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Maternal Trauma Experience on Infant Cortisol Reactivity at 12 months

Michelle Anne Gilchrist
DePaul University, mgilchr2@gmail.com

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Michelle A. Gilchrist

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Thesis Committee

Cecilia Martinez-Torteya, Ph.D. Chairperson

Kathryn Grant, Ph.D.

To Nana, who always inspired me to never give up.

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Biography

The author was born in Rochester, New York. In 2007, she received her Bachelor of Art's degree in Psychology from the University of Rochester. She began her doctoral work in 2014 in the Clinical/Child Psychology Program at DePaul University, Chicago, Illinois. She began her practicum experience at DePaul Family and Community services in 2015 where she currently continues her clinical training.

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Abstract

Intimate Partner Violence (IPV) is a major public health concern in the United States (US). One third of women in the US have experienced rape, physical assault or stalking by a former or current partner (Black et al., 2011). Evidence suggests that women experience increased risk for IPV during the perinatal period and exposure to IPV during and after pregnancy increases risk for adverse physical and mental health outcomes for victims. The "fetal programming" hypothesis proposes that prenatal experiences are also particularly impactful for *offspring* development in the short and long term; prenatal poor nutrition and stress have been linked to negative health outcomes for infants and children, including temperamental difficulties, decreased emotion regulation, and psychopathology (Bergman, Sarkar, O'Connor, Modi & Glover, 2007; Davis et al., 2004; Huizink, de Medina, Mulder, Visser, & Buitelaar, 2002). The Hypothalamic Pituitary Adrenal (HPA) has been identified as a potential mechanism linking prenatal experience and maladaptive outcomes later on. However, there is a paucity of information about the influence of prenatal IPV, a common, often chronic, and highly detrimental stressor, on infant HPA axis development. The current study explores the influence of prenatal IPV exposure on the infant stress response using salivary cortisol, a hormonal indicator of the HPA axis. This study also evaluates the interactions between prenatal IPV exposure, and common co-occurring risk factors, including maternal childhood maltreatment history, and maternal postpartum depressive and PTSD symptoms. A more comprehensive understanding of the biopsychosocial concomitants of prenatal exposure to IPV is needed to inform future prevention and intervention efforts early in life.

Introduction

In the United States, approximately 28.8% of women (34,273,000) report being impacted by Intimate Partner Violence (IPV) at some point in their lifetime. IPV includes the physical, sexual, or psychological assault, threat of assault, or stalking of an individual by a current or previous partner (Black et al., 2011). One in three (35.6%) women in the US reported experiencing physical violence and nearly 1 in 10 (9.4%) reported being raped by an intimate partner over the course of their lifetime (Black et al., 2011). Between 1995 and 2003, the estimated cost of IPV increased from $5.8 billion to approximately $8.3 billion, with money spent on medical care, crisis intervention, and loss of time at work/school (Black, et al., 2011; Centers for Disease Control and Prevention [CDC], 2012). Thus, IPV is a major public health concern in the US.

Women may be particularly vulnerable to IPV during the perinatal period. Violence during pregnancy appears to be highly common with the prevalence ranging from 4 – 8 % (Chu, Goodwin, D'Angel, 2010; Saltzman, Johnson, Gilbert, & Goodwin, 2003; Sharps, Laughon, & Giangrande, 2007). Women who experience IPV during pregnancy are at greater risk of experiencing adverse physical health issues (Silverman, Decker, Reed, & Raj, 2006) as well as mental health problems like anxiety and depression (Ludermir, Lewis, Valongueiro, Araujo, & Araya, 2010; Talley, Heitkemper, Chicz-Demet, & Sandman, 2006). Experiencing IPV has also been associated with preterm births and low birth weight babies (Shah & Shah, 2010), and it places children at greater risk for neonatal and infant mortality and morbidity (Ackerson & Subramanian, 2009; Saifuddin, Koenig, & Stephenson, 2006).

There is a wealth of animal and human research showing that prenatal experience, specifically, stress, may have long-term implications on fetal and infant health well into

adulthood (for review see Glover, O'Connor, & O'Donnell, 2010). Furthermore, many studies have established a link between the experience of psychological stress and changes in physiological markers during pregnancy with a specific focus on psychoneuroendocrine pathways (Latendresse, 2009; Kramer et al., 2009; Rich-Edwards & Gizzard, 2005; Wadhwa, 2005), proposing that dysregulation of the Hypothalamic-Pituitary Adrenal (HPA) axis leads to offspring maladaptation. However, few empirical studies have investigated physiological outcomes of infants exposed to IPV *in utero*, a common, chronic, and noxious stressor. Notably, not all children exposed to traumatic stress or IPV during pregnancy are destined to have poor developmental trajectories. Understanding the contextual factors that may accentuate or attenuate prenatal risk is as equally as important as understanding the mechanisms behind these associations. Few existing studies of prenatal IPV take into account contextual risk factors that are highly comorbid with IPV exposure and may compound risk for infants, such as the mother's own history of childhood maltreatment and postpartum psychological problems.

The primary purpose of this study is to advance the literature examining the biological influence of prenatal IPV and infant stress reactivity. We examined the influence of prenatal IPV exposure on the infant stress response at 12 months using salivary cortisol, a hormonal indicator of the HPA axis. We also evaluated if maternal history of child maltreatment and current psychopathology symptoms moderate the effects of prenatal IPV on infant HPA axis reactivity, such that these additional contextual risk influence the association between IPV and cortisol reactivity.

Impact of Intimate Partner Violence

General physical and mental health. Experiences of IPV are associated with a host of direct and indirect negative physical and psychological outcomes, including but not limited to

facial trauma, loss of consciousness and neurological damage (Campbell, 2002; Campbell & Lewandowski, 1997). There is a substantial literature linking IPV to increased risk of stress related chronic diseases and poor reproductive and sexual health in women (Campbell & Lewandowski, 1997; Kendall-Tackett, Marshall, & Ness, 2003). Unintended pregnancy is both a common consequence and risk factor for the perpetuation of IPV (Campbell & Lewandowski, 1997; Chu, et al., 2010), and other gynecological problems have been reported in women who experience IPV compared to non-exposed women (for review of the literature see Campbell & Lewandowski, 1997). Victims of IPV are also at an increased risk for experiencing a myriad of psychiatric conditions including depression, posttraumatic stress disorder (PTSD), substance abuse, and suicide (Campbell & Lewandowski, 1997; CDC, 2012). Women exposed to violence are three times more likely to report poor mental health compared to women who have not experienced violence (Black et al., 2011).

IPV and pregnancy: maternal and obstetric outcomes. Exposure to IPV during pregnancy has been linked with deleterious obstetric and maternal mental health outcomes in the postpartum period. Women who experience IPV during pregnancy are at an increased risk to engage in unhealthy behaviors (e.g. smoking or drug use), exhibit pregnancy complications (e.g. high blood pressure or infection) and poor birth outcomes including preterm labor, low birthweight, fetal distress, and preeclampsia (Campbell, 2002; Jasinski, 2004; Silverman et al., 2006). A meta-analysis of 30 studies found that women who experience IPV during pregnancy are 1.46 times more likely to deliver early and 1.53 times more likely to have low birth weight infants (Shah & Shah, 2010).

Consistent with more general findings of the relation between IPV and mental health symptoms, violence during pregnancy has been shown to increase maternal risk of mental health

symptoms including depression and PTSD symptoms (Campbell, 2002; Rodríguez et al., 2010). There are a small number of longitudinal studies linking prenatal abuse experiences with postnatal depression (Ansara, 2005; Ludermir, Lewis, Valongueiro, de Araujo, & Araya, 2010). For example, in a group of 210 Latina women, IPV was highly predictive of experiencing depression in the postpartum period, independent of prenatal mental health (Valentine, Rodriguez, Lapeyrouse, & Zhang, 2011). Similarly, Ludermir and colleagues (2000) reported that women who experienced psychological violence during pregnancy were more likely to endorse postnatal depression.

IPV and Offspring outcomes. IPV has also been shown to have a significant impact on the young offspring's socioemotional and physiological development. Specifically, infants whose mothers have experienced prenatal IPV have been shown to be more fearful, and show more internalizing and externalizing problems (Carpenter & Stacks, 2009; Levendosky et al., 2006; O'Campo, Caughy, & Nettles, 2010; Quinlivan & Evans, 2005). A prospective cohort study by Quinlivan & Evans (2005) found that adolescents who experienced IPV during pregnancy were more likely to rate their infants as temperamentally difficult than mothers who were not exposed to violence (Quinlivan & Evans, 2005). To our knowledge only one study has evaluated links between prenatal IPV exposure and infant cortisol reactivity. In a community sample of 182 mother infant dyads, Levendosky and colleagues (2016) reported increased cortisol reactivity and behavior problems in infants exposed to prenatal IPV at 12 months old. However, the same increases were not seen in non-exposed infants when presented with the same laboratory stressor. In this sample of infants cortisol reactivity was related to HPA axis dysregulation in the prenatal IPV exposed group. This result is consistent with the findings of another longitudinal study (Levendosky et al., 2006), which found perinatal IPV (sum of

pregnancy and postpartum experiences) predicted increased infant externalizing behaviors at 12 months old among a community sample of mothers.

In addition, IPV exposure is likely to continue after pregnancy (Harrykissoon, Rickert, & Wiemann, 2002; Sharps, et al., 2007), increasing infant risk for posttraumatic stress symptoms such as disturbances in eating and sleeping patterns (Bogat, DeJonghe, Levendosky, Davidson & von Eye, 2006; Levendosky et al., 2006). A wealth of research documents that exposure to IPV during early childhood leads to increased levels of depression, anxiety, disruptive and aggressive behaviors (Evans et al., 2008; Graham-Bermann & Levendosky, 1997; Kernic et al., 2003). Research has also found alterations in HPA axis activity among children living in households with IPV, but evidence is mixed on the specific pattern of cortisol reactivity that is associated with IPV (Hibel, 2011, Saltzman, 2005; Sturge-Apple, 2012; Towe-Goodman, 2012). Towe-Goodman et al. (2012) used latent profile analysis to determine patterns of infant behavioral and adrenocortical activity in response to interparental aggression. Seven hundred and thirty five seven-month-old children from low-income families were assessed. Results showed that infants from violent homes were more likely to show moderate signs of distress and have high cortisol reactivity than infants from non-violent homes. Similarly, in a study exploring physiological responses of children exposed to intimate partner violence, elevated baseline cortisol in children ages 5-13 who had been exposed to marital violence was observed (Saltzman, 2005). In contrast, Sturge-Apple and colleagues (2012) reported the opposite pattern. In their sample of 201 two-year-old children and their mothers, they found a blunted cortisol response to stress among children exposed to marital violence. This is consistent with the cumulative stress literature, which has noted dysregulated cortisol responses, in the face of chronic stress. Variability of cortisol levels in adults and children have been reported in response to difference in type and

frequency of stress. However, these results suggest that stress, like violent environments, is physiologically taxing and the body physiologically engages to provide balance and stability when under various amounts of pressure (Suglia, Staudenmayer, Cohen, Enlow, Rich-Edwards, & Wright, 2010).

In summary, these results highlight that IPV is a chronic and pervasive stressor that has been associated with a variety of adverse psychological, physical and behavioral outcomes for women and their infants. However, little is known about the influence of pregnancy IPV on the infant HPA axis activity, a potential mechanism of risk that may contribute to the development of behavioral and emotional problems among IPV exposed children. The key role of early HPA axis dysregulation on the development of psychopathology, associations between other indices of prenatal stress and HPA axis alterations, and links between IPV exposure during childhood and stress response alterations, suggest that this is an important avenue of inquiry.

The Hypothalamic Pituitary Adrenal Axis

The HPA axis is a major component of the neuroendocrine system that is known to control and regulate a series of bodily processes in response to stress. The activation of the HPA axis results in a cascade of events that are regulated by a complicated interaction of negative and positive feedback loops. When the organism detects a threat, the hypothalamus increases the production and secretion of corticotrophin releasing hormone (CRH) and arginine vasopressin (AVP). Elevated levels of CRH and AVP signal the pituitary gland to increase production of adrenocorticotropic hormone (ACTH) resulting in an increase of glucocorticoids (GCs, cortisol among humans) by the adrenal gland. When the threat has subsided, the production of excess cortisol stimulates a negative feedback loop allowing the organism to return to baseline levels of HPA activity (for review see Gunnar & Quevedo, 2007).

HPA Axis Dysrgulation. In conjunction with other physiological systems, cortisol plays an influential role in a series of metabolic and immunological responses including growth, reproductive function, as well as cognitive processes (Egliston, McMahon, & Austin, 2007). While the release of cortisol is advantageous in helping manage the short-term effects of stress, prolonged exposure to glucocorticoids have been suggested to have deleterious effects (Duthie & Reynolds, 2013; Lupien, King, Meaney, & McEwen, 2000). For example, the animal literature has demonstrated structural changes in the hippocampus as a result of glucocorticoid exposure (Kim & Diamond, 2002). A similar link has been reported in cognitive processes associated with hippocampal activity in humans. Lupien and colleagues (2001) reported memory and learning impairment in healthy adults when cortisol levels were artificially elevated or reduced. Taken together these results suggest that glucocorticoid activity plays a significant role in a variety of physiological processes and exposure to abnormal concentrations may lead to structural and behavioral changes. The prenatal period is a particularly sensitive time in which excess glucocorticoid exposure may have a profound impact on subsequent development.

HPA axis and pregnancy. Pregnancy is a particularly vulnerable developmental period where the rapidly growing fetus is increasingly more susceptible to changes in the uterine environment. During critical periods of fetal development, over exposure to GCs has been shown to restrict growth and cause structural changes in brain development, specifically in areas with an abundance of cortisol receptors (Egliston et al., 2006; for a review see Seckl & Holmes, 2007). To protect against high levels of glucocorticoid exposure the placenta releases the enzyme 11 β-hydroxysteroid dehydrogenase (11 β-HSD). Although this is a protective process, there is significant variability in production of 11 β-HSD and maternal and fetal cortisol levels are positively correlated, such that maternal levels can explain about 33% of the variance in fetal

cortisol (Gitau, Fisk, Teixeira, Cameron, & Glover, 2001). Thus, even small alterations in maternal cortisol production may have a significant impact on fetal glucocorticoid exposure (Egliston et al., 2006).

Prenatal Stress

Fetal programming. Prenatal experiences have been shown to have lasting effects on both physical and mental health (Barker, Eriksson, Fosen, & Osmond, 2002; Hobel, Goldstein, & Barrett, 2008). Initial observations explored the influence of malnutrition during pregnancy among women who experienced the Dutch famine of 1944. Results showed that women who were pregnant during the famine had increased risk of negative birth outcomes (i.e. lower offspring birth weight, birth length) and their offspring were more likely to experience adverse health conditions in adulthood (i.e. glucose intolerance, obstructive airway disease, obesity, and Coronary Heart Disease; Ravelli et al., 1998; Roseboom et al., 2000; for review see Roseboom, de Rooij, & Painter, 2006). Not only the physical characteristics of the fetal environment seem to be relevant to offspring's health; psychosocial stressors, such as the death of a partner or exposure to a natural disaster (Entringer, Kumsta, Hellhammer, Wadhwa, & Wüst, 2009; for review see Harville, Xiong, & Buekens, 2010; Hobel, Goldstein, & Barrett, 2008) have also been linked with similar adverse obstetric outcomes, as well as long term physiological and psychosocial problems (for review see Talge, Neal & Glover, 2007). These findings suggest that differences in the uterine environment may result in distinct patterns of offspring vulnerability to illness throughout the lifespan, providing the basis for the "fetal programing" theory (Barker et al., 2002).

Fetal programming is a concept that describes the physiological changes a fetus undergoes in response to the *in utero* environment. In order to maintain homeostasis in the face

of specific environmental characteristics (e.g., low nutrient availability, high stress), structural and functional physiological alterations occur during fetal development (for review see Talge et al., 2007). Prenatal influences may be particularly powerful because they occur during a sensitive period, when the organism is undergoing rapid change, increasing vulnerability to various environmental pathogens (Calkins & Devaskar, 2011). Moreover, environmentally induced fetal adaptations inform later adaptation to environmental demands after birth, with some research suggesting effects throughout childhood and even adulthood (Barker, Eriksson, Fosen, & Osmond, 2002; Glover, 2011; Talge, Neal, & Glover, 2007). In regards to prenatal stress and trauma, maternal HPA axis alterations have been proposed as a potent influence, leading to increased levels of fetal cortisol *in utero* exposure, and altering the fetus's physiology and ultimately their ability to regulate stress later on (Gluckman et al, 2007; Gunnar & Quevedo, 2007; O'Connor, Bergman, Sarkar, & Glover, 2013).

Impact of prenatal stress on fetal and infant development. Studies assessing perceived maternal stress, defined broadly to include general stress, distress, anxiety, and depression during pregnancy, report links with a variety of fetal, obstetric and newborn outcomes, including low fetal heart rate, obstetric complications, premature delivery, low birth weight, and small head circumference (Dancause et al., 2011; David, Hirsh, Karin, Toledo & Akselrod, 2007; Dole et al, 2003; Engel, Berkowitz, Wolff & Yehuda, 2005; Lobel et al, 2008; Loomans et al., 2012; Zhu et al., 2010). These outcomes increase the risk for infant mortality, health complications (i.e. asthma, cerebral palsy), poor academic achievement, learning disabilities, and inattention (for review see Hack, Klein, & Taylor; Mwaniki, Atieno, Lawn & Newton, 2012). In a longitudinal study of fetal development, DiPietro and colleagues (2002) found that the fetuses of women who reported more daily life stress had lower fetal heart rate

(FHR), an important indicator of fetal growth and neural integration of the autonomic nervous system (David et al.,2007; Van Leeuwen, Lange, Bettermann, Gronemeyere, & Hatzmann, 1999). Importantly, studies have demonstrated a modest stability in patterns of heart rate from the prenatal period through infancy and have linked variability in patterns of FHR to differences in activity level, motor maturity, and infant irritability (DiPietro et al., 2002; DiPietro, Bornstein, Hahn, Costigan, & Achy-Brou, 2007; DiPietro, Costigan, Pressman &, Doussard-Roosevelt, 2000). Taken together, data from these studies suggest 'maternal distress' during the prenatal period influences key aspects of fetal and newborn development, including offspring growth and central nervous system activity.

Furthermore, maternal stress during pregnancy has also been linked to a variety of negative psychosocial and socioemotional outcomes for offspring during early childhood. Infants exposed to high levels of maternal (self-reported) emotional and perceived stress, negative life events, anxiety and/or depression during pregnancy show increased fearfulness, poor attention regulation, increased behavioral reactivity to novelty, as well as increased crying and fussy behavior (Bergman, Sarkar, O'Connor, Modi & Glover, 2007; Davis et al., 2004; Huizink, de Medina, Mulder, Visser, & Buitelaar, 2002; Mohler, Parzer, Brunner, Wiebel, & Resch, 2006; Wurmser et al, 2006). This pattern remains true even after adjusting for psychosocial risk factors like postnatal maternal mood (e.g., Huizink et al., 2002). In their longitudinal study, Huizink et al. (2002) found that women with higher self-reported stress during pregnancy were more likely to report infant difficult temperament and poor attention regulation when their offspring were three and eight months old. Similarly, a study by Austin, Hadzi-Pavlovic, Leader, Saint and Parker (2005) demonstrated a link between maternal anxiety and infant difficult temperament at 4 and 6 months postpartum.

A smaller number of studies have explored the effects of exposure to a traumatic stressor during pregnancy among young children. Infants whose mothers reported objective stress following a natural disaster were more likely to have lower Bayley scores and decreased language ability than infants whose mothers did not report objective stress (King & Laplante, 2005; Laplante et al., 2004; Laplante Brunet, Schmitz, Ciampi & King, 2008). In regards to behavioral and physiological outcomes, Enlow and colleagues (2009) reported that infants exposed to prenatal traumatic stress were more distressed by a laboratory challenge procedure, experienced more physiological arousal in response to the stressor, and took longer to regulate after the stressor. Efforts to understand the negative effects of prenatal stress and trauma on infants' behavioral and emotional outcomes have focused on the HPA axis as a potential mechanism of risk.

Impact of prenatal stress on infant HPA-axis. Modification of endocrine, immune, and neurobehavioral offspring activity through alterations in the Hypothalamic-Pituitary-Adrenal (HPA) axis have been proposed as an important link between exposure to prenatal stress and temperamental, behavioral, and cognitive child outcomes (Hobel, et al., 2008; Lazinski, Shea, & Steiner, 2008; Nast, Bolten, Meinlschmidt, & Hellhammer, 2013; Wadhwa, 2005). Chronic exposure to stress during pregnancy has been linked to the dysregulation of the HPA axis, resulting in various neuroendocrine patterns (e.g., hyperactivity or hypoactivity) that may increase risk for psychosocial morbidity during childhood and adolescence (Entringer, et al., 2009; O'Connor et al., 2005; Van den Bergh, Van Calster, Puissant, & Van Huffel, 2008). For example, Huizink et al. (2002) reported that altered HPA axis activity during infancy is associated with difficult temperament and motor development in early life among children exposed to prenatal stress. Further, longitudinal links between prenatal stress, HPA axis activity,

and psychosocial problems (e.g., depression) have also been shown in samples of preadolescents and adolescents (Van den Bergh, Van Calster, Smits, Van Huffel & Lagae, 2008; Martinez-Torteya et al., 2015).

Empirical evidence supports the notion that prenatal stress leads to altered offspring HPA axis activity. Prenatal stress exposure predicts elevated baseline, stress-induced, and mean cortisol levels during infancy (Brennan et al, 2008; Hout et al., 2004). Grant and colleagues (2009) used a laboratory relational stressor to assess the link between maternal prenatal anxiety and HPA axis reactivity in a group of 7-month-old infants. They reported higher levels of cortisol reactivity among infants whose mothers reported prenatal anxiety, even when controlling for prenatal depression and current maternal psychiatric symptoms. This association is also observed later, during childhood and adolescence. A prospective study of pre-adolescents, age 10, reported exposure to IPV during pregnancy predicts higher offspring cortisol reactivity to a laboratory psychosocial stressor (Martinez-Torteya et al., 2015). Elevations in basal cortisol levels have also been linked to maternal anxiety during pregnancy among pre-adolescents and adolescents (O'Connor et al., 2005; Van den Bergh et al., 2008).

While there is a large amount of evidence suggesting prenatal stress has long-term implications on development there are several methodological issues that complicate the interpretation of findings related to the cortisol response. Disparities in how prenatal stress is defined and measured (e.g., trauma exposure, vs. self-reported stress levels, vs. depression and anxiety), how HPA activity is measured (e.g., diurnal or basal patterns vs. reactivity), and the developmental period of assessment (e.g., infancy vs. adolescence) limit the conclusions that can be drawn about the role of prenatal stress as a potential influence of the physiological stress response. In addition, there is dearth of information on how offspring outcomes vary based on

stressor characteristics (e.g., type, frequency, or duration). To address these limitations it is necessary to focus on the most common and detrimental stressors that pregnant women face, such as IPV. Also, research is needed to characterize the influence of traumatic stress on early offspring HPA activity while taking into account that many traumatic stressors (such as IPV) do not occur in a vacuum but are frequently co-occurring with additional contextual adversity.

Contextual Risk Factors Associated with IPV and HPA Reactivity.

Based on the literature it is clear that IPV does not occur in isolation. There are numerous environmental factors that could be contributing to the relation between prenatal IPV and infant cortisol reactivity at 12 months. Child maltreatment and maternal postpartum psychopathology have consistently been linked to maladaptive child outcomes and commonly co-occur with IPV (Kendall-Tackett, 2007). For example, a cross-sectional study of 188 women and their infants found that mothers reporting IPV were significantly more likely to be diagnosed with a mood disorder, specifically depression or PTSD, than women with no history of IPV (Cerulli, Talbot, Tan, & Chaudron, 2011). Additionally, history of childhood victimization is considered a significant risk factor for subsequent victimization in adulthood. Both longitudinal and epidemiological studies have demonstrated a robust link between childhood abuse and increased risk of IPV. Desai and colleagues (2002) documented that both men and women with histories of childhood abuse were at an increased risk of being victimized as adults. Specifically, women who experienced childhood physical or sexual abuse were three times more likely to be abused by a romantic partner compared to women without a child maltreatment history. Similarly, there is a robust link between childhood sexual abuse (CSA) and IPV (Coid et al., 2001) after controlling for individual contextual factors (Daigneault, Herbert, & McDuff, 2009).

Childhood maltreatment. The consequences of childhood maltreatment (CM) are profound and have been shown to have long-term deleterious effects on both psychological adjustment and physical health (for review see Norman, Byambaa, De, Butchart, Scott & Vos, 2012; Gilbert, Widom, Browne, Fergusson, Webb & Janson, 2009). Commonly, victimization in childhood has been associated with low self-esteem and increased rates of psychopathology, including depression, PTSD, eating disorders, suicide attempts, and drug and alcohol use during adulthood (Edwards, Holden, Felitti, & Anda, 2003; Heim et al., 2000; Mullen, Martin, Anderson, Romans & Herbison, 1996). Furthermore, CM may increase vulnerability to subsequent stressors. For instance, Horwitz et al. (2001) found that adults with a history of CM and subsequent life stress showed elevated levels of psychopathology, as compared to those exposed to high levels of life stress who did not have a maltreatment history. Similarly, research shows that women who experience multiple traumas generally tend to have worse psychological health outcomes and are at a higher risk for re-victimization than women who had no trauma exposure (Green et al., 2000).

Increased vulnerability to stress, reflected in abnormal HPA axis and autonomic nervous system activity, has also been identified as a potential consequence of abuse and neglect in childhood. A small body of literature links CM history with abnormal cortisol secretion in both a sample of healthy adults (Carpenter et al., 2007) and pregnant women (Bublitz & Stroud, 2012; Shea, Streiner, Fleming, Kamath, Broad, & Steiner, 2007). Moreover, Heim et al. (2000) reported lower baseline cortisol concentrations and greater HPA activity in response to a corticotrophin-releasing-factor laboratory challenge in women who had experienced child abuse as compared to women with no early life adversity. A CM history may also heighten the negative influence of pregnancy stress on child outcomes. Increased physiological distress,

reported by heart rate and respiration, has been observed in infants whose mothers had both high lifetime and perinatal trauma compared to infants whose mothers had lower levels of trauma exposure, even after controlling for current maternal psychopathology (i.e. depression and PTSD; Enlow, Kullowatz, Staudenmayer, Spasojevic, Ritz & Wright, 2009).

Intergenerational effects of child maltreatment. A growing body of research suggests that childhood maltreatment has intergenerational effects, influencing offspring adaptation via environmental and physiological mechanisms. Consistently, research reports that women with a history of CM have offspring with more temperamental and regulatory difficulties in infancy, as well as more externalizing problems during childhood (Bifulco et al., 2002; Lang, Gartstein, Rodgers, & Lebeck, 2010; Roberts, O'Connor, Dunn, & Golding, 2004; Yehuda, Bell, Bierer, & Schmeidler, 2008; Yehuda, Halligan, & Grossman, 2001).

Stress response alterations have also been reported among offspring whose mothers experienced childhood abuse (i.e. cortisol, heart rate, and respiratory response; Brand et al., 2010; Enlow et al., 2009). Brand and colleagues (2010) explored the associations between history of maternal child abuse, maternal psychopathology and cortisol levels, as well as patterns of infant HPA functioning. One hundred and twenty six women and their infants were recruited as part of a larger longitudinal study of perinatal mental health. Results showed that infants whose mothers reported childhood trauma had lower baseline cortisol levels than infants whose mothers reported no trauma. In another study, Martinez-Torteya et al., (2014) reported that abuse and neglect during childhood increased the risk of maternal postpartum depression, which in turn was associated with decreased maternal positive parenting, leading to elevated cortisol response to a laboratory stressor (the Still Face Paradigm) among 7-month-old infants. Taken

together, this evidence suggests that maternal CM may have long-term implications for offspring stress regulation, and may exacerbate the negative effects of prenatal stress exposure.

Maternal postpartum psychopathology. Maternal postpartum psychopathology (i.e. anxiety and depression) has also been shown to play a contributing role in offspring HPA axis functioning. Maternal mood, especially during the first year of life, has significant influence over the parent-child relationship, as well as infant emotional and physiological regulation. Elevated cortisol levels have been reported in infants whose mothers experience high levels of postpartum depression and anxiety (Brennan et al., 2008; Grant et al. 2009; Hout et al., 2004). Lupien and colleagues (2000) reported a strong correlation between infant cortisol in the first year and maternal depressive symptoms. Postpartum psychopathology often impairs caregiving sensitivity, increasing risk for dysregulation of the offspring HPA axis (Martinez-Torteya et al., 2014).

In addition, longitudinal studies have consistently linked maternal postpartum depression to child and adolescent HPA activity. In a sample of 285 mother infant dyads, Essex, Klein, Cho, and Kalin (2002), reported that maternal depression in infancy was the strongest predictor of child afternoon/evening baseline cortisol at four to five years old, with more postpartum depression leading to higher evening cortisol secretion. They also reported that elevated cortisol levels were predictive of mental health symptoms in their sample of children. Similarly, Halligan, Herber, Goodyer, and Murray (2004) reported elevated morning cortisol in 13-year-old adolescents whose mothers had experienced postpartum depression. The effect remained true after controlling for current psychopathology and stressful life events. In sum, postpartum depression, a frequent correlate of IPV exposure during pregnancy, is an additional stressor that may have long-term effects on the infant stress response.

PTSD is another common correlate of IPV. The prevalence of PTSD among women who experience IPV is approximately 57% (Nathanson, Shorey, Tirone, & Rhatigan, 2012). In contrast to depression, PTSD symptoms have been consistently associated with HPA axis hypoactivity among survivors. For example, Olff and colleagues (2006) reported lower plasma cortisol levels in individuals experiencing chronic PTSD compared to healthy controls. However, there is a dearth of studies that have examined the influence of maternal PTSD on infant cortisol reactivity. In one of the only studies to date, Yehuda and colleagues (2005) reported a blunted awakening response and lower bedtime cortisol in infants whose mothers indicated higher PTSD symptoms after the World Trade Center attacks, as compared to trauma-exposed women without PTSD. A second study exploring maternal PTSD symptoms and infant regulation suggested an interaction between prenatal IPV and maternal PTSD symptoms in the first year of life (Ahlfs-Dunn & Huth-Bocks, 2014). In a sample of 120 low-income mother infant dyads, maternal PTSD symptoms moderated the relation between IPV exposure and infant dysregulation at 3 months (Ahlfs-Dunn & Huth-Bocks, 2014).

In sum, research suggests that maternal postpartum depression is an important influence on offspring HPA axis regulation, and, although less well understood, the effect of maternal PTSD symptoms may influence offspring stress reactivity independently or in conjunction with environmental adversity. Thus, maternal postpartum psychopathology needs to be taken into account to obtain a contextually informed understanding of the influence of prenatal trauma exposure on infant cortisol regulation.

Rationale

Infants' prenatal experiences play an important role in the early development of the physiological stress response. Empirical findings have linked prenatal stress to a variety of

negative physical, cognitive, and social outcomes (Bergman, Sarkar, O'Connor, Modi & Glover, 2007; Davis et al., 2004; Huizink, de Medina, Mulder, Visser, & Buitelaar, 2002). The HPA axis has been proposed as a key mechanism of risk, such that prenatal experience contributes to dysregulation of the offspring stress response in the short and long-term and the observed stress response alterations can significantly contribute to psychosocial morbidity. The prenatal stress literature has rarely examined the occurrence of IPV during pregnancy, a highly frequent, chronic, and pervasive stressor that has strong negative effects of both maternal and infant physical and mental health. Only one study to date has evaluated the effect of pregnancy IPV on infant cortisol reactivity (Levendosky et al., 2016), however other studies document HPA alterations among youth exposed to IPV early in life that can be characterized by hypo- and hyper-activity. The prenatal period is particularly sensitive to the development of the infant stress response. Prenatal stress, like IPV, has been proposed to have a "programming" effect making the fetus susceptible to later stress (Levendosky et al., 2016; Talge et al., 2007). Prolonged exposure to environmental stressors impacts the body's ability to regulate to future insults (McEwen, 1998; Braveman et al., 2009). To better understand the link between prenatal IPV and infants' stress response alterations, it is also important to examine relevant contextual factors that may moderate this relationship. History of child maltreatment and maternal postpartum psychopathology are good candidates for moderators of the relation between IPV and infant stress reactivity, because they commonly occur in conjunction with family violence and previous research shows they may potentiate the negative effects of trauma exposure (Enlow et al., 2009; Ahlfs-Dunn & Huth-Bocks, 2014).

The current study examined the effect of prenatal IPV exposure on the infant stress response at 12 months, using salivary cortisol. Potential moderation effects of maternal history

of maltreatment during childhood and postpartum psychopathology were also evaluated. Clinically, it is important to better understand how early negative life experiences, like prenatal IPV, may have long lasting influences on children. This understanding may help inform prevention and intervention efforts targeted at IPV survivors and their children. The ability to identify and provide resources early on may diminish the impact of stressful experiences on child development, addressing the consequences of prenatal risk before they become entrenched.

Statement of Hypotheses

Research Question I. Is IPV exposure during pregnancy associated with infant HPA axis response to stressors?

Hypothesis I. There will be a positive relationship between IPV exposure during pregnancy and infant cortisol reactivity, such that higher prenatal IPV exposure will predict higher cortisol reactivity.

Research Question II. How does exposure to IPV during pregnancy influence infant HPA axis activity in conjunction with frequently co-occurring risk factors, including maternal childhood maltreatment history and postpartum psychopathology?

Hypothesis II. Maternal CM history will moderate the association between prenatal IPV exposure and infant cortisol reactivity, such that CM history will strengthen the effect of IPV exposure on infant cortisol reactivity. Among infants with high maternal CM history, the positive association between IPV exposure and cortisol reactivity will be much stronger than among infants without maternal CM history.

Hypothesis III. The association between prenatal IPV exposure and infant cortisol reactivity will be moderated by maternal postpartum depressive symptoms, such that depressive symptoms will strengthen the effect of IPV exposure on infant cortisol reactivity. Among infants

with high maternal postpartum depressive symptoms, the positive association between IPV exposure and cortisol reactivity will be stronger, whereas this relationship will be weaker among infants of women without postpartum depression.

Hypothesis IV. The association between prenatal IPV exposure and infant cortisol reactivity will be moderated by maternal postpartum PTSD symptoms, such that more PTSD symptoms will reverse the effect of IPV exposure on infant cortisol reactivity. Among infants with high maternal postpartum PTSD symptoms, IPV exposure will have a negative association with cortisol reactivity, reflecting a blunted HPA axis response to stressors. Among infants of women without postpartum PTSD, IPV exposure will have a positive association with cortisol reactivity, similar to that suggested in Hypothesis 1.

Method

Research Participants

A community sample of 102 mother–infant dyads were recruited as part of a larger cross-sectional study exploring the effects of intimate partner violence on relational, behavioral, and physiological infant outcomes. Participants were recruited from community agencies (e.g. Women Infant Child (WIC) centers, laundromats, public libraries, daycare centers, and pediatricians' offices), in Chicago Illinois using flyers and brochures targeting women who may or may not have experienced prenatal IPV. Mothers were eligible to participate if they were between the ages of 18 and 40 years, had no history of schizophrenia, and if they had full custody of infants between the ages of 11 to 14 months old with no significant medical concerns (i.e., no birth defects, serious medical conditions, or developmental delays).

Procedures and Materials

Interested participants completed a brief phone screen with trained undergraduate research assistants to determine eligibility. Women received a description of the study protocol during the phone call. If interested, mothers and their infants were scheduled to complete one in-person session, lasting two and a half to three hours, at DePaul University when their infant was 11–14 months old. During the in-person assessment, mothers signed informed consent documents after a graduate student interviewer advised her of study procedures in detail. Consent was completed outside the interview room, as the dyadic tasks performed during the assessment required the baby to be introduced to a completely novel environment. After consent is provided, the mother and infant were escorted to the interview room, which was furnished with age-appropriate materials and toys. The mother was instructed to engage in a 5-minute free-play task with her infant. Immediately after, an infant baseline saliva sample was collected prior to the dyad beginning a separation and reunion task, the Strange Situation Task (SST; described in detail below). If infants became too distressed by the task, mothers were allowed to discontinue and begin the next activity. Five saliva samples were collected after the SST—10-, 20-, 30-, 40-, and 50-minutes after the task was completed. Finally, the mothers filled out a series of paper-based self-report measures on demographics, maternal lifetime and pregnancy exposure to trauma and violence, parenting style, infant temperament, infant health and maternal current mental health status, and resilience characteristics. Participants were given the opportunity to fill out the questionnaires on their own or complete the questions with assistance of the interviewer (e.g., read aloud), while a research assistant is nearby playing with the infant. Participants were told that they could skip questions they feel uncomfortable answering and can discontinue participation at any point.

Upon completion of the interview, participants received $100 in financial remuneration and a small toy for their infants. Based on answers to self-report questions, child maltreatment, current physical or sexual IPV exposure, and/or suicidal/homicidal ideation were assessed and additional resources were provided (i.e. referrals to Child Protective Services, services for IPV victims, or mental health services). Women who expressed high levels of distress or need for mental health services were also referred to appropriate agencies in their communities.

Measures

Dependent Variables: Stress Challenge and Biological Sample Collection.

Stress Challenge. Mother–infant dyads completed the Strange Situation Task (Ainsworth & Bell, 1970). This task includes a series of three minute episodes during which 1) the mother interacts with her infant, 2) a "friendly stranger" joins them and attempts to play with the infant, 3) the mother exits the room leaving the infant with a stranger and then alone, and 4) the mother-infant dyad is reunited. The room is equipped with a one way mirror where participants was able to see their infants at all times. If the infant became too distressed during the separations, that specific episode was cut short and the next episode is started, allowing the infant to be soothed either by the "friendly stranger" or his/her mother. Infant and mother behavior during the separations and reunions was videotaped and later coded by a clinical psychologist who was trained to classify dyads as secure, insecure, or disorganized. In the present study, this task constitutes the stressor that is expected to mobilize a cortisol response. The SST is the most commonly used separation paradigm assessing the infant stress response (Gunnar, Talge, & Herrera, 2009). It has been shown to consistently induce increases of coritsol in infants between 9 and18 months old (Gunnar, Larson, Hertsgaard, Harris, & Brodersen, 1992; Goldberg et al., 2003; Van Bakel & Riksen-Walraven, 2004).

Cortisol collection. Infant cortisol was assessed as a biological marker of the HPA axis stress response system using saliva samples. The measurement of cortisol through saliva has been shown to be advantageous in working with difficult populations where alternate forms of sample collection, like blood serum or urine, are not possible. Reportedly, salivary cortisol levels are highly correlated with serum cortisol levels (Dorn, Lucke, Loucks, & Berga, 2007; Gozansky, Lynn, Laudenslager & Kohrt, 2005). Saliva was collected using cotton swabs (i.e., Salimetrics Children's Swab) a non-invasive, safe, and well validated (for a review see Levine, Zagoory-Sharon, Feldman, Lewis, & Weller, 2007) sampling technique.

The validity and reliability of cortisol collection in infants has received much attention. Variation in infant stress response as measured by cortisol concentrations is influenced by sample collection method, sleep/wake cycles and feeding patterns, and individual variation in routine (Egliston et al., 2007). Mothers were asked not to feed their infants 60 minutes prior to the start of the visit and throughout the first half of the study protocol to address some of these confounds. If necessary, graham crackers were provided to hungry infants, to eat after the third post-SST sample. The infant's mouth was rinsed with water prior to the next saliva collection. This information was recorded and controlled for in statistical analyses. Maternal report about infant medication use and general health in the past 48 hours, as well as information about when the infant last ate and slept, was recorded and controlled for in statistical analysis, as they have been known to influence cortisol values (Egliston, McMahon, & Austin, 2007; Granger, Hibel, Fortunato, & Kapelewski, 2009). Among infants, resistance to sample collection, inadequate sample volume, and sample contamination can be potential barriers for analysis. To prevent sample attrition caregivers were enlisted to help if infants were to become too fussy during saliva collection (for review see Egliston et al., 2007).

The timing of saliva samples was designed to capture changes from baseline to peak cortisol reactivity and recovery. Prior studies have shown this peak to occur between 15 and 40 minutes following a stressor, depending on infant age (Egliston et al., 2007). The baseline sample was collected post-consent and free play, but prior to the administration of the separation and reunion task. In general, this sample was collected 20 to 30 minutes after arrival to the laboratory. Five consecutive samples were collected every 10 minutes (10, 20, 30, 40, 50 minutes) following the termination of the SST.

Following the interview, samples were stored in a locked -20°C freezer located in the project office until analyzed. For analysis, saliva samples are thawed and centrifuged at 300 rpm for 15 minutes. A commercially available assay with detection range .003 to 3.00 ug/dl and low cross-hormone reactivity (Salimetrics, LLC) was used to determine cortisol concentrations. Samples were assayed in duplicate using Enzyme Linked Immunosorbent Assays (ELISA) at DePaul University (31.66%), the Salimetrics Saliva laboratory (58.29%) and at both laboratories (10.06%) to determine between lab reliability. 11.92% of samples could only be assayed in singleton due to insufficient saliva quantity. Intra-assay (for those in duplicate) coefficients of variability were low (9.11% for DePaul laboratory and 4.36% for Salimetrics laboratory), suggesting excellent reliability.

For the 54 samples that were analyzed at both laboratories, the values provided by Salimetrics were used, except when sample volume was not enough to obtain values for all samples provided by one infant; in that case, the DePaul values were used. To ensure that cortisol levels were equivalent across laboratories, a conversion was provided by the Salimetrics laboratory and applied to all scores for samples assayed at DePaul: (SalimetricsMean = -.014 +

.699(DePaulMean)). Following this conversion, there were no significant differences in mean cortisol scores between labs.

Area under the curve (AUC_G) formula was used to assess the change in infant cortisol over the course of the visit. AUC_G is commonly used to assess neuroendocrine variables (i.e. hormone secretion) over time. It takes into account both change over time and intensity of change. The formula used was 'Area under the curve with respect to the ground' (AUC_G):

$$AUC_G = \sum_{i-1}^{n-1} [t_i * (m_{(i+1)} + m_i)/2]$$

where n equals the number of samples, t_i represents time between measurement, and m_i symbolizing the individual sample marker (Gordis, Granger, Susman, Trickett, 2006; Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003).

Predictor Variables: Maternal Psychosocial Stressors.

Conflicts Tactics Scale **(**CTS2; Straus, Hamby, Boney-McCoy, & Sugarman, 1996). The CTS2 is a 39-item measure designed to assess strategies utilized by a couple during conflict resolution in the past year. It utilizes a 7-point Likert scale, ranging from 0 ("never") to 6 ("more than 20 times") with items like: "My partner punched or hit me with something that could hurt", "My partner used threats to make me have sex", and "My partner suggested a compromise to a disagreement." Participants are asked to rate the frequency in which they experience specific behaviors associated with conflict resolution, both during and following their pregnancy. Postpartum CTS2 was used as a covariate to statistically control for the effects of current relationship conflict.

Items were summed and scored on the following subscales: negotiation, psychological aggression, physical assault, injury and sexual coercion. A Total Abuse Score was generated by

summing the psychological aggression, physical assault, and sexual coercion subscales, with potential scores ranging from 0 to 189. The current study modified the parameters of time in the directions of CTS2 from "in the past year" to "during your pregnancy" and "since your baby's birth". The adaptation of instruction, specifically in regards to the timeframe of the experience, has been done successfully in numerous studies to reflect the needs of the research question (Martin, Beaumont, & Kupper, 2003; Martin, Harris-Britt, Li, Moracco, Kupper & Campbell, 2004).

The CTS2 has been shown to have good internal consistency; psychological aggression $\alpha = .79$, physical assault $\alpha = .86$, sexual coercion $\alpha = .87$, and injury $\alpha = .95$. In addition, an initial sample of female college students provided evidence for strong construct and discriminant validity (Straus et al., 1996). Similar results were reported in several high risk samples including a sample of incarcerated women and women with postpartum depression (Jones, Ji, Beck & Beck, 2002; Newton, Connelly & Landsverk, 2001). Finally, there are reports of strong test-retest reliability. A study by Vega and O'Leary (2007), showed stability of reporting in psychological aggression ($r = .69$), physical assault ($r = .76$), injury ($r = .70$), and negotiation ($r = 0.60$) and moderate association in sexual coercion ($r = 0.30$) in a sample of men enrolled in a domestic violence intervention program.

Childhood Trauma Questionnaire (CTQ; Bernstein & Fink, 1998). The CTQ is a commonly used retrospective self-report measure of childhood abuse and neglect. Consisting of 28-items, the CTQ assesses the frequency of life events prior to the age of 18. Sample items include, "I didn't have enough to eat," "I got hit so hard by someone in my family that I had to see a doctor or go to the hospital," and "My family was a source of strength and support." Participants were asked to respond on a 5-point Likert scale of Never True, Rarely True,

Sometimes True, Often True, and Very Often True. Items yield Physical Abuse, Sexual Abuse, Emotional Abuse, Emotional Neglect, and Physical Neglect scales, as well as a 3-item minimization scale to detect false-negative trauma experiences. Items from all five subscales were summed (after reverse coding for items that describe positive caregiving behaviors) to obtain a Total CM score, ranging from 25 to 125, higher scores indicating maltreatment severity.

The CTQ has been validated in a myriad of general and clinical populations, varying in gender, ethnicity, and socioeconomic status (Bernstein & Fink, 1998). Satisfactory validity was reported of each subscale, ranging from .66 – .92. The CTQ has also shown stability across assessment, with test-retest reliability ranging from .79 - .86 over a 4-month period. Similarly, the CTQ has been shown to have high sensitivity and specificity. It has been highly correlated with other self-report and semi-structured interviews assessing CM including the Childhood Trauma Interview and the Evaluation of Lifetime Stressors, suggesting high convergent and discriminant validity. Finally, the CTQ has been commonly used to assess experience of abuse and neglect in pregnant women (e.g. Lang, Rodgers, & Lebeck, 2006; Madigan, Vaillancourt, McKibbon, & Benoit, 2012).

Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). The CES-D is a 20-item self-report measure designed to assess current depressive symptomatology. Participants were asked to report how often during the past week they experienced common behaviors or feelings of depression characterized by sadness, lack of interest, hopeless attitude about the future, as well as appetite, sleep, or concentration problems. Sample questions include: “I felt depressed,” “I did not feel like eating; my appetite was poor,” or “I thought my life had been a failure.” Interviewees rated their symptoms using a 4-point Likert scale: “Rarely or none of the time (less than 1 day),” “Some or a little of the time (1–2 days),” “Occasionally or a

moderate amount of time (3–4 days)," or "Most or all the time (5–7 days)." Several items are positively worded to minimize the effects of report bias, and are reverse coded. A total score is obtained by summing all items; scores range from 0-60 with higher scores indicating more severe symptomatology. Scores $\geq$16 are considered clinically significant.

The CES-D is considered to be a highly reliable and valid measure in both general and clinical populations. It has been tested across the life span, in ethnically and medically diverse populations and is commonly used in perinatal stress and IPV literature to assess depressive symptoms (DiPietro, Novak, Costigan, Atella, & Reusing, 2006; Hann, Winter, & Jacobsen, 1999; Lang, Stein, Kennedy, & Foy, 2004; Nguyen, Kitner-Triolo, Evans, & Zonderman, 2004). Internal consistency ranges from α = .85 - .90 (Hann et al.,1999; Radloff, 1977). Modest test-retest reliability has been reported. In a survey by Radloff (1977), test-retest reliability over a 2- to 8 week period ranged from r = 0.51 - 0.67, and was r = .32 - .54 over 3- to 12-months. Finally, the CES-D is highly correlated with several other self-report measures of depression suggesting strong convergent validity (Milette, Hudson, Baron, & Thombs, 2010).

Post Traumatic Stress Disorder Checklist – Civilian Version (PCL-C; Weathers, Litz, Herman, Huska, & Keane, 1994). The PCL-C is a 17-item self-report questionnaire assessing current posttraumatic stress symptoms, as assessed by the DSM-IV (American Psychiatric Association, 2000). Women answered each item on a 5-point Likert scale, ranging from 1 ("Not at all") to 5 ("Extremely"), based on their experiences in the past month. Example of items include "Repeated, disturbing memories, thoughts, or images of a stressful experience from the past?" and "Feeling emotionally numb or being unable to have loving feelings for those close to you?" Scores were summed and range from 0-68, higher scores indicating more severe symptomology.

The PCL-C has been shown to have strong psychometric properties in both general and clinical populations. It has excellent internal consistency, $\alpha = .94$, and moderate test- retest reliability 2 weeks following the original assessment, $r = .66$. In addition, the PCL-C has been shown to have strong convergent and discriminant validity (Conybeare, Behar, Solomon, Newman, & Borkovec, 2012; Ruggiero, Del Ben, Scotti, & Rabaliais, 2003).

Covariates: Individual and Contextual Stressors.

Demographics: Education. Socioeconomic status and poverty are chronic stressors that have been associated with a variety of adverse psychosocial outcomes and maladaptive stress responses (Evans & Kim, 2007; Lupien, King, Meaney, & McEwen, 2001). Income and education are commonly used as indicators of both poverty and socioeconomic status. Low-income women are at a disproportionately high risk of experiencing partner conflict (Cunradi, Caetano, Schafer, 2002; Jewkes, 2002). Due to poor question construction and inconsistent response patterns income was not used as a covariate. Rather, self-reported education status was used to control for the influence of socioeconomic stress on the relationship between prenatal experience and cortisol reactivity.

Life Stressor Checklist-Revised (LSC-R; Wolfe, Kimerling, Brown, Chrestman, & Levin, 1996). This questionnaire consists of 30 items assessing history of trauma exposure as well as other stressful life events. The LSC-R asks about a variety of traumatic experiences including exposure to natural disasters, accidents, unexpected loss of a loved one, physical or sexual assault, and other potentially traumatic events.

The measure initially asks respondents to provide a dichotomous response of “yes” or “no” indicating if an event had ever occurred. If the interview endorses an even, they were then prompted to respond to additional follow-up questions regarding age at the time of the event,

duration, and how much the event had affected the individual's life (specifically feelings of horror, hopelessness, or distress). The measure was adapted for the study adding a question asking if the indicated event occurred during the participant's pregnancy. In this study the LSC-R was used to control for additional trauma history, by calculating the sum of traumatic events or victimizations across the participants' lifetime.

The LSC-R demonstrates good construct and criterion validity. The LSC-R shows good face validity as it contains items defined as stressful or traumatic experiences (Criterion A) by the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2000) as well as other daily life hassles. Furthermore, the LSC-R has consistently predicted PTSD symptoms across several diverse populations including women who have experienced IPV and other traumatic experiences related to partner violence (Schumacher et al., 2010). In addition, test-retest reliability is adequate (McHugo et al., 2005).

Statistical Analysis

Preliminary Analyses

Testing the statistical assumptions of regression analyses. Data was assessed to verify that all necessary assumptions are met prior to conducting the study's primary analyses. A visual representation of the data was created to assess for normality and identify outliers. In addition, skewness and kurtosis were used to verify normality and standard methods of data transformation were used to address problems (Field, 2014). Scatterplots were created to visually assess linearity and heteroscedasticity. A Levene's test was completed to assess homogeneity of variance (Field, 2014). Finally, multicollinearity was evaluated to ensure that variables are independent from one another. The Variance Inflation Factor (VIF) was used to assess collinearity between variables (Field, 2013).

Missing data. Little's MCAR test was used to assess patterns of missing data, Missing At Random (MAR) or Missing Completely At Random (MCAR). The Expectation Maximization (EM) imputation was used to handle missingness. EM imputation uses a function to estimate values for missing data based on patterns generated from non-missing values and is superior to listwise deletion methods because it yields less biased estimates (Widaman, 2006).

Hypothesis testing. The primary focus of this study is to evaluate the effect of exposure to IPV during pregnancy on infant cortisol reactivity. The mother's history of abuse during her childhood and postpartum mental health symptomatology (i.e. depression and posttraumatic stress) were tested as possible moderators of the relation between prenatal IPV and infant cortisol reactivity. Linear regressions were used to test each of these relations. To reduce Type I error, the Bonferroni correction was be used to assess significance where α is divided by the number of comparisons (k) (Field, 2013):

$$P_{Crit} = \frac{\propto}{k}$$

$$P_{Crit} = \frac{0.05}{4} = .0125$$

Hypothesis I predicts a main effect between exposure to prenatal IPV and infant cortisol reactivity. To examine whether participants' reports of prenatal IPV predict infant cortisol reactivity, a multiple linear regression was estimated controlling for maternal age and education, additional maternal trauma history, and time of day of cortisol collection.

Hypothesis II predicts that the relation between IPV and infant cortisol reactivity will be moderated by maternal childhood maltreatment. A two-step linear regression was estimated, including the covariates and the main effects of IPV and maternal CM history on the first step of the model. To examine the possible moderation effect, an interaction term was created (product

of centered scores of the continuously scaled pregnancy IPV and CM variables) and included in the second step of the model. Statistically significant interaction terms ($p < .0125$) were plotted following Aiken and West (1991), illustrating the effect of pregnancy IPV on infant cortisol reactivity one SD above, at the mean, and one SD below mean CM history levels (Aiken & West, 1991; Holmbeck, 1997).

Hypothesis III and IV predict that maternal mental health symptomatology (i.e. depression and posttraumatic stress) will each moderate the relation between prenatal IPV and infant cortisol reactivity. An equivalent analysis was run as described in Hypothesis II.

Power analysis. Previous studies investigating IPV and infant morbidity have found a medium effect size for differences in infant outcomes (Manzolli, Nunes, Schmidt, & Ferri, 2012). A similar effect size has been reported in studies between prenatal stress and the infant behavioral outcomes (Goodman, Rouse, Long, Ji, & Brand, 2011). An a priori power analysis was conducted using G*Power Software. G*Power revealed that 86 participants are needed for excellent power to detect a medium effect size $f^2 = .015$, with $\alpha = .0125$, using one predictor (or effect) of interest and up to six additional predictors (Faul, Erdfelder, Lang, & Buchner, 2007).

Results

Data Processing

Outliers (values that were $\pm$ 3 SDs from the mean) were identified and winsorized to preserve rank order. Skewness and kurtosis statistics were used to evaluate the distribution of the data. Values exceeding $\pm$ 3 were considered problematic and those variables (cortisol, prenatal IPV, postnatal IPV) were log transformed (Field, 2013). Following log-transformations, these variables had a normal distribution (Prenatal IPV Skewness = 0.052, Kurtosis = -1.344, Postpartum IPV (Skewness = 0.199, Kurtosis = -1.409).

Missing Data/Imputation

Item and questionnaire level missingness on self-report measures was rare (<1% of all data points) and replaced with the sample mean for each item (Roth, Switzer, & Switzer, 1999). Sum scores were computed using all the available data and the mean imputed values. On the other hand, twelve percent of cortisol data was missing due to lack of saliva for assaying. This was primarily caused by low saliva production or infant distress during collection; Table 1 details how many samples were available at each time point.

Table 1. *Descriptive Statistics of Raw Cortisol Data*

Cortisol Sample	*N*	Missing	Mean	SD
Baseline (T1)	93	9	.190	.178
10 minutes (T2)	90	12	.226	.238
20 minutes (T3)	94	8	.240	.233
30 minutes (T4)	93	9	.240	.245
40 minutes (T5)	88	14	.223	.207
50 minutes (T6)	79	23	.229	.245

Patterns of cortisol missingness were assessed using Little's MCAR test, which revealed that the data was Missing Completely at Random ($X^2 = 465.788$, $p > .05$). Thus, questionnaire and cortisol missing data was imputed using the Expectation Maximization (EM) method. EM imputation has been shown to produce less biased estimates than other imputation techniques or listwise deletion (Widaman, 2006). While the imputed data was used for the analysis presented below, regressions were also conducted with the non-imputed data sets to evaluate whether similar results emerged.

Descriptive Statistics/Correlations

Means and standard deviations for sample characteristics are reported in Appendix A, Table 2 and 3. This community sample was predominantly comprised of minority women (86%) with 26% having a high school education or less. On average women reported over 27 (*M* =

27.26, *SD* = 52.13) incidents of IPV during their pregnancy, 20 (*M* = 20.91, *SD* = 32.82) incidents of IPV during the post-partum year, and more than 7 (*M* = 7.50, *SD* = 4.61) different types of traumatic events throughout their lifetime. The most commonly reported traumatic events across the life span, included the death of someone close (not unexpectedly; 52.9%), seeing violence between family members during childhood (50%), having an abortion or miscarriage (46.1%), being emotionally abused or neglected (44.1%), or having serious financial problems (44.15). Of the 102 women, 32 (32%) reported clinically significant levels of depressive (*n* = 15), PTSD (*n* = 11), or comorbid PTSD and depressive (*n* = 6) symptoms at the time of their 12-month postpartum assessment. Similarly, in this sample women reported high incidences of CM including moderate to severe levels of emotional (26.4%), physical (28.4 %), and sexual (30.4 %) abuse. Intercorrelations among variables are reported in Appendix A, Table 4 and 5. Associations were largely as expected, such that prenatal IPV was significantly associated with postnatal IPV ($r = .62$, $p < .01$), CM ($r = .26$, $p < .01$), lifetime trauma ($r = .37$, $p < .01$), as well as current maternal psychopathology (Depressive symptoms; $r = .33$, $p < .01$; PTSD Symptoms; $r = .45$, $p < .01$)).

Hypothesis Testing

Research Question I. Is IPV exposure during pregnancy associated with infant HPA axis response to stressors?

Hypothesis I. There will be a positive relationship between IPV during pregnancy and infant cortisol reactivity, in which higher prenatal IPV exposure will predict higher cortisol reactivity.

A linear regression was used to test if prenatal IPV significantly predicted infant's cortisol reactivity, as measured by AUC_G, while controlling for maternal lifetime trauma,

education, age, and time of day that infant saliva was collected. The four predictor model explained 16.9% of the variance in infant cortisol reactivity (R^2 = .169, F(5,96) = 3.909, p = .003). Maternal age (b = .32, t(96) = 2.769, p = .007), education (b = -.94, t(96) = -3.040, p = .003) and total maternal lifetime trauma (b = -.30, t(96) = -1.788, p = .046) significantly predicted infant cortisol reactivity as measured by AUC_G. Pregnancy IPV was not a significant predictor of cortisol AUC_G. Results remained unchanged when the non-imputed data were used.

Research Question II. How does exposure to IPV during pregnancy influence infant HPA axis activity in conjunction with frequently co-occurring risk factors, including maternal childhood maltreatment history and postpartum psychopathology?

Hypothesis II. Maternal CM history will moderate the association between prenatal IPV exposure and infant cortisol reactivity, such that CM history will strengthen the effect of IPV exposure on infant cortisol reactivity. Among infants with high maternal CM history, the positive association between IPV exposure and cortisol reactivity will be much stronger than among infants without maternal CM history.

The two-step linear regression included time of day, maternal age, education, and total lifetime trauma as covariates in the first step of the model, as well as the main effects of pregnancy IPV and CM. The mean centered interaction between IPV and CM was entered in the second step of the regression. Neither pregnancy IPV or CM were significant predictors of infant cortisol reactivity. In addition, the interaction term did not improve model fit (ΔF(1, 94) = 0.007, ΔR^2 = .000, p = .93) and was not a significant predictor of AUC_G. Results remained unchanged when the non-imputed data were used.

Hypothesis III. The association between prenatal IPV exposure and infant cortisol reactivity will be moderated by maternal postpartum depressive symptoms, such that depressive

symptoms will strengthen the effect of IPV exposure on infant cortisol reactivity. Among infants with high maternal postpartum depressive symptoms, the positive association between IPV exposure and cortisol reactivity will be stronger, whereas this relationship will be weaker among infants of women without postpartum depression.

Again, a two-step linear regression was conducted including time of day, maternal age, education, and total lifetime trauma as covariates as well as the main effects of pregnancy IPV and current depression in the first step of the model. The centered interaction between IPV and depression symptoms was entered in the second step of the regression. Pregnancy IPV and depression were not significant predictors of cortisol AUC_G. Again, the interaction term did not improve model fit ($\Delta F(1, 94) = .001$, $\Delta R^2 = .000$, p = n.s.) and was not a significant predictor of infant reactivity. Results remained unchanged when the non-imputed data were used.

Hypothesis IV. The association between prenatal IPV exposure and infant cortisol reactivity will be moderated by maternal postpartum PTSD symptoms, such that more PTSD symptoms will reverse the effect of IPV exposure on infant cortisol reactivity. Among infants with high maternal postpartum PTSD symptoms, IPV exposure will have a negative association with cortisol reactivity, reflecting a blunted HPA axis response to stressors. Among infants of women without postpartum PTSD, IPV exposure will have a positive association with cortisol reactivity, similar to that suggested in Hypothesis 1.

A final two-step linear regression, including previously described covariates as well as the main effects of pregnancy IPV and current PTSD symptoms in the first step of the model. The centered interaction between IPV and PTSD symptomatology was entered in the second step of the regression. Pregnancy IPV and PTSD were not significant predictors of infant cortisol reactivity. Moreover, the interaction term did not improve model fit ($\Delta F(1, 94) = 0.346$ $\Delta R^2 =$

.003, p = n.s.) and was not a significant predictor of cortisol AUC_G. Results remained unchanged when the non-imputed data were used.

Post-hoc Analyses

Additional analyses were conducted to address issues related to the measurement of partner violence and cortisol reactivity. Multiple indices have been used to index cortisol reactivity in previous research and consensus is lacking in regards to the best measurement approach (Egliston, McMahon, & Austin, 2007; Goldberg et al., 2003). Although AUC_G is a widely used index, it does not provide information about the shape of the cortisol reaction or how steeply cortisol increases or declines after a stressor (Linden, Earle, Gerin, & Christenfeld, 1997; Myerson, Green & Warusawitherana, 2001). Thus, additional analyses were conducted using an alternative method to capture reactivity – change scores. Change scores represent the difference between baseline/resting cortisol levels, and levels 10 to 30 minutes after experiencing a stressor, a time when mobilization of the cortisol response can be detected in saliva (Goldberg et al., 2003). Although, not without limitations, change scores have the advantage of being easy to calculate and reliable (Linden, Earle, Gerin, & Christenfeld, 1997), and a more direct measure of the steepness of cortisol increase after a stressor. Cortisol levels showed small increases from baseline to 10-, 20-, and 30-minutes post-SST (See Table 6). Correlations between change scores and other variables are shown in Appendix A, Table 7. Cortisol change 10, 20, and 30 minutes after the end of the strange situation were used as outcomes in post-hoc analyses.

Table 6: *Descriptive Statistics of Change Scores (N = 102)*

Cortisol Sample	Mean	SD
Change 10 minutes (T2- T1)	.042	.120
Change 20 minutes (T3-T1)	.042	.118
Change 30 minutes (T4-T1)	.043	.119
AUC_G	11.64	7.63

Second, the measurement of IPV also lacks standardization across researchers. In this study, a widely used scale was utilized (CTS), but previous research with this instrument has used different item combinations to represent IPV (Hibel, Granger, Blair, & Cox, 2011; Saltzman, Holden, & Holahan, 2005). The strengths of using a "cumulative" CTS score are it encompasses a range of events and accounts for the frequency of violence experienced in a romantic relationship (Alhusen, Ray, Sharps & Bullock, 2015; Prospero, 2008). On the other hand, the examination of physical and psychological IPV independently is advantageous, as studies have started documenting differential effects for different types of IPV (Bailey & Daugherty, 2007; Charles & Perreira, 2007). Thus, sub-scores for physical and psychological IPV were used as predictors. Research on sexual violence is very limited, and thus this subscale was not used in additional analyses. Normality, skewness, and kurtosis were also evaluated for the subscores of IPV based on the previously described methods and both prenatal psychological aggression and physical assault scores were log transformed. Intercorrelations among IPV subtypes and other variables are reported in Appendix A, Table 7. Associations were largely as expected, such that both prenatal psychological aggression and physical assault were significantly associated with postnatal IPV ($r = .48$, $p < .01$; $r = .32$, $p < .01$), CM ($r = .21$, $p < .05$; $r = .27$, $p < .01$), lifetime trauma ($r = .30$, $p < .01$; $r = .30$, $p < .01$), as well as current maternal psychopathology (Depressive symptoms; $r = .29$, $p < .01$; $r = .25$, $p < .05$; PTSD Symptoms; $r = .40$, $p < .01$; $r = .30$, $p < .01$). Prenatal aggression and physical assault were also highly correlated with each other ($r = .61$, $p < .01$).

Post Hoc Analysis of Research Question I. Is IPV exposure during pregnancy associated with infant HPA axis response to stressors?

Post hoc analyses followed the previously described methods and used imputed data sets; analyses with unimputed data were conducted only to evaluate whether similar results emerged. Independent linear regressions were conducted using physical and psychological violence as a predictor of change at 10, 20 and 30 minutes post-SST. Time of day, maternal age, education, and lifetime trauma were included as covariates. Physical IPV was not a significant predictor of 10-minute change (b = .019, t(96) = 1.572, p = n.s.), 20-minute change (b = .020, t(96) = 1.530, p = n.s.), or 30-minute change (b = .020, t(96) = 1.530, p = n.s.) in cortisol levels. Similarly, psychological aggression was not a significant predictor of cortisol levels 10 (b = .007, t(96) = .800, p = n.s.), 20-minute (b = .009, t(96) = 1.069, p = n.s.) post-SST. In comparison, psychological aggression was associated with 30-minute change in cortisol levels (b = .020, t(96) = 2.278, p = .025), but this association was not significant using the Bonferroni correction.

Post Hoc Analysis of Research Question 2. How does exposure to IPV during pregnancy influence infant HPA axis activity in conjunction with frequently co-occurring risk factors, including maternal childhood maltreatment history and postpartum psychopathology?

Again, moderation analyses were conducted to determine if commonly co-occurring risk factors (i.e. childhood trauma, maternal depression and PTSD symptoms) influenced the effect of prenatal physical and psychological violence on infant cortisol reactivity as measured by change scores 10, 20, and 30 minutes post-SST.

Post Hoc Analysis: Childhood Maltreatment as the Moderator. Two-step linear regressions were conducted to evaluate whether the interaction between *physical violence* and CM history predicted cortisol change levels at 10-minutes, 20-minutes, and change at 30-minutes post-SST. The first step of the model included time of day, maternal age, education and lifetime trauma history as covariates, as well as the main effects of physical IPV and CM history.

Neither prenatal physical IPV nor CM predicted cortisol change at 10-, 20-, or 30-minutes post-SST. The centered prenatal physical IPV by CM history interaction was entered in the second step of the model. The addition of the interaction term did not improve model fit between prenatal physical IPV and infant cortisol change at 10-minute ($\Delta F(1, 94) = .250$ $\Delta R^2 = .002$, p = n.s.), 20-minute ($\Delta F(1, 94) = 0.474$ $\Delta R^2 = .005$, p = n.s.), or 30 minutes ($\Delta F(1, 94) = 1.628$ $\Delta R^2 = .016$, p = n.s.), post-SST, such that the physical IPV-by-CM interaction did not predict cortisol outcomes.

Similar two-step regressions were used to assess whether the interaction between *psychological violence* and CM history predicted change levels at 10-minutes, 20-minutes, and change at 30-minutes post-SST. Again, The first step of the model included time of day, maternal age, education and lifetime trauma history as covariates as well as the main effects of psychological IPV and CM history. Neither psychological IPV nor CM predicted cortisol change scores. The centered prenatal psychological IPV by CM history interaction was entered in the second step of the model. The addition of the interaction term did not improve model fit for infant cortisol change at 10-minutes ($\Delta F(1, 94) = 2.073$ $\Delta R^2 = .020$, p = n.s.) or 20-minutes post-SST ($\Delta F(1, 94) = 3.082$ $\Delta R^2 = .030$, p = n.s.). The interaction between psychological violence and CM on cortisol reactivity at 30-minutes post-SST ($\Delta F(1, 94) = 4.216$ $\Delta R^2 = .102$, $p = .043$) was trending significance but did not meet standards of the calculated Bonferroni correction of $p < .0125$.

Post Hoc Analysis: PTSD as the Moderator. Similar two-step regressions were used to evaluate whether PTSD moderated the effects of *physical IPV* on infant cortisol levels. For 10 minutes post-SST cortisol levels, the second step of the model improved model fit ($\Delta R^2 = .037$, $\Delta F(1, 94) = 3.927$ $p = .05$) but did not meet the Bonferroni corrected $p < .0125$, and thus the

interaction was not explored further. For 20 minutes post-SST cortisol levels, the second step improved model fit (ΔR^2 = .136, $\Delta F(1, 94) = 7.679$, p =.007). Prenatal physical assault (b = .033 $t(94) = 2.630$, p = .010) significantly predicted infant cortisol reactivity at 20 minutes post stressor, but maternal PTSD and the covariates included in the model did not. Similarly, there was a significant interaction between physical assault and PTSD (b =-.003 $t(94) = -2.771$, p = .007), as shown in Table 8.

Table 8: *Prenatal Physical Assault and PTSD Symptoms Predicting Infant Cortisol Reactivity 20 minutes Post-SST*

Variable	β	Std. Error	*p- value*
Constant	-.017	.093	.859
PTSD	.001	.001	.421
Physical Assault	**.033**	**.001**	**.010***
Maternal Education	.004	.005	.435
Maternal Age	.002	.002	.257
Maternal Lifetime Trauma	-.005	.003	.082
Time of Day	.000	.000	.413
Assault x PTSD	**-.003**	**.001**	**.007***

*$p \leq .05$

Further exploration of the interaction using the PROCESS macro (Hayes, 2013) tested the conditional effects of psychological aggression at three levels of childhood trauma: one standard deviation below the mean, at the mean, and one standard deviation above the mean. Results showed, for individuals with low (b = .067, $t(94) = 3.2448$, p = .002) or average (b = .033, $t(94) = 2.6304$, p = .010) levels of PTSD symptoms, there was a significant relation between prenatal physical assault and their infants' cortisol reactivity, see Appendix B, Figure 1 for visual representation. However, this was not true for women with high levels of PTSD (b = .001, $t(94) = -.080$, p = n.s.).

A similar pattern was observed for 30 minutes post-SST cortisol scores, prenatal physical assault (b = .087, t(94) = 3.37, p = .001) significantly predicted infant cortisol reactivity, but maternal PTSD and the covariates included in the model did not. A significant interaction was observed between physical assault and PTSD (b = -.003 t(94) = -2.90, p = .005), as shown in Table 9. PTSD significantly moderated the relationship between prenatal physical assault and infant cortisol reactivity, such that IPV was associated with more steep increases among infants of women with low (b = .076, t(94) = 3.371, p = .001) and average (b = .037, t(94) = 2.71, p = .008) PTSD symptoms. Again, this was not true for women with high levels of PTSD (b = -.002, t(94) = -.12, p = n.s.), see Figure 2 (Appendix B).

Table 9: *Prenatal Physical Assault and PTSD Symptoms Predicting Infant Cortisol Reactivity 30 minutes Post-SST*

Variable	β	Std. Error	*p- value*
Constant	.013	.104	.898
PTSD	.001	.001	.589
Physical Assault	**.087**	**.026**	**.001***
Maternal Education	.005	.004	.325
Maternal Age	-.001	.002	.655
Maternal Lifetime Trauma	-.001	.003	.746
Time of Day	.000	.000	.592
Assault x PTSD	**-.003**	**.001**	**.005***

*$p \leq .05$

Similarly, a series of two-step linear regressions, including previously described covariates and main effects, were used to assess if *psychological* aggression and PTSD symptoms predicted change levels at 10, 20, 30 minutes post-SST. The interaction term of psychological aggression and PTSD symptoms were entered into the second step of the equation.

There was no significant change in model fit with the addition of the interaction terms at 10 minutes ($\Delta F(1, 94) = .048$ $\Delta R^2 = .000$, p = n.s.), 20 minutes ($\Delta F(1, 94) = .210$ $\Delta R^2 = .002$, p = n.s.), or 30 minutes post-SST ($\Delta F(1, 94) = 2.165$ $\Delta R^2 = .021$, p = n.s.).

Post Hoc Analysis: Depression as the Moderator. A final set of two-step linear regressions, including previously described covariates as well as the main effects, were used to assess whether *physical IPV* and maternal depression symptoms predicted change levels at 10, 20 and 30 minutes post-SST. The centered prenatal physical IPV by depression interaction was entered in the second step of the three regression models. The addition of the interaction term did not improve model fit for infant cortisol change at 10 minutes ($\Delta F(1, 94) = 0.168$ $\Delta R^2 = .002$, p = n.s.), 20 minutes ($\Delta F(1, 94) = .681$ $\Delta R^2 = .007$, p = n.s.), or 30 minutes post-SST ($\Delta F(1, 94) = .00$ $\Delta R^2 = .000$, p = n.s.). Physical IPV, depression, and the interaction effect did not significantly predict infant cortisol reactivity.

Similar regressions were used to assess the interaction between *psychological IPV* and maternal depression. The interaction term did not improve the model fit for cortisol change at 10-minutes ($\Delta F(1, 94) = 1.61$ $\Delta R^2 = .016$, p = n.s.), 20-minutes ($\Delta F(1, 94) = 2.26$ $\Delta R^2 = .021$, p = n.s.), or 30-minutes post-SST ($\Delta F(1, 94) = 2.50$ $\Delta R^2 = .025$, p = n.s.. Neither psychological aggression nor depressive symptoms significantly predicted infant cortisol reactivity.

Discussion

Prenatal stress has been consistently linked to negative infant outcomes, including lower academic achievement, emotion regulation difficulties, and disrupted HPA-axis activity (Mwaniki, Atieno, Lawn & Newton, 2012; Van den Bergh et al., 2005; Hobel, et al., 2008; Wadhwa, 2005). However, few studies have examined offspring outcomes after exposure to prenatal IPV, a relatively common and highly traumatic stressor (Black, et al., 2011; Centers for

Disease Control and Prevention [CDC], 2012). A limited number of studies have reported that prenatal IPV increases infant and child HPA axis reactivity to stressors (Levendosky et al., 2016; Martinez-Torteya et al., 2016) increasing risk for poor emotion regulation and development of future mental health symptoms (Hobel, et al., 2008; Lazinski, Shea, & Steiner, 2008; Nast, Bolten, Meinlschmidt, & Hellhammer, 2013; Wadhwa, 2005) but additional studies are needed to better understand how partner abuse during pregnancy shapes early stress response alterations. The current study aimed to explore the relation between prenatal IPV, commonly co-occurring contextual risk factors (i.e. maternal childhood trauma and current maternal psychopathology), and the infant stress response among 12-month-old infants. The relationships between prenatal IPV, maternal psychopathology, maternal childhood abuse history, and infant cortisol response were complex; these maternal factors did not significantly predict infant cortisol reactivity as measured by $AUC_{G,}$ a commonly used index of cortisol secretion, but jointly influenced post-stressor cortisol increases. These findings expand upon the literature linking prenatal stress and trauma with infants' stress response. Identifying biopsychosocial mechanisms underlying infant stress response alterations can inform evolving theoretical models on the negative effects of early life stress and suggest potential intervention avenues with infants who are at high risk due to prenatal adversity.

Although this sample was recruited from the community, it represented a high-risk group of women; mean levels of trauma exposure from childhood to adulthood were high, and women often reported other psychosocial stressors (i.e. low income, limited academic achievement, and mental health symptoms). Rates of prenatal IPV exposure in this group exceeded previously reported prevalence rates in large nationally representative samples (71.6% in our study compared to 4 to 8% in large epidemiological studies; Chu, Goodwin, D'Angel, 2010; Saltzman,

Johnson, Gilbert, & Goodwin, 2003; Sharps, Laughon, & Giangrande, 2007). However, making comparisons between studies is challenging, as few studies report the frequency or severity of traumatic events endorsed by participants. For example, data on prenatal IPV is commonly reported as the percentage of women reporting one ore more violent event over the course of their pregnancy (Seng, Low, Sperlich, Ronis, & Liberzon, 2009). However, three studies reported 5 to 46.6 IPV incidents during pregnancy on average among other community samples (Alhusen, Bullock, Sharps, Schminkey, Comstock, & Campbell, 2014; Martinez-Torteya et al., 2016; Levendosky et al., 2016), a range that includes the mean reported by our participants. Studies of IPV during pregnancy, similar to ours, commonly recruit women from community organizations that target low income and minority families. These results may be reflective of the experiences of women in these communities that have additional SES stressors and limited access to resources.

Not surprisingly, prenatal IPV often co-occurred with other traumatic experiences and maternal postpartum psychopathology, replicating findings of associations between IPV exposure during pregnancy and past trauma exposure (Dailey, Humphreys, Rankin, & Lee, 2011; Rodriguez et al., 2008), as well as the development of depression and PTSD (Campbell & Lewandoski, 1997; Rodriguez et al., 2008). Associations between CM, traumatic events throughout the lifespan, and postpartum psychopathology were also moderate, consistent with previous reports that CM contributes to the development of postpartum depression independently of prenatal IPV (Malta, McDonald, Hedadoren, Weller & Tough, 2012), and increases risk for subsequent development of PTSD following exposure to additional traumatic experiences later in life (Lewis et al., 2006).

The current study predicted a positive relationship between prenatal IPV and infant cortisol reactivity, in which higher IPV exposure would predict higher cortisol reactivity. Results indicted no significant relation between IPV during pregnancy and the infant cortisol response 12 months postpartum, when the combination of physical, psychological, and sexual partner abuse was used to predict cortisol AUC_G. This was surprising given the substantial literature linking *in utero* experiences, such as maternal stress and psychopathology, with infant cortisol dysregulation (Brennan et al, 2008; Hout et al., 2004). Two previous studies specifically assessed prenatal IPV and offspring cortisol reactivity and reported significant associations (Levendosky et al., 2016; Martinez-Torteya et al., 2016). For example, in a sample of 182 mother-infant dyads, Levendosky and colleagues (2016) reported IPV significantly predicted an increase in cortisol in response to a brief "arm restrain" task at 12 months. Similarly, as part of a longitudinal prospective study of 119 preadolescents, Martinez-Torteya et al. (2016), also reported prenatal IPV exposure significantly predicting elevated cortisol secretion prior to and following a "social stress test." Differences in the laboratory stressor used may account for differences in findings: Levendosky et al.'s (2016) arm restrain is brief and mildly stressful, and only resulted in mobilization of the cortisol response at 20 or 40-minutes post-stress for 65% of the sample. In contrast, the Strange Situation used in the current study is considered a more powerful stressor, consisting of two separation and reunions from the mother, and has resulted in mean-level increases post-SST in previous research (Laurent, Ablow, & Measelle, 2012; Tollenaar, Beijers, Jansen, Riksen-Walraven, & De Weerth, 2011). Both tasks are designed to elicit and evaluate ones response to negative emotions; however, the arm restraint tasks and the strange situation may be capturing different types of negative emotion. For instance, the arm restraint task is meant to elicit anger and frustration while the strange situation focuses on fear.

In addition, infants' responses to the strange situation may be more influenced by the quality of the mother-infant relationship, attachment security has been linked to the severity of distressed displayed when a caregiver leaves the room. For instance, insecurely attached children have grown accustomed to not getting the feedback they need from a caregiver may not become as distressed by their absence (Gunnar, Talge, & Herrera, 2009).

Inconsistent findings may also be due to differences in the characterization of the cortisol response. There is little consensus in regards to the best measurement of cortisol reactivity. AUC_G is commonly used to assess neuroendocrine variables (i.e. hormone secretion) over time. In comparison, change scores have been used as a representation of the mobilization of the cortisol response in which the difference between baseline/resting cortisol levels is calculated (Goldberg et al., 2003). Although both indices have been used and show associations with multiple psychosocial outcomes, they likely reflect different aspects of the activity of the HPA axis. In fact, cortisol change scores were influenced by complex interactions between pregnancy IPV exposure, maternal child maltreatment history, depression, and PTSD in the present study, while AUC_G was not. Additional research comparing measurement of cortisol reactivity is needed to better integrate multiple measurement strategies and address the limitations of various methodologies currently used in stress reactivity research.

In our study only prenatal exposure to *physical* assault predicted increases in infant cortisol, 20- and 30-minutes post-SST among women who reported low and moderate levels of IPV, but physical IPV was not related to infant cortisol among women with severe PTSD symptoms. This is partially consistent with the hypothesized interaction between IPV and PTSD; among women with low to moderate posttraumatic symptoms, physical assault during pregnancy was associated with higher infant cortisol reactivity, mirroring the findings of the two

previous studies of pregnancy IPV that measured offspring cortisol outcomes (Levendosky et al., 2016; Martinez-Torteya et al., 2016). In contrast, among women with high levels of PTSD, pregnancy physical IPV was not associated with infant cortisol reactivity. It may be that, women with high levels of postpartum PTSD already have experienced significant trauma and concomitant physiological stress response alterations, which shape infant HPA axis activity, regardless of prenatal IPV exposure. Alternatively, this may be due to HPA axis hypoactivation related to maternal PTSD. Previous studies have reported a blunted cortisol response in infants exposed to prenatal maternal PTSD symptoms (Yehuda et al., 2005). For example, Yehuda and colleagues (2005) documented a lower cortisol awakening response in infants whose mothers developed PTSD following the World Trade Attacks in New York City compared to infants whose mothers did not develop symptoms. In our study, post-SST cortisol was lower among infants of women with PTSD, suggesting the developing HPA axis is a highly complicated network that responds to competing processes (i.e. increased activation associated with maternal stress and trauma, decreased activation associated with maternal PTSD symptoms). However, it is difficult to compare our findings to those of previous studies, because others have not examined the interaction between the frequency or severity of prenatal trauma exposure and PTSD symptomatology. Of note, previous studies of PTSD focus on natural or man made disasters (e.g. 9/11, hurricanes, and ice storms), and the present study extends these findings to victims of IPV.

Surprisingly, neither maternal CM nor depression was associated with infant cortisol reactivity using AUC_G or post-stress change scores. This is consistent with one study that assessed both prenatal IPV exposure and post-partum depression (Levendosky et al., 2016) but is inconsistent with a number of other studies that document effects of maternal depression and CM

on offspring cortisol response (Brand et al., 2010; Essex, Klein, Chod, & Kalin, 2002; Martinez-Torteya et al, 2014). Timing of symptom assessment is one plausible explanation for the discrepant findings between studies. In this study, mental health symptoms were assessed 12 months postpartum and were reflective of the mother's current functioning. Onset of postpartum mental health symptoms is known to develop between 2 weeks and 12 months following delivery (Stowe, Hostetter, & NewPort, 2005), so our assessment may have failed to identify women who developed depressive symptoms earlier in the postpartum period. For example, Brennan and colleagues (2008) recruited 194 mother infant dyads to participate in a laboratory paradigm at 6 months old. They reported elevated cortisol levels in response to stress in infants exposed to perinatal depressive symptoms, even after controlling for maternal lifetime history of depression. Similarly, Essex and colleagues (2002) followed a group of 282 children from delivery until 12 months and subsequently followed up at 4.5 years. They reported maternal depression during infancy as a significant predictor of infant cortisol 4.5 years old (Essex, Klein, Cho, & Kalin, 2002).

In the current study, maternal education, maternal age, and lifetime trauma were used as covariates as they have all been connected to physiological changes in the HPA-axis (Evans & Kim, 2007; Lupien, King, Meaney, & McEwen, 2001; Schumacher et al., 2010; Enlow et al., 2009; Albers, Marianne Riksen-Walraven, Sweep, & Weerth, 2008). Maternal lifetime trauma history was also associated with a dampened cortisol response. Studies examining maternal lifetime trauma have also been linked with infant adrenocortical alterations, including prolonged reactivity with longer time to recover from stress (Enlow et al., 2009). This pattern is consistent with the adult literature linking chronic trauma exposure with a dampened cortisol response. This is consistent with McEwen's concept of allostasis, which describes how frequent and

prolonged activation caused by stressors (i.e., exposure to multiple stressful or traumatic events) signals the body to down regulate as a way to achieve homeostasis (McEwen, 1998). Cumulative and frequent environmental stressors, like the demands brought upon women living in low SES, have been linked to more negative physiological outcomes (Braveman et al, 2009; Suglia et al., 2010). Ultimately this process results in the dampening of the cortisol response system in response to additional stressors (Heim et al., 2008). Unexpectedly, maternal education and maternal age had opposite effect on infant cortisol secretion; older women had children with higher cortisol $AUC_{G,}$ while women with more education had children with lower cortisol $AUC_{G.}$ Previous literature has used maternal education as an indicator of SES; however, the evidence linking low SES and high cortisol is tenuous, as findings are not consistent across studies (for review of the literature see Dowd, Simanek, & Aiello, 2009).

Implications. This study significantly contributes to the current literature, as few studies have considered the effects of prenatal IPV exposure on infant physiology. It is important to continue to explore how maternal prenatal and postpartum experiences predict infant outcomes. Specifically, there is a need to better understand how prenatal IPV in combination with other negative maternal experiences can alter the stress responses of offspring early on. Understanding patterns of cortisol dysreuglation due to chronic stress exposure may help to identify children at risk for negative physical and mental health problems due to a sensitization of the HPA axis. Additionally, the current study allowed for the exploratory analysis of different types of partner violence on infant stress reactivity. Few studies of this population have examined the relationship between type of maternal prenatal violence exposure on the infant physiological stress response and even fewer have considered the contribution of how additional contextual factors may influence this relationship.

The interactive effect of prenatal physical assault and maternal PTSD symptoms on infant stress reactivity has significant clinical implications. Further exploration of prevention and intervention services for this population of women are needed. Clinically, it is important to consider how different types of IPV may to lead to alternative psychological and physical trajectories for both women and their children. A better understanding of how different types of partner violence and alternative life experiences influence maternal and infant development would be beneficial in identifying the best treatment and services needed for these women and their families.

Relatedly, a large percentage of women in this study also reported significant lifetime trauma histories, suggesting an increased need for better prevention and intervention services. Given the increased frequency of doctor's visits during and after pregnancy, the perinatal period provides a unique opportunity to target these efforts. Increased screening and psychosocial education surrounding violence and mental health symptoms in Obstetric and Gynecology as well as pediatrician's offices may help to identify women at increased risk or in need of services.

Limitations. While this study has several strengths that contribute to the current literature, there are a number of limitations that must also be acknowledged. First, the sample size is relatively small and may reflect experiences of individuals in an urban environment. While racially/ethnically diverse, the frequency and severity of trauma reported across the life span by many of the women in this sample may be reflective of their community.

Secondly, the current study is limited in its measurement of the stress response. Salivary collection of cortisol is considered the least invasive measurement of the HPA axis and most commonly used in studies with infants. However, infants generally produce less saliva and it is often difficult to collect adequate volumes needed for assaying samples. This is particularly true

of infants exposed to mild laboratory stressors (Egliston, McMahon, & Austin, 2007). Due to collection difficulties, many infants in this study were missing the 5th sample, 50 minutes post-SST, or the volumes of the collection were not large enough to run analysis in duplicate.

Similarly, there is some debate in the current literature on the ability of laboratory stressors too appropriately illicit enough discomfort to produce a substantial change in the HPA axis. The Strange Situation generally elevates infant distress by temporarily separating mother and infant. During the task the separations are relatively brief, however if distressed, an unknown adult or their caregiver often comforts the infant. Several studies have demonstrated increased cortisol levels of infants in response to the strange situation (Tollenaar et al., 2011). Yet, there is evidence suggesting by one year infants are more socially aware and can tolerate separation for longer periods of time without becoming distressed; decreasing the likelihood of activating a cortisol response (Gunnar, Talge, Herrera, 2009). In the future, it is hoped multiple measures of the HPA axis (i.e. measurements of reactivity and diurnal patterns collected at simultaneous times points) can be integrated into assessments to get a better understanding of factors contributing to changes in the physiological response to stress over time.

Finally, this study is cross sectional by design therefore limited in the scope of conclusions that can be drawn. Most data was collected using self-reports, and the pregnancy IPV and lifetime trauma data was collected retrospectively. Response bias, social desirability, and imprecise recall are commonly occurring limitations of relying on participant self-reports (Podsakoff, MacKenzie, Jeong-Yeon, 2003). In addition, information about pregnancy and early postpartum psychopathology was not collected; although there is some stability in depressive and PTSD symptoms from pregnancy to the postpartum year (Mora, Bennett, Elo, Mathew, Coyne, & Culhande, 2009; Robertson, Grace, Wallington, & Stewart, 2004), there is evidence that

depression influences infant cortisol response at various time points over the first year of life (Brennan et al., 2008; Field, 2011 for review). Longitudinal assessment of both maternal prenatal and postpartum mental health symptoms is needed to better understand the unique effects of pregnancy IPV on infant HPA axis functioning.

Future Directions. The current findings serve to highlight the continued need of research into the role of early trauma exposure on infant health, specifically intimate partner violence. Considering the complexity and prevalence of trauma exposure across the life span this study also highlights the need for a better understanding of how type, frequency, and severity of trauma influences individual mood and physiology. Additionally, the lack of consistency in the measurement of cortisol response system makes comparisons between studies challenging. Diurnal patterns, awakening response, reactivity, are all used in studies assessing HPA-activity. Future research should use multiple measures to assess HPA-activity as an indicator of the stress response. This is extremely important for women of reproductive age, as maternal experience continues to be shown to have a strong contribution to the development of infant's physiological response to stress.

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Appendix A – Demographic and Correlation Tables

Table 2: *Maternal Demographic Characteristics of the Sample* (*N* = 102)

Variable	***N***	**%**
Age (years)[a]	29.77 ± 6.89	
18-21	11	10.0
22-25	22	22.0
26-29	22	22.0
30-34	23	23.0
35 +	24	23.0
Ethnicity		
White/Caucasian	14	14.0
African-American	35	34.0
Hispanic/Latino	41	40.0
Biracial/Other	10	10.0
Native American	1	1.0
Asian/Pacific Islander	1	1.0
Education (years)[a]	14.61 ± 2.47	
GED or Below	27	26.0
Some College	27	27.0
Trade School/ AA Degree	20	19.0
BA/BS	15	15.0
Some Graduate School	4	4.0
Graduate Degree	8	8.0
Unknown	1	1.0

a. Mean ± SD.

Table 3: *Infant Demographic Characteristics of the Sample* (*N* = 102)

Variable	***N***	**%**
Age (years)[a]	11.85± 0.943	
10	3	2.9
11	38	37.3
12	36	35.3
13	19	18.6
14	6	5.9
Gender		
Male	52	51.0
Female	50	49.0

a. Mean ± SD.

Table 4: *Bivariate Correlations and Descriptives among Study Variables*

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Depression	--											
2. Childhood Trauma	.24*	--										
3. PTSD	.65**	.37**	--									
4. Life Time Trauma	.21*	.51**	.28**	--								
5. Prenatal IPV	.33**	.26**	.45**	.37**	--							
6. Postpartum IPV	.28**	.14	.35**	.28**	.62**	--						
7. Maternal Education	-.22*	-.10	-.16	-.02	-06	.02	--					
8. Maternal Age	-.20*	-.10	-.29**	.01	-.24*	-.17	.34**	--				
9. Maternal Race	.06	-.01	.01	.06	-.01	.12	.19	.18	--			
10. Infant Age	-.02	-.09	-.01	-.11	-.21*	-.01	.03	-.10	.09	--		
11. Infant Cortisol (AUC_G)	-.16	-.04	-.09	-.20*	-14	-.03	-.19	.20*	.19	-.06	--	
12. Lab Analyzed	.11	-.24*	-.03	-.20*	-.07	.01	-.02	-.08	-.14	-.04	.02	--
n	102	102	102	102	102	102	102	102	102	102	102	99
Mean	10.76	59.25	14.73		27.26	20.91	14.61	29.77	--	11.85		
SD	8.21	9.65	11.81		52.13	32.82	2.45	6.89	--	.943		

Note: $^{**}p < .01$, $^{*}p < .05$.

Table 5: *Bivariate Correlations of Cortisol Predictors*

Variable	1	2	3	4	5	6	7
1. Infant Medication	--						
2. Infant Mood	.046	--					
3. Infant Sleep	-.142	.064	--				
4. Infant Sleep Dur.	-.125	.037	.862**	--			
5. Time of Day	.029	.021	-.492**	-.492**	--		
6. Lab Analyzed	.141	.131	-.180	-.262*	.033	--	
7. Infant Cortisol (AUC_G)	.001	-.088	-.043	-.053	.120	.051	--
n	101	102	101	98	102	99	102

Note: $^{**}p < .01$, $^{*}p < .05$.

Table 7: *Bivariate Correlations and Descriptives for Post Hoc Analyses*

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Depression	--																
2. Childhood Trauma	.24*	--															
3. PTSD	.65**	.38**	--														
4. Prenatal IPV	.17	.30**	.28**	--													
5. Postpartum IPV	.26**	.13	.31**	.35**	--												
6. Prenatal Aggression	.20*	.19	.32**	.90**	.43**	--											
7. Prenatal Assault	.01	.31**	.09	.85**	.07	.58**	--										
8. Life Time Trauma	.21*	.51**	.29**	.34**	.25*	.31**	.28**	--									
9. Maternal Education	-.22*	-.10	0.17	-.09	.01	-.06	-.12	-.02	--								
10. Maternal Age	-.20*	-.11	-.27**	-.27**	-.17	-.26*	-.21*	.01	.34**	--							
11. Maternal Race	.06	-.01	.01	.04	.10	.08	-.02	.06	.19	.18	--						
12. Infant Age	-.02	-.08	.01	-.12	-.00	-.2*	-.03	-.11	.03	-.10	.09	--					
13. Infant Cortisol (AUC_{G})	-.16	-.04	-.09	-.15	-.01	-.16	-.1	-.20*	-.19	.20*	.12	-.06	--				
14. Change 10 minutes (T2- T1)	-.12	-.11	-.04	-.04	.03	-.04	-.03	-.19	.03	.13	.01	-.07	.48**	--			
15. Change 20 minutes (T3-T1)	-.18	-.13	-.09	-.01	-.01	.00	.00	-.14	.10	.11	.03	.08	.31**	.82**	--		
16. Change 30 minutes (T4-T1)	-.08	.01	-.04	.09	.03	.13	.04	-.01	.07	-.05	.08	-.11	.30**	.73**	.80**	--	
17. Lab Analyzed	.12	-.24*	-.02	-.07	.02	-.01	-.12	-.20*	-.02	-.08	-.14	-.04	.02	-.09	-.06	-.08	--
n	102	102	102	102	102	102	102	102	102	102	102	102	102	102	102	102	99
Mean	10.76	59.25	14.73	27.26	20.91	18.79	4.93	7.50	14.61	29.77	--	11.85	11.64	.04	.04	.04	--
SD	8.21	9.65	11.81	52.13	32.82	29.39	22.24	4.61	2.45	6.89	--	.94	7.63	.12	.12	.13	--

Note: $^{**}p < .01$, $^{*}p < .05$.

Appendix B – Figures of Significant Interactions

Figure 1.

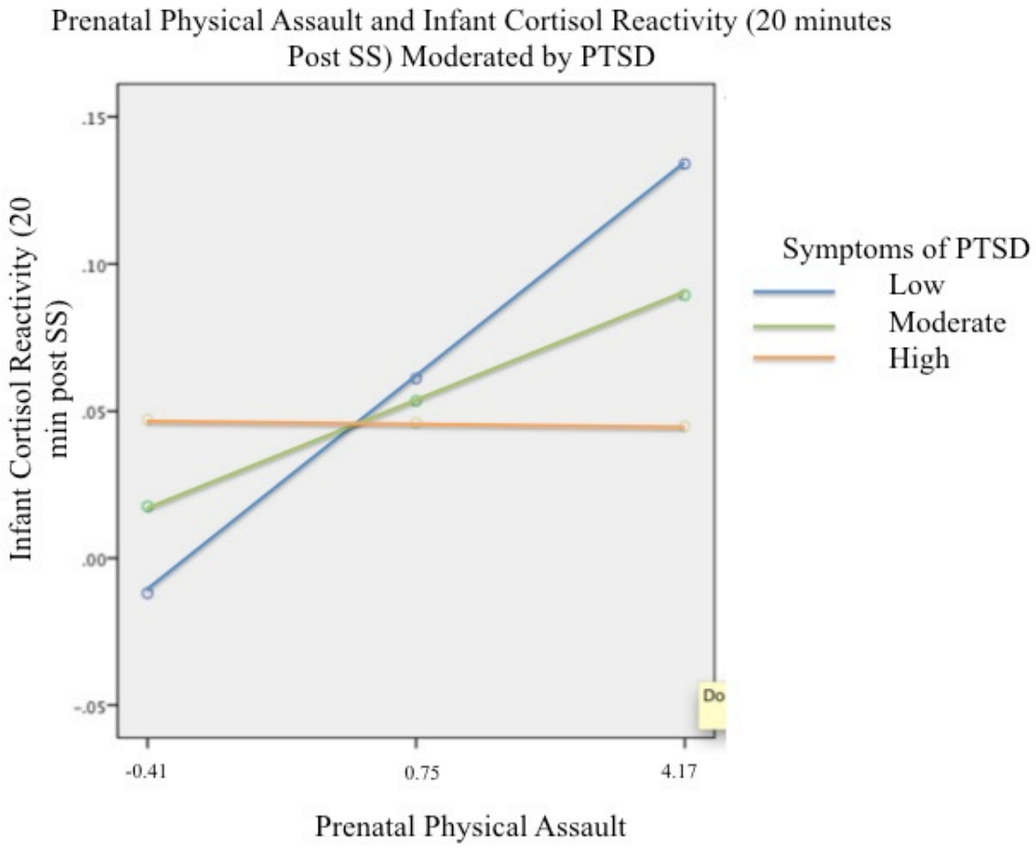
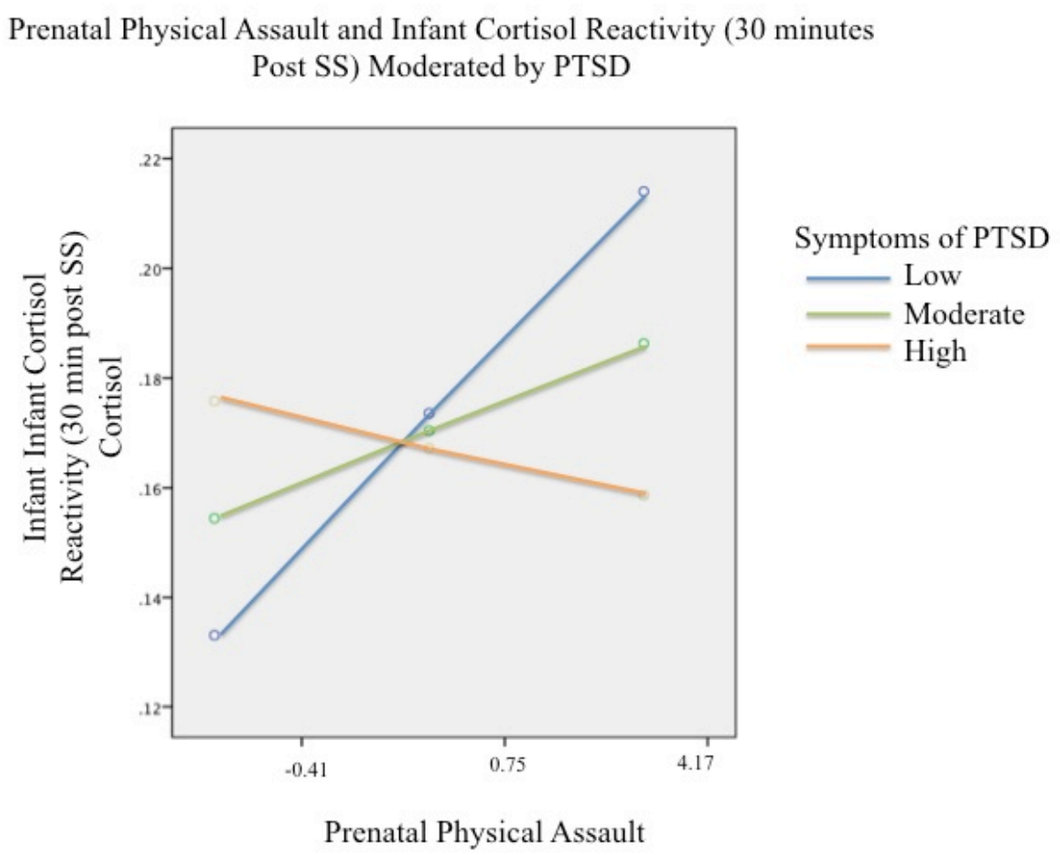


Figure 2.



Appendix C – Self- Report Measures

Conflict Tactics Scale (Pregnancy)

Relationship Behaviors

No matter how well a couple gets along, there are sometimes when they disagree, get annoyed with the other person, want different things from each other, or just have spats or fights because they are in a bad mood, are tired, or for some other reason. Couples also have many different ways of trying to settle their differences. This is a list of things that may happen when you have differences. Please mark how many times your partner did these things to you during your pregnancy with the infant participating in this study. If your partner did not do one of these things during your pregnancy, but it has happened before that, circle "7." If your partner NEVER did these things to you prior to and during your pregnancy, mark "0."

1 = Once during my pregnancy
2 = Twice during my pregnancy
3 = 3-5 times during my pregnancy
4 = 6-10 times during my pregnancy
5 = 11-20 times during my pregnancy
6 = More than 20 times during my pregnancy
7 = Not during my pregnancy, but this has happened before.
0 = My partner has never done this to me

How often did this happen during your pregnancy?	**1= once**	**2= twice**	**3= 3-5 times**	**4= 6-10 times**	**5= 11-20 times**	**6=More than 20 times**	**7= Only before**	**0= never**
1. My partner showed care for me even though we disagreed	1	2	3	4	5	6	7	0
2. My partner explained his or her side of a disagreement to me	1	2	3	4	5	6	7	0
3. My partner insulted or swore at me	1	2	3	4	5	6	7	0
4. My partner threw something at me that could hurt	1	2	3	4	5	6	7	0
5. My partner twisted my arm or hair	1	2	3	4	5	6	7	0
6. I had a sprain, bruise, or small cut because of a fight with my partner	1	2	3	4	5	6	7	0
7. My partner showed respect for my feelings about an issue	1	2	3	4	5	6	7	0
8. My partner made me have sex without a condom	1	2	3	4	5	6	7	0
9. My partner pushed or shoved me	1	2	3	4	5	6	7	0
10. My partner used force (like hitting, holding down, or using	1	2	3	4	5	6	7	0

a weapon) to make me have oral or anal sex								
How often did this happen during your pregnancy?	**1= once**	**2= twice**	**3= 3-5 times**	**4= 6-10 times**	**5= 11-20 times**	**6=More than 20 times**	**7= Only before**	**0= never**
11. My partner used a knife or gun on me	1	2	3	4	5	6	7	0
12. I passed out from being hit in the head by my partner in a fight	1	2	3	4	5	6	7	0
13. My partner called me fat or ugly	1	2	3	4	5	6	7	0
14. My partner punched or hit me with something that could hurt	1	2	3	4	5	6	7	0
15. My partner destroyed something belonging to me	1	2	3	4	5	6	7	0
16. I went to a doctor because of a fight with my partner	1	2	3	4	5	6	7	0
17. My partner choked me	1	2	3	4	5	6	7	0
18. My partner shouted or yelled at me	1	2	3	4	5	6	7	0
19. My partner slammed me against a wall	1	2	3	4	5	6	7	0
20. My partner was sure we could work out a problem	1	2	3	4	5	6	7	0
21. I needed to see a doctor because of a fight with my partner, but I didn't	1	2	3	4	5	6	7	0
22. My partner beat me up	1	2	3	4	5	6	7	0
23. My partner grabbed me	1	2	3	4	5	6	7	0
24. My partner used force (like hitting, holding down, or using a weapon) to make me have sex	1	2	3	4	5	6	7	0
25. My partner stomped out of the room, or house, or yard during a disagreement	1	2	3	4	5	6	7	0
26. My partner insisted on sex when I did not want to (but did not use physical force)	1	2	3	4	5	6	7	0
27. My partner slapped me	1	2	3	4	5	6	7	0
28. I had a broken bone from a fight with my partner	1	2	3	4	5	6	7	0
29. My partner used threats to make me have oral or anal sex	1	2	3	4	5	6	7	0

30. My partner suggested a compromise to a disagreement	1	2	3	4	5	6	7	0
How often did this happen during your pregnancy?	**1= once**	**2= twice**	**3= 3-5 times**	**4= 6-10 times**	**5= 11-20 times**	**6=More than 20 times**	**7= Only before**	**0= never**
31. My partner burned or scalded me on purpose	1	2	3	4	5	6	7	0
32. My partner insisted on oral or anal sex (but did not use physical force)	1	2	3	4	5	6	7	0
33. My partner accused me of being a lousy lover	1	2	3	4	5	6	7	0
34. My partner did something to spite me	1	2	3	4	5	6	7	0
35. My partner threatened to hit or throw something at me	1	2	3	4	5	6	7	0
36. I felt physical pain that still hurt the next day because of a fight with my partner	1	2	3	4	5	6	7	0
37. My partner kicked me	1	2	3	4	5	6	7	0
38. My partner used threats to make me have sex	1	2	3	4	5	6	7	0
39. My partner agreed to a solution I suggested	1	2	3	4	5	6	7	0

Childhood Trauma Questionnaire

The next set of questions asks about some possible experiences you may have had while growing up, before the age of 18. We know that these questions are very personal, and may make you feel somewhat uncomfortable, but please try to answer as honestly as you can. For each question, mark the response that best describes your feelings while growing up as a child and a teenager. The options are: Never True, Rarely True, Sometimes True, Often True and Very Often True.

WHEN I WAS GROWING UP (before the age of 18)…

1. I didn't have enough to eat.
Never True Rarely True Sometimes True Often True Very Often True

2. I knew that there was someone to take care of me and protect me.
Never True Rarely True Sometimes True Often True Very Often True

3. People in my family called me things like "stupid," "lazy," or "ugly."
Never True Rarely True Sometimes True Often True Very Often True

4. My parent(s) were too drunk or high to take care of the family.
Never True Rarely True Sometimes True Often True Very Often True

5. There was someone in my family who helped me feel that I was important or special.
Never True Rarely True Sometimes True Often True Very Often True

6. I had to wear dirty clothes.
Never True Rarely True Sometimes True Often True Very Often True

7. I felt loved.
Never True Rarely True Sometimes True Often True Very Often True

8. I thought that my parent(s) wished I had never been born.
Never True Rarely True Sometimes True Often True Very Often True

9. I got hit so hard by someone in my family that I had to see a doctor or go to the hospital.
Never True Rarely True Sometimes True Often True Very Often True

10. There was nothing I wanted to change about my family.
Never True Rarely True Sometimes True Often True Very Often True

11. People in my family hit me so hard that it left me with bruises or marks.
Never True Rarely True Sometimes True Often True Very Often True

12. I was punished with a belt, a board, a cord, or some other hard object.
Never True Rarely True Sometimes True Often True Very Often True

13. People in my family looked out for each other.
Never True Rarely True Sometimes True Often True Very Often True

14. People in my family said hurtful or insulting things to me.

Never True Rarely True Sometimes True Often True Very Often True

15. I believe that I was physically abused.
Never True Rarely True Sometimes True Often True Very Often True

16. I had the perfect childhood.
Never True Rarely True Sometimes True Often True Very Often True

17. I got hit or beaten so badly that it was noticed by someone like a teacher, neighbor, or doctor.
Never True Rarely True Sometimes True Often True Very Often True

18. I felt that someone in my family hated me.
Never True Rarely True Sometimes True Often True Very Often True

19. People in my family felt close to each other.
Never True Rarely True Sometimes True Often True Very Often True

20. Someone tried to touch me in a sexual way, or tried to make me touch them.
Never True Rarely True Sometimes True Often True Very Often True

21. Someone threatened to hurt me or tell lies about me unless I did something sexual with them.
Never True Rarely True Sometimes True Often True Very Often True

22. I had the best family in the world.
Never True Rarely True Sometimes True Often True Very Often True

23. Someone tried to make me do sexual things or watch sexual things.
Never True Rarely True Sometimes True Often True Very Often True

24. Someone molested me.
Never True Rarely True Sometimes True Often True Very Often True

25. I believe that I was emotionally abused.
Never True Rarely True Sometimes True Often True Very Often True

26. There was someone to take me to the doctor if I needed it.
Never True Rarely True Sometimes True Often True Very Often True

27. I believe that I was sexually abused.
Never True Rarely True Sometimes True Often True Very Often True

28. My family was a source of strength and support.
Never True Rarely True Sometimes True Often True Very Often True

Center for Epidemiologic Studies Depression Scale (CES-D), NIMH

Below is a list of the ways you might have felt or behaved. Please mark how often you have felt this way during the past week.

During the past week…	Rarely or none of the time (less than 1day)	Some or a little of the time (1-2 days)	Occasionally or a moderate amount of time (3-4 days)	Most or all the time (5-7 days)
1. I was bothered by things that usually don't bother me.				
2. I did not feel like eating; my appetite was poor.				
3. I felt that I could not shake off the blues even with help from my family or friends.				
4. I felt I was just as good as other people.				
5. I had trouble keeping my mind on what I was doing.				
6. I felt depressed.				
7. I felt that everything I did was an effort.				
8. I felt hopeful about the future.				
9. I thought my life had been a failure.				
10. I felt fearful.				
11. My sleep was restless.				
12. I was happy.				
13. I talked less than usual.				
14. I felt lonely.				
15. People were unfriendly.				
16. I enjoyed life.				
17. I had crying spells.				
18. I felt sad.				
19. I felt that people dislike me.				
20. I could not get "going."				

Post Traumatic Stress Disorder Checklist – Civilian Version

INSTRUCTIONS: Below is a list of problems and complaints that people sometimes have in response to stressful life experiences. Please read each one carefully, then circle one of the numbers to the right to indicate how much you have been bothered by that problem in the past month.

1 = Not at all
2 = A little bit
3 = Moderately
4 = Quite a bit
5 = Extremely

1. Repeated, disturbing memories, thoughts, or images of a stressful experience from the past? 1 2 3 4 5

2. Repeated, disturbing dreams of a stressful experience from the past? 1 2 3 4 5

3. Suddenly acting or feeling as if a stressful experience were happening again (as if you were reliving it)? 1 2 3 4 5

4. Feeling very upset when something reminded you of a stressful experience from the past? 1 2 3 4 5

5. Having physical reactions (e.g., heart pounding, trouble breathing, sweating) when something reminded you of a stressful experience from the past? 1 2 3 4 5

6. Avoiding thinking about or talking about a stressful experience from the past or avoiding having feelings related to it? 1 2 3 4 5

7. Avoiding activities or situations because they reminded you of a stressful experience from the past? 1 2 3 4 5

8. Trouble remembering important parts of a stressful experience from the past? 1 2 3 4 5

9. Loss of interest in activities that you used to enjoy? 1 2 3 4 5

10. Feeling distant or cut off from other people? 1 2 3 4 5

11. Feeling emotionally numb or being unable to have loving feelings for those close to you? 1 2 3 4 5

12. Feeling as if your future will somehow be cut short? 1 2 3 4 5

13. Trouble falling or staying asleep? 1 2 3 4 5

14. Feeling irritable or having angry outbursts? 1 2 3 4 5

15. Having difficulty concentrating? 1 2 3 4 5

16. Being "super-alert" or watchful or on guard? 1 2 3 4 5

17. Feeling jumpy or easily startled? 1 2 3 4 5

Life Stressor Checklist - Revised

READ THIS FIRST: Now we are going to ask you some questions about events in your life that are frightening, upsetting, or stressful to most people. Please think back over your **whole life** when you answer these questions. Some of these questions may be about upsetting events you don't usually talk about. Your answers are important, but **you do not have to answer any questions that you do not want to**.

1. Have you ever been in a serious disaster (for example, an earthquake, hurricane, large fire, explosion)? YES NO

a. How old were you when this happened? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that ***you or someone else*** could be ***killed*** or seriously ***harmed?*** **YES NO**
d. At the time of the event did you experience feelings of ***intense*** helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

2. Have you ever seen a serious accident (for example, a bad car wreck or an on-the-job accident)? YES NO

a. How old were you when this happened? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that ***you or someone else*** could be ***killed*** or seriously ***harmed?*** **YES NO**
d. At the time of the event did you experience feelings of ***intense*** helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

3. Have you ever had a very serious accident or accident-related injury (for example, a bad car wreck or an on-the-job accident)? YES NO

a. How old were you when this happened? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that ***you or someone else*** could be ***killed*** or seriously ***harmed?*** **YES NO**
d. At the time of the event did you experience feelings of ***intense*** helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

4. Was a close family member ever sent to jail? YES NO

a. How old were you when tis happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**

c. At the time of the event did you believe that ***you or someone else*** could be ***killed*** or seriously ***harmed?*** **YES NO**
d. At the time of the event did you experience feelings of ***intense*** helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

5. Have you ever been sent to jail? YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

6. Were you ever put in foster care or put up for adoption? YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

7. Did your parents ever separate or divorce while you were living with them? YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5

not at all some extremely

8. Have you ever been separated or divorced? YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

9. Have you ever had serious money problems (for example, not enough money for food or place to live)? YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO** c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

10. Have you ever had a very serious physical or mental illness (for example, cancer, heart attack, serious operation, felt like killing yourself, hospitalized because of nerve problems)? YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO** c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

11. Have you ever been emotionally abused or neglected (for example, being frequently shamed, embarrassed, ignored, or repeatedly told that you were "no good")? YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO** c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

12. **Have you ever been physically neglected (for example, not fed, not properly clothed, or left to take care of yourself when you were too young or ill)? YES NO**

a. How old were you when this happened? _____ b. When it ended? _____

ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO** c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**

d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**

e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

13. Have you ever had an abortion or miscarriage (lost your baby)? YES NO

a. How old were you when this happened? _____

ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**

c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**

d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**

e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

14. Have you ever been separated from your child against your will (for example, the loss of custody or visitation or kidnapping? YES NO

a. How old were you when this happened? _____ b. When it ended? _____

ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO** c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**

d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**

e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

15. **Has a baby or child of yours ever had a severe physical or mental handicap (for example, mentally retarded, birth defects, can't hear, see, walk)? YES NO**

a. How old were you when this happened? _____ b. When it ended? _____

ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**

c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**

d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**

e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

16. Have you ever been responsible for taking care of someone close to you (not your child) who had a severe physical or mental handicap (for example, cancer, stroke, AIDS, nerve problems, can't hear, see, walk)? YES NO

a. How old were you when this happened? _____ b. When it ended? _____

ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**

c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**

d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**

e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

17. Has someone close to you died suddenly or unexpectedly (for example, sudden heart attack, murder or suicide)? YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**

e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

18. Has someone close to you died (do NOT include those who died suddenly or unexpectedly)? YES NO

a. How old were you when this happened? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

19.When you were young (before age 16) did you ever see violence between family members (for example, hitting, kicking, slapping, punching)? YES NO

a. How old were you when this happened? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

20. Have you ever seen a robbery, mugging, or attack taking place? YES NO

a. How old were you when this happened? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

21. Have you ever been robbed, mugged, or physically attacked (not sexually) by someone you did not know? YES NO

a. How old were you when this happened? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or

seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

22. *Before age 16*, were you ever abused or physically attacked (not sexually) by someone you knew (for example, a parent, boyfriend, or husband, hit, slapped, choked, burned, or beat you up)?
YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

23. *After age 16*, were you ever abused or physically attacked (not sexually) by someone you knew (for example, a parent, boyfriend, or husband hit, slapped, choked, burned, or beat you up)?
YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

24. Have you ever been bothered or harassed by sexual remarks, jokes, or demands for sexual favors by someone at work or school (for example, a coworker, a boss, a customer, another student, a teacher)? YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

***25. Before age 16*, were you ever touched or made to touch someone else in a sexual way because he/she forced you in some way or threatened to harm you if you didn't? YES NO**

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**

e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

***26. After age 16,* were you ever touched or made to touch someone else in a sexual way because he/she forced you in some way or threatened to harm you if you didn't? YES NO**

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

27. *Before age 16*, did you ever have sex (oral, anal, genital) when you didn't want to because someone forced you in some way or threatened to hurt you if you didn't? YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

28. After age 16, did you ever have sex (oral, anal, genital) when you didn't want to because someone forced you in some way or threatened to harm you if you didn't? YES NO

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

29. Are there any events we did not include that you would like to mention? YES NO
What was the event?_____

a. How old were you when this happened? _____ b. When it ended? _____
ai. Did this happen during your pregnancy (with baby you brought in today)? **YES NO**
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

30. Have any of the events mentioned above ever happened to someone close to you so that even though you didn't see it yourself, you were seriously upset by it? YES NO

What was the event?_____

a. How old were you when this happened? _____ b. When it ended? _____
c. At the time of the event did you believe that *you or someone else* could be *killed* or seriously *harmed?* **YES NO**
d. At the time of the event did you experience feelings of *intense* helplessness, fear, or horror? **YES NO**
e. How much is this affected your life in the past year? 1 2 3 4 5
not at all some extremely

Conflict Tactics Scale (Post-partum)

Relationship Behaviors

No matter how well a couple gets along, there are sometimes when they disagree, get annoyed with the other person, want different things from each other, or just have spats or fights because they are in a bad mood, are tired, or for some other reason. Couples also have many different ways of trying to settle their differences. This is a list of things that may happen when you have differences. Please mark how many times your partner did these things to you since your infant's birth. If your partner did not do one of these things since your baby's birth, mark "0" (even if this has happened during pregnancy or before pregnancy).

1 = Once since my baby's birth
2 = Twice since my baby's birth
3 = 3-5 times since my baby's birth
4 = 6-10 times since my baby's birth
5 = 11-20 times since my baby's birth
6 = More than 20 times since my baby's birth
0 = My partner has not done this to me since my baby's birth

How often did this happen since your baby's birth?	**1= once**	**2= twice**	**3= 3-5 times**	**4= 6-10 times**	**5= 11-20 times**	**6=More than 20 times**	**0= never**
1. My partner showed care for me even though we disagreed	1	2	3	4	5	6	0
2. My partner explained his or her side of a disagreement to me	1	2	3	4	5	6	0
3. My partner insulted or swore at me	1	2	3	4	5	6	0
4. My partner threw something at me that could hurt	1	2	3	4	5	6	0
5. My partner twisted my arm or hair	1	2	3	4	5	6	0
6. I had a sprain, bruise, or small cut because of a fight with my partner	1	2	3	4	5	6	0
7. My partner showed respect for my feelings about an issue	1	2	3	4	5	6	0
8. My partner made me have sex without a condom	1	2	3	4	5	6	0
9. My partner pushed or shoved me	1	2	3	4	5	6	0
10. My partner used force (like hitting, holding down, or using a weapon) to make me have oral or anal sex	1	2	3	4	5	6	0
11. My partner used a knife or gun on me	1	2	3	4	5	6	0

How often did this happen since your baby's birth?	**1= once**	**2= twice**	**3= 3-5 times**	**4= 6-10 times**	**5= 11-20 times**	**6=More than 20 times**	**0= never**
12. I passed out from being hit in the head by my partner in a fight	1	2	3	4	5	6	0
13. My partner called me fat or ugly	1	2	3	4	5	6	0
14. My partner punched or hit me with something that could hurt	1	2	3	4	5	6	0
15. My partner destroyed something belonging to me	1	2	3	4	5	6	0
16. I went to a doctor because of a fight with my partner	1	2	3	4	5	6	0
17. My partner choked me	1	2	3	4	5	6	0
18. My partner shouted or yelled at me	1	2	3	4	5	6	0
19. My partner slammed me against a wall	1	2	3	4	5	6	0
20. My partner was sure we could work out a problem	1	2	3	4	5	6	0
21. I needed to see a doctor because of a fight with my partner, but I didn't	1	2	3	4	5	6	0
22. My partner beat me up	1	2	3	4	5	6	0
23. My partner grabbed me	1	2	3	4	5	6	0
24. My partner used force (like hitting, holding down, or using a weapon to make me have sex	1	2	3	4	5	6	0
25. My partner stomped out of the room, or house, or yard during a disagreement	1	2	3	4	5	6	0
26. My partner insisted on sex when I did not want to (but did not use physical force)	1	2	3	4	5	6	0
27. My partner slapped me	1	2	3	4	5	6	0
28. I had a broken bone from a fight with my partner	1	2	3	4	5	6	0
29. My partner used threats to make me have oral or anal sex	1	2	3	4	5	6	0
30. My partner suggested a compromise to a disagreement	1	2	3	4	5	6	0
31. My partner burned or scalded me on purpose	1	2	3	4	5	6	0
How often did this happen since your baby's birth?	**1= once**	**2= twice**	**3= 3-5**	**4= 6-10**	**5= 11-20**	**6=More than 20**	**0= never**

			times	**times**	**times**	**times**	
32. My partner insisted on oral or anal sex (but did not use physical force)	1	2	3	4	5	6	0
33. My partner accused me of being a lousy lover	1	2	3	4	5	6	0
34. My partner did something to spite me	1	2	3	4	5	6	0
35. My partner threatened to hit or throw something at me	1	2	3	4	5	6	0
36. I felt physical pain that still hurt the next day because of a fight with my partner	1	2	3	4	5	6	0
37. My partner kicked me	1	2	3	4	5	6	0
38. My partner used threats to make me have sex	1	2	3	4	5	6	0
39. My partner agreed to a solution I suggested	1	2	3	4	5	6	0