MATERNAL SENSITIVITY AND CHILD REGULATION IN THE CONTEXT OF INTIMATE PARTNER VIOLENCE

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ABSTRACT

Intimate partner violence (IPV) is defined as physical, sexual, or psychological violence perpetrated by a romantic partner. Estimates of prevalence in heterosexual couples range from 10% to over 20% (Hien & Ruglass, 2009), and pregnant women are at similar risk of IPV compared to other women (Gürkan, 2020). IPV exposure can have negative physical and mental health consequences for both women and children. With children, a robust body of literature links maternal pre- and postnatal IPV exposure to behavioral and physiological self-regulatory difficulties in children (for a review, see Bogat et al., 2023). Maternal sensitivity, on the other hand, is an important parenting factor that promotes children's adaptive self-regulation (for a review, see Deans, 2020). The current study ($N = 123$) examines if maternal sensitivity can protect children from the impact of pre- and postnatal IPV exposure on their regulatory capacities, measured physiologically (resting RSA) and behaviorally (externalizing behavior), at age 2.5 years. Findings revealed that only IPV exposure during infancy significantly predicted increased child externalizing behavior. Furthermore, this effect was moderated by maternal sensitivity such that there was no such association between infancy IPV exposure and higher externalizing behavior for children of mothers rated as highly sensitive. Pre- and postnatal IPV exposure were not significantly associated with child resting RSA. Findings suggest that infancy is a sensitive period in which children are at heightened risk for behavioral dysregulation due to IPV exposure, but that parenting behavior can buffer the impact of IPV on children during this time.

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INTRODUCTION

Intimate partner violence (IPV) is defined as physical, sexual, or psychological violence perpetrated by a romantic partner. Over the lifetime, it is estimated that 36% of US women experience some form of IPV (Smith et al., 2018). Among heterosexual romantic couples, estimates of its prevalence have ranged from 10% to over 20%, depending on how it is defined and the specific population from which data is collected (Field & Caetano, 2005; Hien & Ruglass, 2009). Pregnant women are at similar risk of IPV compared to other women (Gürkan, 2020). With the prevalence rates delineated above, it is no surprise that IPV is considered a significant public health, social, and medical concern. One concern associated with the high rates of IPV is its effect on children. A robust body of literature links IPV to a host of behavioral and emotional issues in children (for a review, see Bogat et al., 2023).

The child's self-regulatory capacity is a critical factor that underlies their emotional and behavioral health. Self-regulation refers to behavioral, personal, and biological processes that manage arousal and support adaptive social and goal-directed responses (Vohs & Baumeister, 2011). Research has shown that children who have deficits in the capacity for self-regulation are more likely to develop symptoms of psychopathology (for a review, see Compas et al., 2017). Various factors can influence children's self-regulatory capacity. Research indicates that IPV is one of those factors (Cummings et al., 2009; Hibel et al., 2011; Martinez-Torteya et al., 2016). When IPV is experienced during the prenatal period, it can derail the fetus' developing stress regulatory systems in a process known as "prenatal programming" (Glover et al., 2010; Gray et al., 2017). During the postnatal period, the presence of IPV can lead to physiological and behavioral dysregulation (i.e., externalizing/internalizing behaviors) through its impact on children's felt emotional security (Davies & Cummings, 1998; DeJonghe et al., 2011).

Furthermore, when witnessed by children, IPV can provoke acute stress responses in children and alter their perceptions of the acceptability of aggression in close relationships (Bandura & Walters, 1977; Levendosky et. al, 2013; Minze et al., 2010). Research suggests that the degree to which children's development of self-regulation is derailed by IPV might be contingent upon parenting factors (Katz & Windecker-Nelson, 2006; Hibel et al., 2011; Manning et al., 2014).

Maternal sensitivity is an important parenting factor that promotes children's adaptive self-regulatory capacity (for a review, see Deans, 2020). When children do not have enough psychological resources to cope with their negative emotions on their own, the sensitive parent helps the child to manage their feelings by providing caregiving responses that help the child to understand, manage, and recover from their affective experiences. Over time, this co-regulation in the dyadic context facilitates children's later confidence and capacity to self-regulate (e.g., Perry et al., 2014; Lunkenheimer et al., 2017). Accordingly, high levels of maternal sensitivity can promote children's psychological development and mental health (Deans, 2020). Further, maternal sensitivity can operate as a protective factor—the sensitive mother can buffer the detrimental effects of environmental stress or psychological risk on children's development (Ku & Blair, 2021; Wong et al., 2019). In the current study, I examine the protective nature of maternal sensitivity in the context of IPV. Specifically, I investigate how maternal sensitivity might buffer the impact of pre- and postnatal IPV on toddlers' capacity for self-regulation.

Child Self-Regulation

Externalizing problems characterize various forms of psychopathology and are thought to stem in part from deficits in self-regulation (Bradley, 2000; Eisenberg et al., 2012). In young children, however, rather than viewed as symptoms of psychopathology as they are with older children, externalizing symptoms are often characterized as deficits in behavioral self-regulation

(Boyce & Ellis, 2005). When considered alongside family and neighborhood risk, toddler externalizing behavior is predictive of problem behavior upon school entry, particularly for boys (for a review, see Campbell et al. 2000). Currently, toddlerhood is seen as a sensitive period in which the budding self-regulatory capacities of the child become readily apparent—a period in which researchers and clinicians can assess the developing regulatory capacity of the child and predict future functioning (for a review, see Skovgaard, 2010).

Externalizing behavior in toddlers takes the form of poor behavioral regulation (i.e., aggression, defiance, and hyperactivity). Boys display significantly higher levels of externalizing problems (Campbell et al., 2000; Mendez et al., 2016; Paz et al., 2021). Furthermore, there is a large body of research that links externalizing problems to inadequate self-regulation (Olson et al., 2017; White et al., 2013). Children with externalizing problems have been characterized as having deficits in effortful control, or "the ability to inhibit a dominant [behavioral] response to perform a subdominant response" (Rothbart & Bates, 1998, p. 137). Examples of how children effortfully control their behavior include adjusting vocal volume, delaying gratification, complying with parent instructions, and resisting temptations. Effortful control is central to positive development, as children must regularly inhibit dominant behavioral responses to engage in a prosocial manner with their parents and their peers (Olson et al., 2005). Without this capacity for regulation, toddlers can be reactive, which can lead to expressions of maladaptive behaviors that confer risk for strained familial relationships, peer rejection, and later academic failure (Campbell et al., 2010; Eisenberg et al., 2000; Kochanska et al., 2000; Tichovolsky et al., 2013). With the maturation of early attentional neural networks, temperamental effortful control is thought to emerge around the time children are 12-months old, with individual differences

becoming more readily observable throughout toddlerhood (Kochanska et al., 2000; Posner & Rothbart, 2000).

Physiological factors make up an additional component of children's self-regulatory profile. In fact, it has been argued that physiological factors *underlie* children's capacity for behavioral regulation (Calkins, 2009; Eisenberg et al., 2012). While a one-to-one mapping between physiology and behavior is not supported by research, there is ample evidence to suggest that physiology is a fundamental component of adaptive self-regulation (Vasilev et al., 2009). The activity of the parasympathetic branch of the autonomic nervous system, specifically, with its action directed towards downregulation and homeostasis, is considered an index of children's self-regulation capacity (Hastings et al., 2014a; Zisner & Beauchaine, 2016).

The autonomic nervous system (ANS) conveys rapid physiological adjustment through the action of both the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The SNS coordinates physiological processes that prepare for nimble and reactive responses to perceived threats. This reaction is balanced by the action of the PNS, which organizes bioregulatory responses, such as lowered heart rate and muscle relaxation, to recover from periods of SNS activity (McCorry, 2007).

PNS activity is often assessed using measures of heart rate variability (HRV) and resting sinus arrythmia (RSA). HRV and RSA are terms often used interchangeably, although there are subtle differences. Heart rate variability (HRV) refers to the natural variability in the interbeat interval between successive heart beats. RSA, on the other hand, involves measuring the natural heart rate variability in response to respiration and can be assessed at rest or in response to stimuli. RSA is an index of vagal tone, which is itself an index of parasympathetic control over the cardiovascular system via the vagus nerve. RSA is often measured via the high-frequency

(HF) band of the heart rate—that which is responsive to respiration (Gray et al., 2017). Nonetheless, even when respiration is not measured, HRV is often referred to as RSA. Following convention, the current study uses RSA and HRV interchangeably.

When measured at rest, cardiac variability is a physiological marker of self-regulation (Bush et al., 2015). That is, high resting HRV is thought to indicate a capacity to flexibly adapt and respond to external environmental demands. Similarly, high RSA signals a greater capacity to upregulate the cardiovascular system in response to threat—a process known as releasing the "vagal break." Low resting HRV, on the other hand, indicates a diminished capacity to upregulate the cardiovascular system as PNS activity (as indexed through RSA) is already low. The ability to flexibly alter one's autonomic activity in response to environmental demands as captured via measures of cardiac variability is considered essential for the promotion of emotion regulation, attention/focus, and social engagement (Bellato et al., 2023; Porges, 1995).

Measures of cardiac activity at baseline have received attention as a physiological index of regulatory capacity in young children (for a review, see Bellato et al., 2023). In both toddlers and older children, high baseline RSA has been associated with behavioral adaptation, positive psychological adjustment, attentional control, emotion regulation, and adaptive distress regulation (Beauchaine, 2015; Bellato et al., 2023; Porges, 2007; Hastings et al., 2014b). Furthermore, there is considerable evidence showing an inverse relation between baseline RSA and externalizing problems in children (Beauchaine, 2015). However, the research is not entirely consistent, with some studies finding no association between resting measures of cardiac variability and self-regulation (Eisenberg et al., 2012; Fanti et al., 2019). Despite the mixed findings, which may be due to sample biases or other methodological factors, recent reviews suggest that vagal tone at rest (i.e., RSA) is a transdiagnostic marker of children's capacity for

self-regulation (Bellato et al., 2023). In sum, past work suggests that high resting HRV is associated with the most adaptive outcomes; that is, resting HRV demonstrates a positive linear relationship with measures of adaptive self-regulation.

Although PNS regulation and the capacity for behavioral regulation are intimately intertwined, they are nonetheless distinguishable processes that coalesce to make up children's overall self-regulatory capacity. For example, a child might be highly physiologically reactive and dysregulated as indexed by their elevated heart rate response to stress but present as behaviorally regulated. On the other hand, a child might be behaviorally disruptive and aggressive while maintaining a regulated physiological profile. In support of this notion, Calkins & Dedmon (2000) detected no differences in resting RSA when comparing toddlers with aggressive/destructive symptoms to those with few such behaviors. Physiological and behavioral indices of dysregulation measure different processes and, when measured separately, assess distinct qualities of the overall profile of self-regulation.

A host of environmental, interpersonal, and psychological risk factors have been established as risk factors for the development of dysregulation in early childhood. For example, neighborhood and socioeconomic risk factors, such as poverty, stressful life events, and residential instability, show associations with childhood externalizing disorders (for a review, see Curtis et al., 2013) and physiological dysregulation (e.g., Roubinov et al., 2018). Furthermore, various parenting factors can confer risk for behavioral and physiological dysregulation in children (for a review, see Zimmer-Gembeck et al, 2022). Overall, the research suggests that environmental stress and instability and parenting/family factors are all important factors to consider when examining the development of behavioral and physiological dysregulation in young children.

IPV and Its Effects on Children

Maternal IPV can confer elevated risk for self-regulatory problems in children. For example, maternal exposure to IPV has been associated with increased withdrawal/internalizing symptoms, externalizing symptoms, and temperamental difficulties in infants and school-aged children (for a review, see Bogat et al., 2023). When children witness IPV themselves, it can lead to short- and long-term behavioral issues that can affect children's relationships throughout development (for a review, see Wood & Sommers, 2011). Interparental conflict, a phenomenon closely related to IPV, has also demonstrated a reliable association with aggression in school-aged children and adolescents (Kinsfogel & Grych, 2004; Minze et al., 2010). Evidence suggests IPV exposure during pregnancy in particular can impact young children's mental health due to the way it derails the developing physiological regulatory systems of the fetus (Lannert et al., 2014; Levendosky et al., 2016).

In terms of physiology, there is a now a significant body of literature linking both pre- and postnatal maternal IPV to children's HPA axis dysregulation (Berg et al., 2022; Hibel et al., 2011; Kuhlman et al., 2018; Martinez-Torteya et al., 2016). Generally, the literature suggest that IPV predicts higher basal cortisol in children, but that patterns of heightened or attenuated cortisol reactivity is contingent upon several other factors, such as children's developmental stage, IPV frequency, as well as the psychological profile of children. In comparison to the work on the HPA axis, there is considerably less research on the impact of pre- and postnatal IPV on children's autonomic nervous system (ANS) functioning. Limited findings do suggest, however, that IPV exposure can lead to heightened ANS reactivity in toddlers and school-aged children (Cummings & Davies, 2002; El-Sheikh et al., 2007). Additionally, several studies found that vagal tone, a marker of parasympathetic activity, moderated the relation between interparental

conflict and child adjustment (e.g., El-Sheikh et al., 2007). Davies and colleagues (2009), however, found that interparental aggression was associated with diminished SNS activity in children. Despite some inconsistent findings, in general, the findings in this area support the sensitization view (Cummings & Davies, 2002); that is, chronic exposure to aggressive conflict can lead to repeated SNS activation and increased reactivity, which overtime can have a dysregulating effect that limits children's ability to respond adaptively to their environment.

When occurring during pregnancy, the impact of IPV on children's stress physiology may be particularly salient. During pregnancy, the fetus rapidly develops, and during this sensitive period, the fetus is particularly susceptible to insults (Charil et al., 2010; Pluess & Belsky, 2011; O'Donnell et al., 2009). Stressful events experienced by the mother, such as IPV, can lead to an influx into the fetal environment of cortisol, epinephrine, and norepinephrine—adrenal hormones that have neurotoxic properties and readily cross the placenta to reach the fetal brain (DiPietro et. al, 2003; LeWinn et al., 2009; Li et al., 2012; Monk et. al, 2000). Researchers have proposed that maternal stress can "program" the hypothalamic-pituitary-adrenal (HPA) axis and autonomic nervous system (ANS) of the fetus, thus influencing the biobehavioral responses after birth (Glover et al., 2010; Gray et al., 2017; O'Donnell et al., 2009).

Beyond prenatal programming, a number of theories have been proposed to explain the correspondence between IPV and child dysregulation. The relational model of trauma suggests that the association between maternal trauma exposure and children's regulatory difficulties may be partially explained by the impact of trauma on the quality of the parent-child relationship (Scheeringa & Zeanah, 2001). The mother's PTSD symptoms may interfere with her ability to perceive and respond to her child's cues in a way that scaffolds adaptive self-regulation for the child. This indirect path has received empirical support (Lannert et al., 2014); however, a direct

pathway has also been proposed in which children's regulatory difficulties emerge due to observing symptoms of dysregulation in the parent (Samuelson et al., 2017). In other words, expressions of fear, anger, and irritability might be learned and internalized by the infant. Infants may be at higher risk for being impacted by dysregulation that IPV exposure causes in the mother due to their close proximity to and dependence on the mother. Furthermore, during infancy, patterns of behavioral and physiological synchrony between the mother and child help to organize the infant's affective experience and lay the foundation for self-regulation (Bell, 2020). If IPV exposure has a dysregulating effect on the mother, it can have a detrimental impact on mother-child synchrony and thus negatively impact the infant's budding self-regulatory capacity (Azhari et al. 2019; Hoyniak et al., 2021). The impact of maternal trauma exposure on children may be pronounced during infancy given infants' rapid learning and development, limited coping skills, and proximity to and dependance on the caregiver (for a review, see De Young et al., 2011).

While infants do not have the cognitive capacity to deduce that IPV has occurred if they were not present when it occurred, toddlers do. As such, toddlers may be at risk to be negatively impacted by IPV as they can infer that interparental violence may have occurred (i.e., by noticing bruises and scratches or picking up on the emotional climate of the family) even if they did not witness or experience it themselves (DeJonghe et al. 2011). Because of this, toddlers may be more likely to experience emotional insecurity and develop behavioral problems in families in which violence is present compared to younger children. It is clear that IPV occurring during infancy, toddlerhood, and beyond can have self-regulatory consequences for children (Howell et al., 2016), however, little research directly compares the impact of IPV exposure during infancy and toddlerhood.

Finally, the context in which IPV typically happens, however, should also be considered as IPV often co-occurs in the context of socioeconomic disadvantage (Kessler et. al, 2001). Parental low education, economic disadvantage, and unemployment are associated with many of the negative mental health outcomes for children that have been empirically linked to IPV exposure. It is possible that these contextual factors account for some of the relationship between IPV and child externalizing and internalizing symptoms.

Maternal Sensitivity

Maternal sensitivity has been linked to a host of positive child outcomes (for a review, see Deans, 2020). Maternal sensitivity can also be a protective factor for children in the context of psychological risk or environmental stress. In several studies, maternal sensitivity protected children with traits associated with risk for mental health problems (i.e., disinhibition, anger proneness) from developing traits of psychopathology (Buck, 2015; Wong et al., 2018). There is also compelling evidence to suggest that some mothers are able to provide sensitive care to their children despite personal hardship, and that this might protect children from the negative effects of such hardship (Bouvette-Turcot et al., 2017; Palermo et al., 2018). For example, maternal sensitivity can be a protective factor for children's psychological development in the context of environmental stressors, such as poverty (Ku & Blair, 2021; Whittaker et al., 2011) and trauma (Borelli et al., 2019; McLaughlin & Lambert, 2017).

The foundation of the mother's ability to parent sensitively is thought to stem from her own experiences of being parented as a child. During childhood, the internal working model (IWM)—the mental scheme that helps individuals predict and understand the environment, engage in survival enhancing behaviors such as proximity maintenance, and develop a psychological sense of security—undergoes rapid development as children navigate their

immediate social world (Bretherton, 1985). The IWM forms in early childhood and goes on to guide the individual's relationship and caregiving behaviors across the lifespan, although caregiving behaviors can change in response to salient relationship experiences (e.g., Casanueva et al., 2008; Doyle & Cicchetti, 2017). For example, some research has established a link between IPV and insensitive parenting (Gustaffson et al., 2015; Levendosky et al., 2012). Studies have also proposed maternal sensitivity as a mediator between mothers' experiences of IPV and child outcomes (e.g., Gustafsson et a., 2012). Other studies, however, have found no relation between IPV and several indices of parenting sensitivity (e.g., Huth-Bocks & Hughes, 2008; Kalil et al., 2003). Furthermore, around 40% of mothers abused in pregnancy have been found to have balanced representations of their child, a concept that is linked to positive parenting and sensitive caregiving (Huth-Bocks et al., 2004b). The inconsistencies in the findings raise the possibility that maternal sensitivity may function as a moderator rather than a mediator in the association between IPV and children's adjustment. This would make sense in theory, since maternal sensitivity is a construct that is fundamentally rooted in mother's early experiences of being parented by their primary caregiver(s) (Benoit & Parker, 1994; Main et al., 1985). Recently, one innovative study showed that mother's experience of being parented by a sensitive caregiver during infancy predicted their electrophysiological neural responses and cognitive appraisals in response to infant cries during midlife, with a history of sensitive caregiving predicting approach-oriented responses and fewer negative appraisals (Martin et al., 2018). As such, while it may be impacted by later adult experiences, the fundamental internal working model underlying mothers' sensitive parenting capacity may be somewhat independent of later relationship experiences.

Building on the body of literature cited above that shows maternal sensitivity to be a protective factor across a variety of domains of risk, some research investigates parenting as a factor that might exacerbate or buffer the negative impact of IPV on children (Katz & Windecker-Nelson, 2006; Hibel et al., 2011; Manning et al., 2014). One study found that maternal sensitivity at 7-months postpartum moderated the association between IPV and cortisol reactivity, such that only children exposed to maternal insensitivity and high violence exposure exhibited increased cortisol reactivity at 2 years old (Hibel et al., 2011). Furthermore, in a study of domestic violence, parental emotion coaching moderated the relation between violence exposure and child behavior problems (Katz & Windecker-Nelson, 2006). Finally, Manning et al. (2014) demonstrated in a longitudinal study how sensitive parenting could buffer the risk posed by interparental violence on children's externalizing behaviors and prosocial development.

The current study builds upon this work by examining if the protective nature of maternal sensitivity is dependent on the timing of maternal trauma exposure. Specifically, the current study tests if maternal sensitivity can be a protective factor for children's development of self-regulation in the context of IPV exposure during pregnancy, infancy (0 – 6-months postpartum), and early toddlerhood (6-months – 2.5-years postpartum). Furthermore, as previous work in this area tests the protective nature of maternal sensitivity in the context of IPV exposure for either children's behavioral or physiological regulation, the current project includes both a physiological and behavioral measure of children's self-regulation as they represent distinct aspects of the child's self-regulatory profile. Findings may reveal sensitive periods in which children are at heightened risk to be negatively affected by IPV. These sensitive periods, if identified, may be considered important developmental stages in which there is a heightened need to assess and intervene upon violence in the home. Furthermore, findings from the current

study may suggest that interventions designed to bolster parenting sensitivity may be particularly called for children of mothers exposed to IPV.

The Present Study

Using a multi-method approach, the current study examines whether maternal sensitivity buffers the impact of maternal IPV exposure on children's self-regulation. Specifically, the current study tests whether maternal sensitivity protects children's self-regulation (assessed at the behavioral and physiological levels) from the detrimental impacts of IPV exposure at three times points: during pregnancy (henceforth referred to as prenatal IPV exposure), from birth – child age 6-months (henceforth referred to as infancy IPV exposure), and from child age 6-months – 2.5-years (henceforth referred to as early toddlerhood IPV exposure). There are three major aims and hypotheses for the current study.

Aim 1

1a. Examine the main effect of prenatal IPV exposure on child externalizing behavior and child resting RSA.

1b. Examine the main effect of infancy IPV exposure on child externalizing behavior and child resting RSA.

1c. Examine the main effect of early toddlerhood IPV exposure on child externalizing behavior and child resting RSA.

Hypotheses 1a – c: Prenatal, infancy, and early toddlerhood IPV exposure are each associated with higher child externalizing behavior and lower child RSA. Drawing on the IPV literature, prenatal IPV has a larger detrimental impact on both indices of child self-regulation than postnatal IPV.

Aim 2

2. Examine the moderating role of maternal sensitivity in the association between prenatal/postnatal IPV and child externalizing behavior.

Hypothesis 2: Maternal sensitivity has a buffering effect, so that the association between IPV (at any point) and (higher) child externalizing behavior are weaker at higher levels of maternal sensitivity.

Aim 3

3. Examine the moderating role of maternal sensitivity in the association between prenatal/postnatal IPV and child resting RSA.

Hypothesis 3: Maternal sensitivity has a buffering effect, so that the association between IPV (at any point) and (lower) child resting RSA are weaker at higher levels of maternal sensitivity.

METHODS

Participants

Data were drawn from the Prenatal Stress Study (PSS), an ongoing longitudinal study of the impact of prenatal stress on children and mothers (MPIs Levendosky, Bogat, Lonstein & Muzik; R01HD085990; R01HD100469: for an overview of study methods, see Levendosky et al., 2021). Participants were recruited from several Midwest cities and towns via flyers posted in obstetrics offices, the community (e.g., Women, Infants, and Children [WIC] offices, laundromats, libraries, public parks), and on social media. To determine study eligibility, women were first screened by phone. Eligibility criteria included being between 15 and 20 weeks pregnant at initial interview, being between 18 and 35 years of age, being involved in a romantic relationship with a man for at least 6 weeks at some point during pregnancy, and being able to read and speak English. Additionally, participants had to either endorse any experiences of IPV in the past year or be Medicaid eligible based on household income and endorse two or more family stressors (i.e., family conflict, neighborhood violence, food insecurity, or other money problems). Because the longitudinal study focuses in part on stress hormones, participants were excluded if they had any medical conditions (e.g., endocrine disorders) or lifestyle factors (e.g., working night shifts) that are known to affect salivary cortisol measures (hormonal data were not included in the present analysis).

One hundred and twenty-three mothers and children were included in the sample based upon having data on (1) the moderator variable (maternal sensitivity), (2) at least one of the outcome variables (child externalizing, resting RSA), and (3) at least one of the predictor variables (prenatal IPV, infancy IPV, early toddlerhood IPV). In this sample, mothers mean age was 27.5 years (*SD* = 4.3). In the study sample, approximately 55% endorsed past-year IPV at

the first pregnancy study visit. Average monthly income was approximately $2,800 (*SD* = 2,324). Other demographic characteristics of the sample (education, employment, race/ethnicity, etc.) are included in Table 1. Two hundred and forty-three mothers were excluded based on not having complete data for all variables in at least one regression model or due to dropping out of the study before outcomes were measured. Chi-squared tests of independence were performed to examine if those who dropped from the study differed on demographic characteristics. The participants in the current study did not differ from those that dropped and/or had missing data in their total family income nor their race/ethnicity (non-white/Hispanic vs. white). However, these groups of participants did differ in their level of education (high school or less vs. college or technical school or more), X^2 (1, N = 396) = 4.26, p = .039. Those participants in the current study were more likely than those who dropped or had missing data to have received college, technical, or graduate education.

Procedures

The larger PSS follows women from pregnancy through the time when their children are four years old with a series of laboratory and at-home study visits. The current study uses data from pregnancy until children are 2.5 years old. Before each study visit, mothers gave their informed consent and were financially compensated after the study visit. Women were first assessed when they were between 15 and 20 weeks pregnant in the project office. During the first study visit, mothers completed survey measures on their demographics and their IPV exposure during pregnancy, as well as on a range of other dimensions not relevant to the current analyses. During study visit two (occurring between the 23rd – 25th week of pregnancy) and study visit three (occurring between the 32nd – 34th week of pregnancy), mothers reported on their IPV exposure since the last study visit and were administered several interviews and self-report

measures not relevant to the current analyses. During study visit four (occurring at one month after birth), mothers reported on their IPV exposure between study visit three and birth. During study visits five and six (occurring when the child was 6 months old and 2.5 years old, respectively), among other study tasks, mothers reported on their postnatal IPV exposure and engaged in a series of behavioral tasks with their child.

During study visit six, each mother and child dyad watched a nature video for two minutes to establish "at rest" physiological activity. ECG data was collected from the child during this time. During the video, they were instructed and reminded not to touch each other. After the video task, each mother and child dyad engaged in a 10-minute free play interaction in a room with age-appropriate toys. Mothers were instructed to play with their child as they typically do so at home.

Measures

Demographics. Demographic information was collected from mothers at each study visit. Maternal race/ethnicity, age, education, relationship status, and family income was collected at study visit one. Based on the notion of cumulative risk (Huth-Bocks, et al., 2004a), these data were used to compute a maternal demographic risk variable that comprised summed dichotomous variables accounting for the presence/absence of racial ethnic minoritized status (i.e., non-white racial/ethnic minority), single, low education (i.e., high school or less), and low family income (i.e., below Medicaid/government aid cutoff). Child sex and race/ethnicity were reported by mothers at visit five.

Pre- and Postnatal IPV Exposure. The *Severity of Violence Against Women Scales (SVAWS; 46-item; Marshall, 1992)* was administered at each study visit to assess mothers' experiences of IPV. The SVAWS assesses participants experience of physical, emotional, and

sexual violence using a 4-point scale ranging from "Never" to "Many Times." Items include "demanded sex whether you wanted it or not" and "kicked you." Severity is commensurate with frequency of experiences as obtained though summing the item scores. Mothers reported on their prenatal IPV exposure at study visits one, two, three, and four. These reports were summed to create the prenatal exposure to IPV variable. Prenatal IPV scores were only computed if SVAWS data was available for at least three of the four timepoints. At study visit five (6 months postpartum), mothers reported on their IPV exposure from birth – 6-months postpartum. At study visit six (2.5 years postpartum), mothers reported on their IPV exposure from 6-months – 2.5-years postpartum. Internal consistencies for the six waves of data collection ranged from .92 to .96.

Maternal Sensitivity. The *National Institute of Child Health and Human Development (NICHD) Qualitative Scales of the Observational Ratings of Mother–Child Interaction Scales* (NICHD Early Child Care Research Network, 1999) were used to code maternal sensitivity from the 10-minute free play interaction. The NICHD scales were developed as an observational measure for use in a national study of childcare and are designed for children between the ages of six months and seven years of age. The NICHD Scales contain global ratings for both mothers and children that focus on behavioral cues and responses between the dyad. The current study utilized the scale that captures the mother's sensitivity to the child's cues when they are not distressed (i.e., "Sensitivity/responsivity to Nondistress"). The Sensitivity to Nondistress scale measures the degree to which the mother responds in a well-paced and appropriate manner to their child's social gestures, play cues, and expressions. The NICHD Scales also contain a "Sensitivity/responsivity to Distress" scale that was not used in the current study. This is because less than 10% of mothers in the sample received this code due to the fact that children rarely

became upset during the free play task. All NICHD parenting codes are rated on a global 5-point scale, ranging from 1 (*not at all*) to 5 (*highly characteristic*). Coders were blind to the family context, including IPV. To establish reliability, coders double coded 25% of the videos, with additional videos being double coded to assure continued reliability throughout. Among all coders, interrater reliability for the Sensitivity to Nondistress scale ranged from .80 to .85.

Child Externalizing Behavior. The *Child Behavior Checklist for Ages 1.5 – 5 (CBCL; 99-item; Achenbach & Rescorla, 2001)* was completed by mothers when children were 2.5 years old. Mothers were given a list of symptoms and asked to indicate how true the statement was for their child within the last six months on a 3-point scale from "Not True" to "Very True or Often True." Higher summed scores on the externalizing scale indicate higher levels of rule-breaking, attentional, and aggressive behavioral problems. Items that contribute to the externalizing score include: "My child is easily frustrated," "My child has temper tantrums," and "My child can't stand waiting and wants everything now." The CBCL is a reliable and valid measure for externalizing symptoms in toddlers (Rescorla, 2005). Cross-cultural studies also support the validity of this measure (Ivanova et al., 2010). Internal consistency for the externalizing scale in the current study was $\alpha = .95$.

Child Resting Sinus Arrythmia. Child resting sinus arrythmia (RSA) was collected using equipment and software from Mindware Technologies. Before the two-minute no-touching video task, used as a baseline measure in this report, electrodes were placed axially on the left and right rib cage and centrally on the stomach. The bioamplifier was set for bandpass filtering at frequencies of .1 and 1000 Hz. The rising edges of R waves were automatically identified with a multiple-pass, self-scaling algorithm and were then visually inspected and corrected manually when possible. Distances between successive R waves were the interbeat intervals (IBI).

RSA data were derived from IBI data using spectral analyses. Prorated IBIs were detrended with a high-pass filter, then Fourier analyses were applied to residual IBI data for each epoch. RSA was estimated from power spectral analysis, using the 0.3–0.8 Hz band (Alkon et al., 2011). Data were non-normal and were log-transformed for analyses.

Plan of Analysis

A moderation model was proposed to test whether maternal sensitivity moderates the effect of pre- and postnatal IPV on child externalizing behavior and child baseline RSA. The proposed model was tested using the following analyses.

Bivariate correlations between all study variables were run to examine the patterns of association. To test the moderation hypotheses, a series of hierarchical linear regressions were run. Maternal demographic risk and child sex were included as covariates in these analyses due to their respective associations with maternal sensitivity (Pungello et al., 2009; Rifkin-Graboi et al., 2015) and child self-regulation (Hastings et al., 2019b; Paz et al., 2021; Rudd et al., 2016).

The hierarchical linear regressions to test the moderating role of maternal sensitivity in the association between prenatal IPV and child outcomes (child externalizing, child resting RSA) included three steps. In the first step of this regression, the covariates were entered. Next, prenatal IPV exposure and maternal sensitivity were included in the model. In the third step of the model, the interaction between prenatal IPV exposure and maternal sensitivity was entered.

Due to the fact that the infancy and early toddlerhood IPV variables measure IPV exposure across very different lengths of time (birth – 6-months postnatal and 6-months – 2.5-years postnatal), they were tested in separate models. The hierarchical linear regressions to test the moderating role of maternal sensitivity on the associations between infancy/early toddlerhood IPV exposure and child outcomes (child externalizing, child resting RSA) included

three steps. In the first step, the covariates were entered. Next, postnatal (infancy/early toddlerhood) IPV exposure and maternal sensitivity were included in the model. In the third step, the interaction between postnatal (infancy/early toddlerhood) IPV and maternal sensitivity was entered. To reduce multicollinearity, all three IPV indices and maternal sensitivity were mean centered prior to analyses. The regression analyses were run in SPSS and employed list-wise deletion. For each regression model, participants that had missing data on one or more of the variables were excluded.

RESULTS

The descriptive statistics of and correlations between study variables are presented in Table 2. There was a significant correlation between prenatal and infancy IPV exposure [$r(148) = .51$, $p < .001$)] as well as between prenatal and early toddlerhood IPV exposure [$r(146) = .32$, $p < .001$)]. Child externalizing behavior was also significantly associated with both prenatal IPV exposure [$r(146) = .18$, $p = .028$)] and infancy IPV exposure [$r(145) = .28$, $p = .006$)]. The results of all hierarchical linear regressions are presented in Tables 3 - 5.

Missing Data

For the participants for whom a prenatal IPV variable was calculated ($N = 120$), ten were missing prenatal IPV data from study visit 1, six were missing prenatal IPV data from study visit 2, ten were missing IPV data from study visit 3, and six were missing IPV data form study visit 4. There were 118 and 119 observations for the infancy and early toddlerhood variable, respectively. Maternal sensitivity data was available for 123 mothers in the current study. In terms of the outcome variables, child externalizing data was available for 119 children and resting RSA data was available for 81 children (RSA data from 42 children were unavailable due to movement artifacts, hardware malfunction, or human error).

While the total participant pool comprised 123 participants, each regression analyses had a different, smaller sample size due to the various combinations of missing data. For example, of the 123 total participants, four did not have a prenatal IPV variable (due to having missing data for 2+ reports of pregnancy IPV) but had data for one or both postnatal IPV indices, and thus could be included in one or both of the analyses involving postnatal IPV but not in the analyses with prenatal IPV. Five participants did not have infancy IPV data yet had data for either prenatal, early toddlerhood IPV, or for both. Four participants who did not have early

toddlerhood IPV data had data for either prenatal IPV, infancy IPV, or for both. Similarly for the outcome variables, there were four children who did not have data for externalizing behavior yet had data for resting RSA. There were 42 children with missing resting RSA data that had data for externalizing behavior. Taking these missing data into account, the sample sizes for each analysis are listed below.

In the analyses with prenatal IPV, there were 115 participants in the model predicting child externalizing behavior and 72 participants in the model predicting child resting RSA. In the analyses with infancy IPV, there were 113 participants in the model predicting child externalizing behavior and 78 participants in the model predicting child resting RSA. In the analyses with early toddlerhood IPV, there were 118 participants in the model predicting child externalizing behavior and 76 participants in the model predicting child resting RSA.

The Effects of Prenatal IPV on Child Outcomes, Moderated by Maternal Sensitivity.

Prenatal IPV exposure, maternal sensitivity, and their interaction were not significantly associated with child externalizing behavior. There was a significant main effect of maternal sensitivity on child resting RSA (β = .245, *SE* = .133, *p* = .045). Prenatal IPV exposure and the interaction between prenatal IPV exposure and maternal sensitivity were not significantly associated with child resting RSA.

The Association Between Postnatal IPV and Child Outcomes, Moderated by Maternal Sensitivity.

There was a significant main effect of infancy IPV exposure (β = .194, *SE* = .210, *p* = .041) on child externalizing behavior. Furthermore, the interaction between infancy IPV exposure and maternal sensitivity (β = -.219, *SE* = .237, *p* = .045) was significantly associated with child externalizing behavior. Maternal sensitivity was not significantly associated with child

externalizing behavior. Together, these variables explained approximately 6% of the variance in child externalizing behavior, $R^2 = .058$, $F(5, 107) = 2.384$, $p = .043$. At low levels of maternal sensitivity (one standard deviation below mean), infancy IPV exposure was associated with increased externalizing symptoms ($B = 1.04$, $p = .005$). At high levels of maternal sensitivity (one standard deviation above mean), there was no such association ($B = .14$, $p = .579$). Plots of interaction simple slopes can be found in Figure 1.

There was a significant main effect of maternal sensitivity ($\beta = .253$, $SE = .133$, $p = .038$) but not infancy IPV exposure on child resting RSA. The interaction between infancy IPV exposure and maternal sensitivity was not significantly associated with child resting RSA.

Neither early toddlerhood IPV exposure, maternal sensitivity, nor their interaction were significantly associated with child externalizing behavior in the full model. There was a significant main effect of maternal sensitivity ($\beta = .259$, $SE = .137$, $p = .038$) but not early toddlerhood IPV exposure on child resting RSA. The interaction between early toddlerhood IPV exposure and maternal sensitivity was not significantly associated with child resting RSA.

DISCUSSION

Overall, the study hypotheses received partial support. IPV exposure during the child's first 6 months of life predicted higher externalizing behavior when children were 2.5 years old. Furthermore, maternal sensitivity moderated this association, such that there was no such link between infancy IPV exposure and elevated externalizing behavior at high levels of maternal sensitivity. Neither prenatal IPV nor early toddlerhood IPV exposure, nor their interaction with maternal sensitivity, were significantly associated with children's externalizing behavior. Finally, child resting RSA was solely predicted by maternal sensitivity, with high levels of maternal sensitivity predicting higher resting RSA. Attrition analyses revealed that those participants in the current sample had higher education (50.6% received education past the high school level) compared to those who dropped out of the study/were excluded due to missing data (40.1 received education past the high school level). Thus, conclusions should be generalized to those with low education with caution.

The hypothesis that IPV would be linked to higher externalizing behavior in children was partially supported, with infancy IPV exposure predicting elevated child externalizing behavior. This was not the case for prenatal IPV exposure nor early toddlerhood IPV exposure. This suggests that infancy is a sensitive period in which there is heightened potential for IPV occurring to the mother to implicate developmental consequences for the child's behavior regulation. At this age, children are highly dependent on their caregiver(s) and are very often physically touching or in close proximity to their mothers. Because of this proximity, infants may be more likely to be exposed to and pick up on symptoms of dysregulation associated with trauma exposure in the mother compared to when they are older, when they spend more time away from the immediate proximity of the mother. The finding in the current study may reflect

how infants of mothers exposed to IPV internalize affective instability and dysregulation in the mother. This interpretation reflects the direct pathway between trauma in the mother and regulatory difficulties in the child that is described in work using the relational PTSD framework (Samuelson et al., 2017; Scheeringa & Zeanah, 2001). In a similar vein, infants might be particularly sensitive to a negative emotional climate in the home due to their limited coping skills. The close proximity of infants to their mother and their limited coping skills helps to explain findings from the current study, in which IPV exposure during infancy seemed to be most detrimental to children's behavioral regulation capacity.

Maternal sensitivity moderated the association between IPV exposure during infancy and children's externalizing behavior. At low levels of maternal sensitivity, there was a positive association between infancy IPV exposure and externalizing behavior, but there was no such association for children of mothers rated as highly sensitive (see Figure 1). In other words, high levels of maternal sensitivity buffered the effect of maternal IPV exposure on children's behavior dysregulation. This suggests that mothers who are highly sensitive caregivers in the context of trauma exposure can shield their children from some of its developmental consequences. This finding builds upon previous research that finds that parenting factors can buffer the impact of IPV exposure on children's adrenocortical reactivity (Hibel et al., 2011), prosocial development (Manning et al., 2014), and behavior problems (Katz & Windecker-Nelson, 2006). Sensitive mothers are consistently responsive to the behavioral cues of their child in a manner that prioritizes the infant's needs and tend to infer internal states as underlying the manifest behavior of the child (Bretherton, 1985). Sensitive responding to the infant's behavioral expression of dysregulation addresses the internal experience of distress and helps the child to return to a calmer state. Overtime, this pattern of co-regulation helps infant develop confidence in their

budding self-regulatory capacity (Perry et al., 2014; Lunkenheimer et al., 2017). The maternal capacity to scaffold self-regulation in this manner may be particularly important in the context of elevated environmental and family stress (Ku & Blair, 2021; Borelli et al., 2019). In such contexts, the sensitive caregiving that the infant receives may shield them from the risk conferred from their broader context. In the current study, sensitive mothers protected children from the behavioral regulation difficulty associated with IPV exposure during infancy.

Both prenatal and early toddlerhood IPV exposure did not significantly predict child externalizing behavior. There are several potential explanations for the pattern of findings regarding the main effects of IPV exposure at the three different timepoints. First, as described above, these findings suggest that early infancy is a sensitive period in which children's developing behavior regulation is particularly susceptible to be disrupted by IPV. Regarding the null findings with prenatal IPV, children's externalizing behavior was reported on by their mothers when the children were 2.5 years old. It is possible that any negative impact of prenatal IPV exposure on children's behavioral regulation would have waned over this length of time, and thus when other proximal factors are included in the model (i.e., demographic risk, maternal sensitivity), there was no significant effect of prenatal IPV. However, given the volume of research linking prenatal IPV to child behavior problems (e.g., Bianchi et al., 2016; Levendosky et al., 2006), this is unlikely. It is noteworthy that the regression coefficients for both the prenatal and early toddlerhood IPV indices are in the hypothesized direction (β = .165 for prenatal IPV; β = .170 for early toddlerhood IPV). As the current study employed list-wise deletion to handle missing data, it is possible that there was insufficient power to detect the small-to-medium sized effect that is typically found when investigating the impact of maternal IPV exposure on child outcomes (e.g., Martinez-Torteya et al., 2016). Similarly, the hypotheses that maternal sensitivity

would buffer the impact of prenatal IPV and toddlerhood IPV on children's externalizing behavior were not supported. This may be explained by the lack of main effects of prenatal and early toddlerhood IPV on externalizing behavior. While moderation can occur for a predictor that has no direct association with an outcome, these findings suggest that there is no significant association between prenatal/early toddlerhood IPV and child externalizing behavior exposure regardless of the level of maternal sensitivity.

Counter to hypotheses, IPV did not predict lower child resting RSA. The current results suggest that pre- and postnatal IPV do not affect children's physiological flexibility to respond to the environment as measured through baseline cardiac variability. This underscores how behavioral and physiological regulation are distinct indices of self-regulation, as infancy IPV exposure predicted higher externalizing symptoms but not lower child resting RSA. It is possible, however, that significant effects of IPV exposure on children's cardiac regulatory activity would have been detected with a different measure of child RSA. For example, recent studies have used growth mixture modeling to identify non-linear profiles of RSA reactivity based on resting and task-based activity (Roubinov et al., 2021). Furthermore, some research in this area adopting the biological sensitivity framework has positioned measures of child RSA as a moderator rather than an outcome variable (El-Sheikh & Hinnant, 2011). For example, children in homes characterized by interparental conflict are at heightened risk for externalizing problems when they also demonstrate low resting RSA (El-Sheikh et al., 2011). It is possible that the association between IPV exposure and child externalizing behavior is driven by those children

who also display lower resting RSA, however a post-hoc analysis of this question in the current study did not support this relationship[1].

Finally, although not part of study hypotheses, maternal sensitivity was associated with higher child resting RSA. In other words, children of mothers who were coded as highly sensitive had higher heart rate variability at rest, signaling a capacity to physiologically adapt and respond to their environment flexibly. This finding is understandable in light of the role that maternal sensitive behavior has in promoting adaptive self-regulation in children (Jones-Mason et al., 2023). The quality of the maternal-child relationship is linked specifically to enhanced vagal regulation (i.e., decreased in RSA when coping or regulation is required; Calkins et al., 2008). Attachment theory posits that one of the roles of the primary caregiver is to act as a secure base from which the infant can explore (Bretherton, 1985). In their interactions with a sensitive caregiver, infants experience increased activation of the PNS and decreased distress which facilitates the exploration of their environment. The higher resting RSA of toddlers of sensitive mothers may reflect how being parented by a sensitive caregiver who consistently meets their needs has allowed them to flexibly explore their environment from a position of security. However, given the cross-sectional nature of the data on RSA and maternal sensitivity, it is also possible that children with high resting RSA elicit more sensitive parenting. Transactional models of parenting acknowledge the contributions of both parent and child characteristics to the parent-child relationship (Hastings et al., 2019a). Previous work has linked low child resting RSA to more restrictive and less supportive parenting, which is theorized to stem from the parent's experience of these children as difficult and rigid (Kennedy et al., 2004). In the current

[1] Post-hoc analysis in the current study revealed that child resting RSA did not significantly moderate the association between IPV exposure during infancy and child externalizing behavior.

study, it is possible that the regulatory capacity and responsiveness of the children with high resting RSA elicited more sensitive parenting.

The current study has several strengths. First, the sample comprised an at-risk, diverse group of women and children. Given the association between IPV exposure and poorer child self-regulation (Berg et al., 2022; Martinez-Torteya et al., 2016; Minze et al., 2010), this is a particularly appropriate sample for investigating the protective nature of maternal sensitivity. Furthermore, data were drawn from a longitudinal study that spanned over three years. This longitudinal data enabled an examination of the effects of IPV exposure at earlier timepoints on child regulation measured at a later timepoint. An additional strength of the current study is the use of multiple types of data. The current study included physiological (child resting RSA), observational (maternal sensitivity), self-report (IPV exposure), and maternal report (child externalizing behavior) data to investigate study questions. This study, however, is not without its limitations. First, all regression analyses employed list-wise deletion. This is particularly relevant given the attrition from the first study visit to the 2.5-year assessment, when child outcomes were measured. There were over double the participants at the first study visit compared to the last, in part due to the fact the larger PSS is an ongoing study, but the current study only used data from those participants that had data from all data assessment visits. With a different missing data strategy (i.e., FIML), it is possible that the effects of prenatal and early toddlerhood IPV exposure, and their interaction with maternal sensitivity, would have crossed the threshold of significance. Finally, use of only a baseline measure of child RSA is a limitation, as task-based cardiac activity represents an additional component of ANS regulation that is unaccounted for in the current study (Roubinov et al., 2021).

Despite these limitations, this study contributes to the existing literature. First, although a bidirectional relationship cannot be ruled out, the current project uncovered an environmental effect on children's stress physiology. Maternal sensitive caregiving was linked to higher resting RSA in children. Furthermore, to the area of research that examines the protective nature of maternal sensitivity in the context of IPV, this study adds a consideration of timing and an assessment of two different forms of self-regulation in children (behavioral and physiological). Findings suggest that infancy is a sensitive period in which IPV can have a heightened impact on children's behavioral dysregulation and that maternal sensitivity is a protective factor for children during this sensitive period. Infancy may be an age at which it is particularly important to assess and intervene upon violence in the home. Findings suggest that interventions designed to bolster parenting sensitivity may be particularly effective for protecting infants from some of the developmental consequences of maternal IPV exposure.

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APPENDIX

Table 1

Sociodemographic Characteristics of Mothers and Children

	%	*Mean*	*SD*
Mother age		27.5	4.3
Monthly family income		2813	2324
Education			
Graduate degree	12.3		
College degree	27.3		
Post high school technical degree	10.4		
High school	42.9		
Less than high school	7.1		
Relationship status			
Single	8.4		
In a relationship	83.8		
Mother race/ethnicity			
White	50.6		
Non-white/Hispanic	49.4		
Child sex			
Male	55.2		
Female	44.8		
Child race/ethnicity			
White	39.0		
Non-white/Hispanic	56.5		

Note: Missing data for relationship status (12 participants), child race/ethnicity (7 participants)

Table 2

Descriptive Statistics and Intercorrelations

Variable	Demographic Risk	Prenatal IPV	Infancy IPV	Early Toddlerhood IPV	Maternal Sensitivity	Child Externalizing	Child Resting RSA
Demographic Risk	1	.04	-.14	-.05	-.06	-.02	-.10
Prenatal IPV		1	.51*	.32*	-.06	.18*	-.03
Infancy IPV			1	.12	.01	.28*	-.01
Early Toddlerhood IPV				1	-.08	.14	.17
Maternal Sensitivity					1	-.14	.17
Child Externalizing						1	.11
Child Resting RSA							1
M		11.71	1.58	4.22	4.03	11.39	5.47
SD		20.15	5.56	8.81	.94	7.95	1.06

*$p < .05$

Table 3

Summary of Regression Analyses with Prenatal IPV, Maternal Sensitivity, and their Interaction Predicting Child Externalizing Behavior

		Child Ext				Child RSA			
		B	*SE B*	*β*	Sig.	*B*	*SE B*	*β*	Sig.
Step 1	Constant	10.35*	1.50	-	< .001	5.36*	.26	-	< .001
	Child sex	1.25	1.39	.09	.371	.11	025	.05	.662
	DemoRisk	-.33	.61	-.05	.595	-.02	.10	-.02	.867
	R^2		.01		.580		.003		.897
Step 2	Constant	10.94*	1.50	-	< .001	5.23*	0.26	-	< .001
	Child sex	.94	1.39	.06	.501	0.26	0.25	0.12	.309
	DemoRisk	-.40	.60	-.06	.508	0.01	0.10	0.01	.961
	Pregnancy IPV	.06	.04	.17	.083	-0.001	0.01	-0.01	.925
	Maternal Sens.	-1.01	.74	-.13	.178	0.27*	0.13	0.25*	.045
	ΔR^2		.04		.210		0.06		.357
Step 3	Constant	10.94	1.51	-	< .001	5.21	026	-	
	Child sex	.96	1.40	.07	.494	.30	.26	.14	.249
	DemoRisk	-.41	.61	-.06	.499	.003	.10	.004	.976

Table 3 (cont'd)

Pregnancy IPV	.06	.04	.16	.127	-.002	.006	-.04	.760
Maternal Sens.	-.98	.76	-.13	.198	.26	.13	.24	.052
Interaction	.01	.04	.02	.824	.01	.01	.13	.267
ΔR^2		.001		.318		0.02		.345

*$p < .05$

Table 4

Summary of Regression Analyses with IPV Exposure at 6 Months, Maternal Sensitivity, and their Interaction Predicting Child Outcomes

		Child Ext				Child RSA			
		B	*SE B*	*β*	Sig.	*B*	*SE B*	*β*	Sig.
Step 1	Constant	10.398*	1.485	-	< .001	5.361*	.257	-	< .001
	Child sex	1.469	1.388	.100	.292	.108	.246	.051	.662
	DemoRisk	-.442	.604	-.069	.466	-.017	.103	-.019	.867
	R^2		.015		.438		.003		.897
Step 2	Constant	11.104*	1.489	-	< .001	5.111*	.327	-	< .001
	Child sex	.971	1.385	.066	.485	.299	.261	.140	.256
	DemoRisk	-.329	.597	-.052	.583	-.003	.103	-.004	.975
	6-month IPV	.435*	.210	.194*	.041	-.054	.093	-.069	.562
	Maternal Sens.	-.944	.734	-.121	.201	.281*	.133	.253*	.038
	ΔR^2		.050		.117		.059		.317
Step 3	Constant	10.597	1.489	-	< .001	5.174*	.363	-	< .001
	Child sex	1.455	1.386	.066	.296	.291	.264	.137	.273
	DemoRisk	-.068	.602	-.011	.911	.001	.104	.001	.996

Table 4 (cont'd)

6-month IPV	.592*	. 221	.156*	.008	-.022	.122	-.029	.854
Maternal Sens.	-1.502	.774	-.125	.055	.185	.273	.167	.499
Interaction	-.481*	.237	.023*	.045	-.050	.123	-.103	.687
ΔR^2		.035*		.043		.002		.434

*$p < .05$

Table 5

Summary of Regression Analyses with IPV Exposure at 2.5 Years, Maternal Sensitivity, and their Interaction Predicting Child Outcomes

		Child Ext				Child RSA			
		B	*SE B*	*β*	Sig.	*B*	*SE B*	*β*	Sig.
Step 1	Constant	10.438*	1.486	-	< .001	5.404*	.261	-	< .001
	Child sex	1.316	1.382	.090	.343	.070	.250	.033	.780
	DemoRisk	-.377	.602	-.059	.532	-.020	.105	-.022	.850
	R^2		.012		.519		.002		.945
Step 2	Constant	10.500*	1.477	-	< .001	5.268*	.262	-	< .001
	Child sex	11.158	1.381	.079	.404	.226	.257	.107	.382
	DemoRisk	-.339	.595	-.053	.570	-.005	.103	-.006	.960
	2.5-year IPV	.151	.083	.170	.072	.008	.014	.067	.569
	Maternal Sens.	-.837	.739	-.107	.260	.290*	.137	.259*	.038
	ΔR^2		.042		.190		.068		.277
Step 3	Constant	10.315	1.481	-	< .001	5.253*	.266	-	< .001
	Child sex	1.370	1.388	.094	.326	.234	.259	.111	.369
	DemoRisk	-.332	.593	-.052	.577	-.004	.103	-.005	.969

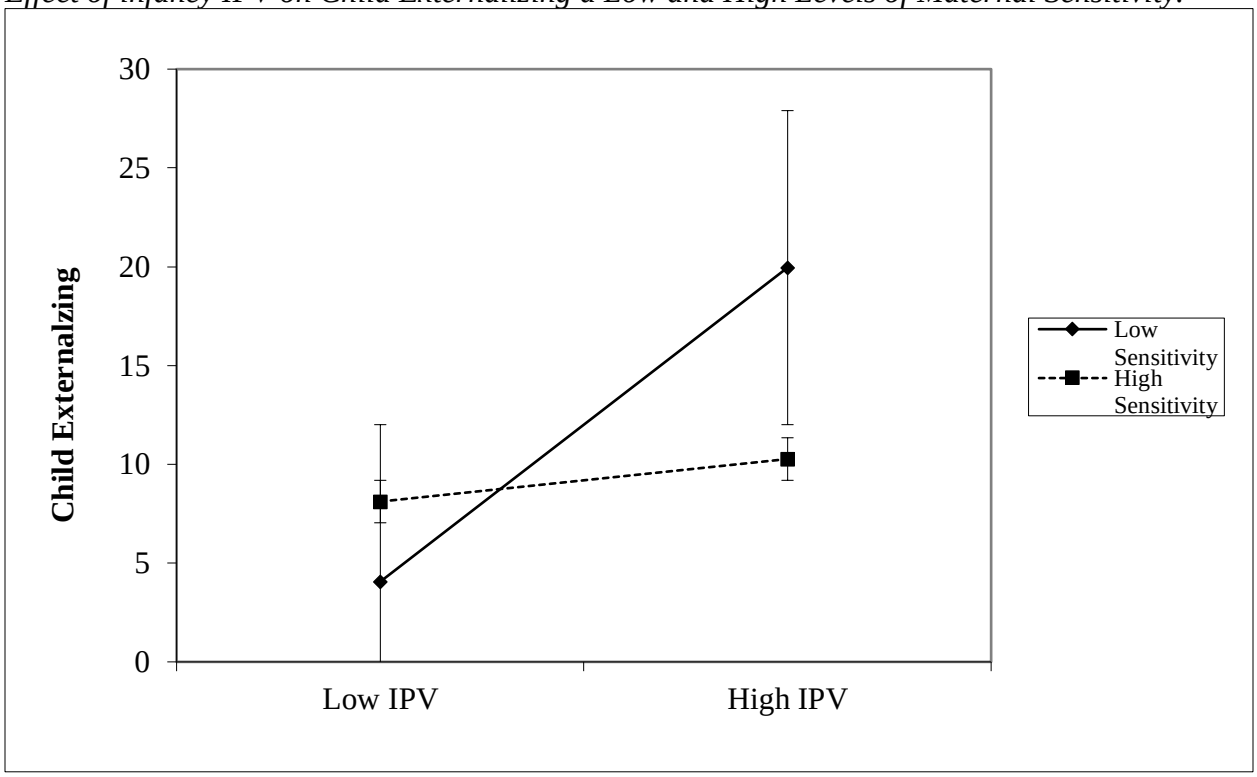
Table 5 (cont'd)

2.5-year IPV	.149	. 083	.167	.076	.005	.016	,041	.759
Maternal Sens.	-.777	.739	-.099	.295	.304*	.141	.271*	.035
Interaction	-.111	.089	-.117	.213	.010	.023	.058	.664
ΔR^2		.014		.175		.003		.385

*$p < .05$

Figure 1

Effect of infancy IPV on Child Externalizing a Low and High Levels of Maternal Sensitivity.



Note: Low/high IPV = 1 SD above/below average infancy IPV; Low/high sensitivity = 1 SD above/below mean maternal sensitivity. Effect is significant only for low sensitivity.