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The Role of Child Adrenocortical Functioning in Pathways Between Interparental Conflict and Child Maladjustment

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Abstract

This study examined the interplay between interparental conflict and child cortisol reactivity to interparental conflict in predicting child maladjustment in a sample of 178 families and their kindergarten children. Consistent with the allostatic load hypothesis (McEwen & Stellar, 1993), results indicated that interparental conflict was indirectly related to child maladjustment through its association with individual differences in child cortisol reactivity. Analyses indicated that the multi-method assessment of interparental conflict was associated with lower levels of child cortisol reactivity to a simulated phone conflict between parents. Diminished cortisol reactivity, in turn, predicted increases in parental reports of child externalizing symptoms over a two-year period. Associations between interparental conflict, child cortisol reactivity, and child externalizing symptoms remained robust even after taking into account demographic factors and other family processes.

Interparental conflict has been shown to be linked with child maladjustment through its association with children's hostile cognitions, negative emotionality, and behavioral distress in the context of interparental conflict (Davies & Cummings, 2006). In identifying the role of social-cognitive responding, Grych and colleagues have repeatedly delineated children's appraisals of threat and self-blame in response to conflict as key intervening mechanisms linking exposure to interparental conflict with children's psychological problems within a variety of designs (e.g., cross-sectional, longitudinal) and samples (e.g., community, families in battered women's shelters) (Grych, Fincham, Jouriles, & McDonald, 2000; Grych, Harold, & Miles, 2003). Highlighting the role of emotional mechanisms, children from high conflict homes also exhibit greater subjective and behavioral distress to interparental conflict in both concurrent and prospective designs (Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings, 2006; Davies, Sturge-Apple, Winter, Cummings, & Farrell, 2006). Negative emotional reactivity to conflict, in turn, is a consistent predictor of children's internalizing and externalizing symptoms (Cummings et al., 2006; Davies, Harold, Goeke-Morey, & Cummings, 2002).

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However, relative to the study of cognitive, emotional, and behavioral functioning, progress in understanding the biological functioning of children exposed to elevated interparental conflict has been slow (El-Sheikh, Harger, & Whitson, 2001). The few studies addressing physiological activity in models of interparental conflict have primarily focused on cardiovascular activity as a marker of sympathetic-adrenomedullary (SAM) functioning (e.g., Ballard, Cummings, & Larkin, 1993; El-Sheikh, 1994; El-Sheikh, Harger, & Whitson, 2001; Katz & Gottman, 1997). Consequently, little is known about the functioning of the limbic-hypothalamic-pituitary-adrenocortical (LHPA) system – another component of the stress system that may be a pivotal process linking early family experiences to the development of children's psychological adjustment (Heim & Nemeroff, 2001; Susman, 2006). In response to calls to integrate the study of the LHPA system in family processes within developmental frameworks (Calkins & Howse, 2004; Cicchetti, 2002; Repetti, Taylor, & Seeman, 2002), this study examines how children's adrenocortical functioning informs the relationship between interparental conflict and child psychological symptoms.

Conceptual frameworks in the broader literature on family risk provide useful guides for understanding children's biological reactivity to interparental conflict (Evans, 2003; Lupien, King, Meaney, & McEwen, 2001; Susman, 2006). Allostasis and allostatic load are primary concepts shared by many of these conceptualizations. Allostasis refers to the process by which biological "set points" in homeostasis are altered to generate physiological resources necessary to promote survival in the face of environmental stress and challenge. However, in highlighting the operation of allostatic load, successive cycles of allostasis engendered by repeated exposure to environmental adversity are theorized to result in wear and tear on the body that undermines the integrity of multiple domains of functioning. Building on the concept of allostatic load, developmental models have postulated that exposure to family adversity disrupts the operation of children's neurobiological systems over time and eventuates in the manifestation of psychological problems (Cicchetti, 2002; Katz, 2001; Repetti et al., 2002; Susman, 2006). Children's neurobiological systems are specifically assumed to be increasingly governed by the goal of preserving their physical and psychological integrity in the stressful context of high conflict homes. Consistent with this premise, experiential histories with stressful stimuli have been shown to cumulatively alter neurobiological responses to subsequent stress and, in the process, affect brain and behavioral functioning (Cahill & McGaugh, 1998). For example, studies support the notion that stress hormones (e.g., epinephrine, glucocorticoids) modify the regulatory effects of the amygdala on memory consolidation of emotionally significant events in other brain regions (e.g., Cahill & McGaugh, 1998; Roozendaal, & McGaugh, 1996).

The LHPA axis is a component of the neuroendocrine stress system that may play a prominent role in understanding the developmental sequelae of family adversity. Cortisol is a hormonal product of a sequence of processes in the LHPA axis and is conceptualized as part of a "second wave" of autonomic responses to aversive or challenging events that follow SAM responses (Cahill & McGaugh, 1998). In response to stressful events, components of the limbic system (e.g., amygdala, hippocampus) involved in processing aversive stimuli stimulate the release of corticotropin-releasing factor (CRF). CRF, in turn, ultimately activates the adrenal gland to secrete cortisol by stimulating the pituitary gland to produce and release of adrenocorticotropic hormone (ACTH) into the bloodstream. Increases in cortisol in response to stress serve a short-term adaptive function of mobilizing energy (e.g., glucose, oxygen), increasing cardiovascular activity, and modulating the processing, learning, and memory consolidation of emotionally significant events (Cahill & McGaugh, 1998; Gold & Chrousos, 2002; Gunnar & Vazquez, 2006; Lupien et al., 2006). Within the bounds of normal LHPA axis functioning, high levels of cortisol set in motion a negative feedback cycle which inhibits the subsequent release of CRF and ACTH, ultimately leading to a decrease in cortisol to basal levels (Gunnar & Vazquez, 2006; Heim &

Nemeroff, 2001; Tsigos & Chrousos, 2002). Given the deleterious consequences of overactivation and underactivation of the LHPA axis for physical, neuropsychological, and psychological functioning (Heim, Ehlert, & Hellhammer, 2000; Tsigos & Chrousos, 2002), healthy functioning is characterized by efficient onset and termination of the LHPA system, tailored to the demands of the context (Meyer, Chrousos, & Gold, 2001).

Building on the allostatic load conceptualization of the LHPA axis as an "environmentally sensitive physiological system" (Granger et al., 1998, p. 709), a primary thesis is that exposure to family adversity, over time, progressively alters the operation of the LHPA system, with the perturbations ultimately having negative repercussions for psychological adjustment (Levine, 1994; Repetti et al., 2002). Further supporting this thesis, the emotional security theory in the family conflict literature postulates that the stressfulness of exposure to interparental conflict leads to child psychological problems by directly undermining children's ability to cope with subsequent stress across multiple response domains (e.g., Davies et al., 2002). In particular, children from high conflict homes are especially likely to feel threatened in the context of interparental conflict, as disagreements in these homes are more likely to continue for longer periods of time, escalate into bouts of hostility and aggression, and proliferate to include the child. Therefore, the LHPA axis is hypothesized to play a primary role in understanding children's reactivity to interparental conflict in light of its significant role in allocating resources to preserve the integrity of organisms in contexts of threat. However, different versions of allostatic load theories provide alternative formulations of how stress alters the neurobiological system and, in turn, increase children's risk for psychological problems.

The *hypercortisolism hypothesis* posits that chronic exposure to environmental adversity may sensitize the LHPA axis to subsequent stressors. Thus, this model postulates that the LHPA axis system becomes increasingly sensitized in its function of marshalling, directing, and sustaining resources to cope with the threat in the face of recurring interparental conflict. Consistent with this hypothesis, several family process theories share the thesis that repeated exposure to interparental conflict sensitizes children's emotional and behavioral responses to subsequent interparental difficulties (e.g., Cummings & Davies, 2002; Davies & Cummings, 2006; Grych & Fincham, 1990). By extension, if sensitization operates broadly across multiple response domains, destructive conflict may engender greater cortisol reactivity to stress. In support of the hypercortisolism hypothesis, parental withdrawal, dysphoria, stress, and some forms of maltreatment (e.g., co-occurrence of physical and sexual abuse) have been linked with higher cortisol levels (Cicchetti & Rogosch, 2001a; 2001b; Essex, Klein, Cho, & Kalin, 2002; Pine & Charney, 2002). In the second link in this process model, the hyperactivity of the LHPA axis system resulting from family discord is posited to increase child vulnerability to mental illness by undermining multiple (e.g., affective, cognitive, neurological, and biological) systems of functioning (Repetti et al., 2002). Consistent with this hypothesis, some studies have shown that children with psychological problems experience elevated cortisol levels (Essex et al., 2002; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001). Thus, according to the hypercortisolism hypothesis, heightened adrenocortical reactivity to interparental discord may be a key mechanism linking interparental conflict with child psychological problems.

In contrast, the *hypocortisolism hypothesis* suggests that the environmental stressors may be linked with child psychological problems through diminished cortisol reactivity to stress. Hypocortisolism may signify the blunting of distressing emotional experiences associated with exposure to adversity (Gunnar & Vazquez, 2001; Heim et al., 2000). Supporting this assumption, the emotional security theory proposes that the dampening of some domains of reactivity to interparental conflict is common in high conflict homes (Davies & Forman, 2002). For example, hypocortisolism may be a neuroendocrine manifestation of strategies to

disengage or disconnect from adverse experiences. At a neurobiological level, the attenuation hypothesis further postulates that diminished cortisol reactivity to stressful events following chronic histories of exposure to adverse socialization contexts serves an adaptive function by thwarting the negative impact of prolonged cortisol elevations on the brain, cardiovascular, and immune functioning (Susman, 2006; Gold & Chrousos, 2002). Despite its proposed adaptive function in discordant homes, the attenuation hypothesis suggests that diminished cortisol reactivity may increase children's risk for developing behavior problems through its association with disruptions in the processing, memories, and emotional reactions to aversive events (Susman, 2006).

Interpreted within these models, destructive interparental conflict may increase children's psychological maladjustment by dampening cortisol reactivity to subsequent stressors. Although research has yet to examine associations between interparental conflict and cortisol functioning, some studies support the role of hypocortisolism in associations between family discord and child behavior problems. For example, reports of negative associations between family risk factors (e.g., family conflict, parenting disturbances) and child cortisol levels are not uncommon in the literature (e.g., Cicchetti & Rogosch, 2001a; 2001b; Granger et al., 1998; Gunnar & Vazquez, 2001). Likewise, low levels of cortisol correlate with greater child psychological problems in some studies (Granger, Weisz, McCracken, Ikeda, & Douglas, 1996; Granger et al., 1998; Hart, Gunnar, & Cicchetti, 1995; Lopez, Vazquez & Olson, 2004; McBurnett et al., 1991).

The viability of the hypercortisolism and hypocortisolism hypotheses may partially depend on the types of psychological symptoms experienced by children. Studies have consistently revealed that lower levels of cortisol activity are associated with greater externalizing symptoms in children (e.g., Granger et al., 1996; 1998; Hart et al., 1995; Shirtcliff, Granger, Booth, & Johnson, 2005). In contrast, data on linkages between cortisol activity and child internalizing symptoms are far from definitive. Whereas cortisol activity or reactivity assessments have been associated with higher levels of internalizing symptoms in some studies (e.g., Granger, Weisz, & Kauneckis, 1994; Klimes-Dougan et al., 2001), other research provides evidence for diminished cortisol activity among children with internalizing symptomatology (e.g., de Haan, Gunnar, Tout, Hart, & Stansbury, 1998; Jansen et al., 1999; Lopez et al., 2004) or negligible associations between cortisol and internalizing symptoms (e.g., Shirtcliff et al. 2005). Due to the discrepancy in the literature with regard to cortisol and forms of child maladjustment, the present study examined child externalizing and internalizing symptoms separately.

The goal of this study is to examine the relative viability of the two versions of the allostatic load hypothesis in understanding pathways among interparental conflict, child cortisol reactivity to interparental discord, and child psychological problems. From the perspective of the emotional security theory, cortisol reactivity to stress or challenge is a more informative assessment of neuorendocrine functioning than basal levels of cortisol because it reflects the LHPA axis' capacity to effectively mobilize and modulate resources (e.g., glucose) necessary to meet the threat posed by stressful events (see Lopez et al., 2004). Thus, given the relevance of individual differences in the ability to garner resources to cope with threat in theories of interparental conflict (Davies & Cummings, 2006), our goal was to delineate the role of cortisol reactivity to parental conflict in associations between interparental conflict and child maladjustment. To increase the ecological validity of our cortisol measures, we assessed child cortisol reactivity in response to a simulated interparental conflict and resolution. Likewise, the predominant use of cross-sectional designs in the cortisol literature cannot adequately test the hypothesis that differences between children in adrenocortical reactivity predict their subsequent psychological functioning (Granger et al., 1996; Repetti et al., 2003). To address this gap, we examine

whether individual differences in the growth curves of children's cortisol reactivity to inteparental conflict predict changes in child psychological maladjustment over two years.

Testing the stability and generalizability of pathways among interparental conflict, child cortisol reactivity, and child psychological problems is another central goal of this paper. Individual differences in family (i.e., parenting), demographic (i.e., race, gender, SES), methodological (i.e., site) characteristics may serve as confounding conditions that reduce, offset, or otherwise alter the magnitude of pathways. Notably, parenting difficulties and variations in socioeconomic status co-vary with interparental conflict (e.g., Almeida, Wethington, & Chandler, 1999; Conger, Ge, Elder, Lorenz, & Simons, 1994) and have also been associated with individual differences in children's cortisol functioning (e.g., Granger et al., 1998; Lupien, Meaney, & McEwen, 2001). Thus, it is plausible that associations between interparental conflict, child cortisol reactivity, and child psychological problems may be artifacts of socioeconomic status or parenting difficulties. In light of these findings, we examine low parental warmth and socioeconomic status as potential covariates in our statistical models.

Previous research also raises the question of whether hypothesized pathways involving child cortisol reactivity may vary with inclusion of child gender or race. Although a recent literature review concluded that studies generally do not report child gender differences in cortisol reactivity to stress, the lack of systematic analyses of the role of child gender in understanding individual differences in cortisol functioning make it difficult to draw conclusions (Kajantie & Phillips, 2006). Moreover, recent conceptual and empirical work highlights the potential utility of exploring gender as a possible covariate and moderator of models of cortisol (e.g., Klimes-Dougan et al., 2001; Loney, Butler, Lima, Counts, & Eckel, 2006, McEwen, 2005). Likewise, identification of differences in adrenocortical functioning between Black and White adults raise the possibility that race may alter associations between interparental conflict, child cortisol reactivity, and child maladjustment as a covariate or moderator (Cohen et al., 2006). Therefore, as a further assessment of the generalizability of our findings, we examined whether our results on child cortisol as an intervening mechanism in models of interparental conflict and child functioning change with inclusion child race and gender as a covariates and moderators.

Method

Participants

The data for this study were drawn from a larger project focusing on linkages between family processes and child coping and psychological adjustment. The original sample of 235 kindergarten children and their families in the first wave of the study were recruited through local school districts and community centers in a moderate-sized metropolitan area in the Northeast and a small city in the Midwest (see Davies, Cummings, & Winter, 2004, for more details). Obtaining a sample of families who, on the whole, exhibited elevated and diverse levels of interparental problems and child vulnerability was a central recruitment goal of the research project. Consistent with this goal, our analyses indicate that 53% of the couples contained at least one maritally dissatisfied partner according to the cutpoint of 100 on the Short Marital Adjustment Test (Crane, Allgood, Larson, & Griffin, 1990; Locke & Wallace, 1958). Further illustrating the range of risk in our sample, proportions of children who scored above the clinical cut point ($t = 63$) on parent reports of internalizing and externalizing symptoms on the Child Behavior Checklist were comparable to or higher than norms derived from a representative sample of U.S. children (Achenbach, 1991; for more sample details, see Davies et al., 2004).

Due to differences in the start dates of the larger project and the supplemental study of child cortisol reactivity, cortisol measures were obtained for 190 of the 235 children. Additional reductions in sample size for this study resulted from incomplete data across the three cortisol assessments (n = 10) and attrition of families over the two-year course of the study (n = 2). The resulting sample consisted of 178 mothers, fathers, and children. Families who met inclusionary criteria for this study did not differ significantly from families who were excluded from the study along any of the primary variables in this study (i.e., interparental conflict, parenting, child maladjustment) or demographic characteristics (e.g., child age and gender, parent education).

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. The average number of years of education completed by mothers and fathers were 14.4 (SD = 2.36) and 14.7 (SD = 2.59), respectively. The majority of the sample identified themselves as White (77.1%), followed by smaller proportions of Black (16.4%), Hispanic (4.5%), Asian (0.8%), and Other/Mixed (1.1%) family members. A large percentage of caregivers reported being the biological parents of their children (92.0%), followed by relatively smaller percentages of stepparents (3.4%), adoptive parents (1.7%), and other types of guardianship (2.9%). The mean age of the children at Wave 1 was 6.0 years (SD = 0.48), with 56% of the sample consisting of girls (n = 99) and 44% consisting of boys (n = 79). At the first wave, mothers, fathers, and children lived in the same household for an average of 5.3 years (SD = 1.04).

Procedures

Data for this longitudinal study were collected at two measurement occasions spaced two years apart. At each wave, families visited the laboratories twice within a one-week period at one of the research sites. The laboratories 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.

Interparental interaction task—At the first visit of Wave 1, mothers and fathers participated in a marital interaction task in which they discussed to common, intense interparental disagreements that they viewed as problematic in their relationship. Following similar procedures in previous research (e,g, Du Rocher Schudlich & Cummings, 2003), each parent was asked to independently select the top three most problematic topics of disagreement in their relationship they felt comfortable discussing. Couples were provided with a list of common disagreements to use as a guide in the selection process. After this procedure, partners conferred to select one topic from each of their lists that they both felt comfortable discussing. The couples subsequently discussed each topic for ten minutes as they normally would at home while they were alone in the laboratory room. Videotaped records of the interactions were coded later for interparental conflict behaviors. Immediately following the interparental interactions, parents completed a survey designed to assess their angry reactions to the interparental conflicts.

Consistent with the use of similar interaction tasks in prior research, the aim of the interparental interaction task was to assess parents' characteristic ways of managing conflict in the interparental relationship (Du Rocher Schudlich & Cummings, 2003). To examine the validity of this assumption, mothers and fathers completed a post-interaction interview in which they individually responded to the question, "Overall, how much did the discussion

resemble disagreements that usually occur between you and your partner at home?" Response alternatives included: (1) a lot more negative, (2) somewhat more negative, (3) a little more negative, (4) about the same, (5) a little more positive, (6) somewhat more positive, and (7) a lot more positive. Supporting the comparability of the interactions to conflicts that occur in the home, the means of mother and father responses fell between "about the same" and "a little more positive" on the seven-point scale ($M = 4.76$, $SD = .90$ and $M = 4.75$, $SD = 1.08$, respectively). In summary, data from our study and prior research converge to support the assumption that the conflict procedures reflected parents' typical methods of managing conflict in the home.

Parent-child interaction task—Observational measures of maternal and paternal parenting practices were obtained through mother and father participation in a play and clean-up task with their child during the first wave of data collection. In the first laboratory visit, fathers participated in the ten-minute task with their children. Fathers were instructed to spend five minutes playing with their child. Following the five minutes, the research assistant provided a pre-arranged signal for the father to have the child clean up the toys. During the second measurement occasion, mothers participated in the same play and clean-up task with their children. Videotaped records of the interactions were subsequently coded for parenting behavior.

Cortisol reactivity during the simulated phone argument task—During the second laboratory visit of the first measurement occasion, children and their mothers participated in the Simulated Phone Argument Task (SPAT) to assess child 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. Interspersed between the free period following the conflict and the resolution was an interview with children regarding their appraisals and reactions to the conflict.

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. For the simulated resolution, the mother was asked to communicate a moderate level of understanding, caring, and warmth in her tone of voice. 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. The validity of the SPAT is supported by significant associations between children's emotional and behavioral distress to the simulated conflict and their prior experiential histories of interparental conflict and family discord and their concurrent and prospective psychological problems (e.g., Davies et al., 2006).

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 all collected in the afternoon or early evening. Average sampling time for pre-task cortisol occurred at 3:46 P.M. (*SD* = 2 hours, 5 minutes; range 12:20 P.M. to 7:20 P.M). The pre-task sample was collected prior to the simulated conflict and resolution while the mother was learning the script for the task.

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. Rather, wide variability is evident across studies in the temporal spacing of post-stressor cortisol measures (Fox, Hane, & Perez-Edgar, 2006). For example, in a recent meta-analysis of cortisol functioning in the context of acute stressors (Dickerson & Kemeny, 2004), collection of cortisol following a stressor varied between 1 and 60 minutes across studies. However, the meta-analytic findings also revealed that cortisol levels across 10-periods following the stressor were highest during the 21-30 and 31-40 minute epochs than any other 10 minute period, leading the authors to conclude that "the peak cortisol response occurs 21-40 minutes from the onset of acute psychological stressors (Dickerson & Kemeny, 2004; p. 368)." Likewise, Douglas Granger (2006; personal communication) voiced a similar conclusion in noting that differences cortisol assessments between 20 and 40 minutes are negligible in the context of individual differences in stress reactivity. Therefore, the two post-conflict saliva samples, which were obtained approximately 25 and 36 minutes after the simulated conflict ending, corresponded with the midpoints of the two 10-minute periods identified in the meta-analysis as evidencing peak levels of cortisol following the stressor.

Following conventional 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. For each cortisol assessment, children chewed Trident original flavor sugarless gum to stimulate saliva flow immediately prior to saliva collection. Children then expurgated through a plastic straw directly into a 20mL collection vial. Saliva samples were immediately stored at −36°C until it was shipped on dry ice to Salimetrics LLC (State College, PA).

Parental assessment of child psychological adjustment—At both measurement occasions, mothers and fathers independently completed questionnaires to assess their children's psychological adjustment problems and family functioning.

Measures

Interparental conflict—We utilized observations derived from the interparental interaction task and parent reports of interparental conflict in the home to obtain a multi-method latent construct of interparental conflict. For the observational component of the measurement battery, trained coders provided behavioral ratings using the Marital Daily Records (MDR) coding system. Prior research supports the validity of the MDR system in capturing interparental conflict characteristics (e.g., Du Rocher Schudlich & Cummings, 2003). Coders rated maternal and paternal anger separately for each of the two interactions along 10-point scales (0 to 9) assessing maternal and paternal anger intensity. Ratings of anger intensity were based on the analysis of maternal and paternal behaviors, verbalizations, and facial expressions during the interactions. Intraclass correlation coefficients, which indexed interrater reliability of ratings of two independent coders were .

84 and .91 for maternal and paternal anger intensity, respectively. Assessments across the interactions were summed to form more comprehensive assessments of maternal anger intensity and paternal anger intensity.

For the self-report component of the measurement battery, each partner completed the O'Leary-Porter scale (OPS; Porter & O'Leary, 1980). The OPS contains 10 items designed to assess the frequency of child exposure to various forms of interparental hostility (e.g., quarrels, sarcasm, physical abuse) on a 5-point Likert-type scale ranging from 0 (*never*) to 4 (*very often*). The scale has been previously demonstrated to have good test-retest reliability, internal consistency, and concurrent validity (Porter & O'Leary, 1980). The measure evidenced acceptable internal consistency for both mother and fathers, $\alpha = .81$ and .78, respectively. Due to the high correspondence between mother and father reports on the OPS, $r(178) = .58$, $p < .001$, maternal and paternal reports were subsequently averaged together to yield a single manifest indicator of the interparental conflict composite.

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 ul of saliva per determination, has a lower limit of sensitivity of 0.003 ug/dl, standard curve range of from 0.007 to 1.8 ug/dl, and average intra-and inter-assay coefficients of variation 5.1% and 8.2% respectively. Method accuracy, determine 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).

Child psychological maladjustment—Mothers and fathers completed the Internalizing and Externalizing scales from the Child Behavior Checklist (CBCL; Achenbach, 1991). The Internalizing scale specifically consisted of the sum of Anxiety/Depressed and Withdrawn subscales from the CBCL, whereas the Delinquency and Aggressive Behavior CBCL subscales comprised the Externalizing scale. The CBCL is a widely used, well-validated measure of child adjustment problems. Internal consistency of the CBCL scales in this sample ranged from .83 to .91. Maternal and paternal reports of internalizing and externalizing symptoms were used as indicators of child psychological maladjustment.

Covariates: Parenting behaviors—Maternal and paternal behaviors reflecting parental warmth during the parent-child interaction task were evaluated using the Warmth/Support and Positive Reinforcement scales from the Iowa Family Interaction Rating Scales (IFIRS; Melby & Conger, 2001). The IFIRS is a global rating scale that assesses the frequency, intensity and proportion of a parent's caregiving behaviors on a 9-point scale, ranging from (1) not at all characteristic to (9) mainly characteristic. In support of its validity, the IFIRS scales have been associated with indices of community risk, family relationship quality, and child functioning in theoretically meaningful ways (Ge, Best, Conger, & Simons, 1996; Melby & Conger, 2001). The Warmth code is specifically designed to assess parental communication of support, affection, and praise toward the child, whereas the Positive Reinforcement code reflects positive parental responses (e.g., praise, approval, rewards) to appropriate or exemplary child behavior. The coders, who were blind to interparental and child functioning, rated mother and father parenting behaviors separately for play and clean up components of the parent-child interaction task. To reduce shared informant variance, different primary coders were used to assess maternal and paternal parenting. Ratings across each interaction were subsequently averaged to form parsimonious, multi-indicator measures paternal and maternal Warmth/Support and Positive Reinforcement. Intraclass correlation coefficients, indexing interrater reliability of ratings of two independent coders for 22% of the parent-child interactions, ranged from .94 to .97.

Covariates: Sociodemographic Characteristics—To examine demographic characteristics as potential covariates and moderators in our model, we quantified parental reports of demographic characteristics into the following dichotomous variables: (a) gender (i.e., girls contrasted with boys), and (b) race (Black children contrasted with White children). To obtain a multi-indicator composite of socioeconomic status, mothers and fathers each reported on (a) their level of education in years, (b) total family income divided into nine categories reflecting increments from the lowest (i.e. less than $6,000) to highest (i.e., $75,000 or more) income categories, and (c) their usual occupation. Descriptions of usual occupation were subsequently coded using the Socioeconomic Index, with higher scores reflecting greater occupational prestige (SEI; Entwisle & Astone, 1994). The six indicators of SES were then standardized and summed to form a single composite of SES (α = .83).

Results

Preliminary and Descriptive Analyses

Table 1 provides the raw means, standard deviations, and correlations among the focal variables in the primary analyses. Supporting the measurement models of our structural equation models, Table 1, as a whole, revealed moderate correlations among proposed indicators of interparental conflict (mean r = .49), wave 1 child internalizing (r = .52) and externalizing symptoms (r = .58), and wave 2 child internalizing (r = .33) and externalizing (r = .45) symptoms. 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 = −2.38, SD = 0.55), post I conflict (M = −2.66, SD = 0.54), and post II conflict (M = −2.74, SD = 0.55).

To assess children's reactivity in response to the simulated interparental conflict, we examined the slope of change across the pre-conflict, post I, and post II cortisol assessments through the use of latent growth curve (LGC) modeling. Figure 1 depicts the results of the unconditional growth model, prior to the inclusion of covariates and predictors. In this analysis, we centered Time so that the intercept provides an estimate of pre-conflict levels of cortisol. The mean slope parameter characterizes the estimate of average, constant change over time in cortisol across the three assessments. To estimate the linear slope, weights of each manifest assessment of cortisol in the model were specified to correspond with the time elapsed since the pre-conflict cortisol measure. Thus, the weight of .36 was assigned to the post I assessment because it occurred approximately 36 minutes after the pre-conflict assessment, whereas the weight of .47 for the post II conflict measure indicates that the assessment took place approximately 47 minutes after the baseline.

The model fit the data well, χ^2 (1, N = 178) = 0.01, p = .95, $RMSEA$ = .000, CFI = 1.00 and χ^2/df ratio = 0.01. The mean level of the intercept was significantly different from 0, (-2.38, z = 57.81). The average slope was also significantly different from 0 (-0.76, z = 9.93). Consistent with previous research on child cortisol reactivity to stressful family events (e.g., Granger et al., 1996; 1998), the direction of the slope estimate indicated that cortisol levels, on average, decreased across sampling occasions. In addition, the intercept and slope factors had statistically significant variances of 0.29 (z = 5.03) and 0.84 (z = 2.84) respectively, indicating that there were significant individual differences in children's pre-conflict cortisol levels and rate of change in cortisol across the three sampling occasions. The covariance between the intercept and slope factor was not significant (-0.22, z = −1.84, p = .07).

Given the diurnal rhythm of cortisol, our next step in preliminary analyses was to introduce time of day of cortisol measurement as a covariate in the unconditional LGC model. Including time of measurement at baseline as a covariate for each of three cortisol measures

yielded a good fit with the data, χ^2 (1, N = 178) = 1.12, p = .29, *RMSEA* = .026, *CFI* = 1.00 and χ^2/df ratio = 1.12. Time of day was significantly associated with pre-conflict (β = −.31, p = .001), post I conflict (β = −.46, p = .001), and post II conflict (β = −.49, p = .001) cortisol assessments. After controlling for time of day, the mean level of the intercept was still significantly different from 0, (-1.097, z = −3.65). Conversely, after taking into account time of day, the average slope changed direction from negative to positive, but the slope was not significant (0.88, z = 1.55). However, examination of the variances for the intercept and slope factors still yielded significant individual differences in the initial levels of cortisol (0.24, z = 4.62) and the slope of cortisol (0.69, z = 2.47). Therefore, given our aim of explicating the role of individual differences in cortisol reactivity to conflict as an intermediary mechanism, we proceeded to examine the growth model of cortisol reactivity in the context of larger structural equation model involving interparental conflict and child maladjustment.

Primary Analyses

In the first step of testing our model, we sought to determine whether interrelations between interparental conflict and child symptoms supported tests of cortisol as a mediating or intervening mechanism. Mediational tests require that a significant association exist between the predictor and outcome, whereas tests of intervening or indirect mechanisms do not require that the proposed predictor be associated with the outcome (Grych et al., 2003; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). To examine whether the data supported tests of mediational or indirect pathways, structural equation models were conducted in which the latent construct of interparental conflict was specified as a predictor of the Time 2 latent construct of child maladjustment after controlling for Time 1 child maladjustment. Paths between interparental conflict and the intercept and slope parameters of cortisol were also estimated. Paths between the slope and intercept factors of cortisol and child maladjustment were constrained to 0 to obtain estimates of the direct paths between interparental conflict and child maladjustment. Separate models were conducted for child internalizing and externalizing symptoms due to high ratio between number of parameters estimated in the models and the sample size. No significant associations were found between interparental conflict and subsequent changes in child internalizing (β = .05) or externalizing (β = .05) symptoms.

Accordingly, the second step of our analyses tested the role of cortisol as an intervening mechanism by examining whether interparental conflict was indirectly related to greater child internalizing and externalizing symptoms through its association with child cortisol reactivity. Figure 1 shows the results of the model in predicting externalizing symptoms. The overall model fit provided an acceptable representation of the data, χ^2 (36, N = 178) = 36.70, p =.44, χ^2/df ratio = 1.02, *RMSEA* = .010, *CFI* = 1.00. All specified indicators in the measurement model loaded significantly (all ps < .001) onto their respective latent constructs. Supporting the role of cortisol reactivity as an intervening mechanism, the results indicated that interparental conflict was associated with decreasing slopes in child cortisol reactivity to conflict, β = −.20, p < .05. Decreasing cortisol reactivity, in turn, predicted increases in child externalizing symptoms over a two year period, β = −.22, p < .05. Follow up tests using procedures for examining indirect pathways outlined by MacKinnon et al. (2002) indicated that cortisol was a significant intervening mechanism in the path between interparental conflict and children's externalizing symptoms, z' = 1.50, p < .05.

The model predicting child internalizing symptoms, which is depicted in Figure 2, also yielded a good representation of the data, χ^2 (36, N = 178) = 40.86, p =.27, χ^2/df ratio = 1.14, *RMSEA* = .028, *CFI* = .99. Consistent with the results for the externalizing symptoms model, all specified indicators in the measurement model loaded significantly (all ps < .001) onto their respective latent constructs. Likewise interparental conflict was significantly

associated with significant declines in the cortisol slope in response to conflict, $\beta = -.19$, $p < .05$. However, in contrast to the externalizing model, the slope of cortisol was not associated with changes in internalizing symptoms over the two year period, $\beta = .01$, *ns*.

To test the generalizability of the pathways in our externalizing and internalizing models, we conducted three additional sets of analyses. First, we examined whether child race, child gender, parental warmth, family socioeconomic status, or research site (i.e., NY and Indiana) might serve as confounding variables or covariates that might alter the associations between interparental conflict, cortisol reactivity, and child externalizing symptoms as covariates. Specifying paths between each of these covariates and child cortisol reactivity and child externalizing symptoms in a successive series of structural equation models failed to alter the significant indirect pathways in our SEM, thereby supporting the stability of our results.

Second, structural paths in Figures 1 and 2 could vary significantly as a functioning child gender, race, and research site. Therefore, to test the moderating role of each these characteristics, we conducted multiple group comparisons in which the data were split according to the dichotomous structure of each of the proposed moderators (e.g., boys and girls within the gender variable). Separate multigroup analyses were conducted for each of the putative moderators. Given the small sample sizes resulting from splitting the data, it was necessary to conduct multiple group path analysis using manifest, rather than latent, composites for our measures of interparental conflict and child externalizing and internalizing symptoms. Manifest variables of these primary constructs were constructed by first standardizing their respective manifest indicators and subsequently summing them to create composites. Each multiple group comparison for the structural paths in Figures 1 and 2 consisted of comparing a model in which all parameters were allowed to vary freely with a model in which comparable paths across the relevant groups (i.e., boys and girls; Black and White children; NY and Indiana research sites) were constrained to equality. Comparisons of the fully constrained and free-to-vary models revealed no difference in fit for any of the group comparisons, thereby indicating that paths in Figure 1 and 2 did not differ as a function child gender, child race, or research site.

Third, it is also possible that prescription and over-the-counter medications may also alter findings in Figures 1 and 2 by significantly affecting cortisol concentrations. Thus, we classified children into medication (i.e., over-the-counter and prescription medication; $n = 26$) and medication-free groups based on parental interview data regarding child use of specific medications. When included as a covariate in the models, the medication variable failed to predict either of the cortisol variables or child maladjustment. Nor did the inclusion of the variable change the findings depicted in Figures 1 and 2. Further tests of medication use as a moderator using multiple group comparisons indicated that the paths in Figure 1 and 2 did not differ as a function of child medication use. Finally, the pattern of findings did not change when the four children who were taking steroid medications were excluded from the analyses. In conclusion, the indirect paths among interparental conflict, child cortisol reactivity, and child externalizing symptoms were robust across a variety of contextual conditions, including research site, child gender, child race, parental warmth, socioeconomic status, and child medication use.

Discussion

Although some progress has been made in understanding children's physiological functioning in the face of interparental conflict (El-Sheikh et al., 2001; Katz, 2001), the predominant focus on child cardiovascular functioning in models of parental conflict has resulted in the relative neglect of other physiological systems that may play a central role in

the regulation of stress (Davies & Cummings, 2006). In addressing this gap, our study examines the role of child LHPA axis functioning in associations between interparental conflict and child psychological maladjustment. Drawing from biopsychosocial models, we specifically examined the viability of two alternative versions of the allostatic load hypothesis. In accordance with the hypocortisolism hypothesis, we tested the prediction that interparental conflict results in *diminished* children's cortisol reactivity to interparental conflict which, in turn, predicts greater child psychological problems over time. Guided by the hypercortisolism hypothesis, we also examined the alternative prediction that interparental conflict is linked with child adjustment problems through its association with *elevated* cortisol reactivity to conflict.

Our results supported the hypocortisolism hypothesis over the hypercortisolism hypothesis. In addressing the first link in our hypothesized indirect pathway, interparental conflict was specifically associated with lower child cortisol reactivity to interparental conflict within the LGC analyses. Thus, these findings suggest that the response of the LHPA axis to stress may become progressively dampened in high conflict homes. Results from analyses of the second link in our hypothesized indirect pathway model varied across the form of psychological maladjustment. Whereas diminished cortisol reactivity to conflict predicted subsequent increases in child externalizing symptoms over a two year period, it was not associated with change in children's internalizing symptoms within that same time period.

Consistent with challenge and resilience models in psychology (Garmezy, Masten, & Tellegen, 1984; Masten, 2001; Rutter, 1985) and psychophysiology (Deinstbier, 1989), one possible explanation for our findings is that exposure to family adversity in manageable doses may actually have steeling effects that serve to enhance coping and inoculate or toughen individuals in the face of subsequent stress. Gunnar and Vazquez (2001) suggest that such a model may explain why greater exposure to stress can result in diminished adrenocortical reactivity. However, questions can be raised about the viability of this explanation. If diminished cortisol reactivity signifies successful adaptation as the challenge model suggests, then it follows that dampened sensitivity of the adrenocortical system should be a prognosticator of better outcomes for at least some domains of functioning. Although authoritative tests of this hypothesis will require assessments of a broader array of health outcomes, the plausibility of this model is challenged by our results indicating that diminished cortisol reactivity was actually associated with increases in child externalizing symptoms and prior empirical support on the psychological and physiological toll of diminished cortisol reactivity (Gunnar & Vasquez, 2001).

In accordance with psychological frameworks, a more plausible explanation is that hypocortisolism may signify some normative form of dissociation or inhibition of the psychological experience of threat. Blunting of psychological experiences and its corresponding reduction in cortisol reactivity may provide a temporary means of attaining a perceived sense of security or control in stressful contexts (Gunnar & Vazquez, 2001). This interpretation is more broadly consistent with the emotional security theory and its assumption that children's affective, social-cognitive, behavioral, and physiological systems reciprocally influence each other toward the goal of preserving security in high conflict homes (Davies & Forman, 2002). In this model, children's effortful attempts to suppress or amplify responses in one system (e.g., subjective experiences of fear and threat; overt expressions of distress) may temporarily facilitate the attainment of security goals and, in the process, trigger decreases in the reactivity of other stress-responsive systems such as the LHPA-axis (Davies & Forman, 2002; Gunnar & Vasquez, 2001; Lopez et al., 2004). The observation that diminished cortisol reactivity predicted subsequent increases in psychological problems lends further support to both the psychological and biological

interpretations that low levels of cortisol production in response to stress may confer some psychological risks (Cicchetti & Rogosch, 2001a; Gunnar & Vasquez, 2001).

Psychobiological models provide another level of interpretation for our findings. As one of the prevailing neurobiological explanations of hypocortisolism, the attenuation hypothesis specifically outlines multiple neurobiological pathways by which dampened cortisol activity resulting from discordant socialization contexts may increase children's vulnerability to externalizing symptoms (Susman, 2006). For example, unpredictability and volatility in discordant homes may dampen physiological and biological responses to threatening events by disrupting the capacity of the limbic system (particularly the amygdala) to process and acquire information on the consequences of emotional events in the family. Therefore, as one form of family discord, prolonged exposure to interparental conflict may undermine fear conditioning in the limbic system that serves to mediate the activation of the LHPA axis. Difficulties in neurobiological processing of and responding to the emotional and fear-relevant parameters of events may engender aggressogenic characteristics such as fearlessness, sensation seeking, and callousness. Adaptation, or the tendency of systems to maintain an internal state of equilibrium, is another mechanism proposed in the attenuation model to explain the genesis of hypocortisolism in the attenuation model. In particular, inhibition of LHPA axis or some of its specific components may reflect the activation of processes designed to prevent chronic overarousal of the stress system in response to danger (Susman, 2006). However, a remaining challenge is to identify the specific mechanisms underlying diminished cortisol reactivity in children exposed to destructive interparental conflict. Research on neurobiological functioning following early traumatic experiences (e.g., abuse) may provide a useful guide in identifying the sources of down-regulation in the LHPA axis (e.g., Heim, Meinischmidt, & Nemeroff, 2003; Heim, Newport, Bonsall, Miller, & Nemeroff, 2001)

Further analysis of the nature of the indirect pathways among interparental conflict, child cortisol reactivity, and child psychological adjustment indicated that child cortisol served as an intervening, rather than mediating, mechanism in models of interparental hostility. Evidence for mediational pathway requires demonstrating that the proposed mediator accounts for a substantial part of the variance in a significant association between the predictor and outcome. However, consistent with results from other longitudinal designs and the existence of considerable heterogeneity in the functioning of children from high conflict homes (e.g., Davies et al., 2002; Grych et al., 2003; Harold, Fincham, Osborne, & Conger, 1997), associations between interparental conflict and changes in child maladjustment were negligible in our study. Accordingly, child cortisol reactivity failed to meet conditions necessary for supporting mediation. Rather, in reflecting another type of indirect effect, the findings indicated that diminished cortisol reactivity served as an intervening mechanism linking interparental conflict to subsequent changes in child maladjustment (MacKinnon et al., 2002). However, identifying interparental conflict as a distal risk factor in the prediction of child psychopathology does not relegate it to a secondary conceptual status. Rather than falling into the reductionist trap of regarding the "the smallest and most proximal event as the ultimate cause (Emery, Fincham, & Cummings, 1992, p. 910)" of child psychopathology, interparental conflict still regarded as playing an integral role as the ultimate cause of unfolding pathogenic processes.

In further supporting the conceptual significance of our findings, documentation of cortisol reactivity as an intervening mechanism is consistent with the risky family framework proffered by Repetti and colleagues (2002). The model specifically postulates that forms of family adversity create disruptions in children's biological systems responses to stress which, in turn, engender poor mental health outcomes in an unfolding cascade of pathogenic processes. In bolstering the role of interparental conflict in the allostatic load model, our

findings indicated that interparental hostility was indirectly associated with increases in externalizing symptoms through its association with children's diminished cortisol reactivity to interparental conflict. Moreover, these paths remained even after inclusion of demographic (i.e., child race and gender, family SES), family (i.e., parental warmth), child (i.e., basal cortisol, child medication use), and methodological (i.e., research site) characteristics as covariates and moderators in the analyses.

However, intriguing questions still remain regarding the pathways between interparental conflict, child cortisol reactivity, and child maladjustment. First, provided our findings are replicated, a next step will be to address the question of how or why children from homes characterized by interparental hostility exhibit lower cortisol reactivity to subsequent interparental conflict. For example, consistent with our psychological explanation for hypocortisolism, it is possible that diminished cortisol reactivity may reflect suppression of children's attempts to suppress subjective experiences of distress and threat (Davies & Forman, 2002; Lopez et al., 2004). Alternatively, allostatic adjustments may be designed to counter what, initially, were elevated levels of cortisol production in response to the stressfulness of exposure to interparental hostility (Gunnar & Vasquez, 2001; McEwen, 1998). Second, in addressing the second link in the chain of processes, another direction will be to further clarify the ill-defined parameters and properties for distinguishing adaptive and maladaptive patterns of low cortisol. Finally, little is known about the underlying mechanisms that explain why individual differences in patterns of cortisol reactivity are associated with child psychological problems. For example, specific patterns of cortisol activity may increase vulnerability to psychopathology by disrupting the efficiency of synaptic pruning, inhibiting neuronal myelination, and damaging the hippocampus (Cicchetti & Rogosch, 2001b) or they may signify children's difficulties in efficiently marshalling physical and psychological resources necessary to successfully resolve stage-salient tasks (e.g., adjustment to school, development of close friendships), with psychological maladjustment being the consequence (Davies & Cummings, 2006).

The results of this study must also be interpreted in the context of methodological limitations. First, although the results of the indirect pathway involving child cortisol reactivity were stable in the context of an array of sociodemographic variables, relationships documented in our community sample of predominantly White families may not necessarily generalize to families with other racial or ethnic backgrounds or families who are facing considerable adversity. Similarly, conclusions about causality are constrained by the limitations of our longitudinal design and the concurrent assessment of interparental conflict and child cortisol reactivity. Thus, although we interpreted the findings as suggesting that interparental hostility indirectly affects child adjustment by disrupting child adrenocortical reactivity to interparental conflict, it is still plausible that the results reflect child effects on the marriage or more complex bidirectional processes. In addition, as is common with multi-method, prospective designs in the family conflict literature (Cummings et al., 2000), indirect pathways among interparental conflict, cortisol reactivity, and child maladjustment were relatively modest in magnitude.

Second, our analysis of child cortisol reactivity occurred in isolation from other neurobiological (e.g., dehydroepiandrosterone and its sulfated ester) and behavioral (e.g., distress) indices of functioning. Therefore, evaluating cortisol reactivity within the broader organization of multiple child response domains may be a central strategy for understanding the developmental meaning of individual differences in LHPA functioning (Granger & Kivlighan, 2003). Moreover, although this study tested predictions that were partially rooted in the allostatic load hypothesis, comprehensive tests of the allostatic load hypothesis will require a wider analysis of different physiological measures.

Third, our latent growth curve analysis of cortisol reactivity holds many advantages over alternative methodologies, but 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 (e.g., Gump & Matthews, 1999; Matthews, Gump, & Owens, 2001). For example, despite the fact that the timing of the post-conflict cortisol assessments corresponded with research on the peak periods of cortisol secretion following stressors (Dickerson & Kemeny, 2004), the trajectories of child cortisol levels following specific interpersonal challenges have yet be thoroughly documented. Likewise, although the simulated resolution following the conflict is ethically necessary to alleviate any residual child distress, it obscures our ability to precisely determine whether dampened cortisol reactivity (particularly for the post-conflict II cortisol assessment) was the product of exposure to the conflict, resolution, or both. The excellent fit of the declining linear slope of the cortisol trajectory across the three sampling occasions in the unconditional LGC analyses does increase our confidence that the dampened cortisol reactivity is not simply an artifact of exposure to a positive interparental resolution. However, increasing the number of cortisol samples within the ill-defined boundaries of the cortisol reactivity and recovery periods in future research are necessary to fully address this limitation.

Despite the limitations, this multi-method, prospective study constitutes the first empirical attempt to identify pathways between interparental conflict, child cortisol reactivity to interparental difficulties, and child adjustment problems. Consistent with the allostatic load hypothesis, interparental conflict was indirectly related to increases in child psychological maladjustment over a two-year period through its association with children's diminished cortisol reactivity to interparental conflict.

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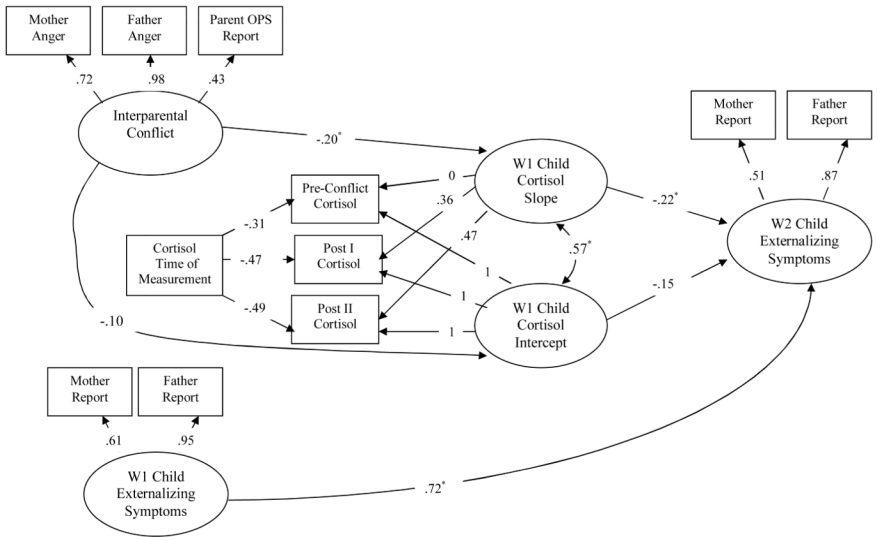

Figure 1.

A structural equation model testing child cortisol reactivity as an intervening mechanism in associations between interparental conflict dimensions and child externalizing symptoms.

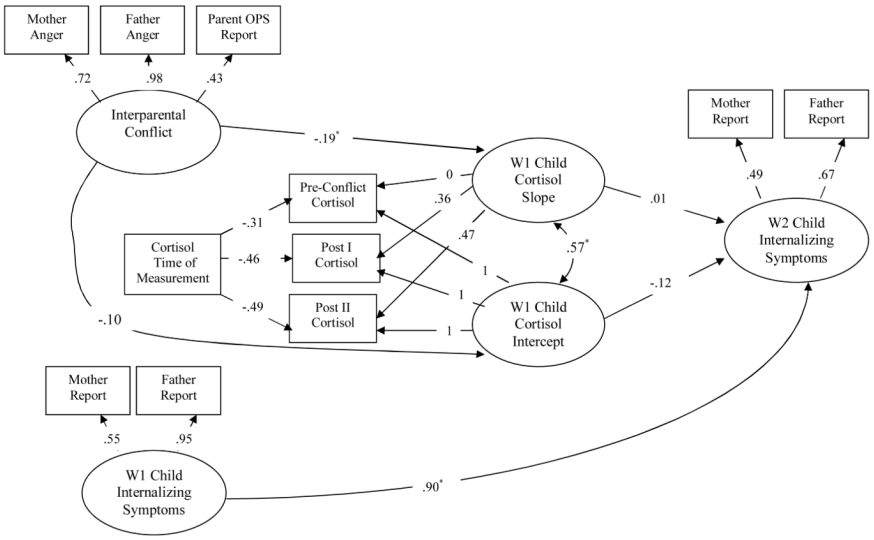

Figure 2.

A structural equation model testing child cortisol reactivity as an intervening mechanism in associations between interparental conflict dimensions and child internalizing symptoms.

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	9	10	11	12	13
Interparental Conflict															
1. Maternal Hostile Behavior	5.18	3.48	--												
2. Husband Host. Behavior	4.30	3.42	.71	--											
3. Parent O'Leary Porter	11.97	4.81	.35	.42	--										
Child Cortisol Reactivity															
4. Pre-Conflict Cortisol (μg/dl)	0.11	0.06	−.05	−.10	.02	--									
5. Post I Cortisol (μg/dl)	0.08	0.05	−.12	−.18	.05	.64	--								
6. Post II Cortisol (μg/dl)	0.08	0.04	−.15	−.20	.00	.62	.84	--							
Wave 1 Child Maladjustment															
7. Internalizing (M)	5.22	4.77	−.04	−.01	.12	−.12	−.07	−.12	--						
8. Externalizing (M)	9.42	7.11	−.09	−.02	.14	−.19	−.07	−.06	.56	--					
9. Internalizing (F)	5.94	5.56	−.04	.01	.08	−.17	−.12	−.16	.52	.30	--				
10. Externalizing (F)	11.03	8.28	−.08	.01	.10	−.17	−.09	−.07	.30	.58	.60	--			
Wave 2 Child Maladjustment															
11. Internalizing (M)	5.97	5.26	−.09	−.07	.07	−.08	−.03	−.02	.66	.40	.43	.21	--		
12. Externalizing (M)	8.40	5.86	−.04	.01	.12	−.16	−.07	−.06	.34	.65	.23	.36	.59	--	
13. Internalizing (F)	5.84	5.22	.02	.12	.17	−.20	−.16	−.19	.34	.27	.58	.41	.33	.26	--
14. Externalizing (F)	9.21	6.31	−.01	.05	.06	−.14	−.15	−.14	.12	.39	.40	.59	.15	.45	.66

Note. Correlations ≥ .15 are significant at $p \leq .05$. (M) and (F) denote mother and father survey reports, respectively.