EMOTION REGULATION AND SENSITIVE CAREGIVING IN TRAUMA-EXPOSED MOTHERS

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ABSTRACT

Sensitive caregiving, or a caregiver's ability to notice and attend to infant signals, interpret them accurately, and respond to them in an appropriate and timely manner, sets the stage for positive trajectories of socioemotional development. Meta-analytic findings suggest that maternal experiences of interpersonal trauma, including histories of childhood maltreatment and intimate partner violence (IPV), are associated with less sensitive caregiving. However, the mechanisms linking women's experiences of interpersonal trauma to parenting behavior are not well understood. Maternal psychopathology and non-balanced maternal representations have been proposed as potential explanatory pathways, both of which share emotion regulation deficits as underlying features. Therefore, the present study aimed to examine whether self-report and physiological (high-frequency heart rate variability; HF-HRV) measures of emotion regulation mediate the associations between maternal experiences of interpersonal trauma and observed caregiving sensitivity. Additionally, current psychopathology and maternal representations were examined as potential moderators of this mediation pathway. The sample consisted of 370 women enrolled in the Michigan Prenatal Stress Study. Participants were oversampled for experiences of interpersonal violence. Assessments of demographic risk, childhood maltreatment, lifetime and pregnancy IPV, and maternal representations were completed during pregnancy. Measures of depression, PTSD, self-reported emotion regulation, baseline and stressed HF-HRV, and observed caregiving sensitivity were collected at 6-months postpartum. A series of structural equation models conducted in Mplus were used to test the study hypotheses. Contrary to expectations, although history of childhood maltreatment was associated with greater self-reported emotion regulation difficulties, neither childhood maltreatment nor IPV significantly predicted caregiving sensitivity directly or indirectly through self-reported emotion

regulation. Regarding physiological emotion regulation, childhood maltreatment was associated with less parasympathetic withdrawal during the stress task, which in turn was associated with less sensitive caregiving. The moderation hypotheses were not supported. Results suggest that early experiences of interpersonal trauma have lasting consequences for women's emotion regulation abilities, and physiological regulation in particular may have bearing on mothers' abilities to engage in sensitive caregiving. Results should be interpreted in the context of the specific methods used in the present study, and an important future direction will be to replicate these findings using a parenting task that is more demanding of mothers' emotion regulation resources. Lastly, cumulative demographic risk was a significant predictor of caregiving sensitivity, highlighting the importance of taking ecological/contextual factors into account when examining mechanisms of intergenerational transmission of risk.

For my parents, who taught me to think like a scientist and feel like a clinician.

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INTRODUCTION

Sensitive caregiving is defined as a caregiver's ability to notice and attend to infant signals, interpret them accurately, and respond to them in an appropriate and timely manner (Ainsworth, Waters, & Wall, 1978). Sensitive caregiving in infancy and early childhood promotes the development of children's attachment security and self-regulation abilities, which together set the stage for trajectories of positive socioemotional development (Calkins, 2011; van der Voort, Juffer, & J. Bakermans-Kranenburg, 2014). On the other hand, less than sensitive caregiving is considered an important mechanism in the intergenerational transmission of attachment insecurity and is associated with a variety of adverse developmental outcomes (Deans, 2018; van IJzendoorn, 1995). Understanding the factors that promote and impede sensitive caregiving is crucial for the prevention of intergenerational transmission of risk.

Trauma that occurs within the context of close relationships, including relationships with one's own caregivers and romantic partners, has the potential to negatively impact an adult's ability to engage in sensitive caregiving. For example, in one study, mothers' cumulative experiences of interpersonal trauma predicted more punitive parenting practices and greater child abuse potential, over and above the effects of demographic risk and mental health diagnoses (Cohen, Hien, & Batchelder, 2008). Indeed, recent meta-analytic findings indicate that maternal experiences of childhood maltreatment are inversely associated with sensitive parenting behaviors (Savage, Tarabulsy, Pearson, Collin-Vézina, & Gagné, 2019), and similar findings have been reported for the association between intimate partner violence (IPV) victimization and parenting (Chiesa et al., 2018). However, effect sizes in both meta-analyses are modest and there was considerable heterogeneity among studies, which may indicate the presence of moderators.

Maternal mental health disorders such as depression and posttraumatic stress disorder (PTSD) and non-balanced maternal representations of the relationship with the child have both been proposed as potential explanatory factors accounting for the link between interpersonal trauma exposure and impaired caregiving sensitivity (e.g., Cooke, Racine, Plamondon, Tough, & Madigan, 2019; Greene, Chan, McCarthy, Wakschlag, & Briggs-Gowan, 2018; Levendosky, Lannert, & Yalch, 2012; Madigan, Wade, Plamondon, & Jenkins, 2015; Muzik et al., 2013). As psychopathology and non-balanced representations both share emotion dysregulation as an underlying feature, the present study sought to clarify the role of maternal emotion regulation in mediating the association between interpersonal trauma and early caregiving sensitivity.

Specifically, the present dissertation uses data from an ongoing longitudinal study of mothers and their infants to test whether the effects of two common forms of interpersonal trauma, childhood maltreatment and IPV, on early caregiving sensitivity are mediated through self-reported and physiological measures of emotion regulation. Additionally, current psychopathology and maternal representations were tested as potential moderators of this indirect path. Increasing our understanding of the mechanistic pathways and specific conditions under which maternal experiences of interpersonal trauma lead to disruptions in early caregiving may aid in identifying mothers at greatest risk for caregiving impairments and shed light on promising targets for intervention.

Effects of Interpersonal Trauma on Caregiving Sensitivity

Developmental psychopathology perspectives suggest that mental health problems resulting from childhood maltreatment (i.e., emotional, physical, or sexual abuse or neglect) may help to explain the link between maternal history of childhood maltreatment and later deficits in caregiving sensitivity. Indeed, experiences of childhood maltreatment are associated with

increased risk for a variety of mental health problems in adulthood including depression and PTSD (Green et al., 2010; McLaughlin et al., 2012, 2013), symptoms of which have the potential to negatively affect early parenting behavior (Bernard, Nissim, Vaccaro, Harris, & Lindhiem, 2018; Greene, Haisley, Wallace, & Ford, 2020; Lovejoy, Graczyk, O'Hare, & Neuman, 2000; Muzik et al., 2017). However, it is important to note that not all those who were maltreated as children go on to develop psychopathology in adulthood, nor do all those who were abused as children go on to perpetrate abuse (Collishaw et al., 2007; Sexton, Davis, Menke, Raggio, & Muzik, 2017). For example, the negative effect of childhood maltreatment on mothers' sense of their own parenting competence was buffered among those who reported low levels of depressive symptoms and high levels of internal resilience factors (Martinez-Torteya, Katsonga-Phiri, Rosenblum, Hamilton, & Muzik, 2018).

Attachment theory provides another possible framework to account for the connection between childhood maltreatment history and parenting difficulties. Bowlby described internal working models as experience-based mental representations of self and other that are formed in the context of early attachment relationships (Bowlby, 1969, 1973, 1980). These representations serve as templates for processing social information and generating expectations about the behavior and intentions of others in subsequent relationships, including relationships with romantic partners and one's own children. Mothers with insecure or "non-balanced" internal working models are expected to interpret ambiguous social information in a more negative manner, consistent with their previous experiences in close relationships (Dykas & Cassidy, 2011). For instance, such mothers might be more likely to interpret infant distress signals as the child being spoiled or manipulative, leading them to respond to the infant less sensitively compared to mothers who make neutral or benevolent attributions about the child's behavior.

Research shows that maltreatment in childhood is associated with increased rates of insecure internal working models in adulthood, which in turn are associated with less sensitive parenting practices including perpetration of child maltreatment (Cort, Toth, Cerulli, & Rogosch, 2011; Lo, Chan, & Ip, 2019; Raby, Labella, Martin, Carlson, & Roisman, 2017). Indeed, one study found that pregnant women with histories of childhood maltreatment and IPV perceived ambiguous infant facial expressions as more negative, which in turn predicted less sensitive parenting behavior in the postpartum period (Dayton, Huth-Bocks, & Busuito, 2016).

However, not all children who experience maltreatment will go on to have lasting attachment difficulties, and secure or "balanced" internal working models may protect against later impairments in caregiving. For example, one study found that maternal attachment representations moderated the association between maternal history of childhood neglect and postpartum bonding difficulties, such that mothers who had been neglected by their own parents reported increased bonding difficulties with their infants only if they also exhibited high levels of attachment insecurity (Julian, Bernard, Handelzalts, Menke, & Muzik, 2021). Similarly, another study found that women with histories of parental emotional rejection and insecure adult attachment representations made more negative attributions about infant cry sounds, whereas secure representations buffered the association between parental rejection and negative attributions about infant distress (Leerkes & Siepak, 2006). Taken together, these findings suggest that "earned secure" internal working models (i.e., secure representations despite negative experiences in relationships) may protect against the intergenerational transmission of insensitive caregiving.

In addition to childhood maltreatment, IPV is another common interpersonal traumatic stressor that can negatively impact early parent-child relationships and maternal caregiving

sensitivity (Chiesa et al., 2018; McIntosh, Tan, Levendosky, & Holtzworth-Munroe, 2019). IPV is defined as physical, sexual, or psychological aggression perpetrated by a current or former romanic partner (Saltzman, Fanslow, McMahon, & Shelley, 2002). Psychological sequalae of IPV victimization such as depression and PTSD symptoms may partially account for the relationship between IPV victimization and reduced maternal sensitivity (e.g., Greene et al., 2018; Levendosky, Huth-Bocks, Shapiro, & Semel, 2003; Schechter et al., 2015). Maternal representations, or internal working models of the relationship with the child, can also be negatively affected by violence in the parental relationship (Huth-Bocks, Levendosky, Theran, & Bogat, 2004; Levendosky et al., 2012). For example, more conflict with the child's father has been associated with greater levels of distortion in the content and quality of maternal representations, which has the potential to impair a mother's abilities to recognize and respond sensitively to the child's needs (Dayton, Levendosky, Davidson, & Bogat, 2010; Schechter et al., 2005; Sokolowski, Hans, Bernstein, & Cox, 2007). Additionally, as maternal representations are child-specific, they are expected to be colored by the quality of the relationship with that child's father, as well as by positive and negative experiences during the pregnancy itself. Therefore, IPV experienced during pregnancy may be expected to have an outsized influence on early maternal caregiving behavior as opposed to preconception IPV, not only because of its recency, but also because of its unique impact on the mother's representations of the specific parent-child relationship.

In summary, maternal experiences of interpersonal trauma, including childhood maltreatment and IPV have the potential to negatively affect caregiving sensitivity. Maternal mental health disorders and maternal representations may influence the strength of these associations. As will be discussed in the following sections, emotion regulation deficits, which

are conceptually related to both psychopathology and internal working models (Cassidy, 1994; Dvir, Ford, Hill, & Frazier, 2014), may serve as an important mediating mechanism linking experiences of interpersonal trauma with maladaptive caregiving.

Interpersonal Trauma, Emotion Regulation, and Caregiving

Emotion regulation refers to the adaptive internal or external processes by which emotional reactions are monitored, evaluated, and modified in order to accomplish one's goals (Thompson, 1994). Caring for a new infant presents substantial emotional challenges, and a parent's ability to modulate their own emotional arousal may underpin their capacity to recognize, accurately interpret, and respond sensitively to the child's needs. Emotion dysregulation on the part of mothers is associated with suboptimal caregiving behavior and increased risk for engaging in child maltreatment (Dix, 1991; Miragoli, Milani, Di Blasio, & Camisasca, 2020). Additionally, a growing body of literature has emerged linking maternal autonomic functioning, a physiological marker of emotion regulation, to early parenting behavior and child adjustment outcomes (e.g., Leerkes, Su, Calkins, O'Brien, & Supple, 2017; Leerkes, Su, Calkins, Supple, & O'Brien, 2016; Ostlund, Measelle, Laurent, Conradt, & Ablow, 2017; Somers, Curci, Winstone, & Luecken, 2021).

Parents' capacity for emotion regulation has origins in their own early developmental histories. Children rely on caregivers to scaffold the development of emotion regulation abilities. This occurs in part through the attachment system, a biobehavioral system that organizes approach and withdrawal behaviors in relation to caregivers in order to maintain a sense of security and physiological homeostasis during times of emotional arousal (Bowlby, 1969, 1973, 1980). By the end of the first year of life, this system is theorized to be "tuned" by experiences in early caregiving relationships. According to attachment theory, children whose distress is

consistently recognized and successfully regulated with the help of a caregiver will begin to internalize these emotional communications and develop the capacity to employ similar regulation strategies on their own. Thus, sensitive caregiving is thought to support the development of emotional self-regulation abilities. Later in life, it is expected that securely attached individuals who benefited from sensitive caregiving will be able to express a full range of emotions, effectively regulate emotions independently, and engage in co-regulation with close others (Cassidy, 1994; Sroufe, 1996).

Experiences of relational trauma such as childhood maltreatment that often occur in the context of parent-child relationships have the potential to disrupt these developmental processes and set the stage for later emotion regulation difficulties (e.g., Briere & Rickards, 2007; Dvir et al., 2014; Greene, McCoach, Briggs-Gowan, & Grasso, 2021; Schore & Schore, 2008). Unsurprisingly, experiences of childhood maltreatment are associated with increased risk of developing insecure and disorganized attachment styles, of which unstable and inadequate emotion regulation is a defining feature (Cassidy, 1994; Mikulincer & Shaver, 2019). As discussed previously, childhood maltreatment is also linked to increased risk of developing a variety of mental health problems that share emotion dysregulation as an underlying deficit (Aldao, Gee, De Los Reyes, & Seager, 2016). The effects of childhood maltreatment on emotion regulation can also be gleaned through quantifiable physiological differences. For example, among adult women, childhood maltreatment history was associated with parasympathetic inflexibility as indicated by lower resting high-frequency heart rate variability (HF-HRV), a proposed biomarker of emotion regulation difficulties (Dale et al., 2018; Meyer et al., 2016; Stone, Amole, Cyranowski, & Swartz, 2018). Emotion regulation difficulties may be one mechanism accounting for the intergenerational transmission of insensitive caregiving. For

example, among mothers of preschoolers, self-reported emotion regulation difficulties mediated the association between maternal history of childhood maltreatment and use of psychological aggression during parenting (Rodriguez, Are, Madden, Shaffer, & Suveg, 2021).

Maternal emotion regulation difficulties may also partially account for the relationship between IPV exposure and deficits in caregiving sensitivity. For example, mothers who have difficulty regulating emotions during parenting may be more likely to project attributes of the abuser onto the infant, resulting in less sensitive responding (Levendosky, Bogat, & Huth-Bocks, 2011; Lieberman, 2007). Emotion regulation difficulties are also associated with PTSD symptom severity in women exposed to IPV (Simpson, Raudales, Reyes, Sullivan, & Weiss, 2021), and may help to account for the negative effects of PTSD on maternal parenting behavior and children's socioemotional outcomes. For exmaple, in families exposed to IPV, maternal emotion regulation difficulties mediated the association between maternal PTSD symptoms and children's self-regulation abilities, highlighting the importnant role that maternal emotion regulation plays in scaffolding children's emotional development (Pat-Horenczyk et al., 2015). There is also some evidence to suggest that maternal autonomic regulation interacts with IPV-related PTSD symptoms to predict positive parenting (Gurtovenko & Katz, 2017). Thus, maternal emotion regulation may underly the effects of IPV-related psychopathology and maternal representations on caregiving sensitvity. In the sections to follow, the theoretical rationale for using autonomic functioning as a physiological index of emotion regulation is presented, and associations with early parenting are discussed.

Heart Rate Variability as a Marker of Emotion Regulation

Emotion regulatory capacity has physiological bases in the autonomic nervous system (ANS). The flexibility with which one is able to transition from a physiological state of high

arousal to low arousal and vice versa in response to situational demands is thought to underly the capacity for regulated emotional responding (Appelhans & Luecken, 2006). Therefore, physiological markers such as high-frequency heart rate variability (HF-HRV), an index of parasympathetic control of the heart, can convey information about one's capacity for emotion regulation (Beauchaine, 2001; Porges, 1995, 2001; Thayer & Lane, 2000). Research has linked high resting HF-HRV with physiological flexibility, self-regulatory capacity, and social-emotional competence, while low resting HF-HRV has been conceptualized as a transdiagnostic marker of emotion dysregulation and psychopathology (Balzarotti, Biassoni, Colombo, & Ciceri, 2017). In addition to basal measures of HF-HRV, transient parasympathetic withdrawal, and corresponding reductions in HF-HRV in response to acute stressors has been linked to more adaptive responding. For example, HF-HRV reductions from baseline in response to child distress cues are associated with more sensitive maternal caregiving behaviors (Ablow, Marks, Feldman, & Huffman, 2013; Joosen et al., 2013; Mills-Koonce et al., 2007, 2009; Moore et al., 2009), suggesting that flexible autonomic regulation may be needed to support sensitive caregiving.

The ANS is subdivided into two complementary branches: the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS). The heart is enervated by both branches of the ANS, and both branches influence the activity of the primary cardiac pacemaker, the sinoatrial node, which generates action potentials that cause the contraction of the myocardium responsible for the heartbeat (Shaffer & Venner, 2013). Engagement of the PNS facilitates a reduction in arousal and heart rate required for maintaining internal homeostatic functions. During times of relative quiescence, the PNS slows the resting heart rate to below the intrinsic firing rate of the sinoatrial node. Conversely, during times of physical or psychological

stress, the influence of the PNS lifts and the excitatory SNS becomes dominant, facilitating increased heart rate and physiological arousal. The SNS serves to mobilize oxygen and other resources to prepare the body to meet environmental challenges. Thus, the relative influence of the PNS and SNS on the heart regulates the length of time between each consecutive heartbeat, also called the interbeat interval. Heart rate variability, or the variance of successive interbeat intervals, indexes the interplay between the sympathetic and parasympathetic influences on heart rate (Berntson et al., 1997).

The sympathetic and parasympathetic influences on the heart are exerted via different mechanisms. Sympathetic influence is mediated by neurotransmission of norepinephrine, which has a relatively slow latency (approximately 4 seconds to peak effect and 20 seconds to return to baseline) compared to the rate of transmission of acetylcholine (peak effect at approximately .05 seconds and return to baseline in 1 second), which mediates parasympathetic influence on cardiac tissue via the vagus nerve (Berntson et al., 1997). Thus, the oscillations in heart rate caused by the two branches occur at different frequencies, allowing the two to be disentangled using spectral analysis, which uses Fourier transformations to decompose heart rate time series data into its low frequency and high frequency components (Berntson et al., 1997). The high frequency (HF) band (.15-.40 Hz for adults) reflects parasympathetic influence on the heart and corresponds to heart rate variations related to the respiration cycle. The act of inhalation temporarily "gates" parasympathetic influence on heart rate, causing heart rate to increase. Upon exhalation, parasympathetic influence is reinstated causing heart rate to decrease (Eckberg, 2003). HF-HRV is used in research as a measure of parasympathetically mediated heart rate variability because only parasympathetic cardiac activity has a mechanism of action rapid enough to covary with the respiration cycle. This high frequency oscillation in heart rate

produced by respiration is also called respiratory sinus arrhythmia (RSA) in the literature. The fact that HF-HRV can be measured non-invasively, in ambulatory settings, using widely available and relatively inexpensive electrocardiography (ECG) equipment, has made it popular among researchers as a proxy measure of parasympathetic functioning.

Several prominent theories including Stephen Porges' polyvagal theory (Porges, 1995, 2001, 2003b, 2007) and Julian Thayer and colleagues' neurovisceral integration model (Thayer & Brosschot, 2005; Thayer, Hansen, Saus-Rose, & Johnsen, 2009; Thayer & Lane, 2000, 2009) attempt to elucidate the relationship between autonomic control of the heart and emotion regulation. Although these models utilize different theoretical frameworks (evolutionary theory and dynamical systems theory, respectively) both conclude that dynamic autonomic cardiac regulation can serve as a physiological marker of emotional health and functioning. Both theories are expanded upon in the sections to follow, as they provide differing but complementary perspectives on the potential relevance of autonomic regulation to the caregiving system.

Polyvagal Theory

Polyvagal theory rests on the idea that two populations of vagal nuclei originating in different parts of the brainstem evolved at different times and under different evolutionary pressures such that they produce opposing responses to environmental challenge (Porges, 1995, 2001, 2003b, 2007). The theory posits that the evolutionarily older, unmyelinated dorsal vagal pathway, originating in the dorsal motor nucleus, promotes a freezing response to environmental challenges characterized by increased vagal tone and reduced metabolic output. By contrast, the evolutionarily newer myelinated pathway, the ventral vagal complex originating in the nucleus ambiguous, allows for the rapid mobilization of energy required for the fight-or-flight response via the removal of the "vagal break" from the heart, resulting in increased heart rate and

availability of oxygen. At rest, the myelinated vagus inhibits sympathetically mediated physiological arousal, and promotes self-regulation. According to Porges, the addition of this second pathway is a unique evolutionary characteristic of mammals that provides the neurophysiological substrate for affective processes necessary for complex social behaviors[1].

Porges further asserts that the insights of polyvagal theory imply a social engagement system that is linked to autonomic control of the heart (Porges, 2001, 2003b, 2003a). He cites anatomical evidence that efferent neurons from the nucleus ambiguous are responsible not only for the heart's adaptive response to environmental challenge, but also for social communication behaviors via projections to the facial muscles, esophagus, pharynx, larynx, and soft palate that are involved in the control of facial expressions and shifts in vocal intonation that convey important information to others about one's current emotional state. Therefore, it is hypothesized that shifts in emotional expression are associated with changes in HF-HRV, as both are mediated by nucleus ambiguous activity. According to this model, primary negative emotions (e.g., scared, angry, sad) are expected to co-occur with vagal withdrawal. Positive emotional states on the other hand are expected to be accompanied by increases in vagal tone and HF-HRV, conditions that are conducive for vegetative functions and affiliative social behaviors. As a consequence of this integrated system, withdrawal of the ventral vagal complex and accompanying visceral arousal and fight-or-flight behaviors limit one's ability to express social engagement behaviors (e.g., positive and contingent facial expressions, prosody of voice, sustained eye contact, control of muscles of the inner ear to optimize extraction of human voice from background noise), whereas increased activity of the ventral vagal complex promotes calm emotional states and

[1] It should be noted that the scientific basis for the phylogenic hierarchy of the ventral vagus has been disputed in the literature (Grossman & Taylor, 2007) and myelinated nerve fibers connecting the brain and the heart have been reported in non-mammalian species (e.g., Monteiro et al., 2018).

prosocial engagement while dampening the neural circuits involved in responding to threat. Thus, the social engagement system is thought to be compromised when the environment is perceived as unsafe. As it relates to caregiving, this theory implies that temporary parasympathetic withdrawal on the part of the mother, as indexed by a reduction in HF-HRV, would be expected to facilitate appropriate behavioral responses under conditions of threat or acute infant distress. However, low basal HF-HRV under non-stressful conditions may indicate impairment in the mother's ability to access the social engagement system and to promote calm, co-regulated states with the infant.

Neurovisceral Integration Model

Inspired by findings linking HF-HRV to attentional and affective control, Thayer and colleagues proposed the neurovisceral integration model (Thayer & Lane, 2000, 2009). Like polyvagal theory, the neurovisceral integration model attempts to explain the role of autonomic functioning in affective and self-regulatory processes. Taking a dynamical systems perspective, these authors focused on identifying the functional and structural circuits involved in coordinating behavioral systems. They emphasized the importance of the central autonomic network (Benarroch, 1993) which describes the bidirectional links between forebrain structures (e.g., the anterior cingulate, insula, ventromedial prefrontal cortex, amygdala, and hypothalamus) and the nucleus of the solitary tract in the brainstem, output from which enervate the sinoatrial node of the heart via the stellate ganglia and the vagus nerve. Thus, the integrated output of the central autonomic network is responsible for sympathetic and parasympathetically mediated changes in heart rate variability.

Bolstered by neuroimaging findings suggesting that activity in the medial prefrontal cortex suppresses activation of the amygdala, and in turn influences the heart via connected

brainstem structures, the authors concluded that resting HF-HRV may index top-down self-regulatory capacity, including capacity for emotion regulation and executive functioning (e.g., Davidson, 2000; Sakaki et al., 2016; Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). In other words, resting HF-HRV may be a useful proxy measure of the efficiency of neural feedback mechanisms between the central and ANS such that resting HF-HRV is positively associated with prefrontal control and negatively associated with the activation of limbic structures, overactivity of which have been implicated in problems with emotion regulation and psychopathology.

Thayer and colleagues advanced the view that emotional responses emerge from interactions between multiple neural subsystems in response to environmental demands and serve to facilitate the coordination of goal-directed behavior. They proposed that discrete emotions may represent preferred configurations or "attractor" states of valance and arousal in the state-space of the organism, and that such attractors help to define the organism's behavioral repertoire (Thayer & Lane, 2000). Emotion dysregulation and related psychopathology may therefore result from an individual being "stuck" in a particular emotional state or behavioral pattern that is inappropriate for the demands of the current environment. The neurovisceral integration model also emphasizes the importance of attentional selection for adaptability and efficient emotion regulation, with hypervigilance to threat and negativity bias being prime examples of disinhibition of attentional selection that occur in disorders such as PTSD and depression. According to this model, it is adaptive for the prefrontal cortex to go "offline" temporarily during times of stress, facilitating the rapid withdrawal of parasympathetic control of the heart to allow for excitatory sympathetic influences needed to mount a fight-or-flight response. However, if the inhibitory influence of the prefrontal cortex is disrupted for prolonged

periods, the flexibility of the ANS to respond to environmental changes is reduced, and rigid cognitive and behavioral patterns may occur (Thayer & Lane, 2009). Therefore, the neurovisceral integration model suggests that baseline HF-HRV is expected to be positively associated with emotion regulation and executive control (Thayer et al., 2009). Extending this theory to apply to the caregiving system, it follows that mothers exposed to chronic environmental threats or psychopathology may have more difficulty flexibly modulating their autonomic, attentional, and behavioral responses during caregiving.

Interpersonal Trauma, HF-HRV, and Caregiving

While the polyvagal theory and neurovisceral integration models provide different perspectives on the role of parasympathetic cardiac control in emotion regulation, they tend to agree that 1) high resting HF-HRV is expected to be associated with better emotional and social functioning, 2) perception of threat or challenge is expected to be associated with temporary parasympathetic withdrawal and decreased HF-HRV, allowing for sympathetic dominance and context-appropriate energetic and behavioral mobilization. Conversely, low basal HF-HRV and inflexible parasympathetic reactivity in response to environmental demands are expected to be associated with lack of access to calm socially-engaged states and greater emotion regulation difficulties.

Although neither the polyvagal theory nor the neurovisceral integration model speak explicitly to the factors that determine individual differences in HF-HRV, both imply that chronic experiences of threatening and unsafe environments might play a role. Indeed, there is growing evidence to suggest that the development of the autonomic stress response system is influenced by early life experiences, including relationships with caregivers (Calkins, 2011; Calkins, Graziano, Berdan, Keane, & Degnan, 2008; M. Johnson et al., 2017; Kaplan, Evans, &

Monk, 2008; McLaughlin et al., 2015; Propper, 2012; Skowron et al., 2011). For example, Calkins (2008) found that the quality of the mother-child relationship at age 2 predicted greater parasympathetic reactivity (parasympathetic withdrawal during a challenge task) among children at age 5. Additionally, children who spent longer in institutionalized care settings as infants displayed blunted autonomic reactivity at age 12 in response to a social evaluation stressor compared to those who were placed in foster care earlier or were never exposed to institutional care (McLaughlin et al., 2015).

Based on the above findings, it would be reasonable to expect that individuals with histories of childhood maltreatment might display less flexible autonomic regulation in adulthood. Indeed, in several studies, women's self-reported experiences of childhood maltreatment were associated with lower resting HF-HRV in adulthood (Dale et al., 2018; Meyer et al., 2016). Both hyper- and hypo-reactivity of the ANS in response to stress have been reported in adults with childhood maltreatment histories. For example, among pregnant women, adverse childhood experiences including childhood maltreatment predicted less parasympathetic withdrawal in response to infant cry sounds (Oosterman, Schuengel, Forrer, & De Moor, 2019). Additionally, in a sample of undergraduate students with and without abuse histories, those with self-reported childhood physical, emotional, or sexual abuse displayed less cardiovascular reactivity (heart rate and systolic/diastolic blood pressure) to a judged serial subtraction task compared to non-abused peers (Ginty, Masters, Nelson, Kaye, & Conklin, 2017). By contrast, in a similar study of young adults, those with maltreatment histories evidenced heightened cardiovascular reactivity (heart rate) to mental and physical stressors, and longer latencies to recovery compared to the non-exposed group (Beilharz et al., 2020). Prolonged cardiovascular arousal in response to infant distress signals has also been observed among mothers with

histories of childhood neglect (Buisman et al., 2018), and there is some evidence for differential effects of threat (emotional and physical abuse) versus deprivation (emotional and physical neglect) experiences on autonomic functioning (Buisman et al., 2019), however such experiences frequently co-occur.

These differences in findings may be due to the differential focus on sympathetic vs. parasympathetic arousal. Differences in ANS functioning in pregnant versus non-pregnant participants should also be considered when comparing across studies. Maternal ANS functioning changes over the course of pregnancy to accommodate increased blood volume and uroplacental blood flow (Heiskanen et al., 2008). These changes involve increased resting sympathetic cardiac activity and decreased vagally-mediated parasympathetic activity with increased gestational age (Kolovetsiou-Kreiner et al., 2018; Kuo, Chen, Yang, Lo, & Tsai, 2000). Resting HF-HRV parameters increase after delivery as they return to pre-pregnancy levels (Heiskanen et al., 2008; Sarhaddi et al., 2022). Brown et al. (2021) found that HF-HRV increased significantly from late pregnancy to 4-6 weeks postpartum, and then plateaued for the remainder of the postpartum year. Others suggest that the ANS returns to baseline functioning within 3 months after delivery (Chen, Kuo, Yang, Lo, & Tsai, 1999). Given the potential differences in HF-HRV pre- and post-pregnancy, care must be taken to consider the nature and timing of such autonomic changes when comparing findings from studies that measure maternal autonomic functioning during the perinatal period.

Although maternal autonomic functioning has rarely been studied in IPV-exposed samples, there is some evidence to suggest that emotion regulation as indexed by HF-HRV is associated with parenting behavior in IPV-exposed mothers. For example, in one study of mothers of school-aged children recruited from domestic violence agencies, parasympathetic

withdrawal during a conflict discussion with their child moderated the association between mothers' PTSD symptoms and non-supportive parenting practices (Gurtovenko & Katz, 2017). Mothers with more severe posttraumatic stress symptoms were more likely to use non-supportive parenting practices only if they also exhibited low levels of parasympathetic withdrawal in response to the conflict discussion task. Mothers who demonstrated high levels of parasympathetic withdrawal during the conflict discussion task were able to engage in supportive parenting despite their PTSD symptoms, suggesting that flexible autonomic regulation may serve as a protective factor that buffers against the negative effects of IPV-related trauma on parenting. Another study by Molina et al. (2022) failed to replicate the moderating role of vagally-mediated HRV on the association between IPV exposure and maternal parenting, nor was a competing mediation model significant. Instead, they reported that more maternal experiences of verbal IPV were associated with less positive observed parenting behavior, while larger increases in HF-HRV during a joint-puzzle task with their preschooler independently predicted more positive parenting. This discrepancy in findings could be due to the use of IPV experiences as the predictor instead of PTSD symptoms as in the Gurtovenko & Katz (2017) study, as well as differences in the emotional context of the challenge tasks (i.e., conflict vs. teaching). Taken together, greater flexibility in HF-HRV (either augmentation or suppression depending on the emotional context of the task) appears to support positive parenting in IPV-exposed mothers.

It is yet unclear whether maternal parasympathetic regulation observed in more general stress contexts (i.e., ones that do not involve a parent-child interaction) are able to predict differences in parenting. Most studies examining the influence of maternal autonomic regulation on parenting measure maternal physiology in the context of structured parent-child interactions (e.g., still-face, strange situation paradigm, clean-up, joint puzzle completion) (Hill-Soderlund et

al., 2008; Moore et al., 2009; Oppenheimer, Measelle, Laurent, & Ablow, 2013; Smith, Woodhouse, Clark, & Skowron, 2016; Sturge-Apple, Skibo, Rogosch, Ignjatovic, & Heinzelman, 2011). Although these paradigms benefit from their ecological validity, they may be confounded by in-the-moment differences in parent and child behavior, which make it difficult to interpret whether between-person differences in maternal HF-HRV are due to the mothers' regulatory capacities or differences in the child's response. In addition, some interactive paradigms may not be sufficiently stressful to necessitate physiological stress regulation in all mothers, again making it difficult to draw generalizable conclusions about the role of parasympathetic reactivity in parenting.

Although comparisons of HF-HRV reactivity in response to parenting stressors and non-parenting social stressors have not been examined directly, there is some evidence to suggest that the emotion regulation strategies developed and used in the context of close relationships generalize to inform the ways that individuals respond affectively and physiologically to non-attachment-related stressors (e.g., Maunder, Lancee, Nolan, Hunter, & Tannenbaum, 2006; Mikulincer & Shaver, 2019; Movahed Abtahi & Kerns, 2017). Additionally, self-report measures of emotion regulation such as the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004), which are not specific to parenting contexts, are nonetheless found to be associated with observed maternal sensitivity (Leerkes, Su, & Sommers, 2020). Therefore, generalized deficits in parasympathetic regulation might likewise be associated with parenting outcomes. In the present study, maternal HF-HRV was measured at baseline and during a (non-parenting) standardized social stress task to determine whether generalized parasympathetic dysregulation confers risk for insensitive caregiving. By including both self-report and physiological indices of emotion regulation, the present study aimed to clarify whether the two

methods of measuring emotion regulation yield similar or differing associations with parenting outcomes.

Moderators of the Associations Between Interpersonal Trauma, Maternal Emotion Regulation, and Parenting

While experiences of interpersonal trauma are thought to increase risk for emotion regulation difficulties, which in turn are expected to have negative consequences for parenting, it is not a foregone conclusion. Risk and protective factors may influence the strength of the associations among interpersonal trauma, emotion regulation, and caregiving sensitivity. These factors include maternal psychopathology and maternal representations of the relationship with the child.

Maternal Mental Health

Trauma-related psychopathology, including depression and PTSD, are consistently associated with deficits in emotion regulation (Chang, Kaczkurkin, McLean, & Foa, 2018; Ehring & Quack, 2010; Joormann & Stanton, 2016; McLean & Foa, 2017). However, not all who are exposed to trauma will go on to develop clinically significant symptoms (Collishaw et al., 2007). One might expect that women who develop clinically significant symptoms of depression or PTSD after trauma exposure would be more likely to exhibit emotion regulation difficulties compared to those who do not develop psychopathology, and therefore may be more likely to engage in suboptimal parenting.

Indeed, there is evidence to suggest that exposure to interpersonal trauma interacts with psychopathology to predict physiological aspects of emotion regulation. For example, Stone and colleagues (2018) found that histories of childhood emotional abuse interacted with depression to predict resting HF-HRV, such that depressed women with child abuse histories had lower resting

HF-HRV compared to depressed women without childhood maltreatment histories. Similarly, in another study childhood maltreatment predicted depressive symptoms only in adults who exhibited low resting HF-HRV (Zhang, Luo, Davis, & Zhang, 2021). Extending such findings to parenting outcomes, another study found that maternal HF-HRV reactivity to a challenging parenting scenario interacted with IPV-related PTSD symptoms to predict observed parenting behavior (Gurtovenko & Katz, 2017).

In the present study, maternal psychopathology was tested as a moderator of the direct and indirect effects of maternal interpersonal trauma history on caregiving sensitivity through emotion regulation. Support for the hypothesis that the negative effects of interpersonal trauma exposure on caregiving sensitivity are strengthened for those exhibiting clinical levels of depression and PTSD symptoms would suggest that screening new mothers for trauma-related psychopathology would be an appropriate way to identify those at greatest need of parenting support.

Maternal Representations

A mothers' relational schemas likewise may influence the degree to which experiences of interpersonal trauma affect her parenting. In adults, relational schemas or internal working models are primarily assessed via semi-structured interviews, such as the Adult Attachment Interview (AAI; George, Kaplan, & Main, 1985), which ask respondents to reflect on their relationships with caregivers, descriptions of which are subsequently coded for the individual's state of mind regarding attachment. The Working Model of the Child Interview (WMCI; Zeanah, Benoit, & Barton, 1986) is a similar measure that assesses parents' representations of their relationship with a particular child, either during pregnancy or after birth. The narrative produced during the interview is subsequently coded into "balanced," "non-balanced disengaged," or

"non-balanced distorted" classifications that are conceptually analogous to the secure-autonomous, insecure-dismissing, and insecure-preoccupied classifications of the AAI. Balanced maternal representations are characterized by rich and coherent descriptions of the child and the relationship, a high degree of acceptance of the child, and acknowledgement of both the positive and negative aspects of parenting. Non-balanced representations are characterized either by minimization of affect and limited descriptions of the infant (non-balanced disengaged), or by heightened affect and incoherent descriptions of the infant (non-balanced distorted).

As would be expected based on the assumptions of attachment theory, research indicates that adult women's attachment styles toward their own caregivers, as assessed by the AAI, largely corresponded with prenatal maternal representations measured by the WMCI, such that women who had secure attachments to caregivers were 25 times more likely to have balanced representations of their relationship with their unborn child (Madigan, Hawkins, Plamondon, Moran, & Benoit, 2015). In line with this finding, mothers who experienced childhood maltreatment, specifically physical neglect, were more likely to have non-balanced prenatal representations (Malone, Levendosky, Dayton, & Bogat, 2010). Additionally, maternal representations may be influenced by salient relational experiences in adulthood, for example experiences of IPV with the child's father (Huth-Bocks et al., 2004).

Although women exposed to interpersonal trauma in childhood or adulthood are expected to be at greater risk of having non-balanced prenatal representations, there is still variability among these groups. As discussed previously, attachment style or internal working models seem to be related to one's ability to engage in flexible emotion regulation (Cassidy, 1994; Pietromonaco & Barrett, 2000; Zimmermann, 1999). Women who maintain secure/balanced internal working models despite histories of relational trauma are expected to have greater

emotion regulation resources compared to those with non-balanced internal working models and may be less susceptible to the detrimental effects of interpersonal trauma on parenting. For example, previous research suggests that mothers' internal working models interact with current psychological distress to predict caregiving sensitivity, such that high levels of psychological distress were associated with less sensitive caregiving only for insecurely attached mothers (Mills-Koonce et al., 2011). Additionally, multiple studies report that mothers with difficult relationships with their own parents, but who were categorized as having secure internal working models on the AAI (i.e., "earned secure"), displayed parenting that was more like those with continuous secure classifications as opposed to those with insecure classifications, even when under high levels of stress (Pearson, Cohn, Cowan, & Cowan, 1994; Phelps, Belsky, & Crnic, 1998). Similarly, mothers who experienced neglect in their own childhoods were more likely to experience postpartum bonding difficulties only if they also endorsed high levels of attachment insecurity, suggesting that balanced internal representations of close relationships may exert a protective influence on early parenting for those who experienced childhood maltreatment (Julian et al., 2021). There is less research examining the role of maternal internal working models on caregiving sensitivity among IPV-exposed mothers. That which does exist suggests that mothers with balanced prenatal working models display more positive parenting compared to non-balanced mothers regardless of IPV experiences (Dayton et al., 2010).

In the present study prenatal representations were tested as a moderator of the effects of interpersonal trauma history on maternal sensitivity directly and indirectly through emotion regulation to examine whether balanced representations buffer against the negative effects of interpersonal trauma on caregiving sensitivity, and whether this buffering effect operates through adaptive emotion regulation. Support for the hypothesis that balanced working models are

protective against the detrimental effects of interpersonal trauma exposure on caregiving sensitivity would lend additional support for attachment-based parenting interventions that aim to improve maternal representations.

Covariates

Demographic risk factors such as low socioeconomic status have the potential to influence maternal caregiving sensitivity, for example through increased parenting stress, lack of time and resources, or limited developmental knowledge (e.g., Raikes & Thompson, 2005; Slack, Holl, McDaniel, Yoo, & Bolger, 2004). Additionally, demographic risk factors are associated with increased exposure to chronic ecological stressors such as financial stress, food or housing insecurity, neighborhood crime, and discrimination. Chronic stress increases allostatic load and can lead to changes in the functioning of physiological stress systems including the ANS (McEwen, 1998). For example, in a large urban sample from the Netherlands, exposure to childhood maltreatment was associated with lower resting HF-HRV, but this association was no longer significant after the inclusion of demographic risk covariates (Bakema et al., 2020). Similarly, in a study of mothers of toddlers, socioeconomic adversity was associated with a profile of autonomic hypoarousal, which in turn was associated with insensitive and disengaged parenting (Sturge-Apple et al., 2011). Therefore, demographic risk was controlled for in the present study by including a cumulative risk variable comprised of low income, low educational attainment, single parenthood, and racial/ethnic minority status as a covariate in the analyses.

The Present Study

Experiences of interpersonal trauma in childhood and adulthood have been linked to emotion regulation difficulties. Mothers with interpersonal trauma histories also seem to have more difficulty interpreting and responding sensitively to infant cues (e.g., Dayton et al., 2016).

Maternal emotion regulation is one possible mechanism through which maternal experiences of trauma influence parenting (e.g., Cabecinha-Alati, Langevin, & Montreuil, 2020). Self-reported emotion regulation difficulties have been linked to less sensitive parenting (Carreras, Carter, Heberle, Forbes, & Gray, 2019; Leerkes et al., 2020; Miragoli et al., 2020). In addition, there is evidence that physiological indices of emotion regulation such as HF-HRV are associated with differences in caregiving behavior (e.g., Hill-Soderlund et al., 2008; Leerkes et al., 2016; Mills-Koonce et al., 2009; Moore et al., 2009; Skowron, Cipriano-Essel, Benjamin, Pincus, & Van Ryzin, 2013). However, it is not yet clear whether maternal emotion regulation difficulties account for the association between interpersonal trauma exposure and deficits in early caregiving sensitivity. Nor is it clear whether these pathways look similar for interpersonal trauma that occurs in different developmental periods (e.g., childhood maltreatment, IPV in adolescence and adulthood, IPV during pregnancy). Therefore, the first goal of the present study was to clarify whether maternal trauma in the context of relationships with parents and romantic partners negatively affects caregiving sensitivity in the early postpartum period through emotion regulation deficits.

Maternal emotion regulation was indexed using multiple methods including a self-report measure and physiological measures of parasympathetic functioning (HF-HRV). Most studies examining associations between maternal physiological regulation and parenting outcomes have measured maternal physiology during parenting-specific stress tasks such as parent-child interaction paradigms or infant cry recordings. However, experiences of interpersonal trauma are thought to affect generalized emotion regulation abilities (Dykas & Cassidy, 2011; Mikulincer & Shaver, 2019), and measures of HF-HRV during non-parenting standardized stress paradigms may be less susceptible to confounds due to differences in infant behavioral reactivity.

Therefore, in the present study, HF-HRV was measured at baseline and in response to a non-parenting social evaluation threat task, the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), to determine whether generalized physiological dysregulation likewise confers parenting risk. Concordance between self-report and physiological measures of emotion regulation and their associations with observed caregiving sensitivity would lend additional empirical support for the utility of a widely available self-report measure of emotion regulation (DERS; Gratz & Roemer, 2004) to identify parents who may stand to benefit from intervention.

Furthermore, although interpersonal trauma is thought to confer parenting risk, not all mothers who experience trauma will exhibit maladaptive parenting (e.g., Sexton et al., 2017). The heterogeneity and small effect sizes found in research linking childhood maltreatment and IPV to parenting outcomes suggest that moderators may be relevant (e.g., Chiesa et al., 2018; Savage et al., 2019). Therefore, an additional goal of the study was to investigate potential risk and protective factors that influence the strength of the associations between interpersonal trauma and caregiving sensitivity. Maternal mental health is one possible moderator. In the present study clinical levels of maternal depression and PTSD symptoms were tested as moderators of the effects of interpersonal trauma on caregiving sensitivity both directly and indirectly through emotion regulation. Mothers who go on to experience psychopathology following trauma exposure were expected to display more emotion regulation difficulties and less sensitive parenting compared to those who did not exhibit clinically significant symptoms.

Maternal representations are another potential moderator. The caregiving system is thought to be influenced in part by the mother's internal working model of the relationship with the child (George & Solomon, 2008). Maternal representations may be influenced by the mother's experiences with her own parents and with romantic partners, including the child's

father, putting those who have experienced trauma in these relationships at greater risk for having non-balanced maternal representations (Huth-Bocks et al., 2004; Madigan, Hawkins, et al., 2015; Malone et al., 2010). Non-balanced maternal representations are associated with reduced caregiving sensitivity (Dayton et al., 2010). However, the detrimental effects of interpersonal trauma on maternal representations may be counteracted by benevolent experiences in close relationships. For example, someone with negative caregiving experiences may be able to maintain secure attachment representations due to the presence of a stable and protective relationship with another adult (Roisman, Padrón, Sroufe, & Egeland, 2002). Similarly, those who have insecure attachments in early life may be able to transition to secure classifications over time through positive changes in the family environment or corrective experiences in adult relationships (Waters, Weinfield, & Hamilton, 2000). Balanced maternal representations may confer parenting resilience in the face of interpersonal trauma. Therefore, in the present study, balanced maternal representations were tested as a moderator of the effect of interpersonal trauma on caregiving sensitivity directly and indirectly through emotion regulation.

The present study leverages data from an ongoing longitudinal study of prenatal and postnatal stress to address the questions of how and under what conditions do maternal experiences of interpersonal trauma affect early caregiving sensitivity. The sample is uniquely positioned to answer these research questions as the participants are characterized by high levels of interpersonal violence exposure and sociodemographic risk. Additionally, including measures of multiple forms of interpersonal trauma made it possible to examine whether the effects of interpersonal trauma on caregiving sensitivity differed based on the type and timing of interpersonal trauma exposure.

In summary, the study adds to the existing literature by examining specific mechanisms through which experiences of trauma in childhood and adult relationships affect early parenting. Generalized deficits in maternal emotion regulation, as measured by self-report and parasympathetic functioning at baseline and during a non-parenting stress task, were investigated as potential mediators of the association between interpersonal trauma exposure and caregiving sensitivity. Additionally, maternal mental health and maternal representations were examined as potential moderators. Increasing our understanding of how and for whom experiences of interpersonal trauma lead to parenting deficits will help clinicians more accurately identify parents who are in need of intervention, as well as clarify whether emotion regulation and physiological components thereof represent appropriate treatment targets, as has been suggested in the literature (e.g., Braeken, Jones, Otte, Nyklíček, & Van den Bergh, 2017; Crandall, Deater-Deckard, & Riley, 2015; Zalewski, Lewis, & Martin, 2018).

Hypotheses

Aim 1: To examine the associations between self-report and physiological measures of emotion regulation.

Hypothesis 1. HF-HRV is considered a physiological index of emotion regulation (Appelhans & Luecken, 2006; Beauchaine, 2015; Thayer et al., 2009). Low resting HF-HRV and lack of HF-HRV reactivity to environmental stressors are suggestive of reduced autonomic flexibility and emotion regulation deficits. There is literature suggesting that greater self-reported emotion regulation difficulties are associated with lower resting HF-HRV (Visted et al., 2017; Williams et al., 2015) and less HF-HRV suppression from baseline to challenge task (Leerkes et al., 2020). Therefore, in the present study it was expected that greater self-reported emotion

regulation difficulties would be significantly correlated with lower resting HF-HRV and less HF-HRV suppression from baseline to the TSST.

Aim 2: To examine whether maternal histories of childhood maltreatment and IPV affect caregiving sensitivity via maternal emotion regulation.

Hypothesis 2a. Interpersonal trauma experiences are linked to impairments in emotion regulation and sensitive caregiving (Chiesa et al., 2018; Rodriguez et al., 2021; Savage et al., 2019). There is a growing literature to suggest that maternal emotion regulation is an essential prerequisite for sensitive parenting (e.g., Crandall et al., 2015). Therefore, it was expected that more experiences of interpersonal trauma in childhood (i.e., childhood maltreatment) and adulthood (i.e., lifetime and pregnancy IPV) would predict lower levels of maternal sensitivity, and their effects would be partially mediated through greater self-reported emotion regulation difficulties.

Hypothesis 2b: Interpersonal trauma exposure is also linked to impairments in physiological regulation. For example, history of childhood maltreatment is correlated with lower resting HF-HRV in women (Dale et al., 2018; Meyer et al., 2016). Additionally, flexible HF-HRV reactivity to parenting stressors has been associated with more sensitive parenting behavior (Ablow et al., 2013; Balzarotti et al., 2017; Hill-Soderlund et al., 2008; Katz & Gurtovenko, 2015; Leerkes et al., 2016; Mills-Koonce et al., 2007; Moore et al., 2009). Therefore, in the present study I expected that more experiences of interpersonal trauma in childhood and adulthood would be associated with less maternal sensitivity, and these effects would be partially mediated through lower HF-HRV at baseline and higher HF-HRV during the TSST.

Aim 3: Examine whether the indirect effects of interpersonal trauma on caregiving sensitivity through emotion regulation are conditional on maternal mental health.

Hypothesis 3. Maternal experiences of interpersonal trauma are associated with increased emotion regulation difficulties (Greene et al., 2021; Pat-Horenczyk et al., 2015). Experiences of interpersonal trauma also increase the likelihood that mothers will exhibit psychopathology such as depression and PTSD in the postpartum period, although resilience is also common (Collishaw et al., 2007). I expected that clinical levels of mental health symptoms would interact with interpersonal trauma histories to predict mothers' self-reported emotion regulation difficulties, such that the effect would be strengthened for mothers with clinical depression and PTSD symptoms. In turn, I expected that the indirect effects of interpersonal trauma on caregiving sensitivity through self-reported emotion dysregulation would be strengthened for mothers with clinical levels of depression and PTSD.

Aim 4: Examine whether the indirect effects of interpersonal trauma on caregiving sensitivity through emotion regulation are conditional on maternal representations.

Hypothesis 4. There is some work suggesting that having secure or balanced representations of self and other in close relationships confers parenting resilience in the face of trauma (Dayton et al., 2010; Julian et al., 2021; Pearson et al., 1994; Phelps et al., 1998). As balanced representations are characterized by flexible and adaptive emotion regulation strategies, I expected that the effects of childhood maltreatment and IPV on self-reported emotion regulation difficulties would be buffered among women with maternal balanced representations, and thus the indirect effects of interpersonal trauma on caregiving sensitivity through emotion regulation difficulties would be weakened for mothers with balanced prenatal representations of their infants.

METHODS

Participants

The present study used data collected as a part of a larger longitudinal study of the effects of prenatal stress on mothers and infants. Participants were recruited early in their pregnancies from Midwest cities and towns. Participants were oversampled for experiences of IPV and other stressors including financial stress, family conflict, neighborhood violence and food insecurity. Participants completed three waves of data collection during pregnancy, as well as subsequent waves at 1- and 6-months postpartum. Participants were compensated financially for their participation at all waves. All procedures were approved by the university's Institutional Review Board (IRB). The main dependent variables of the proposed study, namely maternal HF-HRV, self-reported emotion regulation, and observed caregiving sensitivity were collected at the 6-month postpartum visit. As data collection for the larger study is ongoing, only data from participants who entered the study between April, 2017 and December, 2020 who had the possibility of completing their 6-month postpartum visit by December 31st, 2021were included in the present analyses. There were 370 women who enrolled in the study during this period. Of these participants, 234 completed the 6-month postpartum assessment. The 37% attrition rate is similar to other longitudinal studies with high-risk community samples (e.g., Cicchetti, Rogosch, & Toth, 2006; Nuttall, Valentino, & Borkowski, 2012). Additionally, due to the HF-HRV measure being added after the start of data collection, as well as pauses to physiological data collection due to the COVID-19 pandemic, usable HF-HRV data was collected from a subsample of 93 participants.

Demographics for the overall sample are presented in Table 1. The average age of participants at enrollment was 26.69 (*SD* = 4.36, range = 18-35). Of the mothers, 32.4% were

primiparous. The racial makeup of the sample was 45.7% white/European American, 35.9% Black/African American, 9.5% multi-racial, 1.6% Asian American/Pacific Islander, .3% Native American, and 7% other/not specified. With regard to ethnicity, 7.6% of the sample identified as Hispanic/Latina. Over half of the sample (55.9%) had a high school degree or less education. The majority of participants had never been married (61.4%), and most were living with a partner (71.6%). The median monthly household income of the sample was $2,300 ($M$ = 2,758.26, SD = 2,290.18, range = 0-17,000). Two-tailed independent samples t-tests were used to assess group differences between participants who completed the 6-month postpartum assessment and those who attritted early. Participants who completed the 6-months postpartum assessment did not significantly differ from those who dropped out prematurely with regard to age [$t(367) = -.173$, $p = .863$], household income [$t(365) = -.697$, $p = .486$], childhood maltreatment [$t(355) = .823$, $p = .411$], or lifetime IPV experiences [$t(327) = .336$, $p = .737$].

Procedures

The study was advertised to potential participants via flyers posted in the local community, on social media websites, and through an Ob-Gyn Perinatal Registry. Women were screened over the phone for study eligibility. Women were eligible to participate if they were below 20 weeks pregnant and in a relationship with a man for at least part of their pregnancy. Additionally, in an effort to demographically match IPV and non-IPV participants, potential participants had to either endorse any experiences of IPV in the past year *or* be eligible for Medicaid based on household income and endorse two or more family stressors. Participants were further excluded if they endorsed medical conditions (e.g., Cushing's disease) or lifestyle factors (e.g., working night shifts) that have the potential to affect salivary cortisol measures, as

cortisol was a variable of interest in the overall study (hormone data was not included in the current research).

The first study visit took place when women were between 15 and 20 weeks pregnant. At this visit, informed consent to participate in the study was obtained and participants reported on demographic information, responded to questionnaires about experiences of childhood maltreatment and IPV in their lifetime and since becoming pregnant, and completed additional measures and procedures that are not the focus of the present study. Experiences of IPV since the last study visit were assessed at all subsequent visits, which took place at 23-25 weeks gestation, 32-34 weeks gestation, and at 1-month and 6-months postpartum. At the third pregnancy visit, participants were also administered the Working Model of the Child Interview (Zeanah et al., 1986) which was coded to assess prenatal representations.

At the 6-month postpartum visit, women arrived at the study offices with their infants. Mothers were fitted with ambulatory heart rate monitors which they wore for the duration of the visit. A two-minute baseline HF-HRV recoding was taken while mothers held their infants on their laps facing away from them and completed non-stressful questionnaires. Next, mothers and infants took part in an 8-minute free play interaction in which mothers were given a variety of age-appropriate toys and instructed to play with their infant as they would at home. This interaction was video recorded and later coded for maternal parenting behavior. After this, mothers and infants were brought to separate rooms where they underwent stress induction paradigms. For mothers, this was the TSST (Kirschbaum et al., 1993) which consisted of three minutes of speech preparation, immediately followed by five minutes of public speaking (mock job interview) and five minutes of mental arithmetic performed in front of two judges who prompted them to continue or start over if they made a mistake. This task has been shown to

reliably produce physiological stress responses, including changes in HRV (Hamidovic et al., 2020). Following the completion of these tasks, women were reunited with their infants and filled out additional questionnaires regarding current symptoms of psychopathology, emotion regulation, and IPV.

Measures

Interpersonal Trauma

Childhood Maltreatment. To assess maternal history of childhood maltreatment, participants completed the short form of the *Childhood Trauma Questionnaire* (CTQ; Bernstein et al., 2003) at the first pregnancy visit. The CTQ is a 28-item questionnaire that assesses perceived childhood maltreatment. It includes five maltreatment subscales assessing physical, emotional, and sexual abuse, physical and emotional neglect, and a validity minimization/denial subscale. Each of the five maltreatment subscales are assessed using five items each ranging from 1 - "Never true" to 5 - "Very often true." The sum of the five subscales yields a total childhood maltreatment score ranging from 25 –125. Example items include "People in my family hit me so hard that it left me with bruises or marks" and "I believe that I was emotionally abused." This scale has demonstrated factorial invariance across community and clinical samples, and has good criterion validity (Bernstein et al., 2003). For the present analyses the sum of the five subscales was used as a measure of overall maltreatment severity, with higher scores indicating more childhood maltreatment ($M = 48.81$, $SD = 21.08$, range = 25–119). In the present sample, Cronbach's alpha for the CTQ total score was $\alpha = .95$ indicating high internal consistency.

IPV. To assess IPV victimization, women completed the *Severity of Violence Against Women Scales* (SVAWS; Marshall, 1992) at each study visit. The SVAWS is a 46-item measure

that assesses psychological, physical, and sexual violence. Example items include "Threated to hurt you" and "Beat you up." Women rated each item on a 4-point frequency scale ranging from 0 - "Never" to 3 - "Many times". High internal consistency has been reported for the subscales (α = .89–.96) as well as for the total score (α = .97) (Huth-Bocks, Levendosky, & Semel, 2001; Marshall, 1992). At the first pregnancy visit, participants responded about IPV experiences ever since they started dating and since they became pregnant. At all subsequent visits they completed the measure for the interval since their last study visit, or in the case of the 1-month postpartum visit, the interval between their last visit and when they gave birth. Item responses for the measure were summed at each administration to produce a total IPV score for the specified time period, with higher scores indicating greater frequency of violence. In the present analyses, alternate models were run to examine the effects of lifetime and pregnancy IPV. Lifetime IPV was measured using the "ever since started dating" SVAWS administration given at the first pregnancy visit (M = 19.12, SD = 27.51, range = 0–138). Pregnancy IPV was measured by summing SVAWS scores across administrations at the first (i.e., "since becoming pregnant"), second and third pregnancy visits, and the one-month postpartum visit (i.e., "since your last interview until you gave birth") to capture IPV experienced during the duration of pregnancy. Pregnancy IPV scores were only computed if SVAWS data was available for at least three of the four timepoints (n = 211, M = 12.57, SD = 23.01, range = 0–188).

Emotion Regulation

Self-reported Emotion Regulation. Maternal self-reported emotion regulation was measured via the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) that was administered at the 6-month postpartum assessment. The scale consists of 36 items rated on a 5-point scale ranging from 1 - "Almost never" to 5 - "Almost Always". Sample items include

"I experience my emotions as overwhelming and out of control" and "When I'm upset, I take time to figure out what I'm really feeling (reverse scored)." The scale yields 6 subscales assessing non-acceptance of emotional responses, difficulty engaging in goal-directed behavior, impulse control difficulties, lack of emotional awareness, limited access to emotion regulation strategies, and lack of emotional clarity, as well as a total score ranging from 36–180, with higher scores representing greater emotion regulation difficulties. High internal consistency (α = .93) and adequate construct and predictive validity has been reported for the DERS (Gratz & Roemer, 2004). In the present sample, the reliability for the total score was high and similar to prior studies (α = .94). The total score was used as the measure of self-reported emotion regulation difficulties in the present study, (M = 78.94, SD = 23.04, range = 36–136).

Heart Rate Variability. ECG data were collected for a subsample of participants during the 6-month postpartum assessment using the Bittium Faros 360 ambulatory cardiac monitoring device (Bittium Corp., Oulu, Finland). The mother's skin was prepared with an alcohol swab, and 3 electrodes were attached on her left and right clavicles and in the center of the sternum. For the baseline recording, mothers held their infants in their laps facing away from them for two minutes while they filled out a standard set of non-stressful questionnaires. Continuous recordings were subsequently taken while mothers participated in the TSST. ECG data was collected at a sampling rate of 1,000 Hz for the majority of participants. Due to experimenter error, data was collected at a sampling rate of 250 Hz for 27 participants. As excellent concordance has been reported for frequency-domain HRV measures collected at 1,000 Hz and 250 Hz, all data was included in the analyses regardless of the sampling frequency (Kwon et al., 2018).

Following data collection, ECG data were imported into Kubios HRV Premium 3.4.3 software for HRV analysis (Tarvainen, Niskanen, Lipponen, Ranta-Aho, & Karjalainen, 2014). R-spikes were initially detected using a built-in automated algorithm. Prior to analysis, data were visually inspected and manually edited for artifacts. Noisy segments that could not be reliably corrected were excluded from analysis. An epoch length of 30 seconds is generally considered the minimum time needed to reliably estimate HF-HRV (Berntson, Quigley, Norman, & Lozano, 2016), therefore, HF-HRV was calculated for each consecutive 30-second interval during the baseline period and TSST, resulting in 4 total epochs for the baseline measurement and 26 epochs for the TSST. The frequency domain parameter HF band power was used as the primary measure of HF-HRV in all analyses. The high frequency bandpass filter was set to 0.15–0.40 Hz to correspond to the recommended respiratory frequency range for adults (Task Force, 1996). HF-HRV was averaged across the two-minute baseline recording, which served as the baseline measure. HF-HRV was also averaged across the epochs of the TSST to produce a task average. All HRV measures were natural log transformed to reduce skewness prior to analysis.

Of the 101 participants from whom ECG data was collected, 93 had usable HRV data from either the baseline period (n = 77) or stress task (n = 88). Regarding the missing data for the baseline, 9 participants did not have a baseline measurement taken due to it not yet being added to the visit procedures, 12 yielded an ECG signal that was too noisy to reliably clean, 2 had a time synching issue, and for 1 participant the heart rate monitor was not turned on correctly. Regarding missing HRV data for the TSST, 7 participants did not complete the TSST during the visit, 4 yielded an ECG signal that was too noisy to reliably clean, 1 participant had a time synching issue, and for 1 participant the heart rate monitor was not turned on correctly.

Maternal Sensitivity

Observed maternal sensitivity was coded from video recordings of the 8-minute mother-infant free play interactions that took place during the 6-month postpartum assessment. The coding scheme was adapted from the NICHD Study of Early Childcare Mother-Infant Interaction scales (Owen, 1992) by Cynthia Frosch and Margaret Owen and is appropriate for use with infants 3-15 months of age. The scheme consists of global ratings of seven domains of maternal behavior: sensitivity to distress, sensitivity to non-distress, intrusiveness, detachment, stimulation of development, positive regard, and negative regard. Items were rated on a five-point scale ranging from 1 - "Not at all characteristic" to 5 - "Highly characteristic." Sensitivity to distress and non-distress ratings reflected the parents' awareness of, interest in, and demonstration of well-timed and appropriate responses to the infant's emotional expressions, interests, and capabilities. Intrusiveness referred to parent-focused overstimulation of the child, or overcontrol at the expense of the infant's autonomous exploration. The detachment scale captured parents' lack of emotional involvement with the infant, passivity, and failure to appropriately scaffold the interaction. The positive regard scale ratings reflected the parent's expression of genuine delight in the infant, whereas the negative regard scale reflected verbal or nonverbal expressions of harshness, disapproval, or nonacceptance of the infant. Intrusiveness, detachment, and negative regard scales were reverse scored such that higher scores on all scales indicated more positive parenting. The scales were used to create a latent maternal sensitivity factor, with higher scores indicating greater maternal sensitivity.

Ratings were made by three trained reliable coders who were blinded to other participant data. A masters-level clinician with prior experience coding mother-child interactions served as the primary coder and oversaw training and reliability. Initial interrater reliability was

established by obtaining interclass correlations of .8 or above with the primary coder on at least 20% of videos, after which coders were allowed to code independently. The coding team met weekly to code videos together and discuss challenging codes to prevent coder drift. The final sample included codes from 219 mother-infant dyads. Regarding missing data, 7 dyads did not complete the free play during their visit, 1 participant did not consent to video recordings, and 7 videos could not be coded due to recording equipment failures or interviewer error.

Maternal Mental Health

Depression. Maternal depression was assessed via the *Edinburgh Postnatal Depression Scale* (EPDS; Cox, Holden, & Sagovsky, 1987) administered at the 6-month postpartum visit. The EPDS is a 10-item self-report questionnaire on which participants rated how often they experienced each symptom over the past week. Example items include "I have been so unhappy that I have had difficulty sleeping" and "I have blamed myself unnecessarily when things went wrong." Items are rated on a 4-point scale, resulting in a total score ranging from 0-30. Scores ranged from 0–26 in the present sample ($M = 9.02$, $SD = 5.91$). Total scores were then dichotomized into Depressed = 1 and Non-depressed = 0 groups based on the recommended clinical cut-off score of 13 or above as indicating probable depression (Cox et al., 1987). In the present sample 27.5% (n = 61) of participants scored in the clinical range for depression.

PTSD. Maternal PTSD symptoms were measured via the *PTSD Checklist for DSM-5* administered at the 6-month postpartum visit (PCL-5; Blevins, Weathers, Davis, Witte, & Domino, 2015). The PCL-5 is a 20-item scale assessing how much the respondent has been bothered by symptoms of PTSD in the past month. Items are rated on a 5-point scale ranging from 1- "Not at all" to 5- "Extremely." Example items include "Having strong physical reactions when something reminded you of the stressful experience (for example, heart pounding, trouble

breathing, sweating)?" and "Avoiding memories, thoughts, or feelings related to the stressful experience?" A total symptom severity score was obtained by summing the scores for each of the 20 items. In the present sample, scores ranged from 0–80 (M = 19.19, SD = 19.06). Total scores were then dichotomized into PTSD = 1 and non-PTSD = 0 groups based on the recommended clinical cuff-off score of 31 or above as indicating probable PTSD (Blevins et al., 2015). In the present sample 24.1% (n = 52) of participants scored in the clinical range for PTSD.

Maternal Representations

The *Working Model of the Child Interview* (WMCI; Zeanah et al., 1986) is a semi-structured interview used to examine mothers' perceptions about their infant's personality and their relationship with the child. The WMCI has been adapted to be administered prenatally to assess maternal representations about the unborn child (Benoit, Parker, & Zeanah, 1997). Prenatal WMCI classifications have demonstrated significant stability with postnatal classifications at 12 months (Benoit et al., 1997), and significantly predict maternal parenting behaviors and infant attachment classifications in theoretically consistent ways (Benoit et al., 1997; Dayton et al., 2010). In the present study, the interview was administered during the third pregnancy visit when women were 32-34 weeks pregnant, resulting in a total of 254 interviews. Interviews, which took about 45 minutes to administer, were audio recorded and transcribed for coding purposes. Transcripts were coded using the coding scheme developed by Zeanah and colleagues (1993).

Interviews are coded into three typologies that conceptually parallel adult attachment classifications. *Balanced representations* are characterized by descriptions conveying emotional warmth and acceptance. Mothers classified as balanced were able to imagine their infant in detail, convincingly described being drawn to comfort their infant during times of distress, and

express a range of emotions in a well-regulated manner. *Disengaged representations* were characterized by emotional deactivation and distancing from the infant. Descriptions of the infant were stereotyped or lacking in detail. *Distorted representations* were characterized by pervasive distortion in the representation of the infant or the relationship in the form of excessive distraction, self-involvement, helpless confusion, or role-reversal. Narratives were lacking in coherence and organization, and descriptions of the infant and relationship were emotionally charged and dysregulated. Representational classifications were dichotomized into Balanced = 1 or Non-Balanced = 0 prior to analysis.

Transcripts were coded by two graduate-level research assistants who received specialized training by a consultant who herself trained to reliability with the first author of the instrument. Before coding the interviews from the current sample, both coders achieved reliability (operationalized as 80% agreement on 10 consecutive transcripts) on a set of interviews that had been consensus coded by the consultant and the developer of the measure, which were treated as the gold standard. Percent agreement for the two coders and Cohen's kappa analyses were used to assess inter-rater reliability for the current sample. Prior to coding independently, reliability analyses were completed on 50 double-coded interviews. Percent agreement for the Balanced/Non-Balanced typology classification was 86%, yielding a kappa value of .63, indicating good agreement (Cicchetti, 1994). Differences in typological classifications were resolved by conferencing, and the resulting consensus classifications were used in the analyses. In the present sample, 39.0% (n = 99) of participants were coded as having Balanced prenatal representations.

Demographic Risk

Given the potential effects of demographic risk factors on maternal parenting and autonomic regulation, demographic risk was controlled for in the analyses. Participants provided information regarding their race/ethnicity, marital status, education, and income (assessed via Medicaid status) during the first pregnancy interview. These variables were coded dichotomously with a score of "1" indicating increased risk (i.e., single, below Medicaid, racial/ethnic minority, high school or less education). Previous research has supported the use of cumulative risk scores over the use of individual risk variables (Sameroff, Seifer, Baldwin, & Baldwin, 1993). Presence or absence of each risk variable was summed to create a cumulative risk score ranging from 0 - 4, (M = 2.53, SD = 1.25).

Data Analytic Plan

Missing data mechanisms were an important consideration for the data analytic plan. Physiological measures were only collected for a subsample of participants as these measures were added when the study was already underway, and collection of physiological data was paused during COVID-19 restrictions. Data are typically considered missing completely at random (MCAR) when a measure is added late to a study, as the order of participant enrollment in the study is theoretically random (Bogat et al., 2021). Additionally, as there was no dependency between the timing of the COVID-19 pandemic and the participants' progression through the study waves, missingness due to the pandemic is also thought to have an MCAR mechanism. As such, modern missing data estimation techniques such as full information maximum likelihood estimation (FIML) can be assumed to produce unbiased parameter estimates (Lang & Little, 2018). However, given the large amount of missing data, all analyses that included the HF-HRV variables, were run separately using the full sample (n = 370) and

with the only the subsample of participants who had HF-HRV data available (n = 93) to ensure consistency of the results.

Statistical power was another important consideration. As the small number of cases with HF-HRV data limits the power to detect significant effects, where possible hypotheses were tested separately using both self-reported and physiological indices of emotion regulation, and a preliminary aim was included to assess the relationship between self-report and physiological indices of emotion regulation (Aim 1). Additionally, the moderated mediation hypotheses were tested only in the self-report models. The hypotheses and corresponding analytic plans are outlined in detail below.

Hypothesis 1

Hypothesis 1 stated that self-reported emotion regulation difficulties would be significantly correlated with lower resting HF-HRV and less HF-HRV suppression from baseline to the TSST. Descriptive statistics and bivariate correlations are presented for all variables of interest, and correlations between self-report and physiological measures of emotion regulation were used to assess Hypothesis 1. A significant negative correlation between DERS total score and baseline HF-HRV will be interpreted as indicating support for this hypothesis. A significant positive correlation between DERS and TSST HF-HRV and a significant negative correlation between the DERS and HF-HRV reactivity (baseline HF-HRV minus TSST HF-HRV) will also indicate support (i.e., as DERS increases, HF-HRV *suppression* in response to stress decreases). As small to moderate correlations between the DERS total score and resting HF-HRV ($r = –.27$, Visted et al., 2017; $r = –.33$, Williams et al. 2015) and HF-HRV reactivity ($r = –.17$, Leerkes et al., 2020) have been reported in the literature, a power analysis was conducted using an effect size of .3 using G*Power (Faul, Erdfelder, Buchner, & Lang, 2009). Given a sample size of 88

(number of participants with TSST HF-HRV data), the power to detect a moderate correlation is .82.

Hypothesis 2a

Hypothesis 2a states that more experiences of interpersonal trauma in childhood and adulthood would predict lower levels of maternal sensitivity, and these effects would be partially mediated through greater self-reported emotion regulation difficulties. This and the following hypotheses were tested using structural equation modeling (SEM) conducted in Mplus version 8 software (Muthén & Muthén, 2017). Prior to conducting the main analyses, confirmatory factor analysis (CFA) was used to create a latent factor for the maternal caregiving sensitivity construct. In order to test the hypothesis, three separate SEM mediation models were fit in which childhood maltreatment, lifetime IPV, and pregnancy IPV, respectively, predicted maternal sensitivity through self-reported emotion regulation. Full information maximum likelihood estimation (FIML) was used to account for missing data due to lack of participation at a wave of data collection or failure to complete a relevant measure (Enders & Bandalos, 2001). Cumulative demographic risk was included as a covariate in all models testing mediation hypotheses. Model fit was assessed using multiple indices including the Chi-square index, comparative fit index (CFI), the Tucker-Lewis index (TLI), and the root mean square error of approximation (RMSEA). CFI and TLI values above .95 and RMSEA values below 0.08 indicate acceptable model fit (Hu & Bentler, 1999). Indirect effects were tested using the percentile bootstrap method with 1,000 resamples to produce 95% confidence intervals around the product coefficients (Falk, 2018). Effects are considered significant if the confidence interval does not contain zero. A simple mediation power analysis conducted in MedPower given the sample size

of n = 370 and assuming moderate effect sizes of the a and b paths indicated that the study is adequately powered to detect a significant indirect effect (Kenny, 2017).

Hypothesis 2b

Hypothesis 2b stated that more experiences of interpersonal trauma in childhood and adulthood would predict lower levels of maternal sensitivity, and these effects would be partially mediated through lower HF-HRV at baseline, and higher HF-HRV during the social stress task. Similar to the above, to test this hypothesis, three separate SEM mediation models were fit in which childhood maltreatment, lifetime IPV, and pregnancy IPV, respectively, predicted maternal sensitivity through both resting and challenged HF-HRV, which were allowed to covary. Cumulative demographic risk was included as a covariate in the models. Because HF-HRV data was only available for a subsample of participants, models were run using the full sample and using only the subsample of participants that had usable HF-HRV data. Model fit and indirect effects were assessed using the same procedures described above.

Hypothesis 3

Hypothesis 3 stated that the indirect effects of interpersonal trauma on caregiving sensitivity through self-reported emotion dysregulation would be strengthened for mothers with clinical levels of depression and PTSD symptoms. To test this hypothesis, six moderated mediation SEM models were specified. Models were identical to those fit for Hypothesis 2a, with the addition of either clinical Depression or PTSD moderating both the a and c' paths. This allowed a test of whether the presence of maternal psychopathology interacts with interpersonal trauma to predict decreased maternal sensitivity directly or indirectly through increased emotion regulation difficulties. Any significant interactions were probed and plotted to aid in

interpretation (Hayes, 2017). Due to sample size limitations, the moderated mediation analyses were not repeated in the HF-HRV models.

Hypothesis 4

Lastly, hypothesis four stated that the indirect effects of interpersonal trauma on caregiving sensitivity through self-reported emotion regulation difficulties would be weakened for mothers with balanced representations. To test this hypothesis, three moderated mediation SEM models were specified. Models were identical to those in Hypothesis 2a, with the addition of Balanced representations moderating both the a and c' paths. This allowed a test of whether the presence of balanced prenatal representations interacts with interpersonal trauma to predict increased maternal sensitivity directly or indirectly through emotion regulation. Any significant interactions were probed and plotted to aid in interpretation (Hayes, 2017).

RESULTS

For ease of presentation the results section is organized by hypothesis. The results of each hypothesis are followed by a summary of the major findings.

Hypothesis 1

Bivariate correlations between the DERS total score and HF-HRV at baseline, HF-HRV during TSST, and change in HF-HRV from baseline to TSST (baseline HF-HRV minus TSST HF-HRV) were conducted. Variable means, standard deviations, and correlations are presented in Table 2. Contrary to the hypothesis, self-reported emotion dysregulation was not significantly correlated with any of the HF-HRV measures.

Hypothesis 2a

Descriptive statistics were calculated for each variable. Means, standard deviations, and correlations are presented in Table 3. Prior to fitting the structural models, a confirmatory factor analysis (CFA) was conducted in Mplus version 8 software (Muthén & Muthén, 2017) to create the maternal sensitivity latent variable. The maternal sensitivity latent factor was predicted to have all seven rating scales [sensitivity to distress, sensitivity to non-distress, stimulation of development, intrusiveness (reverse coded), detachment (reverse coded), positive regard, and negative regard (reverse coded)] as indicators. However, as the free play was not designed to be a distress-eliciting task, sensitivity to distress was only able to be observed and coded for n = 61 dyads, therefore this code was excluded from the analysis. The marker indicator approach to model identification was employed, such that the factor loading of the first indicator (sensitivity to non-distress) was fixed to 1. The resulting latent factor with six indicators (sensitivity to non-distress, stimulation of development, intrusiveness, detachment, positive regard, and negative regard) produced a warning that the residual covariance matrix was not positive definite, and

therefore, the results could not be interpreted. In an effort to improve the model, an exploratory factor analysis (EFA) was run, which revealed that a two-factor model was a better fit to the data compared to the one factor model, $\chi^2\Delta(5) = 89.34$, $p < .001$. Results of the two-factor model indicated that all scales loaded primarily on the first factor, with the exception of intrusiveness which loaded primarily on its own factor. Therefore, the decision was made to drop intrusiveness as an indicator of the maternal sensitivity latent variable. Additionally, as the negative regard code exhibited low variability ($M = 4.72$, $SD = 0.51$, range = 3-5) and had a low factor loading, the decision was made to drop this indicator for parsimony. A one-factor CFA was rerun with sensitivity to non-distress, stimulation of development, detachment (reverse coded), and positive regard as indicators of the latent maternal caregiving sensitivity factor (Figure 1). This model demonstrated good fit to the data and was used as the maternal sensitivity factor in all subsequent analyses, $\chi^2(2) = 4.81$, $p = 0.79$, CFI = 1.00, TLI = 1.03, RMSEA < .001.

Childhood Maltreatment Model

In the first model, childhood maltreatment was modeled as a predictor of maternal sensitivity through self-reported emotion dysregulation. Cumulative demographic risk was included as a covariate in the model. Both predictors (childhood maltreatment and cumulative risk scores) were grand mean centered prior to their inclusion in the model. Maternal sensitivity was included as a latent factor identified by fixing the first factor loading to 1, while all other variables were treated as observed. Model fit was good ($\chi^2(11) = 12.57$, $p = 0.32$, CFI = .99, TLI = .98, RMSEA = .02). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 4. Figure 2 presents a path diagram of the model displaying standardized path coefficient estimates. History of childhood maltreatment was significantly associated with greater self-reported emotion regulation difficulties ($b = 0.190$, s.e. = 0.069, $p = .006$). However,

contrary to the hypothesis, maternal history of childhood maltreatment did not significantly predict observed maternal caregiving sensitivity (b = –0.003, s.e. = 0.003, p = .297), nor did self-reported emotion regulation difficulties predict observed maternal caregiving sensitivity (b = 0.000, s.e. = 0.003, p = .886). Cumulative demographic risk was significantly associated with less sensitive caregiving (b = –0.181, s.e. = 0.052, p = .001).

Tests of the indirect effects revealed that self-reported emotion regulation was not a significant mediator of the effect of childhood maltreatment history on early maternal caregiving sensitivity, 95% CI = [–0.001, 0.001]. The direct effect of childhood maltreatment on sensitivity was also not significant, 95% CI = [–0.008, 0.003]. The indirect effect of demographic risk on maternal sensitivity through emotion regulation was not significant, 95% CI = [–0.007, 0.011]. However, there was a significant direct effect of cumulative demographic risk on maternal sensitivity, 95% CI = [–0.291, –0.079] such that more risk factors predicted lower sensitivity.

Lifetime IPV Model

Next a model was run to test whether lifetime history of IPV predicted maternal sensitivity through self-reported emotion regulation difficulties. Again, cumulative demographic risk was included as a covariate in the model, and both predictors (lifetime IPV and cumulative demographic risk) were grand mean centered. Maternal sensitivity was included as a latent factor identified by fixing the first factor loading to 1, while all other variables were observed. Model fit was good ($\chi^2(11) = 14.16$, $p = 0.22$, CFI = .98, TLI = .97, RMSEA = .03). Unstandardized path coefficient estimates, standard errors, and p–values are presented in Table 5. Figure 3 presents a path diagram of the model displaying standardized path coefficient estimates. In this model cumulative demographic risk was the only significant predictor of maternal sensitivity such that more demographic risk factors predicted less sensitive caregiving behavior (b = –0.193,

s.e. = 0.054, $p < 0.001$). Neither lifetime exposure to IPV ($b = 0.001$, s.e. = 0.002, $p = 0.570$) nor self-reported emotion regulation difficulties (b = –0.001, s.e. = 0.003, $p = 0.679$) were significant predictors of maternal sensitivity. Lifetime IPV also did not significantly predict emotion regulation difficulties ($b = 0.096$, s.e. = 0.068, $p = 0.159$).

Tests of the indirect effects revealed that self-reported emotion regulation difficulties did not a significantly mediate the effect of lifetime IPV exposure on maternal caregiving sensitivity, 95% CI = [–0.001, 0.000]. The direct effect of lifetime IPV on sensitivity was also not significant, 95% CI = [–0.003, 0.005]. The indirect effect of demographic risk on maternal sensitivity through self-reported emotion regulation was also not significant, 95% CI = [–0.005, 0.010]. However, there was a significant negative direct effect of demographic risk on maternal sensitivity, 95% CI = [–0.305, –0.090].

Pregnancy IPV Model

A third model was run to test whether IPV that occurred during the pregnancy period predicted maternal sensitivity through self-reported emotion regulation difficulties. Cumulative demographic risk was included as a covariate in the model, and both predictors (pregnancy IPV and cumulative demographic risk) were grand mean centered. Maternal sensitivity was included as a latent factor identified by fixing the first factor loading to 1, while all other variables were observed. Model fit was good ($\chi^2(11) = 12.41$, $p = 0.33$, CFI = .99, TLI = .99, RMSEA = .02). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 6. Figure 4 presents a path diagram of the model displaying standardized path coefficient estimates. As in the lifetime IPV model, cumulative demographic risk was the only significant predictor of maternal sensitivity such that more demographic risk factors predicted less sensitive caregiving behavior ($b = –0.193$, s.e. = 0.054, $p < 0.001$). Neither IPV during pregnancy ($b =$

0.001, s.e. = 0.002, p = 0.717) nor self-reported emotion regulation difficulties (b = –0.001, s.e. = 0.003, p = 0.715) were significantly associated with maternal sensitivity. Pregnancy IPV also did not significantly predict emotion regulation difficulties (b = 0.107, s.e. = 0.105, p = 0.308).

Tests of the indirect effects revealed that self-reported emotion regulation was not a significant mediator of the effect of pregnancy IPV on early maternal caregiving sensitivity, 95% CI = [–0.001, 0.000], nor was there a significant direct effect of pregnancy IPV on sensitivity, 95% CI = [–0.004, 0.007]. The indirect effect of demographic risk on maternal sensitivity through self-reported emotion regulation difficulties was also not significant, 95% CI = [–0.006, 0.011], but there was a significant direct effect of demographic risk on sensitivity, 95% CI = [–0.303, –0.089].

Hypothesis 2b

Childhood Maltreatment Model

A model was fit in which childhood maltreatment predicted maternal sensitivity through baseline and challenged HF-HRV[2]. Cumulative demographic risk was included as a covariate in the model. Both predictors (childhood maltreatment and demographic risk scores) were grand mean centered prior to their inclusion in the model. Maternal sensitivity was included as a latent factor identified by fixing the first factor loading to 1, while all other variables were treated as observed. Covariances were allowed between the predictors and between the two HF-HRV measures. Model fit was good ($\chi^2(14) = 14.34$, p = 0.42, CFI = 1.00, TLI = 1.00, RMSEA = .01). Unstandardized path coefficient estimates, standard errors, and p–values are presented in Table

[2] Due to the high proportion of missing data for the HF-HRV variables, for all analyses using HF-HRV data, models using the full sample (N = 370) were compared to models using only the subsample of participants who had any usable HRV data (N = 93). As the findings and interpretations did not differ substantively between these models, the results of the full sample models are presented throughout.

7. Figure 5 presents a path diagram of the model displaying standardized path coefficient estimates.

As in the previous models, cumulative demographic risk was significantly associated with less sensitive caregiving ($b = –0.139$, s.e. = 0.059, $p = .019$). The effect of childhood maltreatment on maternal sensitivity was not significant ($b = –0.001$, s.e. = 0.003, $p = 0.752$). History of childhood maltreatment was also not significantly associated with baseline HF-HRV ($b = 0.011$, s.e. = 0.008, $p = 0.176$); however, childhood maltreatment was significantly associated with HF-HRV during the TSST such that women who reported more childhood maltreatment had higher HF-HRV on average during the stress task ($b = 0.012$, s.e. = 0.006, $p = 0.044$). In turn, baseline HF-HRV was not a significant predictor of maternal sensitivity ($b = 0.239$, s.e. = 0.140, $p = 0.089$), but HF-HRV during the TSST did significantly predict maternal sensitivity in the expected direction such that women who had higher HF-HRV during the stress task exhibited less sensitivity when interacting with their infants ($b = –0.393, 0.183, p = 0.032$).

Tests of the indirect effects revealed that the effect of childhood maltreatment on early maternal caregiving sensitivity was not significantly mediated through baseline HF-HRV, 95% CI = [–0.002, 0.009], nor challenged HF-HRV, 95% CI = [–0.012, 0.000]. The direct effect of childhood maltreatment on sensitivity was also not significant 95% CI = [–0.007, 0.005]. The indirect effects of demographic risk on maternal sensitivity through baseline and challenged HF-HRV were not significant [95% CI = (–0.083, 0.044) and 95% CI = (–0.094, 0.033); respectively], but there was a significant direct effect of demographic risk on maternal sensitivity, 95% CI = [–0.258, –0.025].

Lifetime IPV Model

Next, a model was fit in which lifetime history of IPV predicted maternal sensitivity through baseline and challenged HF-HRV. Cumulative demographic risk was included as a covariate in the model. Both predictors (lifetime IPV and cumulative risk scores) were grand mean centered prior to their inclusion in the model. Maternal sensitivity was included as a latent factor identified by fixing the first factor loading to 1, while all other variables were treated as observed. Covariances were allowed between the predictors and between the HF-HRV measures. Model fit was good ($\chi^2(14) = 14.87$, $p = 0.38$, CFI = 1.00, TLI = 0.99, RMSEA = .01). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 8. Figure 6 presents a path diagram of the model displaying standardized path coefficient estimates.

In this model, cumulative demographic risk was significantly associated with less sensitive caregiving ($b = –0.145$, s.e. = 0.063, $p = .021$) while the effect of lifetime IPV exposure on maternal sensitivity was not significant ($b = 0.003$, s.e. = 0.003, $p = 0.258$). Lifetime IPV exposure was not significantly associated with either baseline HF-HRV (b = 0.003, s.e. = 0.008, $p = 0.646$) or HF-HRV during the stress task ($b = 0.007$, s.e. = 0.005, $p = 0.189$). Baseline HF-HRV was not a significant predictor of maternal sensitivity ($b = 0.239$, s.e. = 0.154, $p = 0.121$), however HF-HRV during the TSST did significantly predict maternal sensitivity in the expected direction such that women who had higher HF-HRV during the stress task exhibited less sensitivity when interacting with their infants ($b = –0.398$, s.e. = 0.194, $p = 0.039$).

Tests of the indirect effects revealed that the effect of lifetime IPV on early maternal caregiving sensitivity was not significantly mediated through baseline HF-HRV, 95% CI = [–0.003, 0.006], nor challenged HF-HRV, 95% CI = [–0.009, 0.001], and the direct effect of

lifetime IPV on maternal sensitivity was not significant 95% CI = [–0.002, 0.008]. The indirect effects of demographic risk on maternal sensitivity through baseline and challenged HF-HRV were also not significant, 95% CI = [–0.081, 0.060] and 95% CI = [–0.134, 0.025], respectively. However, there was a significant direct effect of demographic risk on sensitivity in the negative direction, 95% CI = [–0.264, –0.024].

Pregnancy IPV Model

Lastly, a model was fit in which IPV during pregnancy was modeled as a predictor of maternal sensitivity through baseline and challenged HF-HRV. Cumulative demographic risk was included as a covariate in the model. Both predictors (pregnancy IPV and cumulative risk scores) were grand mean centered prior to their inclusion in the model. Maternal sensitivity was included as a latent factor identified by fixing the first factor loading to 1, while all other variables were treated as observed. Covariances were allowed between the predictors and between the two HF-HRV measures. Model fit was good ($\chi^2(14) = 13.24$, $p = 0.51$, CFI = 1.00, TLI = 1.00, RMSEA = <.01). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 9. Figure 7 presents a path diagram of the model displaying standardized path coefficient estimates.

In this model, cumulative demographic risk was significantly associated with less sensitive caregiving ($b = –0.145$, s.e. = 0.062, $p = .019$). The direct effect of pregnancy IPV exposure on maternal sensitivity was not significant ($b = 0.001$, s.e. = 0.003, $p = 0.757$). Pregnancy IPV exposure was also not significantly associated with either baseline HF-HRV ($b = –0.004$, s.e. = 0.007, $p = 0.617$) or HF-HRV during the stress task ($b = –0.002$, s.e. = 0.005, $p = 0.764$). Baseline HF-HRV was not a significant predictor of maternal sensitivity ($b = 0.229$, s.e. = 0.151, $p = 0.129$), however HF-HRV during the TSST did significantly predict maternal

sensitivity in the expected direction such that women who had higher HF-HRV during the stress task exhibited less sensitivity when interacting with their infants (b = –0.382, s.e. = 0.185, p = 0.039).

Tests of the indirect effects revealed that the effect of pregnancy IPV on maternal caregiving sensitivity was not significantly mediated through baseline HF-HRV, 95% CI = [–0.005, 0.005], nor challenged HF-HRV, 95% CI = [–0.003, 0.007]. The direct effect of pregnancy IPV on maternal sensitivity was also not significant, 95% CI = [–0.008, 0.006]. The indirect effects of demographic risk on maternal sensitivity through baseline and challenged HF-HRV were not significant, 95% CI = [–0.081, 0.061] and 95% CI = [–0.134, 0.026], respectively. However, there was a significant negative direct effect of demographic risk on maternal sensitivity, 95% CI = [–0.264, –0.028].

Hypothesis 2 Summary

Overall, the hypothesis that maternal experiences of interpersonal trauma in childhood, adulthood, and during pregnancy would negatively affect early caregiving sensitivity through self-reported emotion regulation difficulties was not supported. Although childhood maltreatment significantly predicted increased self-reported emotion regulation difficulties, neither childhood maltreatment nor IPV (lifetime or during pregnancy) significantly predicted observed caregiving sensitivity during the free play. Self-reported emotion regulation difficulties were also not significantly associated with observed maternal sensitivity. Indirect effects of interpersonal trauma variables on maternal sensitivity through emotion regulation were not significant, therefore there was no evidence for mediation. Cumulative demographic risk did significantly predict maternal sensitivity such that women with more demographic risk factors

demonstrated less sensitive caregiving behavior with their 6-month-olds during the free play task.

There was some support for the hypothesis that physiological dysregulation would predict less sensitive caregiving. Controlling for baseline HF-HRV, higher HF-HRV (i.e., less vagal withdrawal) during the stress task predicted less sensitivity during the free play. Additionally, childhood maltreatment (but not lifetime or pregnancy IPV) predicted higher HF-HRV during the stress task. However, the indirect effect of childhood maltreatment on maternal caregiving sensitivity through challenged HF-HRV was not significant, and therefore the evidence fell short of supporting mediation.

In summary, experiences of childhood maltreatment predicted both self-reported and physiological emotion regulation difficulties. IPV experiences throughout one's dating history and during the most recent pregnancy were not associated with either self-reported or physiological indices of emotion dysregulation. Physiological dysregulation during the social stress task was associated with lower maternal sensitivity during the mother-infant interaction. Lastly, having more demographic risk factors also predicted lower maternal sensitivity during the mother-infant interaction.

Hypothesis 3

Descriptive statistics were calculated for each variable. Means, standard deviations, and correlations are presented in Table 10. Endorsing clinical levels of depression symptoms was significantly positively correlated with childhood maltreatment severity ($r = .183$, $p = .006$) and lifetime IPV severity ($r = .146$, $p = .038$). Clinical depression was also positively correlated with self-reported emotion regulation difficulties ($r = .492$, $p < .001$). Endorsing clinical levels of PTSD symptoms was significantly positively correlated with childhood maltreatment history ($r =$

.142, $p = .038$) as well as self-reported emotion regulation difficulties ($r = .261$, $p < .001$). Clinical depression and PTSD were also positively correlated ($r = .425$, $p < .001$).

Childhood Maltreatment x Depression Model

In the first moderated mediation model, childhood maltreatment, clinical depression, and their interaction were included as predictors of maternal sensitivity directly and indirectly through self-reported emotion dysregulation (i.e., moderation of both the a and c' paths). Cumulative demographic risk was included as a covariate in the model. Childhood maltreatment and cumulative risk scores were grand mean centered prior to their inclusion in the model. Maternal sensitivity was included as a latent factor identified by fixing the first factor loading to 1, while all other variables were observed. Model fit was acceptable ($\chi^2(17) = 27.47$, $p = 0.05$, CFI = .96, TLI = .92, RMSEA = .04). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 11.

Current depression was positively associated with self-reported emotion regulation difficulties ($b = 24.734$, s.e. = 2.702, $p < .001$), but it was not significantly associated with caregiving sensitivity ($b = 0.000$, s.e. = 0.164, $p = 1.000$). Contrary to the hypothesis, the interaction between childhood maltreatment and current depression was not a significant predictor of emotion regulation difficulties ($b = –0.028$, s.e. = 0.116, $p = .808$) nor did the interaction significantly predict caregiving sensitivity ($b = 0.004$, s.e. = 0.007, $p = .506$). With depression included in the model, the indirect effect of the childhood maltreatment on sensitivity through self-reported emotion regulation difficulties was not significant, 95% CI = [–0.001, 0.001], nor was there a significant direct effect of childhood maltreatment on sensitivity 95% CI = [–0.011, 0.003]. Therefore, there was no support for clinical depression as a moderator of the hypothesized model.

Lifetime IPV x Depression Model.

The analyses were identical to the prior model with Lifetime IPV substituted for Childhood Maltreatment. Model fit was good ($\chi^2(17) = 21.507$, $p = 0.204$, CFI = .98, TLI = .97, RMSEA = .03). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 12.

Current depression was positively associated with self-reported emotion regulation difficulties ($b = 24.974$, s.e. = 2.826, $p < .001$), but it was not significantly associated with caregiving sensitivity ($b = –0.012$, s.e. = 0.169, $p = 0.944$). Contrary to the hypothesis, the interaction between lifetime IPV and current depression was not a significant predictor of emotion regulation difficulties ($b = 0.058$, s.e. = 0.114, $p = .612$) nor did the interaction significantly predict caregiving sensitivity ($b = 0.001$, s.e. = 0.005, $p = 0.874$). With depression included in the model, the indirect effect of the lifetime IPV on sensitivity through self-reported emotion regulation difficulties was not significant, 95% CI = [–0.001, 0.000], nor was there a significant direct effect of lifetime IPV on sensitivity, 95% CI = [–0.004, 0.007]. Therefore, there was no support for clinical depression as a moderator of the hypothesized model.

Pregnancy IPV x Depression Model

The analyses were identical to the prior model with Pregnancy IPV substituted for Lifetime IPV. Model fit was good ($\chi^2(17) = 17.141$, $p = 0.445$, CFI = 1.00, TLI = 1.00, RMSEA = .01). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 13.

Current depression was positively associated with self-reported emotion regulation difficulties ($b = 25.104$, s.e. = 2.805, $p < .001$) but was not significantly associated with caregiving sensitivity ($b = –0.012$, s.e. = 0.163, $p = 0.942$). Contrary to the hypothesis, the

interaction between pregnancy IPV and depression was not a significant predictor of emotion regulation difficulties ($b = 0.026$, s.e. = 0.164 $p = .875$), nor did the interaction significantly predict caregiving sensitivity ($b = 0.007$, s.e. = 0.007, $p = 0.294$). With depression in the model, the indirect effect of the pregnancy IPV on sensitivity through self-reported emotion regulation difficulties was not significant, 95% CI = [–0.001, 0.001], nor was the direct effect of pregnancy IPV on sensitivity, 95% CI = [–0.007, 0.006]. Therefore, there was no support for clinical depression as a moderator of the hypothesized model.

Childhood Maltreatment x PTSD Model

The analyses were identical to the moderated mediation models above with PTSD substituted for Depression. Model fit was acceptable ($\chi^2(17) = 18.10$, $p = 0.38$, CFI = .99, TLI = .99, RMSEA = .01). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 14.

Current PTSD was positively associated with self-reported emotion regulation difficulties ($b = 13.599$, s.e. = 3.685, $p < .001$), but it was not significantly associated with caregiving sensitivity ($b = –0.091$, s.e. = 0.145, $p = .528$). Contrary to the hypothesis, the interaction between childhood maltreatment and PTSD was not a significant predictor of emotion regulation difficulties ($b = –0.087$, s.e. = 0.152, $p = .567$), nor did the interaction significantly predict caregiving sensitivity ($b = 0.001$, s.e. = 0.006, $p = 0.927$). With PTSD included in the model, the indirect effect of childhood maltreatment on sensitivity through self-reported emotion regulation was not significant, 95% CI = [–0.001, 0.001], nor was there a significant direct effect of childhood maltreatment on sensitivity 95% CI = [–0.010, 0.005]. Therefore, there was no support for PTSD as a moderator of the hypothesized model.

Lifetime IPV x PTSD Model

Model fit was good ($\chi^2(17) = 22.410$, $p = 0.170$, CFI = .97, TLI = .95, RMSEA = .03). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 15. Current PTSD was positively associated with self-reported emotion regulation difficulties ($b = 13.024$, s.e. = 3.656, $p < .001$), but it was not significantly associated with caregiving sensitivity ($b = -0.123$, s.e. = 0.142, $p = 0.384$). Contrary to the hypothesis, the interaction between lifetime IPV and PTSD was not a significant predictor of emotion regulation difficulties ($b = 0.246$, s.e. = 0.128, $p = .055$) nor did the interaction significantly predict caregiving sensitivity ($b = 0.001$, s.e. = 0.005, $p = 0.795$). With PTSD included in the model, the indirect effect of the lifetime IPV on sensitivity through self-reported emotion regulation was not significant, 95% CI = [–0.001, 0.000], nor was there a significant direct effect of lifetime IPV on sensitivity, 95% CI = [–0.004, 0.006]. Therefore, there was no support for PTSD as a moderator of the hypothesized model.

Pregnancy IPV x PTSD Model

Model fit was good ($\chi^2(17) = 17.049$, $p = 0.451$, CFI = 1.00, TLI = 1.00, RMSEA = .003). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 16. Current PTSD was positively associated with self-reported emotion regulation difficulties ($b = 13.098$, s.e. = 3.779, $p = .001$), but it was not significantly associated with caregiving sensitivity ($b = -0.157$, s.e. = 0.137, $p = 0.253$). Contrary to the hypothesis, the interaction between pregnancy IPV and PTSD was not a significant predictor of emotion regulation difficulties ($b = 0.168$, s.e. = 0.241 $p = .486$) nor did the interaction significantly predict caregiving sensitivity ($b = 0.013$, s.e. = 0.008, $p = 0.088$). With PTSD included in the model, the indirect effect of pregnancy IPV on sensitivity through self-reported emotion

regulation difficulties was not significant, 95% CI = [–0.001, 0.001], nor was the direct effect of pregnancy IPV on sensitivity 95% CI = [–0.012, 0.003]. However, there was a significant direct effect of the pregnancy IPV by PTSD interaction on maternal sensitivity, 95% CI = [0.003, 0.033].

Given the significance of this interaction according to the bootstrapped confidence interval, the interaction was plotted (Figure 8) and simple slope analyses were conducted to aid in interpretation. For mothers without clinical levels of PTSD symptoms, the simple slope of the effect of pregnancy IPV on caregiving sensitivity was $b = –0.002$, s.e. = 0.004, $p = 0.512$. For mothers reporting clinical levels of PTSD symptoms, the simple slope of the effect of pregnancy IPV on caregiving sensitivity was $b = 0.011$, s.e. = 0.006 $p = 0.095$. Therefore, the effects of pregnancy IPV on sensitivity did not differ from zero at either clinical or subclinical levels of PTSD symptoms, however, the effects did differ from one another such that at high levels of pregnancy IPV, those with clinical PTSD symptoms displayed more sensitive caregiving. The Johnson-Neyman procedure was used to test regions of significance (P. O. Johnson & Neyman, 1936). The plot (Figure 9) revealed that the positive effect of PTSD on maternal sensitivity was significant only when pregnancy IPV was greater than two standard deviations above the mean.

Hypothesis 3 Summary

In summary, hypothesis 3 was generally not supported. While clinical levels of depression and PTSD symptoms were associated with greater self-reported emotion regulation difficulties, the interactions between maternal psychopathology and interpersonal trauma did not significantly predict either self-reported emotion regulation difficulties or maternal sensitivity. There was however evidence that current PTSD moderated the effect of pregnancy IPV on

maternal sensitivity, such that mothers with clinical PTSD symptoms were more sensitive in their interactions with their infants if they also experienced high levels of IPV during pregnancy.

Hypothesis 4

Descriptive statistics were calculated for each variable. Means, standard deviations, and correlations are presented in Table 17. Balanced representations were significantly negatively correlated with pregnancy IPV ($r = –.151$, $p = .032$) and cumulative demographic risk ($r = –.203$, $p = .001$). Balanced maternal representations were significantly positively correlated with sensitivity to non-distress ($r = .162$, $p = .021$), stimulation of development ($r = .169$, $p = .015$), reverse coded detachment ($r = .230$, $p < .001$), and positive regard ($r = .180$, $p = .010$).

Childhood Maltreatment x Maternal Representation Model

In this moderated mediation model, childhood maltreatment, maternal representations, and their interaction were included as predictors of maternal sensitivity directly and indirectly through self-reported emotion regulation difficulties (i.e., moderation of both the a and c' paths). Cumulative demographic risk was included as a covariate in the model. Childhood maltreatment and cumulative risk scores were grand mean centered prior to their inclusion in the model. Maternal sensitivity was included as a latent factor identified by fixing the first factor loading to 1, while all other variables were observed. Model fit was acceptable ($\chi^2(17) = 22.69$, $p = 0.16$, CFI = .97, TLI = .94, RMSEA = .03). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 18.

Balanced representations were positively associated with observed maternal sensitivity ($b = 0.305$, s.e. = 0.120, $p = .011$) but they were not significantly associated with self-reported emotion regulation difficulties ($b = –1.947$, s.e. = 1.333, $p = .492$). Contrary to the hypothesis, the interaction between childhood maltreatment and maternal representations was not a

significant predictor of emotion regulation difficulties (b = –0.131, s.e. = 0.154, p = .395) nor did the interaction significantly predict caregiving sensitivity (b = –0.004, s.e. = 0.006, p = .510). With maternal representations included in the model, the indirect effect of childhood maltreatment on sensitivity through self-reported emotion regulation was not significant, 95% CI = [–0.002, 0.001] nor was there a significant direct effect of childhood maltreatment on sensitivity 95% CI = [–0.008, 0.007]. Therefore, there was no support for maternal representations as a moderator of the hypothesized model. There was however a significant direct effect of balanced representations on maternal sensitivity, 95% CI = [0.071, 0.530], such that having balanced prenatal representations predicted greater observed caregiving sensitivity. The indirect effect of balanced representations on maternal sensitivity through self-reported emotion regulation difficulties was not significant, 95% CI = [–0.017, 0.027].

Lifetime IPV x Maternal Representation Model

The analyses for this model were identical to the prior one with Lifetime IPV substituted for Child Maltreatment. Model fit was good ($\chi^2(17)$ = 19.416, p = 0.305, CFI = .99, TLI = .98, RMSEA = .02). Unstandardized path coefficient estimates, standard errors, and p–values are presented in Table 19.

Balanced representations were positively associated with observed maternal sensitivity (b = 0.324, s.e. = 0.116, p = .005) but they were not significantly associated with self-reported emotion regulation difficulties (b = –2.653, s.e. = 3.486, p = .447). Contrary to the hypothesis, the interaction between lifetime IPV and maternal representations was not a significant predictor of emotion regulation difficulties (b = –0.105, s.e. = 0.151, p = .488) nor did the interaction significantly predict caregiving sensitivity (b = 0.005, s.e. = 0.004, p = .205). With maternal representations included in the model, the indirect effect of lifetime IPV on sensitivity through

self-reported emotion regulation was not significant, 95% CI = [–0.001, 0.001], nor was there a significant direct effect of lifetime IPV on sensitivity, 95% CI = [–0.006, 0.004]. Therefore, there was no support for maternal representations as a moderator of the hypothesized model. There was however a significant direct effect of balanced representations on maternal sensitivity, 95% CI = [0.089, 0.541], such that having balanced prenatal representations predicted greater observed caregiving sensitivity. The indirect effect of balanced representations on maternal sensitivity through self-reported emotion regulation difficulties was not significant, 95% CI = [–0.027, 0.030].

Pregnancy IPV x Maternal Representation Model

The analyses for this model were identical to the prior one with Pregnancy IPV substituted for Lifetime IPV. Model fit was good ($\chi^2(17) = 17.157$, $p = 0.444$, CFI = 1.00, TLI = 1.00, RMSEA = .01). Unstandardized path coefficient estimates, standard errors, and *p*–values are presented in Table 20.

Balanced representations were positively associated with observed maternal sensitivity ($b = 0.321$, s.e. = 0.121, $p = .008$), but they were not significantly associated with self-reported emotion regulation difficulties ($b = –2.487$, s.e. = 4.198, $p = .554$). Contrary to the hypothesis, the interaction between pregnancy IPV and maternal representations was not a significant predictor of emotion regulation difficulties ($b = –0.221$, s.e. = 0.354, $p = .532$) nor did the interaction significantly predict caregiving sensitivity ($b = –0.004$, s.e. = 0.008, $p = .596$). With maternal representations included in the model, the indirect effect of pregnancy IPV on sensitivity through self-reported emotion regulation was not significant, 95% CI = [–0.002, 0.001], nor was there a significant direct effect of pregnancy IPV on sensitivity 95% CI = [–0.003, 0.009]. Therefore, there was no support for maternal representations as a moderator of the

hypothesized model. There was however a significant direct effect of balanced representations on maternal sensitivity, 95% CI = [0.075, 0.550], such that having balanced prenatal representations predicted greater observed caregiving sensitivity. The indirect effect of balanced representations on maternal sensitivity through self-reported emotion regulation difficulties was not significant, 95% CI = [–0.027, 0.033].

Hypothesis 4 Summary

The hypothesis that balanced prenatal representations would buffer against the negative effects of interpersonal trauma on maternal sensitivity through emotion regulation was not supported. In all three models, the interaction between balanced representations and maternal experiences of trauma were not significant predictors of either self-reported emotion regulation difficulties or maternal sensitivity. However, in all three models, having balanced prenatal representations directly predicted greater observed caregiving sensitivity during the 6-month postpartum free play task. This effect was not mediated through self-reported emotion regulation.

DISCUSSION

The goal of the present study was to examine potential mechanisms through which experiences of interpersonal trauma including childhood maltreatment and IPV affect early caregiving sensitivity. In particular, I chose to examine the mediating role of maternal emotion regulation, as both interpersonal trauma and insensitive caregiving have been linked to emotion regulation deficits (e.g., Carreras et al., 2019; Dvir et al., 2014; Greene et al., 2021; Leerkes et al., 2016; Miragoli et al., 2020; Pat-Horenczyk et al., 2015). Both self-report and physiological measures of emotion regulation were included in the study in the hopes of better understanding their concordance and their ability to predict parenting outcomes (Leerkes, 2020). In addition, maternal psychopathology, including clinical depression and PTSD, and maternal representations were examined as potential moderators of the effects of interpersonal trauma on caregiving sensitivity, as both have been highlighted in the literature as relevant to parenting outcomes in the context of trauma, and both are theoretically related to emotion regulation abilities (Aldao et al., 2016; Mikulincer & Shaver, 2019). Overall, the expectation that greater exposure to interpersonal trauma in childhood and adulthood would predict less sensitive caregiving was not supported, nor were the mediation or moderated mediation hypotheses. However, several important takeaways can be gleaned from the study results.

Contrary to expectations, none of the HF-HRV measures (baseline HF-HRV, challenged HF-HRV, or HF-HRV reactivity) were significantly correlated with women's self-reported emotion regulation difficulties. This was surprising given that previous studies found significant associations between the DERS and HF-HRV measures. For example, both Williams et al. (2015) and Visted et al. (2017) found that resting vagally-mediated HRV was negatively correlated with total scores on the DERS. Procedural differences in baseline HF-HRV

measurement between this research and prior research might account for the lack of association in the present sample. Rather than sitting alone and staring at a gray screen during the baseline period, participants in the present study had their infants in their laps and completed non-arousing questionnaires while the baseline measurement was being taken. It is possible that the increased cognitive load affected the quality of our baseline measurement, as HF-HRV is known to vary in relation to cognitive demand (Luque-Casado, Perales, Cárdenas, & Sanabria, 2016). Additionally, although several studies report that maternal autonomic functioning returns to normal within 3-4 months postpartum, there may be individual differences. Thus, possible physiological differences between recently pregnant participants versus non-perinatal participants could have played a role in the discrepant findings (R. L. Brown, Fagundes, Thayer, & Christian, 2021; Chen et al., 1999).

With regard to HF-HRV reactivity, Leerkes (2020) found that mothers who reported greater emotion regulation difficulties on the DERS, exhibited less vagal withdrawal from baseline to distress-eliciting interactions with their 6-month-old infants (i.e., less HF-HRV reduction from baseline to the average of arm restraint, novel toy presentation, and still-face reengagement tasks). We were unable to replicate these results using the TSST as the stress induction task. There is not a consensus in the literature as to what degree of change in HF-HRV constitutes reactivity, and comparisons between studies are hindered by inconsistencies in the types of HRV parameters reported (e.g., time vs. frequency domain) so it is not clear whether the level of reactivity seen in the present study was comparable to other samples. Most studies report mean reductions in HRV from baseline to the challenge tasks such as the TSST (e.g., Eagle, Rash, Tice, & Proeschold-Bell, 2021; Giese-Davis et al., 2006; Mackersie & Calderon-Moultrie, 2016; Rohleder, Wolf, Maldonado, & Kirschbaum, 2006); however, others report mean increases

similar to that seen in the present study (e.g., Dale et al., 2018; Shinba, 2014; Yim, Quas, Rush, Granger, & Skoluda, 2015). In the present sample, 78% of participants displayed an increase in HF-HRV in response to the stressor while the other 22% showed the expected decrease. Other studies have likewise failed to find evidence for convergence of self-report and physiological reactivity measures. For example, Ostlund et al. (2019) reported that among expectant mothers, vagal withdrawal in response to infant cry videos was not significantly associated with self-reported emotion dysregulation; however, both physiological and self-report indices of maternal emotion regulation were independently associated with infant neurobehavioral outcomes. Another study found that mother's HRV suppression from baseline to a parenting-themed speech task was not significantly associated with self-reported emotion regulation difficulties on the DERS (Davis, Suveg, & Shaffer, 2015). Given these discrepant findings, it is possible that stressful interactions with one's own infant results in autonomic responses that are more aligned with self-report measures compared to simulated or non-parenting stressors such as the one used in the present study. Nonetheless, our results suggest that self-reported emotion regulation cannot be assumed to be a good proxy for physiological measures of autonomic regulation. Interestingly, self-report and autonomic measures of emotion regulation seemed to operate differently with respect to parenting outcomes in the present study, which reinforces the importance of utilizing multiple methods of measurement in future research.

The second hypothesis stated that maternal experiences of interpersonal trauma, including childhood maltreatment and lifetime and pregnancy IPV, would predict less maternal sensitivity, and that these effects would be partially mediated through self-reported and physiological measures of emotion regulation. Overall, this hypothesis was not supported. Neither childhood maltreatment nor IPV significantly predicted observed caregiving sensitivity,

suggesting that, in the present sample, women's abilities to engage in sensitive caregiving during a naturalistic free-play task was largely resilient to experiences of interpersonal trauma. This finding contradicts meta-analytic work that suggests relational trauma is associated with less positive and more negative parenting practices (Chiesa et al., 2018; Savage et al., 2019). The studies included in these analyses encompassed a wide range of children's ages (0-11 and 0-6 respectively) and used a variety of parenting measures, which could help to explain this discrepancy. However, at least in the case of childhood maltreatment, the authors failed to find evidence of moderation by child age or by self-report vs. observational parenting measures (Savage et al., 2019). Effect sizes in both meta-analyses were small (ranging from $r = -.08$ to $r = .17$) and tended to be larger for the effects of trauma on negative parenting behaviors (e.g., physical aggression), which were not the focus of the present study. Additionally, publication bias may have suppressed non-significant findings like those reported in the present study.

With regard to the hypothesized mediators, maternal history of childhood maltreatment, but not lifetime or pregnancy IPV, was associated with greater self-reported and physiological emotion dysregulation. This finding adds to the large body of literature suggesting that the development of emotion regulation abilities is influenced by the quality of relationships with caregivers in early life (Dvir et al., 2014). It also adds to the growing literature examining the relationship between childhood maltreatment and physiological regulation. The finding that mother's childhood maltreatment histories positively predicted stressed HF-HRV when controlling for baseline measures is consistent with previous studies reporting that women's adverse childhood experiences were associated with less parasympathetic withdrawal during stressors such as infant cry (Oosterman et al., 2019). Additionally, although the specific indirect effect of childhood maltreatment on maternal sensitivity through challenged HF-HRV was not

significant, and therefore the mediation hypothesis was not supported, higher challenged HF-HRV was associated with less observed maternal sensitivity. This suggests that less responsive autonomic regulation in the face of external stressors, even ones that are not parenting related, is associated with less sensitive caregiving. This finding is consistent with the assumptions of the neurovisceral integration model, which suggests that increased flexibility of the central autonomic network, as indexed by increased vagally-mediated HRV reactivity to external demands, will be associated with greater cognitive and behavioral flexibility that supports the selection of contextually appropriate responses (Thayer & Lane, 2000). This finding is also consistent with a number of studies that report that less vagal withdrawal during infant distress is associated with less sensitive parenting behaviors (Ablow et al., 2013; Leerkes et al., 2016; Mills-Koonce et al., 2009; Skowron et al., 2013). Thus, less flexible autonomic responding may represent a physiological risk marker for parenting difficulties.

Interestingly, self-reported emotion regulation difficulties were not associated with observed parenting in the present study, which calls into question the utility of self-report measures of emotion regulation for identifying mothers who may benefit from parenting interventions. However, one important difference between the present study and others that have found associations between self-reported emotion dysregulation and maternal sensitivity was the type of parenting task used. Ours was a brief free-play interaction with an infant who is not yet walking or talking. Presumably, this type of caregiving interaction may be less demanding of mother's emotion regulation resources compared to other commonly used paradigms that elicit infant distress (e.g., Strange Situation or Still-Face reunion) or place demands on the child (e.g., joint puzzle or clean up tasks). For example, Leerkes et al. (2020) reported that mother's self-reported emotion dysregulation on the DERS was correlated with less observed maternal

sensitivity during mother-child interactions following procedures designed to elicit infant distress (arm restraint and novel toy approach). Indeed, some have argued that caregiving sensitivity during infant distress and non-distress contexts should be considered separate constructs altogether, as they seem to have different predictors and associated outcomes (Leerkes, Weaver, & O'Brien, 2012; McElwain & Booth-Laforce, 2006). Therefore, it is important to interpret the findings in the context of the specific methods employed in the present study, and the results should not be assumed to generalize to other parenting contexts. An important future direction will be to test the current hypotheses using a more emotionally demanding parenting task.

The third hypothesis examined the role of maternal psychopathology as a potential enhancer of the effects of interpersonal trauma on maternal sensitivity through emotion regulation. The expectation that clinical levels of depression and PTSD symptoms would strengthen the indirect effects of interpersonal trauma on sensitivity through self-reported emotion regulation was not borne out in the data. Although current depression and PTSD were both associated with greater self-reported emotion regulation difficulties, neither PTSD nor depression interacted with interpersonal trauma to predict self-reported emotion dysregulation, nor did they significantly moderate the indirect effects of childhood maltreatment or IPV on maternal sensitivity through self-reported emotion regulation difficulties.

The possibility that clinical levels of psychopathology would moderate the direct effects of interpersonal trauma exposure on maternal sensitivity was also explored. Maternal depression did not moderate the effects of any type of interpersonal trauma exposure on caregiving sensitivity. However, clinical levels of PTSD symptoms did interact with pregnancy IPV to predict maternal sensitivity. The direction of this effect was somewhat counterintuitive, such that clinical levels of PTSD symptoms seemed to confer protective effects on caregiving sensitivity,

but this effect was significant only at very high levels of pregnancy IPV. One way to interpret this finding is that women who are exposed to severe and psychologically impactful IPV may attempt to compensate for the negative impact of IPV on their children by being highly attentive in their parenting. Indeed, there is evidence to suggest that sensitive parenting can buffer against the negative effects of maternal PTSD (and presumably, in the case of IPV, the trauma that caused it) on young children's mental health outcomes (Greene, McCarthy, Estabrook, Wakschlag, & Briggs-Gowa, 2020). The fact that this pattern was only seen for IPV that occurred during pregnancy and not for preconception IPV suggests that this type of compensation is specific to recent or ongoing violence. Mothers' efforts to compensate for ongoing IPV in their parenting has previously been documented in qualitative studies (e.g., Scrafford, Miller-Graff, Umunyana, Schwartz, & Howell, 2022), as well as in quantitative work with IPV-exposed samples. For example, Levendosky et al. (2003) found that more severe IPV exposure was positively associated with women's reports of parenting effectiveness with preschool-aged children (although in this study maternal psychopathology had detrimental effects on parenting). Similarly, Greeson et al. (2014) found that among some mothers, more severe IPV was associated with increased use of authoritative parenting practices. That this pattern was seen only for clinical levels of PTSD symptoms and not for depressive symptoms in the present study may indicate that increased parenting responsiveness in the context of IPV is facilitated by the anxious and hypervigilant qualities of posttraumatic psychopathology, rather than by negative mood-related symptoms, which may be more likely to result in increased detachment during parenting.

The hypothesis that balanced maternal representations would buffer against the negative impact of interpersonal trauma on emotion regulation and parenting sensitivity was not

supported. Maternal representations did not interact with any type of trauma exposure to predict self-reported emotion regulation, nor were any of the indirect effects of interpersonal trauma on maternal sensitivity through emotion regulation moderated by having balanced representations. There was however a direct effect of maternal representations on maternal sensitivity, such that having a balanced representation of the relationship with the child was associated with greater maternal sensitivity during the free play interaction. This finding is consistent with previous studies that report associations between balanced maternal representations and more sensitive parenting in infancy and early childhood (Dayton et al., 2010; Sokolowski et al., 2007; Zajac, Raby, & Dozier, 2019). Despite the theorized relationship between internal working models and emotion regulation abilities, the two constructs were not associated in the present sample, and only internal working models had predictive power in relation to observed parenting behavior (Mikulincer & Shaver, 2019). This could indicate that affect regulation in attachment-priming contexts, such as during the WMCI and during parenting, are distinct from general emotion regulation abilities, such as those measured by the DERS. Alternatively, as both the WMCI and the free play were rated by independent coders, while the DERS was self-report, it could indicate that women in the present sample lacked the insight needed to reliably report on their own emotion regulation abilities. If this were the case, it could also help to explain why objective physiological measures of emotion regulation were more predictive of parenting behavior compared to the self-report measure.

Finally, in all of the models tested, cumulative demographic risk (comprised of low income, high school or less education, single parenthood, and racial/ethnic minority status) was a significant predictor of less sensitive caregiving, over and above the effects of maternal trauma exposure. This is consistent with other studies that find that demographic risk factors are strongly

associated with maternal parenting sensitivity (Leerkes et al., 2012; Popp, Spinrad, & Smith, 2008). It is important to note that the effects of demographic risk on sensitivity did not operate through self-reported or physiological measures of emotion regulation, which suggests that the obstacles that demographic risk factors pose to sensitive caregiving are not likely to be overcome with psychological interventions alone. This finding highlights the importance of taking ecological factors into consideration when researching mechanisms involved in intergenerational transmission of risk.

Limitations

The results of the present study should be considered in the context of several limitations. One of the major limitations of the present study was that observed parenting behavior was only measured during a relatively low-demand free play context. Theoretically, one might expect the effects of maternal emotion regulation on parenting behavior to be more apparent during affectively charged parenting scenarios, for example during infant distress. Additionally, as maternal sensitivity to infant distress is more predictive of children's self-regulation abilities compared to maternal sensitivity in non-distress contexts, maternal parenting behavior during infant distress may represent a more potent measure of parenting risk (Leerkes, Nayena Blankson, & O'Brien, 2009). Future studies should strive to include observations of parenting in multiple emotional contexts.

Additionally, while our stress task was more standardized than many of the parenting stressors commonly used in the literature that must also contend with differences in infant's reactivity, it also had some drawbacks. First, women's physiological reactions to the speech and mental arithmetic tasks may have been influenced by their educational attainment or comfort with public speaking. This risk was mitigated by controlling for cumulative demographic risk in

the analyses, and cumulative demographic risk was not found to be correlated with challenged HF-HRV in the present sample. Second, because separation from the infant preceded the administration of the stress task, we were not able to fully distinguish physiological reactivity in response to the separation from physiological reactivity in response to the TSST. The convergence between our findings regarding the association between challenged HF-HRV and maternal sensitivity and those that used infant cry stimuli as the stressor suggest that physiological responses to general and parenting-specific stressors may have similar associations with parenting. However, this conclusion should be further clarified by studies that are able to more cleanly distinguish between attachment related and non-attachment related stressors.

Another limitation was that because ECG data was only available for a small subsample of participants, we were unable to test the moderated mediation hypotheses in the HF-HRV models. Lastly, the current research focused solely on maternal caregivers' experiences of trauma and parenting. The larger longitudinal study that the data were drawn from did not include assessments of fathers, and it is not clear the fathers would show the same pattern of results (Lunkenheimer, Brown, & Fuchs, 2021). This is an important direction for future research.

The present study also had several notable strengths. First, the sample was racially diverse, increasing the generalizability of the findings, and was over-sampled for IPV exposure, making it uniquely suited to answer questions about the effects of interpersonal trauma on early parenting. Additionally, by including measures of childhood maltreatment, lifetime-, and pregnancy IPV exposure, we were able to test whether interpersonal trauma exposure during different developmental periods had differential effects on emotion regulation and caregiving. Another important strength was that multiple methods were used to measure emotion regulation,

which allowed the correspondence between self-report and physiological measures to be examined, and their unique relationships to predictor and outcome variables to be compared. Including an observed measure of caregiving sensitivity was also a methodological strength, as self-report measures of parenting are subject to social desirability bias, the effects of which may be especially pronounced among IPV-exposed mothers (Kobayashi et al., 2021).

Clinical Implications

The findings of the present study have implications for clinical practice. Calls have been made in the literature for parenting interventions to explicitly target parents' emotion regulation, as emotion regulation is considered foundational for effective parenting (Maliken & Katz, 2013; Zalewski et al., 2018). The findings from the present study provide further support for the utility of such approaches, especially ones that may be effective at increasing parents' autonomic regulation and flexibility. For example, Dialectical Behavioral Therapy (DBT; Linehan, 1993), a modality that emphasizes mindfulness and emotion regulation skills training, has been adapted for used with perinatal populations and components have been incorporated into parenting interventions (Zalewski et al., 2020). A pilot study of one such treatment for IPV-exposed mothers that integrates DBT emotion regulation skills training with an emotion coaching parenting intervention found that mothers in the treatment group demonstrated improvements in emotion regulation, as measured by increased baseline RSA, as well as increased emotional awareness and emotion coaching, increased use of validation and decreased use of scolding, and increased perceptions of parenting self-efficacy compared to those in the waitlist control group (Katz et al., 2020). While several studies have reported positive effects of parenting interventions on children's autonomic development, Katz et al.'s research is among the first to report changes in mothers' autonomic functioning as a result of participation in treatment. Whether

improvements in mothers' autonomic regulation are causally related to changes in parenting sensitivity is an important area for future research.

In addition to emotion regulation skills training, interventions that target autonomic regulation directly, such as HRV biofeedback, are potentially fruitful avenues for exploration. HRV biofeedback uses paced breathing practice combined with real-time monitoring of HRV to improve parasympathetic regulation. The few studies of HRV biofeedback in peripartum populations suggest that biofeedback is associated with reductions in perceived stress and improvements in anxiety symptoms among pregnant and postpartum women (Beckham, Greene, & Meltzer-Brody, 2013; Herbell & Zauszniewski, 2019; Kudo, Shinohara, & Kodama, 2014; Siepmann et al., 2014; van der Zwan, Huizink, Lehrer, Koot, & de Vente, 2019). Despite recommendations in the literature, the effects of HRV biofeedback on parenting sensitivity have yet to be investigated (Reijman et al., 2016).

Mindfulness-based interventions might also theoretically be expected to positively impact parents' autonomic regulation. For example, among pregnant women trait mindfulness was associated with less pre- and postpartum emotional distress and higher resting HF-HRV (Braeken et al., 2017). However, evidence that mindfulness or meditation-based interventions are effective at producing lasting changes in autonomic functioning is currently lacking (L. Brown et al., 2021). Mindfulness interventions in the perinatal period have shown some promise for reducing stress and improving psychological well-being, but adequately powered randomized controlled trials are needed to draw firm conclusions (Lever Taylor, Cavanagh, & Strauss, 2016). A review of the effects of mindfulness-based interventions on parenting outcomes suggests that these interventions may be helpful in reducing parenting stress and improving children's

psychological functioning, although the authors caution that the extant literature is subject to methodological limitations (Burgdorf, Szabó, & Abbott, 2019).

Emerging evidence for intergenerational effects as a result of mindfulness interventions is particularly exciting. For example, Noroña-Zhou et al. (2022) found that 6-month-old infants of women who participated in a group mindfulness-based intervention during pregnancy exhibited more adaptive physiological and behavioral stress regulation compared to infants whose mothers received treatment as usual. It is not yet clear whether these effects were driven by intrauterine factors or postnatal parenting.

In summary, incorporating pre- and post-measures of maternal autonomic functioning into parenting intervention research will be an important contribution to the literature, one that has the potential to shed new light on the psychophysiological underpinnings of sensitive caregiving and the mechanisms by which parenting interventions are effective. Future research is needed to clarify whether changes in maternal autonomic physiology correspond to sustained improvements in parenting sensitivity, and whether incorporating parent-focused emotion regulation skills and somatic practices such as biofeedback or meditation into traditional parenting interventions might provide added benefits.

The results of the present study also reaffirm the association between balanced maternal representations and caregiving sensitivity, independent of maternal emotion regulation abilities. Maternal representations, though influenced by past and current experiences in close relationships, may be modifiable by targeted interventions. For example, participation in Mom Power, a trauma-informed attachment-based multifamily group intervention aimed at improving parent-child relationships, resulted in significant improvements in maternal representations as measured by changes in WMCI classification and reflective functioning (Rosenblum et al.,

2018). Although changes in WMCI classification as a result of interventions have yet to be causally linked to changes in caregiving sensitivity, a number of attachment-based parenting interventions positively influence caregiving sensitivity and infant attachment (Bakermans-Kranenburg, van IJzendoorn, & Juffer, 2003; Mountain, Cahill, & Thorpe, 2017). The present study provides further support for the utility of attachment-based parenting interventions in improving parenting sensitivity.

Finally, the fact that cumulative demographic risk was associated with less sensitive caregiving, over and above the effects of maternal trauma exposure and emotion regulation difficulties, highlights the need for systems-level efforts that prevent the intergenerational transmission of risk. For example, social policies that relieve financial stress for families with young children, promote racial equity, and reduce barriers to post-high school educational attainment may all be expected to support parent-child relationship quality at the population level. The effectiveness of poverty reduction interventions, such as unrestricted cash transfer programs for low-income mothers, at improving family functioning and child developmental outcomes are currently being investigated in randomized controlled trials (e.g., Troller-Renfree et al., 2022). Primary prevention strategies, such as brief universal postnatal nurse home-visiting programs, have already demonstrated success at increasing positive parenting behaviors, and reducing maternal mental health problems, infant emergency care visits, and suspected child maltreatment (Dodge et al., 2014; Goodman, Dodge, Bai, Murphy, & O'Donnell, 2021). More research is needed to understand the mechanisms through which cumulative demographic risk affects maternal sensitivity, but it is clear that addressing social determinants has an important role in promoting maternal and infant outcomes.

Conclusion

The present study examined maternal emotion regulation as a potential mechanism linking multiple forms of interpersonal trauma to early caregiving sensitivity. Overall, the findings suggest that histories of childhood maltreatment predict increased subjective and physiological emotion dysregulation among mothers in the postpartum period. In turn, impaired emotion regulation, as evidenced by higher HF-HRV during a standardized stress task, was associated with less sensitive caregiving. This finding lends support to the idea, explicated both in polyvagal theory and the neurovisceral integration model, that flexible and responsive parasympathetic adaptation to contextual demands supports adaptive behavioral responding, and extends it to the context of parenting. Additionally, maternal representations of the relationship with the infant and socioecological factors influenced early caregiving behavior, independent of maternal emotion regulation. Taken together the findings suggest that multiple mechanistic pathways subserve early parenting behavior, each of which may represent appropriate targets for intervention.

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APPENDIX A: TABLES

Table 1

Sample Demographics

Variable	Mean (SD)
1. Age	26.69 (4.36)
2. Monthly household income	$2,758.26 ($2,290.18)
	N (%)
3. Marital status	
Never married	227 (61.4%)
Married	128 (34.6%)
Separated	7 (1.9%)
Divorced	6 (1.6%)
Widowed	1 (0.3%)
4. Cohabitating with a partner	265 (71.6%)
5. Primiparous	120 (32.4%)
6. Education Level	
Less than high school	39 (10.5%)
High school diploma/GED	168 (45.4%)
Post-high school technical degree	41 (11.1%)
College degree (AA, BA, BS)	91 (24.6%)
Graduate degree	30 (8.1%)
7. Race	
White	169 (45.7%)
Black or African American	133 (35.9%)
Asian American or Pacific Islander	6 (1.6%)
American Indian or Alaska Native	1 (0.3%)
Multi-racial	35 (9.5%)
Other/unspecified	25 (6.8%)
8. Hispanic/Latina	29 (7.6%)

Table 2

Bivariate correlations and descriptive statistics for Aim 1

	1.	2.	3.	4.
1. DERS total	-			
2. Baseline HF-HRV	.009	-		
3. TSST HF-HRV	.019	.790**	-	
4. Baseline minus TSST HF-HRV	.026	.685**	.095	-
N	223	77	88	72
Mean	78.94	5.49	6.19	–0.74
Standard deviation	23.04	1.34	0.94	0.83

Note. DERS = Difficulties in Emotion Regulation Scale; HF-HRV = high-frequency heart rate variability; TSST = Trier Social Stress Test. All HF-HRV values are log transformed. *$p < 0.05$, **$p < 0.01$.

Table 3

Bivariate correlations and descriptive statistics for Aim 2

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Childhood maltreatment	-										
2. Lifetime IPV	.137*	-									
3. Pregnancy IPV	.271**	.333**	-								
4. Demographic risk	.174**	.046	.068	-							
5. DERS total	.174**	.104	.105	–.010	-						
6. Baseline HF-HRV	.208	.054	–058	.018	.009	-					
7. TSST HF-HRV	.260*	.156	–.036	.184	.019	.790**	-				
8. Sensitivity to non-distress	–.086	.010	–.008	–.313**	–.001	–.076	–.147	-			
9. Stimulation of development	–.119	.003	–.021	–.118	–.020	–.052	–.127	.433**	-		
10. Detachment (reversed)	–.140*	–.021	.003	–.165*	–.084	.011	–.147	.479**	.413**	-	
11. Positive regard	–.032	.126	.095	–.185**	.061	.068	–.087	.326**	.306**	.285**	-
N	357	329	211	367	223	77	88	219	219	219	219
Mean	48.81	19.12	12.58	2.53	78.94	5.49	6.19	2.89	3.36	3.98	3.50
Standard deviation	21.08	27.51	23.01	1.25	23.04	1.34	0.94	0.99	1.12	0.88	1.06

**Note*. IPV = Intimate partner violence; DERS = Difficulties in Emotion Regulation Scale. **p* < 0.05, ***p* < 0.01.

Table 4

Unstandardized model estimates for the effects of childhood maltreatment on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	**Estimates**	**SE**	***p*–values**
Effects of predictors on maternal sensitivity			
Childhood maltreatment→Maternal sensitivity	–0.003	0.003	0.297
DERS→Maternal sensitivity	0.000	0.003	0.886
Demographic risk→Maternal sensitivity	–0.181	0.052	0.001**
Effects of predictors on emotion regulation			
Childhood maltreatment→DERS	0.190	0.069	0.006**
Demographic Risk→DERS	–0.887	1.260	0.481
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.915	0.163	<0.001**
Detachment (reversed) loading	0.777	0.120	<0.001**
Positive regard loading	0.664	0.135	<0.001**
Maternal sensitivity factor residual variance	0.471	0.083	<0.001**
Covariance between predictor and covariate			
Childhood maltreatment with Demographic risk	4.570	1.330	0.001**

Note. DERS = Difficulties in Emotion Regulation Scale. *$p < 0.05$, **$p < 0.01$.

Table 5

Unstandardized model estimates for the effects of lifetime IPV on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	Estimates	SE	*p*–values
Effects of predictors on maternal sensitivity			
Lifetime IPV→Maternal sensitivity	0.001	0.002	0.570
DERS→Maternal sensitivity	–0.001	0.003	0.679
Demographic risk→Maternal sensitivity	–0.193	0.054	<0.001**
Effects of predictors on emotion regulation			
Lifetime IPV→DERS	0.096	0.068	0.159
Demographic Risk→DERS	–0.332	1.256	0.791
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.904	0.156	<0.001**
Detachment (reversed) loading	0.767	0.125	<0.001**
Positive regard loading	0.664	0.129	<0.001**
Maternal sensitivity factor residual variance	0.479	0.083	<0.001**
Covariance between predictor and covariate			
Lifetime IPV with Demographic risk	1.467	1.742	0.400

Note. IPV = Intimate partner violence; DERS = Difficulties in Emotion Regulation Scale. *$p < 0.05$, **$p < 0.01$.

Table 6

Unstandardized model estimates for the effects of pregnancy IPV on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	Estimates	SE	*p*–values
Effects of predictors on maternal sensitivity			
Pregnancy IPV→Maternal sensitivity	0.001	0.002	0.717
DERS→Maternal sensitivity	–0.001	0.003	0.715
Demographic risk→Maternal sensitivity	–0.193	0.054	<0.001**
Effects of predictors on emotion regulation			
Pregnancy IPV→DERS	0.107	0.105	0.308
Demographic risk→DERS	–0.396	1.258	0.753
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.905	0.157	<0.001**
Detachment (reversed) loading	0.768	0.125	<0.001**
Positive regard loading	0.662	0.129	<0.001**
Maternal sensitivity factor residual variance	0.480	0.083	<0.001**
Covariance between predictor and covariate			
Pregnancy IPV with Demographic risk	1.977	1.229	0.108

Note. IPV = Intimate partner violence; DERS = Difficulties in Emotion Regulation Scale. *$p < 0.05$, **$p < 0.01$.

Table 7

Unstandardized model estimates for the effects of childhood maltreatment on maternal sensitivity through high-frequency heart rate variability

Parameters	Estimates	SE	*p*–values
Effects of predictors on maternal sensitivity			
Childhood maltreatment→Maternal sensitivity	–0.001	0.003	0.752
Baseline HF-HRV→Maternal sensitivity	0.239	0.140	0.089
TSST HF-HRV→Maternal sensitivity	–0.393	0.183	0.032*
Demographic risk→Maternal sensitivity	–0.139	0.059	0.019*
Effects of predictors on HF-HRV			
Childhood maltreatment→Baseline HF-HRV	0.011	0.008	0.176
Childhood maltreatment→TSST HF-HRV	0.012	0.006	0.044*
Demographic risk→ Baseline HF-HRV	–0.045	0.110	0.686
Demographic risk→ TSST HF-HRV	0.064	0.071	0.368
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.945	0.171	<0.001**
Detachment (reversed) loading	0.800	0.124	<0.001**
Positive regard loading	0.692	0.143	<0.001**
Maternal sensitivity factor residual variance	0.404	0.099	<0.001**
Covariances			
Childhood maltreatment with Demographic risk	4.593	1.331	0.001**
Baseline HF-HRV with TSST HF-HRV	0.896	0.147	0.000**

Note. HF-HRV = High-frequency heart rate variability; TSST = Trier Social Stress Test. *p < 0.05, **p < 0.01.

Table 8

Unstandardized model estimates for the effects of lifetime IPV on maternal sensitivity through high-frequency heart rate variability

Parameters	Estimates	SE	*p*–values
Effects of predictors on maternal sensitivity			
Lifetime IPV→Maternal sensitivity	0.003	0.003	0.258
Baseline HF-HRV→Maternal sensitivity	0.239	0.154	0.121
TSST HF-HRV→Maternal sensitivity	–0.398	0.194	0.039*
Demographic risk→Maternal sensitivity	–0.145	0.063	0.021*
Effects of predictors on HF-HRV			
Lifetime IPV→Baseline HF-HRV	0.003	0.008	0.646
Lifetime IPV→TSST HF-HRV	0.007	0.005	0.189
Demographic risk→ Baseline HF-HRV	–0.005	0.122	0.966
Demographic risk→ TSST HF-HRV	0.104	0.078	0.186
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.931	0.160	<0.001**
Detachment (reversed) loading	0.787	0.129	<0.001**
Positive regard loading	0.689	0.135	<0.001**
Maternal sensitivity factor residual variance	0.415	0.097	<0.001**
Covariances			
Lifetime IPV with Demographic risk	1.615	1.744	0.354
Baseline HF-HRV with TSST HF-HRV	0.934	0.172	0.000**

Note. IPV = Intimate partner violence; HF-HRV = High-frequency heart rate variability; TSST = Trier Social Stress Test. *$p < 0.05$, **$p < 0.01$.

Table 9

Unstandardized model estimates for the effects of pregnancy IPV on maternal sensitivity through high-frequency heart rate variability

Parameters	Estimates	SE	*p*–values
Effects of predictors on maternal sensitivity			
Pregnancy IPV→Maternal sensitivity	0.001	0.003	0.757
Baseline HF-HRV→Maternal sensitivity	0.229	0.151	0.129
TSST HF-HRV→Maternal sensitivity	–0.382	0.185	0.039*
Demographic risk→Maternal sensitivity	–0.145	0.062	0.019*
Effects of predictors on HF-HRV			
Pregnancy IPV→Baseline HF-HRV	–0.004	0.007	0.617
Pregnancy IPV→TSST HF-HRV	–0.002	0.005	0.764
Demographic risk→ Baseline HF-HRV	–0.009	0.123	0.941
Demographic risk→ TSST HF-HRV	0.107	0.081	0.187
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.930	0.160	<0.001**
Detachment (reversed) lading	0.786	0.129	<0.001**
Positive regard loading	0.688	0.135	<0.001**
Maternal sensitivity factor residual variance	0.417	0.097	<0.001**
Covariances			
Pregnancy IPV with Demographic risk	1.978	1.235	0.109
Baseline HF-HRV with TSST HF-HRV	0.943	0.185	0.000**

Note. IPV = Intimate partner violence; HF-HRV = High-frequency heart rate variability; TSST = Trier Social Stress Test. *$p < 0.05$, **$p < 0.01$.

Table 10

Bivariate correlations and descriptive statistics for Aim 3

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Childhood maltreatment	-												
2. Lifetime IPV	.137*	-											
3. Pregnancy IPV	.271**	.333**	-										
4. Demographic risk	.174**	.046	.068	-									
5. DERS total	.174**	.104	.105	–.010	-								
6. Baseline HF-HRV	.208	.054	–058	.018	.009	-							
7. TSST HF-HRV	.260*	.156	–.036	.184	.019	.790**	-						
8. Sensitivity to non-distress	–.086	.010	–.008	–.313**	–.001	–.076	–.147	-					

Table 10 (cont'd)

9. Stimulation of development	–.119	.003	–.021	–.118	–.020	–.052	–.127	.433**	-				
10. Detachment (reversed)	–.140*	–.021	.003	–.165*	–.084	.011	–.147	.479**	.413**	-			
11. Positive regard	–.032	.126	.095	–.185**	.061	.068	–.087	.326**	.306**	.285**	-		
12. Depression	.183**	.146*	.083	.037	.492**	–.075	.011	–.012	–.075	.025	.006	-	
13. PTSD	.142*	.112	.140	.067	.261**	–.145	–.068	–.024	–.085	–.078	–.091	.425**	-
N	357	329	211	367	223	77	88	219	219	219	219	222	216
Mean	48.81	19.12	12.58	2.53	78.94	5.49	6.19	2.89	3.36	3.98	3.50	0.27	0.24
Standard deviation	21.08	27.51	23.01	1.25	23.04	1.34	0.94	0.99	1.12	0.88	1.06	0.45	0.43

**Note*. IPV = Intimate partner violence; DERS = Difficulties in Emotion Regulation Scale. **p* < 0.05, ***p* < 0.01.

Table 11

Unstandardized model estimates for the effects of childhood maltreatment, depression, and their interaction on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	**Estimates**	**SE**	***p*–values**
Effects of predictors on maternal sensitivity			
Childhood maltreatment→Maternal sensitivity	–0.004	0.003	0.215
Depression→Maternal sensitivity	0.000	0.164	1.000
Childhood maltreatment x depression→Maternal sensitivity	0.004	0.007	0.506
DERS→Maternal sensitivity	–0.001	0.003	0.871
Demographic risk→Maternal sensitivity	–0.178	0.053	0.001**
Effects of predictors on emotion regulation			
Childhood maltreatment→DERS	0.108	0.072	0.135
Depression→DERS	24.734	2.702	<0.001**
Childhood maltreatment x depression→ DERS	–0.028	0.116	0.808
Demographic Risk→DERS	–0.907	1.103	0.411
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.914	0.166	<0.001**
Detachment (reversed) loading	0.774	0.124	<0.001**
Positive regard loading	0.654	0.139	<0.001**
Maternal sensitivity factor residual variance	0.472	0.083	<0.001**
Covariances			
Childhood maltreatment with Demographic risk	4.574	1.329	0.001**
Childhood maltreatment with Depression	1.670	0.628	0.008**
Childhood maltreatment with Childhood Maltreatment x depression	142.013	31.068	<0.001**
Depression with Demographic risk	0.019	0.034	0.578
Depression with Childhood maltreatment x depression	1.388	0.564	0.014*

Table 11 (cont'd)

Demographic risk with Childhood maltreatment x depression	0.432	0.794	0.586

Note. DERS = Difficulties in Emotion Regulation Scale. *$p < 0.05$, **$p < 0.01$.

Table 12

Unstandardized model estimates for the effects of lifetime IPV, depression, and their interaction on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	**Estimates**	**SE**	***p*–values**
Effects of predictors on maternal sensitivity			
Lifetime IPV→Maternal sensitivity	0.001	0.003	0.720
Depression→Maternal sensitivity	–0.012	0.169	0.944
Lifetime IPV x depression→Maternal sensitivity	0.001	0.005	0.874
DERS→Maternal sensitivity	–0.001	0.003	0.759
Demographic risk→Maternal sensitivity	–0.192	0.054	<0.001**
Effects of predictors on emotion regulation			
Lifetime IPV →DERS	0.013	0.067	0.846
Depression→DERS	24.974	2.826	<0.001**
Lifetime IPV x depression→ DERS	0.058	0.114	0.612
Demographic Risk→DERS	–0.577	1.118	0.606
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.906	0.158	<0.001**
Detachment (reversed) loading	0.768	0.129	<0.001**
Positive regard loading	0.666	0.131	<0.001**
Maternal sensitivity factor residual variance	0.478	0.085	<0.001**
Covariances			
Lifetime IPV with Demographic risk	1.450	1.745	0.406
Lifetime IPV with Depression	1.862	1.011	0.065
Lifetime IPV with Lifetime IPV x depression	–242.112	62.126	<0.001**
Depression with Demographic risk	0.021	0.034	0.527
Depression with Lifetime IPV x depression	1.130	0.745	0.129
Demographic risk with Lifetime IPV x depression	–0.488	1.078	0.651

Table 12 (cont'd)

Note. IPV = Intimate Partner Violence; DERS = Difficulties in Emotion Regulation Scale. *p < 0.05, **p < 0.01.

Table 13

Unstandardized model estimates for the effects of pregnancy IPV, depression, and their interaction on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	**Estimates**	**SE**	***p*–values**
Effects of predictors on maternal sensitivity			
Pregnancy IPV→Maternal sensitivity	–0.001	0.003	0.782
Depression→Maternal sensitivity	–0.012	0.163	0.942
Pregnancy IPV x depression→Maternal sensitivity	0.007	0.007	0.294
DERS→Maternal sensitivity	–0.001	0.003	0.746
Demographic risk→Maternal sensitivity	–0.190	0.054	<0.001**
Effects of predictors on emotion regulation			
Pregnancy IPV →DERS	0.058	0.106	0.586
Depression→DERS	25.104	2.805	<0.001**
Pregnancy IPV x depression→ DERS	0.026	0.164	0.875
Demographic Risk→DERS	–0.640	1.106	0.563
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.903	0.160	<0.001**
Detachment (reversed) loading	0.772	0.129	<0.001**
Positive regard loading	0.663	0.129	<0.001**
Maternal sensitivity factor residual variance	0.474	0.083	<0.001**
Covariances			
Pregnancy IPV with Demographic risk	1.975	1.229	0.108
Pregnancy IPV with Depression	0.882	0.806	0.274
Pregnancy IPV with Pregnancy IPV x depression	137.093	69.127	0.047*
Depression with Demographic risk	0.019	0.034	0.565
Depression with Pregnancy IPV x depression	0.614	0.664	0.355
Demographic risk with Pregnancy IPV x depression	–0.069	0.769	0.929

Table 13 (cont'd)

Note. IPV = Intimate Partner Violence; DERS = Difficulties in Emotion Regulation Scale. *p < 0.05, **p < 0.01.

Table 14

Unstandardized model estimates for the effects of childhood maltreatment, PTSD, and their interaction on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	**Estimates**	**SE**	***p*–values**
Effects of predictors on maternal sensitivity			
Childhood maltreatment→Maternal sensitivity	–0.003	0.004	0.504
PTSD→Maternal sensitivity	–0.091	0.145	0.528
Childhood maltreatment x PTSD→Maternal sensitivity	–0.001	0.006	0.927
DERS→Maternal sensitivity	0.000	0.003	0.990
Demographic risk→Maternal sensitivity	–0.178	0.054	0.001**
Effects of predictors on emotion regulation			
Childhood maltreatment→DERS	0.181	0.073	0.013*
PTSD→DERS	13.599	3.685	<0.001**
Childhood maltreatment x PTSD→ DERS	–0.087	0.152	0.567
Demographic Risk→DERS	–1.108	1.226	0.366
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.922	0.166	<0.001**
Detachment (reversed) loading	0.780	0.126	<0.001**
Positive regard loading	0.670	0.138	<0.001**
Maternal sensitivity factor residual variance	0.466	0.084	<0.001**
Covariances			
Childhood maltreatment with Demographic risk	4.567	1.328	0.001**
Childhood maltreatment with PTSD	1.263	0.659	0.056
Childhood maltreatment with Childhood Maltreatment x PTSD	–135.141	29.569	<0.001**
PTSD with Demographic risk	0.036	0.034	0.283
PTSD with Childhood maltreatment x PTSD	1.140	0.623	0.067

Table 14 (cont'd)

Demographic risk with Childhood maltreatment x PTSD	0.610	0.943	0.518

Note. PTSD = Posttraumatic Stress Disorder; DERS = Difficulties in Emotion Regulation Scale.

*$p < 0.05$, **$p < 0.01$.

Table 15

Unstandardized model estimates for the effects of lifetime IPV, PTSD, and their interaction on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	Estimates	SE	*p*–values
Effects of predictors on maternal sensitivity			
Lifetime IPV→Maternal sensitivity	0.001	0.003	0.672
PTSD→Maternal sensitivity	–0.123	0.142	0.384
Lifetime IPV x PTSD →Maternal sensitivity	0.001	0.005	0.795
DERS→Maternal sensitivity	–0.001	0.003	0.813
Demographic risk→Maternal sensitivity	–0.187	0.055	<0.001**
Effects of predictors on emotion regulation			
Lifetime IPV →DERS	–0.002	0.073	0.976
PTSD →DERS	13.024	3.656	<0.001**
Lifetime IPV x PTSD → DERS	0.246	0.128	0.055
Demographic Risk→DERS	–0.546	1.222	0.655
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.914	0.161	<0.001**
Detachment (reversed) loading	0.774	0.128	<0.001**
Positive regard loading	0.671	0.133	<0.001**
Maternal sensitivity factor residual variance	0.471	0.085	<0.001**
Covariances			
Lifetime IPV with Demographic risk	1.467	1.747	0.401
Lifetime IPV with PTSD	1.451	0.923	0.116
Lifetime IPV with Lifetime IPV x PTSD	217.406	60.545	<0.001**
PTSD with Demographic risk	0.039	0.034	0.258
PTSD with Lifetime IPV x PTSD	0.840	0.721	0.244
Demographic risk with Lifetime IPV x PTSD	–0.077	1.071	0.943

Note. PTSD = Posttraumatic Stress Disorder; IPV = Intimate Partner Violence; DERS = Difficulties in Emotion Regulation Scale. *$p < 0.05$, **$p < 0.01$.

Table 16

Unstandardized model estimates for the effects of pregnancy IPV, PTSD, and their interaction on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	**Estimates**	**SE**	***p*–values**
Effects of predictors on maternal sensitivity			
Pregnancy IPV→Maternal sensitivity	–0.002	0.004	0.512
PTSD→Maternal sensitivity	–0.157	0.137	0.253
Pregnancy IPV x PTSD →Maternal sensitivity	0.013	0.008	0.088
DERS→Maternal sensitivity	–0.001	0.003	0.729
Demographic risk→Maternal sensitivity	–0.187	0.055	<0.001**
Effects of predictors on emotion regulation			
Pregnancy IPV →DERS	0.018	0.126	0.886
PTSD →DERS	13.098	3.779	0.001**
Pregnancy IPV x PTSD → DERS	0.168	0.241	0.486
Demographic Risk→DERS	–0.617	1.232	0.616
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.901	0.164	<0.001**
Detachment (reversed) loading	0.770	0.127	<0.001**
Positive regard loading	0.660	0.130	<0.001**
Maternal sensitivity factor residual variance	0.461	0.083	<0.001**
Covariances			
Pregnancy IPV with Demographic risk	1.963	1.232	0.111
Pregnancy IPV with PTSD	1.438	0.933	0.123
Pregnancy IPV with Lifetime IPV x PTSD	154.956	78.547	0.049*
PTSD with Demographic risk	0.037	0.034	0.286
PTSD with Pregnancy IPV x PTSD	0.944	0.752	0.209
Demographic risk with Pregnancy IPV x PTSD	0.375	0.695	0.590

Note. PTSD = Posttraumatic Stress Disorder; IPV = Intimate Partner Violence; DERS =

Table 16 (cont'd)

Difficulties in Emotion Regulation Scale. $*p < 0.05$, $**p < 0.01$.

Table 17

Bivariate correlations and descriptive statistics for Aim 4

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Childhood maltreatment	-											
2. Lifetime IPV	.137*	-										
3. Pregnancy IPV	.271**	.333**	-									
4. Demographic risk	.174**	.046	.068	-								
5. DERS total	.174**	.104	.105	–.010	-							
6. Baseline HF-HRV	.208	.054	–058	.018	.009	-						
7. TSST HF-HRV	.260*	.156	–.036	.184	.019	.790**	-					
8. Sensitivity to non-distress	–.086	.010	–.008	–.313**	–.001	–.076	–.147	-				
9. Stimulation of development	–.119	.003	–.021	–.118	–.020	–.052	–.127	.433**	-			
10. Detachment (reversed)	–.140*	–.021	.003	–.165*	–.084	.011	–.147	.479**	.413**	-		
11. Positive regard	–.032	.126	.095	–.185**	.061	.068	–.087	.326**	.306**	.285**	-	
12. Balanced representation	–.090	–.008	–.151*	–.203**	–.046	.020	.022	.162*	.169*	.230**	.180*	-

Table 17 (cont'd)

N	357	329	211	367	223	77	88	219	219	219	219	254
Mean	48.81	19.12	12.58	2.53	78.94	5.49	6.19	2.89	3.36	3.98	3.50	0.39
Standard deviation	21.08	27.51	23.01	1.25	23.04	1.34	0.94	0.99	1.12	0.88	1.06	0.49

**Note*. IPV = Intimate partner violence; DERS = Difficulties in Emotion Regulation Scale. *$p < 0.05$, **$p < 0.01$.

Table 18

Unstandardized model estimates for the effects of childhood maltreatment, maternal representations, and their interaction on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	**Estimates**	**SE**	***p*–values**
Effects of predictors on maternal sensitivity			
Childhood maltreatment→Maternal sensitivity	–0.001	0.004	0.792
Balanced representation →Maternal sensitivity	0.305	0.120	0.011*
Childhood maltreatment x Balanced representation →Maternal sensitivity	–0.004	0.006	0.510
DERS→Maternal sensitivity	0.000	0.003	0.901
Demographic risk→Maternal sensitivity	–0.149	0.051	0.004**
Effects of predictors on emotion regulation			
Childhood maltreatment→DERS	0.233	0.081	0.004**
Balanced representation →DERS	–1.947	3.501	0.578
Childhood maltreatment x Balanced representation → DERS	–0.131	0.154	0.395
Demographic Risk→DERS	–1.108	1.226	0.366
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.937	0.164	<0.001**
Detachment (reversed) loading	0.796	0.125	<0.001**
Positive regard loading	0.679	0.141	<0.001**
Maternal sensitivity factor residual variance	0.435	0.083	<0.001**
Covariances			
Childhood maltreatment with Demographic risk	4.590	1.332	0.001**
Childhood maltreatment with Balanced representation	–0.872	0.632	0.167

Table 18 (cont'd)

Childhood maltreatment with Childhood Maltreatment x Balanced representation	163.376	22.995	<0.001**
Balanced representation with Demographic risk	–0.120	0.040	0.002**
Balanced representation with Childhood maltreatment x Balanced representation	–0.625	0.483	0.196
Demographic risk with Childhood maltreatment x Balanced representation	2.775	1.116	0.013*

Note. DERS = Difficulties in Emotion Regulation Scale. *$p < 0.05$, **$p < 0.01$.

Table 19

Unstandardized model estimates for the effects of lifetime IPV, maternal representations, and their interaction on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	**Estimates**	**SE**	***p*–values**
Effects of predictors on maternal sensitivity			
Lifetime IPV→Maternal sensitivity	–0.001	0.003	0.801
Balanced representation →Maternal sensitivity	0.324	0.116	0.005**
Lifetime IPV x Balanced representation →Maternal sensitivity	0.005	0.004	0.205
DERS→Maternal sensitivity	–0.001	0.003	0.781
Demographic risk→Maternal sensitivity	–0.164	0.052	0.002**
Effects of predictors on emotion regulation			
Lifetime IPV →DERS	0.141	0.092	0.124
Balanced representation →DERS	–2.653	3.486	0.447
Lifetime IPV x Balanced representation → DERS	–0.105	0.151	0.488
Demographic Risk→DERS	–0.467	1.337	0.727
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.923	0.151	<0.001**
Detachment (reversed) loading	0.794	0.125	<0.001**
Positive regard loading	0.683	0.131	<0.001**
Maternal sensitivity factor residual variance	0.435	0.078	<0.001**
Covariances			
Lifetime IPV with Demographic risk	1.465	1.740	0.400
Lifetime IPV with Balanced representation	– 0.007	0.907	0.994
Lifetime IPV with Lifetime IPV x Balanced representation	299.554	68.217	<0.001**
Balanced representation with Demographic risk	–0.119	0.037	0.001**

Table 19 (cont'd)

Balanced representation with Lifetime IPV x Balanced representation	–0.040	0.655	0.952
Demographic risk with Lifetime IPV x Balanced representation	0.997	1.287	0.439

Note. IPV = Intimate partner violence; DERS = Difficulties in Emotion Regulation Scale. *p < 0.05, **p < 0.01.

Table 20

Unstandardized model estimates for the effects of pregnancy IPV, maternal representations, and their interaction on maternal sensitivity through self-reported emotion regulation difficulties

Parameters	**Estimates**	**SE**	***p*–values**
Effects of predictors on maternal sensitivity			
Pregnancy IPV→Maternal sensitivity	0.003	0.003	0.373
Balanced representation →Maternal sensitivity	0.321	0.121	0.008**
Pregnancy IPV x Balanced representation →Maternal sensitivity	–0.004	0.008	0.596
DERS→Maternal sensitivity	–0.001	0.003	0.728
Demographic risk→Maternal sensitivity	–0.158	0.051	0.002**
Effects of predictors on emotion regulation			
Pregnancy IPV →DERS	0.139	0.124	0.264
Balanced representation →DERS	–2.487	4.198	0.554
Pregnancy IPV x Balanced representation → DERS	–0.221	0.354	0.532
Demographic Risk→DERS	–0.512	1.334	0.701
Latent factor parameter estimates			
Sensitivity to non-distress loading	1.000	0.000	--
Stimulation of development loading	0.938	0.154	<0.001**
Detachment (reversed) loading	0.805	0.126	<0.001**
Positive regard loading	0.691	0.131	<0.001**
Maternal sensitivity factor residual variance	0.430	0.080	<0.001**
Covariances			
Pregnancy IPV with Demographic risk	2.169	1.257	0.084
Pregnancy IPV with Balanced representation	– 1.921	0.969	0.047*
Pregnancy IPV with Pregnancy IPV x Balanced representation	110.495	53.455	0.039*
Balanced representation with Demographic risk	–0.120	0.037	0.001**

Table 20 (cont'd)

Balanced representation with Pregnancy IPV x Balanced representation	–1.197	0.417	0.004**
Demographic risk with Pregnancy IPV x Balanced representation	1.063	0.534	0.046*

Note. IPV = Intimate partner violence; DERS = Difficulties in Emotion Regulation Scale. *p < 0.05, **p < 0.01.

APPENIDX B: FIGURES

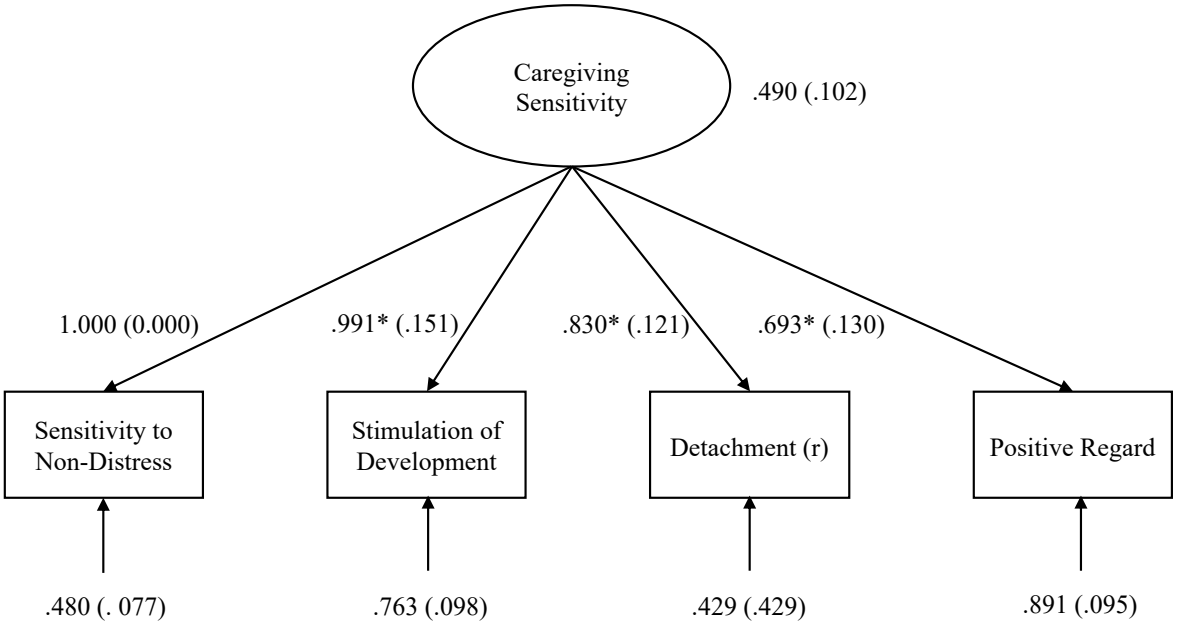

Figure 1. Maternal caregiving sensitivity factor model. Factor was identified by fixing the first factor loading to 1. Diagram presents unstandardized estimates and standard errors. Model fit: $\chi^2(2) = 4.81, p = 0.786$, CFI = 1.00, TLI = 1.03, RMSEA < .001. *$p < .05$.

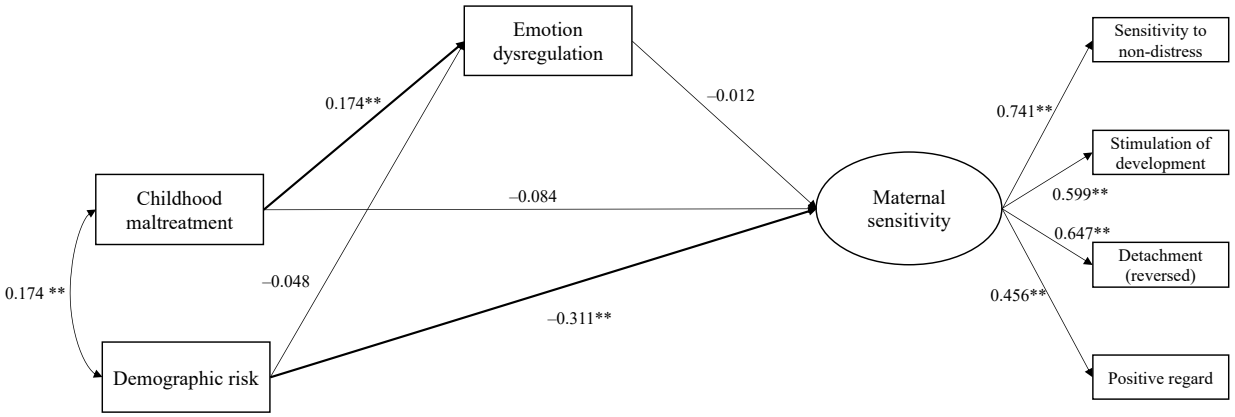

Figure 2. Path diagram for the effect of childhood maltreatment on maternal sensitivity through self-reported emotion regulation difficulties. Diagram presents standardized path estimates. Factor was identified by fixing the first factor loading to 1. Model fit: $\chi^2(11) = 12.57$, $p = 0.32$, CFI = .99, TLI = .98, RMSEA = .02. Means, variances, and residual variances are omitted for ease of interpretation. Bolded lines represent significant structural paths. *$p < .05$, **$p < .01$.

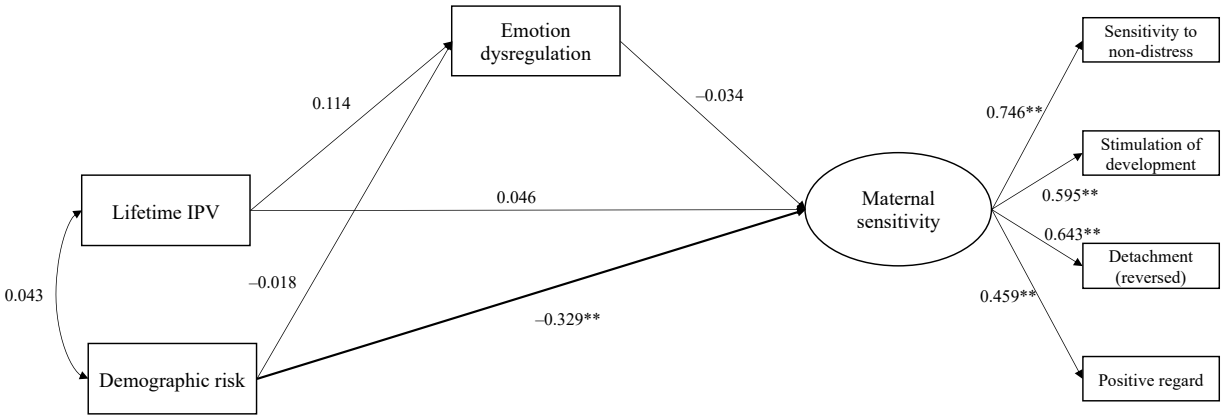

Figure 3. Path diagram for the effect of lifetime IPV exposure on maternal sensitivity through self-reported emotion regulation difficulties. Diagram presents standardized path estimates. Factor was identified by fixing the first factor loading to 1. Model fit: $\chi^2(11) = 14.16$, $p = 0.22$, CFI = .98, TLI = .97, RMSEA = .03. Means, variances, and residual variances are omitted for ease of interpretation. Bolded lines represent significant structural paths. *$p < .05$, **$p < .01$.

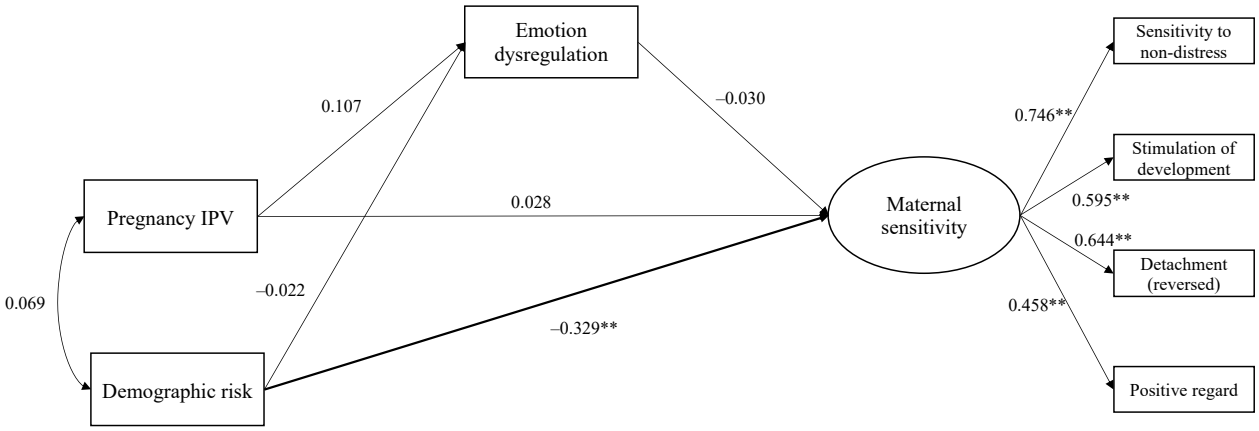

Figure 4. Path diagram for the effect of pregnancy IPV exposure on maternal sensitivity through self-reported emotion regulation difficulties. Diagram presents standardized path estimates. Factor was identified by fixing the first factor loading to 1. Model fit: $\chi^2(11) = 12.41, p = 0.33$, CFI = .99, TLI = .99, RMSEA = .02. Means, variances, and residual variances are omitted for ease of interpretation. Bolded lines represent significant structural paths. *$p < .05$, **$p < .01$.

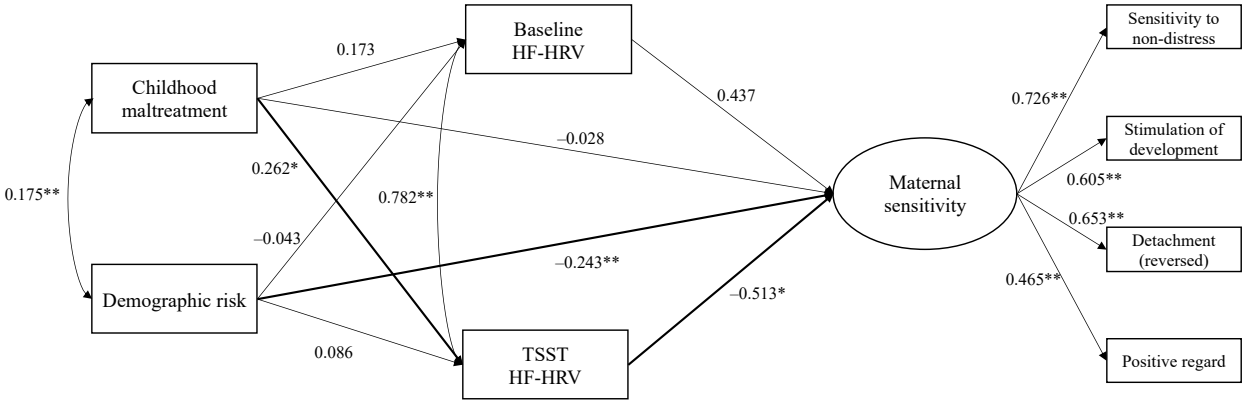

Figure 5. Path diagram for the effect of childhood maltreatment on maternal sensitivity through baseline and challenged HF-HRV. Diagram presents standardized path estimates. Factor was identified by fixing the first factor loading to 1. Model fit: $\chi^2(14) = 14.34$, $p = 0.42$, CFI = 1.00, TLI = 1.00, RMSEA = .01. Means, variances, and residual variances are omitted for ease of interpretation. Bolded lines represent significant structural paths. *$p < .05$, **$p < .01$.

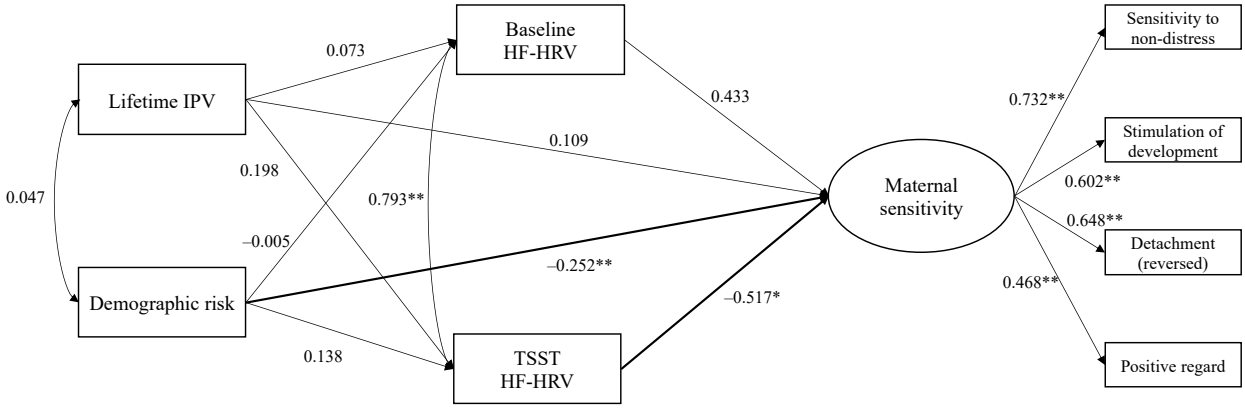

Figure 6. Path diagram for the effect of lifetime IPV on maternal sensitivity through baseline and challenged HF-HRV. Diagram presents standardized path estimates. Factor was identified by fixing the first factor loading to 1. Model fit: $\chi^2(14) = 14.87$, $p = 0.38$, CFI = 1.00, TLI = 0.99, RMSEA = .01. Means, variances, and residual variances are omitted for ease of interpretation. Bolded lines represent significant structural paths. *$p < .05$, **$p < .01$.

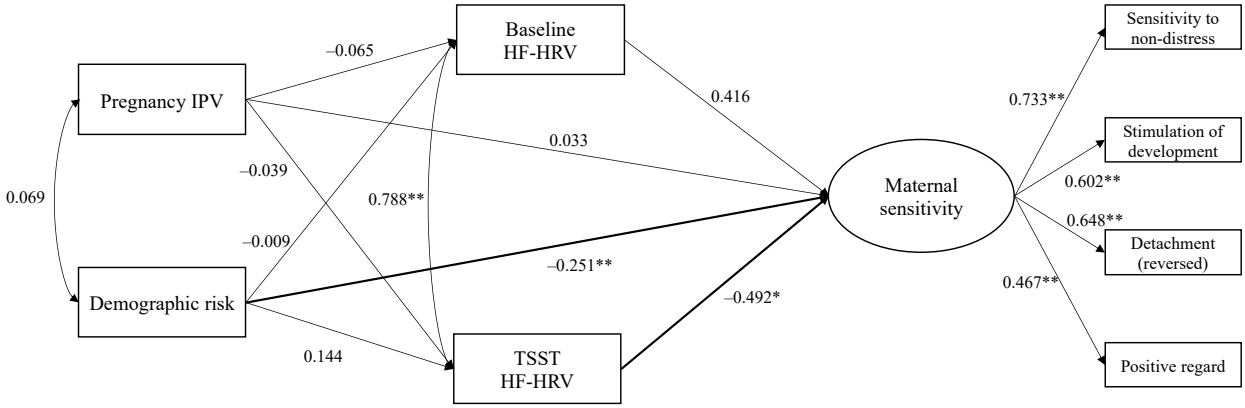

Figure 7. Path diagram for the effect of pregnancy IPV on maternal sensitivity through baseline and challenged HF-HRV. Diagram presents standardized path estimates. Maternal sensitivity factor was identified by fixing the first factor loading to 1. Model fit: $\chi^2(14) = 13.24$, $p = 0.51$, CFI = 1.00, TLI = 1.00, RMSEA = <.01. Means, variances, and residual variances are omitted for ease of interpretation. Bolded lines represent significant structural paths. *$p < .05$, **$p < .01$.

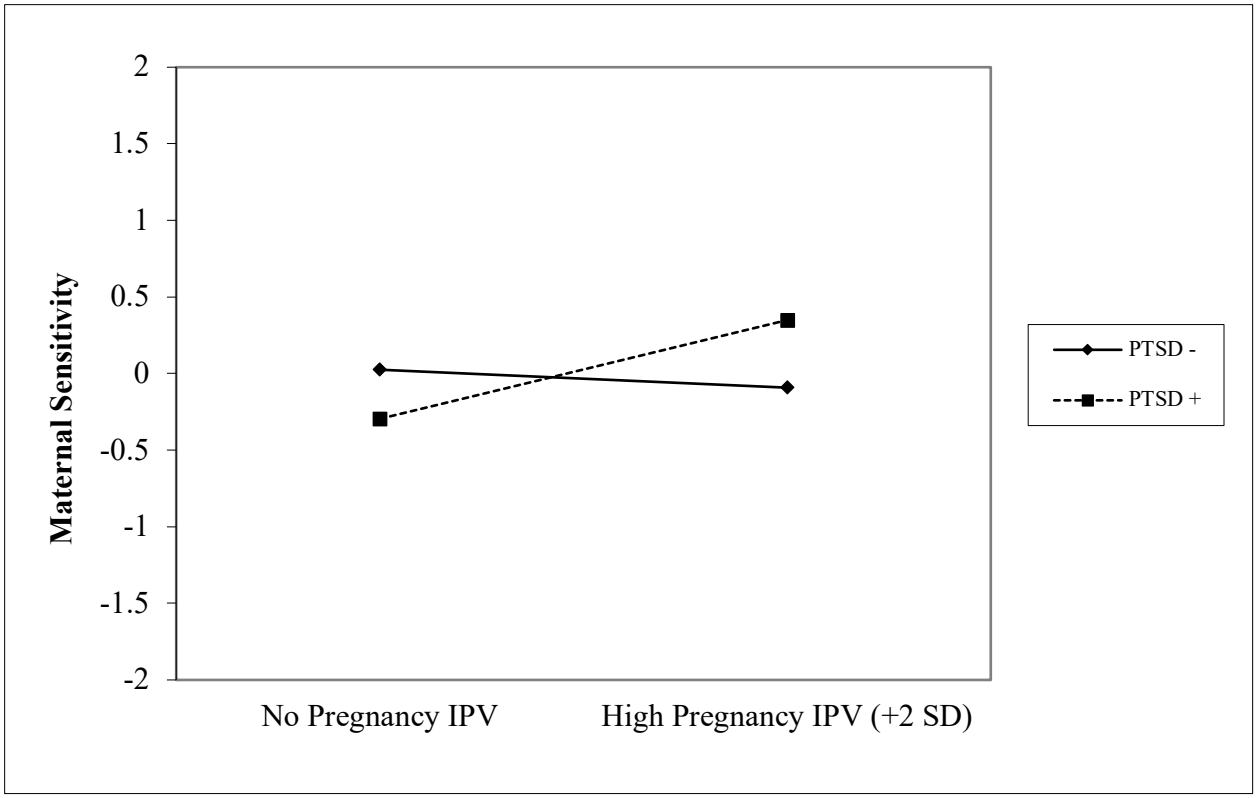

Figure 8. Graph of the effect of pregnancy IPV on maternal sensitivity with (PTSD +) and without (PTSD –) clinical levels of PTSD symptoms. Note: IPV = intimate partner violence; PTSD = Posttraumatic stress disorder.

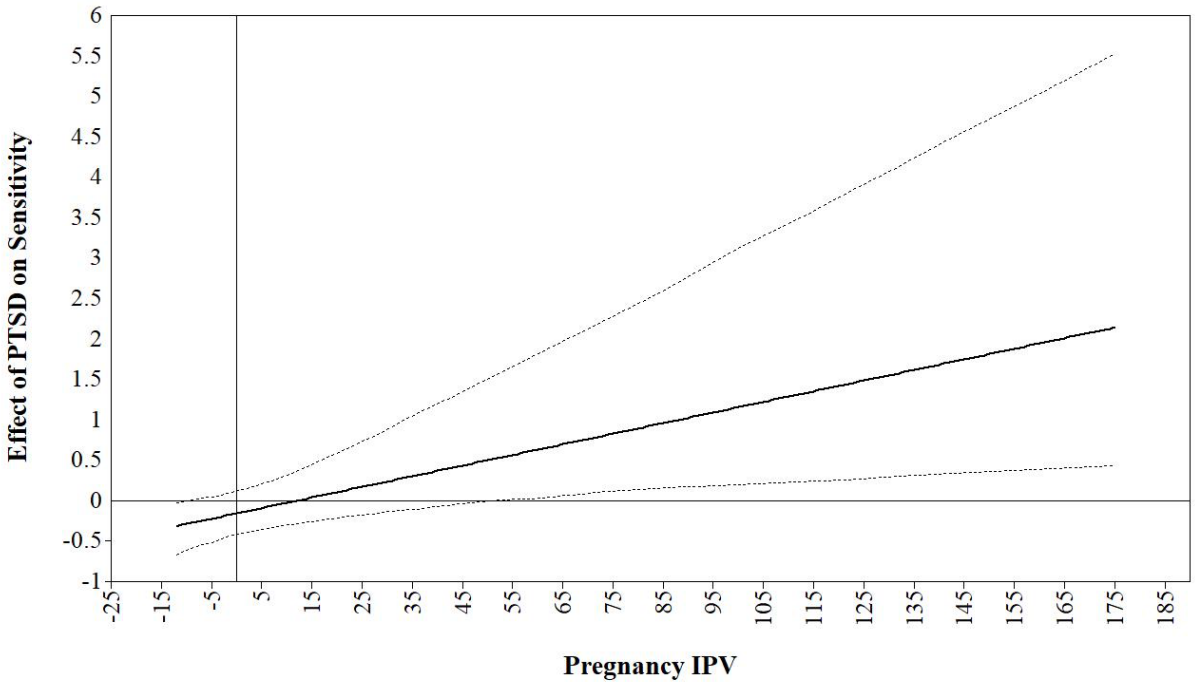

Figure 9. Johnson-Neyman plot of the effect of clinical levels of PTSD symptoms on maternal sensitivity by pregnancy IPV. Pregnancy IPV is grand mean centered. Dashed lines represent 95% confidence bands. Effect is considered significant for regions where confidence bands do not include zero. Note: IPV = intimate partner violence; PTSD = Posttraumatic stress disorder.