gunnar et al., 1988; Gunnar & Donzella, 1999). The present analyses illustrate the potential for adversity to independently affect the behavioral and adrenocortical systems, and thus, examining their dissociation in the context of risk may be particularly informative in understanding the development of pathology. It has been argued that dissociations between behavioral and biological indices represent increased adaptability by promoting the individual's ability to respond differently, and thus more specifically, to the challenge at hand (Quas et al., 2000). However, at least two studies have found insensitive and unresponsive maternal behavior to be related to bio-behavioral dissociations (Gunnar et al., 1988; Towe-Goodman et al., under review). Thus, dissociations may be indicative of inconsistent or inadequate external regulation, or in the case of the current analyses, inconsistent environmental signals (i.e., positive maternal behavior but chronic exposure to violence).

According to epigenetic and evolutionary-developmental models, surrounding ecological characteristics shape an individual's biology to be distinctive from individuals in other ecologies. Maternal behavior is thought to be a mechanism for this biological structuring, portraying information about the surrounding environment and serving as a signal of the environment's quality. Thus, the maternal behaviors *program* the offspring's lifelong phenotypic responses to be uniquely sensitive to the specific contextual and environmental demands in which the

offspring is raised. In this perspective, unpredictable environments induce harsh maternal behavior facilitating the highly reactive physiological profiles necessary to respond to increased environmental demands (e.g., Boyce & Ellis, 2005). The present analyses support this view, with children exposed to chronic stress (in the form of inter-parental violence) and low maternal sensitivity exhibiting heightened cortisol reactivity. Heightened adrenocortical reactivity has been conceptualized as increased "biological sensitivity to context" (Boyce & Ellis, 2005) or a physiological profile that allows the individual to garner information from the environment and respond appropriately. However, Boyce and Ellis extend the epigenetic findings to also hypothesize high reactive profiles to be facilitated in highly *supportive* environments, allowing the individual to acquire the benefits of the positive interactions. The current findings do not support Boyce and Ellis' suggestion that non- violent contexts and high maternal sensitivity (i.e., a highly supportive environment) result in high physiological reactivity. In fact, within this population, these were the children that did not exhibit any reactivity to the emotional challenge.

Keeping within an evolutionary framework, it may be misleading to only view the adrenocortical reactivity exhibited by violence exposed children as maladaptive and potentiating negative mental and physical health outcomes. By definition, an adaptation is an environmentally induced change in anatomy, physiology, or behavior contributing to an individual's ability to survive and reproduce (Williams, 1966). Thus, to assess whether heightened reactivity truly is maladaptive it must be determined if this response confers a disadvantage over lower reactivity profiles, *specifically in the context of violence* (e.g., Hofer, 2006). In other words, while over activity of the HPA axis may be related to poor mental and physical health outcomes longitudinally, within in the immediate context of violence, it may be adaptive and necessary for survival. Adrenocortical reactivity functions to prepare and aid in the organism's response to

challenge. These children may be inundated with stressors and thus a higher level of resources may be needed to behaviorally, mentally, and physically cope. Thus, heightened reactivity in the violence exposed children may be an adaptive response, necessary for successfully navigating the extreme stressors of their day-to-day lives. Recently, Hofer (2006) has discussed this issue and its implications for intervention. In sum, viewing these negative responses as adaptive does not diminish the need to intervene on behalf of the child, but underscores the necessity of intervening at the maternal or familial levels. Teaching a child more positive coping styles may be futile if these responses to stress are ineffective in helping the child deal with aversive family situation. Only if these stressors are at least partially alleviated will intervention efforts be successful (e.g., Field, 1998).

The Family Life Project

Being born into a poor and rural population represents profound risk, jeopardizing the mental and physical development of children. Unfortunately, this population has been under studied, and therefore the characteristics of the communities, families, and individuals that shape child development in the poor rural context, and potentially serve as factors of risk and resilience, are unknown. Data for the present analyses come from the Family Life Project, an epidemiological sample representative of children living in rural Appalachia and the Black South. Data for these analyses come from the Family Life Project, an epidemiological sample representative of children living in rural Appalachia and the Black South. The epidemiological sample allows for greater generalizability to individuals living in these conditions. As such, there is increased importance in integrating findings across reports from this multi-site program project

in an effort to reveal how these observations collectively advance our understanding of the concomitants and sequelae of early individual differences in HPA reactivity and recovery.

Our first report (Blair et al., 2008) revealed engaged maternal behaviors to be associated with increased cortisol reactivity at 7-months and with overall lower levels of cortisol at 15-months. The next paper in our series (Towe-Goodman et al, under review) explored self-regulatory strategies and parenting behaviors associated with profiles of associated and disassociated emotional and adrenocortical reactivity at the 7-month assessment. Similar to the Blair et al analyses, Towe-Goodman et al found evidence that infants with less engaged mothers were more likely to show reduced adrenocortical reactivity but high behavioral reactivity. Another paper coming out of this project (Fortunato et al., under review) found early engaged maternal behavior to result in consistent rapid adrenocortical recovery to the challenge tasks at both the 7- and 15- months assessments. Together, these papers highlight the importance of early maternal caregiving in structuring reactivity of the HPA axis, recovery from activation, and HPA axis coordination with behavior. The present findings extend these observations in important ways. Primarily, the analyses present here were the first to examine chronic exposure to adversity in relation to infant adrenocortical levels and reactivity. This non-urban impoverished sample was chosen specifically because the children are at high risk for the development of maladjustment. Therefore, examining prevalent and chronic negative contextual influences to which this population is exposed is particularly important in furthering our understanding of what constitutes 'risk' in the lives of these children. Secondly, the present analyses reveal the influence of early maternal behavior to be critical in facilitating long-term physiological regulation, even in the face of adversity. Not all children exposed to risk develop problems and the identification of factors that may protect children elucidates potential pathways of

intervention. In sum, while past analyses on these data have underscored the influence of maternal behavior, the present analyses highlight the potential for positive maternal behavior to promote physiological resilience in risky environments (e.g., family violence).

During early childhood (i.e., infancy and toddlerhood) the world that a child directly interacts with is limited. Larger socioeconomic and demographic pressures may not have a direct influence on the child's development. However, previous research has shown these factors to be a significant source of family stress and therefore may indirectly influence the developing child through changes in parent-parent and parent-child interactions (Conger et al., 1994). Similarly, during infancy, the impact of negative community, or as seen in the current analyses, familial characteristics may only be detrimental in the context of poor parenting behavior particularly during sensitive periods of development.

Limitations and Future Directions

This study had several limitations. First, researchers have emphasized the importance of using multiple measures to assess the psychobiology of stress (e.g. Bauer, et al., 2002). The stress response is a multi-system response that is characterized by both an autonomic nervous system response [comprised of the sympathetic (SNS) and parasympathetic nervous systems (PNS)] and an HPA axis response. These physiological systems are in a constant process of coordinated adjustment and fine-tuning, meeting the individual's needs in responding to an ever changing environment. Examining measures of SNS and PNS arousal would give further insight into the physiological processes associated with verbal and physical aggression exposure. Past studies have found outcomes associated with exposure to conflict and violence to be moderated by activation of the SNS, the PNS, and the interaction of these two systems (see El-Sheikh et al.,

2009 for review). Given the HPA axis activation may also play an important role in the link between violence exposure and maladjustment, future studies should assess both the autonomic nervous system and the HPA axis to determine if each individual system or the coordination of the two systems facilitates risk or resilience. Similarly, other indices of behavioral responses should be examined into future studies. The present analysis was limited in that only behavioral reactivity was explored as a mediator of adrenocortical reactivity. Individual differences in behavioral *regulation* (e.g., gaze aversion, thumb sucking) may be more indicative how effective the behavioral surface is at responding, and may prove to be a more useful tool in understanding when physiological responses are evoked. Second, despite controlling for sampling time of day in all analyses, the large variation in sampling time may have contributed to a portion of the unexplained variance. This may have interfered with attempts to estimate the relation of family and child characteristics to cortisol levels and cortisol reactivity. Third, this set of analyses is preliminary in determining the influence of conflict and violence exposure on individual difference in stress responsive physiology. Clearly, the next step is to examine the mediating or moderating role of stress physiology in the development of mental and physical health problems. Longitudinal studies, such as this one, are especially important given the dynamic nature of conflicted families (e.g., frequent changes in cohabitation, waves of increase and decrease in conflict and violence). While the present analyses indicate there may be differences in adrenocortical activation based on risky family exposure, it is not known if these changes are lasting, independent of continued exposure or if future experiences might buffer or potentiate these differences. Given the dynamic nature of families, more research needs to be done examining intra-familial differences in interactions to determine what particular styles present the largest risk factors for the children exposed. Fourth, the sample for this analysis is an

epidemiologically valid representation of children and families living in the areas from which it is drawn. As such, it is representative of a substantial segment of the low-income population in the United States in non-urban areas. While this is a major strength in allowing the findings to be generalizable to a large portion of the rural United States, the generalizability does not extend to urban populations. Future studies should be aimed at other populations to increase our understanding of how these processes operate across contexts.

Conclusions

Developmental scientists are aware of the repercussions of acute and chronic exposure to adversity on human development. Extreme trauma at any point in an individual's life has the potential to exert life-long psychological, behavioral, or emotional changes. However, two agendas are clear within the field of developmental psychopathology; 1) identify risk factors that potentiate the development of pathology and 2) identify individual, social, and environmental factors that contribute to resilience (e.g., Rutter, 1980). The present analyses may further our progress toward both goals. In support of a growing body of evidence, children are unduly susceptible to the powerful influence of violent, hostile, and coercive interaction styles. However, even in families with these interaction styles, certain positive exchanges may occur. This presents a unique opportunity to uncover and understand potential moderating factors; factors that encourage resilience despite exposure to extreme negativity. The current work underscores the need for a detailed understanding of the caregiver qualities that are critical for fostering the life-long physiological and behavioral regulatory and coping abilities needed to overcome adversity.

CHAPTER 4

General Discussion

In the life of a young child, the family is of central importance. Human infants and toddlers are completely dependent on their caretakers for food, shelter, protection, and garnering information that will facilitate survival. Thus, it would only make sense that the human brain would evolve special sensitivities to familial and caregiver interactions, particularly during infancy and early childhood. In families characterized by positive, warm, and supporting relationships, children learn and develop appropriate socio-emotional competencies and benefit from better overall mental and physical health. Conversely, exposure to early family stress may jeopardize both short- and long-term well-being by creating deficits in behavioral, emotional, and physiological regulation. Furthermore, stressful circumstances during childhood may be particularly detrimental as resources are diverted from important growth and development functions and shunted into more survival-based processes.

During the first two years of life the human brain nearly triples in size due to cell growth, and takes up almost half of the infant's caloric requirements. Brain components that control stress responsive physiological systems have an exceptionally large proportion of this post-natal growth. During this period of growth, environmental and contextual experiences have a huge impact on how the neuronal connections are made, how sensitive they are to stimulations, and on the behavioral and neuroendocrine outputs of these systems. As expected, emotional and physical stressors such as abuse, neglect, parental divorce, maternal deprivation, and malnutrition (see Tarullo & Gunnar, 2006 for review) and as found in the present analyses, inter-parental conflict, can influence physiological stress responses.

Integrating the findings across the analyses in the previous two chapters reveals a theme consistent with a developmental perspective and a family systems framework. Primarily, during infancy a child's world is limited to interactions with family members and other caregivers. It is therefore understandable that adverse contextual (Congers et al., 1994) or family interaction patterns would only have implications when paired with caregiver dysfunction. In Study 1, when the children were in late infancy, it was only within the context of certain maternal behavioral patterns did inter-parental conflict and violence result in heightened adrenocortical levels. Analyses revealed the combination of high verbal aggression and low maternal sensitivity was related to higher levels of cortisol across all three sample collection points (pre and post task). Similarly, high physical aggression and low maternal negative-intrusion was related to higher overall levels of cortisol. While these two findings may seem contradictory (see chapter 2 concluding comments for a discussion aimed at disentangling the pairing of aggression type with the type of maternal behavior), the pattern of findings clearly indicate parent-child interactions and the inter-parental context in which they occur may be important in understanding how familiar process combine to shape and influence physiological development.

Analyses in Study 2 examined adrenocortical functioning in these same children as they grew into toddlers. Toddlers have an increased understanding of the world around them and a better comprehension of causes and consequences, thus, there is greater potential for inter-parental violence to directly influence these children. The analyses for Study 2 supported this notion, finding exposure to chronic violence over the first two years of life to be directly related to heightened cortisol reactivity during toddlerhood. Moreover, children no exposed to high levels of cumulative violence exhibited no reactivity to the task, potentially indicating a physiologically inappropriate response. Caregiving did still prove important, with positive

caregiving during infancy buffering the toddlers from heightened cortisol reactivity even in the face of inter-parental violence. Thus, children that were exposed to the cumulative violence but had mothers that were sensitive and responsive to their needs, did not show cortisol reactivity to the challenge tasks.

At first glance there may seem to be a discrepancy between the heightened overall cortisol *levels* at the infant visit and the heightened *reactivity* at the toddler visit in relation to family conflict and violence exposure. However, it is possible that increased reactivity is exhibited at both visits. Infants develop wariness to strangers sometime in the second half of their first year of life, roughly between 6- and 9- months of age. Stranger anxiety is thought to coincide with the infant's ability to move independently and represents the infant's understanding that not all people are the infant's primary caregiver (see Thompson & Limber, 1990 for review). Stranger anxiety is thought to peak between 12 and 15 months of age, and the infant visit (at approximately 15-months) was specifically designed to capture this time of heightened fear reactivity (e.g., Scarr & Salapatek, 1970). It is therefore possible that the higher cortisol levels in the infants exposed to verbal and physical aggression could have been increased cortisol reactivity to the presence of a stranger (i.e., the home visitors conducting the visit and interview). The stranger-induced cortisol reactivity may have masked any subsequent adrenocortical reactivity during the challenge tasks. While this is speculative, there is evidence that risk increases stranger anxiety (e.g., Main, 1983; Thompson & Lamb, 1983) and stranger/separation anxiety is related to cortisol reactivity (Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996). Thus, it is possible that both analyses represent increased adrenocortical reactivity. Regardless, both sets of analyses highlight the importance of family conflict and violence in the development of the HPA axis and the potential for exposure to these negative

interaction patterns to dysregulate adrenocortical activity. Given the prevalence of violence and children exposed to these traumatic events, these findings should not be taken lightly.

The United States is the most violent nation in the industrialized world, prompting violence to be considered "a defining characteristic of American society" (Cicchetti & Lynch, 1993). This is especially relevant for young people. Firearms account for the majority of deaths in teens and young adults. Children in the United States are exposed to violence everywhere; in their homes, in their neighborhoods, and even in their schools. Our children are steeped in violence, conflict, hostility, and aggression. The short- and long-term repercussions on America's youth are astonishing. In short, violence begets violence. Each generation is raised to be more violent than the next as children are exposed to more and more traumatizing and horrific scenes that become common day occurrences. In fact, it is harder to find children raised in urban environments that have *not* witnessed a stabbing, a shooting, a mugging, or some other sort of violent crime by the time they are in elementary school than those that have witnessed these traumatic events (Bell & Jenkins, 1993). And in the end violence is internalized, and repeated.

Exposure to violence not only increases the likelihood of violent behavior, but also a wide range of mental and physical health problems. Exposure to abuse or household dysfunction during childhood has been related to several of the leading causes of death in adults. Adults with multiple exposures to these adversity had 4- to 12-fold increased health risks for alcoholism, drug abuse, depression, and suicide attempt; a 2- to 4-fold increase in smoking, poor self-rated health, and sexually transmitted disease; and a 1.4- to 1.6-fold increase in severe obesity. Furthermore, the number of categories of adverse childhood exposures showed a graded relationship to the presence of adult diseases including ischemic heart disease, cancer, chronic lung disease, skeletal fractures, and liver disease (Felitti et al., 1998). In effect, more and more

children are being raised surrounded by adversity only to grow up to be burdened with severe mental and physical health problems.

The current governmental and privatized system in place to deal with health concerns is a system of health *care*. In short, what this means, usually, is treatment is not begun until after an individual is diagnosed with a disease or illness. The top three killers in the United States are 1) heart disease, 2) cancer, and 3) cerebrovascular disease (National Center for Health Statistics, 2006). All three of these illnesses are considered chronic diseases and require extensive and expensive long-term care. The response of the present day health *care* system is to diagnose then intervene, providing continued care to chronically ill patients. Unfortunately, such efforts are too little, too late and diagnosed individuals are rarely "cured". The United States spends 6 times more on end-of-life care, such as this, than on healthcare services geared toward children (National Center for Health Statistics, 2006). A person thinking within the confines of the current health care paradigm would say, "of course we spent more money on the old, the elderly is the population with the most disease". However, thinking within a *health* paradigm one would not be interested in simply ameliorating symptoms and treating the disease, but stopping the disease before it occurred. We are currently wholly focused on health *care*: delivering health services to individuals whom are already sick. But what if, instead of caring for the sick, we *prevented* the disease?

During the 1990s massive popular media attention was directed at the notion that the first two to three years of life were critical in facilitating healthy brain and behavioral development (see Nelson & Bosquet, 2005, for review). A plethora of studies revealed critical periods for the development of cognitive, emotional, linguistic, physical, immunological, and physiological functioning (e.g., Albers et al., 1997; Carlson & Earls, 1997; Cicchetti & Rogosch, 2001; Fisher,

Ames, Chisholm, & Savoic, 1997). While it is apparent that the brain continuously develops well into adulthood, the brain shows considerable plasticity and therefore increased susceptibility during early childhood. It would follow that the pathways to healthy habits, behaviors, and attitudes are paved during infancy. To adequately address the current trends in health (i.e., the prevalence of chronic diseases) precipitated by early stress, a fundamental shift in public policy spending must occur, prioritizing early positive socio-emotional development. Ameliorating early stress has the potential to drastically alter health trajectories and set individuals on a route to better mental and physical health throughout life.

Violence and hostility sets in motion detrimental physiological processes ultimately leading to illness, disease, and death. Children, the innocent bystanders of adult aggression, carry this burden. The analyses presented in the previous chapters reveal infants exposed to conflict and violence to have heightened cortisol levels and reactivity. Infants! Children as young as 15 months are already exhibiting physiological dysregulation in relation to violence exposure. Together, these analyses along with past research indicate that the pre-disease pathways leading to chronic diseases such as heart disease, could be starting as early as infancy. Specifically highlighted in these analyses is the importance of early negative familial relationships in determining these maladaptive trajectories, and the possibility that sensitive maternal caretaking may mitigate this risk.

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Curriculum Vitae

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Education

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Grants

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Selected Publications

Hibel, L.C., Granger, D. A., Blair, C., Cox, M., & The Family Life Project Investigators (in press). Intimate partner violence moderates the relationship between mother-infant adrenocortical responses to an emotional challenge. *Journal of Family Psychology*

Granger, D. A., **Hibel, L. C.,** & Fortunato, C. K., (in press). Medication effects on salivary cortisol: Mechanisms of action, a "watch list", and tactics to minimize impact in biobehavioral research. *Psychoneuroendocrinology*

Blair, C., Granger, D. A., Kivlighan, K. T., Willoughby, M., Greenberg, M., **Hibel, L.C**., Fortunato, C. K. & 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*. 44,1095-109.

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