Brief Report

Childhood Interparental Conflict and HPA Axis Activity in Young Adulthood: Examining Nonlinear Relations

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ABSTRACT: *Relations between early adversity and the neuroendocrine stress response are most often tested in a linear framework. Findings from studies of nonlinear relations between early stress and reactivity in childhood are suggestive, but curvilinear associations between childhood family stress and stress reactivity at later developmental stages remain unexplored. The current study examined curvilinear relations between childhood interparental conflict (IPC) and cortisol reactivity in young adulthood. Participants ($n = 91$; Mean age = 18.7, SD = .97; 59% White, 25% Hispanic) reported on the frequency and intensity of childhood exposure to IPC and salivary cortisol was sampled before and after a challenging interpersonal role-play task. Significant curvilinear relations were found such that higher total cortisol and cortisol reactivity during the task was observed among youth reporting lower and higher frequency of IPC, suggesting that moderate IPC exposure may be associated with lower cortisol activity at a later developmental stage. © 2013 Wiley Periodicals, Inc.* Dev Psychobiol 9999: XX–XX, 2013.

Keywords: *interparental conflict; cortisol; curvilinear; stress-inoculation; young adulthood*

INTRODUCTION

Children's psychological and physiological development unfolds within the context of multiple systems, perhaps the most critical of which is the family environment. Interparental conflict (IPC) is a particularly pervasive form of family stress that has been shown to increase risk of psychosocial maladjustment (Cummings & Davies, 2002; Fosco & Grych, 2008; Hair et al., 2009; Paradis et al., 2009; Rhoades, 2008) and physiological dysregulation (Davies, Sturge-Apple, Cicchetti, & Cummings, 2007; Davies, Sturge-Apple, Cicchetti, & Cummings, 2008; Granger et al., 1998). The hypothalamic–pituitary–adrenal (HPA) axis, one of the primary mediators of the neuroendocrine stress response system, is a critical link between childhood adversity and later health. The calibration of the HPA axis is highly sensitive to environmental risks and resources; as evidenced by different levels of the hormone cortisol, the quality of early life experiences contributes to a range of alterations to HPA axis functioning (Gunnar & Quevedo, 2007; McCrory, De Brito, & Viding, 2010). Children exposed to IPC, for example, have been shown to exhibit higher basal cortisol (Davies, Sturge-Apple, Cicchetti, Manning, & Zale, 2009) and both higher (Davies et al., 2008) and lower cortisol stress responses (Davies et al., 2007; Granger et al., 1998).

Moving Beyond Linear Relations

Notably, prior studies have evaluated linear relations between conflict exposure and physiological functioning based on the assumption of a direct relation

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between exposure and maladjustment. However, meta-analyses find that the percentage of variance that IPC accounts for in youth outcomes is moderate at best (Buehler et al., 1997; Rhoades, 2008). It is plausible that IPC influences development in a curvilinear manner, such that moderate amounts of IPC exposure in childhood lead to beneficial outcomes relative to extremes in either direction. Such relations are supported by the concept of stress inoculation, which posits that moderately stressful experiences, compared to overly protective or highly stressful environments, may promote more adaptive physiological stress responses to later stressors (Lyons, Parker, Katz, & Schatzberg, 2009). Illustratively, a burgeoning literature has demonstrated a curvilinear relationship between early exposure to stress and later stress reactivity such that navigating challenging, moderate levels of stress during development inoculates against the negative impact of future stress (e.g., Ellis, Essex, & Boyce, 2005; Khoshaba & Maddi, 1999; Lyons & Parker, 2007). While inoculation hypotheses have not been directly considered within the context of childhood IPC and reactivity in young adulthood, researchers have observed that exposure to family environments that are not extreme in either direction can down-regulate reactivity in childhood (e.g., Ellis et al., 2005; Gunnar, Frenn, Wewerka, & Van Ryzin, 2009).

The Current Study

Although preliminary findings from studies examining nonlinear models of stress and reactivity are suggestive, curvilinear relations between childhood family stress and physiological stress responses at later developmental stages remain relatively unexplored. The current study addresses this gap by testing a curvilinear relation between childhood exposure to IPC and the cortisol stress response in young adulthood. We hypothesized that higher and lower self-reported childhood exposure to IPC, relative to the mean, would be associated with a greater cortisol response to and across a stressful interpersonal task. In addition, we evaluated whether the impact of past IPC was above and beyond current conditions, potential negative reporting biases, or psychological symptoms by controlling for reports of current family conflict, perceived stress, and mental health symptoms. Finally, prior research has frequently combined properties of IPC to provide a summary measure of the construct; however, distinct aspects of IPC exposure may exert unique effects on children's adjustment (Cummings & Davies, 1994). There is empirical precedent for differential effects on the mental health of youth from intact families, in particular (e.g., Wymbs, Pelham, Molina, & Gnagy, 2008). Moreover, as reviewed by Miller, Chen, and Zhou (2007) different forms of stress can result in distinct neuroendocrine response patterns. As such, we conducted analyses in which we examined intensity and frequency of IPC separately.

METHOD

Participants

The current sample is drawn from a larger study of family conflict among young adults from intact families. Participants were recruited from Introduction to Psychology courses at a large southwestern public university in the United States. Data were collected over two semesters in 2008–2009. Young adults were eligible to participate based on their responses to a large screening survey that included the conflict subscale of the *Moos Family Environment Scale* (FES-C; Moos & Moos, 1994) and basic demographic questions related to family structure. Specifically, respondents were asked to complete the FES-C in reference to their family environment prior to age 16 and were eligible if they scored in the highest or lowest quartiles of the FES-C. The original study used an extreme groups design to maximize the power to detect linear associations between family conflict (not IPC) and stress reactivity in a relatively small sample, if a relation truly existed (Kagan, Snidman, & Arus, 1998).[1] In addition, only those from continuously married families were invited to participate. This selection strategy was chosen in order to avoid potential confounding introduced by marital (divorce, separation), custodial, or family (blended family, one-parent family, multiple families) status, as well as variable exposure to interparental interactions across childhood and adolescence.

One hundred six young adults were invited to a lab session 1–3 months after completing the screening survey, at which time they again completed the FES-C in reference to their family relationships prior to 16. To increase reliability of the retrospective report on the childhood family environment, only participants who scored in the same FES-C quartile on both administrations were invited to participate in the full protocol ($n = 93$). Due to equipment failure, two of these individuals did not participate in the role-play task and

[1] This sampling methodology did not restrict the range of IPC exposure in the current sample. Past FES-C and past total IPC were only moderately correlated, $r = .53$, $p < .01$, and graphical displays of the IPC data indicate that the full range of scores are represented.

did not provide saliva samples. Demographic characteristics of the final sample ($n = 91$) are displayed in Table 1.

Procedure

Participation took place between 2:00 p.m. and 6:30 p.m. (mean starting time = 3:15 p.m., $SD = 1$ hr), in order to best capture cortisol reactivity, as recommended by Nicolson's (2008) methodological guidelines. Participants were instructed to abstain from alcohol for 24 hr before testing and exercising, eating, smoking, and drinking caffeinated beverages for 2 hr before testing. They were queried about compliance and rescheduled if they did not comply. Participants first read and signed informed consent forms. After a 20-min resting period, the first saliva sample (T1) was collected. Participants then completed a 10-min role-play task, described below. Immediately after the task (10 min after the baseline saliva sample), participants provided a second sample (T2). Twenty minutes after the end of the task, participants gave a third sample (T3). Participants then responded to all self-report questionnaires. The fourth sample (T4) was collected 40 min after the end of the task. Participants were compensated with research participation credits and debriefed prior to leaving.

Table 1. Sample Demographics and Study Variable Descriptives

Characteristic	*n* (%)
Sex	
Female	44 (51.6%)
Male	47 (48.4%)
Ethnicity	
White, non-Hispanic	54 (59.3%)
Hispanic	23 (25.3%)
African-American	6 (6.6%)
Asian	4 (4.4%)
Other	4 (4.4%)
Family income	
$0–29,999	4 (4.4%)
$30,000–44,999	8 (8.8%)
$45,000–59,999	11 (12.1%)
$60,000–79,999	16 (17.6%)
$80,000–99,999	19 (20.9%)
$100,000+	29 (31.8%)
No answer	4 (4.4%)
Characteristic	***M* (*SD*)**
Age (range 18–22)	18.7 (.97)
BMI (17–44)	23.1 (4.2)
CPIC frequency (6–18)	10.4 (4.0)
CPIC intensity (7–21)	11.5 (4.3)
ASR externalizing (1–30)	12.2 (7.0)
ASR internalizing (1–42)	14.9 (10.1)
Total mental health problems (2–67)	26.9 (14.5)
Cortisol AUCg (6.2–78)	29.9 (12.7)
Individual cortisol samples	
Baseline (1.3–20.2)	7.27 (4.06)
0 min post-task (1.8–25.4)	8.75 (4.42)
20 min post-task (1.7–34)	8.65 (5.09)
40 min post-task (2.3–41.3)	7.76 (6.03)

Note: Total mental health problems reflect the sum of the individual's internalizing and externalizing symptoms, as measured by Adult Self Report (Achenbach & Rescorla, 2003). Cortisol values are nontransformed and in nmol/L.

Role-Play Task. While there are many ways to induce a physiological stress response, Smith (2003) emphasizes the need to use "conceptually relevant laboratory stress paradigms" (p. 257). The role-play task, chosen for its high relevance for college students and salience to the experience of conflict during childhood, exposed young adults to a 10-min uncontrollable interpersonal conflict discussion modeled after a task described by Semenchuk and Larkin (1993). Immediately following the collection of the first saliva sample, participants role-played a situation in which they were trying to study for an important exam but their neighbor was playing his/her music too loud. The participant was instructed to ask his/her "neighbor" to turn down the music. The neighbor was played by a gender-matched undergraduate research assistant who maintained a neutral facial expression and followed a scripted series of responses indicating refusal to cooperate. This role-play task has been demonstrated in previous studies to result in significant physiological reactivity in young adults (Hernandez, Larkin, & Whited, 2009; Luecken, Kraft, & Hagan, 2009; Luecken & Roubinov, 2012). A repeated measures ANOVA indicated significant cortisol reactivity to the task in the current study, $F = 38.627$, $p < .001$. Of the 91 participants, 64 (70%) showed an increase in cortisol level from baseline. Assessment of participant moods pre- and post-task revealed significant increases from baseline in ratings of anger, hostility, nervous, and upset affect (p's $< .001$).

Measures

Cortisol Activity. Saliva samples, collected using the Salivette device (Sarstedt, Rommelsdorf, Germany), were frozen at 0°F and shipped to Dresden Lab Services (Dresden, Germany) for analysis. High-sensitive enzyme immunoassays were performed in duplicate to analyze free cortisol. The range of sensitivity of the assay is .4–1.0 nmol/L. The intraassay coefficient of variation is 2.5% and the average interassay coefficient of variation is 6.0%.

Childhood Exposure to Interparental Conflict (IPC). Young adults' perceptions of childhood (prior to age 16) exposure to IPC were measured with the Children's Perception of Interparental Conflict Scale (CPIC; Grych, Seid, & Fincham, 1992). Scores on the six-item Frequency (e.g., "I never saw my parents argue or disagreeing." $\alpha = .92$) and seven-item Intensity (e.g., "My parents got really mad when they argued." $\alpha = .91$) subscales were summed, with higher scores reflecting higher exposure to IPC.

Current Mental Health Problems. Internalizing and externalizing symptoms were assessed using the Adult Self-Report Form (ASR; Achenbach & Rescorla, 2003; Internalizing subscale, $\alpha = .91$; Externalizing subscale $\alpha = .83$). A summary score was created to capture the total number of mental health problems.[2]

Current Perceived Stress. Young adults reported on the degree to which they felt stressed by life situations in the past month using the 10-item Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983; $\alpha = .62$). Sample items include: "In the last month, how often have you felt that you were unable to control the important things in you life?" and "In the last month, how often have you found that you could not cope with all the things you had to do?" Responses to items ranged from 0 (*never*) to 5 (*very often*). Item responses were summed to create a total score (Range = 20–58; $M = 31.05$, $SD = 7.74$).

Current Family Environment. To assess characteristics of their current family relationships, young adults completed the nine-item Conflict subscale of the *Moos Family Environment Scale* (FES-C; Moos & Moos, 1994; e.g., "We fight a lot in our family." $\alpha = .80$). Items were rated 0 (*false*) or 1 (*true*), and the scores were summed; higher values reflected greater current family conflict (range = 0–9; $M = 2.86$, $SD = 2.44$).

Statistical Analysis

Analyses evaluated total cortisol output across the role-play task as well as the magnitude of cortisol reactivity (change from T1 to T3), both of which are theoretically meaningful aspects of cortisol response (Nicolson, 2008). Given the inclusion of higher order terms in the prediction of cortisol, two summary formulas were chosen to measure cortisol output and reactivity. For analyses of the impact of IPC on cortisol output across the task, area under the curve with respect to ground (AUCg) was computed (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003) and log-transformed to correct for deviations from normality. AUCg has been identified as valid for capturing total hormonal output during a specified period of time and correlates highly with other measures of average secretion (Fekedulegn et al., 2007). For the evaluation of reactivity, raw cortisol values were log-transformed, and residualized change scores were computed by regressing the 20-min post-task (T3) cortisol sample on the baseline cortisol sample (T1) and identified covariates. Because the cortisol response peaks approximately 20 min after exposure to stress, the T3 sample was used to best capture reactivity (change from baseline). Residualized change scores adjust for the baseline sample but avoid some of the reliability concerns with difference scores (MacKinnon, 2008). The change score was used instead of area under the curve with respect to increase (AUCi) because AUCi is not applicable to all patterns of cortisol output, particularly when it results in negative values (Fekedulegn et al., 2007). A number of factors that can influence cortisol activity were considered for inclusion as covariates in the main analyses, including time since waking (in minutes), sex, age, smoking status, caffeine use, body mass index (BMI), hormonal contraception use (Yes/No), and days since last menstrual period began; only those significantly related to cortisol were included as covariates.

Prior to the main analyses, all predictors and covariates were mean-centered for the purpose of reducing error related to multicollinearity (Aiken & West, 1991). Although variables fell within normal ranges of skewness and kurtosis, Kolmogorov–Smirnov tests revealed significant non-normality in several study variables. Raw cortisol values were inspected for outliers. None were above normal physiological range (i.e., none were >45 nmol/L). Regression diagnostics conducted to identify potential influential cases identified several cases with high externally studentized residuals (>2; Neter, Wasserman, & Kutner, 1989). To address potential undue influence from multivariate outliers and univariate non-normality, models were tested using the SAS ROBUSTREG procedure with least trimmed squares (LTS) estimation (SAS Institute, 2011). The ROBUSTREG procedure computes LTS estimates that are used to detect multivariate outliers, which are then down-weighted in the final weighted least square regression, yielding a test statistic

[2] Given that externalizing and internalizing problems may be differentially related to cortisol, we also tested the models controlling for either externalizing or internalizing symptoms. Parameter estimates and standard errors did not change and neither internalizing nor externalizing predicted total cortisol output or cortisol reactivity.

that follows a Chi-square distribution. To test the hypothesis that there would be a curvilinear relation between childhood exposure to IPC and cortisol activity in young adulthood, cortisol activity (either AUCg or the residualized change score) was regressed on IPC, IPC^2 and identified covariates, and the statistical significance of the beta associated with the quadratic term (IPC^2) was examined.

RESULTS

Preliminary Analyses

Descriptive statistics are included in Table 1. Kendall's τ correlations, a nonparametric measure appropriate for small data sets with a large number of tied ranks, indicated that BMI, smoking, age, days since beginning of last menstrual period (females only), and use of hormonal contraception (females only) were not correlated with total cortisol output (all p's $> .13$). Only time since waking ($\tau = -.24$, $p < .001$) and participant sex ($\tau = -.22$, $p = .01$) were significantly correlated with total cortisol output; therefore, they were included as covariates in models predicting AUCg and in the computation of residualized change scores. Total IPC was not associated with participant age, family income, or race/ethnicity (all p's $> .55$). Zero-order correlations between study variables are shown in Table 2. IPC frequency and intensity were moderately correlated, and both were significantly correlated with total mental health symptoms. Internalizing and externalizing symptoms, current family conflict, and current perceived stress were not significantly correlated with cortisol.

Primary Analyses

First, we tested the hypothesis that exposure to both higher and lower total IPC would be associated with higher AUCg. As predicted, robust multiple regression analyses revealed a significant curvilinear association between total IPC and AUCg, controlling for time since waking and participant sex, $B = .002$, $SE = .004$, $\chi^2 = 3.86$, $p < .05$, $R^2 = .45$ (Table 3), such that individuals who reported either low or high IPC exhibited greater AUCg relative to the reduced AUCg observed in individuals who reported moderate IPC (see Fig. 1). The relation remained significant after controlling for current family conflict and perceived stress, and neither current family conflict nor perceived stress predicted AUCg. Results also did not change when controlling for mental health symptoms, and mental health symptoms did not predict AUCg. Next, we tested whether this curvilinear relation was specific to either frequency or intensity of IPC. Multiple regression analyses revealed a significant curvilinear association between IPC frequency and AUCg, $B = .01$, $SE = .004$, $\chi^2 = 8.65$, $p = .003$, $R^2 = .43$ (Table 3), with higher AUCg evident among young adults exposed to both lower and higher IPC frequency in childhood. The relation remained significant after controlling for current family conflict, perceived stress, and mental health symptoms. The intensity of IPC did not exhibit a curvilinear ($p = .31$) or linear ($p = .25$) association with AUCg. However, when current family conflict and perceived stress were entered into the model, there was a significant positive linear association between IPC intensity and cortisol AUCg, $B = .04$, $SE = .02$, $\chi^2 = 6.15$, $p = .005$, $R^2 = .43$. This result did not change when controlling for mental health problems.

Table 2. Zero-Order Correlations Among Study Variables

	1	2	3	4	5	6	7	8
1. AUCg	1.0							
2. Reactivity	.43*	1.0						
3. CPIC-F	.03	−.02	1.0					
4. CPIC-I	.10	.04	.63*	1.0				
5. ASR-T	−.05	−.06	.30*	.24*	1.0			
6. Sex	−.22**	−.14***	−.05	−.06	.11	1.0		
7. Wake time	−.24*	−.11	−.04	−.07	−.02	.13	1.0	
8. FES-C	.01	.01	.58*	.59*	.33*	−.01	−.04	1.0
9. PSS	.02	.04	.04	.01	.11	−.12	.01	.03

Note: $n = 91$. Due to non-normality of data, Kendall's Tau correlation coefficients are reported above. AUCg = nontransformed total cortisol output. Reactivity = a residualized change score computed by regressing the third cortisol sample on the first sample. ASR-T = sum of internalizing and externalizing symptoms. Sex is coded 0 = male, 1 = female. Wake time = minutes between when the individual reported waking up and the first cortisol sample. FES-C = Conflict subscale score of Family Environment Scale measuring current family conflict. PSS = Perceived Stress Scale.

*$p < .01$.

**$p < .05$.

Table 3. Relations Between Self-Reported Frequency of Childhood Interparental Conflict and Cortisol Activity in Young Adulthood

	Cortisol AUCg				Cortisol Reactivity			
	B	*SE*	χ^2	*p*	*B*	*SE*	χ^2	*p*
Intercept	2.94	.07	1,580	<.001	−.26	.16	2.43	.12
Wake Time	−.001	.0004	8.43	.004	—	—	—	—
Sex	−.23	.09	6.32	.01	—	—	—	—
IPC-Freq	−.02	.01	1.20	.27	−.04	.03	1.54	.22
IPC-Freq^2	.01	.004	8.65	.003	.02	.01	3.88	.048
	R^2	.43	$R^2\Delta$[a]	.06	R^2	.04	$R^2\Delta$	.04

Note: Cortisol reactivity was measured by computing a residualized change score; given that wake time and sex were included in the computation of the residualized change score, they were not included again in the analysis of cortisol reactivity.

[a]The change in R^2 when IPC^2 added to the model.

Next, we tested a curvilinear relation between total IPC exposure and cortisol reactivity to the task. The curvilinear association was not significant ($p = .40$) and remained nonsignificant when current family conflict, perceived stress, or mental health symptoms were included in the model. We then examined frequency and intensity of IPC separately. Whereas IPC frequency exhibited a curvilinear relation with cortisol reactivity, $B = .02$, $SE = .01$, $\chi^2 = 3.88$, $p < .049$, $R^2 = .04$ similar to the pattern observed with AUCg (see Fig. 1), IPC intensity did not exhibit a linear ($p = .84$) or curvilinear ($p = .48$) association with reactivity. Controlling for current family conflict, perceived stress, or mental health symptoms did not change the pattern of results and these variables were not significantly related to cortisol reactivity.

DISCUSSION

Young adulthood, a developmental period of increasing risk for stress-related disorders and the emergence of psychopathology, is an opportune time to evaluate the impact of childhood exposure to IPC on patterns of cortisol reactivity later in life. Exposure to IPC has long been considered a stressor linearly related to youth well being, with minimal exposure presumed to predict optimal adjustment and greater exposure directly associated with maladjustment (Rhoades, 2008). Yet the complexity of the stress response system and preliminary evidence showing that moderate exposure to stressors can down-regulate the stress response system suggests that evaluation of more complicated relations between childhood adversity and later physiological reactivity is warranted. We hypothesized that perceived exposure to childhood IPC would be related to cortisol reactivity in young adulthood in a curvilinear manner. In line with this hypothesis, we found that young adults who reported lower *and* higher exposure to childhood IPC exhibited greater cortisol output during a stressful interpersonal task compared to youth who reported moderate levels of IPC. This finding appeared to be driven by the frequency of IPC exposure rather than intensity of exposure. To our knowledge, the current study is the first to support curvilinear relations between childhood IPC and cortisol stress reactivity in young adulthood.

Curvilinear Relation Between Childhood IPC Frequency and Later Stress Reactivity

Models of stress inoculation suggest that exposure to moderate adversity early in life may exert a "steeling" or "buffering" effect, reducing physiological and emotional responses to subsequent stressors (Lyons et al., 2009). The U-shaped relation between childhood IPC exposure and later stress reactivity found in the current study is consistent with this phenomenon. Findings from research based on both animal and human models suggest that moderate exposure to stressors may support the development of adaptive behaviors over the long term. Nonhuman primates exposed to intermittent interpersonal stress early in life exhibit a more adaptive physiological stress response and greater cognitive control of behavior in young adulthood relative to those without exposure to stress and those exposed to extreme adversity (e.g., Levine & Mody, 2003; Parker, Buckmaster, Schatzberg, & Lyons, 2004; Parker, Buckmaster, Sundlass, Schatzberg, & Lyons, 2006; Parker, Buckmaster, Lindley, Schatzberg, & Lyons, 2011). In humans, the experience of mild stressful events in childhood has been associated with decreased cardiovascular response in a laboratory stress test (Boyce & Chesterman, 1990) as well as enhanced coping with stressors in adulthood (Khoshaba & Maddi, 1999). Illustratively, Gunnar et al. (2009) demonstrated that periadolescent youth who had experi-

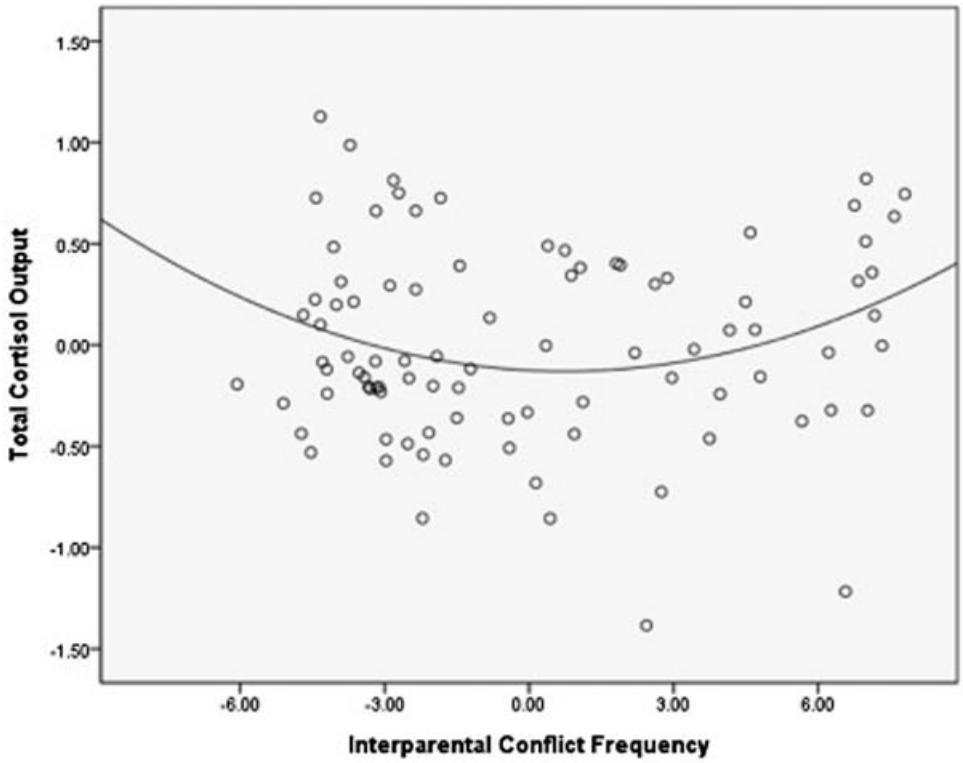

FIGURE 1 Cortisol AUCG by mean-centered frequency of conflict (quadratic fit), controlling for minutes since waking and sex.

enced early adoption and consequently moderate levels of early life stress exhibited a lower cortisol stress response compared to non-adopted children possibly reflecting a more adaptive stress response.

The curvilinear association identified in the current study is also in line with theories of differential susceptibility and conditional adaptation. The theory of differential susceptibility suggests that high reactivity associated with low and high early life stress environments increases children's susceptibility to those environmental conditions "for better or worse" (Belsky et al., 2007). Relatedly, conditional adaptation models (e.g., biological sensitivity to context, Ellis et al., 2005; adaptive calibration model, Del Giudice et al., 2011) suggest elevated physiological reactivity develops within both stressful and supportive childhood environments to provide a match to the prevailing conditions (Del Giudice et al., 2011; Ellis et al., 2005). Such patterns of biological reactivity have been proposed to facilitate children's effective responses to challenges in adverse environments and promote children's use of the resources available to them in supportive environments. Interpreted through the lens of conditional adaptation, the current findings suggest that elevated cortisol activity may have emerged within conditions of lower and higher IPC exposure as a means toward successful navigation of these environments. Low levels of IPC, however, do not necessarily constitute the existence of an exceptionally supportive environment, thus we are careful not to overspeculate.

Linear Relation Between Childhood IPC Intensity and Later Stress Reactivity

When distinct aspects of IPC were examined, the curvilinear relation between IPC exposure and cortisol activity was only observed for the measure of frequency, whereas intensity of IPC was positively associated with total cortisol output when current family conflict and perceived stress were also controlled for. Although not hypothesized, this result is consistent with recent accounts of different types of conflict leading to a variety of cortisol patterns in youth (e.g., Koss et al., 2013). It is also in line with the phenomenon of resonant frequency observed in other biological systems (e.g., stimulation of the cardiovascular system at an ideal rate to maximize heart rate variability), which demonstrates that a particular frequency of exposure, rather than intensity of a stimulus, has a beneficial effect (Vaschillo, Vaschillo, & Lehrer, 2006). Interpar-

ental conflict can vary in its intensity and frequency such that stress within the family environment may be more acute (time-limited periods of intense conflict) or chronic (conflict that occurs frequently) in nature. It has been demonstrated that children are sensitive to the differences between these two characteristics of conflict (Grych & Fincham, 1990), and among intact families there is evidence that frequency of IPC, compared to conflict intensity, leads to differential outcomes in youth (Wymbs et al., 2008). Distinguishing between frequent and intense conflict is also relevant for cortisol regulation given that childhood exposure to stressors can uniquely alter neuroendocrine regulation and reactivity to new stressors depending on time course or characteristics of the stressor (Hagan et al., 2010; Miller et al., 2007). The current findings suggest that the unique influences of specific properties of IPC on HPA axis functioning are worthy of attention in future research.

Limitations

Limitations of the current study should be considered when interpreting the results. First, participants were students from intact married families. As such, the range of adversity experiences may have been restricted in the current sample, and results may not generalize to broader community samples or young adults who experienced parental divorce, parental death, or severe interparental aggression during childhood. Relatedly, although reflective of the geographic area from which it was drawn, the study sample was predominately White (59%) or Hispanic (25%), limiting generalizability of results to other cultural groups. Second, participants were asked to retrospectively recall experiences of IPC. Although we cannot determine participants' "true" exposure, their continuing perceptions may be a more powerful indicator of psychobiological reactivity than observer, parent, or childhood accounts, which suffer from their own limitations. Importantly, recent research suggests measurement error associated with childhood retrospective reports does not significantly affect estimates of models that examine the long-term impact of early adversity (Fergusson, Horwood, & Boden, 2011). As additional controls, we only included participants whose reports of characteristics of the childhood family environment were consistent across two administrations of the FES scales. To minimize concerns regarding negativity biases (e.g., Krueger, Markon, & Bouchard, 2003) or confounding by current experiences, we confirmed that results held after controlling for current mental health symptoms, current family conflict, and perceived life stress.

Third, the cross-sectional design precludes conclusions about causality, and we cannot determine when these patterns of cortisol reactivity emerged or their stability across the lifespan. The development of physiological reactivity is a dynamic process that unfolds over time and in response to the variety of environmental and social contexts to which children are exposed (Obradovíc, Bush, & Boyce, 2011). Intervening factors not evaluated in the current study (e.g., economic hardship, child maltreatment, peer influences, parenting or the parent–child relationship) may have contributed to the observed patterns of cortisol activity. Future studies that test mediators of the impact of childhood family experiences on adult HPA activity are critical for advancement of theory and the development of effective interventions. Finally, the present study did not evaluate potential moderators such as gender or socioeconomic status, which would be important considerations for future studies with larger sample sizes. Although meta-analyses have not found significant gender differences with regard to relations between IPC and psychological adjustment (e.g., Buehler et al., 1997; Rhoades, 2008), there is theoretical and empirical precedent for finding distinct gender effects of adversity on physiological reactivity (e.g., Del Giudice et al., 2011).

Despite these limitations, the current study makes a substantial contribution to the growing literature on the neurobiological correlates of exposure to family stressors in childhood. The majority of existing studies have examined linear relations between childhood experiences and physiological systems, however the presumption of a linear association may mask more complex relationships between the family environment and later stress reactivity (Krause, 1995). Indeed, different childhood experiences have been linked to exaggerated as well as blunted stress reactivity. Although the potential for nonlinear relations between the family environment and physiological activity is gaining interest, previous investigations have primarily examined effects during childhood and focused on broader measures of childhood adversity or sympathetic/parasympathetic responses in relation to marital conflict in childhood (e.g., El-Sheikh, Harger, & Whitson, 2001; El-Sheikh, Keller, & Erath, 2007). The present study extends this literature by examining a specific form of stress in the childhood environment, interparental conflict, and HPA activity in young adulthood. Furthermore, results underscore the importance of examining individual patterns of stress responsivity within a context-dependent framework.

CONCLUSION

The current findings indicate that a moderate level of exposure to IPC during childhood may down-regulate the stress response in young adulthood, whereas either very limited conflict exposure or frequent IPC may lead to similar patterns of heightened reactivity. Parents engaging in moderate levels of conflict may have other characteristics that further benefit the developing stress response system, such as increased warmth, or may provide models for resolving conflict, which might be less likely in families with no conflict or very frequent conflict. Collectively, the current results highlight the dynamic interplay between childhood environmental conditions and individual biological factors that shape the physiological stress response and suggest future directions in the study of nonlinear relations between early childhood environments and HPA activity in young adulthood.

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