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Relational trauma in the context of intimate partner violence☆

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ABSTRACT

The relational model of trauma (Scheeringa & Zeanah, 2001) proposes that infants' trauma symptoms may be influenced by their mothers' trauma symptoms and disruptions in caregiving behavior, although the mechanisms by which this occurs are less well understood. In this research, we examined the direct and indirect effects of a traumatic event (maternal intimate partner violence [IPV]), maternal trauma symptoms, and impaired (harsh and neglectful) parenting on infant trauma symptoms in a sample of mother–infant dyads ($N = 182$) using structural equation modeling. Mothers completed questionnaires on IPV experienced during pregnancy and the child's first year of life, their past-month trauma symptoms, their child's past-month trauma symptoms, and their parenting behaviors. Results indicated that the effects of prenatal IPV on infant trauma symptoms were partially mediated by maternal trauma symptoms, and the relationship between maternal and infant trauma symptoms was fully mediated by neglectful parenting. Postnatal IPV did not affect maternal or infant trauma symptoms. Findings support the application of the relational model to IPV-exposed mother–infant dyads, with regard to IPV experienced during pregnancy, and help identify potential foci of intervention for professionals working with mothers and children.

Living in a household where intimate partner violence (IPV) occurs likely threatens an infant's safety and well-being and may undermine early social-emotional functioning. The majority of children are first exposed to IPV in infancy (Graham-Bermann & Perkins, 2010), and the dysregulating effects of IPV can manifest as early as age one (Bogat, DeJonghe, Levendosky, Davidson, & von Eye, 2006; Levendosky, Bogat, & Martinez-Torteya, 2013). Mothers exposed to IPV also frequently exhibit symptoms of posttraumatic stress, which can affect caregiving functioning (Dutton et al., 2006; Martinez-Torteya, Bogat, von Eye, Davidson, & Levendosky, 2009). The relational model of trauma (Scheeringa & Zeanah, 2001) predicts that maternal trauma symptoms following exposure to a traumatic event can impair interactions between mothers and their children, thus eliciting or exacerbating infant traumatic stress symptoms. In this research, we used the relational trauma model to test hypotheses about the influence of maternal IPV exposure and mediating maternal factors on infant trauma symptoms to advance our understanding of the mechanisms by which IPV confers risk of emotional and behavioral dysregulation on very young children.

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IPV prevalence rates are as high as 30% in young, low-income, unmarried women with young children (Carpenter & Stacks, 2009; Tallieu & Brownridge, 2010), and the prevalence of posttraumatic stress disorder (PTSD) symptoms in exposed women ranges from 30% to 84% (for a review, see Woods, 2005). One in eight children in the United States is exposed to IPV before the age of five (Hamby, Finkelhor, Turner, & Omrod, 2011), and children exposed to IPV are more likely than non-exposed children to exhibit a range of psychological and behavioral problems, including PTSD and trauma symptoms (Briggs-Gowan, Carter, & Ford, 2010; Carpenter & Stacks, 2009; Chan & Yeung, 2009; Kitzmann, Gaylord, Holt, & Kenny, 2003; Levendosky et al., 2013). Meta-analytic data indicate a medium-sized association between IPV exposure and PTSD in children ($z = 0.35$; Chan & Yeung, 2009). The effects of maternal exposure to IPV on children are both direct (Kitzmann et al., 2003) and indirect, and prior research indicates that children's susceptibility to negative outcomes may be conferred via parental factors such as maternal PTSD symptoms (Scheeringa & Zeanah, 2001) and parenting behaviors (Krishnakumar & Buehler, 2000).

Although the majority of research on trauma symptoms in young children has focused on children ages 3 and older (e.g., Briggs-Gowan et al., 2010; Graham-Bermann & Levendosky, 1998), there is evidence that children possess the developmental capacity to both process an event as traumatic and display trauma symptoms as early as infancy (for a review, see De Young, Kenardy, & Cobham, 2011). Infants may be particularly at risk for the development of dysregulation consistent with trauma symptoms following IPV exposure given their limited coping skills, rapid developmental change, and reliance on primary caregivers who may themselves be impaired and whose caregiving may suffer as a consequence. As noted by De Young and colleagues, infant symptoms following trauma exposure typically present as a range of physiological, affective and behavioral dysregulation that is observable by parent report. There is also growing evidence that infants exposed to the specific stressor of IPV can show symptoms similar to those seen in older children and adults (Bogat et al., 2006; DeJonghe, Bogat, Levendosky, Von Eye, & Davidson, 2005; Levendosky et al., 2013). Developmentally sensitive diagnostic criteria for evaluating PTSD in infants and toddlers include symptoms such as exaggerated startle response, extreme fussiness, social withdrawal, and restricted range of affect following exposure to traumatic events (Zero to Three, 2005). In the present research, the term "trauma symptoms" will be used to refer to the physiological, affective, and behavioral signs of dysregulation characteristic of changes in infant regulatory functioning following trauma exposure. The study of trauma symptoms in infants has been given little attention in the literature to date, which is of concern given the effects that early trauma may have on child development and the role of trauma symptoms as a risk factor for PTSD in later childhood and adulthood (De Young et al., 2011; Schore, 2001).

The relational model of PTSD proposed by Scheeringa and Zeanah (2001) suggests pathways by which maternal trauma symptoms may influence infants' trauma symptoms. The model emerged from observations that trauma symptoms frequently co-occur between infants and caregivers (Famularo, Fenton, Kinscherff, Ayoub, & Barnum, 1994; Laor, Wolmer, Mayes, & Gershon, 1997; Sack, Clarke, & Seeley, 1995). It purports that maternal trauma symptoms influence infant trauma symptoms through impaired maternal caregiving. That is, a mother's trauma symptoms may make it difficult for her to read cues of distress in her infant and respond effectively (Scheeringa & Zeanah, 2001). Several empirical studies have supported the link between maternal and child trauma symptoms, for example, in dyads exposed to natural disasters (e.g., Hurricane Katrina: Scheeringa & Zeanah, 2008), war (Kaufman-Shriqui et al., 2013), traumatic physical injury (Nugent, Ostrowski, Christopher, & Delahanty, 2007), and childhood cancer (Graf, Bergstraesser, & Landolt, 2013).

In the relational PTSD model, maternal trauma symptoms lead to multiple forms of impaired caregiving, including neglectful or harsh parenting (Scheeringa & Zeanah, 2001). Indeed, a mother's emotional response to the traumatic event may contribute to neglectful parenting by leading her to withdraw and be less responsive and emotionally available to her child, leaving the infant unregulated when emotionally aroused and exacerbating the infant's trauma symptoms. Harsh parenting, such as overly-controlling behavior or use of physical punishment, may also occur as a result of maternal trauma exposure (Lieberman, 2004). For example, a mother's experience of irritability, anxiety, or excessive feelings of guilt as a function of her own trauma symptoms may lead to overly controlling or otherwise harsh caregiving, and her recurrent heightened emotional arousal may distress the infant. Thus, implicit in the model of relational trauma is the assertion that parenting behaviors partially mediate maternal and infant trauma symptoms (De Young et al., 2011).

Prior research provides some support for the applicability of the relational trauma model to IPV-exposed mother–infant dyads, specifically. For example, research suggests that mother and infant PTSD symptoms often co-occur in IPV-exposed samples (Bogat et al., 2006; Levendosky et al., 2013). Threats to a caregiver are also more likely to result in more severe trauma symptoms in infants, including a diagnosis of PTSD, than are traumatic events not involving a threat to a caregiver (Scheeringa, Wright, Hunt, & Zeanah, 2006; Scheeringa & Zeanah, 1994). Further, maternal psychopathology and well-being has been implicated as a mediator of IPV exposure and parenting across multiple studies, consistent with the relational PTSD model (Huang, Wang, & Warrener, 2010; Nicklas & Mackenzie, 2013). Lastly, some mothers experiencing IPV are more likely to engage in parenting that is less effective or potentially harmful for developing infants, such as harsh or neglectful parenting (Holden & Ritchie, 1991; Renner & Slack, 2006; Zolotor, Theodore, Coyne-Beasley, & Runyan, 2007).

A growing number of studies examining relations between IPV exposure, parenting, and mother and infant outcomes have examined IPV exposure occurring in infancy and early childhood (e.g., Bogat et al., 2006). However, risk of IPV increases during pregnancy, is associated with more severe violence compared to IPV that occurs outside of pregnancy, and may also have deleterious effects on children (Glover, O'Connor, & O'Donnell, 2010; Huth-Bocks, Levendosky, Theran, & Bogat, 2004b; Taillieu & Brownridge, 2010). Maternal exposure to trauma such as IPV may directly alter the developing hypothalamic-pituitary-adrenal (HPA) axis of the fetus via increased corticosteroid exposure, leading to affective and behavioral dysregulation in

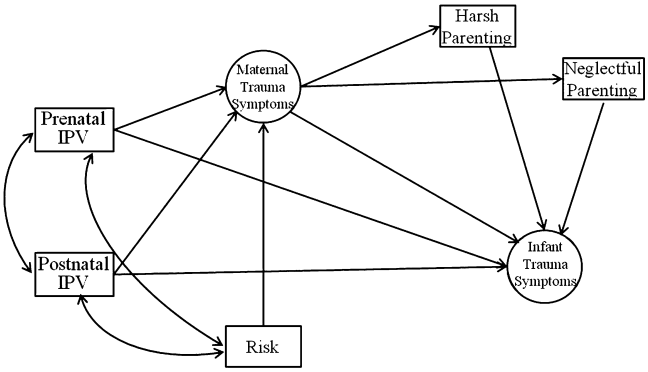

Fig. 1. Hypothesized path model.

infancy (Glover et al., 2010; Bergh, Mulder, Mennes, & Glover, 2005). Indirectly, exposure to IPV during pregnancy contributes to maternal trauma symptoms and can negatively influence parenting (Huth-Bocks, Levendosky, Theran, & Bogat, 2004a). Thus, although prenatal IPV exposure would not meet diagnostic criteria as a traumatic event for the fetus, according to current nosology, prior research supports the contention that infant regulatory functioning is affected by prenatal IPV through both the direct physiological impact of trauma *in utero* as well as exposure to maternal trauma symptoms and associated parenting deficits after birth. To date, no studies have examined the influence of prenatal IPV exposure and mediating parental factors on infant trauma symptoms. Thus, the present research also explored whether, similar to postnatal IPV, maternal exposure to IPV during pregnancy directly or indirectly influences infant trauma symptoms.

In this research, we examined the direct effects of IPV exposure (prenatal and postnatal), and its indirect effects via relational factors (maternal trauma symptoms, harsh and neglectful parenting) on infant trauma symptoms. We hypothesized that (1) prenatal and postnatal IPV would have a direct effect on maternal trauma symptoms, (2) maternal trauma symptoms would increase harsh and neglectful parenting, and (3) maternal trauma symptoms and parenting would mediate the effect of prenatal and postnatal IPV on infant trauma symptoms. We further hypothesized that these effects would remain statistically significant when controlling for other risk factors (e.g., partner status, substance use, and other stressful life events). The proposed model is presented in Fig. 1.

Method

Participants

Participants were 182 mother–infant dyads, recruited from urban, suburban, and rural areas in the Midwestern U.S. as part of a larger research project. Women were recruited with fliers describing the study posted in local businesses throughout the recruitment areas, organizations that specifically serve families with young children, and organizations that serve women experiencing IPV. Electronic media including Craigslist™ and Facebook™ were also used. Potential participants were screened by telephone to ensure they met several criteria based on the aims of the larger study: (1) English-speaking, (2) 18 to 34 years old, (3) not pregnant, (4) not lactating or willing to not breast feed child for 2 hours prior to assessment, (5) *without* endocrine or other disorders associated with abnormal glucocorticoid release, (6) involved in a heterosexual romantic relationship for at least 6 weeks during the pregnancy; and (7) no premature delivery (i.e., <37 weeks). We also over-sampled for IPV exposure: women were considered to have IPV pre- and/or postnatally if they endorsed any items on the violence questionnaire (Severity of Violence Against Women Scales; SVAWS) indicating experiences of IPV that were, at a minimum, threats of moderate physical violence (see Measures below for a fuller description). In the present sample, 73% ($N = 133$) of participants met this criteria.

The demographic characteristics of the mothers were as follows: average age of 24.5 years (range = 18–40), average monthly income of $1170 (median = $900), 51% single and living alone, and 51% had a high school diploma or less. Mothers' ethnicity was 15% multiracial, 33% African American, 43% white and 9% Latina. Children had a mean age of 11.77 months (SD = 2.44), were evenly distributed by gender (50% girls), and were ethnically diverse (36% multiracial, 29% African American, 28% white, and 7% in other groups).

Measures

Infant Trauma Symptoms. Infants were assessed for trauma symptoms with the *Infant Social and Emotional Assessment Trauma-Related Symptoms Scale* (ITSEA-TRSS; Briggs-Gowan & Carter, 2007). The ITSEA-TRSS has been used previously with samples of 1-year-old children (Mongillo, Briggs-Gowan, Ford, & Carter, 2009). Higher scores on the subscales of this measure have been associated with exposure to traumatic events (e.g., Briggs-Gowan, Carter, & Ford, 2011). This measure consists of 39 items that mothers rate on a 3-point scale from "not true/rarely" to "very true/often," describing behaviors they observed in their infant

Table 1
Means, standard deviations, intercorrelations, and scale reliability coefficients of study variables.

	1	2	3	4	5	6	7	8	9	10	11
1. Prenatal IPV	(.98)										
2. Postnatal IPV	.68*	(.98)									
3. Maternal trauma (re-experiencing)	.45*	.45*	(.79)								
4. Maternal trauma (avoidance)	.42*	.38*	.76*	(.83)							
5. Maternal trauma (arousal)	.50*	.36*	.73*	.77*	(.84)						
6. Infant trauma (re-experiencing)	.31*	.13	.13	.19*	.16*	(.34)					
7. Infant trauma (avoidance)	.18*	.05	.10	.11	.08	.20*	(.47)				
8. Infant trauma (arousal)	.39*	.26*	.28*	.29*	.36*	.51*	.41*	(.82)			
9. Harsh parenting	.20*	.18*	.15*	.17*	.19*	.11	.10	.25*	(.61)		
10. Neglectful parenting	.04	.03	.24*	.29*	.19*	.02	.27*	.20*	.10	(.60)	
11. Cumulative risk (composite)	.44*	.38*	.37*	.41*	.37*	.20*	.15*	.28*	.30*	.15*	—
Minimum	0	0	0	0	0	0	0	2	0	0	0
Maximum	126	138	15	19	15	7	7	31	23	15	5
Mean	20.72	12.57	2.61	3.93	3.94	1.68	1.38	11.63	4.58	3.28	2.26
Standard deviation	28.34	21.82	3	4.49	4.22	1.51	1.57	5.69	4.84	2.98	1.25
Skew	1.83	2.97	1.26	1.18	.94	1.04	1.19	.65	1.52	1.32	−.09
Kurtosis	3.02	10.88	1.24	.55	-.11	1.13	.96	.09	2.37	2.17	−.63

Note:
* $p < .05$.

in the prior month. Sum scores for three trauma symptom subscales (arousal, re-experiencing, and avoidance/numbing) were used in the present study. Sample arousal items include "is easily startled" and "is bothered by loud noises or bright lights." Example re-experiencing items include "repeated same action over and over again without enjoyment" and "wakes up from scary dreams or nightmares." Sample avoidance/numbing items include "less fun than other children" and "avoids physical contact." Scale reliabilities (Cronbach's alpha) for this and other study measures are presented in Table 1.

Intimate Partner Violence

Women's exposure to IPV was assessed with the *Severity of Violence Against Women Scales* (SVAWS; Marshall, 1992). This measure consists of 46 items rated on a 4-point scale ranging from "Never" to "Many Times." Items range from symbolic threats (such as shaking a figure) to threats of mild, moderate, and severe violence, actual mild, moderate and severe violence, and sexual violence. An example of severe violence includes "punched you," and sexual violence includes "demanded sex whether you wanted to or not." Women completed the SVAWS twice: once for experiences of IPV from male partners during pregnancy (prenatal IPV exposure) and once for IPV from male partners in the first year after the child's birth (postnatal IPV exposure). To improve the retrospective reporting of IPV, a life events calendar was administered (Belli, 1998; Kessler & Wethington, 1991) prior to women completing both the prenatal and postnatal SVAWS.

Maternal Trauma Symptoms

Women were assessed for trauma symptoms resulting from IPV or other lifetime traumatic events with the *Modified PTSD Symptom Scale–Self Report (MPSS-SR;* Falsetti, Resnick, Resick, & Kilpatrick, 1993). This self-report scale measures the frequency of trauma symptoms present in the prior 2 weeks on three subscales: re-experiencing, avoidance/numbing, and arousal symptoms. Eighteen percent of women displayed "probable" PTSD, as assessed by the recommended cut-off score >13 (Coffey, Gudmundsdottir, Beck, Palyo, & Miller, 2006).

Maternal Parenting

Neglectful parenting was measured using the *Multidimensional Neglectful Behavior Scale – Parent Report* (MNBS-PR; Kaufman Kantor et al., 2004). This 15-item measure assesses the frequency of neglectful parenting behaviors during the prior 6 months on a 4-point scale, ranging from "never happened" to "always happened." For the current study, items with poor inter-item reliability were dropped, and remaining items ($n = 12$) were summed. To assess for harsh parenting behaviors, women were administered the *Parent-Child Conflict Tactics Scales* (CTSPC; Straus, Hamby, Finkelhor, Moore, & Runyan, 1998). This 22-item measure assesses the frequency of harsh parenting on a 7-point scale ranging from "has never happened" to "more than 20 times" within the prior year. The current study used a sum score of 18 items from the psychological and physical aggression subscales.

Cumulative Risk. A cumulative risk variable was created to control for additional sources of demographic and environmental risk known to influence child outcomes (Sameroff, Seifer, Baldwin, & Baldwin, 1993), using a method recommended for assessing multiple social risk factors when other factors are of primary interest (Burchinal, Roberts, Hooper, & Zeisel, 2000). The cumulative risk variable was computed by summing 5 dichotomous scores (0 = low risk; 1 = high risk), including income (below Medicaid poverty cut-off = 1), marital status (single = 1), age (≤22 years = 1), negative life events using the Life Experiences Survey (Sarason, Johnson, & Siegel, 1978; highest 25% percentile = 1), and drug use rated by the Perinatal Risk Assessment Monitoring Survey (Gilbert, Shulman, Fischer, & Rogers, 1999; any street drug use pre- or postnatal = 1). The cumulative risk score could range from 0 to 5.

Procedures

Women who were interested in the study telephoned the project office to complete an intake questionnaire that determined participant eligibility. Based on eligibility and consent, women were then scheduled for interviews with their children. All research visits occurred in project offices when children were approximately 1 year of age (range 11–15 months). Interviews were administered by two trained graduate and/or undergraduate students. Mothers signed informed consent for themselves and their infant. At the end of the interview mothers were financially compensated for their participation, and the infants were given a small stuffed animal.

Results

Descriptive statistics for the variables of interest are in Table 1. Correlations ranged from small to large. Prenatal and postnatal IPV were highly correlated with each other, and both were also moderately correlated with cumulative risk. Prenatal and postnatal IPV were each also correlated with both maternal and infant trauma symptoms, with correlations ranging from small to large. Maternal trauma symptom clusters exhibited large intercorrelations, and infant trauma symptom clusters exhibited small to moderate intercorrelations. Of note, maternal trauma symptoms exhibited only modest correlations with infant trauma symptoms. For example, avoidance/numbing infant trauma symptoms exhibited the smallest correlations both with the other infant trauma symptoms and with maternal trauma symptoms.

To confirm the viability of the trauma symptom clusters with which we intended to test our hypotheses, we performed confirmatory factor analyses (CFAs) separately for maternal and infant trauma symptoms. In order to account for the skew and kurtosis in our data, we used robust maximum likelihood estimation in these and subsequent structural analyses, which uses a sandwich estimator to compute standard errors and is robust to non-normal data (Muthén & Muthén, 2010; Tomer & Pugesek, 2003). For each model, we used the observed measurements of the three PTSD symptom clusters (re-experiencing, avoidance/numbing, and arousal), setting the variance of the first indicator in each (re-experiencing cluster) to 1.00. We evaluated the models with maximum likelihood estimation using Mplus (version 6; Muthén & Muthén, 2010) for these and subsequent analyses. For each CFA model, overall fit was excellent (RMSEA = .00, CFI = 1.00, SRMR = .00), indicating that the latent measurements of both maternal and infant trauma symptoms would be amenable to further analysis in more inclusive structural models.

In our first structural equation model, we tested whether maternal trauma symptoms mediated the effect of prenatal and postnatal IPV symptoms on infant trauma symptoms while controlling for cumulative risk (see Fig. 2). Of note, in this

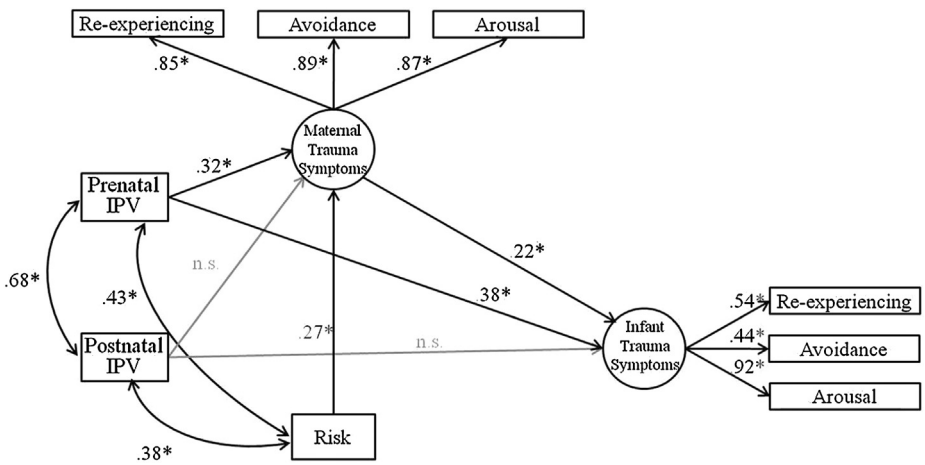

Fig. 2. Partial mediation model including prenatal and postnatal IPV, maternal and infant trauma symptoms, and cumulative risk.
Notes: Standardized coefficients listed in model; $^{*}p < .05$

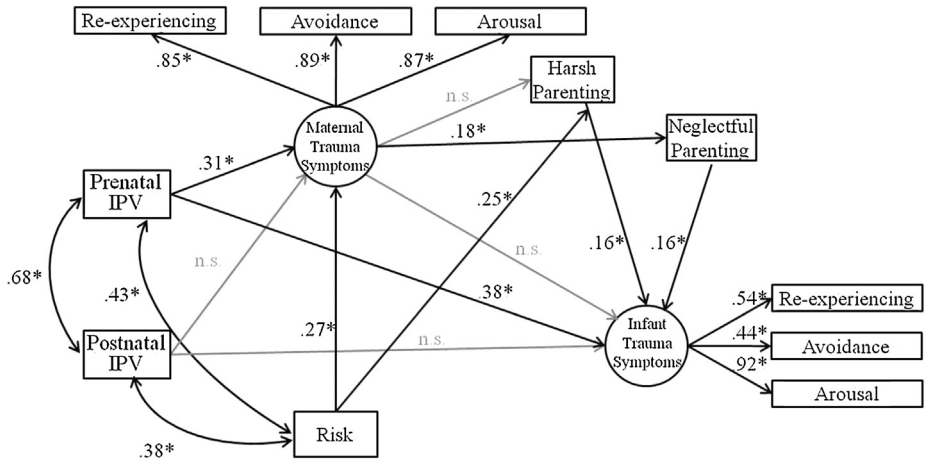

Fig. 3. Complete mediation model including prenatal and postnatal IPV, maternal and infant trauma symptoms, harsh and neglectful parenting, and cumulative risk.
Notes: Standardized coefficients listed in model; *$p < .05$

and our second structural model, we retained all paths from the base model (all paths estimated) that were statistically significant in the structural model and removed those paths that were hypothesized but not statistically significant. The χ^2 test of model fit was statistically significant ($\chi^2 = 34.08$, df = 21, $p = .04$), indicating possible model misfit. However, the χ^2 test does not take sample size or model complexity into account (Tanaka, 1993). Indeed, other fit indices consistently indicated that overall model fit was good (RMSEA = .06, CFI = .97, SRMR = .03). This model exhibited better comparative fit than the base model with all paths estimated (AIC = 3949.72, BIC = 4054.91). As hypothesized, maternal trauma symptoms exerted a significant main effect on infant trauma symptoms. Furthermore, maternal trauma symptoms partially mediated the effect of prenatal IPV on infant trauma symptoms. Postnatal IPV did not exert a statistically significant effect on either maternal or infant trauma symptoms. Cumulative risk was associated with both types of IPV, as well as with maternal trauma symptoms.

In our second structural equation model, we tested whether harsh and neglectful parenting behaviors mediated the effects of maternal trauma symptoms on infant trauma symptoms (see Fig. 3). The paths between risk and infant trauma symptoms, between harsh and neglectful parenting, and between IPV (both prenatal and postnatal) and parenting (both harsh and neglectful) were neither theoretically predicted nor statistically significant in the base model with all paths estimated; as such, we did not estimate these paths in our final model. We also did not estimate the path between cumulative risk and neglectful parenting, which was statistically insignificant in the base model. However, cumulative risk was associated with harsh parenting in the base model, so we estimated it in our final model. In this model, the χ^2 test of model fit was not statistically significant ($\chi^2 = 49.24$, df = 35, $p = 06$), and all other indices similarly indicated that overall model fit was good (RMSEA = .05, CFI = .97, SRMR = .04). Additionally, the constraints we imposed on our final model resulted in an improvement in comparative model fit over the base model with all paths estimated (AIC = 4952.18, BIC = 5086.05).

With parenting variables included in the model, prenatal IPV retained its consistent effect on both maternal and infant trauma symptoms. However, the effect of maternal trauma symptoms on infant trauma symptoms was no longer significant; rather, this effect was fully mediated by neglectful parenting. Both types of parenting behaviors exhibited modest effects on infant trauma symptoms. However, contrary to our hypotheses, maternal trauma symptoms were not associated with harsh parenting. Lastly, both maternal trauma symptoms and harsh parenting were associated with cumulative risk.

Discussion

This research examined the relational model of trauma in mother–infant dyads studied in the context of IPV. Three main findings emerged. First, maternal prenatal IPV had a direct effect on maternal trauma symptoms, whereas the most proximal IPV (that which occurred during the infant's first year of life) did not. Second, the predictors of harsh and neglectful parenting were different. Maternal trauma symptoms predicted neglectful parenting, whereas general risk factors predicted harsh parenting. And, third, infant trauma symptoms were directly predicted by prenatal IPV and maternal trauma symptoms and neglectful parenting mediated this relationship. These findings have important implications for understanding the relational model of trauma as well as helping women and infants cope with exposure to IPV.

Postnatal IPV exposure was neither associated with maternal trauma symptoms nor infant trauma symptoms when prenatal IPV was taken into account. This finding on the surface seems inconsistent with previous research demonstrating deleterious direct effects of postnatal IPV on infant trauma symptoms (Bogat et al., 2006; Levendosky, Bogat,

& Martinez-Torteya, 2013), but prenatal IPV was not examined in these studies. It is possible, therefore, that when maternal exposure to IPV in pregnancy is taken into account, IPV exposure in the first year of life may not uniquely contribute to infant trauma symptoms. Postnatal IPV exposure may also have negative effects that are not apparent in infancy but which may become apparent later in development. For example, one longitudinal investigation that examined both prenatal and postnatal IPV found that IPV during both time periods was associated with depressive symptoms at age 10 (Martinez-Torteya, Bogat, von Eye, & Levendosky, 2014). Two other explanations may account for the absence of a relationship between postnatal IPV exposure and maternal and infant trauma symptoms. First, as the average frequency of IPV was higher in the prenatal period than the postnatal period, it is possible that the significant effects of prenatal IPV are partially explained by its relatively high frequency observed in the present sample. Second, prenatal IPV may uniquely affect child outcomes via a series of proximal (*in utero*) and distal (parental) physiological and psychological mechanisms that postnatal IPV cannot, as discussed below.

Scheeringa and Zeanah (2001) emphasized that trauma symptoms involving avoidance and withdrawal may lead to neglectful parenting, with negative outcomes for infants. The present results support this contention of the relational model and suggest that disruptions in parenting behavior can also be influenced by IPV in pregnancy. Results indicated an association between maternal prenatal IPV and maternal trauma symptoms, which were both related to infant trauma symptoms. Consistent with the relational model, the effect of maternal trauma symptoms on infant trauma symptoms was fully mediated by neglectful parenting. Thus, maternal trauma symptoms and neglectful parenting both emerged as important mechanisms explaining the link between prenatal IPV and infant trauma symptoms. These findings suggest that traumatic events experienced by the mother during pregnancy can negatively influence later infant functioning via relational factors such as impaired maternal mental health and caregiving behaviors. Results are consistent with studies of the postnatal period that indicate a relationship between maternal trauma and infant trauma symptoms (Bogat et al., 2006) and find that maternal mental health can partially mediate the relationship between IPV and infant externalizing behavior (Levendosky, Leahy, Bogat, Davidson, & von Eye, 2006).

We predicted that both harsh and neglectful parenting would partially mediate the relationship between IPV and infant trauma symptoms; however, whereas neglectful parenting mediated the association between maternal and infant trauma symptoms, harsh parenting mediated the association between cumulative demographic risk and infant trauma symptoms. While one previous study found that maternal PTSD predicted greater levels of physical discipline (Cohen, Hien, & Batchelder, 2008), literature on maternal trauma symptoms and their association with specific types of parenting behaviors is otherwise scarce. The current findings suggest that harsh parenting may be a consequence of general risk factors that are associated with IPV exposure, rather than a direct result of maternal trauma symptoms. Although only parental neglect was predicted by maternal trauma symptoms, both parenting types were associated with increased infant distress. These results highlight the importance of mother–infant interactions in supporting infant social-emotional functioning. Infants exposed to IPV rely on caregivers to help them cope with emergent dysregulation, and impairments in parenting can sustain or exacerbate infant trauma symptoms.

In addition to the mediating effects of maternal trauma symptoms and parenting, results revealed a direct relationship between prenatal IPV and infant trauma symptoms. One possible mechanism may be physiological dysregulation (Yehuda & Bierer, 2007). Stress (including traumatic stress) activates the maternal hypothalamic-pituitary-adrenal (HPA) axis and results in a cascade of hormonal responses, culminating in the release of glucocorticosteroids into the bloodstream that can cross the placental barrier and affect fetal brain development (e.g., Van den Bergh, Mulder, Mennes, & Glover, 2005). Prenatal exposure to maternal HPA axis byproducts is associated with epigenetic changes to the fetal stress response system that impair the child's HPA axis functioning (Seckl & Meaney, 2006). The dysregulating effects of fetal exposure to glucocorticosteroids predispose children to a range of psychopathology, including anxiety (Huizink, Mulder, & Buitelaar, 2004). This is one mechanism by which intergenerational transmission of trauma symptom risk has been proposed to occur (Yehuda & Bierer, 2007). Consistent with prior research suggesting that *in utero* exposure to IPV may have negative neurodevelopmental consequences for children (e.g., Glover et al., 2010), our results indicate that mother's trauma exposure during the prenatal period is associated with the expression of infant trauma symptoms after birth.

There were some limitations to the current study. First, the study was cross-sectional and retrospectively measured prenatal IPV exposure. This approach limits conclusions about both the causality of relationships among variables and the course of the development of infant trauma symptoms from the prenatal period into infancy. One could argue that mothers may have had difficulty accurately reporting on incidents of IPV that occurred in the past. However, in order to enhance the reliability of the mother's retrospective reporting, we incorporated an event history calendar into the protocol. An event history calendar supports autobiographical memory retrieval by introducing a visual aid and inquiring about participants' experiences in relation to major activities, holidays, and events (Belli, 1998). This method improves the reporting of IPV compared to a standard interview about violent events (Yoshihama, Gillespie, Hammock, Belli, & Tolman, 2005), and has demonstrated improved recall over standard interview methods in both record-check (van der Vaart & Glasner, 2007) and longitudinal (Belli, Shay, & Stafford, 2001) designs in which the accuracy of participant reports could be assessed with a high degree of confidence.

An additional limitation is that all measures relied on maternal report of IPV and maternal and infant functioning, and, as such, maternal characteristics or response bias may have influenced reporting. Future studies including behavioral observations of parenting and infant functioning are important to conduct. Furthermore, although the use of community-based recruitment methods allowed for the recruitment of a diverse sample of women, and oversampling for IPV provided statistical

power to test the present hypotheses, it is unclear to what extent these results may generalize across the broader population of individuals experiencing IPV. For example, individuals recruited from family violence shelters may differ from those recruited through community-based methods with regard to the frequency and severity of IPV experienced (Kelly & Johnson, 2008), which may differentially impact both trauma symptoms and parenting. Future studies may benefit from varying sampling methods to examine how these results may replicate within different groups of women experiencing IPV.

The conceptualization and measurement of infant trauma symptoms in the present study has implications for future research. The use of the ITSEA-TRSS allowed for the measurement of arousal, re-experiencing, and avoidance/numbing symptoms in all infants who participated in the study, thereby providing an index of infant dysregulation consistent with prior research on infant responses following traumatic stress (for a review, see De Young et al., 2011). Results on this measure were moderately correlated with infant trauma symptoms on a PTSD Semi-Structured Interview (PTSDSSI; $r = 0.43$, $p < 0.05$) that was administered for a subset of infants ($N = 95$) in the present sample who had been exposed to any traumatic event in the first year of life, suggesting that the behaviors assessed by the ITSEA-TRSS and PTSDSSI reflect similar and overlapping domains of maladaptive responses to trauma. Further investigation of the construct overlap between the items of the ITSEA-TRSS and diagnostic criteria is recommended.

An additional implication of the current study is the need to study the development of infant trauma symptoms over time, starting with exposure during the prenatal period. Future research should include prospective assessments of the mechanisms of prenatal IPV exposure on developing infants and would benefit from a closer examination of proposed prenatal mechanisms of risk, such as HPA axis dysregulation. In addition, the present study examined the construct of harsh parenting, which includes a range of behaviors from threatening physical punishment to pinching, slapping, or kicking a child. The most severe forms of harsh parenting may be characterized as child abuse, with significant implications for children's emotional health (Famularo et al., 1994; Jouriles, McDonald, Slep, Heyman, & Garrido, 2008); however, documented instances of child maltreatment for participants were not available. It is possible that our participants were unwilling to endorse the more severe items on the harsh parenting subscale, thus biasing our results. Future research should attempt to include additional measures of child maltreatment, including official reports.

The results of our research may have implications for professionals working with mothers and children. Findings emphasize the importance of clinicians attending to women's experiences of pre- and/or postnatal IPV and resulting trauma symptoms. If maternal trauma symptoms are present, women may engage in neglectful parenting. Demographic risk should also be assessed, as it may predict both IPV exposure and harsh parenting. Clinicians should consider assessing prenatal IPV exposure as a potentially dysregulating source of stress when assessing infant trauma symptoms. Intervening in family violence during the prenatal period may be especially important because relational and physiological mechanisms both likely contribute to infant outcomes. Our research findings highlight the importance of the primary caregiving relationship in infant outcomes following exposure to trauma. When such symptoms are identified, clinicians should attend to parental mental health, risk factors, and parenting in treating infant trauma symptoms.

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