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Children's Patterns of Emotional Reactivity to Conflict as Explanatory Mechanisms in Links Between Interpartner Aggression and Child Physiological Functioning

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Abstract

Background—This paper examined children's fearful, sad, and angry reactivity to interparental conflict as mediators of associations between their exposure to interparental aggression and physiological functioning.

Methods—Participants included 200 toddlers and their mothers. Assessments of interparental aggression and children's emotional reactivity were derived from maternal surveys and a semi-structured interview. Cortisol levels and cardiac indices of sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) activity were used to assess toddler physiological functioning.

Results—Results indicated that toddler exposure to interparental aggression was associated with greater cortisol levels and PNS activity and diminished SNS activity. Toddler angry emotional reactivity mediated associations between interparental aggression and cortisol and PNS functioning. Fearful emotional reactivity was a mediator of the link between interparental aggression and SNS functioning.

Conclusions—The results are interpreted within conceptualizations of how exposure and reactivity to family risk organizing individual differences in physiological functioning.

Interparental aggression is a significant public health concern in the lives of young children. Children exposed to hostility between parents are vulnerable to an array of psychological and physical health problems (Grych & Fincham, 2001). Consistent with the theme of this special issue, emotional security theory (EST) regards emotion regulation processes as central explanatory mechanisms in pathways between interparental conflict and child adjustment (Davies & Sturge-Apple, 2007). At another level of analysis, neurobiological models share the assumption that family risk factors, including interparental aggression, undermine the general functioning of multiple physiological systems early in life (Heim,

Meinischmidt, & Nemeroff, 2003; Repetti, Taylor, & Seeman, 2002). In integrating these levels of analysis, EST postulates that children's patterns of emotional regulation to interparental conflict initially develop from early experiences with aggression between parents and, in turn, set the stage for changes in the homeostatic functioning of stress-responsive biological systems (Davies, Winter, & Cicchetti, 2006). Accordingly, this paper examines children's patterns of emotional reactivity as intervening mechanisms in associations between their exposure to interparental aggression and the homeostatic set points of multiple physiological indices in a sample of toddlers experiencing disproportionate levels of domestic violence.

Many prevailing conceptualizations highlight emotional responding to family stress as explanatory mechanisms in links between family discord and indices of physiological functioning (e.g., Luecken, Applehans, Kraft, & Brown, 2006; Repetti et al., 2002; Troxel & Matthews, 2004). Consistent with these frameworks, EST postulates that emotional reactivity to interparental conflict organize and reflect physiological, perceptual-cognitive, and behavioral action tendencies that, over time, alter the operating range of stress-responsive physiological systems (Davies et al., 2006; Davies & Sturge-Apple, 2007). Thus, through the process of allostasis, it is plausible to hypothesize that emotional distress resulting from experiences with interparental aggression are associated with physiological responses that are designed to generate resources necessary to protect individuals from environmental stress. However, successive cycles of allostasis engendered by emotional responses to pernicious interpersonal contexts may, over time, broaden and deepen into changes in the overall homeostatic functioning of physiological systems (Repetti et al., 2002; Susman, 2006). Thus, in accordance with the concept of allostatic load, EST proposes that emotional dysregulation may result in physiological perturbations and accompanying problems maintaining homeostasis in several biological systems. A derivative hypothesis is that children's emotional responses to threatening family events serve as risk mechanisms that account, in part, for why early life experiences with interparental aggression may be associated with subsequent individual differences in general physiological activity.

In documenting the risk posed by interparental aggression, EST highlights the value of distinguishing fearful, sad, and angry forms of conflict reactivity as mediators of associations between interparental conflict and child functioning. EST maintains that relationship difficulties between adult caregivers increase children's exposure to threat and activate their social defense system which serves to minimize threat within family and social networks (Davies & Sturge-Apple, 2007). Within EST, children's experiential histories of interparental hostility result in the development of a flexible repertoire of action tendencies serving the defense module which are manifested in dispositions to exhibit specific patterns of fearful, dysphoric, and hostile responses. In turn, specific emotion patterns are presumed to reflect underlying individual differences in physiological responses that cumulatively exert impact on the general operation of stress-responsive, physiological systems. As an initial foray into identifying the emotional and physiological processes associated with exposure to domestic violence, the primary aim of this paper was to examine young children's fearful, sad, and angry reactivity to interparental conflict as mediating mechanisms in links between their exposure to interparental aggression and physiological indices of autonomic nervous system (SNS & PNS) and hypothalamic-pituitary-adrenocortical (HPA) activity.

The selection of specific physiological indices for this study was guided by evidence that these axes are keenly sensitive to family risk factors. The SNS reflects the first wave of autonomic responding to stressful events that helps prime the body for fight-flight responses by increasing cardiac output, oxygen flow, and blood glucose levels (Repetti et al., 2002). In contrast, the PNS has an inhibitory effect on the SNS in shifting resources from fight-flight

responding to restorative, homeostatic functions. Therefore, adaptation to stressful contexts requires a delicate balance in the operation of both the SNS and PNS, as damping down the PNS system in response to adverse events allows individuals to attend to and cope with stressors without having to tax resources through excessive recruitment of the SNS system (Quigley & Stifter, 2006).

Through its role as a second wave of autonomic responding to stress (Gunnar & Vazquez, 2006), the HPA axis is regarded as a central system for advancing an understanding of children's adaptation to interparental conflict (Saltzman, Holden, & Holahan, 2005). In response to stressful events, increases in cortisol serve to mobilize energy (e.g., glucose, oxygen), increase cardiovascular activity, and modulate the processing, learning, and memory consolidation of emotionally significant events (Gunnar & Vazquez, 2006). In accordance with central role the HPA axis plays in allocating resources necessary to process and react to stressful events, children's basal cortisol functioning may provide a particularly informative index for understanding how children adapt to interparental aggression.

Our decision to examine the affective and physiological correlates of interparental aggression during toddlerhood was based on several developmental considerations. First, early childhood is regarded as a significant period of neurobiological plasticity within the HPA, PNS, and SNS systems (Gunnar & Quevedo, 2006; Susman, 2006). Second, emotional reactivity to conflict may play a particularly pronounced role as a mediator in pathways between interparental aggression and physiological functioning during the toddler period. As a central developmental task during toddlerhood, regulating emotion is challenging even in normative socialization contexts, but may be especially difficult for children who must also contend with emotion-laden bouts of aggression between parents (Davies et al., 2006). Third, because young children's resulting susceptibility to distress is theorized to be largely rooted in affective structures that are outside of the child's conscious processing or verbal articulation, they may leave lasting imprints on physiological systems that are designed to defend against threat (Davies & Sturge-Apple, 2007).

The proposed meditational model is further examined in relation to two alternative mechanisms. First, because low socioeconomic status (SES) has been associated with emotion regulation difficulties and alterations in the set points of physiological systems (Lupien et al., 2006), a plausible alternative to our conceptualization is that exposure to interparental aggression is only spuriously related to children's emotional reactivity to conflict and their physiological functioning by virtue of its association with low SES. Second, neurogenetic risk models postulate that underlying temperamental vulnerabilities of children from high conflict homes may explain why they experience greater emotional reactivity to conflict and differences in their general physiological functioning (Harden et al., 2007). Thus, SES and difficult temperament are specified as covariates in the analysis of pathways among interparental violence, children's emotional reactivity to conflict, and their physiological functioning.

Moreover, the sparse, complex body of prior research on young children does not provide a sufficient base for specific hypotheses about the nature of associations between exposure and reactivity to interparental aggression and physiological functioning. Empirical attempts to identify the physiological underpinnings of toddler negative affect are rare (e.g., SNS indices; Quigley & Stifter, 2006) or inconclusive (i.e., cortisol, cardiac PNS; Beauchaine, 2001; Gunnar & Vazquez, 2006). Thus, although emotional reactivity to conflict is hypothesized to mediate links between interparental aggression and physiological functioning, no specific hypotheses are offered regarding the specificity or nature of associations in these pathways.

Method

Participants

Participants included 200 two-year-old children and their mothers in a moderately-sized metropolitan area in the Northeast. A two-step recruitment process was implemented to maximize individual differences in the experience of interparental aggression while minimizing heterogeneity in sociodemographic adversity. In the first step, we recruited participants through agencies serving disadvantaged children and families, including Women, Infants, and Children, Temporary Assistance to Needy Families rosters from the Department of Human and Health Services, and the county family court system. In the second step, we administered the abbreviated version of the Physical Assault Scale of the Conflict Tactics Scale 2 (CTS2; Straus et al., 1996) to insure that roughly equal proportions of participating mothers experienced (a) no violence, (b) mild/moderate physical violence, and (c) severe physical violence in the interpartner relationship during the last year. Additional inclusionary consisted of: (a) the female caregiver is the biological mother; (b) the child participant is 27-months old (± 5 months) and has no serious developmental disabilities, and (c) the male partner had regular contact with the mother and toddler over the past year.

Median annual income for the family household was \$18,300 (US) per year and a substantial minority of mothers (30%) and their partners (24%) did not complete high school. The mean age of the children was 26 months ($SD = 1.69$), with 44% of the sample consisting of girls ($n = 92$). The majority of mothers and children were Black (56%), followed by smaller proportions of family members who identified as White (23%), Latino (11%), Multi-Racial (7%), and “Other” (3%).

Procedures

Mothers and their toddlers made three visits to our laboratory within a one- to two-week time period to obtain the primary measures. The research procedures were approved by the Institutional Review Board at the research site prior to conducting the study.

Interparental Disagreement Interview and Questionnaires—Mothers completed survey assessments of interparental aggression and participated in the Interparental Disagreement Interview (IDI) in a separate room from the child. Adapted from an established assessment of interparental conflict processes (Crockenberg & Langrock, 2001), the IDI is a semi-structured, maternal interview designed to obtain maternal narratives of the quality and course of common interparental conflicts and children’s affective responses to the conflicts.

Salivary cortisol collection—Basal cortisol measures were obtained through saliva samples collected from each child in the presence of the mother upon arrival to the laboratory on two of the three laboratory visits. To limit the effects of diurnal patterns, samples were collected within a narrow time window in the morning ($M = 9:37$ A.M.; $SD = 30$ minutes). To further reduce extraneous variability in cortisol, saliva samples were collected after toddlers were awake for at least one hour and refrained from eating or drinking for at least 30 minutes. After holding a sorbette under the child’s tongue for one minute to obtain a sufficient quantity of saliva, a research assistant immediately stored the sorbette in a 2 mL cryovial at -80°C until it was shipped on dry ice to Salimetrics, LLC. (State College, PA).

Cardiac autonomic functioning—Basal heart rate data were collected on each child at the beginning of the second laboratory visit using a MiniLogger 2010 Series (Mini Mitter,

Inc., Bend, OR). The MiniLogger detects each R-Wave and records every interval between successive R-Waves to the nearest millisecond. Mothers held their children on their laps while a trained experimenter placed two electrodes on the child's chest; one medially on the right collar bone and one on the child's left side below the rib cage. Consistent with prior research, children were given low arousal activities during recording to keep them calm and facilitate baseline data collection (Hastings et al., 2008).

Measures

Interparental aggression—Four measures were administered to mothers to provide a multi-method assessment of interparental aggression over the past year. The first two indices of aggression were derived from maternal reports on the Physical Assault and Injury Subscales of the Revised Conflict Tactics Scale (CTS2; Straus et al., 1996). Following scoring guidelines, prevalence scores were calculated for each of the two scales based on the sum of whether (1 = one or more occurrences) or not (0 = no occurrence) the specific act in each of the items occurred in the past year. Internal consistency for the Physical Assault ($\alpha = .90$) and Injury ($\alpha = .83$) Subscales were satisfactory. As the third measure, mothers completed the Physical Aggression Subscale of the Conflict and Problem-Solving Scales (CPS; Kerig, 1996). Reliability of the CPS Physical Aggression was satisfactory ($\alpha = .92$) and prior research supports its validity (Kerig, 1996).

As the final indicator of interparental hostility, maternal narratives in response to IDI questions about the frequency and quality of interparental conflicts were coded along a seven-point Aggression scale for mothers and their partners separately. To establish reliability, two coders independently coded 26% of the interviews. Because indices of maternal and partner aggression were highly correlated ($r = .54$, $p < .001$), the measures were summed to form a single, parsimonious index of IDI aggression. Interrater agreement for ratings of IDI aggression index was satisfactory, $ICC = .85$.

Child emotional reactivity to conflict—Another pair of judges independently coded the maternal narratives on the IDI to obtain three indicators of the angry, fearful, and sad forms of emotional reactivity. First, mothers provided their own descriptions of how their children felt during and following the conflict along with intensity ratings for each descriptor. Judges subsequently coded the descriptors and intensity ratings to obtain indices of children's intensity of sadness, fear, and anger along six point scales (0 = none; 5 = a whole lot) during and immediately following the conflict. Mean intensity ratings across both the conflict and post-conflict periods were averaged to obtain indices of fear, sadness, and anger. Intraclass correlation coefficients, reflecting agreement between two independent raters on 25% of the interviews, ranged from .87 to 1.0 for maternal *intensity* ratings of child fearful, sad, and angry reactivity.

Second, judges coded for the presence (1) and absence (0) of child fearful, sad, or angry behaviors based on maternal narrative accounts of children's behaviors both (a) during and (b) following the disagreements. Ratings were aggregated across the conflict and post conflict periods to generate measures of fearful, sad, and angry *behavior* (0 = not present; 1 = present in one period; 2 = present in both periods). Kappa coefficients indexing interrater agreement ranged from .92 to 1.0.

Third, coders provided molar ratings of children's fearful distress, sadness, and hostility along seven-point scales based on the quality and organization of the children's behavior during and following conflicts. Narratives containing no indications of the specific form of emotionality were coded as "0", whereas "6" on the molar scale reflected that children were exhibiting dysregulated patterns of reactivity. Coder ratings were summed across the conflict and post-conflict periods to obtain overall *molar* assessments of fearful distress,

sadness, and hostility in response to interparental conflicts. Intraclass correlation coefficients, reflecting agreement between the two raters on 25% of the interviews, ranged from .93 to .96.

The validity of IDI indices of emotional distress is supported by its moderate associations with child and parent reports of child emotional reactivity to conflict on established questionnaires in a large sample of families with elementary school children (Davies & Cummings, 2004).

Cortisol—All samples were assayed for salivary cortisol in duplicate using a highly sensitive enzyme immunoassay (Salimetrics, PA). The test uses 25 µl of saliva per determination, has a lower limit of sensitivity of 0.003 µg/dl, standard curve range from 0.012 to 3.0 µg/dl, and average intra- and inter-assay coefficients of variation 3.5 % and 5.1 % respectively. Method accuracy, determined by spike and recovery, and linearity, determined by serial dilution are, 100.8 % and 91.7 %. Values from matched serum and saliva samples show the expected strong linear relationship, $r(63) = 0.89$, $p < 0.0001$ (Salimetrics, 2005). Cortisol variables were calculated based on a common residualized scoring technique that controls for sample collection time (Granger et al., 1994). A cortisol score was created by averaging the log-transformed scores of the two collection samples.

PNS and SNS Cardiac Functioning—Interbeat interval (IBI) data were extracted from the EKG assessment using Mini-Log 2000W software (Mini Mitter, Inc., 2001), edited for artifacts, and analyzed using CMet software (Allen, 2002). Measures of PNS and SNS activity were calculated using a method developed by Toichi et al. (1997). Calculations are based on a Lorenz plot of the IBI data (Allen et al, 2007). In the resulting ellipsoidal graph, the transverse axis (T) reflects variability from one IBI to the next while the longitudinal axis (L) reflects the range of variability in all of the IBIs. The cardiac sympathetic index (CSI) is calculated as the ratio L/T , while the cardiac vagal index (CVI) is measured as the $\log_{10}(L \times T)$. Toichi et al. found CSI is unaffected by parasympathetic blockade due to atropine, but decreases with administration of propranolol indicating that it is a measure of sympathetic nervous system activity. Conversely, CVI is subject to action by atropine, but is unaffected by sympathetic blockade due to propranolol. Toichi's CSI and CVI have been used as measures of autonomic activity in physiological research with young children (Diego et al., 2005).

Difficult temperament—To obtain an index of difficult temperament, mothers completed the Discomfort and Anger/Frustration Subscales of the Child Behavior Questionnaire (CBQ; Rothbart, Ahadi, Hersey, & Fisher, 2001). The Discomfort scale is designed to capture dispositions to experience negative affect in response to various stimuli ($\alpha = .61$), whereas the Anger subscale captures negative affect dispositions in response to interruption of goals and activities ($\alpha = .76$). The two measures were standardized and averaged together to form a composite of difficult temperament.

SES—Caregiver reports of their own and their partner's educational level and occupational status were coded using the Hollingshead Four Factor Index of Social Class. Higher scores reflect higher SES (Hollingshead, 1975).

Results

For descriptive purposes, Table 1 provides the means, standard deviations, and intercorrelations among the measures of main constructs. Consistent with the low correlations between sadness and the primary variables in Table 1, sadness was not associated with interparental aggression or physiological functioning in preliminary

structural equation modeling analyses (SEM). Consequently, sadness was dropped from the primary analyses to increase model parsimony.

Primary Analyses

SEM was utilized to test hypothesized process models. Models were estimated using the full-information maximum likelihood method (FIML) through the AMOS 7.0 statistical software (Arbuckle, 2006). Because some mediational guidelines require that a significant association exist between the predictor and outcome, our first step was to explore whether interparental aggression was directly associated with children's physiological functioning prior to estimating paths between our proposed mediators and children's physiological functioning. Thus, the model in Figure 1 was estimated while constraining paths between emotional reactivity and physiological functioning to 0. The bracketed path coefficients in Figure 1 denote the resulting parameter estimates in the model. Interparental aggression was significantly associated with greater cortisol levels, higher PNS activity, and dampened SNS functioning.

Given the documented paths between interparental aggression and children's physiological functioning, we next examined the mediational model depicted in Figure 1. The model fit the data adequately, $\chi^2(53, N = 200) = 103.67, p < .005, \chi^2/df$ ratio = 1.96, CFI = .96, and RMSEA = .069. The results indicated that interparental aggression was associated with greater toddler fear responses to conflict which, in turn, were associated with lower SNS activity. In support of two additional mediational pathways, interparental aggression was associated with toddler angry reactivity to conflict which, in turn, predicted higher cortisol levels and PNS activity. As a further test of the mediational role of angry and fearful emotional reactivity, we conducted follow up analyses using MacKinnon and colleagues (2007) procedures for calculating the significance of the indirect effects. Results indicated that all three indirect pathways were significant, including links between interparental aggression and (a) fearful reactivity and SNS activity, 95% CI = .017, .002, $p < .05$; (b) angry reactivity and cortisol, 95% CI = .00012, .005, $p < .05$; and (c) angry reactivity and PNS functioning, 95% CI = .0004, .01, $p = .01$.

Alternative Models

To examine the robustness of the three mediational pathways, we conducted two additional sets of analyses. First, given the potential moderating role of child gender in models of emotion regulation, we examined whether the proposed mediational pathways differed as a function of gender by simultaneously estimating the models for boys and girls using multiple-group analysis. Model comparisons revealed no significant difference in fit for each of the models, thereby indicating that child gender did not moderate the proposed pathways. Second, we also tested the model in Figure 1 by including difficult temperament and SES variables as covariates. Specifying paths between each of these covariates and children's emotional reactivity and physiological functioning failed to alter the significant indirect pathways in our SEM, thereby supporting the stability of our results.

Discussion

Our results provide support for family risk models and their premise that emotional responses to family stress are key mechanisms in the link between early exposure to family discord and homeostatic set points of physiological functioning (Repetti et al., 2002). Underscoring the significance of interparental aggression in the lives of children, exposure to violence and hostility between parents was associated with child HPA, SNS, and PNS functioning. Mediational analyses further indicated that toddler fearful and angry emotional reactivity mediated pathways between interparental conflict and their physiological activity

after controlling for the roles of SES and child temperament as potential explanatory mechanisms.

Consistent with prior research in middle childhood and adolescence (Pendry & Adam, 2007; Saltzman et al., 2005), interparental discord was associated with greater child cortisol levels. Drawing on prior conceptual models (Davies & Sturge-Apple 2007; Pendry & Adam, 2007), a plausible interpretation is that the tense environment created by quarrelling parents alters the HPA axis set point by engendering heightened anticipatory vigilance, threat, and emotionality in children. For example, according to the sensitization hypothesis (Davies & Sturge-Apple, 2007), exposure to interparental discord progressively increases children's emotional reactivity to conflict and, ultimately, leads to stable individual differences in their daily physiological and psychological functioning. However, providing compelling support for this interpretation requires extending documentation of associations between interparental conflict and cortisol levels to explore children's patterns of reactivity to conflict as intervening mechanisms in these pathways. As a first foray into testing this proposition, our findings indicated that children's angry conflict reactivity was a primary intermediary mechanism underlying associations between interparental conflict and children's cortisol levels.

A similar pattern of associations between interparental aggression, angry reactivity to conflict, and heightened physiological activity was also identified in the prediction of cardiac PNS functioning. Interparental aggression was associated with greater PNS functioning prior to estimating paths between emotional reactivity and the physiological variables. Moreover, angry emotional reactivity was a significant mediator of the relationship between interparental aggression and greater PNS activity. Our results do not readily support conceptualizations of greater cardiac PNS activity as markers of developmentally proficient patterns of emotional affect regulation (Beauchaine, 2001). Specifically, the angry reactivity composite, which is designed to capture prolonged, dysregulated anger and hostility, should not predict heightened PNS activity. The positive association between PNS and interparental aggression, a well-established risk factor, is also inconsistent with the interpretation that high cardiac PNS is a physiological marker of developmentally appropriate patterns of adaptation to the environment. Rather, our results are more consistent with prior empirical documentation of associations between higher cardiac PNS activity and young children's exposure to other known family risk (e.g., negative parenting, maternal depression) factors (Ashman, Dawson, & Panagiotides, 2008; Gottman & Katz, 1989; Field et al., 2001) and their negative emotionality and behavioral difficulties (Bell & Deater-Deckard, 2007).

If these findings are replicated, then modification of some conceptualizations of the PNS activity may be warranted. For example, it is possible that successive reorganizations in biological systems in early childhood may result in qualitative changes in the functions of PNS activity across later developmental periods. Consistent with this assumption, negative behavioral functioning is associated with greater cardiac PNS in the first two years of life (Bell & Deater-Deckard, 2007). However, in reflecting a possible developmental shift, higher PNS functioning is a reliable correlate of self-regulation, adaptation, and competence in early and middle childhood.

Individual differences in toddler cardiac SNS activity were explained by a different family pathway. Witnessing elevated interparental aggression was associated with lower SNS activity. The results further indicated that children's fearful reactivity to interparental conflict was a mediator of the relationship between interparental aggression and SNS functioning. Interparental aggression was associated with greater fearful reactivity to interparental conflict which, in turn, predicted dampened SNS functioning. Although the

limited knowledge base on the family process correlates of SNS activity hinders the formulation of well-developed explanations of these findings, it is consistent with the premise that “SNS tone might be low (p. 500, Porges, 1992)” in stress-vulnerable children. In keeping with this interpretation, the attenuation model postulates that adaptation, or the tendency of systems to maintain an internal state of equilibrium, may explain the coupling of lower SNS activity with toddler experiences of interparental aggression and frightened behaviors (Susman, 2006). The lowering of the set point of the SNS system may specifically reflect the activation of processes designed to prevent the toxic effects of the chronic overarousal in response to threat. However, if this process is operating, then empirical documentation of associations between indices of low SNS activity and greater externalizing symptoms suggests that it incurs costs in the form of psychological difficulties (Lorber, 2004).

The study findings must also be interpreted in relation to its limitations. First, the cross-sectional design cannot definitely address the temporal ordering of relationships in our model. Our results do not rule out a plausible alternative hypothesis that physiological functioning (e.g., PNS, cortisol) may mediate links between interparental aggression and children’s emotional responding to conflict. However, at this early stage of research, theoretical and methodological considerations support the appropriateness of our design and interpretation of findings. Bolstering the theoretical viability of our conclusions, several family process models postulate that indices of emotional reactivity to family stress are central intervening processes in links between family discord and broader indices of child outcomes, regardless of whether outcomes are manifested in physiological or psychological functioning (Davies et al., 2006; Lueken et al., 2006; Repetti et al., 2002). Furthermore, because the measurement battery was designed to delineate children’s emotional reactivity within a temporal window that preceded assessments of physiological functioning, analyzing emotional reactivity as an explanatory mechanism rather than an outcome of physiological functioning was also more valid from a methodological perspective.

Second, measurement issues further qualify conclusions that can be drawn from the findings. Although the multi-method measurement (i.e., maternal questionnaires, interview ratings, physiology) battery in this study has advantages in reducing method variance, common method variance may still be operating due to our reliance on maternal accounts of interparental aggression and child emotional reactivity. Similarly, in spite of documentation of the validity of maternal reports of child conflict reactivity, the stressfulness and emotional salience being directly involved in interparental disputes may compromise the accuracy of maternal reports of child reactivity. Thus, replicating our results using other methods within a longitudinal design is an important research direction.

Despite these limitations, our multi-method, multiple level of analysis study represents one of the first attempts to identify pathways between young children’s exposure to interparental aggression, their emotional reactivity to conflict, and their physiological functioning. Consistent with multiple theories, our results supported a model in which interparental aggression was associated with individual differences in HPA, SNS, and PNS functioning through its link with heightened angry and fearful patterns of responding to interparental conflict.

KEY POINTS

- Children exposed to interparental aggression are at risk for developing psychological and physical health problems.

- Children exposed to higher levels of interparental aggression had greater cortisol levels and parasympathetic nervous system activity and diminished sympathetic nervous system activity.
- Toddler angry emotional reactivity was an explanatory mechanism in associations between interparental aggression and cortisol and parasympathetic nervous system functioning.
- Toddler fearful emotional reactivity accounted, in part, for the link between interparental aggression and SNS functioning.
- Understanding emotional reactivity and physiological correlates of interparental aggression may assist in the identifying children and families who are most in need of intervention and formulating clinical objectives and tools designed to counteract the pathogenic processes.

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Figure 1.
A structural equation model testing forms of emotional reactivity as mediators in associations between interparental aggression and child physiological functioning. Parameter estimates for the structural paths are standardized path coefficients. * $p < .05$.

Table 1
Means, standard deviations, and intercorrelations of the main variables in the primary analyses

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
<u>Interparental Aggression</u>																
1. CPS – Phys. Viol.	--															
2. CTS2- Phys. Assault	.61*	--														
3. IDI Aggression	.54*	.39*	--													
4. CTS- Injury	.55*	.69*	.35*	--												
<u>Emotional Reactivity</u>																
5. Fear (Behavior)	.25*	.16*	.42*	.16*	--											
6. Fear (Molar)	.26*	.12	.37*	.16*	.87*	--										
7. Fear (Intensity)	.17*	.07	.10	.07	.51*	.55*	--									
8. Sad (Behavior)	.03	.03	.12	.10	.16*	.16*	.05	--								
9. Sad (Molar)	.03	.00	.10	.10	.16*	.19*	.05	.92*	--							
10. Sad (Intensity)	.10	.07	.14*	.19*	.15*	.19*	.05	.87*	.84*	--						
11. Anger (Behavior)	.07	.19*	.11	.08	-.04	-.04	-.07	-.04	-.11	-.04	--					
12. Anger (Molar)	.13	.21*	.18*	.08	-.06	-.02	-.08	-.03	-.10	-.04	.87*	--				
13. Anger (Intensity)	.07	.15*	.05	.02	-.01	-.01	-.01	-.15*	-.15*	-.15*	.63*	.58*	--			
<u>Physiological Activity</u>																
14. PNS	.11	.16*	-.04	.15*	-.01	-.01	-.08	-.05	-.10	.02	.18*	.20*	.14*	--		
15. SNS	-.15*	-.11	-.13	-.08	-.20*	-.21*	-.07	-.09	-.14*	-.12	-.05	-.04	-.05	-.43*	--	
16. Cortisol	.13	.28*	.06	.11	-.06	-.02	.05	.08	.03	.08	.19*	.21*	.18*	.14	-.09	--
<i>M</i>	2.49	2.75	3.97	0.41	0.79	2.87	0.46	0.32	0.79	0.41	0.42	1.70	0.28	4.60	1.48	-0.74
<i>SD</i>	3.56	4.23	3.60	1.21	0.73	2.58	0.83	0.56	1.55	0.78	0.62	2.44	0.69	0.52	0.56	0.27

* *Note.* $p < .05$.