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Interparental Violence, Maternal Emotional Unavailability and Children's Cortisol Functioning in Family Contexts

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Abstract

The goal of the present study was to examine the specificity of pathways among interparental violence, maternal emotional unavailability, and children's cortisol reactivity to emotional stressors within the interparental and parent-child relationships. The study also tested whether detrimental family contexts were associated on average with hypocortisolism or hypercortisolism responses to stressful family interactions in young children. Participants included 201 toddlers and their mothers from impoverished backgrounds who experienced disproportionate levels of family violence. Assessments of interparental violence were derived from maternal surveys and interviews, whereas maternal emotional unavailability was assessed through maternal reports and observer ratings of caregiving. Salivary cortisol levels were sampled at three timepoints before and after laboratory paradigms designed to elicit children's reactivity to stressful interparental and parent-child contexts. Results indicated that interparental violence and mother's emotional unavailability were differentially associated with children's adrenocorticol stress reactivity. Furthermore, these family risk contexts predicted lower cortisol change in response to distress. The results are interpreted in the context of risky family and emotional security theory conceptualizations that underscore how family contexts differentially impact children's physiological regulatory capacities.

For young children, the family environment is considered a primary agent in shaping their ontogenetic development. Optimal rearing environments characterized by family relationships that are nurturing, engaged, and responsive have been associated with positive developmental outcomes in children. In contrast, harsh, fractured and unpredictable family contexts have been associated with altered functioning (e.g., Cicchetti & Howes, 1991; Cummings & Davies, 1994; Dunn & Davies, 2001; Sturge-Apple, Davies, & Cummings, 2006). Over the past several decades, developmental research couched within family frameworks has endeavored to explicate the underlying mechanisms that might account for such associations. Correspondingly, research has delineated the explanatory role of children's emotionality (e.g., Cummings, Schermerhorn, Davies, Goeke-Morey, &

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Cummings, 2006), cognitive functioning (Grych, Harold, & Miles, 2003; Jouriles, Brown, McDonald, Rosenfield, & Leahy, 2008; Sturge-Apple, Davies, Cummings, Winter, & Schermerhorn, 2008), and behavioral regulation (e.g., Gordis, Margolin, & John, 2001) in these family process models. However, relatively little is known about children's physiological functioning in the context of multiple family relationships. This gap is particularly significant given the central role ascribed to children's physiological functioning in specific family contexts by prevailing conceptualizations of family risk (e.g., Boyce & Ellis, 2005; Repetti, Taylor, & Seeman, 2002). Towards the goal of identifying family correlates of aberrations in young children's physiological functioning, we specifically explore whether interparental violence and maternal caregiving difficulties are differentially predictive of children's adrenocorticol reactivity to standardized, laboratory procedures designed to elicit children's distress in the interparental and parent-child relationship.

Neurobiological frameworks highlight the significance of understanding the functioning of the hypothalamic-pituitary-adrenal (HPA) axis stress response system in risky family environments (e.g., Cicchetti, 2002; Repetti, Taylor, & Saxbe, 2007; Susman, 2006). The HPA axis serves as a primary means for marshaling resources in response to environmental threat and stress. The end product of HPA activation is the glutocorticoid hormone cortisol. Increases in cortisol levels in response to an environmental stressor serve the adaptive function of amplifying cognitive processing of emotionally significant events, as well as mobilizing energy and physiological resources towards addressing the stressor (e.g., Gold & Chrousos, 2002; Gunnar & Quevedo, 2007). For young children, interparental aggression and insensitive harsh caregiving behaviors are regarded as particularly salient environmental pathogens by virtue of their pernicious implications for the preservation children's personal safety and well-being (Cicchetti & Rogosch, 2001; Margolin, 2001). Thus, in light of the stress-sensitive nature of the HPA axis, family risk conceptualizations postulate that both interparental aggression and emotional unavailability by caregivers are potent predictors of individual differences in children's adrenocortical functioning.

Our ability to simultaneously document associations among interparental and parenting risk factors and children's cortisol functioning allows powerful tests of two contrasting models of the stress response. First, a stress-generality model proposes that the HPA response is uniform across stressors (e.g., Selye, 1975) suggesting that children are at increased risk for dysregulation regardless of the specific stressor in the environment. Translated to understanding pathways between family stress and children's physiological reactivity, a primary prediction of this approach is that exposure to difficulties in any specific family relationship will evoke a physiological response that is comparable across stressors in multiple family relationships. In attesting to the lasting influence of this model in physiological research, assessment strategies in studies have predominantly relied on measurement of children's physiological reactivity to a single stressor (e.g., Trier task) that is conceptually unrelated to the proposed risk factor (e.g., parenting difficulties, interparental conflict). However, more recent conceptualizations emphasizing specificity in the nature and implications of stressors have increasingly challenged these earlier assumptions (e.g., Dickerson, Gruenewald, & Kemeny, 2004; Wiener, 1992). According to this perspective, specific environmental experiences act as specific signals that elicit reactivity patterns that are exquisitely designed to assist individuals in coping with comparable threats in subsequent contexts (Kemeny, 2003).Toward our aim of understanding pathways between family risk factors and children's physiological reactivity to stressors, a derivative prediction is that earlier experiences in specific family relationships may assume particular significance in predicting children's patterns of neurobiological reactivity in those relationship contexts rather than to a wide array of stressful events. Accordingly, the present study is, to our knowledge, the first empirical foray into examining the relative utility of these alternative

frameworks in delineating pathways between family processes and children's physiological reactivity to stressors in multiple family relationships.

These two broad perspectives yield some general predictions about the nature of associations between experiential histories of stress and stress reactivity, but they offer little specific guidance on hypothesizing how and why stress and threat within family contexts may elicit a specific profile of physiological stress reactivity in young children. Emotional security theory (EST; e.g., Davies & Cummings, 1994; Davies & Sturge-Apple, 2007) may provide valuable direction and precision in determining the relative viability of the stress-generality and stress-specificity models in family frameworks. EST views emotional security in interparental and parent-child relationships as a primary goal for children. By extension, children's experiential histories of difficulties within interparental and parent-child relationship systems undermine their emotional security within these contexts. First, EST proposes that children's exposure to bouts of violence, aggression, and conflict between parental figures is a particularly potent threat to children, amplifying their concerns about safety and security within this context. In contrast, EST proposes parental difficulties in providing emotionally available, sensitive, and responsive caregiving undermine children's emotional security when confronted with external threat and without a supportive and consistent caregiver to turn to (e.g.,Cicchetti, Rogosch, & Toth, 1998; Levondosky & Graham-Bermann, 2000). Given its emphasis on differentiated pathways in family processes, EST provides a conceptual blueprint for expecting specificity in associations between children's experiential histories in the interparental and parent-child relationship and their physiological reactivity to laboratory paradigms designed to activate children's concerns about emotional security within these specific family relationships.

A small corpus of findings examining children's HPA regulation within risky family contexts have independently supported associations among emotionally salient and threatening events within the family context and children's HPA functioning. Davies and colleagues (2007) reported that interparental hostility specifically predicted children's lower cortisol reactivity in response to witnessing a simulated phone disagreement between parents even after taking into account the negligible role of warm and supportive parenting as a predictor. With respect to linkages associated between emotionally available and responsive parenting and children's HPA activation to stress associated with the attachment system, studies exploring components of the hypothesis provide some support for expecting associations. For example, maternal insensitive, harsh and hostile parenting has been associated with elevated HPA reactivity to maternal separation in infancy and toddlerhood (e.g., Bugental, Martorell, & Barraza, 2003; Spangler & Grossman, 1993).

Models of allostatic load propose two possible pathways by which exposure to interparental violence and maternal emotional unavailability may eventuate in aberrations in children's HPA system functioning. First, the *hypercortisolism hypothesis* proposes that chronic exposure to environmental challenges may result in the HPA axis system becoming increasingly sensitive in its function of marshalling resources to cope with the threat. Thus, repeated exposure to interparental violence or caregiving deficits may lead children's HPA reactivity may be heightened or amplified in stressful or threatening family contexts. In contrast, the *hypocortisolism hypothesis* suggests that chronic environmental stressors may eventuate in suppressing, rather than amplifying, adrenocortical reactivity to stress. Interpreted within this framework, more destructive forms of family interactions may be associated with hypocortisolism or lower than expected levels of cortisol activity.

Our decision to examine associations between interparental violence, parenting, and children's physiological reactivity during toddlerhood was based on several developmental considerations. First, to our knowledge no study has examined these associations in a sample

of preschool children. The salience of this developmental period for understanding children's adaptation is highlighted by empirical evidence that young children are disproportionately exposed to interparental violence and are more vulnerable to experiencing psychological difficulties following exposure to interparental aggression than are older children (Kitzmann, Gaylord, Holt, & Kenny, 2003; Fantuzzo, Boruch, Beriama, Atkins, & Marcus, 1997; McDonald, Jouriles, Briggs-Gowan, Rosenfield, & Carter, 2007). Second, children's developing autonomy bids and exploratory competencies hinge upon their perceptions of caregiving figures to respond responsively to distress and threat. Parenting that is emotionally unavailable, insensitive, and overreactive undermine children's ability to flexibly respond to, explore, and adapt to challenging environments. Third, early childhood is consistently posited as a significant period of developmental plasticity in children including rapid changes in neurobiological development and emotion regulation and thus may be a particularly vulnerable period to family adversity (e.g., Edwards & Liu, 2002).

In summary, the present study was designed to provide the first test of the viability of the stress specificity hypothesis for understanding children's physiological functioning within family contexts. In accordance with EST conceptualizations of differentiation in children's emotional security in family systems, we hypothesized that interparental violence would be primarily associated with children's cortisol reactivity to a standardized laboratory paradigm in which children were exposed to a simulated interparental conflict. We further hypothesized that maternal emotional unavailability would be primarily associated with children's reactivity in the Strange Situation paradigm, an established assessment of children's confidence in their caregiver as a source of protection. In line with relationship specificity models, we also hypothesized that the strength of the covariation in children's cortisol reactivity to the two different paradigms of different family contexts would be modest in magnitude reflecting context-specific distress and not generalized manifestations of physiological stress and coping. A final aim was to identify if hypocortisolism or hypercortisolism hypotheses were supported in family risk models. Given the elevated levels of threat to emotional security in children experiencing interparental violence and problematic parenting, we hypothesized that children would evidence lower physiological responses to our laboratory stressor paradigms. To accomplish these aims, we utilized a multi-method measurement battery within an analytic model that integrated path analysis with latent growth trajectories of children's cortisol reactivity.

Method

Participants

Participants included 201 two-year-old children and their mothers in a moderately-sized metropolitan area in the northeastern United States. A two-step recruitment process was implemented to maximize individual differences in the experience of interparental aggression while minimizing heterogeneity in sociodemographic adversity. In the first step, we recruited participants through agencies who serve disadvantaged children and families, including Women, Infants, and Children, Temporary Assistance to Needy Families rosters from the Department of Human and Health Services, and the county family court system. In the second step, we administered the abbreviated version of the Physical Assault Scale of the Conflict Tactics Scale 2 (CTS2; Straus et al., 1996) to insure that roughly equal proportions of participating mothers experienced (a) no violence (40%), (b) mild/moderate physical violence (24%), and (c) severe physical violence (36%) in the interpartner relationship during the last year. Additional inclusionary criteria for the study consisted of: (a) the adult female participant is the biological mother and primary caregiver of the target child; (b) the child participant is 27-months old (+/− 5 months); (c) the child has no serious cognitive,

sensory, or motor impairments, and (d) the male partner had regular contact with the mother and toddler over the past year.

Average annual income for the family household was $20,807 (US; *SD* = 12,278) per year and a substantial minority of mothers (33%) and their partners (22%) did not complete high school. The mean age of the children was 25.4 months (*SD* = 1.55), with 56% of the sample consisting of girls (*n* = 117) and 44% consisting of boys (*n* = 91). Mothers, fathers, and children lived in the same household for an average of 6.4 years (*SD* = 1.09). The majority of the sample of mothers and children were African-American (56%), followed by smaller proportions of family members who identified as European-American (25%), Multi-Racial (8%), Latino (7%), and "Other" (4%).

Procedures

Mothers and their toddlers visited our laboratory three times within a one- to two-week time period. During the first visit to the laboratory, the Strange Situation (SS) procedure was administered. A free-play, compliance task was administered during the second visit, and the administration of a Simulated Phone Argument Task (SPAT) was completed during the third visit. The assessments were spaced accordingly to minimize potential overlap across paradigms. Mothers also completed questionnaires and interviews across the three visits. Procedures were standardized across participants.

Strange Situation—The standard Strange Situation (SS) Paradigm was conducted as described in Ainsworth and Wittig (1969) and Ainsworth et al. (1978). The SS consists of seven episodes each of which last for three minutes. The episodes were as follows: (1) Mother and child entered the paradigm room in which there were various toys and two bean bag chairs. Mother was directed to sit in the bean bag chair and remain seated for the duration of the time she was in the room. She was directed that she could respond to her child's bids for attention, but should not initiate play herself. (2) A stranger entered the room and sat in a chair directly across from the mother. She remained silent for one minute and did not interact with mother or child. At the second minute of the episode, the stranger engaged the mother in a conversation, and at the third minute of the episode the stranger attempted to engage the child in play. (3) Mom left her child in the room with the stranger. (4) The mother called to the child two times from outside the room and then entered the room. Mothers were instructed to greet their child, return to her designated chair, and if necessary attempt to soothe the child as they normally would. (5) The mother leaves for a second time and the child is left alone in the room. (6) The stranger reenters the room and sits in her chair. (7) Similar to episode 4. The mother called to her child two times from outside the door, entered the room and greeted her child, and sat in her chair. If the child became extremely upset during the two separation episodes (3 and 5), such as intense crying for at least 30 seconds, or if the mom was unable to handle seeing her child distressed, the episode was terminated early and the next episode began.

Simulated phone argument task—During the third laboratory visit, children and their mothers participated in the Simulated Phone Argument Task (SPAT; e.g., Davies, Cummings, & Winter, 2004) to assess children's reactivity to interparental conflict. During this procedure, children witnessed live simulations of their parents engaging in a conflict and a subsequent resolution over the telephone. Each exchange lasted approximately 1 minute and was interspersed by a three-minute free period. The conflict script revolved around a relatively trivial disagreement regarding whether the father had completed a task requested by the mother (i.e., stopped at the store or made a phone call or an appointment). The mothers were instructed to convey mild irritation, frustration, and anger toward their partner as they normally would at home. Although the simulations indicated to the child that

the father was on the other end of the phone, an experimenter was actually on the phone feeding the mother the lines from the script.

Several procedures were instituted during a pre-simulation briefing and training session to insure that mothers accurately followed the script and expressed the desired level and type of affect for each emotional exchange. First, mothers listened to a standard, audiotaped sample of the content and affective tone of the conflict and resolution. Second, mothers practiced the script with the experimenter until they were able to convey accurately the content and affective tone of the exchanges. Third, mothers were encouraged to convey the same emotional level and quality of their successful practice run in the actual task with their children. Fourth, in feeding the mother the lines during the procedure, the experimenter simulated the affective tone and level for the mother to emulate.

For the actual SPAT procedure, the mother and child were led into a room with a desk, a phone, and toys on the floor. The child was told that their mother had some surveys to complete and they could play with the toys. The “surveys” were cross-word puzzles meant to occupy the mother until the SPAT procedure commenced. The mothers then told their child that they were going to call the father on the phone to discuss a concern. The mother dialed the phone in the room in full view of the child. In addition, a simulated resolution was also instituted after the argument section to insure that children did not experience any prolonged, intense distress.

The validity of the SPAT for simulating conflicts between parents with the child as an observer is supported by its utilization in previous research projects which have identified associations between children’s distress reactions to the simulated conflict, their exposure to family conflict, and their concurrent and prospective psychological problems (e.g., Davies, et al., 2004; Davies, Sturge-Apple, Cicchetti, & Cummings, 2007, 2008).

Mother-Child Interaction Task—Mothers and their children participated in an observational free-play/compliance task at the laboratory which was videotaped for later coding. The mothers were instructed to play with their child as they would at home after the dyad was escorted into a room containing several developmentally appropriate toys. After seven minutes, an experimenter knocked on the door to signal the end of the free-play session to the mother. Mothers were then instructed to ask their children to stop playing and clean up the toys without providing assistance. The experimenter continued to knock on the door at one-minute intervals, up to three minutes, if the child appeared to be off-task. By the third knock mothers were told that they could provide assistance to their child with picking up the toys. The compliance portion of the task was recorded for six minutes, regardless of progress, making the entire session approximately 13 minutes.

Salivary cortisol collection—Cortisol measures were obtained through saliva samples collected from each child participant. To limit the effects of daily routines and afternoon naps on child cortisol levels, all visits were conducted within a two-hour window during the morning hours. Baseline samples were collected on average within the first 15 minutes upon arrival to the laboratory, after maternal consent had been obtained. All toddlers had been awake at least one hour prior to providing the morning saliva samples, thus avoiding the period of the dynamic cortisol awakening response (Susman et. al., 2007). During the visit, toddlers were monitored to insure they did not eat or drink for 30 minutes before the post-task sample collection was taken in order to limit saliva contamination. Due to the age of the participants, a sorbette was held under the child’s tongue by a research assistant for one minute to ensure a sufficient quantity of saliva was obtained. Each sorbette was placed in a 2 mL cryovial and immediately stored at −80°C until shipped on dry ice to Salimetrics, LLC. (State College, PA).

Measures

Interparental violence—Multi-modal assessments were used as our manifest indicators of a latent construct of interpartner violence. First, mothers self-report of interpartner violence were assessed using subscales from the Revised Conflict Tactics Scale (CTS2; Straus et al., 1996). The CTS2 Physical Assault subscale contains 24 items designed to assess maternal and partner acts of physical violence toward each other in the interpartner relationship. Items vary from relatively mild (e.g., "I pushed or shoved my partner.") to severe (e.g., "I used a knife or gun on my partner") forms of assault. The correlation between maternal reports of self and partner on the measure was $r = .83$, $p < .001$. The CTS2 Injury subscale contains 8 items which ask how often four types of injury occur as a result of physical attacks by a partner, a need for medical attention, or pain continuing for a day or more. Following guidelines, prevalence scores were calculated the scale based on the sum of the occurrences of acts (1 = act occurred one or more times; 0= specific act did not occur). Reliability was satisfactory for both scales in this sample (α ranged from .89–.92) and research supports the validity of the measures (El-Sheikh, Cummings, Kouros, Elmore-Staton, & Buckhalt, 2008; Kerig, 1996).

The remaining indicator of interparental violence was derived from the Interparental Conflict Characteristics (ICC) Module of the Interparental Disagreement Interview (IDI). Based on a prior interview developed in previous research (Crockenberg & Langrock, 2001), the ICC module of the IDI is a semi-structured, narrative interview with the mother designed to assess the frequency, nature, course, and aftermath of interpartner conflicts witnessed by child participants. Support for the validity of the IDI is reflected in its associations with established measures of interparental discord and child psychological functioning (Davies, Sturge-Apple, Cicchetti, Manning, 2009). Video records of the interview were coded for interparental aggression. Coders specifically rated the level of maternal and partner aggression during the conflicts along seven-point scales. Aggression was operationally defined as the level of hostility and aggression directed toward the partner in the interparental relationship. At one extreme, no aggression (0 = none) was characterized by no evidence of any overt form of aggression or hostility direct toward the partner. At the other extreme, high (6) aggression reflects high levels of aggression that reflect considerable dysregulation, disorganization, and/or loss of control on the part of the parent that reflects a clear risk to the psychological or physical welfare of the child. The intraclass correlation coefficient reflecting interrater agreement for ratings of aggression was .92.

Maternal Emotional Unavailability—Indices of maternal emotional unavailability were assessed by behavioral observation. Observer ratings during the free play and compliance tasks were completed using the Iowa Family Interaction Rating Scales (IFIRS; Melby & Conger, 2001). Ratings were assessed on nine-point Likert-type scales ranging from 1 (not characteristic at all) to 9 (mainly characteristic). In line with previous operationalizations of emotional availability (e.g., Biringen, 2000), the insensitive/parent-centeredness, disengagement, hostility, and low affective warmth scales were utilized to form a composite of maternal parenting. *Insensitive/parent-centeredness* assessed the extent to which the mothers lack an awareness of the child's needs, moods, interests, and capabilities. As such, interactions with the child are paced to the parent's behavior and mood and not well-timed, the mother appears to "miss" opportunities to appropriately engage child, and enforces rules, regulations, and constraints without considering the child's age-appropriate choice, control, or autonomy. *Disengagement* was measured by the extent to which mothers displayed behaviors that put physical or emotional distance between them and their child. Examples include ignoring the child, choosing not to participate in play with the child, showing a lethargic or apathetic attitude toward the child. Maternal behaviors that indicated care or support toward the child (e.g., giving praise, smiling or laughing, and showing physical

affection) were assessed via the *Warmth* scale. Warmth was rescaled such that higher scores indicated lower affection and lower levels of positive affect. Finally, *Hostility* was measured by mother's behaviors that reflect anger, contempt, or harsh rejection of her child. Examples include angry or contemptuous facial expressions, sarcastic tone of voice, and menacing/threatening body posture. The intraclass correlation coefficients, which reflect the inter-rater reliability of three independent coders for 25% of the interactions, ranged from .84 to .94 across the three coders across the two interactions.

Cortisol – SSP—Saliva samples were collected from the children at three points during the strange situation procedure to obtain cortisol reactivity measures. In the initial 20 minutes of the visit, prior to the collection of the pre-task saliva sample, the experimenters developed rapport with the families, obtained parental consent and child assent, and invited children to play with toys to get acquainted with the laboratory. Children then followed conventional sampling procedures in preparation of saliva sampling procedures (Schwartz, Granger, Susman, Gunnar, & Laird, 1998). Average sampling time for pre-task cortisol occurred at 9:47 AM. (*SD* = 0 hours, 28 minutes). Two post-task saliva samples were also obtained to assess trajectories of cortisol change across three assessments. Following guidelines established in previous research (e.g., Dickerson & Kemeny, 2004), the two post-task saliva samples were obtained approximately 25 and 50 minutes after the end of the 5^{h} episode in the strange situation paradigm in which the mother leaves and the child is alone in the room. This is designed to be the most stressful episode of the procedure for the child. The three saliva samples were immediately stored at −36°C until it was shipped on dry ice to Salimetrics LC (State College, PA). All samples are assayed for salivary cortisol in duplicate using a highly sensitive enzyme immunoassay (Salimetrics, PA). The test uses 25 µl of saliva per determination, has a lower limit of sensitivity of 0.003 µg/dl, standard curve range from 0.012 to 3.0 µg/dl, and average intra-and inter-assay coefficients of variation 3.5 % and 5.1 % respectively. Method accuracy, determined by spike and recovery, and linearity, determined by serial dilution are 100.8 % and 91.7 %. Values from matched serum and saliva samples show the expected strong linear relationship, $r(63) = 0.89$, $p < 0.0001$ (Salimetrics, 2005).

Cortisol – SPAT—Cortisol collection procedures for the SPAT paradigm followed similar guidelines to those for the SSP. Average sampling time for pre-task cortisol for the SPAT visit occurred at 9:28 AM. (*SD* = 0 hours, 30.6 minutes). Two post-task saliva samples were also obtained to assess trajectories of cortisol change across three assessments. The two post-task saliva samples were obtained approximately 25 and 37 minutes after the end of the simulated disagreement, the period in the SPAT to correspond roughly with the midpoints of the two peak periods of cortisol reactivity to stressors (Dickerson & Kemeny, 2004).

Socioeconomic Status—Mothers completed a demographic interview to obtain three measures of socioeconomic status. First, mother's reported on her educational level. Second, mother's occupational status was used to derive the occupational prestige score on the Hollingshead Occupation Score. Third, mothers reported on the earned annual income of the family unit without consideration of public assistance supplements. These three measures were used as indicators in our latent construct of SES.

Results

Initial Analyses

Table 1 provides the raw means, standard deviations, and correlations among the focal variables in the primary analyses. Cortisol data were checked for possible outliers, and 14 subjects (7%) evidenced values greater than 3.5 standard deviations away from the mean.

These values were removed. A maximum of 13% of the data was missing due to data loss across the different measures (unreliable cortisol assessments, equipment malfunctions), and we utilized missing data estimation available in Mplus 6.0 to retain the full sample for analyses (Muthen & Muthen, 2001). Data screening also featured assessment of univariate skewness and kurtosis. Large values of these statistics indicate non-normality of data for analysis variables, which may prove to be problematic in structural equation modeling analyses. To account for the possible effects of non-normality, standard errors were derived using bootstrap procedures in MPlus. Bootstrapping techniques creates multiple subsamples from the original to derive a sampling distribution which is not limited by assumptions of normality and yields more accurate standard error estimations of model parameters (West, Finch, & Curran, 1995). Models were run requesting a ML bootstrap on 500 samples with a 90% bias-corrected confidence interval and significance was determined using bootstrapped standard errors.

Modeling Cortisol Reactivity

Unconditional LGC Model—As a first step in examining the relationships among family contexts and children's cortisol reactivity to distress, we examined unconditional latent growth curve models (LGC) for children's cortisol reactivity using MPlus 6.0 statistical software. Unconditional LGC are examinations of trajectories free from any predictors of change and establish both the existence and significance of average intercepts and slopes as well as the presence of significant individual variability around these means. Because children were assessed in the morning to limit the effects of daily routines and napping on cortisol levels, average cortisol values decreased across the tasks (Table 1). Cortisol evidences a strong diurnal pattern whereby highest values are evidenced upon wake-up in the morning followed by a steep decline over the morning hours, waning in the afternoon. Thus, average cortisol values in the present study include both the steep decline in the morning juxtaposed against the stress response to the laboratory paradigms. To disentangle the diurnal pattern from children's reactivity to laboratory assessments, we followed previous recommendations for controlling for the impact of time in the growth curve analyses (e.g., Davies, et al., 2007; 2008). To accomplish this, children's wake-up hour as reported by mothers (SS *M* = 7:50 AM, Range = 4:00 AM – 9:50 AM; SPAT *M* = 7:55 AM, Range = 5:20 AM – 10 AM) were regressed onto the manifest cortisol indicators of the respective latent growth curve (see Figure 1). This practice effectively separates the variability in the cortisol value into that attributed to length between child wake-up time and assessment time and that attributed to reactivity to stressor paradigms via the factor loadings of the growth curve constructs. Parameterization of the growth curve was defined by a linear slope which weighted each manifest cortisol measurement to correspond to the number of minutes elapsed from the collection of the baseline sample. Factor loadings on the growth curve were centered on the initial cortisol assessment, identifying the intercept value of the growth function to reflect mean cortisol levels at baseline.

The unconditional LGC model of children's cortisol reactivity to the interparental disagreement task and the strange situation paradigm fit the data well, χ^2 (15, N = 201) = 29.64, $p < .05$, RMSEA = .07, CFI = .98) indicating that a linear trajectory was an acceptable model for the reactivity paradigm. Parameter estimates, including means and variances of the intercept and slope factors are presented in Figure 1. Findings indicated significant individual variability within the intercept values ($\sigma_{SPAT} = .02$; $\sigma_{SS} = .01$). These findings suggested significant individual differences in children on the initial pre-task cortisol assessments across both assessment days. Examining the latent slope factors for cortisol change in the SPAT and SS paradigms revealed that linear change in children's cortisol levels across both tasks were positive on average. These findings suggest children's cortisol levels increased from baseline to post-paradigm assessment in both the SPAT and

the SS paradigms. This increase was significant in the SS ($p < .001$) and marginally significant in the SPAT ($p = .08$).

To inspect how many children evidenced a positive slope, or positive change in cortisol levels over the SPAT and SS tasks, we utilized an option available in MPlus which calculates and saves the actual factor score for each individual. Thus, a score for each child on the slope factor in the latent growth model is calculated for the SS and SPAT and examined to determine how many children showed evidence of a positive change in cortisol levels in response to the tasks. Examination of the slope factor scores revealed that every child but one evidenced a positive slope score in the SS (Range = −.11 to 1.14), and all but 10 children evidenced a positive slope factor score in the SPAT (Range = −.14 to .79). Thus, children evidenced mild to moderate positive cortisol reactivity to the stress paradigms as parameterized in our latent growth model. The presence of significant variability in the slope factors allowed for the examination of family context predictors of children's differences in cortisol reactivity within each stress paradigm (e.g., Davies et al., 2007; 2008).

Finally, we examined the strength of the correlation between the growth model factors of the two family stressor paradigms. Both the SS and the SPAT intercept factors were significantly albeit modestly correlated with one another ($r = .14$, $p = .01$), which indicated that initial pre-task cortisol levels were associated with one another even though they were assessed on two different visits. Thus, modest stability was evident in initial cortisol levels across the two different assessment days. In addition, analyses revealed a non-significant correlation between the slope factors for the two family paradigms (r = −.02, p = .80). In line with the stress specificity hypothesis, this finding suggests that individual differences in children's cortisol reactivity to the stress paradigms in the current study were context specific to a large extent.

Conditional LGC Model—Given the presence of significant individual differences in cortisol trajectories in both the SS and the SPAT paradigms, our next set of analyses examined interparental violence and maternal emotional unavailability as predictors of children's cortisol functioning in interparental and parent-child contexts (Figure 2). In addition, although the fairly homogeneous demographic backgrounds of the families were designed to limit the impact of background variables on the study findings, the possibility of differences across socio-economic covariates in model processes may still exist. Thus, we also entered socio-economic status as a predictor of cortisol reactivity to explore if pathways between IPV and maternal emotional unavailability were still significant in the presence of this factor. The model featured latent constructs of interparental violence, maternal emotional unavailability, and social economic status. A confirmatory factor model analysis of these latent variables revealed that the manifest indicators loaded significantly and singularly on their respective factors (absolute values of λ's ranged from .42 – .86 ($p < .001$)). Given the convergence of the factor model, interparental violence, maternal emotional unavailability, and socio-economic factors were simultaneously regressed onto intercept and slope constructs for both the SPAT and SS paradigms. This model provides a test of the specificity or generalizability of effects. It assesses the predictive ability of a family context variable to account for significant individual variation in children's cortisol reactivity to the lab paradigm tasks when considered simultaneously with the other context variable.

The model represented the data adequately, χ^2 (132, N = 201) = 277.31, $p = .01$, RMSEA = .07, CFI = .90.) Results are presented in Figure 3. Although all possible pathways between interparental violence, socio-economic factors, and maternal emotional unavailability were estimated, only significant pathways are presented in the figure for clarity of significant effects. Analyses revealed that interparental violence was a significant predictor of

children's cortisol reactivity in the SPAT paradigm (β = -.21, p = .039). This pathway accounted for 8% of the variability in cortisol reactivity in the SPAT paradigm. The beta for this effect was negative which indicates that as interparental violence increased, children's cortisol reactivity was lower on average. This finding supports the hypocortisolism hypothesis for the interparental context. Attesting to the stress specificity hypothesis, IPV was not significantly associated with the SPAT intercept (β = .14, p = .12), the SS intercept (β = .06, p =.44), or the SS slope factors (β = −.04, p = .64).

Examination of the pathways associated with maternal emotional unavailability revealed that this construct was significantly associated with both of the intercept and slope factors for children's cortisol activity in the SS paradigm. Maternal emotional unavailability predicted higher cortisol levels at baseline for the first visit (β = .28, p < .01). This finding was not expected and warrants further discussion. In accordance with study hypotheses, maternal emotional unavailability was negatively associated with cortisol change in response to the SS paradigm (β = −.25, p < .01). This finding suggested that as mothers evidenced higher levels of insensitive, disengaged and hostile caregiving behaviors, children's cortisol reactivity to threats associated with maternal separation and being left alone was lower. These pathways accounted for 11% of the variability in the intercept factor and 7% of the variability in the slope factor in the SS paradigm. In further support of the stress specificity hypothesis, maternal emotional unavailability was not significantly associated with the SPAT intercept (β = .10, p = .21) or slope factors (β = −.08, p =.46).

Our primary aim was to chart specificity in pathways between interparental violence, maternal emotional unavailability, and children's cortisol reactivity to family paradigms, however maternal parenting behaviors may serve as potentiators or protective factors in the relationships between interparental violence and children's functioning (e.g., Levendosky, Huth-Bocks, Shapiro, and Semel, 2003). To explore the possibility of interactive effects, we utilized the capabilities of MPlus to model interactions between latent continuous variables using full-information likelihood estimates which increase the efficiency and power to detect effects (Klein & Moosbrugger, 2000). Thus a model in which a latent interaction between our latent interparental violence and maternal emotional unavailability in predicting both cortisol intercepts and slopes was analyzed. Results indicated that the interaction factor did not significantly predict children's cortisol intercepts or slopes for both the SPAT and SS paradigms (*p* values ranged from .32 to .95, ns). Furthermore, all main effects previously reported remained significant when taking into account the possible interactive effects. These results suggested that compounding or interactive effects across family risk variables did not operate in our process model.

Although fairly homogeneous demographic backgrounds of the families were designed to limit the impact of background variables on the study findings, the possibility of differences across socio-economic covariates in model processes may still exist. Thus, we also entered socio-economic status as a predictor of cortisol reactivity to explore if pathways between IPV and maternal emotional unavailability were still significant in the presence of this factor. Analyses revealed that the strength of the primary model pathways between family predictors and cortisol factors was not attenuated when socio-economic risk was included. However, socio-economic risk was a marginally significant predictor of children's initial cortisol levels during the Strange Situation paradigm (β = −.20, p = .08), such that higher risk was associated with higher basal cortisol levels.

Finally, associations identified in the primary analyses may differ for boys and girls (e.g., Davies & Lindsay, 2001; Leaper, 2002). Consequently, we examined child gender as a potential moderator using a multiple-group approach where the model was estimated simultaneously for boys and girls. We constrained measurement model parameters to be

equal to each other in the two models (e.g., error variances, factor loadings) but tested for differences in structural parameters (e.g., means, variances and predictor pathways). First, in our unconditional latent growth model of cortisol trajectories, holding all measurement parameters equal to one another for boys and girls did not produce any significant differences in the mean levels and individual variability of cortisol reactivity for the two different paradigms. Thus, boys and girls did not differ from one another on cortisol reactivity in the interparental or parent-child tasks. Although no differences in cortisol activity were found between boys and girls, gender differences may still operate in the structural paths of the family process model (e.g. associations between IPV and cortisol in the SPAT task). To test this, we used the same multiple group approach for examining whether the impact of interparental violence or maternal parenting on children's cortisol reactivity in the two laboratory paradigms was different for boys and girls. We again assumed that all measurement model parameters were invariant across group (e.g., error variances, factor loadings). We tested for significant differences between boys and girls in the strength of the process pathways between IPV and maternal emotional unavailability and cortisol reactivity in family paradigms. No significant differences in these structural model pathways were found between boys and girls. Thus in our sample, child gender did not moderate model pathways.

Discussion

Through our attempt to delineate the biological consequences of exposure to adverse rearing contexts, the present study breaks new ground by simultaneously examining children's experiences with interparental and parenting difficulties as predictors of their neuroendocrine reactivity to stressors within the interparental and parent-child relationships. Results supported a stress-specificity perspective and the more specific hypotheses derived from an emotional security approach to differentiating the family correlates of children's reactivity to interparental and parent-child stress (e.g., Davies & Sturge-Apple, 2007). Whereas exposure to interparental aggression was the only predictor with individual differences in children's cortisol reactivity to a laboratory simulation of interparental conflict, children's cortisol reactivity to the parent-child relationship challenge (i.e., Strange Situation) was only predicted by their experiences with emotionally unavailable caregiving. Consistent with the hypocortisolism hypothesis, greater levels of adversity in these family relationships were associated with children's lower cortisol reactivity to the family stressors.

Results from the present study support EST contentions that interparental violence represents a proximal, salient, and uncontrollable threat to young children (e.g., Davies & Sturge-Apple, 2006). In line with previous work documenting associations between interparental conflict and violence with cortisol reactivity (e.g, Davies, et al., 2007), the present study adds further evidence that living in homes characterized by the presence of interparental violence may alter children's emotional security in ways that prime physiological systems to respond to possible violence and aggression within the interparental relationship. Cast in the EST framework, it may be that the need for increased vigilance, anxiety, and threat in the context of IPV is a hallmark symptom of children's concerns about emotional security manifested at a physiological level of analysis (Cummings & Davies, 1996; Davies & Sturge-Apple, 2007).

Findings also revealed that maternal caregiving difficulties have specific implications for children's ability to utilize caretakers a source of support and security during times of stress and threat. The salience of maternal emotional unavailability for children's HPA functioning in the mother-child context was evident in the present study. During infancy and early childhood, parents function as external regulators, assisting young children in modulating arousal and setting the stage for effective emotion regulation in the presence of stressful and

challenging events. A mother's inability to provide children with sensitive, consistent, and responsive caregiving behaviors may fail to inoculate children during stressful experiences, which in turn may inhibit the normative development of regulatory processes in response to stress.

Additional model tests provided supplementary support to the notion that children's physiological reactivity to threat within the family system is governed by different emotional security systems. As indicated in the figure, both intercept factors were significantly correlated with one another, suggesting stability across assessment intervals in basal cortisol levels. This was not unexpected and highlights the stability in HPA activity in the children in the sample across the span of the visits. However, also of note is the lack of a significant correlation between the slope factors for the two family paradigms. This finding suggests that children's physiological arousal to the stressors in the current study were, to some extent, context specific. These findings highlight how our ability to simultaneously explore associations with different family contexts has important methodological and substantive implications for future research focused on understanding family risk and children's functioning. From a substantive perspective, our findings support the application of stress specificity models from physiological research to consideration of children's physiological pattern of responding to different family contexts. Specifically, they underscore how children's physiological functioning in response to stress and threat in different family systems represent dispositional and state forms of reactivity, and are not a function of an underlying trait-like response. From a methodological standpoint, the findings here illuminate the necessity of considering the ecological validity of laboratory assessments. Further progress in investigating associations between family contexts and children's functioning hinges upon research which utilizes paradigms that are tailored to activate children's experiential histories associated with specific family systems (Dickerson & Kemeny, 2004; Gunnar, Talge, & Hererra, 2009).

The present study also revealed that interparental violence and maternal emotional unavailability were associated with lower cortisol reactivity in the research paradigms. Psychophysiological literature postulates that the LHPA system becomes increasingly sensitized in its function of marshalling, directing, and sustaining resources associated with chronic exposure to environmental stress eventuating in hyporeactivity of the LHPA system. Given the adverse childrearing contexts present in the current study, lower cortisol reactivity associated with interparental violence and maternal emotional unavailability may signify to some extent an adaptive form of dissociation or inhibition of the psychological experience of threat (Gunnar & Vasquez, 2001; Heim, Ehlert, & Hellhammer, 2000). Thus, the blunting of cortisol reactivity may be indicative of children attempting to downregulate the psychological impact and provide a temporary means of attaining a perceived sense of security or control in stressful contexts (Gunnar & Vazquez, 2001). Although the findings in the current study are in accordance with previous research exploring reactivity in interparental (Davies, et al., 2006), and mother-child contexts (e.g., Blair, et al., 2008), firm conclusions regarding directionality of these effects must be tempered against the backdrop of relatively little research in the interparental context and somewhat disparate findings in the mother-child context across research studies.

Of additional note is that model analyses also revealed associations between socio-economic risk and basal cortisol constructs. Our sample evidenced higher levels of socio-economic adversity and our findings of links between socio-economic risk and elevated basal cortisol is consistent with a large literature documenting this association, particularly in young children (e.g., Lupien, King, Meaney, & McEwen, 2001). According to the "stress gets under your skin" hypothesis, lower SES (defined as lower income-to-needs ratio, single parenting, low maternal education, housing problems, e.g., Evans, Kim, Ting, Tesher, &

Shannis, 2007) is implicated in physiological models of allostatic load (e.g., Juster, McEwen, & Lupien, 2009; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). To date, the literature seems to converge on the consensus that SES is associated with manifestations of allostatic load in physiological systems in young children and the findings here lend additional support to this body of work. Against the backdrop of socioeconomic adversity however, our findings revealed that children's experiential histories in family contexts remained potent predictors of their physiological reactivity to our laboratory paradigms.

Finally, although we made no a priori hypotheses regarding associations between family predictor variables and children's initial cortisol levels, our analyses further revealed that maternal emotional unavailability significantly predicted children's higher intercept cortisol levels prior to conducting the Strange Situation. This association was only evident for one of the two assessments of basal cortisol and extensive interpretation of the findings requires replication. However, recent studies have reported similar associations as found here in which family risk variables were positively associated with elevated initial cortisol levels and negatively associated with cortisol reactivity to stressor tasks (e.g., Bugental, Schwartz, & Lynch, 2010; Cutuli, Wiik, Herbers, Gunnar, & Masten, 2010, Blair, et al., 2007). Although these studies did not delve into interpretations of these findings, given their presence in the literature some speculation as to why these differential associations were found here is warranted. From a substantive standpoint, this differential associations with intercept and slope levels of cortisol activity may indicate the complex interplay between basal cortisol levels and cortisol reactivity in young children (e.g., Tarullo & Gunnar, 2006). While the specific mechanisms underlying the association have yet to be articulated, one plausible hypothesis may involve differential associations between primary glutocorticoid (GC) receptors associated with activity in the LHPA. Recent work outlining the stress response has suggested that mineralocorticoid receptors are primarily associated with basal concentrations of GCs while glucocorticoid receptors primarily mediate the stress effects on GCs (Gunnar & Vazquez, 2006). Thus, the vulnerability present in both basal and reactivity levels may be a differential function of the activation of these receptors. While exploring this is beyond the scope of the current project, it highlights the importance of future research to more fully delineate children's overall LHPA response systems in risky family contexts (e.g., Cutuli, et al., 2010).

Fully interpreting the results of our study requires consideration of the methodological limitations. First, because fathers were not assessed in the present study, we were not able to examine the role of the father-child relationship in associations between interparental violence and children's physiological reactivity to stress. Given previous work demonstrating the impact of interparental violence on father-child relationships (e.g., Mahoney, et al., 2003), it will be important for future research to include samples of fathers experiencing interparental violence to determine whether our findings based on mother-child data are comparable for father-child relationships. Second, the cross-sectional design cannot definitively address the temporal ordering of relationships in our process model. Our conclusions would be bolstered through examining the model processes over time. Third, the present study focused on the HPA axis; however, future research on family-wide process models examining children's physiological functioning would benefit by increasing the rigor of physiological assessments. For example, contextualizing the study of cortisol within broader profiles of functioning across multiple physiological systems may also advance psychophysiological models of children's coping with family conflict (El-Sheikh, Erath, Buckhalt, Granger, & Mize, 2008; Gordis, et al., 2006). Fourth, children's behavioral indicators of distress were not examined in the present study. It would be informative for future research to better explore how external markers of distress are associated with physiological functioning.

Through delineating the physiological correlates of children's exposure to interparental violence and caregiving deficits, the present study emphasizes the centrality of children's physiological response systems in risky family models and family frameworks. If replicated, the present findings have important implications for clinical interventions involving children exposed to interparental violence. Children's HPA activity in the context of detrimental functioning across family systems may increase children's vulnerability for subsequent mental health difficulties. Recent research has illuminated links between adverse early rearing environments and neurobiological development (Cicchetti, Rogosch, Gunnar, & Toth, 2010; Repetti, et al., 2007). From a public health standpoint, the present research also raises concerns about the physical health outcomes of cortisol levels in children living in the context of interparental violence and disrupted caregiving. The early years of life is a period of rapid neurobiological development (Cicchetti, 2002; Thompson & Nelson, 2001) and this development is occurring when children are frightened, vigilant, and completely dependent on their caretakers who are engaged in interparental violence. Over time, dysregulation in the LHPA system can have a deleterious impact on children's physical health including immune suppression, increased risk for diabetes, and neurotoxic effects (e.g., Danese, Pariante, Caspi, Taylor, & Poulton, 2007; McEwen, 1998; Repetti et al., 2002; Sapolsky, 2000).

In conclusion, the present findings supplement important discoveries of the explanatory role of children's behavioral strategies (Gordis, Margolin, & John, 2001) and representational models (e.g., Sturge-Apple, et al., 2008) of dealing with conflict and stress in family relationships, by delineating the physiological consequences of living in homes characterized by interparental violence and diminished caregiving. Disruptions to homeostatic set-points in biological stress response systems in young children may set the stage for long-term difficulties across a variety of outcomes including emotion regulation, social development, as well as physical and mental health difficulties (Repetti, et al., 2009). Taken together, the convergence of findings across this multiple levels of analysis investigation lend support to the assertion that understanding children's psychobiological functioning in family contexts has important implications for our understanding of children's development.

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Task Order

Visit 1	Visit 2	Visit 3
Baseline Cortisol Assay	Mother-Child Free Play Task	Baseline Cortisol Assay
Strange Situation Task (20 minutes)		SPAT Task (10 minutes)
Post-I Cortisol Assay (20 minutes post peak of stressor)		Post-I Cortisol Assay (25 minutes post peak of stressor)
Post-II Cortisol Assay (40 minutes post peak of stressor)		Post-II Cortisol Assay (35 minutes post peak of stressor)
Mother Questionnaires	Mother Questionnaires	Mother Questionnaires

Figure 1.
Timeline for task procedures and cortisol samples across study visits.

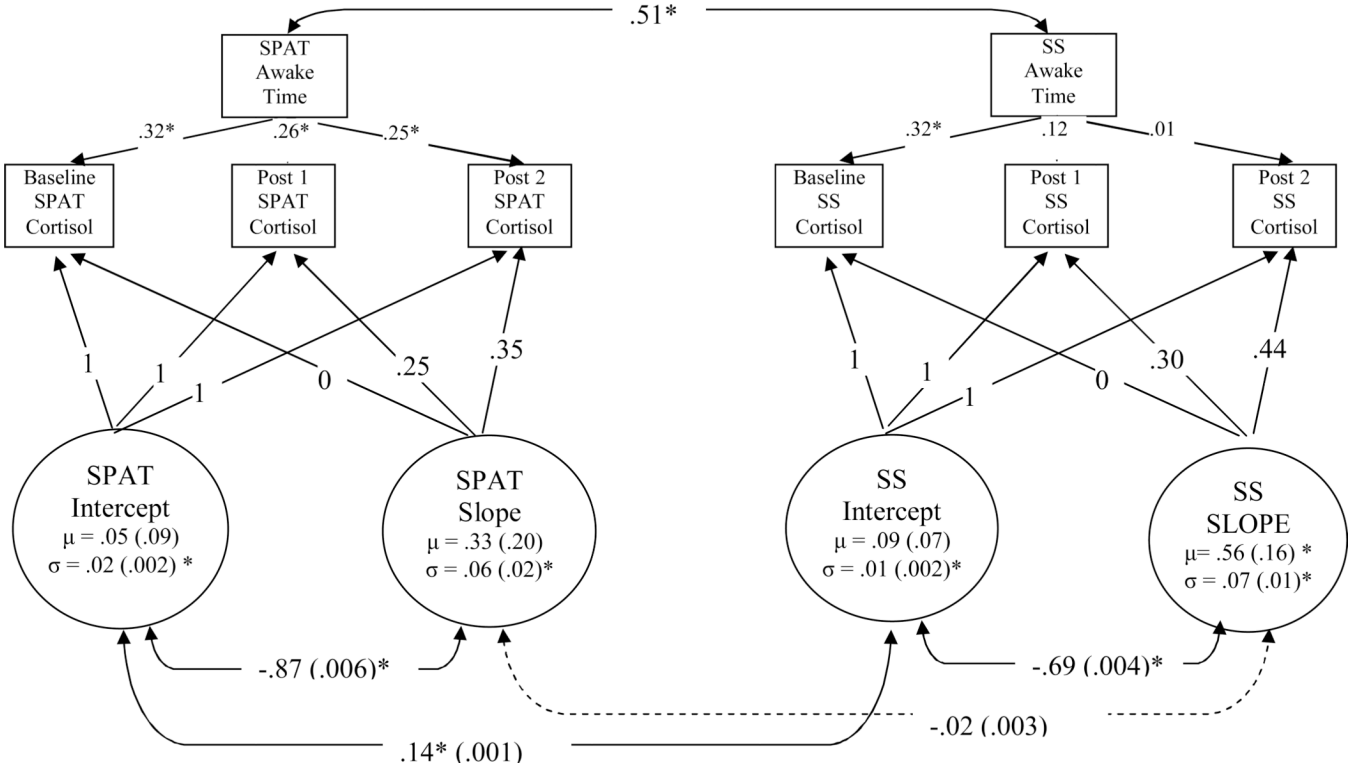

Figure 2.

Unconditional latent growth curve model of children's trajectories of cortisol reactivity to the interaparental disagreement task and the strange situation paradigm. SPAT = interparental disagreement task, SS = Strange Situation, Time = Time of day, μ = sample average, σ = sample variance. † $p < .10$, $^{*}p < .05$.

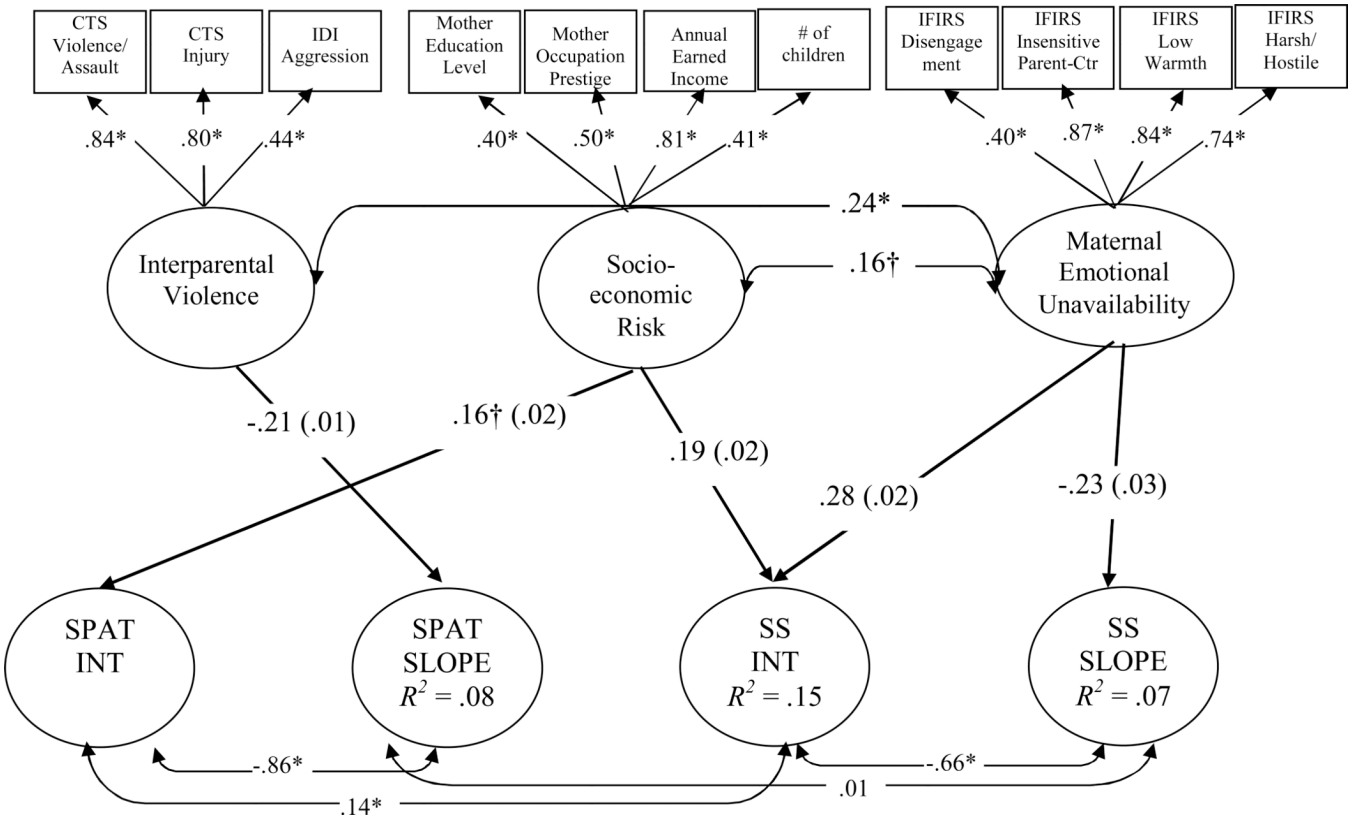

Figure 3.

A structural equation model testing associations among interparental violence, maternal emotional unavailability, family SES, and children's cortisol reactivity within stressor paradigms. Parameter estimates for the structural paths are standardized path coefficients and robust standard errors are presented in (). All possible pathways between the three predictor variables and the four cortisol variables were estimated, however for ease of presentation only significant pathways are presented in the figure. In addition, the model presented in Figure 1 was estimated within this process model, however to reduce the complexity of the model within the figure, only the latent variables for cortisol constructs are included. Pathways constrained (time loadings on growth curve variables) and freely estimated within Figure 1 were modeled the same way in the process model presented in Figure 2. † $p < .10$, * $p < .05$.

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. CTS Violence	--																
2. CTS Injury	.67*	--															
3. IDI Aggression	.37*	.35*	--														
4. Mat Low Warmth	.16*	.15*	.07	--													
5. MatHostility	.23*	.11	.12	.62*	--												
6. MatInsensitivity	.16*	.16*	.08	.73*	.65*	--											
7. Matl Disengagment	.11	.26*	.07	.46*	.15*	.29*	--										
8. Annual Earned Inc	−.08	−.06	.06	−.07	−.09	−.13*	.02	.--									
9. Education Level	−.09	−.07	.11	−.34*	−.24*	−.36*	−.20*	.29*	--								
10. Occupation Prestige	−.11	−.05	.02	−.06	−.07	−.13*	.03	.37*	.38*	--							
11. Number of children	−.14*	−.03	.02	−.12	−.13*	−.15*	−.09	−.40*	.07	−.17*	--						
12. Pre SS Cortisol	.11	.13*	.19*	.28*	.14*	.26*	.20*	−.14*	−.26*	−.04	.06	--					
13. Post I SS Cortisol	.05	.05	.09	.22*	.16*	.18*	.14*	−.11	−.18*	−.10	.08	.52*	--				
14. Post II SS Cortisol	.04	.04	.03	.03	.02	.09	.03	−.05	−.12	−.04	−.03	.29*	.73*	--			
15. Pre SPAT Cortisol	.10	.14*	−.01	.20*	.06	.12	.22*	−.15*	−.19*	.06	.01	.30*	.14*	.14*	--		
16. Post I SPAT Cortisol	.05	.12	−.02	.08	.08	.10	.08	−.15	−.18*	−.01	.03	.30*	.22*	.15*	.80*	--	
17. Post II SPAT Cortisol	−.01	.07	−.02	.12	.12	.14*	.13*	−.11	−.18*	.02	.02	.29*	.28*	.23	.70*	.83*	--
M	1.33	0.41	3.94	5.39	2.39	4.98	2.91	1.74	3.99	2.01	2.55	0.41	0.35	0.34	0.49	0.36	0.35
SD	3.21	1.21	3.55	1.56	1.52	1.75	1.42	0.49	1.08	1.57	1.49	0.13	0.09	0.08	.015	0.10	0.08