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Adrenocortical Underpinnings of Children's Psychological Reactivity to Interparental Conflict

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Abstract

This study examined interrelationships among children's cortisol reactivity and their psychological reactivity to interparental conflict in a sample of 208 first-graders (mean age = 6.6 years). Assessments of children's psychological reactivity to conflict distinguished among their distress, hostile, and involvement responses across multiple methods (i.e., observation, questionnaire) and informants (i.e., observer, parent). Relative to other forms of conflict reactivity, children's distress responses to interparental conflict were consistent, unique predictors of their elevated cortisol reactivity to interparental conflict even after inclusion of demographic factors as moderators and covariates. Moderator analyses further revealed that associations between distress and elevated cortisol levels in response to interparental conflict were particularly pronounced when children exhibited high levels of involvement in conflicts.

Understanding how children respond to interparental conflict is a pressing priority for public health (NIH, 2003, PAR-03–096). Interparental conflict increases children's vulnerability to a wide range of behavioral, emotional, and social problems. In attempting to identify the processes associated with the risk experienced by children from high conflict homes, conceptual models have underscored the significance of children's responses to conflict across multiple levels of analysis (Davies & Cummings, 2006). For example, emotional security theory (EST) regards children's elevated distress responses to conflict as developing from repeated exposure to destructive interparental conflict and, in turn, increasing the likelihood of psychological problems (Davies & Cummings, 1994). At another level of analysis, research on children's physiological reactivity to conflict has elucidated pathways between interparental conflict history and child adjustment problems (e.g., see review by Katz, 2001).

The approach commonly shared by the psychological and psychophysiological research traditions involves analyzing children's responses to conflict at one level of analysis (e.g., psychological) in isolation from children's response patterns at other levels of analysis (e.g., physiological). For example, empirical tests of psychological theories of interparental conflict have primarily focused on the analysis of behavioral and social-cognitive reactions to interparental conflict to the relative exclusion of physiological assessments (Davies & Cummings, 1994; Grych & Fincham, 1990). Likewise, studies conducted on children's physiological functioning in the context of adult or parental conflict are commonly rooted in a different body of literature addressing children's physiological reactivity to broad array of

stressful events (Katz, 2001). Therefore, little is known about the interplay among children's psychological and physiological reactivity to interparental conflict.

In reflecting the repeated calls to integrate children's physiological and psychological responses to family stress (Cummings, Davies, & Campbell, 2000; Fox, Hane, & Perez-Edgar, 2006; Katz, 2001), Katz (2001) concluded: "It is only through integrating our understanding of both biological and behavioral processes that we can get a complete picture of the effects of marital conflict on the whole child (p. 207)." Therefore, although dissociations between physiological and psychological indices of functioning are common in the literature, fully understanding children's responses to conflict at multiple levels of analysis requires further examination of how indices of physiological reactivity to conflict correspond concurrently with comparable assessments of psychological responses to conflict (Fox et al., 2006).

To address the paucity of research on the relationship between children's physiological and psychological reactivity to interparental conflict, we examine associations between children's behavioral reactivity and their limbic-hypothalamic-pituitary-adrenocortical (LHPA) system reactivity to interparental conflict. Through its role as a second wave of autonomic responding to stress (Cahill & McGaugh, 1998), the LHPA axis is regarded as a useful means of advancing an understanding of children's reactivity to interparental conflict (Saltzman, Holden, & Holahan, 2005). In response to stressful events, components of the limbic system (e.g., amygdala, hippocampus) involved in processing aversive stimuli modulate the release of corticotropin-releasing factor (CRF) by the hypothalamus. CRF, in turn, activates the adrenal gland to secrete cortisol by stimulating the pituitary gland to produce and release adrenocorticotrophic hormone (ACTH) into the bloodstream. Increases in cortisol in response to stress mobilize energy (e.g., glucose, oxygen), increase cardiovascular activity, and modulate the processing, learning, and memory consolidation of emotionally significant events (Cahill & McGaugh, 1998; Gunnar & Vazquez, 2006). In accord with the role that the LHPA axis plays in allocating resources to process and cope with stressful events, cortisol reactivity may be a critical component of children's responses to the stress of witnessing interparental conflict. Thus, the purpose of this study was to examine the concomitant correspondence between children's cortisol reactivity to interparental conflict and three of the most common forms of children's responses to conflict: distress, hostility, and involvement (Cummings & Davies, 1994).

EST provides a foundation for expecting associations between children's behavioral and physiological responses to interparental conflict. According to EST, preserving a sense of security is an important goal for children in contexts of interparental conflict (Cummings & Davies, 1996). When children witness interparental conflict, elevations in their distress characterized by anxiety, vigilance, and emotional upset is theorized to be one of the most reliable barometers of children's insecurity in the interparental relationship. Drawing on the role of the LHPA axis in marshalling resources to cope with threatening events, EST proposes that distress indicative of children's concerns about security may be linked with changes in the ability of the LHPA axis to mount physiological responses to interparental conflict. These individual differences in the LHPA axis are hypothesized to insidiously undermine children's physical and mental health over time (Davies, Winter, & Cicchetti, 2006).

Because EST offers little guidance with regard to the nature of the proposed link between behavioral and cortisol reactivity, theories on the physiological underpinnings of individual psychological functioning may sharpen predictions on the interplay between children's psychological and cortisol reactivity to interparental conflict. First, consistent with the emphasis on distress in EST, some physiological conceptualizations postulate that elevated

cortisol reactivity to stressful events is most likely to occur when children are predisposed toward exhibiting submissive behavioral reactions characterized by high levels of emotional distress, anxiety, and inhibition (e.g., Fox et al., 2006; Kagan, Reznick, & Snidman, 1998; Spangler & Grossman, 1993). Thus, in the context of understanding children's responses to interparental conflict, this "distress specificity" model would predict that children's distress should evidence unique and specific associations with cortisol reactivity relative to other forms of behavioral responding (i.e., involvement, hostility). Second, the negative affect hypothesis postulates that greater cortisol reactivity underlies more general forms of negative emotional responses to stressful events, including irritability and hostility (Spangler & Grossmann, 1993). Therefore, in contrast to the distress specificity model, the prediction of the negative affect hypothesis is that both distress and hostility would evidence stronger relations to elevated cortisol reactivity, especially in relation to involvement. Third, within a general arousal model, any type of behavioral activation or engagement with the stressor is expected to be associated with elevated cortisol reactivity (see Gunnar & Vazquez, 2006). Thus, as forms of behavioral activation, another hypothesis is that distress, hostility, and involvement may each be linked with greater cortisol reactivity.

Some support is evident for the predictions of the distress specificity model. Children with elevated internalizing symptoms have been shown to exhibit concurrent elevations in cortisol reactivity to mild laboratory stressors (e.g., Ashman, Dawson, Panagiotides, Yamada, & Wilkinson, 2002; Granger, Weisz, & Kauneckis, 1994). Tests of the coupling of behavioral and adrenocortical activity in response to the same stressor also provide support to the distress specificity hypothesis. For example, children's behavioral distress in response to social stressors (e.g., caregiver separation, transition to day care) have been contemporaneously linked with greater cortisol reactivity to social stressors (e.g., caregiver separation, transition to day care) (Dettling, Gunnar, & Donzella, 1999; Gunnar, Larson, Hertsgaard, Harris, & Brodersen, 1992). Likewise, patterns of distress reflecting insecure attachment patterns are associated with children's greater cortisol reactivity to the series of separations and reunions during the Strange Situation (Hertsgaard, Gunnar, Erickson, & Nachmias, 1995; Spangler & Grossmann, 1993).

At the same time, other studies do not definitively support the distress specificity hypothesis. Inclusion of indicators of anger in assessments of distress in some of the studies described above cannot disentangle the distinct associations between forms of negative emotional and adrenocortical reactivity as outlined in the distress specificity hypothesis (e.g., Dettling et al., 1999). In fact, some studies have revealed that indices of child aggression, anger, and global negative affect in child care settings were correlated with increases in cortisol activity across the day (Dettling et al., 1999; Dettling Parker, Lane, Sebanc, & Gunnar, 2000; Gunnar & Donzella, 2000; Tout, deHaan, Kipp-Campbell, & Gunnar, 1998). Therefore, in support of the negative affect model, one alternative hypothesis would be that high levels of cortisol reactivity are coupled with any form of negative reactivity to interparental conflict. Given that some of the studies on preschool adjustment to day care have also reported that impulsive and approach behaviors are associated with elevated child cortisol reactivity (e.g., Dettling et al., 1999), the general arousal hypothesis that any effortful response to interparental conflict may covary with elevated cortisol is also plausible.

In light of inconsistencies in associations between behavioral and adrenocortical reactivity to other stressful events, rigorously testing the distress specificity hypothesis hinges on examining its relative viability in relation to the negative affect and general arousal hypotheses. Thus, the present study simultaneously examines the specificity and commonality of patterns of associations between children's distress, hostile, and involvement responses to interparental conflict and elevated cortisol reactivity to interparental conflict. Consistent with the organizational perspective on development

(Cicchetti & Tucker, 1994), the associations between any form of behavioral responding and adrenocortical reactivity may depend on children's broader patterns of behavioral responses to interparental conflict. For example, the EST suggests that distress responses may signify more pronounced threats to children's security if they are coupled with high levels of involvement in interparental conflicts (Davies et al., 2006). Accordingly, we also employ moderator models to explore whether associations between distress and cortisol reactivity are moderated by involvement and hostility. Toward the goal of examining the stability of our results, a final step is to examine the role of child and demographic characteristics (i.e., gender, race, blood contamination in saliva samples, medication use, research site) that may alter associations between children's psychological and cortisol reactivity to conflict as covariates and moderators (Cohen et al., 2006; Hibel, Granger, Cicchetti, & Rogosch, 2007; Granger et al., 2007; Loney, Butler, Lima, Counts, & Eckel, 2006).

Method

Participants

The data for this study were collected from the second measurement occasion of a longitudinal project on children's coping with family stress. The original sample of 235 kindergarten children and their families in the first measurement occasion of the study were recruited through local school districts and community centers at two research sites located in moderate-sized cities in the Northeast and the Midwest. The sample for this study consisted of 208 mothers and their 1st grade children. Reductions in the size of the original sample resulted from attrition of families during the one year span between measurement occasions ($n = 8$), incomplete data across the three cortisol assessments ($n = 2$), and exclusion of children who were identified as outliers (i.e., ± 3 standard deviations from the mean) on cortisol assessments ($n = 8$). Due to the potential effects of psychotropic and glucocorticoid drugs on cortisol levels (Hibel et al., 2007; Masharani et al., 2005), nine additional children who were taking these medications were excluded from the final sample. Thus, the sample used in this paper consisted of 208 mothers and their children. Statistical comparisons of the present sample ($n = 208$) and participants in the larger sample who were excluded from this study ($n = 27$) along 16 demographic (e.g., family income, occupational status, race-ethnicity, marital status, age) variables yielded no significant differences.

Sociodemographic data reflected that the participating families were similar to the households in the counties from which our sample was drawn (i.e., St. Joseph County, IN; Monroe County, NY). Median annual family income of the families was between \$40,000 and \$54,999. Mothers, on average, completed some college. The majority of the sample of mothers and children were White (75%), followed by smaller proportions of Black (15%), Multi-Racial (7%), and Hispanic (3%) family members. A large percentage of female caregivers reported being the biological parents of their children (95%), followed by relatively smaller percentages of step or adoptive parents (3%), and other types of guardianship (2%). The mean age of the children was 6.6 years ($SD = 0.52$), 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 ($SD = 1.09$).

Procedures

Following the completion of two visits to the laboratory during the first measurement occasion, mothers and children attended two more laboratory visits at one of the research sites during second measurement occasion one year later. Data for the current study were drawn from the second visit of the second measurement occasion (i.e., the fourth family visit to our laboratory). The laboratories at each site were designed to be comparable in size and quality and included: (a) an observation room that was designed to resemble a family room

(e.g., couch, pictures, lamps, end tables) and equipped with audiovisual equipment to capture family interactions, and (b) interview rooms for completing confidential survey measures. The research was approved by the Institutional Review Boards at both sites prior to conducting the study.

Simulated phone argument task—During the visit, children and their mothers participated in the Simulated Phone Argument Task (SPAT) to assess child reactivity to interparental conflict. During this procedure, children witnessed a live simulated conflict and resolution between their parents over the telephone. Each exchange lasted approximately 1 minute and was interspersed by a three-minute free period to assess prolonged or delayed responses of children. 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 simulation 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. Videotaped records were later coded for child overt reactivity to the interparental conflict. For ethical purposes of allaying any child distress, the resolution with the father over the telephone consisted of mother communicating a moderate level of understanding, caring, and warmth in her tone of voice. Interspersed between the free period following the conflict and the resolution were two five-minute interviews with children designed to assess their reactions to the conflict and resolution, respectively.

Several procedures were instituted during a pre-simulation training session to maximize the validity of the SPAT. First, to maximize the accuracy of the simulations, mothers listened to a standard, audiotaped sample of the conflict and resolution and practiced the script with the experimenter until they were able to convey accurately the content and affective tone of the exchanges. Moreover, in feeding the mother the lines during the procedure, the experimenter simulated the affective tone and level for the mother to emulate. Second, to increase the ecological validity of the exchanges, mothers: (a) scheduled the visit during a time in which the father was accessible by phone; (b) chose one of three script options that most realistically reflected the nature of interparental activities, and (c) had the opportunity to alter the scripts to reflect the content and nature of actual interparental conflicts and resolutions that occur in the home. Script changes were only implemented if they did not significantly alter the intensity, meaning, and affective tone of the disagreement or resolution. Thus, this procedure provided important controls over the nature of interparental conflict stimuli, while at the same time presenting conflicts that were for all purposes live and real from the observing child's perspective. The validity of the SPAT is supported by associations between children's distress reactions to the simulated conflict and their exposure to family conflict and their concurrent and prospective psychological problems (e.g., Davies, Cummings, & Winter, 2004).

Salivary cortisol collection—Saliva samples were collected from the children at three points during the simulated conflict procedure to obtain cortisol measures. Cortisol levels tend to evidence more gradual declines during the afternoon and evening hours than during the morning hours (Knutson et al., 1997; Stansbury & Gunnar, 1994). Thus, to limit the effects of time of day on cortisol assessments, sample collection times for cortisol were, with one exception, collected in the afternoon or early evening. Average sampling time for pre-task cortisol occurred at 3:50 P.M. ($SD = 2$ hours, 1 minute).

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 re-acquainted with the laboratory. While

the mother was learning the script for the SPAT, the children then followed conventional sampling procedures in preparation of saliva sampling procedures (Schwartz, Granger, Susman, Gunnar, & Laird, 1998). Children rinsed their mouths with water prior to the baseline assessment to limit the undue influence of various contaminants during the assay process. Following a seven minute period in which they were invited to play with toys, children chewed Trident original flavor sugarless gum for approximately 3 minutes to stimulate saliva flow immediately prior to saliva collection. The pre-task sample was then collected prior to the simulated conflict by having children expurgate through a plastic straw directly into a 20mL collection vial.

Two post-conflict saliva samples were also obtained to assess trajectories of cortisol change across three assessments. No definitive guidelines are available for precisely identifying the timing of peak cortisol levels following stressors. However, a recent meta-analysis of cortisol functioning revealed that cortisol levels across 10-minute periods following the stressor were highest during the 21–30 and 31–40 minute epochs than any other 10 minute period (Dickerson & Kemeny, 2004). Therefore, the two post-conflict saliva samples were obtained approximately 25 and 35 minutes after the simulated conflict to correspond with the midpoints of the two 10-minute peak periods of cortisol reactivity to stressors. Consistent with the pre-conflict sampling procedures, children chewed Trident original flavor sugarless gum to stimulate saliva flow and then expectorated through a plastic straw directly into a 20mL collection vial. In the interim between the SPAT and the two post-conflict sampling procedures, children were invited to play with toys in the room to limit extraneous stress and challenge that may result from inclusion of tasks or measures. The three saliva samples were immediately stored at –36°C until it was shipped on dry ice to Salimetrics LC (State College, PA).

Reports of Child Reactivity to Conflict—Following a brief break after the SPAT, mothers completed a measure of children's behavioral reactivity to interparental conflict in the home as part of a battery of questionnaires.

Measures

Observer Ratings of Child Behavioral Reactivity to Conflict—Children's behavioral reactions to the simulated conflict during SPAT were coded for distress, hostility, and involvement. To assess children's behavioral reactivity to interparental discord, judges coded the presence of specific types of the three behaviors (i.e., distress, hostility, involvement) for eight 30-second intervals during the 4 min. videotaped segment of the conflict and post-conflict periods. Specific behaviors indicative of distress reactions included: (1) freezing - tense, motionless, or “fixed in place” for 5 or more seconds; (2) anxiety - expressions of anxiety through facial expressions and/or physical gestures; and (3) verbal concern - verbalizations of concern about the interparental conflict. Measures of hostile behavior consisted of: (1) anger - facial expressions or postures reflecting anger; (2) interpersonal aggression - verbal or physical hostility directed toward the self or others, (3) object aggression - verbal or physical aggression directed toward objects. Finally, specific indicators of involvement were as follows: (1) comforting the parent: verbal or physical attempts to comfort or mediate the conflict, (2) inquiries about parent feelings or relationships- questions about the emotional state of the parent or quality of the interparental relationship (e.g., “Mom, are you okay?; “Is dad mad?”), and (3) triangulation - intervention strategies that reflect efforts to ally with one parent against the other parent. Although codes within each composite were designed to economically capture complementary dimensions (i.e., distinct with modest overlap) of the larger construct, codes were not mutually exclusive. Therefore, multiple behavioral codes (e.g., anger, interpersonal aggression) could be rated as present within any interval. Frequency counts of the specific indicators of each of

the three respective constructs were summed together to form composites of distress, hostile, and involvement behaviors. To establish reliability, two coders independently coded 25% of the videotapes. Intraclass correlation coefficients, reflecting interrater agreement in the frequency counts of each of the nine specific behavioral codes ranged from .64 to 1.0. Average intraclass coefficients were .86 for indicators of distress .74 for the hostility codes, and .91 for the involvement behaviors.

Maternal Reports of Behavioral Reactivity to Conflict—Mothers completed questionnaire items derived from the Security in the Marital Subsystem – Parent Report Inventory (SIMS-PR) to obtain indices of children's distress, hostile, and involvement responses to conflict in the home over the last year that were comparable to behavioral codes in the SPAT (Davies, Forman, Rasi, & Stevens, 2002). For the current report, three modified versions of the SIMS-PR subscales were utilized. The Distress (or Emotional Reactivity) scale consists of six items that reflect anxiety, distress and submissiveness (i.e., “appears frightened,” “keeps very still (almost as if he or she is frozen),” “still seems upset after we argue,” “appears upset,” “tries to hide feelings,” “repeatedly brings up questions and concerns about arguments after its over”). The Hostility scale contains five items that characterize angry and aggressive reactions to interparental conflict in the home (i.e., “yells at family members,” “says unkind things to family members,” “starts hitting, kicking, slapping, or throwing things at family members,” “appears angry,” “causes trouble”). The Involvement scale is comprised of five items that index children's behavioral intervention in interparental conflicts and their aftermath (e.g., “tries to help us solve the problem,” “tries to comfort one or both of us,” “watches and listens very closely,” “tells us to stop arguing,” “gets involved in the argument”). In further support of the factor structure of the SIMS-PR, an exploratory factor analysis of the 16 items generated a three factor solution that supported the specification of each of the items onto three respective subscales. Internal consistencies for the Distress, Hostility, and Involvement scales, as indexed by alpha coefficients, were .78, .78, and .73, respectively. The validity of the original SIMS-PR scales is supported by its theoretically meaningful associations with children's exposure to family conflict, coping difficulties with family stress, and psychological problems (e.g., Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings, 2006; Davies, Harold, Goeke-Morey, & Cummings, 2002).

Cortisol—All samples were assayed for salivary cortisol at Salimetrics, Inc. in duplicate using a highly-sensitive enzyme immunoassay (Salimetrics, PA). The test used 25 µl of saliva per determination, has a lower limit of sensitivity of 0.003 µg/dl, standard curve range of 0.007 to 1.8 µg/dl, and average intra- and inter-assay coefficients of variation 5.1% and 8.2%. Method accuracy, determined by spike recovery, and linearity, determined by serial dilution are 103 % and 96 %. Values from matched serum and saliva samples show the expected strong linear relationship, $r(63) = 0.89$, $p < 0.0001$ (Salimetrics, 2000).

Demographic and child characteristics—Five demographic and child variables were examined as potential covariates or moderators in the model: (a) blood contamination in saliva, (b) child medication use, (c) research site, (d) child gender, and (e) child race. First, to obtain an assessment of blood contamination, saliva samples were assayed for transferrin through immunoassays conducted by Salimetrics. The test, which uses 20 µl of saliva per assay, has a minimum detection limit of 0.12 mg/dl. Mean blood contamination levels ($M = .33$ mg/dl; $SD = .35$ mg/dl; range = .05 to 3.22 mg/dl) in this study were generally comparable to, if not lower than, prior studies (Kivlighan et al., 2004). Second, consistent with prior work (e.g., Hibel et al., 2006), mothers also reported on children's over-the-counter and prescription medication use. Children who were taking at least one medication (20%) were compared with children who were medication free (80%). The most frequently

used medications included acetaminophen or ibuprofen (6.7%) and non-steroidal anti-inflammatory drugs (6.7%), followed by relatively lower levels of over-the-counter cough/cold medicine (2.4%), vitamins (2.4%), antibiotics (1.4%), antacids (0.9%), and other medications (2.9%). Third, research site was coded as a dichotomous variable (i.e., Northeast site contrasted with Midwest site). Finally, in addition to examining child gender (i.e., girls contrasted with boys), child race was also explored as a fifth potential variable based on prior LHPA axis differences between White and Black adults (Black children contrasted with White children; Cohen et al., 2006).

Results

Descriptive Analyses

Table 1 provides the raw means, standard deviations, and correlations among the focal variables in the primary analyses. Correlations between distress, hostility, and involvement within each method of assessment (i.e., SPAT observation, maternal report) were modest in magnitude (mean $r = .20$; range = $-.03$ to $.35$). As expected, associations among conceptually related constructs (e.g., involvement) derived from different methods were also modest and significant (mean $r = .20$; range = $.15$ to $.24$). Modest correspondence across different informant reports of child psychological distress is very common in developmental and clinical research (De Los Reyes & Kazdin, 2005). As with prior research on child psychological problems, the multiple methods were derived from informants who amassed different types of information about children from different contexts (Achenbach, 2006). Log transformations of the three cortisol measures were used to reduce skewness and normalize distributions prior to the primary analyses, yielding the following means and standard deviations: pre-conflict ($M = -2.44$, $SD = 0.47$), post I conflict ($M = -2.70$, $SD = 0.44$), and post II conflict ($M = -2.75$, $SD = 0.43$).

Preliminary Analyses

The predominant strategy of assessing cortisol reactivity to stressors through the piecewise calculation of difference scores between pre-stressor and each post-stressor cortisol level in analysis of variance or regression models fails to: (a) disentangle measurement error from measures of reactivity and (b) effectively capture individual differences in cortisol trajectories across three or more measurement occasions (Hruschka, Kohrt, & Worthman, 2005). Because growth modeling addresses these limitations (for an example, see Powers, Pietromonaco, Gunlicks, & Sayer, 2006), we assessed the slope of change across the pre-conflict, post I, and post II cortisol assessments through the use of latent growth curve (LGC) modeling.

Given the diurnal rhythm of cortisol, our first step in LGC modeling involved specifying the effects of time of measurement, prior to the inclusion of the main predictors or any covariates or moderators. LGC specification was identical to the model depicted in Figure 1 without the inclusion of the three forms of conflict reactivity as predictors. The model fit the data well, $\chi^2(7, N = 208) = 2.84$, $p = .90$, $RMSEA = .000$, $CFI = 1.00$ and χ^2/df ratio = 0.41. As an estimate of pre-conflict cortisol levels, the mean level of the intercept was significantly different from 0 ($\mu_i = -1.60$, $z = 6.37$). The mean slope parameter, which characterizes average, constant change in cortisol across the three assessments, was positive but not significant ($\mu_s = 0.44$, $z = 1.67$). Notably, analysis of the variances for the intercept and slope factors yielded significant individual differences in the initial levels of cortisol ($\zeta_i = 0.18$, $z = 6.60$) and change in cortisol ($\zeta_s = 0.18$, $z = 4.23$). Therefore, the results support our aim of examining associations between individual differences in children's psychological and cortisol responses to interparental conflict.

Primary Analyses

Although associations between behavioral and adrenocortical functioning may reflect bidirectional influences between the systems, our goal was to identify the specificity and uniqueness of associations between children's patterns of behavioral reactivity and their cortisol reactivity. Accordingly, to examine the relative and distinct patterns of correspondence between behavioral and cortisol reactivity, we simultaneously examined children's distress, hostility, and involvement as predictors of cortisol activity in a series of growth models. The first model involved the specification of pathways from SPAT observational ratings of distress, hostility, and involvement to the initial levels (intercept) and change in cortisol in response to the conflict (slope). The model, which is depicted in Figure 1, provided an acceptable representation of the data, $\chi^2(19, N = 208) = 15.34, p = .70$, χ^2/df ratio = 0.81, $RMSEA = .000$, $CFI = 1.00$. Paths between children's behavioral responses and the cortisol slope are of particular relevance in light of our focus on children's cortisol reactivity to conflict. Consistent with hypotheses, children's distress was positively associated with cortisol reactivity to the simulated interparental conflict, $\beta = .23, p < .01$. In contrast, hostility and involvement were not associated with initial levels or change in cortisol reactivity. As a further test of the specificity of associations, we calculated pairwise parameter comparisons of the magnitude of paths running from each form of child psychological reactivity to child cortisol reactivity. The results indicated that the association between distress and cortisol reactivity was marginally stronger than comparable pathways involving hostility, $z = 1.60, p = .055$ (one-tailed), and involvement, $z = 1.60, p = .055$ (one-tailed).

To explore the viability of this model for children's psychological reactivity in the home, we also examined associations between maternal reports of children's reactivity to conflict in the home and their cortisol reactivity to the simulated interparental conflict. As shown in Figure 2, the model yielded a good representation of the data, $\chi^2(19, N = 208) = 34.16, p = .02$, χ^2/df ratio = 1.80, $RMSEA = .062$, $CFI = .99$. Consistent with the findings from the first model, maternal reports of children's distress responses to interparental conflict in the home were significantly associated with greater cortisol reactivity to conflict, $\beta = .27, p < .001$ and lower pre-conflict levels of cortisol, $\beta = -.18, p < .05$. Conversely, dampened cortisol reactivity was related to maternal reports of child hostility, $\beta = -.20, p = .01$. Analysis of pairwise comparisons among the pathways running from behavioral reactivity to the cortisol slope revealed that distress was a stronger predictor of greater cortisol reactivity than either hostility, $z = 3.53, p < .01$, or involvement, $z = 3.08, p < .01$.

Tests of the Stability of the Primary Findings

As a further test the generalizability of the results, we examined whether blood contamination in saliva, child medication use, research site, child gender, and child race may serve as both covariates and moderators that alter the nature of associations between children's distress and their cortisol reactivity to conflict. First, for both the SPAT and maternal report models of child reactivity depicted in Figures 1 and 2, we conducted five additional covariate analyses incorporating paths between each of the five demographic variables and the intercept and slope of child cortisol. Supporting the stability of the findings, all paths between the covariates and the intercept and slope of cortisol were nonsignificant. Moreover, inclusion of the demographic variables did not alter the significant paths between children's distress and child cortisol reactivity for either the SPAT or maternal ratings of children's responses to conflict. Second, moderator models examined whether the associations between distress and cortisol varied across the five demographic variables. We conducted separate models for each predictor and demographic variable pairing. In these analyses, the main effects of the distress variable and the specific demographic factor along with the two-way interaction term were all included as predictors

of the cortisol intercept and slope. The results of the analyses produced no more moderating effects for the five demographic variables (1 of 20 interactions were significant) than would be expected by chance. In conclusion, the results were consistent across a variety of contextual conditions, including research site, child gender, child race, blood contamination levels, and child medication use.

Child Involvement and Hostility as Moderators of Distress

Guided by our conceptual focus on distress, our final analytic model selectively examined whether associations between distress and child cortisol reactivity to conflict were moderated by children's involvement or hostile reactions to the conflict. Distress, involvement, and the distress $\times$ proposed moderator (i.e., either hostility or involvement) interaction variables were simultaneously estimated as predictors of the cortisol intercept and slope in the LGC analyses. Four separate statistical models were specified for each of the two measures (SPAT, maternal ratings) of the two proposed moderators after centering the predictors to reduce problems with multicollinearity (Aiken & West, 1991). For the sake of brevity, we focus on the results that directly address the paths between moderator terms and cortisol reactivity. Of the four moderator analyses conducted, only the interaction term involving SPAT observations of distress and involvement was a significant predictor of child cortisol reactivity ($B = .07, t = 2.38, p < .05$). To clarify the interaction in latent growth curve analyses of the SPAT, we followed the procedures outlined by Preacher, Curran and Bauer (2006) for: (a) plotting regression slopes of child distress at high (1 SD above the mean) and low (1 SD below the mean) levels of involvement and (b) conducting simple slope analyses to examine whether associations between distress and cortisol reactivity were significantly different from 0 at high (+ 1 SD) and low (−1 SD) levels of involvement. As the graphical plot in Figure 3 shows, the association between distress and the cortisol slope is particularly pronounced at high rather than low levels of involvement. In support of this interpretation, simple slope analyses indicated that observations of child distress predicted greater cortisol reactivity when levels of child involvement in the conflict were high, ($B = .15, t = 4.24, p < .001$), but not low ($B = -.01, t = 0.25, p = ns$).

Discussion

Various conceptualizations of children's coping with interparental conflict and children's psychobiological reactivity to social stressors converge to highlight the potential interplay between children's psychological and cortisol responses to interparental conflict (Davies et al., 2006; Gunnar & Vazquez, 2006; Levine & Weiner, 1988; Spangler & Schieche, 1998). However, little is known about the concurrent interconnections between children's reactivity at these different levels of analysis. Accordingly, this study aimed to break new ground by identifying the nature of the associations between multiple dimensions of children's psychological reactivity to interparental conflict and their cortisol functioning in response to interparental conflict.

Simultaneous analysis of pathways among children's distress, hostile, and involvement responses to interparental conflict and their cortisol reactivity to interparental conflict indicated that distress was significantly and uniquely associated with higher levels of cortisol reactivity to interparental conflict. Confidence in the validity of our findings is bolstered by the considerable stability of pathways between distress responding and cortisol reactivity to conflict across several conditions. First, the specificity of associations between cortisol and distress reactivity to conflict were consistent across measures of child reactivity to conflict that varied in use of method (i.e., observation, questionnaire), informant (i.e., observer, mother), and context (i.e., laboratory, home). Thus, children's heightened cortisol reactivity to simulated interparental conflict was not only specifically associated with observational ratings of children's distress responses within the same conflict context, but it

was also predicted by higher levels of maternal questionnaire reports of children's reactivity to interparental conflict in the home. Second, tests of differences in the magnitude of links between maternal reports of the three forms of psychological responding and cortisol reactivity indicated that the association between distress and cortisol reactivity was stronger than comparable paths involving child hostile and involvement responses to conflict. Third, paths between distress and cortisol reactivity were consistent even after including several demographic and child characteristics as confounding factors or moderators in the analyses.

Coherence in the results raises a central question: Why is distress reactivity to conflict uniquely associated with elevated cortisol responding to interparental conflict? Among the conceptualizations of linkages between psychological and physiological responding, the general arousal and negative affect models do not hold the precision necessary to readily explain the associations between distress and cortisol reactivity. In the general arousal model, the assumption that many forms of effortful responding to interparental conflict are coupled with greater LHPA activity would specifically suggest that distress, hostility, and involvement are relatively similar in their roles as predictors of cortisol levels. Correspondingly, the negative affect model does not distinguish between hostility and distress and assumes that negative emotionality is broad correlate of greater cortisol reactivity.

Rather, the distinctive confluence of children's heightened distress and cortisol reactivity to conflict found in the present study supports a distress specificity model and its main premise that distress is distinctly coupled with greater cortisol reactivity (Susman, Dorn, Inoff-Germain, Nottelmann, & Chrousos, 1997). Profiles characterized by elevated distress and LHPA activity in the context of interparental conflict may signify a hyper-reactive threat detection system (Gunnar & Vazquez, 2006). Supporting this interpretation, EST posits that elevated distress in response to interparental conflict is a hallmark symptom of children's concerns about security (Cummings & Davies, 1996).

In further informing the distress specificity model, EST also suggests that hypersensitivity to threat may be particularly evident when distress responses to interparental conflict occur in conjunction with high levels of involvement in the conflict. In this context, attempts to intervene in adult problems by children who are distressed by interparental conflict may signify a last resort, defense strategy for preserving security when alternative solutions and resources are not accessible (Davies et al., 2006). Thus, if the blending of distress and involvement does reflect extreme considerable alarm, then distress should be a particularly potent predictor of cortisol reactivity to conflict when child involvement levels are also high. In partially supporting this interpretation, follow up analyses of an interaction between child distress and involvement revealed that children's distress in the simulated conflict was associated with elevated cortisol reactivity to conflict only when they were also highly involved in the parental problems.

Conceptualizations of threat and challenge help to further address the mechanisms responsible for the confluence of distress and elevated cortisol reactivity. For example, one hypothesis derived from the distress specificity model is that elevations in cortisol are most likely to occur when children cannot access the resources necessary to cope with the stressor or perceive the stressor as beyond their control (Levine & Weiner, 1988; Spangler & Schieche, 1998). Applied to our findings, underlying doubts about the ability to utilize strategies necessary to preserve goals of safety and security may underlie the association between children's distress and adrenocortical reactivity (Saarni, Mumme, & Campos, 2006). Similarly, social psychological frameworks draw important distinctions between threat and challenge. Whereas the experience of threat is theorized to reflect appraisals that stressful contexts exceed coping resources available to the individual, challenges reflect

perceptions that skills and resources available will be sufficient to cope with the demands of the context (Blascovich & Mendes, 2000; Dienstbier, 1989). According to these models, threat is hypothesized to be linked with elevated LHPA activity (Dienstbier, 1989; Mendes, Blascovitch, Lickel, & Hunter, 2002). Integrated with the EST assumption that distress is a key indicator of concerns about safety, one interpretation is that the coupling of distress and cortisol reactivity to conflict may be rooted in the common underlying experience of threat. Although still speculative at this early stage of research, there are some bases for expecting that this response pattern may increase children's adjustment problems, particularly in the form of internalizing symptoms. For example, independent lines of research have identified children's internalizing symptoms as common sequelae of children's heightened adrenocortical reactivity to stressful events (Gunnar & Vazquez, 2006) and their elevated distress responses to interparental conflict (Davies & Cummings, 2006).

Two additional, but inconsistent, findings from our analyses raise more speculative questions. First, maternal report of child hostility was associated with dampened cortisol reactivity to the simulated interparental conflict. Given that this pathway was only identified in one of the two main analytic models, considerable care should be taken in interpreting the results. Nevertheless, one tentative explanation derived from social psychological conceptualizations of challenge is that high levels of confidence in the ability to weather the stressfulness of interparental conflict may underlie the experience of hostile and dampened cortisol reactions to interparental conflict (e.g., Blascovitch & Mendes, 2000). A related interpretation drawn from the attenuation hypothesis is that hostility may be considered dominant, bold, or callous response that may be associated with underarousal of the LHPA system in response to interparental conflict (Susman, 2006). Assuming this finding is replicated in the future, a critical step in future research is to identify the long-term implications of this response pattern. For example, within the attenuation hypothesis (Susman, 2006) the co-occurrence of hostility and dampened cortisol responses may reflect underlying disturbances in neurobiological processing of emotional and fear-relevant parameters of events that ultimately deepen and expand into broader externalizing problems. Second, the findings also indicated that maternal reports of distress were associated with lower pre-conflict levels of cortisol. However, a comparable path between SPAT distress and pre-conflict cortisol was not significant. Thus, the findings, as a whole, suggest that distress responses to interparental conflict are not consistent correlates of a broad, trait-like condition of hypercortisolism across multiple contexts. Rather, distress responses to conflict evidenced specificity in its power to predict greater cortisol reactivity to conflict.

Fully interpreting the results of our study also requires consideration of the methodological limitations. Future research on children's coping with family conflict and their cortisol functioning would benefit by increasing the rigor of psychological and physiological assessments. For example, our identification of pathways between distress reactivity and the intercept and slope parameters of cortisol functioning does not afford a thorough analysis of specificity or breadth of the adrenocortical correlates of distress responses to conflict. One important step will be to determine whether distress responding to interparental conflict is specifically associated with cortisol reactivity to interparental conflict or broader indices of cortisol reactivity to a wide array of family, interpersonal, or even more global (i.e., physical and psychosocial) stressors. Likewise, guided by conceptualizations of emotional reactivity in EST, our assessments of emotional distress were largely comprised of assessments of anxiety, fear, and general distress. As a result, our measurement battery did not permit a full analysis of associations between adrenocortical reactivity and other forms of negative emotion such as sadness. In addition, contextualizing the study of cortisol within broader profiles of functioning across multiple physiological systems may also advance psychophysiological models of coping with family conflict (Doussard-Roosevelt et al., 2003; Gordis, Granger, Susman, & Trickett, 2006; Granger et al., 2006). For example, the

modest to moderate magnitude of associations between psychological and physiological reactivity to conflict may be augmented by analyses of synchrony in functioning between the LHPA axis and the parasympathetic (e.g., Doussard-Roosevelt et al., 2003) or sympathetic (e.g., Gordis et al., 2006) nervous systems.

Future advances in identifying links between children's behavioral and physiological reactivity to conflict will also benefit from modifications in the temporal spacing of assessments. For example, the number and timing of our cortisol assessments do not permit a definitive analysis of the pattern of cortisol reactivity across stressor and recovery periods (Matthews, Gump, & Owens, 2001). Greater numbers of cortisol assessments in future research would afford flexibility in analyzing both linear and curvilinear trajectories of cortisol over time. Moreover, delineation of individual differences in recovery of cortisol may be achieved by lengthening the temporal spacing between the stressor and some of the cortisol assessments. However, relative to more common methods of assessing cortisol reactivity (e.g., difference scores between pre-conflict and a single post-conflict measure of cortisol), our latent growth curve analysis generated a richer characterization of cortisol reactivity through its ability to incorporate multiple assessments of cortisol that are sensitive to the direction and shape of the trajectory. Likewise, although our use of cross-sectional design is well-suited to address the goal of examining the concurrent correspondence between cortisol and behavioral reactivity to conflict, use of longitudinal designs will afford tests of complementary questions about possible transactional influences among behavioral and adrenocortical reactivity over longer time periods.

Finally, although the demographic characteristics of our sample of families were highly comparable to the Northeast and Midwest regions of the U.S., the findings in this study were derived from a community sample of predominantly White families. Therefore, the results of our study may not necessarily generalize to families with other racial, ethnic, or high-risk backgrounds. Nevertheless, the failure of a wide array of demographic characteristics to alter as covariates or moderators the pathways found between children's distress and cortisol reactivity to interparental conflict provides some bases for the generalizability of the results.

In conclusion, our multi-method, multiple level of analysis study is the first foray into identifying the adrenocortical underpinnings of children's behavioral and psychological responses to interparental conflict. Consistent with the distress specificity model in the psychophysiological literature, children's distress responding to interparental conflict was consistently linked with latent growth curve analyses of greater cortisol reactivity to interparental conflict across multiple methods (i.e., observations, questionnaires), informants (i.e., observer, mother), and contexts (i.e., laboratory, home). Moreover, the pathways were distinctive when also taking into consideration other forms of children's reactivity (i.e., hostility, involvement) to conflict and a range of sociodemographic variables as potential confounding factors. In broader accord with EST, children's security in the interparental relationship may be a central goal system organizing the confluence of distress and heightened cortisol reactivity to conflict.

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References

- Achenbach TM. As others see us: Clinical and research implications of cross-informant correlations for psychopathology. *Current Directions in Psychological Science*. 2006; 15:94–98.
- Aiken, LS.; West, SG. Multiple regression: Testing and interpreting interactions. Sage; London: 1991.
- Ashman SB, Dawson G, Panagiotides H, Yamada E, Wilkinson CW. Stress hormone levels of children of depressed mothers. *Development and Psychopathology*. 2002; 14:333–349. [PubMed: 12030695]
- Blascovich, J.; Mendes, WB. Challenge and threat appraisals: The role of affective cues.. In: Forgas, J., editor. *Feeling and thinking: The role of affect in social cognition*. Cambridge University Press; Paris: 2000. p. 59-82.
- Cahill L, McGaugh JL. Mechanisms of emotional arousal and lasting declarative memory. *Trends in Neurosciences*. 1998; 21:294–299. [PubMed: 9683321]
- Cicchetti D, Tucker D. Development and self-regulatory structures of the mind. *Development and Psychopathology*. 1994; 6:533–549.
- Cohen S, Schwartz JE, Epel E, Kirschbaum C, Sidney S, Seeman T. Socioeconomic status, race, and diurnal cortisol decline in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Psychosomatic Medicine*. 2006; 68:41–50. [PubMed: 16449410]
- Cummings, EM.; Davies, PT. Children and marital conflict: The impact of family dispute and resolution. Guilford Press; New York: 1994.
- Cummings EM, Davies P. Emotional security as a regulatory process in normal development and the development of psychopathology. *Development and Psychopathology*. 1996; 8:123–139.
- Cummings, EM.; Davies, P.; Campbell, SB. Developmental psychopathology and family process: Theory, research, and clinical implications. Guilford Press; New York: 2000.
- Cummings EM, Schermerhorn AC, Davies PT, Goeke-Morey MC, Cummings JS. Interparental discord and child adjustment: Prospective investigations of emotional security as an explanatory mechanism. *Child Development*. 2006; 77:132–152. [PubMed: 16460530]
- Davies PT, Cummings EM. Marital conflict and child adjustment: An emotional security hypothesis. *Psychological Bulletin*. 1994; 116:387–411. [PubMed: 7809306]
- Davies, PT.; Cummings, EM. Interparental discord, family process, and developmental psychopathology.. In: Cicchetti, D.; Cohen, DJ., editors. *Developmental Psychopathology: Vol. 3: Risk, Disorder, and Adaptation*. 2nd ed.. Wiley & Sons; New York: 2006. p. 86-128.
- Davies PT, Cummings EM, Winter MA. Pathways between profiles of family functioning, child security in the interparental subsystem, and child psychological problems. *Development and Psychopathology*. 2004; 16:525–550. [PubMed: 15605624]
- Davies PT, Forman EM, Rasi JA, Stevens KI. Assessing children's emotional security in the interparental subsystem: The Security in the Interparental Subsystem (SIS) Scales. *Child Development*. 2002; 73:544–562. [PubMed: 11949908]
- Davies PT, Harold GT, Goeke-Morey M, Cummings EM. Child emotional security and interparental conflict. *Monographs of the Society for Research in Child Development*. 2002; 67 3, Serial No. 270.
- Davies PT, Winter MA, Cicchetti D. The implications of emotional security theory for understanding and treating childhood psychopathology. *Development and Psychopathology*. 2006; 18:707–735. [PubMed: 17152397]
- De Los Reyes A, Kazdin AE. Informant discrepancies in the assessment of childhood psychopathology: A critical review, theoretical framework, and recommendations for further study. *Psychological Bulletin*. 2005; 131:483–509. [PubMed: 16060799]
- Deinstbier RA. Arousal and physiological toughness: Implications for mental and physical health. *Psychological Review*. 1989; 96:84–100. [PubMed: 2538855]
- Dettling AC, Gunnar MR, Donzella B. Cortisol levels of young children in full-day childcare centers: Relations with age and temperament. *Psychoneuroendocrinology*. 1999; 24:519–536. [PubMed: 10378239]
- Dettling AC, Parker SW, Lane S, Sebanc A, Gunnar MR. Quality of care and temperament determine changes in cortisol concentrations over the day for young children in child care. *Psychoneuroendocrinology*. 2000; 25:819–836.

- Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*. 2004; 130:355–391. [PubMed: 15122924]
- Doussard-Roosevelt JA, Montgomery LA, Porges SW. Short-term stability of physiological measures in kindergarten children: Respiratory sinus arrhythmia, heart period, and cortisol. *Developmental Psychobiology*. 2003; 43:231–242.
- Fox, NA.; Hane, AA.; Perez-Edgar, K. Psychophysiological methods for the study of developmental psychopathology.. In: Cicchetti, D.; Cohen, DJ., editors. *Developmental Psychopathology: Vol. 2: Developmental Neuroscience*. 2nd ed.. Wiley; New York: 2006. p. 381-426.
- Gordis EB, Granger DA, Susman EJ, Trickett PK. Asymmetry between salivary cortisol and alpha - amylase reactivity to stress: Relation to aggressive behavior in adolescents. *Psychoneuroendocrinology*. 2006; 31:976–987. Elsevier Science. [PubMed: 16879926]
- Granger DA, Cicchetti D, Rogosch FA, Hibel LC, Teisl M, Flores E. Blood contamination in children's saliva: Prevalence, stability, and impact on the measurement of salivary cortisol, testosterone, and dehydroepiandrosterone. *Psychoneuroendocrinology*. 2007; 32:724–733. [PubMed: 17582690]
- Granger DA, Kivlighan KT, Blair C, El-Sheikh M, Mize J, Lisonbee JA, Buckhalt JA, Stroud LR, Handwerker K, Schwartz EB. Integrating the measurement of salivary alpha -amylase into studies of child health, development, and social relationships. *Journal of Social and Personal Relationships*. 2006; 23:267–290.
- Granger DA, Weisz JR, Kauneckis D. Neuroendocrine reactivity, internalizing behavior problems, and control-related cognitions in clinic-referred children and adolescents. *Journal of Abnormal Psychology*. 1994; 103:267–276. [PubMed: 8040496]
- Grych JH, Fincham FD. Marital conflict and children's adjustment: A cognitive-contextual framework. *Psychological Bulletin*. 1990; 108:267–290. [PubMed: 2236384]
- Gunnar MR, Donzella B. Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology*. 2000; 27:199–220. [PubMed: 11750779]
- Gunnar M, Larson M, Hertzgaard L, Harris M, Brodersen L. The stressfulness of separation among 9-month-old infants: Effects of social context variables and infant temperament. *Child Development*. 1992; 63:290–303. [PubMed: 1611934]
- Gunnar, MR.; Vazquez, DM. Stress neurobiology and developmental psychopathology.. In: Cicchetti, D.; Cohen, DJ., editors. *Developmental Psychopathology: Vol. 2: Developmental Neuroscience*. 2nd ed.. Wiley & Sons; New York: 2006. p. 533-577.
- Hertzgaard L, Gunnar M, Erickson MF, Nachmias M. Adrenocortical responses to the Strange Situation in infants with disorganized/disoriented attachment relationships. *Child Development*. 1995; 66:1100–1106. [PubMed: 7671652]
- Hibel LC, Granger DA, Kivlighan KT, Blair C, the Family Life Investigators. Individual differences in salivary cortisol: Associations with common over-the-counter and prescription medication status in infants and their mothers. *Hormones and Behavior*. 2006; 50:293–300. [PubMed: 16682032]
- Hibel LC, Granger DA, Cicchetti D, Rogosch F. Salivary biomarker levels and diurnal variation: Associations with medications prescribed to control children's problem behaviors. *Child Development*. 2007; 78:927–937. [PubMed: 17517013]
- Hruschka DJ, Kohrt BA, Worthman CM. Estimating between- and within-individual variation in cortisol levels using multilevel models. *Psychoneuroendocrinology*. 2005; 30:698–714. [PubMed: 15854786]
- Kagan J, Reznick JS, Snidman N. Biological bases of childhood shyness. *Science*. 1998; 240:167–171. [PubMed: 3353713]
- Katz, LF. Physiological processes and mediators of the impact of marital conflict on children.. In: Grych, JH.; Fincham, FD., editors. *Interparental conflict and child development: Theory, research, and applications*. Cambridge University Press; New York: 2001. p. 188-212.
- Knutson U, Dahlgreen J, Marcus C, Rosberg S, Bronnegard M, Stierna P, Albertsson-Wikland K. Circadian cortisol rhythms in healthy boys and girls: Relationships with age, growth, body composition, and pubertal development. *Journal of Clinical Endocrinology and Metabolism*. 1997; 82:536–540. [PubMed: 9024250]

- Levine S, Weiner SG. Psychoendocrine aspects of mother-infant relationships in nonhuman primates. *Psychoneuroendocrinology*. 1988; 13:143–154. [PubMed: 3287413]
- Loney BR, Butler MA, Lima EN, Counts CA, Eckel LA. The relation between salivary cortisol, callous-unemotional traits, and conduct problems in an adolescent non-referred sample. *Journal of Child Psychology and Psychiatry*. 2006; 47:30–36. [PubMed: 16405638]
- Masharani U, Shiboski S, Eisner MD, Katz PP, Janson SL, Granger DA, Blanc PD. Impact of exogenous glucocorticoid use on salivary cortisol measurements among adults with asthma and rhinitis. *Psychoneuroendocrinology*. 2005; 30:744–752. [PubMed: 15919580]
- Matthews KA, Gump BB, Owens JF. Chronic stress influences cardiovascular and neuroendocrine responses during acute stress and recovery, especially for men. *Health Psychology*. 2001; 20:403–410. [PubMed: 11714181]
- Mendes WB, Blascovich J, Hunter SB, Lickel B, Jost JT. Threatened by the unexpected: Physiological responses during social interactions with expectancy-violating partners. *Journal of Personality and Social Psychology*. 2007; 92:698–716. [PubMed: 17469953]
- National Institutes of Health. Program announcement: Research on children exposed to violence (PAR-03–096). 2003 [December 23, 2007]. from http://grants.nih.gov/grants/guide/search_results.htm?year=active&scope=pa
- Powers SI, Pietromonaco PR, Gunlicks M, Sayer A. Dating couples' attachment styles and patterns of cortisol reactivity and recovery in response to a relationship conflict. *Journal of Personality and Social Psychology*. 2006; 90:613–628. [PubMed: 16649858]
- Preacher KJ, Curran PJ, Bauer DJ. Computational tools for probing interaction effects in multiple linear regression, multilevel modeling, and latent curve analysis. *Journal of Educational and Behavioral Statistics*. 2006; 31:437–448.
- Sarni, C.; Campos, JJ.; Camras, LA.; Witherington, D. Emotional development: Action, communication, and understanding.. In: Eisenberg, N.; Damon, W.; Lerner, RM., editors. *Handbook of child psychology: Vol. 3. Social, emotional, and personality development*. 6th ed.. Wiley; Hoboken, NJ: 2006. p. 226-299.
- Salimetrics, LLC. HS cortisol kit information. State College; PA: 2000. Unpublished manuscript
- Saltzman KM, Holden GW, Holahan CJ. The psychobiology of children exposed to marital violence. *Journal of Clinical Child and Adolescent Psychology*. 2005; 34:129–139. [PubMed: 15677287]
- Schwartz EP, Granger DA, Susman EJ, Gunnar MR, Laird B. Assessing salivary cortisol in studies of child development. *Child Development*. 1998; 69:1503–1513. [PubMed: 9914636]
- Spangler G, Grossman KE. Biobehavioral organization in securely and insecurely attached infants. *Child Development*. 1993; 64:1439–1450. [PubMed: 8222882]
- Spangler G, Schieche M. Emotional and adrenocortical responses of infants to the Strange Situation: The differential function of emotional expression. *International Journal of Behavioral Development*. 1998; 22:681–706.
- Stansbury K, Gunnar MR, Fox NA. Adrenocortical activity and emotion regulation. *The Development of Emotion Regulation: Biological and behavioral considerations*. Monographs of the Society for Research in Child Development. 1994; 59:108–134.
- Susman EJ. Psychobiology of persistent antisocial behavior: Stress, early vulnerabilities, and the attenuation hypothesis. *Neuroscience and Biobehavioral Reviews*. 2006; 30:376–389. [PubMed: 16239030]
- Susman EJ, Dorn LD, Inoff-Germain G, Nottelmann ED, Chrousos GP. Cortisol reactivity, distress behavior, and behavioral and psychological problems in young adolescents: A longitudinal perspective. *Journal of Research on Adolescence*. 1997; 7:81–105.
- Tout K, de Haan M, Campbell EK, Gunnar MR. Social behavior correlates of cortisol activity in child care: Gender differences and time-of-day effects. *Child Development*. 1998; 69:1247–1262. [PubMed: 9839413]

Figure 1. Associations between children's observed psychological reactivity and their cortisol reactivity to the simulated interparental conflict. SPAT = Simulated Phone Argument Task. * $p < .05$; ** $p < .01$

Figure 2.

Associations between maternal reports of children's psychological reactivity to interparental conflict in the home and their cortisol reactivity to the simulated interparental conflict. * $p < .05$; ** $p < .01$

Figure 3.

A graphical plot of the significant interaction between observations of children's distress and involvement responses to interparental conflict predicting their cortisol reactivity to the simulated interparental conflict. SPAT = Simulated Phone Argument Task.

Table 1

Means, standard deviations, and intercorrelations of the primary variables in the study.

Variables	Mean	SD	1	2	3	4	5	6	7	8
Child Interparental Conflict Reactivity (SPAT Observation)										
1. Distress	1.32	1.39	--							
2. Hostility	0.39	1.04	-.03	--						
3. Involvement	0.45	0.71	.28*	-.02	--					
Child Interparental Conflict Reactivity in the Home (Maternal Report)										
4. Distress	9.75	3.84	.15*	.07	.07	--				
5. Hostility	7.00	2.74	.04	.24*	-.02	.34*	--			
6. Involvement	12.40	4.45	.09	-.05	.20*	.35*	.25*	--		
Child Cortisol Reactivity										
7. Pre-Conflict (µg/dl)	0.097	0.044	-.11	.05	.00	-.06	.03	.02	--	
8. Post I (µg/dl)	0.073	0.032	.01	.00	.05	.04	-.03	.03	.71*	--
9. Post II (µg/dl)	0.070	0.030	.01	.03	-.01	-.03	-.03	-.03	.60*	.86*

Note.

*
 $p < .05$.