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Cascades of Risk Linking Intimate Partner Violence and Adverse Childhood Experiences to Less Sensitive Caregiving During Infancy

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Abstract

This study evaluated if maternal intimate partner violence (IPV) had indirect effects on sensitive parenting in infancy through prenatal depressive symptoms and postpartum parenting stress and if maternal adverse childhood experiences (ACEs) moderated these indirect effects. We hypothesized that: (a) IPV would be associated with greater prenatal depressive symptoms, which would predict greater postpartum parenting stress, and ultimately less sensitive parenting and (b) the link between IPV and depressive symptoms would be strongest for mothers with high ACEs. Participants included 295 mothers and their infants who were assessed prenatally and at 12 months postpartum. Path analyses indicated that mothers with higher IPV endorsed greater prenatal depressive symptoms, which was in turn associated with postpartum parenting stress, and ultimately less sensitive parenting behavior. Moderation analyses revealed that these indirect effects varied as a function of maternal ACEs, with the link between IPV and depressive symptoms only present for mothers who reported high ACEs. Because less sensitive caregiving is often an early indicator of child maltreatment risk, understanding precursors to sensitivity is critical to increase precision in parenting interventions designed to reduce risk for maltreatment. Results may inform evidence-based preventive interventions for mothers and infants at high-risk for child abuse and neglect.

Keywords

domestic/intimate partner violence; adverse childhood experiences; parental sensitivity; perinatal depressive symptoms; parent-infant dyads

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Intimate partner violence (IPV), conceptualized as physical, sexual, or psychological abuse by or toward a romantic partner (Capaldi et al., 2012), is a pervasive public health concern and a well-established risk factor for a number of mental, physical, and socioemotional problems (Sugg, 2015). Parental IPV also puts children at risk for experiencing maltreatment (Coulter & Mercado-Crespo, 2015). It is estimated that in approximately 40% of IPV cases, child abuse and neglect is also present in the home (Finkelhor et al., 2009). Furthermore, some states consider exposure to IPV to be a form of child neglect or emotional abuse. As one potential explanation for this co-occurrence of types of trauma, research has consistently demonstrated that IPV increases caregivers' risk for engaging in potentially harmful parenting practices (e.g., harsh psychological and physical discipline, neglect) with their children (Chiesa et al., 2018). However, little is known about what underlying processes explain the link between IPV and parenting difficulties and what factors lead some caregivers to be more vulnerable than others.

Maternal sensitivity during infancy, characterized by the mother's capacity to accurately interpret and respond to infant cues, provides the foundation for positive parent-child relationships across the life span and promotes healthy socioemotional development (de Wolff & van IJzendoorn, 1997). Mothers who are less sensitive with their infants may show ineffective or delayed responses to infants' distress or bids for attention or have interactions with their infants defined by intrusive, harsh, or withdrawn behaviors (de Wolff & van IJzendoorn, 1997). Experiencing IPV is a significant risk factor for less sensitive maternal behavior during infancy, with IPV consistently associated with harsh and intrusive parenting and dyssynchronous parent-infant interactions (Gustafsson et al., 2012). Lower levels of sensitive caregiving not only increases risk for infant socioemotional difficulties, but can also be an early indicator of parents' risk for engaging in child abuse or neglect (Cicchetti & Toth, 2005; Lindhiem et al., 2011; Valentino, 2017). For example, in prior research, maltreating mothers tend to show less sensitive parenting than their non-maltreating peers (Cicchetti et al., 2006), and theorists consistently argue that difficulties recognizing and appropriately responding to children's cues and needs is a central component of parent-child relationship dysfunction (Lindhiem et al., 2011). Furthermore, increasing sensitivity has been established as a crucial goal of parenting interventions designed to reduce risk for child maltreatment (Baggett et al., 2017; Lindhiem et al., 2011). It is therefore critical to better understand the psychosocial factors that interfere with mothers' abilities to provide sensitive parenting in the earliest months of life.

Depressive Symptoms and Parenting Stress as Mechanisms Linking IPV to Parenting

Models of family spillover posit that negativity transfers from one relationship to another. Thus, distress and emotional turmoil arising from IPV may "spill over" into the parent-child relationship, setting in motion processes that undermine caregivers' ability to engage in child-sensitive parenting behaviors (Erel & Burman, 1995). It is possible that maternal mental health in pregnancy and feelings of parenting-related stress in the early postpartum period may serve as intermediary mechanisms underlying "spillover" from IPV to insensitive caregiving. IPV has consistently been identified as a salient risk factor

for depression, particularly among women (Beydoun et al., 2012). In fact, some research suggests that these links may be especially significant during pregnancy (Martin et al., 2006), and prenatal depression has been associated with a number of negative health, parenting, and offspring outcomes postnatally (Bonari et al., 2004; Field, 2011). Prenatal depression may also initiate a number of cognitive, biological, and behavioral responses that carry over into the early months of parenting (Huizink et al., 2017). As such, mothers with a history of depression may be more likely to feel high parenting stress, conceptualized as the degree to which a parent feels overwhelmed by the demands of parenting (Deater-Deckard, 2004). One study found that depression during the third trimester of pregnancy was associated with higher parenting stress at 3 and 6 months postpartum (Misri et al., 2010). However, limited research has examined whether this extends beyond 6 months postpartum.

Two salient aspects of parenting stress that may be especially affected by prenatal depression are: (1) perceptions that the infant and interactions with the infant do not meet the parent's expectations or are not enjoyable (e.g., stress around parent-child dysfunctional interaction) and (2) feelings of distress that stem from personal factors directly related to parenting (e.g., general parental distress; Abidin, 2012). In support of examining distinct dimensions of stress, Huizink and colleagues (2017) found that depression in pregnancy was specifically associated with child attachment-related parenting stress as opposed to other dimensions of stress. Furthermore, when mothers feel heightened stress about their new role as a caregiver, it can undermine their ability to engage in sensitive parenting (Deater-Deckard, 2004). When mothers feel stress that stems from perceptions of dysfunctional interactions with their child, they may be especially at risk for less optimal caregiving (Smith & Stephens, 2018). For example, if a mother approaches an interaction with her infant with negative expectations, this may guide her parenting behaviors (e.g., less sensitivity), which will result in infant misbehavior that loops back into her negative cognitions and behaviors (Sturge-Apple et al., 2014). Consistent with this notion, studies have shown that stress related to parent-child dysfunctional interaction is specifically linked to less maternal sensitivity (Dau et al., 2019; Smith & Stephens, 2018). However, no prior research has examined if this dimension of stress is a mechanism linking prenatal depression to subsequent maternal sensitivity, and very few studies have directly tested it as a unique predictor of sensitivity when compared to other forms of parenting stress.

Although there is piecemeal support for our hypothesized cascade of processes linking IPV to less sensitive caregiving through prenatal depressive symptoms and parenting stress, no study has directly tested all four of these links simultaneously in a sample of low-income mothers transitioning from pregnancy to parenting an infant. Consequently, we only have a partial understanding of how these important constructs may fit together to explain how and why IPV increases mothers' risk for engaging in less sensitive caregiving with their infant.

The Potential Role of Maternal ACEs in Cascades of Risk

Finding potential sources of variability in indirect pathways is important for identifying which mothers are at highest risk for prenatal depressive symptoms, which then may lead to parenting stress and less sensitivity. Mothers' own adverse childhood experiences (ACEs), including abuse, neglect, household dysfunction, and parental loss (Felitti et al., 1998),

may be a critical source of variability. Depression is considered one of the most common health risks of an accumulation of ACEs (Hughes et al., 2017). Furthermore, research has shown that women with a history of ACEs may be especially vulnerable to experiencing depression during the perinatal period (McDonnell & Valentino, 2016) and parenting stress and parenting difficulties after giving birth (Banyard et al., 2003; Lang et al., 2010).

Drawing on the stress sensitization hypothesis, the present study examined maternal ACEs as a moderator of risk. The stress sensitization hypothesis posits that early adversity contributes to psychopathology in adulthood by heightening an individual's overall sensitivity to later stressors such as IPV (Hammen et al., 2000). Experiencing IPV, after adversity exposure in childhood, may provoke heightened sensitivity to stress and exacerbate feelings of hopelessness, shame, and sadness that were present since childhood. This may ultimately manifest in clinically significant depressive symptoms, particularly during pregnancy when reproductive hormones influence the stress response (Brummelte & Galea, 2010) such that women are especially vulnerable to depression and IPV. Extant evidence shows synergistic effects of early adversity and later IPV on prenatal depressive symptoms (Tung et al., 2019). For example, Tung and colleagues (2019) found that associations between childhood adversity and emotional distress in pregnancy were moderated by prenatal IPV in a pattern consistent with stress sensitization, highlighting the importance of their multiplicative contribution to mental health. Therefore, ACEs may exacerbate links between IPV and depression, which in turn sets in motion a further cascade of risk (e.g., increased parenting stress) leading to less sensitive parenting.

The Present Study

This study evaluates if (1) maternal exposure to IPV has indirect effects on sensitive parenting in infancy through prenatal depressive symptoms and postpartum parenting stress and (2) maternal ACEs moderate these indirect effects. Consistent with stress sensitization (Hammen et al., 2000), we hypothesized that (1) IPV would be linked with greater prenatal depressive symptoms, which would in turn be associated with greater parenting stress at 12 months postpartum and ultimately less sensitive caregiving, and (2) the link between IPV and depressive symptoms would be especially pronounced for mothers with a history of ACEs. Furthermore, we tested if these effects are specific to stress around dysfunctional parent-child interactions or generalizable to general parental distress. Because the stressors of living in poverty (e.g., housing instability, child care challenges, food insecurity, limited access to resources) increase families' risk for experiencing IPV and child maltreatment (e.g., Drake & Jonson-Reid, 2013; Jewkes, 2002), we tested our hypotheses in a large sample of low-income mothers and their infants assessed prenatally and at 12 months postpartum. To more conclusively determine the temporal ordering of the hypothesized cascade of processes, we included assessments of key constructs during both the prenatal and postpartum period when possible (i.e., IPV, depressive symptoms).

Method

Participants

Participants were drawn from a larger longitudinal project designed to examine the impact of maternal ACEs on maternal enrollment and retention in home visiting programs and the extent to which home visiting programs enhance parenting and infant health and developmental outcomes. Pregnant women were recruited from Women, Infants, and Children (WIC) clinics. To be eligible for the study, women had to be: (1) referred to family visiting services, (2) pregnant with a singleton, (3) at least 18 years of age, and (4) fluent in English or Spanish. Participants included 295 mothers and their infants who were assessed prenatally and at 12 months postpartum (retention = 84%). Of mothers, 40% were Hispanic, 42% White, 19% Black, 7% biracial, and 32% other races. Mothers ranged in age from 18 to 44 years ($M = 25.61$ years; $SD = 5.33$ years), 23% had less than a high school degree, 37% completed high school, 36% had some postsecondary education, and 4% had a bachelor's degree. At the prenatal assessment, gestational age (GA) ranged from 44 to 283 days ($M = 185.60$ days; $SD = 59.96$ days). Sixty-five percent of mothers were unemployed in pregnancy, 44% were single parents, and 38% were first time mothers. Ten percent of infants were born premature, 62% of mothers reported at least one health problem in pregnancy (e.g., preeclampsia, gestational diabetes, anemia), and 41% reported their infant had at least one chronic health condition in the first year of life (e.g., respiratory issues, gastrointestinal issues). All families received public assistance, and 39% enrolled in evidence-based home visiting services over the course of the study.

Procedure

Expectant mothers completed baseline assessments at a prenatal enrollment visit, and mothers and their infants participated in a follow-up assessment at 12 months postpartum. Assessments were completed in participants' homes. Mothers provided written consent for themselves and their infant to participate. Ethical permission for the study was granted by the Institutional Review Board at Lifespan. Mothers were compensated monetarily for participation.

Measures

Demographics and potential covariates.—Prenatally, mothers reported their age, educational level, race and ethnicity, marital status (single vs. two-parent household), employment status (unemployed vs. employed), due date, and whether they were first time mothers. GA was calculated as the number of days between the last menstrual period (approximated based on due date) and the date of the prenatal assessment. Consistent with prior research (e.g., Tyrka et al., 2015), indicators of socioeconomic risk (education high school diploma, unemployment, single parenthood) were summed to provide a parsimonious assessment of socioeconomic adversity (possible scores ranged from 0 to 3). At the postpartum assessment, mothers reported their infant's actual birth date, if they experienced any health problems in pregnancy, and if their infant had any chronic health problems. Infant premature birth was determined from the difference between the due date and actual birth date.

Intimate partner violence (IPV).—At the prenatal assessment, mothers reported on their experiences of IPV in the past year using the Short Form of the Revised Conflict Tactics Scales (CTS2-SF; Straus & Douglas, 2004). For each conflict tactic, mothers were first asked the frequency of their own behavior (e.g., “I pushed, shoved, or slapped my partner”) and then for the frequency of the same behavior committed by their partner (e.g., “my partner pushed, shoved, or slapped me”). Response options ranged from 0 (“never”) to 6 (“more than 20 times”). Subscales included Assault (4 items; e.g., “my partner punched or kicked or beat me up”), Injury (four items; e.g., “I had a sprain, bruise, or small cut, or felt pain the next day because of a fight with my partner”), Psychological Aggression (four items; e.g., “my partner insulted or swore or shouted or yelled at me”), and Sexual Coercion (four items; e.g., “my partner insisted on sex when I did not want to or insisted on sex without a condom [but did not use physical force]”). The total past IPV score consisted of the sum of the 16 items from the four subscales ($\alpha = .77$). Mothers also completed the CTS2-SF at 12 months postpartum to assess IPV in the past 6 months. Items and scoring procedures were the same for the total IPV score at this time-point ($\alpha = .71$). The CTS2-SF is widely used to assess IPV and has been shown to have strong psychometric properties (Straus & Douglas, 2004).

Adverse childhood experiences (ACEs).—Prenatally, mothers completed the Adverse Childhood Experiences Questionnaire (Felitti et al., 1998). This 10-item measure assessed mothers’ exposure to adversity before age 18, including abuse (emotional, physical, sexual), neglect (physical, emotional), and household dysfunction (household mental illness, substance abuse, physical violence, separation/divorce, incarceration). Responses to each item were coded as 0 (not experienced) or 1 (experienced), and possible ACE score ranged from 0 to 10. The percentage of mothers who reported exposure to each ACE was as follows: 26% verbal abuse, 16% physical abuse, 17% sexual abuse, 32% emotional abuse, 13% neglect, 61% parental separation/divorce, 11% parental domestic violence, 28% parental substance abuse, 25% parental mental illness, and 14% parental incarceration. Of mothers, 22% reported zero ACEs, 26% reported one ACE, 14% reported two ACEs, 11% reported three ACEs, 11% reported four ACEs, and 16% reported five or more ACEs. Prior research suggests that an ACE score of 4 or more carries significant risk for health problems (Felitti et al., 1998).

Depressive symptoms.—Prenatally, mothers completed the Patient Health Questionnaire–9 (PHQ-9; Kroenke et al., 2001). This nine-item screening tool assessed symptoms of depression (e.g., “little interest or pleasure in doing things”; “feeling down, depressed, or hopeless”) in the last 2 weeks. Each item was rated along a 4-point scale ranging from 0 (“not at all”) to 3 (“nearly every day”). Items were summed ($\alpha = .81$), with higher scores indicating greater depressive symptoms. Mothers also completed the PHQ-9 at 12 months postpartum. Scale items and scoring were the same at this time-point ($\alpha = .89$). The PHQ-9 is widely used by researchers and practitioners to assess depression; its items align with *DSM-5* criteria for identifying depressive disorders (Beard et al., 2016); it has been validated for use in samples of pregnant women (e.g., Sidebottom et al., 2012).

Parenting stress.—At 12 months postpartum, mothers completed the Parenting Stress Index-Short Form (PSI-SF; Abidin, 2012). Subscales used in the current study included Parent-Child Dysfunctional Interaction and Parental Distress. The Parent-Child Dysfunctional Interaction scale assessed the degree to which mothers perceived their child as not meeting expectations and viewed interactions with their child as not reinforcing (12 items; $\alpha = .86$). The Parental Distress scale assessed the level of distress mothers experienced as a function of personal factors that were directly related to parenting (12 items; $\alpha = .90$). Percentile scores were used in analysis. The PSI-SF is widely used and has demonstrated strong psychometric properties (Haskett et al., 2006).

Sensitive parenting.—Sensitive parenting was assessed from observations of mother-infant free play tasks completed at 12 months postpartum. Mothers were instructed to play with the infant for 7 min using developmentally appropriate toys (e.g., toy telephone, car, rings, blocks) provided by our research team. The interaction was video-recorded by a research assistant using a handheld camera. Using the Sensitivity scale from the Emotional Availability Scales (Biringen et al., 2000), two trained raters (rater reliability .80 or above) coded maternal sensitivity during the task. The Sensitivity scale is a 9-point scale that ranges from 1 (“highly insensitive”) to 9 (“highly sensitive”) and assesses the degree to which mothers display a behavioral style that is responsive to infant cues, affectively positive, flexible, interactive, and emotionally connected (e.g., showing genuine interest and positive affect toward infant, reading infant’s signals accurately and responding appropriately, clear emotional connection between infant and mother).

Data Analysis

SPSS Version 25 was used to examine descriptive statistics, correlations between the substantive variables of interest, and potential covariates (socioeconomic adversity, ethnicity, race, age, first time mother, enrollment in home visiting intervention, GA, infant premature birth, maternal health problems in pregnancy, infant chronic health conditions). The few values on primary variables that were greater than 3.29 standard deviations from the mean (one value for ACEs and prenatal depressive symptoms, two values for postpartum depressive symptoms) were winsorized (Tabachnick & Fidell, 2012). However, the two IPV variables were log-transformed instead of having outliers winsorized because of high skew and kurtosis. Five potential covariates (socioeconomic adversity, age, ethnicity, first time mother, infant premature birth) were related to IPV, depressive symptoms, parenting stress, or sensitivity. However, when included in the larger model, infant premature birth was no longer a significant predictor of any variables, and its inclusion did not alter the pattern of results. Therefore, to maximize parsimony, the only covariates included in final models were socioeconomic adversity (specified as a predictor of sensitivity), ethnicity (specified as a predictor of IPV, depressive symptoms, and parenting stress), age (specified as a predictor of depressive symptoms), and first time mother (specified as a predictor of parenting stress). Families who participated in both time-points did not differ from those lost to attrition on any primary variables or potential covariates. When examining whether rates of missingness in the dataset were associated with any of the primary study variables or potential covariates, only three analyses were significant: higher rates of missingness were associated with higher scores on parental distress ($r = .15$, $p = .02$) and socioeconomic adversity ($r = .15$, $p = .008$),

and mothers who reported experiencing health problems in pregnancy had less missing data than those who did not, $t(293) = 3.36, p = .001$.

Primary hypotheses were tested using path analysis in Mplus Version 7.4 (Muthen & Muthen, 1998–2012). Full-information maximum likelihood (FIML) estimation was used to accommodate missing data (10.51% of values). Model fit was assessed using standard criteria (Hu & Bentler, 1999), including chi-square (χ^2) tests, the root mean square error of approximation (RMSEA; values $< .08$ indicate acceptable fit), comparative fit index (CFI; values $> .90$ indicate acceptable fit), and the standardized root mean square residual (SRMR; values $< .08$ indicate acceptable fit).

We first specified a model that examined if past experiences of IPV and ACEs uniquely had indirect effects on sensitive parenting at 12 months postpartum through prenatal depressive symptoms and two dimensions of parenting stress (perceptions of parent–child dysfunctional interaction, parental distress) at 12 months postpartum. IPV, ACEs, and identified covariates were specified as predictors of prenatal depressive symptoms, dimensions of parenting stress, and sensitive parenting. Continuous predictors were mean-centered to avoid potential issues of multicollinearity. Paths were also specified between prenatal depressive symptoms and parenting stress and sensitive parenting, and between parenting stress and sensitive parenting. To rule out the potential operation of concurrent depressive symptoms and IPV as confounding predictors of sensitive parenting, assessments of these constructs at 12 months postpartum were also included in the model (i.e., specified in same portion of model as 12-month parenting stress variables; see Figure 1). Correlations were specified among exogenous predictors and among residual errors on intermediary variables assessed at 12 months postpartum (parenting stress, postpartum IPV, postpartum depressive symptoms). To test for hypothesized indirect effects, we utilized Mplus capabilities to assess models using 10,000 bootstrap replicates to obtain bias-corrected bootstrap confidence intervals for the indirect effects (MacKinnon et al., 2004). Then, to examine whether there was a significant interaction between past experiences of IPV and ACEs, we specified an additional model identical to the first model but with the addition of the interaction between IPV and ACEs as a predictor of each variable identified in the developmental cascade of processes (i.e., prenatal depressive symptoms, parent-child dysfunctional interaction, sensitive parenting). Significant interactions were probed using procedures outlined by Aiken and colleagues (1991); simple slopes were calculated at low ($-1\ SD$) and high ($+1\ SD$) levels of the moderator variable.

Results

Descriptive Analyses

Table 1 provides means, standard deviations, and correlations for primary variables.

Primary Analyses

Indirect effects of IPV and ACEs on sensitive parenting.—The model testing unique indirect effects of IPV and ACEs on sensitive parenting (Figure 1) provided an acceptable fit to the data, $\chi^2(14) = 19.88, p = .13, RMSEA = .04, CFI = .99, SRMR = .03$.

Of relevance to our aims, higher ratings of IPV, $\beta = .24$, $p < .001$, and a higher number of ACEs, $\beta = .17$, $p = .008$, were each associated with greater prenatal depressive symptoms. In turn, prenatal depressive symptoms were associated with maternal perceptions of parent–child dysfunctional interaction, $\beta = .21$, $p = .002$, and parental distress, $\beta = .39$, $p < .001$, at 12 months postpartum. However, while maternal perceptions of parent–child dysfunctional interaction were associated with less sensitive parenting, $\beta = -.24$, $p = .008$, parental distress was not, $\beta = .17$, $p = .13$. Bias-corrected bootstrapping tests indicated that the indirect path involving past IPV, prenatal depressive symptoms, parent–child dysfunctional interaction, and sensitive parenting was significantly different from zero, 95% CI $[-.102, -.009]$. The indirect path involving ACEs, prenatal depressive symptoms, parent–child dysfunctional interaction, and sensitive parenting was also significantly different from zero, 95% CI $[-.016, -.001]$.

Interaction between IPV and ACEs.—The model testing the interaction between IPV and ACEs (Figure 2) provided an acceptable fit to the data, $\chi^2(17) = 29.05$, $p = .03$, RMSEA = .05, CFI = .97, SRMR = .04. The interaction between IPV and ACEs predicted prenatal depressive symptoms, $\beta = .14$, $p = .03$. Prenatal depressive symptoms were associated with maternal perceptions of parent–child dysfunctional interaction, $\beta = .21$, $p = .002$, which were associated with less sensitive parenting, $\beta = -.24$, $p = .009$. Simple slopes revealed that IPV was significantly associated with prenatal depressive symptoms when mothers reported high, $b = 3.62$, $p < .001$, but not low, $b = .92$, $p = .31$, ACEs (Figure 3). Bias-corrected bootstrapping tests indicated that the indirect path involving the interaction between IPV and ACEs, prenatal depressive symptoms, parent–child dysfunctional interaction, and sensitive parenting was significantly different from zero, 95% CI $[-.030, -.001]$. The indirect effect of IPV on sensitive parenting was significant only when ACEs were high, 95% CI $[-.147, -.014]$, not low, 95% CI $[-.073, -.007]$.

Discussion

IPV is a salient risk factor for parenting difficulties in infancy, but little is known about what mechanisms underlie this link and what factors make some caregivers more vulnerable than others. The primary aims of this study were to: (1) test if IPV had indirect effects on sensitive parenting in infancy through prenatal depressive symptoms and postpartum parenting stress and (2) evaluate if maternal ACEs moderated these indirect effects. We found that mothers who experienced IPV reported greater prenatal depressive symptoms, which was associated with stress around parent–child dysfunctional interaction at 12 months postpartum, and ultimately less sensitive parenting. Analyses revealed that indirect effects were moderated by maternal ACEs, such that the link between IPV and prenatal depressive symptoms was only present for mothers who reported high ACEs in their own childhood. These findings expand our understanding of the dynamic interplay between multiple forms of risk in undermining maternal sensitivity during the transition from pregnancy to parenting an infant. Because prior theory and research point to less sensitive caregiving as a major risk factor for child abuse and neglect (Cicchetti & Toth, 2005; Lindhiem et al., 2011), our findings may have important implications for understanding the earliest cascades of risk from maternal exposure to IPV to risk of child maltreatment.

As hypothesized, the link between IPV and depressive symptoms was particularly pronounced for mothers with a history of ACEs, such that IPV was associated with prenatal depressive symptoms only when mothers reported high, but not low, ACEs. This finding is consistent with the stress sensitization hypothesis, wherein exposure to adversity in childhood may heighten mothers' overall sensitivity to later stressors, especially IPV, leading to increases in depressive symptoms (Hammen et al., 2000). When mothers with a history of ACEs experience IPV in adulthood, it may amplify already existing negative emotions (e.g., hopelessness, shame, sadness), which ultimately increases their risk for depressive symptoms. This risk may be most pronounced during pregnancy when women undergo psychosocial changes (e.g., role transition, relationship changes), neural changes, and dramatic shifts in reproductive hormones (Brummelte & Galea, 2010; Misra et al., 2003). However, it is also possible that IPV may serve as a trauma reminder for mothers with a history of ACEs, which may exacerbate symptoms of posttraumatic stress (e.g., negative alterations in cognition and mood; American Psychiatric Association, 2013). Given that depression and posttraumatic stress symptoms are often comorbid, future work is needed to further disentangle these pathways.

Notably, although prenatal depressive symptoms were associated with both forms of parenting stress at 12 months postpartum, only stress around parent–child dysfunctional interaction was associated with less sensitive parenting. Depression's association with both forms of stress is somewhat unsurprising given that prior work has demonstrated associations between depression and parenting stress in mothers (e.g., Gelfand et al., 1992). However, the finding that an association between stress and sensitive parenting was only evident for stress related to parent–child dysfunctional interaction speaks to the crucial role of child-related cognitions as driving parenting behavior. Consistent with cognitive models of parenting (Sturge-Apple et al., 2014), it is possible that when a mother approaches an interaction with her infant with preexisting feelings that her infant does not live up to her expectations and that the interaction will not be rewarding, she may use this as a guide in how she interacts with her infant (e.g., demonstrate less sensitive caregiving behavior). Indeed, recent studies found that stress related to parent–child dysfunctional interaction is specifically linked to less maternal sensitivity (Dau et al., 2019; Smith & Stephens, 2018). However, our findings build on this work by examining this link in the context of a broader cascade model stemming from maternal exposure to IPV in the context of the transition from pregnancy to parenting an infant. This may point to multiple opportunities for intervention with mothers at high risk for child maltreatment.

It is important that our findings be interpreted in the context of the study's limitations. First, because all mothers in this study were living in low-income households, results may not generalize to caregivers with less socioeconomic disadvantage or high affluence. Additionally, our study focused specifically on mothers, but it will be critical for future research to examine how other caregivers (e.g., fathers) are affected by the stressors examined in this study. Second, we drew upon observational assessments of maternal sensitivity, but most of the measures used to assess key constructs relied on maternal report, which could introduce informant bias in the nature and strength of the associations. It will be important for future work to test whether these findings replicate using multiple methods and informants (e.g., diagnostic interviews, health records, practitioner report).

Third, our longitudinal design is a strength of this study, but it will be critical for future work to test this developmental cascade using more than two time-points to more definitively determine directionality and timing of indirect effects. Fourth, although our focus on examining maternal ACEs as a moderator of these indirect effects was guided by prior research and theory, it will be critical for future research to examine whether other factors (e.g., social support, clinical service utilization, child temperament) may buffer or exacerbate links in this cascade of risk. We also used the 10-item ACEs Questionnaire; future research should test our hypotheses using more in depth assessments of maternal childhood adversity. Similarly, our study focused on depressive symptoms as a mental health mechanism, but it will be important for future research to examine how other forms of psychopathology commonly associated with IPV and ACEs (e.g., posttraumatic stress symptoms, anxiety symptoms, substance use disorders) may play a role in these processes. Furthermore, mothers' neurobiology (e.g., brain activation in response to infant cues) and internal representations of their infant and the mother-infant relationship may also be important intermediary mechanisms that warrant future study. Fifth, the PHQ-9 has been validated in samples of pregnant women (e.g., Sidebottom et al., 2012), but it is important to acknowledge that this is a screening tool, and some of its items may overlap with normative experiences related to pregnancy (e.g., feeling tired or having little energy, poor appetite or overeating). Therefore, our findings should be replicated with other assessments of depression (e.g., Hamilton Depression Rating Scale, Beck Depression Inventory, Edinburgh Postnatal Depression Scale). Furthermore, because mothers were recruited in pregnancy, we did not obtain assessments of depression prior to pregnancy, which will be important to account for in future studies. Finally, our main aim in this paper was to examine a cascade of risk that may lead to later child maltreatment, but it will be important for future work to directly test whether these effects are present in samples of children with direct assessments of maltreatment and/or substantiated reports of abuse and neglect (e.g., child protective services involvement).

In summary, our study was designed to examine whether prenatal IPV had indirect effects on less sensitive caregiving at 12 months postpartum through prenatal depression and postpartum parenting stress. Findings revealed that mothers who experienced IPV endorsed greater prenatal depressive symptoms, which was in turn linked with higher ratings of parenting stress around parent-child dysfunctional interaction, which was associated with less sensitive parenting behavior during a mother-infant free play task. Interestingly, these indirect effects were strongest for mothers who had a history of ACEs. Beyond the singular effects of one type of adversity, our findings add to the small body of literature demonstrating the multiplicative effects of ACEs and IPV on prenatal depressive symptoms (Tung et al., 2019). Results show that mothers exposed to both ACEs and IPV are at the greatest risk for experiencing depressive symptoms during pregnancy, which then increases parenting stress and ultimately less sensitive parenting of their infants. Considered in the context of intergenerational transmission of trauma or stress, this may be one initial pathway by which adversity in the parent generation contributes to maltreatment in the child generation, via maternal insensitivity (Lehrner & Yehuda, 2018).

Our findings may have important clinical implications for promoting maternal and infant health and reducing risk for future child maltreatment. An initial step may be to screen

women receiving perinatal care at obstetrics/gynecology clinics for multiple risk factors, including IPV, ACEs, and depressive symptoms, given that each of these risk factors are rarely asked about simultaneously (Connelly et al., 2013), yet their combination launches a cascade of risk for poor health outcomes and future parenting difficulties. Another important avenue for intervention may be through home visiting programs, which serve mothers of infants with the goal of promoting positive parent-child interactions and healthy infant development, while reducing risk for child maltreatment (Duffee et al., 2017). One of the major goals of most home visiting programs is to enhance sensitive parenting behaviors. Our findings suggest that considering how mothers' experiences of IPV and ACEs, and in turn prenatal depression and postnatal parenting stress, impact their ability to parent sensitively is paramount. Without directly taking these factors into account and addressing them both prenatally and postnatally, it may be difficult for home visiting providers to alter maternal sensitivity through psychoeducation and modeling alone. These findings may also inform the development and refinement of evidence-based interventions for maternal depression (e.g., considering and addressing current IPV and past ACEs in treatment goals) and for infants living in contexts of risk (e.g., identifying ways to reduce parenting stress in mothers who have been depressed; advancing trauma-informed interventions for parents). For example, existing interventions aimed at promoting healthy parent-child relationships that have been effective in promoting sensitive parenting and positive outcomes for both parents and infants have adopted trauma-informed approaches (e.g., Mom Power; Child-Parent Psychotherapy; Lieberman et al., 2015; Rosenblum et al., 2018). This may illuminate potential pathways for prevention of child maltreatment.

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Figure 1.

Path model examining indirect effects of IPV and ACEs on sensitive parenting through depressive symptoms and parenting stress. Parameter estimates for the structural paths are standardized path coefficients. For clarity, only significant paths and correlations are shown. Note: IPV = intimate partner violence; ACE = adverse childhood experience; PS = parenting stress, P-C = parent-child. * $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 2.

Path model examining moderated indirect effect of IPV by ACEs on sensitive parenting through depressive symptoms and parenting stress. Parameter estimates for the structural paths are standardized path coefficients. For clarity, only significant paths and correlations are shown.

Note: IPV = intimate partner violence; ACE = adverse childhood experience; PS = parenting stress, P-C = parent-child. * $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 3.

Graphical plot of the interaction between IPV and ACEs in predicting prenatal depressive symptoms.

Note. IPV = intimate partner violence; ACE = adverse childhood experience. *** indicates that simple slope is significant at $p < .001$; *ns* indicates that simple slope is not statistically significant.

Table 1.

Descriptive Statistics and Correlations Among Substantive Variables of Interest.

Variable	1	2	3	4	5	6	7	8	9	10	11	12
Prenatal												
1. IPV	–											
2. ACEs	.22*	–										
3. Depressive Symptoms	.27*	.25*	–									
12 Months Postpartum												
4. PS: P-C Dysfunctional Interaction	.01	.07	.20*	–								
5. PS: Parental Distress	.20*	.18*	.43*	.56*	–							
6. Maternal Sensitivity	.12	.01	.03	–.16*	.05	–						
7. IPV	.43*	.09	.29*	.11	.32*	.12	–					
8. Depressive Symptoms	.15*	.25*	.50*	.20*	.59*	.03	.32*	–				
Covariates Included												
9. Socioeconomic Adversity	.03	.01	.06	.08	–.05	–.21*	–.13	–.01	–			
10. Age	.03	.14*	.20*	.01	.08	.07	.04	.10	–.19*	–		
11. Ethnicity (% Hispanic)	–.14*	–.10	–.07	–.07	–.14*	–.01	–.18*	–.17*	.08	–.16*	–	
12. First Time Mother (% yes)	.11	.07	.01	–.13*	–.00	–.04	.12	–.04	–.03	–.25*	–.15*	–
Mean/n	.55	2.34	5.55	30.83	41.04	5.45	.32	3.47	1.69	25.61	118	111
SD/%	.46	2.22	4.58	25.20	29.58	1.47	.40	4.67	.91	5.33	40%	38%
Minimum	.00	.00	.00	5.00	3.00	2.00	.00	.00	.00	18.00	0	0
Maximum	1.77	9.00	20.00	99.00	99.00	8.00	1.46	18.00	3.00	44.00	1	1

Note. IPV = intimate partner violence; ACE = adverse childhood experience; PS = parenting stress, P-C = parent-child. Values reported are based on winsorized and log-transformed data used in primary analyses.

* $p < .05$.