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Interparental Conflict and Child HPA-Axis Responses to Acute Stress: Insights Using Intensive Repeated Measures

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Interparental conflict is a common source of psychosocial stress in the lives of children. The purpose of this study was to examine the association between recent interparental conflict and one component of the physiological stress response system, the hypothalamic-pituitary-adrenal (HPA)-axis. Parents of 42 children (ages 8-13 years) completed daily diaries of interparental conflict for 8 weeks. At the end of the 8 weeks, youth participated in the Trier Social Stress Test for Children (TSST-C) while providing 2 preand 4 poststress salivary cortisol samples. Youth whose fathers reported a pattern of increasing interparental conflict over the past 8 weeks demonstrated an exaggerated HPA-axis response to acute stress. Mother-reported interparental conflict was not associated with children's HPA-axis responses without accounting for fathers' reports. When accounting for fathers' reports, the offspring of mothers reporting higher average daily interparental conflict demonstrated an attenuated HPA-axis response to the stressor. By estimating both average exposure and recent patterns of change in exposure to conflict, we address the circumstances that may prompt attenuation versus sensitization of the HPA-axis in the context of interparental conflict. We conclude that the HPA-axis is sensitive to proximal increases in interparental conflict which may be one pathway through which stress affects health across development, and that incorporating father's reports is important to understanding the role of the family environment in stress responses. This study further demonstrates the value of using intensive repeated measures and multiple reporters to characterize children's psychosocial environment.

Keywords: cortisol, TSST-C, interparental conflict, marital conflict, daily diary

Interparental conflict is a common source of psychosocial stress for children and adolescents (Cummings & Davies, 2002; Davies & Cummings, 1994). High conflict family environments are pro-

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spectively associated with negative health outcomes (Masarik & Conger, 2017; Mechanic & Hansell, 1989; Montgomery, Bartley, & Wilkinson, 1997; Raposa, Hammen, Brennan, O'Callaghan, & Najman, 2014). One critical component of the body's physiological stress response, the HPA-axis, is socially regulated during childhood (Gunnar & Donzella, 2002; Hostinar, Sullivan, & Gunnar, 2014). Thus, one pathway through which interparental conflict may lead to degradations in health is through alterations to the functioning of this system (Hostinar & Gunnar, 2013; Repetti, Taylor, & Seeman, 2002). Yet, published research on this topic to date has focused predominantly on trait-level measures of interparental conflict and has relied on mothers' reports of psychosocial dynamics in the child's home. The purpose of this study was to determine how recent patterns of high or increasing interparental conflict was associated with variability in children's HPA-axis responses to acute laboratory stress. Further, we sought to clarify the distinct contributions of mothers' and fathers' perceptions of interparental conflict to this phenomenon.

There are two, seemingly competing, hypotheses on how family conflict may influence the HPA-axis response to stress. One suggests that greater exposure to family conflict will be associated with exaggerated, or sensitized, responses to stress (Doom & Gunnar, 2013; Gunnar & Vazquez, 2006). The other hypothesis is that greater exposure to family conflict will be associated with attenuated responses to stress (Susman, 2006). This attenuated pattern may indicate that the child has habituated to social threats like interparental conflict because they have been exposed repeatedly over time (Seeman & McEwen, 1996). Further, there is a well-established theoretical model that suggests that HPA-axis responses to stressors depend upon a child's emotional reaction to that stressor (Davies & Martin, 2013). Indeed, children who respond with more anger to interparental aggression also exhibit greater HPA-axis reactivity (Davies, Sturge-Apple, Cicchetti, Manning, & Zale, 2009). Importantly, this theoretical model hypothesizes that children's exposure to interparental conflict may alter their response to social threats more broadly (i.e., outside of the home; Davies & Martin, 2013), which may have long-term implications for the child's health and development.

There is evidence for both sensitization and attenuation of the HPA-axis in the context of interparental conflict. Some of these studies have examined youth HPA-axis responses to simulated interparental conflict in the laboratory with mixed results. For example, greater interparental conflict has been associated with attenuated cortisol responses to simulated marital conflict in the lab (Davies, Sturge-Apple, Cicchetti, & Cummings, 2007). However, HPA-axis responses to simulated conflict in the lab are exaggerated when the child also exhibited distress (Davies, Sturge-Apple, Cicchetti, & Cummings, 2008; Koss et al., 2013). In parallel, a number of studies have emerged examining variability in interparental conflict and youth responses to a novel psychosocial stress paradigm. These studies, which also return mixed results, speak to how exposure to interparental conflict may shape responses to stress both in and outside of the home. For example, adolescents exposed to more family aggression demonstrated attenuated cortisol concentrations during novel laboratory stress (Lucas-Thompson, 2012; Saxbe, Margolin, Spies Shapiro, & Baucom, 2012), and young adults exposed to both very high and very low interparental conflict exhibited exaggerated HPA-axis responses to acute stress (Hagan, Roubinov, Purdom Marreiro, & Luecken, 2014). One potential explanation for these equivocal findings is the dynamic sensitivity of the HPA-axis to different social contexts during childhood. Thus, rather than focusing on trait differences between families in the frequency and intensity of interparental conflict, equal attention should be focused on whether interparental conflict has been increasing or decreasing in the recent past.

Yet, the predominant model for examining the association between interparental conflict and child HPA-axis reactivity is to use traitlike measures of conflict. These measures typically make use of retrospectively reported conflict via questionnaires (e.g., Hagan et al., 2014), observer-rated interparental conflict in the laboratory (e.g., Koss et al., 2013), or a combination of both (Davies et al., 2007; Lucas-Thompson, 2012; Saxbe et al., 2012). These approaches to measuring interparental conflict pay less attention to how responsivity of the HPA-axis may change as a function of changes in interparental conflict over time. Indeed, both experimental evidence in animals and correlational research in humans has shown that recent stress has the potential to sensitize an individual to subsequent stressors (Kuhlman, Chiang, Horn, & Bower, 2017). Indeed, children exhibit upregulated cortisol on days they experience stress at home or at school (Bai, Robles, Reynolds, & Repetti, 2017), and increases in family conflict from week to week also correspond to upregulations in a child's

diurnal regulation of cortisol (Kuhlman, Repetti, Reynolds, & Robles, 2016). These findings suggest that similar upregulations in HPA-axis responses to acute stress may also occur in the wake of increases in interparental conflict. The present study aims to address this gap in the existing literature by using intensive repeated measures to prospectively assess daily interparental conflict over 8 weeks and determine its association with HPA-axis reactivity to acute psychosocial stress. This method enabled us to differentiate between two indicators of exposure to conflict between parents, typical levels of exposure and recent patterns of change (increases or decreases) in conflict, which may clarify some of the equivocal findings in the literature pertaining to attenuation and sensitization.

Furthermore, more studies are needed to characterize how social processes in families are associated with HPA-axis responses to acute stress in early adolescence. To date, relatively few of the existing studies examining the role of interparental conflict in HPA-axis responses to acute psychosocial stress have been conducted in adolescent samples (Koss et al., 2011, 2014; Lucas-Thompson, 2012; Lucas-Thompson & Granger, 2014; Saxbe et al., 2012), and these samples tended to be older adolescents. During adolescence, neurobiological processes are relatively more sensitive to environmental influences (Fuhrmann, Knoll, & Blakemore, 2015) and may set the stage for physiological responses to stress well into adulthood. These differences begin as early as age 9 (Dahl, 2004). This is an important gap in our understanding because the predominant theoretical model focuses on family psychosocial environments in early childhood as important in shaping the long-term functioning of physiological stress response systems (Gunnar & Donzella, 2002; Meaney, 2001). Determining HPA-axis sensitivity in late childhood and early adolescence to short-term changes in family conflict may illuminate psychosocial intervention targets for adolescents that mitigate the long-term negative health sequelae of living in a family characterized by high conflict.

The purpose of this study was to extend our knowledge of the associations between interparental conflict and HPA-axis responses to acute stress among youth. To do this, we measured daily mother- and father-reported interparental conflict for 8 weeks. From these diary assessments, we computed two different indices: typical interparental conflict and recent patterns of change in interparental conflict. We then examined the associations between these indices and their child's HPA-axis response to a novel psychosocial stressor at the end of the 8 weeks. Based upon the existing literature, we hypothesized that higher average interparental conflict would be associated with attenuated HPA-axis responses to stress. However, we also hypothesized that recent patterns of increasing interparental conflict would be associated with stress sensitization, or more exaggerated HPA-axis responses to acute stress.

Method

Participants

Participants in this study were 42 youth (25 girls) and their parents (41 mothers and 39 fathers) who participated in a study designed to investigate the association between family interactions and health. Inclusion criteria for the study were youth between the ages of 8 and 13 years, currently living in two-parent households,

with at least one biological parent willing to participate. Eligibility was not limited to legally married parenting couples, nor heterosexual parent couples, although only heterosexual couples participated. Youth were excluded from the larger study if they had any major medical conditions that are known to affect the HPA-axis or immune system. Among the 47 youth participating in the larger study, 44 completed the laboratory stress protocol, and 42 provided sufficient cortisol samples to model stress responses. Median personal income reported by each parent was within the \$31,851 to \$64,250 tax bracket, and 57% of mothers (59% of fathers) attained at least a bachelor's degree or higher. Youth were between the ages of 8-13 ($M_{age} = 11.09$, $SD_{age} = 1.64$), 46.8% non-Hispanic white, 23.4% Latino/Hispanic, 23.4% African American, 4.3% Asian, and 2.1% Other.

Procedures

Participants were recruited from public schools, pediatric clinics, community centers, newspaper advertisements, and direct mailings. This study was approved by the University of California Los Angeles Institutional Review Board; parents provided written consent to study procedures and youth provided assent. Active data collection for this study took place exclusively between September and May in order to avoid summer vacation. For 8 weeks, participating parents completed online daily diaries before bed. Diaries assessed multiple facets of daily life including their experiences of conflict with their spouses. After the 8-week daily diary phase, youth were invited to participate in a standardized laboratory stress task. Throughout this task, youth provided six saliva samples at 35 min and immediately before the stressor and then 20, 30, 40, and 50 min after stress initiation.

Measures

Interparental conflict. Daily interparental conflict was assessed via online diaries completed by parents each evening for 8 weeks; all 42 youth had interparental conflict data from at least one parent. The 14 conflict items from the Adult Home Data Questionnaire (Timmons & Margolin, 2015) included six statements describing the individual's behavior ("I expressed anger or irritation at my partner"; "I felt distant or withdrawn from my partner"; "I hit, pushed, or shoved my partner"; "I nagged my partner"; "I ignored my partner's wishes or needs"; "I took my partner's feelings lightly"), two statements describing the dyad's behavior ("My partner and I disagreed about a child-related issue," "My partner and I disagreed about an issue unrelated to children"), and six statements describing the partner's behavior ("My partner . . . expressed anger or irritation at me"; " . . . seemed distant or withdrawn from me"; " . . . hit, pushed, or shoved me"; " . . . nagged me"; "... ignored my wishes or needs"; and "... took my feelings lightly"). Parents used a 3-point response scale to rate the degree to which each statement described their interactions with their partners that day (1 = not at all, 2 = some, and 3 = a lot). Responses to the 14 items were averaged to compute a daily score for each parent, with lower values indicating less conflict that day.

Daily diary completion in this study was excellent; 95% and 94% for mothers and fathers, respectively. All 56 days of diary ratings were available for 30% of mothers (range = 27–56 days) with complete diaries), and 26% of fathers (range = 20–56 days),

and the average $\pm SD$ number of completed daily diaries was 49.4 \pm 8.8 for mothers and 48.5 \pm 10 for fathers. Both betweenperson and within-person reliability of daily diary measured interparental conflict across the 8 weeks were excellent (Sears, Repetti, Reynolds, Robles, & Krull, 2016). The number of diaries completed by mothers and fathers was not associated with child age, p > .55, family income, p > .69, child-reported family conflict, p > .82, slope of mother-reported conflict, p > .40, mean fatherreported conflict, p > .29, slope of father-reported conflict, p > .20, baseline cortisol, p > .28, or peak cortisol, p > .61. Mothers who reported higher scores on their child's pubertal development completed fewer diaries, r = -.34, p = .02, and mothers who reported higher average daily interparental conflict across the 8-week assessment phase had husbands who completed fewer daily diaries, r = -.33, p = .044.

Youth participating in this study also completed the Conflict Properties subscale of the Children's Perception of Interparental Conflict Scale (CPIC) at the baseline assessment (Grych, Seid, & Fincham, 1992). The CPIC is a 48-item self-report questionnaire comprised of 3 subscales: Conflict Properties, Threat, and Self-Blame. In the 19-item Conflict Properties subscale the child responds with *true, sort of or sometimes true,* or *false* to statements about the frequency (e.g., how often parents argue), intensity (e.g., the extent of yelling or physical contact during fights), and resolution of conflict (e.g., whether parents remain angry after arguments have ended). Responses were averaged across all items in the subscale. Reliability for the CPIC within our sample was excellent, $\alpha = .90$.

Pubertal development. Mothers completed the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988) in terms of growth spurt, skin changes, body hair growth, and breast growth for daughters. Responses on the 4-point scale $(1 = has not yet begun, 2 = has barely begun, 3 = has definitely begun, 4 = has been completed) were averaged. The PDS is a valid and reliable measure that correlates with physician reports of pubertal development (Dorn & Biro, 2011). Reliability for the mother-reported PDS within our sample was good, <math>\alpha = .81$.

HPA-axis response to acute stress. Physiological stress reactivity and recovery was measured by salivary cortisol responses to the TSST-C (Buske-Kirschbaum et al., 1997). All laboratory sessions took place in the afternoon and early evening. In front of a camera and trained judges, youth were given the beginning of a story and were given 5 min to prepare an "exciting ending." After the speech task, children were asked to serially subtract 7 from 758 as quickly and as accurately as possible for 5 min and prompted to start over when they made a mistake. This task was adapted for use in child populations from the Trier Social Stress Test (TSST; Kudielka, Hellhammer, & Kirschbaum, 2007) and is a reliable HPA-axis activator (Foley & Kirschbaum, 2010).

Salivary cortisol was assayed from six samples of saliva collected 35 min and immediately prior to stress onset (baseline) and 20, 30, 40, and 50 min after stress initiation. Of the 42 youth included in our analyses, 32 (76.2%) had complete data for salivary cortisol, seven (16.7%) were missing one sample, and three (7.1%) were missing two samples. Saliva samples were frozen after collection and shipped on dry ice to the TUD Biopsychology Laboratory in Dresden, Germany directed by Clemens Kirschbaum. Cortisol concentrations were measured using a commercially available enzyme immunoassay. All samples from the same person were assayed on the same plate (50 μ l saliva required; minimum detection limit <.003 μ g/dL, mean intra- and interassay coefficients of variance below 10%). Reactivity to the laboratory stressor was indexed by the linear rate of increase in cortisol following the initiation of stress. Recovery from the laboratory stressor was indexed by the quadratic effect of time on cortisol, indicating the rate of cortisol decline following peak response to the stressor.

Data Analysis

All analyses were conducted using SPSS Version 24. All continuous variables were tested for normality. We identified three cortisol values that slightly exceeded 4 standard deviations from the mean, and these values were winsorized to maximize available data. For all multivariate analyses, cortisol values were transformed using the natural log transformation to reduce skew and kurtosis. Two parameters of perceived interparental conflict were computed for each parent: (a) the average daily report of interparental conflict, and (b) a slope that reflected the pattern of change in conflict during the 8-week data collection period. The slope for each respondent was estimated using multiple linear regression predicting interparental conflict from study day (1–56).

The initial saliva sample (35 min prior to stress initiation) was not included in cortisol response models. Linear mixed models with an unstructured covariance matrix and maximum likelihood estimation were used to examine how cortisol, nested within individuals, changed from baseline (immediately prior to stress) to 20, 30, 40, and 50 min after the stress initiation. We first tested whether a linear model (with only a linear parameter), a quadratic model (with linear and quadratic parameters), or a cubic model (with linear, quadratic, and cubic terms) best fit the pattern of cortisol change over time. The quadratic model was the best fit to the data (linear Akaike information criterion [AIC] = 329.4; quadratic AIC = 295.1; cubic AIC = 296.2), thus all subsequent models included a linear (reactivity) parameter and a quadratic (recovery) parameter. Following stress exposure, cortisol concentrations demonstrated a linear increase over time, b = .027, SE =.004, p < .001, that reached an inflection point (peak response) and began to decline, b = -.0004, SE = .00007, p < .001. All subsequent models were then compared with this cortisol-only model to determine the additional variance accounted for by the inclusion of our hypothesized predictors.

We then examined whether sex, age, pubertal status, ethnic group (0 = non-Hispanic white, 1 = all other ethnic groups), and youth reports of interparental conflict at baseline were associated with the pattern of HPA-axis response to the stressor. See below for details on these analyses. Only age and sex were associated with youth cortisol responses to stress, therefore subsequent models tested whether significant associations persist above and beyond the effects of sex and age.

We then examined patterns of cortisol response to stress as a function of time, the interparental conflict indices, and the interaction of time and interparental conflict indices. We separately tested the models of interparental conflict indices as reported by mothers and fathers, and finally a model of mother and father indices of interparental conflict simultaneously. Models using only mother-reported predictors reflected data from 41 families, only father-reported predictors reflected data from 39 families.

Specifically, age accounted for 2% of variance in HPA-axis response to the stressor (AIC = 289.64). Older youth demonstrated a slower linear increase in cortisol following stress, b = -.009, SE = .003, p < .001, and a more marked decline after reaching their peak cortisol response, b = .0001, SE = .00004, p =.005, but age was not associated with cortisol concentrations at baseline, b = .053, SE = .064, p = .41. Sex accounted for 4% of variance in HPA-axis responses to the stressor (AIC = 281.88). Compared with male participants, female participants had lower cortisol values prior to stress exposure, b = -.46, SE = .20, p =.028, and also demonstrated a faster increase in cortisol following stress, b = .04, SE = .008, p < .001, and a delayed recovery from their peak cortisol response, b = -.0004, SE = .0001, p = .003. Puberty did not account for any variance in HPA-axis response to the stressor (AIC = 300.64). Mother-reported pubertal status was not associated with cortisol concentrations at baseline, b = .009, SE = .12, p = .94, nor patterns of change in cortisol over time, linear b = -.003, SE = .005, p = .51, quadratic b = .00003, SE =.0001, p = .72. Ethnic group did not account for any variance in HPA-axis response to the stressor (AIC = 299.16). Ethnic group was not associated with cortisol concentrations at baseline, b = -.032, SE = .08, p = .67, nor patterns of change in cortisol over time, linear b = -.004, SE = .003, p = .25, quadratic b =.00001, SE = .000005, p = .25. Youth-reported interparental conflict at baseline (CPIC) accounted for 22% of variance in HPA-axis responses to stress (AIC = 229.99), and was not associated with cortisol concentrations at baseline, b = .213, SE = .26, p = .42, nor patterns of change in cortisol over time, linear b = -.003, SE = .01, p = .79, quadratic b = .00002, SE = .0002, p = .86.

Results

The most frequently endorsed interparental conflict items for this sample were "I felt distant or withdrawn from my partner" on 16.3% of diaries, "I expressed anger or irritation at my partner" on 15.5% of diaries, "My partner expressed anger or irritation at me" on 14.9% of diaries, "My partner seemed distant or withdrawn from me" on 14.3% of diaries, "My partner took my feelings lightly" on 13.5% of diaries, "My partner and I disagreed about an issue unrelated to children" on 12.2% of diaries, and "My partner ignored my wishes or needs" on 11.5% of diaries. There were no items in our daily interparental conflict that were never endorsed throughout the 8-week assessment, including those referencing physical violence.

Table 1 presents descriptive characteristics of key study variables as well as bivariate correlations between them. Daily reports of interparental conflict were low for both mothers and fathers in this sample, slightly higher in mothers than fathers, t = -2.13, p = .040, and did not change significantly across the 8-week diary phase, t = -1.54, p = .124 and t = -1.93, p = .053 for mothers and fathers, respectively. There was a positive correlation between mother and father-reported mean interparental conflict, and the slopes for mother and father reports of conflict were not significantly correlated within one another. In addition, children's descriptions of greater interparental conflict were associated with a pattern of increasing maternal reports of conflict. Associations between children's reports of conflict and mother- and father-reported interparental conflict averaged over the 8-week assess-

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Table 1	
Means and Standard Deviations for All Key Study Variable	es and Bivariate Correlations Between Them

Variable	M (SD)	1	2	3	4	5	6	7	8	9
1. Age	11.09 (1.64)	1.00								
2. PDS	2.14 (.83)	082	1.00							
3. Family income ^a	2.75 (1.04)	.014	123	1.00						
4. Child-reported conflict properties										
scale (CPIC)	1.53 (.38)	131	109	.243	1.00					
5. Mother-reported mean marital										
conflict (daily diary)	1.12 (.14)	.034	.039	.313*	.271†	1.00				
6. Mother-reported slope of marital										
conflict (daily diary)	06 (.21)	344*	084	125	.312*	.199	1.00			
7. Father-reported mean marital										
conflict (daily diary)	1.08 (.08)	150	.088	074	.314†	.497**	.259	1.00		
8. Father-reported slope of marital										
conflict (daily diary)	10 (.22)	261	.139	252	.149	020	.199	.130	1.00	
9. Baseline cortisol (nmol/L)	4.25 (3.99)	.176	.000	.275†	.192	046	.034	178	004	1.00
10. Peak cortisol (+30 min)	8.96 (9.15)	29^{\dagger}	133	.126	.117	237	084	199	.113	.127

Note. Cases for bivariate correlations were deleted pairwise (n = 41 for mother-reported data; n = 39 for father-reported data; n = 42 with hypothalamic-pituitary-adrenal (HPA)-axis reactivity; n = 38 youth with complete mother-reported, father-reported, and HPA-axis reactivity data. PDS = Pubertal Development Scale; CPIC = Children's Perception of Interparental Conflict Scale.

^a Average parent income was reported in tax brackets where $1 = \langle \$7, \$25, 3 = \$31, \$51-64, 250, and 5 = \rangle \$171, \$50$.

p < .10. p < .05. p < .01.

ment phase were positive, although these associations were non-significant.

Youth in this study demonstrated a robust, average increase in cortisol from 4.25 nmol/L immediately prestress to 8.96 nmol/L at the mode peak response (30 min poststress initiation); 64.3% (n = 27) of participants demonstrated an increase in cortisol following the TSST-C. Figure 1 displays mean (*SEM*) cortisol concentrations throughout the stress task.

Mother-Reported Interparental Conflict and Youth HPA-Axis Response to Acute Stress

We first examined the associations between mother-reported measures of interparental conflict and changes in cortisol following acute stress. Mother-reported mean interparental conflict across the 8-week daily diary phase accounted for 23% of variance in HPA-axis response to the stressor (AIC = 227.28). However, mother-reported mean interparental conflict was not associated

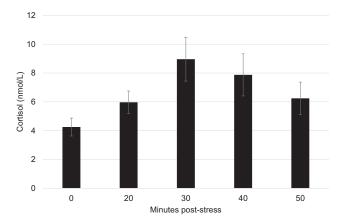


Figure 1. Mean (± SEM) cortisol following stress onset.

with cortisol at baseline, b = -.490, SE = .702, p = .49, nor patterns of change in cortisol over time, linear b = -.040, SE = .026, p = .13, quadratic b = .0006, SE = .0004, p = .14. In this model, it is important to note that the standard error of the intercept increased from .10 to .80 compared with the unadjusted model of cortisol response, which decreased the reliability of this term from p < .001 to p = .026. Similar changes in the estimates for the linear and quadratic terms for change in cortisol across the protocol also occurred. Thus, it appears that mean mother-reported conflict is accounting for some of the variance accounted for by the timing of the samples, despite the timing of the samples remaining a better predictor of those cortisol values. Change in mother-reported interparental conflict over the past 8 weeks (slope) accounted for less than 1% of variance in HPA-axis response to the stressor (AIC = 293.05), and was not associated with cortisol at baseline, b = -.199, SE = .498, p = .69, nor patterns of change in cortisol over time, linear b = -.0005, SE = .02, p =.98, quadratic b = -.00001, SE = .0003, p = .97.

We then tested a model including both average and slope indices of mother-reported interparental conflict simultaneously, which accounted for 23% of variance in HPA-axis responses to the stressor (AIC = 226.1). Again, neither index of mother-reported conflict was associated with their child's HPA-axis response, all ps > .20. This pattern of results was unchanged when accounting for the number of diaries completed by each mother in our sample.

Father-Reported Interparental Conflict and Youth HPA-Axis Response to Stress

We separately examined the associations between fatherreported measures of interparental conflict and cortisol response to acute stress. Father-reported mean interparental conflict across the 8-week daily diary phase accounted for 8% of variance in HPAaxis response (AIC = 271.21). However, father-reported mean interparental conflict was not associated with cortisol at baseline, b = -2.80, SE = 1.46, p = .062, nor patterns of change in cortisol over time, linear b = .040, SE = .061, p = .52, quadratic b = -.0005, SE = .001, p = .63. Change in father-reported interparental conflict over the past 8 weeks (slope) accounted for 10% of variance in HPA-axis response to the stressor (AIC = 266.33). A pattern of increasing father-reported interparental conflict during the 8 weeks prior to the laboratory stressor was associated with faster cortisol acceleration in response to stress, linear b = .050, SE = .022, p = .028, and nonsignificantly slower recovery from the stressor, quadratic b = -.0007, SE = .0004, p = .065, but was not associated with cortisol at baseline, b = .118, SE = .557, p = .83.

We then tested a model that included the mean and slope of father-reported interparental conflict simultaneously, which accounted for 18% of variance in HPA-axis responses to the stressor. In this model, higher average father-reported conflict was nonsignificantly associated with lower cortisol at baseline, b = -2.92, SE = 1.50, p = .059, and not associated with cortisol responses to the stress task, p > .76. Consistent with the unadjusted model, a slope of increasing interparental conflict was associated with exaggerated activation of the HPA-axis following stress, linear b = .050, SE = .023, p = .030, quadratic b = -.001, SE = .0004, p = .073. This pattern of results was unchanged when accounting for the number of diaries completed by each father in our sample.

Combined Parent-Reported Interparental Conflict and Youth HPA-Axis Response

Finally, we simultaneously examined the associations between both mother- and father-reported patterns of interparental conflict (mean and slope) and cortisol responses to the stressor. See Table 2 for the estimates of HPA-axis response to acute stress by motherand father-reported means and slope of interparental conflict across the preceding 8 weeks. This simultaneous model accounted for 32% of variability in cortisol responses to stress (AIC = 202.1). Perhaps more notably, adding father-reported indices of

Table 2

Simultaneous, Unstandardized Coefficient Estimates Predicting Change in Cortisol in Response to Acute Stress from Fatherand Mother-Reported Indices of Daily Interparental Conflict Over the Preceding 8 Weeks

Predictor	B (SE)	t
Intercept	3.81 (1.70)	2.25*
Time	.09 (.06)	1.46
Time ²	002(.001)	-1.87^{+}
Daily interparental conflict		
Mean (mother)	55 (1.16)	47
Mean (Mother) \times Time	08(.04)	-2.02^{*}
Mean (Mother) \times Time ²	.001 (.001)	2.02^{*}
Mean (father)	-1.77(1.67)	1.06
Mean (Father) \times Time	.03 (.06)	.47
Mean (Father) \times Time ²	0001(.001)	11
Slope (mother)	.29 (.55)	.52
Slope (Mother) \times Time	01(.02)	29
Slope (Mother) \times Time ²	.00002 (.0003)	.06
Slope (father)	.12 (.51)	.23
Slope (Father) \times Time	.05 (.02)	2.67**
Slope (Father) \times Time ²	001 (.0003)	-2.27^{*}

p < .10. p < .05. p < .01.

interparental conflict to the model of mother-reported indices accounted for an additional 8% of variance in cortisol responses to stress. In this model, higher average mother-reported interparental conflict was associated with an attenuated cortisol response following stress, linear b = -.08, SE = .04, p = .046, quadratic b =.001, SE = .001, p = .047. Further, a pattern of increasing father-reported interparental conflict was associated with an exaggerated cortisol response to stress, linear b = .05, SE = .02, p =.009, quadratic b = -.001, SE = .0003, p = .026. These associations remained after accounting for the effect of age, sex, and number of diaries completed by each parent in the model. Figure 2 displays the effect of mother-reported mean conflict and fatherreported slope of conflict on cortisol response to stress.

Discussion

This study examined HPA-axis responses to acute stress among youth as a function of recent exposure to interparental conflict over 8 weeks. We did not find evidence that interparental conflict was associated with youth HPA-axis responses using conflict reported by mothers alone. However, youth whose fathers reported a pattern of increasing interparental conflict demonstrated exaggerated HPA-axis reactivity to acute stress. When looking at youth HPAaxis responses as a function of both mother- and father-reported indices of interparental conflict, higher average mother-reported interparental conflict was associated with an attenuated cortisol response to the task. These findings highlight HPA-axis sensitivity to recent changes in the psychosocial environment that may shed light on neurobiological risk and resilience to stress during the transition to adolescence. Further, these results may clarify mixed findings in the literature by demonstrating that frequent, daily exposure to psychosocial stress such as interparental conflict may be associated with attenuated responses to stress, whereas recent increases in the same stressor may be associated with sensitization of the HPA-axis to acute stress. Finally, the findings highlight the value of intensive repeated measures as an approach to understanding interparental conflict, which is currently the best available method of capturing recent changes in the family environment.

Youth whose fathers reported a pattern of *increasing* interparental conflict during the 8 weeks preceding the laboratory stressor demonstrated a faster activation and slower HPA-axis recovery following stress compared with youth whose fathers reported stable patterns of interparental conflict over the same period. Thus recent increases in interparental conflict may sensitize the child's HPA-axis to incidences of social threat. Among the existing studies of adolescent HPA-axis responses to acute stress in the context of interparental conflict (Koss et al., 2011, 2014; Lucas-Thompson, 2012; Lucas-Thompson & Granger, 2014; Saxbe et al., 2012), none have used daily diary or other intensive repeated measures approaches, nor have they included reports of interparental conflict from fathers. The results of the present study underscore the added benefit of fathers' reports of escalating conflict in the home as a key predictor of stress physiology in their children. These data add to the evidence that the HPA-axis is sensitive to proximal changes in the social environment, not only when in the home as measured by diurnal cortisol (Kuhlman et al., 2016), but may also alter the way the body responds to psychological stress outside the home. Yet, it is important to note that we did not assess HPA-axis functioning before and after the 8-week

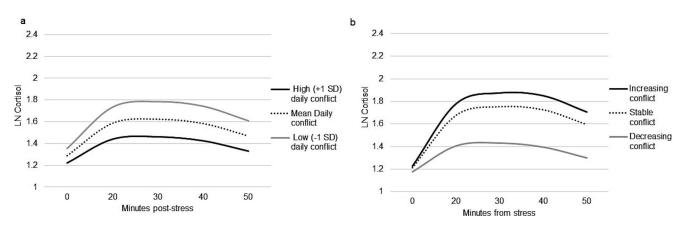


Figure 2. Cortisol response to acute psychological stress by mother-reported average interparental conflict and father-reported slope interparental conflict. Cortisol response to acute psychological stress by (a) mean $(\pm 1 SD)$ mother-reported interparental conflict, and (b) slope $(\pm 1 SD)$ of father-reported interparental conflict over the past 8 weeks.

interparental conflict phase and thus cannot speak to withinsubject changes in stress reactivity. Future investigations should also consider that youth cortisol responses to stress did not vary as a function of child-reported interparental conflict. There are several possible explanations for the lack of association in our sample. The child-report questionnaire was administered once at baseline, 8 weeks prior to the acute laboratory stressor. The differential predictive power of the child and parent report variables may therefore reflect the importance of recent patterns of conflict for child cortisol responses to stress. Further, items included in the child report of interparental conflict assess explicit conflict between parents (such as arguing) that is salient to the child, whereas the diary reports from parents assess daily experiences of interparental conflict that may not be explicitly recognized by the child but nonetheless contribute to the family environment (e.g., feeling distant or withdrawn, expressing irritation, ignoring feelings or taking them lightly). That being said, we are hesitant to overinterpret this null result and replication in a larger sample is warranted.

Previous studies have found interparental conflict to be associated with both exaggerated (Hagan et al., 2014) and attenuated (Davies et al., 2007; Lucas-Thompson, 2012; Saxbe et al., 2012) HPA-axis responses to acute stress. The present study contributes to this knowledge by demonstrating that recent changes in exposure to interparental conflict in the home and average exposure may be exerting opposite influences on HPA-axis functioning. For example, while patterns of increasing conflict as reported by fathers were associated with exaggerated cortisol responses to our stressor, higher average interparental conflict as reported by mothers was associated with attenuation of the cortisol response. It may be the case that an association between high average interparental conflict and attenuated HPA-axis responses to stress is only observable among families with stable, recent patterns of conflict. These findings highlight the importance of taking the sensitivity of the HPA-axis to changes in the psychosocial environment into consideration when designing future studies, such as incorporating intensive repeated measures that can capture nuanced changes in family relationships.

This study has important implications for our understanding of how stress "gets under the skin" to promote disease across the life

span. Specifically, social regulation of the HPA-axis can disproportionately advantage or disadvantage youth living in different types of home environments in other domains of life including peer interactions and academic settings (Gunnar & Donzella, 2002; Hostinar et al., 2014). The availability of intensive repeated measures of interparental conflict over a relatively prolonged period of time may have captured nuanced patterns in family social dynamics that have implications for the development of biobehavioral responses to stress in general. Saxbe and colleagues (2012) posit that a diminished HPA-axis response to stress, which they observed among adolescents living in families characterized by more aggression, may promote resilience in children exposed to chronic family conflict. Indeed, chronic stress has been linked to both exaggerated and attenuated HPA-axis functioning (Miller, Chen, & Zhou, 2007). Attenuated cortisol responses to simulated interparental conflict in the lab have been linked to more active coping strategies for dealing with the stressor (Sturge-Apple, Davies, Cicchetti, & Manning, 2012). Most salient to the methods in this study are the implications of these findings for the youth's ability to manage stress in a school setting. The HPA-axis response to the TSST-C most closely approximates how youth might respond to psychosocial challenges in academic settings. Thus, these results may indicate the ways in which recent increases in conflict in the family environment spill over into other social contexts.

Youth HPA-axis responses did not vary as a function of motherreported indices of interparental conflict without also accounting for father-reported indices. Yet, models using indices of interparental conflict from both mothers and fathers accounted for improvements in model fit from those using mother reports alone. Mothers and fathers may be meaningful barometers for different aspects of the social environment that are important for their child's development. Further, infants synchronize with their mothers and fathers differently for different emotions (Feldman, 2003), and adolescent emotional synchrony with one parent over another has implications for HPA-axis responses (Saxbe et al., 2014). Despite their critical role in psychosocial development (Cabrera & Tamis-LeMonda, 2013; Parke, 2000; Suh et al., 2016), inclusion of fathers in psychological research on children and families is still the exception rather than the norm. It is possible that recent increases in interparental conflict mask associations between average household conflict and youth HPA-axis responses. It is also possible, and even likely, that mother and father experiences of conflict moderate one another which will be an important question to explore in future studies that are powered to do so. Our results build upon the existing literature by demonstrating that variability in *fathers*' perceptions of interparental conflict were more robust predictors of youth physiological response to the environment than were mothers' perceptions of conflict. Greater efforts to incorporate fathers into child development research are greatly needed to interrogate the role of the family environment in biopsychosocial development.

Younger and female participants exhibited greater cortisol responses to the stress task compared with older or male participants. Age, sex, and pubertal status are key developmental characteristics associated with the body's response to stress (Dorn et al., 2009; Kudielka & Kirschbaum, 2005; Oldehinkel & Bouma, 2011). Exaggerated reactivity and delayed recovery of the HPA-axis in females compared with males in this study is consistent with long-standing observations in animal research (Kudielka & Kirschbaum, 2005). This may extend existing evidence that females, particularly during adolescence, are more sensitive than males to socially evaluative stress (Oldehinkel & Bouma, 2011). We also observed more robust activation among younger participants in our sample (ages 8-10) compared with our early adolescent participants (ages 11-13). This may reflect the increases in effective strategies for coping with stressful experiences that come with age (Steinberg, 2005). Puberty was not associated with HPA-axis responses to stress in this sample. In studies sampling youth from a wider age range, pubertal status does account for some between-subjects differences in HPA-axis functioning (Blumenthal, Leen-Feldner, Badour, Trainor, & Babson, 2014; Dorn et al., 2009). Therefore, it is possible that the age range of our participants restricted our power to detect an effect of pubertal status on cortisol response to stress.

These results should be interpreted in the context of their limitations. First, this study was designed to gather comprehensive psychosocial information over 8 weeks from a relatively small number of families (n = 42), and therefore our sample size may have precluded our ability to detect small effects and warrants replication. The sample size and measures used in this study also limited our ability to test more nuanced relationships, such as who initiated the conflict and whether the type or topic of the conflict influences the strength of the link with child cortisol responses to stress. Further, the results represent associations in intact families with low and stable interparental conflict and further investigations in higher conflict families are necessary. Fewer fathers completed the daily diary than mothers in our sample. Therefore, estimates of interparental conflict for mothers may be slightly more reliable than those from fathers. We also only examined HPA-axis responses to the TSST-C which may not reflect how the youth in this study respond to all psychological or social stressors in their lives. Different types of acute stress result in different patterns of the HPA-axis response (Dickerson & Kemeny, 2004; Kuhlman, Olson, & Lopez-Duran, 2014), and therefore more research is needed to characterize the extent of, and limitations to, the generalizability of responses to the TSST-C in daily experiences. We also only examined cortisol up to 50 min poststress initiation which limits our ability to speak to recovery of the HPA-axis from acute stress. Finally, there is an emerging body of literature showing that parents and children exhibit HPA-axis synchrony that varies as a function of marital quality and conflict (Saxbe et al., 2014). However, parents in this study did not complete the TSST and therefore we are unable to test the role of parent-child synchrony in our observations.

Both stress and social relationships have robust effects on health, which may occur in part through the gradual upregulation of physiological stress response systems that presage disease (Seeman & McEwen, 1996; Seeman, McEwen, Rowe, & Singer, 2001). The effects of stress appear to be exaggerated when exposure occurs during childhood (Repetti, Robles, & Reynolds, 2011; Repetti et al., 2002), yet the neurobiological pathways through which psychosocial stress starts these allostatic processes remains unclear. In the present study, youth HPA-axis responses appear to vary as a function of recent patterns of change in interparental conflict and average daily exposure to interparental conflict. These findings extend our understanding of how psychosocial stress in the family contributes to changes in physiological processes by demonstrating that recent increases in conflict in the home are associated with corresponding increases in HPA-axis responses to stress. When accounting for this, higher stable conflict may attenuate physiological responses to stress. These results may help to disentangle the role of chronic and recent conflict in youth stress responses. Further, this study demonstrates the importance and value of intensive repeated measures approaches to understanding psychosocial environments in the lives of children, particularly as reported by fathers who are currently underrepresented in psychological research.

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