Conflict Appraisals and Trauma Symptoms in Children Exposed to Intimate Partner Violence

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Children exposed to intimate partner violence (IPV) are at risk for posttraumatic stress symptoms (PTSS) and alterations in hypothalamic-pituitary-adrenal (HPA) axis functioning. The current study investigated children's threat and self-blame appraisals about parental conflict as potential mechanisms leading to these adverse outcomes. Parent-child relationships were also examined. The sample consisted of 119 10-year-olds and their mothers who were recruited from the community as part of a larger study of IPV. Children's reports of IPV directed at their mother in the past year were not associated with PTSS; however, IPV exposure was also associated with greater threat appraisals and poorer quality parent-child relationships. These results provide further evidence that witnessing IPV is threatening for children, has negative consequences for parent-child relationships, and can impact children's HPA axis functioning.

Keywords: domestic violence; cortisol; HPA axis; PTSD

Exposure to intimate partner violence (IPV) directed towards a parent is a prevalent and often chronic form of child maltreatment. According to the National Survey of Children's Exposure to Violence, 7.9% of children ages 10–13 were exposed to IPV during the past year, and 17.9% during their lifetimes (Hamby et al., 2011). Exposure to IPV in childhood confers increased risk for a variety of adverse health outcomes, including post-traumatic stress symptoms (PTSS) (e.g., Evans et al., 2008) and alterations in physiological stress response (De Bellis & Zisk, 2014). Unfortunately, the mechanisms through which these outcomes emerge are still poorly understood. There is some research to suggest that children's appraisals about parental conflict are important

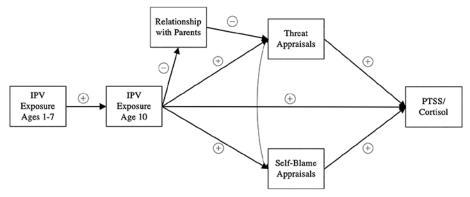


FIGURE 1. Conceptual model depicting hypothesized associations among children's exposure to adult IPV, children's appraisals about conflict and parent–child relationships, and trauma sequalae (i.e., posttraumatic stress symptoms and cortisol output).

factors in determining children's immediate behavioral and physiological responses to conflict, as well as distal mental health and adjustment outcomes (Grych & Fincham, 1990; Davies & Cummings, 1994). However, research linking appraisals to adjustment outcomes generally focuses on broad bands of child behavior, such as internalizing and externalizing problems, rather than on trauma symptoms, specifically. Because appraisals of traumatic events are also thought to play a role in the development and maintenance of PTSS (Ehlers & Clark, 2000), the present study investigated whether school-aged children's threat and self-blame appraisals about adult conflict mediated the association between IPV exposure and PTSS, and between IPV exposure and physiological stress response (Figure 1).

Trauma Symptoms Result From Exposure to IPV

Children exposed to IPV are vulnerable to developing posttraumatic stress disorder (PSTD) (e.g., Graham-Bermann et al., 2012; Koolick et al., 2016; Levendosky et al., 2013). However, reported rates of PTSD in IPV-exposed children vary widely between studies, due in part to differences in sample characteristics (e.g., shelter vs. community samples), the measures used to assess symptoms, and the fact that some children are resilient even in the face of exposure to IPV. However, even when the threshold for a PTSD diagnosis is not met, clinically significant symptoms are often present that can have far-reaching consequences for children. For example, children's PTSS are associated with both internalizing and externalizing behavioral problems (e.g., Milot et al., 2010), and among adolescents, PTSS mediate the association between family violence exposure and poor school functioning (McGill et al., 2014). In addition, PTSS are predictive of children's physical health problems over and above the contributions of violence exposure and demographic risk (Graham-Bermann & Seng, 2005); this may reflect alterations in neuroendocrine or immune function as a result of chronic stress. Because positive associations between IPV exposure and childhood trauma symptoms have previously been reported in the literature (e.g., Evans et al., 2008), we sought to examine this association in the present sample.

Exposure to IPV Affects the HPA Axis

Childhood trauma not only affects the emotional response of an individual (e.g., PTSS or PTSD), but it can also alter stress physiology, including dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis (e.g., De Bellis & Zisk, 2014). In healthy individuals, activation of the HPA axis in response to a physical or psychological stressor results in a tonic release of cortisol, which facilitates the mobilization of energy among other adaptations aimed at helping the individual respond to the challenge. When the challenge is met or the threat is resolved, cortisol, which is self-regulating via negative feedback mechanisms, returns to baseline levels. Research findings regarding the link between violence exposure and children's HPA axis functioning have been mixed. Increased cortisol reactivity to a laboratory stressor has been observed in children exposed to IPV (Saltzman et al., 2005) and community violence (Peckins et al., 2012). However, there is also evidence of attenuated physiological stress reactivity in children exposed to IPV (Sturge-Apple et al., 2012; Saxbe et al., 2012; Cordero et al., 2017). This blunting of cortisol secretion may reflect the eventual down-regulation of a chronically aroused system, such that over time individuals dealing with chronic stressors in their immediate environments will transition from a highly reactive to an inhibited response due to enhanced negative feedback inhibition of the HPA axis or other mechanisms (Gunnar & Vasquez, 2001). The present study aims to build on this literature by examining the relationship between recent IPV exposure and children's cortisol output in response to an acute social stressor.

Children's Appraisals of Adult Conflict and IPV

While it is clear that PTSS are an adverse outcome commonly associated with IPV exposure, the mechanisms through which some children develop PTSS in the context of IPV are still not well understood. The ways that individuals interpret potentially traumatic events are thought to have consequences for their subsequent psychological adjustment and wellbeing. For example, the cognitive model of PTSD posits that maladaptive appraisals about the traumatic event and its consequences serve to maintain the persistent sense of current threat that characterizes the diagnosis and inhibit the processing of traumatic memories (Ehlers & Clark, 2000; Meiser-Stedman, 2002). Children's appraisals about interparental conflict are also thought to be associated with child adjustment outcomes, with particular emphasis placed on the role of children's perceptions of threat and attributions of selfblame (Grych & Fincham, 1990). In both IPV and non-violent samples, research finds that school-aged children's appraisals of threat (i.e., how personally threatening is the conflict) and self-blame (i.e., how much they are to blame for the conflict) mediate the association between exposure to interparental conflict and children's internalizing problems (e.g., Gerard et al., 2005; Grych et al., 2000). However, measuring only broad bands of behavior does not address one of the primary outcomes associated with exposure to IPV, namely PTSS. As of yet there is relatively little research examining whether children's appraisals of IPV influence the development of PTSS, specifically, however, some investigators have pointed to children's threat processing, personalization, and feelings of security/insecurity within the parent-child relationship as potential mediators of trauma-related psychopathology (Fosco et al., 2007; McLaughlin & Lambert, 2017).

For children exposed to IPV, parental conflict may be perceived as highly threatening as it carries the potential to cause harm to important attachment figures and exceed children's abilities to cope effectively. Generalized threat appraisals in response to signs of parental conflict may be a salient pathway leading to PTSS. Self-blaming attributions, which are related to feelings of guilt and shame, have been associated with the development of PTSD in individuals exposed to IPV and other forms of child maltreatment (e.g., Lehmann, 1997; Filipas & Ullman, 2006). The only study to test whether threat and self-blame appraisals mediated the relationship between IPV and PTSS failed to find a significant effect (Kilpatrick & Williams, 1998). However, these researchers used a small sample size and a single item to assess each appraisal, perhaps limiting their ability to detect the hypothesized relationships.

Likewise, few studies have examined the associations among IPV, appraisals, and physiological arousal. However, we might hypothesize that children's appraisals about IPV influence their physiological arousal in response to the events. If witnessing violence against a parent is accompanied by an overwhelming sense of threat and self-blaming attributions, which heighten the personal salience of the events, high levels of physiological arousal could be expected. For example, one study found that among 8-11-year-old children exposed to high levels of marital conflict, those who endorsed high levels of threat and self-blame appraisals demonstrated increased cardiovascular reactivity in response to a conflict-related psychosocial stressor (El-Sheik & Harger, 2001). With regard to the HPA axis, (Koss et al., 2013) found that in a community sample of first- and second-graders (not specifically selected for IPV exposure), threat appraisals were associated with a rising pattern of cortisol secretion in response to a staged marital dispute. However, an attenuated profile of cortisol reactivity was also observed and was associated with parents' use of destructive conflict tactics, leaving open the possibility that the eventual downregulation of the HPA axis may result from repeated exposure to hostile interactions between parental figures. In the present research, children's threat and self-blame appraisals will be tested as potential mediators of both PTSS and cortisol.

Parent–Child Relationships

Family-level factors also influence children's adjustment in the face of traumatic events. The spillover hypothesis suggests that destructive conflict in the parental relationship will have a negative impact on parent-child relationships as a result of parents being less emotionally available, less sensitive to children's needs, and less effective in their parenting practices (Erel & Burman, 1995). This spillover effect may be particularly harmful for children exposed to IPV, as evidenced by the high rates of co-occurrence among IPV exposure, harsh parenting practices, and child maltreatment (Grasso et al., 2016). In addition, mothers coping with IPV are likely to be depressed or to have PTSS themselves, which can negatively influence their ability to engage in sensitive parenting (Greene et al., 2018). If a child is not able to count on their parent to be a secure emotional base, either because the parent is emotionally unavailable or actively menacing, the child may feel even more threatened and less able to regulate when witnessing violence in the home (e.g., Boeckel et al., 2017). Therefore, parent-child relationship quality will be tested as a mediator of the effect of IPV exposure on children's threat appraisals in the present study.

The Present Study

As stated above, the present study focused on understanding whether threat and self-blame appraisals mediate the relationship between IPV and child PTSS as well as challenged cortisol output (see Figure 1). Data were drawn from a larger longitudinal study of IPV (see Bogat et al., 1999; Levendosky et al., 2000). Information about mothers' experiences of IPV victimization were collected annually when children were ages 1–7. When

children were 10 years old, an in-person follow up assessment took place, during which children reported on their experiences of witnessing IPV in the past year, their perceptions about adult conflict in the household, and the quality of their relationships with caregivers. Children also participated in a standardized laboratory stress task consisting of public speaking and mental arithmetic (Buske-Kirschbaum et al., 1997). Children provided saliva samples before and after this task to be assayed for cortisol. At the visit mothers also provided information about children's socioemotional functioning, including PTSS. We tested the following research questions: (1) Does past year IPV exposure positively predict children's PTSS and challenged cortisol production at age 10? (2) Are associations between IPV exposure and children's PTSS and cortisol production mediated by children's threat and self-blame appraisals? (3) Is the association between IPV exposure and children's threat appraisals mediated by parent-child relationship quality?

METHOD

Participants

Participants were 119 mother-child dyads, representing a subsample of the 206 women who were initially recruited during the third trimester of pregnancy to participate in a longitudinal study of IPV. Women were recruited from mid-Michigan communities—mainly small and medium sized towns and cities. They were oversampled for experiences of IPV such that half of the sample endorsed physical IPV during pregnancy. Participants were eligible for the study if they were 18–40 years of age, involved with a male romantic partner for at least 6 weeks during pregnancy, and English speaking. After the initial pregnancy interview, women were contacted annually at the child's first through seventh birthdays for follow-up assessments. When children were 10 years old, attempts to re-contact the original participants were made. Only the 119 mother-child dyads who participated in the 10-year assessment were included in the present analyses.

The 119 dyads did not differ from those who were part of the original longitudinal study on any demographic variables except monthly income. Non-participants had significantly lower monthly family income at the intake assessment, F(1, 203) = 4.12, p = .04. Re-contacted children included 65 boys and 54 girls, mean age 10 years and 6 months (SD = 3 months). The children's races were 50% White, 23% African American, 23% multiracial, 2% Latino, 1% Native American, and 1% Asian American. Mean monthly family income was \$3,196 (SD = \$2,805), and 46% reported that they were receiving supplemental nutrition assistance. Of the mothers, 11% did not complete high school, 28% completed high school, 42% had some college or trade school, 11% had a bachelor's degree, and 6% had some graduate school or a graduate degree. Relationship status of the mothers was 53% married, 24% never married, and 23% separated, divorced, or widowed.

Measures

Age 10 IPV Exposure. Children completed a 31-item version of the Revised Conflict Tactics Scale (CTS2; Straus et al., 1996) which assessed the frequency of psychological and physical aggression toward their mother in the past year. The negotiation subscale was not included in the total score. Example items include "father insulted or swore at mother" and "father slammed mother against a wall." Children rated each item on a 7-point scale ranging from 0 = This has never happened to 6 = More than 20 times in the past year, and

endorsements were summed. The scale has good internal consistency (.79 to .95; Straus et al., 1996); it was also good in the present sample ($\alpha = .84$).

Past IPV. The 46-item Severity of Violence Against Women Scale (SVAWS; Marshall, 1992) assesses physical, psychological, and sexual IPV experiences of women. Example items include "Threated to hurt you" and "Beat you up." Women rated each item on a 4-point scale ranging from 0 = never to 3 = many times. Mothers completed the SVAWS annually when children were ages 1–7 and coefficient alphas ranged from .91 to .99. The frequency of IPV at each age was summed to produce the past IPV variable.

Parent–Child Relationship Quality. The 9-item Relations with Parents subscale from the BASC-2 Self-Report of Personality (SRP; Reynolds & Kamphaus, 2002) was administered. It assesses positive regard toward parents and feeling valued by them. Statements such as "My parents are proud of me" were rated on a 4-point scale (0 = Never to 3 = Almost Always) and the answers were summed. The scale demonstrated good internal consistency in the present sample ($\alpha = 0.80$).

Appraisals of Interparental Conflict. The Children's Perceptions of Interparental Conflict Scale (CPIC; Grych, Seid & Fincham, 1992) assesses marital conflict from the child's perspective. The subscales used in this study, threat and self-blame, have good internal consistency and test-retest reliability (Grych et al., 1992). The threat subscale includes items such as "I get scared when my parents argue" and the self-blame subscale includes items such as "Even if they don't say it, I know I'm to blame when my parents argue." Children rated each item on a 3-point scale (0 = True, 1 = Sort of True, and 2 = False). Items were reverse coded such that higher scores indicated higher levels of threat and self-blame, and responses were averaged to produce the subscales. In the current sample, the threat subscale demonstrated good internal consistency ($\alpha = .84$), as did the self-blame subscale ($\alpha = .74$) after two poorly performing items were eliminated.

Posttraumatic Stress Symptoms. Children's posttraumatic stress symptoms were measured via the PTSD/Dissociation subscale derived from the Child Behavior Checklist (CBCL 6-18; Achenback & Rescorla, 2001). The PTSD/Dissociation subscale, developed by Sim and colleagues (2005) and further validated by Milot et al. (2013), has demonstrated construct validity via strong associations with the Trauma Symptoms Checklist for Young Children and the Child Dissociative Checklist (Milot et al., 2013). The scale consists of the sum of 16 items from the CBCL that experts rated as representative of trauma symptoms. Example items include "Nervous, high strung, or tense," and "Nightmares." Mothers rated how true each item was for their child during the past 6 months (0 = Not True, 1 = Somewhat or Sometimes True, or 2 = Very True or Often True). The scale has demonstrated strong internal consistency ($\alpha = .80$; Milot et al., 2013), as it did in the present sample ($\alpha = .83$).

Salivary Cortisol and Experimental Manipulation. The Trier Social Stress Test for Children (TSST-C) is a standardized laboratory stress task shown to elicit a reliable physiological stress response in children ages 9 to 14 (Buske-Kirschbaum et al., 1997). In this task, the child is given 5 minutes in which to prepare an exciting ending to a story. They are told their presentation must last for 5 minutes and their performance will be videotaped and reviewed by a judge. After the story task, the child is immediately asked to serially subtract the number 7 from 758 as fast as possible and is told to start over from the beginning if a mistake is made. The task is discontinued after four mistakes. Children are then debriefed and given positive feedback about their performance. In our research, the task was perceived as stressful by most children (M = 3.24, SD = 1.05 on a 1–5 scale).

HPA axis functioning was measured by salivary cortisol. Saliva samples of 2 ml were obtained from the children using the passive drool method. The baseline sample was obtained 20 minutes after arrival at the project offices. The second and third samples were taken 20 and 40 minutes after the TSST-C. Samples were frozen and then shipped to Salimetrics, LLC for duplicate assays. The range of detection is $0.003-3.0 \mu g/dl$, and the inter-assay and intra-assay coefficients of variability were less than 10% and 15%, respectively. Area under the curve with respect to ground (AUCg), a measure of total cortisol output capturing both starting levels and change in response to the challenge, was used in the present analyses (Khoury et al., 2015). Cortisol values were log transformed prior to calculating area under the curve to reduce skew.

Procedure

As cortisol secretion follows a circadian rhythm, the time of day of the assessment was standardized across participants. All study visits commenced between 4:00 and 5:00 pm, this time was chosen to best accommodate participant's school/work hours. Upon arrival at the project offices, mothers and children completed informed consent and informed assent procedures together, and then were brought to separate rooms for the various assessments.

Data Analytic Strategy

In order to test study hypotheses, two path analysis models were fit in Mplus version 8 (Muthén & Muthén, 1998–2017): one with PTSS as the outcome, the other with children's cortisol production (Hayes, 2018). Missing data were handled using full information maximum likelihood estimation (Enders & Bandalos, 2001). Model fit was assessed using multiple indices including the chi-square index, the comparative fit index (CFI) and the root mean square error of approximation (RMSEA). CFI values above .90 and RMSEA values below 0.08 indicate acceptable model fit (MacCallum et al., 1996). In order to test our mediation hypotheses, direct and indirect effects were tested using the percentile bootstrap method with 1,000 resamples to produce 95% confidence intervals around the product coefficients (Falk, 2018); effects are considered significant if the confidence interval does not contain zero.

RESULTS

Descriptive Statistics and Bivariate Associations

Means, standard deviations, and bivariate correlations among study variables are presented in Table 1. On average, women reported 26 incidents of domestic violence during the first seven years of their child's life, and children reported about 4 IPV incidents during the past year, with much variation (SD = 50.94 and 6.46 respectively). Seventy percent (n = 79) of mothers reported having experienced physical, psychological, or sexual IPV in at least one prior wave of the study, with 40.2 % (n = 45) reporting having experienced IPV at 3 or more waves. Of the 96 children who completed the Revised Conflict Tactics Scale, 59.4% (n = 57) reported at least one incident of IPV perpetrated against their mother in the past year. Of these, 28.1% (n = 16) endorsed incidents of both physical and psychological IPV, while the remaining 71.9% (n = 41) endorsed psychological aggression only. With regard

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	1.	2.	3.	4.	5.	6.	7.
1. Past IPV	-						
2. Age 10 IPV	.420**	-					
3. Threat	.206*	.400**	-				
4. Self-Blame	.068	.194	.226*	-			
5. Parent–Child Relationship	260**	268**	265**	.038	-		
6. PTSS	.093	.005	.161	.172	109	-	
7. Cortisol (AUCg)	069	185	096	072	.004	014	-
Valid N	119	96	97	97	100	119	99
Mean	25.83	4.32	.62	.12	21.92	3.21	4.5
SD	50.94	6.46	.47	.24	4.54	3.82	3.21
Minimum	0	0	0	0	9	0	.83
Maximum	335	36	2	1.43	27	25	17.76
n < 0.05 $n < 0.01$							

 TABLE 1.
 Intercorrelations and Descriptive Statistics of Study Variables

 $p^* < 0.05$. $p^* < 0.01$.

to PTSS, while the majority endorsed low levels of symptoms, 27.7% (n = 33) of mothers reported that their child was exhibiting four or more symptoms.

IPV Exposure and PTSS

The first model examined whether children's appraisals about parent's conflict mediated the association between IPV exposure and PTSS (see Table 2 for unstandardized parameter estimates; standardized estimates are presented in text). The model fit was good, $\chi^2(6) = 5.64$, p = 0.47, CFI = 1.00, TLI = 1.02, RMSEA < .01. There was a significant positive association between mothers' past IPV experiences and children's reports of IPV at age 10 (b = .41, p < .01). Age 10 IPV was significantly positively associated with threat appraisals (b = .35, p < .01), and negatively associated with parent–child relationship quality (b = -.27, p < .01), such that children who reported more frequent exposure to parental IPV over the past year reported worse relationships with parents. The association between age 10 IPV exposure and self-blame was nonsignificant (b = .20, p = .10), as was the association between IPV exposure and PTSS (b = -.09, p = .36).

With regard to the first hypothesis, the direct effect of IPV exposure on children's PTSS was estimated to lie between –.29 and .11 with 95% confidence, indicating that it was not significantly different from zero at the .05 level. This suggests that contrary to our expectations, past year IPV exposure did not predict increased PTSS. The indirect effect of recent IPV exposure on PTSS through children's threat appraisals was estimated to lie between –.03 and .18 with 95% confidence, suggesting that the indirect effect was also not significantly different from zero at the .05 level. Therefore, there is no evidence that children's threat appraisals mediate the association between IPV exposure and PTSS. The indirect effect of recent IPV exposure on PTSS through self-blame was estimated to lie between –.02 and .12 with 95% confidence, again suggesting that PTSS were not significantly mediated by children's self-blame appraisals. There was however a significant direct effect of IPV exposure on children's threat appraisals, 95% CI [.16, .53], as well

TABLE 2. Unstandaruzeu 1 atri Coefficients								
Model 1 - PTSS	Estimate	SE	р					
Ages $1-7 \rightarrow IPV$ Age 10 IPV	.057	.015	<.001**					
Age 10 IPV \rightarrow Threat	.026	.008	.001**					
Age 10 IPV \rightarrow Self-blame	.007	.004	.084					
Age 10 IPV \rightarrow Relationship with parents	189	.059	.001**					
Age 10 IPV \rightarrow PTSS	051	.061	.407					
Relationship with Parents \rightarrow threat	.026	.008	.001**					
Threat \rightarrow PTSS	1.532	1.177	.193					
Self-blame \rightarrow PTSS	2.787	2.130	.191					
Threat with self-blame	.019	.015	.209					
Model 2 - Cortisol	Estimate	SE	р					
Ages $1-7 \rightarrow IPV$ Age 10 IPV	.057	.015	<.001**					
Age 10 IPV \rightarrow Threat	.025	.008	.001**					
Age 10 IPV \rightarrow Self-blame	.007	.004	.094					
Age 10 IPV \rightarrow Relationship with parents	188	.058	.001**					
Age 10 IPV \rightarrow Cortisol	009	.005	.097					
Relationship with parents \rightarrow Threat	019	.009	.031*					
Threat \rightarrow Cortisol	.039	.055	.478					
Self-blame Cortisol	002	.115	.984					
Threat with self-blame	.018	.014	.198					

TABLE 2. Unstandardized Path Coefficients

Note. IPV = intimate partner violence; PTSS = posttraumatic stress symptoms. ${}^{*}p < 0.05$. ${}^{**}p < 0.01$.

as a significant indirect effect through relationship with parents, 95% CI [.01, .11], such that that parent–child relationship quality partially mediated the association between IPV exposure and children's threat appraisals. The sequential indirect effect of IPV exposure on PTSS through relationship with parents and threat appraisals was nonsignificant, 95% CI [.00, .03].

IPV Exposure and Challenged Cortisol

The second path analysis examined IPV exposure and children's total cortisol production in response to the Trier Social Stress Test. The model was a good fit to the data, $\chi^2(6) =$ 4.66, p = 0.59, CFI = 1.00, TLI = 1.07, RMSEA <.01. Using percentile bootstrapping, the direct effect of IPV exposure on cortisol area under the curve was estimated to lie between -.41 and -.01 with 95% confidence, suggesting a significant effect at the .05 level such that more frequent exposure to parental IPV within the last year was associated with lower levels of cortisol output in response to the stress task. (The bootstrapped confidence interval of the direct effect is considered to be more accurate than the path estimate, which was only approaching significance, [b = -.20, p = .05], because it takes into account any nonnormality in the sampling distribution.) Tests of specific indirect effects revealed that the effect of recent IPV exposure on children's cortisol output was not significantly mediated by children's threat, 95% CI [-.04, .10], nor self-blame appraisals, 95% CI [-.05, .05].

DISCUSSION

The present research sought to extend our understanding of the influence that children's appraisals about IPV have on behavioral and physiological outcomes associated with trauma. Our findings indicate that children exposed to more IPV within the past year tend to display lower levels of cortisol output in response to acute social stressors. In addition, IPV exposure was positively associated with children's threat appraisals about parents' conflict, and this association was partially mediated by children's perceptions of having poorer-quality relationships with their parents. Contrary to our expectations, there was no evidence that children's threat or self-blame appraisals mediated the association between IPV exposure and either cortisol or PTSS. Our inability to detect the latter relationship may have been hampered by the fact that IPV was not associated with PTSS in the present sample. Overall, our findings suggest that although exposure to IPV directed at one's mother is experienced as frightening and threatens children's sense of personal safety, these perceptions in middle childhood do not necessarily contribute to the development of PTSS or changes in the physiological stress response.

A wealth of prior research, including previous findings with the present sample, indicate that IPV exposed children are at increased risk for internalizing and externalizing problems (e.g., Bair-Merritt et al., 2015; Martinez-Torteya et al., 2016). There is less research examining the relationship between IPV exposure and childhood trauma symptoms, but that which does exist typically (though not universally, see Lamers-Winkelman et al., 2012 and Telman et al., 2016) finds a positive association between IPV and PTSS. For example, in a meta-analysis by Evans et al. (2008), a large effect size was found for the association between IPV exposure and childhood trauma symptoms, although these results were based on only six studies. We found no evidence for this association. There are multiple possible reasons for the discrepancy between our findings and prior research. First, as the children in the present sample primarily reported exposure to verbal aggression between parents, it is possible that the violence experienced in this community sample was not severe enough to result in PTSS. However, psychological and physical aggression are highly correlated in our sample and in others, and both have the potential to be overwhelming and traumatic to children, especially considering that many of the children in our study were exposed to physical IPV prior to age 10. Differences may also be due to the measures used to assess PTSS. The PTSD/Dissociation subscale of the CBCL used in the present study does not require the identification of an index trauma, and therefore may not capture the construct with optimal specificity. In addition, mothers may not be accurate reporters of children's PTSS in middle childhood. For example, there is some literature to suggest that mother's reports of children's trauma symptoms are more associated with mother's own PTSS than with children's self-reports of PTSS (e.g., Valentino et al., 2010). Finally, an alternative explanation for these null findings is that PTSS in this sample were driven primarily by traumatic events not included in the model, such as experiences of direct victimization or exposure to community violence. Given what is known about the prevalence of polyvictimization (Hamby et al., 2010), confounding traumatic experiences cannot be ruled out.

The hypothesis that IPV exposure would be associated with children's challenged cortisol was supported, but the direction of the effect was opposite to what we had anticipated. However, reduced HPA axis responding to acute stressors in trauma-exposed children has been previously reported in the literature (e.g., Cordero et al., 2017; Saxbe et al., 2012; Sturge-Apple et al., 2012). This blunting of HPA axis reactivity may represent an adaptation to living in a chronically stressful environment that mitigates the harmful effects of prolonged exposure to high levels of glucocorticoids (e.g., Fries et al., 2005; Gunnar & Vazquez, 2001). Our findings are consistent with those of Saxbe et al. (2012) who found that, among adolescents, higher levels of family aggression, on average, predicted blunted physiological reactivity to a conflict discussion with parents. They also observed that those with high levels of family aggression and high cortisol reactivity were at the greatest risk of PTSS, a finding that could help explain why, in the present sample, blunted cortisol output was observed alongside a null association between IPV exposure and PTSS. While this finding supports the idea that hypocortisolism following childhood adversity may confer resilience to future stressors (e.g., Gunnar et al., 2009), blunted HPA axis reactivity has also been linked to greater socioemotional difficulties in maltreated youth (Ouellet-Morin et al., 2011). It is important to note that our analyses do not speak to possibility of subgroups of children who may exhibit higher levels of cortisol reactivity subsequent to IPV. The fact that other researchers report such subgroups suggests that there may be wide-ranging individual differences in children's emotional and physiological responses to IPV (cf. Davies & Martin, 2014), something that clinicians who work with IPV-exposed children should keep in mind. More research is needed to understand the functional consequences of both sensitized and attenuated profiles of HPA axis responding in IPV-exposed children.

Regarding the role of appraisals, the present study provides further evidence that exposure to IPV is associated with increased perceptions of threat to self. That is, children who were exposed to IPV were more likely to report that they are frightened by parents' arguments and that they lack confidence in their ability to cope when arguments occur. Self-blame on the other hand was not significantly associated with recent IPV exposure, perhaps indicating that by middle childhood, children are more apt to attribute blame accurately to perpetrators rather than themselves. Indeed, Jouriles and colleagues (2000) found that younger children (ages 8-9) endorsed significantly more self-blame than did older children (ages 10-12). Furthermore, there was no evidence for the hypothesized indirect effects of IPV exposure on PTSS through children's threat and self-blame appraisals. The present findings replicate the results of Kilpatrick & Williams (1998), which also failed to find support for the mediating role of self-blame and threat appraisals on PTSD in a community sample of 6- to 12-year-olds. Their results and ours suggest that children's appraisals about IPV do not influence PTSS as would be predicted by the cognitive model of PTSD. Children's appraisals also did not mediate the association between IPV exposure and decreased HPA axis output in response to a social stressor. While it is possible that appraisals of threat and/or self-blame about parental conflict influence cortisol mobilization in response to IPV situations specifically, rather than to generic social stressors like the Trier Social Stress Test (see Davies et al., 2008), our results indicate that children's appraisals of threat salience and attributions of blame do not explain the effect of IPV exposure on blunted HPA axis functioning.

As expected, IPV exposure was associated with poorer quality parent-child relationships. This is likely due to a combination of direct and indirect insults that IPV incurs. For example, child witnesses of IPV are themselves at risk of becoming targets of violence. In addition, the toll of IPV on maternal parenting stress and mental health might render mothers less emotionally available to children and more likely to use negative parenting practices (e.g., Green et al., 2018). Worse child-rated relationships with parents partially mediated the association between IPV exposure and children's threat appraisals. These findings lend credence to the idea that IPV can be detrimental to children's sense of security within the parent–child subsystem, which in turn can lead them to feel more vulnerable and less able to regulate in the face of family conflict. This finding highlights the need for interventions with at-risk children and families that focus on supporting positive parent–child relationships.

The results should be considered in the context of several limitations. Because we could not run latent variable models, we cannot assume that all variables included in the path analyses are free from measurement error. Another limitation is that we did not have children's ratings of PTSS, which by middle childhood may be more accurate than parents' reports. Additionally, exposure to traumatic events other than IPV (e.g., community violence, child abuse/neglect) were not assessed. Future research should strive to assess multiple types of potentially traumatic events throughout the child's life. Finally, protective factors were also not included in our model. There may be subgroups of children whose negative outcomes were mitigated by such factors. This is an important direction for future research.

The present research also had many strengths. First, the inclusion of a racially and socioeconomically diverse community sample enhanced the generalizability and ecological validity of the findings. Second, measuring both behavioral symptoms of trauma (PTSS) and physiological indicators of stress response (cortisol) allowed for examination of multiple domains of children's functioning. Third, we used children's self-reports about exposure to IPV, conflict appraisals, and relationship with parents. Much of the existing research on IPV relies solely on maternal report, which may be inaccurate or biased by demands of social desirability. Finally, we were able to assess children's history of IPV from the first year of their lives and include this data in our models.

The present study extends the literature by examining whether children's threat and selfblame appraisals about parental IPV lead to the development of PTSS. In contrast to prior research examining only internalizing outcomes, we found no evidence for the mediating role of appraisals on PTSS. However, the lack of association between IPV exposure and PTSS in the present sample does not negate or diminish the need for continued attention, research, and support for issues of family violence, especially in light of the finding that IPV can lead to generalized changes to children's physiological stress response systems.

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