

THE RELATIONSHIP BETWEEN SYMPATHETIC NERVOUS SYSTEM ATTUNEMENT  
AND INTIMATE PARTNER VIOLENCE IN MOTHER-INFANT DYADS

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## **ABSTRACT**

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When mothers and infants interact socially, they also interact on a physiological level, which can lead them to have attuned physiological reactions. However, studies have shown that chronic stressors may negatively affect attunement. One such stressor is intimate partner violence (IPV). In physiological attunement, the negative effect of a stressor like IPV may be seen in the dysregulated or exaggerated sympathetic nervous system (SNS) responses of mothers and their infants, which may make it more difficult for them to attune to one another. This study hypothesized that IPV would negatively affect SNS attunement such that dyads who experienced IPV both pre- and postnatally (chronic IPV) would be less attuned than dyads who did not. In addition, it was hypothesized that maternal warmth would have a positive relationship with attunement in dyads who experienced chronic IPV. IPV, maternal warmth and SNS reactivity (via salivary alpha amylase: sAA) were assessed during a laboratory stress task in 182 mother-infant dyads. IPV was assessed for both the pre- and postnatal periods. The attunement literature operationalizes and analyses attunement in several ways. Therefore, three multilevel modeling (MLM) analyses that correspond to three common conceptualizations of attunement were run to test each hypothesis. Overall, the results did not support the hypotheses, suggesting that IPV does not influence mother-infant SNS attunement. The results also suggest that the way in which attunement is conceptualized and analyzed may differentially affect results, which may help to explain the variation in outcomes in the mother-infant physiological attunement literature.

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## INTRODUCTION

When mothers and infants interact with one another, their actions, facial expressions, and vocalizations are often complementary. This continuous and reciprocal exchange of physiological, behavioral and affective signals brings mother and infant into attunement, which serves to promote infant well-being and social-emotional growth (Bornstein, 2007). This attunement helps a mother respond appropriately to her child's needs and aids in the development of infant self-regulation (Feldman, 2007; Middlemiss, Granger, Goldberg, & Nathans, 2012). Attunement between mother and infant begins at a critical time in infant development in which physiological/biological systems, affect, and the environment are integrated to form the basis of lasting patterns in the infant's personality, emotional expression, self-regulation, and stress responsivity (Bornstein, 2014; Fogel, 1993). Although physiological responses are important to the infant's developing experiential framework, physiological attunement in human infants has not yet been well-studied (Van Bakel & Riksen-Walraven, 2008). One type of physiological attunement is attunement of the sympathetic nervous system (SNS), which is one of the body's stress response systems (Laurent, Ablow, & Measelle, 2012).

In SNS attunement, mother and infant may show similar response patterns in indicators of SNS activation (e.g., increases in heart rate, skin conductance, norepinephrine or adrenaline). SNS attunement can also be measured using sAA, a salivary analyte that is indicative of SNS activation (Chatterton, Vogelsson, Lu, Ellman, & Hudgens, 1996). When an infant feels distressed, this can activate the infant's SNS and increase the concentration of sAA in the infant's saliva (Granger et al., 2006). An attuned and emotionally well-regulated mother would theoretically also experience distress, SNS innervation, and sAA increases, but to a lesser extent than the baby. This would allow the mother to be empathic to her baby's distress but also help

her to calm the infant by modeling self-regulation. If the infant is able to attune to the mother, the infant's stress response and sAA will decrease as the baby mirrors the decreasing distress of the mother (Pally, 2010). Attunement may be a concurrent process, in which mother and infant experience physiological changes at about the same time and in the same direction; a reciprocal and lagged process, in which mothers' and infants' physiological arousal influences the others' subsequent physiological response; or it may be led by the mother, such that a mother's physiological response is predictive of her infant's response and the process is not reciprocal. Regardless of how attunement is conceptualized, mother-infant attunement likely plays a role in the development of adaptive infant emotional self-regulation.

Physiological attunement may be negatively influenced by stressors that elicit strong, maladaptive physiological responses and/or lead to emotional dysregulation in the mother, in the infant, or in both. One such stressor is intimate partner violence (IPV: defined here as sexual, physical, or psychological violence perpetrated by a male towards a female partner). With a lifetime prevalence rate of around 25% (Tjaden & Thoennes, 2000), IPV is a common stressor that many women face. Four to eight percent of women report experiencing IPV during pregnancy (Gazmararian et al., 2000), and this percentage may be even higher among ethnic minority and low income women (Charles & Perreira, 2007). IPV is also more common in young adults and typically decreases with age (O'Leary, 1999). Young adults often have young children, and each year millions of children are exposed to IPV (McDonald, Jouriles, Ramisetty-Mikler, Caetano, Green, & 2006). IPV may affect SNS attunement prenatally or postnatally by differentially influencing the maternal and/or infant SNS response (Diego et al., 2004) or by affecting the mother's parenting and mental health (Kessler, Molnar, Feurer, & Applebaum, 2001; Leung & Slep, 2006; Levendosky, Leahy, Bogat, Davidson, & von Eye, 2006). However,



the negative effect of IPV on mother-infant attunement and infant outcomes may be moderated by maternal warmth. Indeed, Skopp and colleagues (2007) found that when mothers were warm (i.e. responding sensitively to their infants and being encouraging), children developed fewer problematic behaviors despite being exposed to IPV.

No published studies have explored whether IPV could negatively impact a child's development by influencing mother-infant SNS attunement using sAA. This kind of research may be important to better understand whether attunement is one of the mechanisms through which IPV influences the mother-infant relationship. Moreover, studying physiological attunement and maternal warmth may help to create new testable hypotheses as to why some mothers and infants who are exposed to IPV have better outcomes than others. The aim of the current study is to examine the effects of IPV on mother-infant SNS attunement in response to a laboratory stressor. This study had four *a priori* IPV groups – a control group with no IPV, a prenatal IPV only group, a postnatal IPV only group, and a pre and postnatal IPV group. Exploring the effect of IPV on mother-infant sAA attunement by IPV grouping would allow for the examination of which time period (i.e. pre- or post-natal) might have the greatest effect on mother-infant SNS attunement.

### **Attunement**

From birth onwards, infants depend on a primary attachment figure, most often the mother, to attune to their needs and respond appropriately (Bowlby, 1969; Leerkes, Blankson, & O'Brien, 2009). Bornstein (2007) defined attunement as a dyadic process in which one or both parties are sensitive to the needs and emotions of the other person and respond by mirroring, co-regulating, harmonizing, and synchronizing. Attunement can be behavioral, in which the mother and child mirror one another's behaviors; affective, in which the mother and infant express

similar emotions; or biological/physiological, in which the mother and infant have similar physiological response patterns (Feldman, 2007). Attunement can be intentional or unintentional and can occur during both stressful and nonstressful situations. Attunement may be concurrent, reciprocal and delayed, or mother-led and non-reciprocal (Laurent et al., 2012; Sethre-Hofstad, Stansbury, & Rice, 2002; Stenius et al., 2008). This attunement of behavior, affect, and physiology between mother and infant occurs at a time in which patterns of personality, intellect, social functioning, and stress reactivity begin to develop that are evident across the infant's life (Bornstein, 2014; Meaney, 2001). These early social and environmental conditions may also modify gene expression and influence physiological regulatory mechanisms. Therefore, it is not surprising that mother-infant attunement affects infant self-regulatory behaviors, attachment, cognition, and social-emotional development (Feldman, 2007; Middlemiss et al., 2012). A lack of attunement over an extended time period with an attachment figure can lead to a disruption in infant development as evidenced by physiological dysregulation, depression, and reduced responsiveness (Field, 1985). Thus, attunement is a vital component of normative infant development and the mother-infant relationship, and it has been found to be predictive of infant outcomes, such as attachment security (Isabella & Belsky, 1991).

Initially, the mother must adapt to her infant's affective state to achieve attunement; by the age of 9 months, however, infants also adapt to their mothers' affect to achieve attunement (Feldman, Greenbaum, & Yirmiya, 1999). Thus, attunement becomes a dyadic process and may indicate shared emotional and physiological states. Conversely, when a mother and her infant are not attuned, this may indicate, that at that moment, the mother is unable to attend to her child's needs and promote the infant's emotional regulation and/or the infant is unable to match the mother's emotional state, leading to discordant or mismatched affective states. Across the

attunement literature, attunement has been conceptualized in different ways. It can be seen as a reciprocal and lagged process in which each member of the dyad influences the other member's subsequent response, or as a concurrent process in which both members of the dyad respond similarly and contemporaneously. In addition, some researchers have specified that the mother leads the attunement process, while other researchers have indicated that either mother or infant can lead.

Mother-infant attunement may be important in the development of infant self-regulation. Feldman et al. (1999) found evidence for the relationship between affective attunement and the transition from mutual mother-infant affective regulation to infant self-regulation. The researchers speculated that dyadic attunement provided the infants with a “contained” context in which to feel increased levels of arousal without fear of dysregulation. Pally (2010) noted that in this contained context, when the baby shows distress, the mother can attune to the baby's feelings and reflect back the baby's emotions in a toned-down way. This simultaneously helps the baby to feel understood while also providing the baby with a model for down-regulating emotional distress. Field (1982) found that when mothers were less attentive to their infants' behavioral signals, the infants who were already at higher risk of hyper-arousal and disturbed interactions were more likely to avoid eye contact with their mothers and experience higher arousal. Thus, both the mother and the infant demonstrated an inability to attune to the other and consequently the babies were more aroused. However, an additional consideration is whether attunement is always advantageous or adaptive (Laurent et al., 2012). If an infant attunes to his mother when the mother is insensitive to the infant's distress or impaired by her own distress, the infant may become more avoidant of his emotions or become increasingly distressed and dysregulated without the modeling and boundaries provided by a sensitive mother (Waters,

West, & Mendes, 2014). In other words, mother and infant may be attuned behaviorally, affectively and/or physiologically, but this attunement may be based on maladaptive responses. Thus, attunement does not always reflect an adaptive response because the response may be inappropriate given the context or the infant's needs (Laurent et al., 2012).

## **Maternal Warmth**

Maternal warmth may also play an important role in the mother-infant relationship. When a mother is affectionate and displays positive affect toward her child, she gives the child an opportunity to learn and practice appropriate affect expression and regulation (Isley, O'Neil, Clatfelter, & Parke, 1999). In this way, maternal warmth may promote attunement by fostering an environment in which attunement may be likely to occur. When a mother is warm and attentive towards her child, her child may be better able to attend and respond to his or her mother. Maternal warmth may also have developmental implications. Maternal warmth in infancy is associated with better later relationships with peers (Davidov & Grusec, 2006), a greater ability to express emotions appropriately (Isley et al., 1999), higher self-esteem (Clark & Symons, 2000), better emotional self-regulation during a frustration task (Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002), and higher IQ at age 4 (Pearson et al., 2011).

Maternal warmth has also been studied in the context of IPV and was found to moderate the relationship between IPV and children's externalizing behaviors, such that IPV and externalizing behaviors were positively related at low levels, but not at high levels, of maternal warmth (Skopp et al., 2007). This suggests that increased maternal warmth may decrease the negative effects of IPV on child outcomes. If maternal warmth moderates the relationship between IPV and child outcomes – outcomes which are also related to disruptions in the mother-

child relationship (Johnson & Lieberman, 2007) – and if IPV negatively impacts mother-infant attunement (as hypothesized in this study), it stands to reason that maternal warmth may also moderate the effect of IPV on attunement. Whereas IPV may disrupt attunement, maternal warmth may foster it, thereby lessening the strength of the hypothesized relationship between IPV and attunement.

### **Sympathetic Nervous System**

The nervous system is responsible for carrying out voluntary and involuntary actions in response to internal and external cues. As outlined by Moore et al. (1988), the human nervous system begins to develop during gestation and is composed of the central (CNS) and the peripheral nervous system (PNS). The CNS is made up of the brain and spinal cord while the PNS consists of the remaining nerves outside of the brain and spinal cord. The PNS is further subdivided into the autonomic and somatic nervous systems. The autonomic nervous system (ANS) has two main components and controls homeostasis, stress responses and arousal by influencing heart rate, sweating, respiration, pupil dilation and digestion. The sympathetic nervous system (SNS), the “activating” portion of the ANS, is one of the main components of the mammalian biological stress system. The SNS controls the body’s “fight or flight” response by releasing two catecholamines, epinephrine and norepinephrine. These hormones activate the body’s stress response by increasing heart rate, increasing blood flow to muscles, and dilating pupils, which better enable the organism to confront or escape a stressor (Cannon, 1914). The SNS is complemented by another component of the ANS, the parasympathetic nervous system (PSNS). The PSNS slows the heart rate, promotes digestion, and increases salivation. It is often referred to as the “rest and digest” division of the ANS.

The SNS can be activated by both physical and psychological stress, from exercise (Chatterton et al., 1996) and submersion in cold water (Speirs, Herring, Cooper, Hardy, & Hind, 1974), to the Trier Social Stress Test (Kirschbaum, Pirke, & Hellhammer, 1993) and exposure to audio recordings of interadult conflict in children (El-Sheikh & Harger, 2001). The development of the SNS begins during gestation and continues after birth (Brodal, 2004). During the final months of pregnancy and into the first year of life, the SNS must adjust quickly to allow the fetus or infant to adapt to the immediate environment (Porges & Furman, 2011; Young & Morrison, 1998). There is evidence of prenatal programming, which may occur when changes to the fetus in the prenatal period contribute to postnatal outcomes of the SNS. Maternal hormones released during an SNS stress response may affect placental blood flow and influence the development of the fetus and the fetal SNS (Giannakouloupoulos, Teixeira, Fisk, & Glover, 1999). These developmental influences may still be apparent in the postnatal period. Indeed, studies have found that women with higher levels of these hormones during pregnancy tend to have infants who showed elevated postnatal levels (Diego et al., 2004; Field et al., 2004). This prenatal programming of the SNS will be discussed further as it relates to SNS indicators, intimate partner violence as a specific stressor, and the mother-infant relationship.

Although this study examines the SNS, there are two primary stress response systems: the SNS and the hypothalamic-pituitary-adrenal (HPA) axis. Both systems are responsible for maintaining and reinstating homeostasis (Chrousos, 2009). Research suggests that both systems are interrelated, although it is unclear whether they interact or have an additive effect on one another (Granger, Kivlighan, El-Sheikh, Gordis, & Stroud, 2007). However, they are associated with different stress reaction profiles (Chrousos & Gold, 1992) and may also be sensitive to different kinds of stress (Allwood, Handwerger, Kivlighan, Granger, & Stroud, 2011). Still, the

HPA axis has been more extensively studied in the physiological attunement literature, and as such, this paper will cite several studies that measured HPA axis activity in the discussion of physiological attunement.

### **Salivary Alpha Amylase**

Indicators of physiological reactivity can be found in saliva, which has long been seen as an informative “diagnostic fluid” (see review in Malamud & Rodriguez-Chavez, 2011). Recently it was found that a known salivary analyte, alpha amylase (sAA), reflects SNS activity (Chatterton et al., 1996). Multiple studies have found a positive correlation between sAA increases and other SNS indicators, and a negative correlation between sAA and PSNS indicators (Bosch, De Geus, Veerman, Hoogstraten, & Nieuw Amerongen, 2003; El-Sheikh, Erath, Buckhalt, Granger, & Mize, 2008; Granger et al., 2006; Nater et al., 2006), leading Nater and Rohleder (2009) to conclude that “the pathways that lead to the secretion of sAA are clearly sympathetic/parasympathetic in nature” (p.492). An increase in SNS activity leads to higher levels of alpha-amylase production, which can be measured by examining saliva samples (Granger et al., 2007). sAA levels in saliva peak within 5-10 minutes of SNS activation and return to baseline within about 20 minutes (Davis & Granger, 2009). Thus, samples can be collected quickly and easily, which is ideal for use with infants and children. Although newborns experience SNS innervation and release norepinephrine, SNS activity cannot be measured using sAA until several months after birth. Newborns do not yet have sAA in their oral mucosa because it begins to develop shortly after birth. By 6 months of age, however, an increase in sAA due to stress can already be measured; between 5 and 6 years of age, sAA activity has reached the maximum level (Davis & Granger, 2009).

Multiple studies have examined the relationship between norepinephrine and sAA because both are associated with SNS activation. However, the results have been inconsistent. Batzri et al. (1971) found that catecholamines (including norepinephrine) were associated with sAA secretion in rat salivary glands, and Speirs et al. (1974) conducted a similar experiment in several humans with comparable results. Chatterton and colleagues (1996) concluded that the concentration of alpha amylase in saliva was predictive of blood plasma levels of norepinephrine in 47 males who underwent either a physical or a psychological stressor. In contrast, Nater et al. (2006) examined 30 men and did not find plasma catecholamines to be associated with sAA concentrations. However, in a 2012 study, Thoma and colleagues examined 66 men and women and found a significant relationship between norepinephrine and sAA following a Trier Social Stress Test. Moreover, the relationship was stronger between norepinephrine and sAA than between norepinephrine and epinephrine. Thus, sAA and norepinephrine were more correlated than two already well-established indicators of the SNS. In sum, the literature generally supports sAA as a valid indicator of the SNS and an association between sAA and norepinephrine.

As a biomarker of the SNS stress response, sAA can be used to measure physiological attunement of the SNS in mothers and their infants. Although mothers and their infants can be attuned during day-to-day activities, physiological attunement may be particularly important for the development of infant emotional self-regulation during distressing situations. When infants are upset and experience an increase in SNS activity, they may need their mothers to attune to their physiological state. This allows the mother to understand her infant's distress and model self-regulatory behavior by helping the infant to downregulate his or her stress response. Several studies have examined physiological attunement during stressful situations by activating the SNS with a frustrating or alarming laboratory task. In a study by Laurent et al. (2012), mothers and



their infants from a high-risk, low SES sample showed greater sAA synchrony during a cleanup task and an emotion battery (Lab-TAB) task than during the Strange Situation, showing the importance of stressor type on the level of physiological SNS attunement. Shea et al. (2006) used a burst of noise and infant arm restraint to elicit a stress response from mothers and infants. The researchers found that sAA levels in mothers and infants were positively related. Gordis et al. (2010) found that mothers and their adolescent children displayed sAA attunement at baseline and when discussing interparental conflict except when the family had a history of interparental aggression. Mothers and their children were not attuned during the discussion in families with interparental aggression. Thus, the degree of physiological attunement depended on two potential stressors: the type of discussion (neutral/negative) and the amount of conflict present in each family (absent/present). These results suggest that mother, child, or both were unable to attend to physiological changes in the other due to the stress caused by the discussion. These studies suggest that mother-child SNS attunement is greatly affected by the type of stressor and the quality of the mother-child relationship (especially in high-risk samples).

Past research on stress responses has often relied on measuring increases in salivary cortisol, an indicator of the HPA axis stress response system. While salivary cortisol has proven to be a reliable indicator of HPA activation, several studies have found that it does not adequately encapsulate a person's stress response to all types of stressors, especially when looking at attunement. For example, Laurent et al. (2012) found that mother-infant dyads had greater cortisol attunement after an attachment stressor, but greater sAA attunement after a challenge task. Moreover, a study by Allwood et al. (2011) found results suggesting that sAA is more reactive to laboratory stressors (performance or peer rejection tasks) than cortisol. These studies suggest that the HPA axis and the SNS respond differentially to different types of

stressors. Researchers have hypothesized that the HPA axis is more reactive to threatening stressors while the SNS is responsive to a wider range of stressors to enable an immediate response to a challenge (Frankenhauser, 1982; Nigg, 2006). Thus, the SNS is more likely to respond to both a stressor like IPV and a stressful lab task. This study will therefore examine the SNS response because the lab challenge used in this study is more likely to elicit frustration and activate the SNS than it is to feel threatening and activate the HPA axis of mothers and their infants.

### **Stress Effects on the SNS**

Psychological factors, such as the physical and emotional stress caused by IPV, have the potential to influence infant SNS development pre- and postnatally, which could lead to maladaptive changes in SNS functioning (Young, 2002). Indeed, Field et al. (2004) found that changes in maternal norepinephrine levels were predictive of changes in fetal and neonate norepinephrine levels. Diego and colleagues (2004) had a similar finding when they looked at the effect of pre- and postpartum maternal depression on norepinephrine dysregulation. They found that depressed women had higher levels of norepinephrine and their infants did as well. The effect of maternal depression on infant SNS activity was greatest when mothers had both pre-and postpartum depression. These results suggest that an emotional response (here, maternal depression) to a stressor that increases norepinephrine release can influence the development of the fetal norepinephrine response. Because norepinephrine and sAA are both associated with SNS activity, higher plasma norepinephrine may be indicative of greater SNS sensitivity to stress and thus may also be related to increased sAA responsivity. Thus, increased maternal norepinephrine may lead to prenatal programming of the child's SNS, which may be associated with greater stress reactivity postpartum (Phillips & Barker, 1997; Phillips & Jones, 2006).

Moreover, the results also indicate that the chronicity or the timing of the stressor may be important. The authors speculate that the effect of increased maternal SNS activity on fetal SNS development may be indirect. Maternal norepinephrine can restrict uterine blood flow, which in turn precipitates the activation of the fetal stress response (Giannakouloupoulos et al., 1999). Thus, the effect of a stress-induced increase in maternal SNS activity during pregnancy may be critical to the development of the infant's SNS during and after parturition. It may also lead to lasting changes in the child's SNS reactivity. Moreover, these changes in SNS functioning and norepinephrine levels may be accompanied by changes in sAA concentrations due to the positive relationship between SNS activation and sAA levels.

Chronic or more extreme trauma can also have a blunting effect on a person's SNS. D'Andrea and colleagues (2013) found that during an acoustic startle paradigm, individuals with moderate trauma exposure showed increased SNS reactivity, whereas individuals who had more chronic exposure or worse trauma had blunted SNS reactivity and increased PSNS reactivity. These results suggest that individuals tend to respond to a stressor with SNS activation. If the stressor becomes chronic and/or significantly worsens, however, the SNS response may become blunted and the individual may dissociate and not have a normative physiological response to the stressor. Thus, women who experience more chronic IPV may have initially responded to a stressor with SNS hyper-reactivity, but over time, this response may dampen until they no longer experience SNS arousal when faced with a stressful situation that would normally elicit an SNS response. If a mother who experienced chronic IPV were to dissociate in the face of a stressful situation, she may not be able to attend to her baby or respond empathically because she may not feel the same physiological arousal that the infant is feeling. Therefore, examining the chronicity of a stressor like IPV may be crucial in understanding the effects on SNS reactivity and the

implications for dyadic attunement. Importantly, the existing mother-infant physiological attunement literature has not yet examined the effect of chronic versus non-chronic trauma on attunement. However, research on the effects of duration of trauma on the SNS would suggest that differentiating between chronic and non-chronic trauma in studies of trauma and attunement may be important.

IPV could disrupt the nascent mother-infant bond during this important developmental stage in the infant's life. Women who are victims of IPV are at a greater risk of developing mental health problems, such as depression, posttraumatic stress disorder, suicidality, and anxiety (Kessler et al., 2001; Pico-Alfonso et al., 2006). They are also more likely to endorse physical and mental health problems (Coker et al., 2002). These negative mental health outcomes are associated with harsh parenting and less sensitive caregiving (Leung & Slep, 2006). For example, depressed mothers tend to be less warm and consistent in their caregiving (Holmes, 2013; Leung & Slep, 2006) and are less likely to perceive their parental role as being positive (Black et al., 2002). Furthermore, if a mother is herself emotionally dysregulated, she may be less able to model adaptive self-regulatory behaviors to her child (Low & Stocker, 2005). IPV specifically is associated with inconsistent parenting, less physical affection, more hostile parenting, and child abuse (Casanueva & Martin, 2007; Levendosky et al., 2006; Ritchie & Holden, 1998; Rossman & Rea, 2005). Moreover, women who have experienced IPV are more likely to have distorted, incomplete, and unrealistic views of their children (Sokolowski, Hans, Bernstein, & Cox, 2007), and this distorted representation is associated with insecure infant attachment (Huth-Bocks, Levendosky, Theran, & Bogat, 2004). Thus, IPV may negatively influence maternal warmth and caregiving, which in turn may reduce a mother's sensitivity to her infant's needs and disrupt mother-infant attunement. Conversely, Levendosky and colleagues

(2003) found that some women who experience IPV “compensate” by being more warm and attentive parents. In this case, IPV may be associated with increased mother-infant attunement due to more sensitive caregiving.

The effect of IPV on mother-infant physiological attunement has not been well studied, especially in infants aged one or younger. Still, given the relationship between IPV and parenting problems and between IPV and distorted maternal representations of their infants, it seems feasible that IPV would be associated with changes in attunement. In light of the research that suggests that younger children may be more affected by IPV, the relationship between IPV and mother-infant attunement difficulties may be stronger in infants. An important thing to consider is that this relationship may vary in mother-infant dyads depending on the type of behavioral, emotional or physiological response - women who have experienced IPV may be more able to attune to their infants if the infant is expressing a positive behavior or emotion because this would not alarm the mother and she would be less likely to respond with harsh parenting or become distracted by her own response. If the infant is distressed, however, there may be a greater difference in the quality of the subsequent mother-infant attunement between women who have and have not experienced IPV.

There is a paucity of research studying physiological attunement, IPV, and maternal factors. Johnson & Lieberman (2007) examined affective attunement in mothers and their preschool-aged children who had been exposed to IPV and found that IPV was not related to affective attunement. In a study by Hibel and colleagues (2009), the authors studied physiological attunement in mother-infant dyads, some of whom had experienced IPV. Mothers and infants from IPV-exposed dyads showed more physiological attunement during an arm restraint task than did dyads not exposed to IPV, which suggests that the context of the early

mother-infant relationship may affect the coordination between the mother's and the infant's stress response. However, physiological attunement in this study was measured by using cortisol as a marker of HPA reactivity, which means that the results cannot be applied to physiological attunement as measured by sAA as a marker of the SNS. Despite the fact that the relationship between IPV, physiological attunement, and maternal factors has not been well studied, this area of research may be important for understanding one pathway through which IPV influences infant development and the mother-infant relationship. The goal of the present study is to explore how IPV and maternal warmth influence mother-infant attunement. This information may help to inform intervention efforts that aim to minimize the negative effects of IPV on child outcomes by adding to pre-existing knowledge of protective and risk factors.

This study is designed to address several of the limitations of the existing attunement literature that have been reviewed here. Firstly, this study aims to not only examine the effect of trauma on mother-infant physiological attunement, but also to explore whether the effect differs between chronic and non-chronic trauma. In addition, to address the problem of differing definitions and analyses of physiological attunement, the data in this study will be analyzed using three different analyses, which correspond to three often-used conceptualizations of attunement, to examine whether the different conceptualizations of attunement would significantly influence and differentiate the results.

## **Hypothesis 1**

The first hypothesis is that there will be differences in patterns of mother-infant attunement across the four IPV groups.

The control group is expected to show the greatest attunement because mothers will be most able to model resilience and regulate their own emotions due to not having experienced recent and/or ongoing IPV and infants will be most able to attune to their mothers because they have not been prenatally programmed and/or postnatally exposed to IPV. Mothers and infants will be able to implement adaptive self-regulatory strategies that reduce the infant's negative emotions and increase positive emotions (Schoore, 2005).

Mothers and infants in the prenatal IPV only group are predicted to be less attuned than the control group. While mothers may not have any difficulty modeling self-regulation because their experience of IPV is not recent, infants in this group may be prenatally programmed to over-react to a stressor. This prenatal programming may make it more difficult for infants to attend to their mothers.

The postnatal IPV only group is also hypothesized to be less attuned than the control group. The recency of IPV may lead to hyper-arousal of the SNS, particularly in the mothers. These mothers may react more strongly to a perceived threat and have greater difficulty reducing their distress. Consequently, they may not have been able to help their infants self-regulate in the past. Thus, the infants may react more strongly to the in-lab stress task and be less capable of self-soothing. Although the mothers may be aware of the infants' distress, they may be distracted by their own physiological response to the stressful environment and the infants' crying. As a result, they may not be able to attune to the child. Therefore, the dysregulation of the stress response of both dyad members will result in less physiological attunement.

Mother-infant dyads in the prenatal/postnatal (chronic) IPV group are hypothesized to be the least attuned. Due to the chronicity of IPV, mothers may be less reactive to stress as a whole and have a delayed and attenuated physiological response. Like the infants in the prenatal IPV

only group, the infants in this group may have also been prenatally programmed, which is associated with an increased stress response. Thus, the infants in this group may react more strongly to a stressor, while their mothers do not. Moreover, because the mothers may be less reactive to stressors, they may also be less reactive and sensitive to their infants' needs or distress. If this is the case, these mothers may have been unable to show their infants how to regulate emotions during previous distressing situations. These mothers would then also be less able to attune to their infants during the lab stress task. Consequently, mothers and infants in this group may be less attuned because mothers may have a blunted response to stress while infants may react more strongly to stress due to prenatal programming and an inability to self-regulate.

Alternatively, the postnatal IPV group and the chronic IPV group may have the most attuned mothers and infants because they may have more shared stressful situations at home, causing them to react similarly to a stressful laboratory task. Dyads without postnatal IPV may not react in a similar way to a stress task because they did not have the same kind of shared stressful experiences to base their responses on.

## **Hypothesis 2**

The relationship between maternal warmth and attunement is hypothesized to be stronger in dyads with prenatal/postnatal IPV compared to dyads with no IPV, prenatal IPV only, or postnatal IPV only. Infants in dyads with more chronic IPV may respond more strongly to stress due to both prenatal programming and the stressful postnatal environment, and thus may be more dependent on their mothers to help with self-regulation and adaptation. Mothers from these dyads may tend to have a blunted SNS stress response, making it more difficult for them to respond warmly and appropriately to their distressed infant and model adaptive self-regulatory



behaviors. However, when mothers are warm, they may be able to attend to their babies' needs in spite of the IPV exposure, causing the dyad to be attuned. In dyads with prenatal/postnatal IPV, maternal warmth may be more crucial for attunement to occur because the child may be predisposed to an over-reactive stress response and there are more distressing moments in which the child can become dysregulated emotionally and physiologically. In dyads with no IPV, prenatal IPV only or postnatal IPV only, mother and infant may be less prone to responding inappropriately to a stressor, so they may be more likely to respond in an attuned way regardless of whether the mother is high in warmth or not. Thus, the relationship between maternal warmth and attunement is expected to be strongest in the prenatal/postnatal IPV group.

## **METHODS**

### **Participants**

182 mother-infant dyads participated in the study. They were recruited from three counties in Michigan using fliers, Craigslist, and Facebook. These fliers were posted in stores, restaurants, libraries, medical offices (pediatrician and OBGYN), daycares, and preschools. To recruit IPV-exposed dyads, fliers were also placed in domestic violence shelters and given to agencies that help victims of domestic violence. The study planned to recruit 40 women for each group (prenatal IPV, postnatal IPV, pre- and postnatal IPV and control) who were matched on demographic measures. In total, 914 women were screened, of whom 182 were eligible for the study.

### **Procedures**

Women were first screened over the phone for eligibility. In order to be included in the study, they needed to be (1) English speakers, (2) between the ages of 18 and 34 years old, (3) not currently pregnant, (4) fine with not breastfeeding their child for 2 hours before the assessment, (5) free from endocrine disorders, cancer, or currently receiving treatment for cancer, (6) in a heterosexual romantic relationship for at least 6 weeks during the pregnancy, and (7) their children were not born prematurely before 37 weeks gestation.

Because of the difficulty in finding women who had experienced postnatal IPV only and prenatal IPV only, the number of participants in each group varied. The control group had 58 women (31.9%), the prenatal IPV only group had 34 women (18.7%), the postnatal IPV only group had 12 women (6.6%), and the pre- and postnatal group had 78 women (42.9%).

Mothers and their infants were interviewed when infants were around one year old, and

all interviews were conducted in the afternoon to minimize the effect of sampling time on the diurnal rhythm of salivary alpha amylase (see Nater, Rohleder, Schlotz, Ehlert, & Kirschbaum, 2007). Mothers were asked not to drink alcohol 12 hours prior to the visit, not to brush their teeth, chew gum or consume a large meal 1 hour before the visit, and not to ingest sugary or acidic foods 20 minutes before the visit. The interviews were conducted by two undergraduate students. Interviewers inquired about maternal and infant health and took forehead temperatures to confirm that mother and infant were not ill. Then they collected a pre-task saliva sample from both mother and infant. Mothers were asked to “chew” to collect saliva and then pass the saliva into a cryogenic vial. Saliva was obtained from infants by placing two micro sponge devices under the infant’s tongue for 60-90 seconds. These microsponges were transferred to cryogenic vials. Then the Modified Lab-TAB Arm Restraint task was completed to assess sympathetic nervous system activation and regulation. Five minutes after the task, when sAA normally peaks, saliva was collected again from mothers and infants. Then mothers filled out non-stressful questionnaires (so as not to influence sAA levels). 20 minutes after the task, when sAA usually returns to baseline or near-baseline levels (recovery), saliva was collected from mothers and infants. After saliva was collected, mothers filled out the remaining questionnaires and were compensated for their participation. Infants received a stuffed animal.

## **Measures**

*Intimate Partner Violence:* Women were first assessed during the telephone screening using questions 9-19 of the *Conflict Tactics Scale (CTS; Straus, 1979)* to determine the timing of IPV and preliminarily place women in one of the four groups. When women came in for the in-person interview, the *Severity of Violence Against Women Scales (SVAWS; Marshall, 1992)* was given to determine the final classification of women into the IPV groups. The SVAWS is a 46-

item measure that assesses psychological, physical, and sexual violence on a 4-point scale.

Women also indicated how distressing each item was on a scale of 1 to 3. Women were asked to complete the SVAWS twice: once for prenatal IPV experiences and once for postnatal IPV experiences. Women were grouped based on their responses to #14 and higher (these items assess minor, moderate, serious and sexual violence). They were also asked to indicate whether any violence had occurred in the past 48 hours and if their infants had witnessed or heard any of the violence. In the present sample, for the prenatal SVAWS,  $\alpha = .978$ , and for the postnatal SVAWS,  $\alpha = .975$ .

*Parenting Behavior Checklist (PBC; Fox, 1992):* The PBC is a 50-item parenting self-report measure used for women with children ages 1-4. There are 3 subscales: Expectations, Discipline, and Nurturing. Only the nurturing subscale ( $\alpha = .826$ ) was used in this study, and it measures how much mothers stimulate and encourage the psychological growth of their children (maternal warmth).

*Challenged Salivary Alpha Amylase: The Modified Lab-TAB Arm Restraint task* (Goldsmith & Rothbart, 1999) was used to assess mother and infant stress responses and regulation via sympathetic nervous system activation. In the Arm Restraint task, which is used to assess child temperament, an infant sits in a highchair and is given a toy to play with. Usually the mother holds the child's arms, but in the modified version used for this study, the interviewer restrained the child's arms gently after 2 minutes of play. The interviewer sat directly behind the child and the mother sat behind and to the left of the infant. The infant could see neither the interviewer nor the mother, but the mother could see the child. The interviewer held the infant's arms at the infant's sides for 2 minutes or until the infant cried hard for 20 seconds. The mother was told beforehand that if the infant became too upset by the task, the interviewer would stop.

The mother could also stop the task at any time. After the task, the infant was taken out of the chair and given back to his or her mother.

Mother and infant saliva samples were collected pre-task, 5 minutes after the task, and 20 minutes after the task. The saliva samples were kept in a freezer until they could be assayed and analyzed. Commercially available kinetic reaction assays were used. Saliva samples were analyzed by pipetting samples (8 ul diluted 1:200) onto 96-well plates. These plates contained 2-chloro-p-nitrophenol and maltotriose, which react spectrophotometrically with sAA such that the amount of sAA in the sample was proportional to the optical density of the product. The intra-assay variation was computed using the mean of 30 replicate tests and was less than 7.5%. The inter-assay variation was calculated using the mean of average duplicates of 16 runs and was less than 6%.

### **Proposed Analyses**

Other studies have analyzed physiological attunement data using multiple methods, such as difference score correlations, regression, and multilevel modeling (MLM). As mentioned previously, they also had different conceptualizations of attunement. However, the data analytic strategies in these studies were not always in accordance with the definition of attunement given in the study (e.g., operationalizing attunement as lagged process but analyzing the data as if it were concurrent). More recent studies have tended to forgo the more simplistic and flawed analytical methods (e.g., difference score correlations) in favor of multilevel modeling (MLM), which is recommended for this kind of data analysis. MLM can be used to analyze data in which individuals are nested within dyads, and individual data is crossed with time due to the multiple data collection time points. Therefore, MLM will be used to analyze the present data.

Three MLM analyses, which correspond to the aforementioned three conceptualizations of attunement, will be run for each hypothesis. Firstly, a correlated growth model will be run, which corresponds to the conceptualization of attunement as concurrent and similar changes in mother and infant across time. Then, a cross-lagged analysis will be conducted, which corresponds to the definition of attunement as being a reciprocal and lagged process, in which both members of the dyad influence the other's subsequent response. The last analysis will be one in which maternal values predict infant values over time, which corresponds to the conceptualization of attunement as a non-reciprocal process.

### **Exploratory Post-Hoc Examinations**

If there are no significant differences among the four *a priori* groups or if there is not enough variability within each group to determine whether they are attuned or not, *post-hoc* examinations will be done.

## RESULTS

### Missing Data

Of the 182 dyads, 23 had one or more missing sAA values. This may have been due to mothers and infants not being able to produce enough saliva needed for the measurement at one or more collection time points, or infants being too upset for a sample to be taken. Missing sAA values were not imputed because this was not necessary for the planned analyses as MLM is robust to missing data. There were no missing items for any dyads on the Nurturing subscale of the PBC or for the pre- and postnatal SVAWS.

To determine whether the absent sAA values were missing at random, each dyad was coded with either a “0” to indicate no missing data, or a “1” to indicate that 1 or more sAA values were missing in a dyad. This was then correlated with the other measures that were used and important demographic variables. IPV grouping, the presence of IPV at any time point (dichotomously coded), SVAWS scores, maternal warmth, maternal education, maternal age, infant gender, monthly income, maternal ethnicity, maternal mental health issues (depression, anxiety, PTSD), and maternal drug and alcohol use were not significantly correlated with the presence or absence of missing data. Therefore, there does not appear to be a pattern to the missing data, and the data will be considered to be missing at random (e.g., see Counts, Nigg, Stawicki, Rappley, & von Eye, 2005).

### Hypothesis 1

- (1) *Does the timing and duration of IPV exposure affect mother-infant attunement in an acutely stressful situation?*

It was hypothesized that there would be differences in mother-infant attunement among the four *a priori* IPV groups. Dyads from the control group were expected to show the greatest attunement, the prenatal IPV only group and the postnatal IPV only group were expected to be less attuned, and the prenatal/postnatal IPV group was expected to be the least attuned. However, the four group model would not run because there was not enough variability in the sAA values across time, particularly among the infants. The model was then simplified by dividing the dyads into two groups rather than four. Given that chronic IPV was expected to be the most disruptive to attunement, the IPV groups were then divided to examine the relationship between chronic versus non-chronic IPV and attunement. The first group was made up of dyads who had either experienced no IPV, prenatal IPV only, or postnatal IPV only ( $n = 104$ ) (i.e. no or some IPV). Dyads with both pre- and postnatal IPV were in the second group ( $n = 78$ ) (i.e. chronic IPV). The means and standard deviations are presented below, in Table 1.

**Correlated Growth Analysis.** The correlated growth analysis evaluates attunement as a concurrent process. For this analysis, the fixed effects model included separate intercepts and slopes for mothers and babies. The intercept estimated the person's average sAA and the slope measured the linear change in sAA as time increased by one unit. In the random effects part of the model, different mother and baby intercept and slope variances in each of the two groups were specified. The model then attempted to estimate the correlation between mother and baby slopes separately for the two *a priori* groups. The IPV variable was effects coded (no/some IPV or chronic IPV). In this model, individuals were nested within dyads, and this was crossed with time. The fixed effects of the model included separate intercepts and slopes for mothers and babies, as well as interactions between the intercepts and slopes and the level two IPV variable. The IPV by intercept interaction would indicate whether there were mean differences in average



sAA for the two groups, while the IPV by slope interaction would show whether there were average differences in the change in sAA over time as a function of IPV. The model also included separate random effects for the two IPV groups. For each group the model included variances in the intercepts and slopes of the mothers, variances in the intercepts and slopes of the infants, the covariance of mother and infant intercepts, the covariance of mother and infant slopes, and the residual variances.

Results for the fixed effects from the two-grouping growth model showed that there was a significant mean difference in average maternal sAA for the two IPV groups ( $\beta = -.844, p < .001$ ), but there was no significant mean difference between both IPV groups in average infant sAA ( $\beta = .213, p = .236$ )

Results for the random effects are presented in the Table 3. They indicated that mothers' intercepts and slopes varied significantly in both groups. There was significant variance in the babies' intercepts in both groups, but variance was not significant in babies' slopes in both groups. Mothers' and babies' intercepts covaried significantly in both groups such that mothers who were relatively high in sAA on average had babies who were also relatively high on average. Mothers' and infants' slopes did not covary in either group, suggesting that mothers and infants were not attuned and that the chronicity of IPV did not have a significant and differential effect on attunement.

**Cross-Lagged Analysis.** The reciprocal and lagged definition of attunement can be tested in a cross-lagged model like the one depicted in Figure 1. The horizontal arrows represent stability (e.g., a mother's sAA value at T1 will be related to her sAA value at T2), and the

diagonal arrows represent the influence of one member of the dyad's past response on the other's subsequent response (e.g., a baby's sAA value at T1 is related to the mother's sAA value at T2).

Mothers and infants were treated as both "actors" and "partners" within the model to test the reciprocal and lagged influence of mother and infant sAA, while controlling for the effect of an individual's past response on that individual's next response. Actor and partner scores were grand mean centered, and mother and infant were dummy coded. IPV was effects coded (no/some IPV or chronic IPV). Dyad ID and time were entered as repeated effects. The dependent variable was actor sAA. The residual structure was specified as compound heterogeneous symmetry (CSH), which allowed for unequal variances.

According to the results, a person's previous sAA was a good predictor of that person's subsequent sAA for both mothers and infants (infants:  $\beta = .895$ ,  $p < .001$ ; mothers:  $\beta = .824$ ,  $p < .001$ ), indicating that there was stability. However, neither mother nor infant sAA values influenced the other person's subsequent sAA value (Baby:  $\beta = -.009$ ,  $p = .646$ ; Mother:  $\beta = .068$ ,  $p = .156$ ), suggesting that influence was not significant. Moreover, IPV did not moderate the relationship between actor and partner sAA, nor was IPV related to maternal or infant sAA across time. Thus, IPV did not significantly affect the stability or influence of maternal or infant sAA values.

Thus, the results suggest that mother and infant sAA values were stable, such that a person's past sAA value significantly predicted the person's subsequent sAA value. However, mothers' previous sAA values did not predict infants' subsequent sAA values, and infants' previous sAA values did not predict subsequent maternal sAA values. This partner effect remained nonsignificant even when IPV was added as a moderator. In addition, IPV did not affect maternal or infant sAA values across time.

**MLM – Mom Predicts Baby Analysis.** The conceptualization of attunement as non-reciprocal can be examined with a mixed model analysis in SPSS, in which the values of one dyad member are predictors of the values of the other dyad member. Here, maternal sAA was the predictor, and infant sAA was the dependent variable. Thus, this analysis examined whether a mother's sAA at a particular time point predicted her infant's sAA at that same time point.

In this model, the intercept, maternal sAA, IPV group, and the maternal sAA x IPV interaction were entered as fixed effects predicting the infant's sAA. The intercept and maternal sAA were also entered as random effects. The fixed effects indicated whether mothers and infants were similarly high or low at specific time points; the random effects gave measures of intercept, residual and maternal sAA variance. Maternal sAA was centered and IPV was effects coded (no/some IPV and chronic IPV). The residual structure was specified as variance components (VC), which specifies variances for intercepts and for the effect of maternal sAA on infant sAA and residual, but fixes the covariances between these parameters to zero.

Results of this analysis suggested that maternal sAA predicted infant sAA, such that if the mother had high sAA at a time point, the baby tended to have high sAA at that time point ( $\beta = .09, p = .007$ ). IPV did not predict infant sAA ( $\beta = .257, p = .142$ ), and the strength of the relationship between maternal sAA and infant sAA was not significantly changed by the presence or absence of chronic IPV ( $\beta = -.021, p = .521$ ). However, the estimate of maternal sAA variance suggested that the degree to which a mother's sAA predicted her baby's sAA differed from family to family ( $\beta = .039, p = .005$ ).

**Brief Summary of Results.** The results of all three analyses suggest that IPV did not moderate mother-infant attunement in this sample. There was slight variation in the results from each. The correlated growth model indicated that the change in maternal sAA over time differed

between the no/some IPV group and the chronic IPV group, suggesting a blunting of the maternal SNS response in the chronic IPV group which was a prediction of this hypothesis. This is consistent with research that suggests that chronic trauma is associated with a decreased SNS stress response. However, infants' sAA did not differ between both groups and also did not significantly vary across time, and so dyads did not have attuned stress responses in either group. The cross-lagged analysis only indicated that mother and infant sAA values across time were stable. Maternal and infant past sAA values did not significantly influence the other's subsequent response, suggesting that mothers and infants were not attuned. Stability and influence did not differ between both IPV groups, indicating that IPV did not have a significant effect on sAA values. In the analysis in which maternal sAA was examined as a predictor of infant sAA, maternal sAA was a significant predictor of infant sAA, and the strength of maternal sAA as a predictor varied from dyad to dyad. However, this variance was not accounted for by the presence or absence of chronic IPV. The results of the last analysis may have differed from the other results because it was not a dyadic or lagged measure of attunement and did not treat the mother and infant SNS responses as reciprocal.

Because the arm restraint task was terminated early if an infant cried hard for 20 seconds, a regression was run to determine whether the lack of results was due in part to the infants of one IPV group becoming significantly more distressed and needing to end the arm restraint task early. The regression suggested that IPV grouping did not predict early termination of the arm restraint task ( $\beta = .082, p = .274$ ). Therefore, the lack of a significant difference between infants of both IPV groups is likely not a function of early task termination due to excessive infant distress.

## Hypothesis 2

*(2) Does maternal warmth influence the relationship between the maternal SNS stress response and the infant SNS stress response? Does the relationship differ in the context of exposure to both pre- and postnatal IPV?*

It was hypothesized that in dyads with both prenatal and postnatal (chronic) IPV, higher levels of maternal warmth would be associated with increased mother-infant attunement; in dyads with no IPV, prenatal IPV only, or postnatal IPV only, the relationship between maternal warmth and mother-infant attunement was not expected to be as strong. Given that there was not enough variability in sAA within the *a priori* groups, and that some of the groups were too small, the original analyses that were planned to test this hypothesis were not possible. Instead, the data were examined using the three analyses from the first hypothesis. All analyses were run in SPSS.

**Correlated Growth Analysis.** The first analysis was a correlated growth model. IPV was effects coded (1 = chronic IPV, -1 = no/some IPV), and time and maternal warmth were both grand-mean centered. IPV, time, maternal warmth, maternal sAA, infant sAA, and their interactions were entered as fixed effects into the model. The mom by time (centered) interaction and the baby by time (centered) interaction were entered as random effects. The dependent variable was a person's sAA. Dyad ID by time was entered as a repeated effect. The covariance structure was specified as CSH. For more information about the model, please see Hypothesis 1.

Maternal warmth was not a significant predictor of maternal or infant average sAA values or sAA slopes across time, even when the chronicity of IPV was taken into account. These values are presented in Table 6.

**Cross-Lagged Analysis.** As mentioned in the first hypothesis, in the cross-lagged model, mothers and infants were entered as both “actors” and “partners” into the model. For this analysis, maternal warmth was also entered into the model as a fixed effect. Actor and partner scores were grand mean centered, as was maternal warmth. Mother and infant were dummy coded, and IPV was effects coded (1 = chronic IPV, -1 = no/some IPV). Dyad ID by time was entered as a repeated effect. The dependent variable was actor sAA. The residual structure was specified as CSH.

The relationship between a partner’s past sAA and the actor’s present sAA (which could indicate attunement) remained nonsignificant when maternal warmth and IPV were included in the model (mother as partner,  $\beta = -.005$ ,  $p = .40$  and baby as partner,  $\beta = -.002$ ,  $p = .46$ ). The estimates of all fixed effects in this analysis are presented in Table 7.

**MLM – Mom Predicts Baby Analysis.** In this analysis, maternal sAA, maternal warmth, and IPV were examined as predictors of infant sAA. The maternal variables, IPV and their interactions were entered as fixed effects, and infant sAA was specified as the dependent variable. The intercept and maternal sAA were entered as random effects. Maternal sAA and maternal warmth were grand-mean centered. IPV was effects coded to indicate the presence of chronic or no/some IPV. The residual structure was VC. For additional details, please see the corresponding analysis for the first hypothesis.

Maternal warmth, IPV, and their interactions with maternal sAA were not predictors of infant sAA. The estimates of the fixed effects are presented in Table 8.

**Brief Summary of Results.** All three analyses indicated that maternal warmth did not significantly influence mother-infant attunement in the presence or absence of chronic IPV.

Moreover, maternal warmth was not significantly related to maternal or infant sAA over the course of the laboratory stress task.

## DISCUSSION

The present study examined the relationship between IPV, mother-infant physiological attunement, and maternal warmth using six MLM analyses. This was the first study to explore mother-infant sympathetic nervous system attunement in an IPV-exposed population and the first to examine the association between maternal warmth and mother-infant attunement in the context of IPV. In addition, given the multiple conceptualizations of attunement within the literature and the variability within the analyses used to determine attunement, this study was an important step towards examining whether differences within the conceptualization and analysis of attunement lead to different results.

This study had two research questions. Specifically, it aimed to examine the relationship between the timing and duration of IPV exposure and mother-infant physiological attunement during a stressful situation; and to examine the relationship between maternal warmth and mother-infant physiological attunement during a stressful situation, and whether this relationship differed depending on the IPV experiences of a dyad.

It was hypothesized that IPV would be related to mother-infant physiological attunement during a stressful situation that was unrelated to IPV. However, the initially planned analyses could not be run using the four group model. Instead, the dyads were split into two groups: a group who had experienced either no IPV or IPV during only one time period, and a group who had experienced IPV during both the pre- and postnatal periods. Three analyses were run that corresponded to the three main conceptualizations of attunement from the physiological attunement literature. Overall, the findings from all three analyses did not support this hypothesis. The final analysis did suggest a relationship between the maternal stress response



and the infant stress response, although this relationship did not vary between both IPV groups as hypothesized. It did vary between dyads, suggesting that other intra-family factors may account for attunement differences.

Due to the lack of research in this area, a null finding is difficult to interpret. It could be that the null finding is valid and that the results are real or it could also be that the lack of findings is due to a methodological issue. Both possibilities will be explored more in depth here.

Mothers and infants who had experienced IPV were expected to be less physiologically attuned when stressed, particularly if the IPV exposure was both prenatal and postnatal. However, the results indicated that attunement was not related to IPV exposure. If these results are valid, they suggest that IPV may not influence mother-infant physiological attunement. The presence or absence of chronic IPV did not differentiate infants in the study, but mothers who experienced chronic IPV had lower levels of sAA than mothers who did not. This suggests that there may be important differences between mothers and infants that leaves mothers more vulnerable to SNS changes following exposure to chronic IPV. This may be because the mothers were the direct targets of the IPV, whereas the infants may have experienced the IPV more indirectly. There may also be age-related differences in the effect of IPV on SNS reactivity. Perhaps IPV does not affect SNS reactivity in infants (via prenatal programming and postnatal exposure) but does affect reactivity in adults. However, potential reasons for this are unclear given that prenatal programming of the infant SNS has been found in another study that examined the effect of prenatal exposure to a stressor on postnatal SNS responsivity. This inconsistency suggests that prenatal programming of the SNS should be further studied.

The null finding for the effect of IPV on attunement is inconsistent with attunement findings in a similar sample when the HPA axis was the stress system of interest. Hibel and colleagues (2009) found that IPV affected HPA axis attunement in exposed mother-infant dyads. Thus, it may be that IPV only influences mother-infant physiological attunement of the HPA axis and not of the SNS stress response. This may be because sAA responsivity varies based on age. Granger and colleagues (2006) found differences among infants, school-aged children, adolescents, and adults in sAA responsivity to social stressors. The infants and children did not show an increase in sAA in response to a stressor while the adolescents and adults did. These results are similar to the findings of this study: infants did not show significant variability in their sAA reactivity, while the mothers showed more variability. Thus, the null findings may be attributable in part to infant sAA not yet being significantly responsive to a short social stressor (arm restraint).

However, it is also possible that the lack of findings is due to a methodological issue. For example, there may have been problems with the activation or measurement of SNS reactivity. There was not enough variability in sAA, which could mean that the arm-restraint task was not stressful enough to elicit a sufficient stress response from both mother and infant. While the task itself was not expected to be directly stressful for mothers, it was thought that the task might be indirectly stressful via the infants' response to the task. However, the infants may have felt more irritated by the arm restraint than threatened, which in turn may not have been sufficiently stressful to elicit the mothers' stress responses. Thus, the arm restraint task may not have been stressful enough for a one-year-old to increase the amount of sAA in the infant's oral mucosa. The lack of sAA variability in infants in this study is consistent with the results of Granger and colleagues (2006), who also did not find changes in sAA following stressful lab tasks (including

the arm restraint task) in infants six to ten months of age. Laurent and colleagues (2012) used multiple Lab-TAB tasks to elicit an SNS stress response, suggesting that perhaps the one task used in this study was insufficient. Still, it is important to note that in this sample, the arm restraint task was stressful enough to activate the infant HPA axis stress response. This finding is difficult to explain, given that the SNS is typically more easily activated and responds to a wider range of stressors than the HPA axis.

The lack of findings may also have been due to the sample. Firstly, it was a high risk sample, which means that dyads within the *a priori* groups may not have been significantly different in terms of overall stress that they experienced, which was not controlled for. Secondly, the *a priori* group sizes did not turn out as initially planned. They were unequal in size and two of the groups were smaller than had been initially planned. Specifically, it was difficult to find women who had experienced IPV only during the prenatal period or only during the postnatal period. In relationships plagued by IPV, it is less common for IPV to suddenly end or suddenly begin when pregnancy is over unless a woman leaves or begins a relationship with an abusive partner around that time. Consequently, it was difficult to recruit an equal and sufficient number of women for each IPV group. If there had been enough women in each group, the initially planned analyses might have run, and this study may have been better able to illuminate how the timing and duration of IPV is related to mother-infant physiological attunement. As this was not the case, this study could not explore whether the timing of IPV (pre- or postnatal) was differentially related to mother-infant attunement.

The second hypothesis of the study was that maternal warmth would be associated with mother-infant physiological attunement, and that the strength of this association would be related to IPV. The same three analyses from the first hypothesis were also run here. None of the

analyses showed an association between maternal warmth and mother-infant physiological attunement, regardless of whether the dyad had experienced chronic IPV or not.

The results of this analysis may be an accurate reflection of the relationship (or lack thereof) among physiological attunement, IPV and maternal warmth. If the results are valid, they could suggest that warm and nurturing parenting is not related to mother-infant physiological attunement and that this relationship is not influenced by the presence or absence of chronic IPV.

However, these results may not be valid and the lack of findings may be due to methodological issues. Firstly, the measurement of maternal warmth was a self-report measure, and thus it may not have captured maternal warmth in the same way that an objective measure would have. Secondly, this hypothesis builds off of the first hypothesis. Thus, if the null findings for the first hypothesis were due to a methodological issue, this would have a direct impact on the findings for the second hypothesis.

The limitations addressed here provide direction for future research, which may create a context within which to understand the results of this study. Given the lack of consensus in the physiological attunement literature as to how to define and determine attunement, this study aimed to examine not only whether mother-infant dyads were attuned, but also whether the results stayed the same across different conceptualizations and analyses of attunement. Although the results of this study were similar across the three types of analyses used, the analyses should be replicated in other samples. Until then, studies that conceptualize and analyze attunement differently cannot necessarily be compared because they may be measuring different things or using analyses that treat the data differently.

Additionally, future studies could break up the prenatal and postnatal periods. The effect of IPV on fetal and infant SNS development and on mother-infant attunement may differ depending on when during development the IPV occurs. There may be windows during pregnancy, for example, where the fetus is more susceptible to prenatal programming such that if IPV were to take place during that time, the development of the fetus would be more likely to be altered. While several studies have suggested different sensitive periods during gestation for specific psychological outcomes in later life, research has not yet examined sensitive periods for infant SNS development or mother-infant attunement.

This study did not find evidence for or against prenatal programming of the SNS because the initially planned analyses could not be run. However, other studies have found evidence in support of prenatal programming, although the mechanisms by which the fetal SNS may be prenatally programmed are unclear since maternal SNS hormones do not pass into the placenta. It has been suggested that maternal norepinephrine restricts blood flow to the fetus, which could activate and program the fetal SNS stress response. Future research should continue to explore this potential mechanism and others to better understand prenatal programming of the SNS.

Despite the limitations of this study, it represents a contribution to the nascent literature on mother-infant physiological attunement in IPV-exposed dyads and may help to inform the methodology used in future research. Previous research has examined physiological attunement via the HPA axis in IPV-affected mother-infant pairs, but this is the first study to examine attunement in this population using the SNS. Moreover, it is also one of the first to address the issues of multiple definitions of attunement and multiple analytical approaches that muddle the physiological attunement literature. Although further research is needed to examine the potential

differential effects of timing and chronicity of IPV exposure on mother-infant attunement, this is one of the first studies to propose and attempt to do so.

## **APPENDICES**

## **APPENDIX A**

### **TABLES**



**Table 1.** sAA Means and Standard Deviations for Mothers and Infants

	<b>Total</b>	<b>No/Some IPV</b>	<b>Chronic IPV</b>
Mother sAA Time 1	7.253 (3.82)	8.036 (4.10)	6.216 (3.15)
Mother sAA Time 2	7.248 (3.77)	8.065 (3.86)	6.179 (3.40)
Mother sAA Time 3	7.345 (3.78)	7.989 (3.71)	6.494 (3.76)
Infant sAA Time 1	6.200 (2.49)	6.011 (2.68)	6.457 (2.19)
Infant sAA Time 2	6.29 (2.58)	6.110 (2.64)	6.532 (2.49)
Infant sAA Time 3	6.375 (2.55)	6.213 (2.67)	6.582 (2.37)

**Table 2.** Fixed Effects of the Correlated Growth Model

	<b>Role: Mother</b>		<b>Role: Infant</b>	
	<b>Estimate</b>	<b><i>t</i></b>	<b>Estimate</b>	<b><i>t</i></b>
<b>Intercept</b>	7.156**	28.500	6.268**	35.030
<b>IPV Status</b>	-.844**	-3.360	.213	1.190
<b>Time</b>	.061	.580	.055	1.050
<b>IPV x Time</b>	.054	.510	-.018	-.350

\*\* $p < .001$

**Table 3.** Random Effects of the Correlated Growth Model

	No/Some IPV		Chronic IPV	
	Estimate	Z-Value	Estimate	Z-Value
<b>Maternal Intercept Variance</b>	12.849**	6.81	8.764**	5.79
<b>Infant Intercept Variance</b>	6.194**	6.84	4.771**	5.92
<b>Maternal Slope Variance</b>	.470*	2.03	1.344**	3.41
<b>Infant Slope Variance</b>	.078	1.08	.132	1.51
<b>Maternal/Infant Intercept Covariance</b>	2.891*	2.98	1.605*	2.00
<b>Maternal/Infant Slope Covariance</b>	.023	.24	.177	1.34

\*\* $p < .001$ , \* $p < .05$

**Table 4.** Fixed Effects of the Cross-Lagged Analysis

	<b>Role: Mother</b>		<b>Role: Infant</b>	
	<b>Estimate</b>	<b><i>t</i></b>	<b>Estimate</b>	<b><i>t</i></b>
<b>Role</b>	6.958**	57.144	6.778**	96.932
<b>Actor Previous sAA x Role</b>	.824**	24.614	.895**	31.875
<b>Partner Previous sAA x Role</b>	.0684	1.423	-.009	-.459
<b>IPV x Role</b>	-.097	-.798	.0004	.006
<b>Actor Previous sAA x IPV x Role</b>	.001	.028	-.008	-.299
<b>Partner Previous sAA x IPV x Role</b>	.033	.688	-.013	-.650

\*\* $p < .001$ , \* $p < .05$

**Table 5.** Fixed and Random Effects for Maternal sAA Predicting Infant sAA

	<b>Estimate</b>	<b>Standard Error</b>	<b><i>df</i></b>	<b><i>t</i></b>
<b>Intercept</b>	6.272**	.174	170.815	35.986
<b>Maternal sAA</b>	.090*	.033	127.145	2.747
<b>IPV</b>	.257	.174	170.815	1.478
<b>Maternal sAA x IPV</b>	-.021	.032	127.145	-.644
<b>Intercept Variance</b>	4.609**	.577	-	-
<b>Maternal sAA Variance</b>	.039*	.014	-	-

\*\* $p < .001$ , \* $p < .05$

Table 6: Fixed Effects of the Correlated Growth Model

	Role: Mother		Role: Infant	
	Estimate	<i>t</i>	Estimate	<i>t</i>
<b>Intercept</b>	7.150**	27.433	6.287**	34.311
<b>IPV</b>	-.825*	-3.166	.193	1.053
<b>Time</b>	.059	.570	.058	1.110
<b>IPV x Time</b>	.065	.631	-.021	-.409
<b>Maternal Warmth</b>	.025	.847	-.024	-1.152
<b>Time x Maternal Warmth</b>	.013	1.123	-.005	-.803
<b>IPV x Maternal Warmth</b>	-.008	-.264	.025	1.216
<b>IPV x Time x Maternal Warmth</b>	-.003	-.248	.004	.597

\*\* $p < .001$ , \* $p < .05$

**Table 7.** Fixed Effects for the Cross-Lagged Analysis

	<b>Role: Mother</b>		<b>Role: Infant</b>	
	<b>Estimate</b>	<b><i>t</i></b>	<b>Estimate</b>	<b><i>t</i></b>
<b>Intercept</b>	6.976**	56.490	6.783**	95.578
<b>Actor Previous sAA</b>	.821**	23.299	.906**	30.017
<b>Partner Previous sAA</b>	.079	1.544	-.010	-.475
<b>IPV</b>	-.130	-1.050	.005	.071
<b>Maternal Warmth</b>	.021	1.470	-.008	-.952
<b>Actor Previous sAA x IPV</b>	-.003	-.074	.015	.511
<b>Partner Previous sAA x IPV</b>	.018	.360	-.023	-1.142
<b>Actor Previous sAA x Maternal Warmth</b>	-.001	-2.34	.003	1.040
<b>Partner Previous sAA x Maternal Warmth</b>	.007	1.265	-.003	-1.347
<b>Maternal Warmth x IPV</b>	-.016	-1.141	.009	1.122
<b>Actor Previous sAA x Maternal Warmth x IPV</b>	.004	1.168	.003	1.080
<b>Partner Previous sAA x Maternal Warmth x Postnatal IPV</b>	-.005	-.844	-.002	-.745

\*\* $p < .001$

**Table 8.** Fixed Effects for Maternal sAA, Maternal Warmth, and IPV as Predictors

	<b>Estimate</b>	<b><i>t</i></b>
<b>Intercept</b>	6.286**	35.955
<b>Maternal sAA</b>	.102*	3.145
<b>Maternal Warmth</b>	-.018	-.922
<b>IPV</b>	.244	1.395
<b>Maternal sAA x Maternal Warmth</b>	.003	.936
<b>Maternal sAA x IPV</b>	-.014	-.433
<b>Maternal Warmth x IPV</b>	.023	1.184
<b>Maternal sAA x Maternal Warmth x IPV</b>	.006	1.888

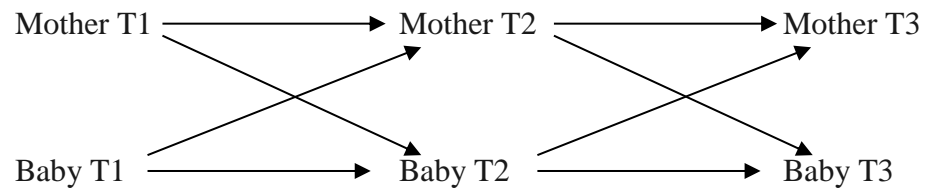
\*\* $p < .001$ , \* $p < .05$



## **APPENDIX B**

### **FIGURES**

**Figure 1.** Cross-Lagged Model



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