BEHAVIORAL AND PHYSIOLOGICAL REACTIVITY TO STRESS IN INFANTS EXPOSED TO INTIMATE PARTNER VIOLENCE

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ABSTRACT

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Abnormal physiological and behavioral responses to stress as a result of early life stress may predispose children to adverse mental health consequences, such as PTSD, in response to new traumatic events during adolescence and adulthood. Research also indicates that maternal stress in the prenatal period may affect biobehavioral alterations in the offspring. Interestingly, infants who experience physiological hyperreactivity do not necessarily display heightened behavioral distress, and the regulation of distress may be associated with high cortisol reactivity. More work is needed to understand how physiological and behavioral components of the stress response system are integrated. The current study examined whether 12-month-old infants exposed to specific stressful events, intimate partner violence (IPV) prenatally and/or postnatally, compared to nonexposed infants, display a distinct pattern of behavior and cortisol reactivity when undergoing a laboratory stress task. Latent profile analysis was used to create profiles of infants' (N=182) behavioral and cortisol responses; a three-class solution emerged. While prenatal IPV predicted membership in a profile characterized by high cortisol reactivity, emotion reactivity and regulation did not distinguish the groups. Post-hoc analyses indicated that exposure to both pre- and postnatal IPV is associated with cortisol reactivity *and* emotion stability. Thus, while exposure to prenatal IPV alone may be a risk factor for physiological reactivity, exposure to chronic IPV in the prenatal and postnatal period may be more likely to affect the regulation of emotional responses. Findings are discussed in relation to the Emotional Security Hypothesis.

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INTRODUCTION

Examining relationships among the biological and behavioral indicators of coping in early childhood may illuminate pathways of vulnerability and resilience to stress across the lifespan (Davies, Sturge-Apple, Cicchetti, & Cummings, 2007; Gunnar & Quevedo, 2007a). Past trauma is a known risk factor for maladaptive reactions to trauma later in life (Delahanty, & Nugent, 2006; Schoedl, Costa, Fossaluza, Mari, & Mello, 2013). Alterations in cortisol and behavioral responses as a result of dealing with early stress may predispose children to adverse mental health consequences, such as PTSD, in adolescence and adulthood, when they are exposed to a new traumatic event (e.g., Bomyea, Risbrough, & Lang, 2012). Research also indicates that maternal stress in the prenatal period may affect biobehavioral alterations in the offspring (Davis, Glynn, Schetter, Hobel, Chicz-Demet, & Sandman, 2007; Glover, O'Connor, & O'Donnell, 2010). Extant studies of early trauma typically examine either physiology or overt behavior, but more work is needed to understand how these different components of the stress response system are integrated (Granger & Kivlighan, 2003). Intimate partner violence (IPV) is a common stressor for pregnant and parenting women that has deleterious effects on infant emotional and physiological functioning (Carpenter & Stacks, 2009). The current study examined whether infants who are exposed to IPV prenatally and/or postnatally display a distinct pattern of behavior and cortisol reactivity, compared to non-exposed infants, when undergoing a laboratory stress task.

Cortisol

The hypothalamic-pituitary-adrenal (HPA) axis is a complex system that, in part, regulates the body's response to stress. However, baseline levels (basal) of cortisol are present at all times and circulate in the bloodstream based on circadian rhythms that are stable by two years

of age (Stansbury & Gunnar, 1994). Upon perception of stress, the hypothalamus sets in motion a chain of events that leads to the release of cortisol into the bloodstream from the adrenal gland (Gunnar & Quevedo, 2007b). This physiological stress response helps the body respond to stress in the short term, but repeated or chronic elevations in cortisol production can contribute to adverse outcomes in physical and psychological functioning (e.g., Charmandari, Tsigos, & Chrousos, 2005).

Developmentally, the HPA axis response to stress becomes less reactive over the first year of life in typically developing infants (Jansen, Beijers, Riksen-Walraven, & de Weerth, 2010; Tollenaar, Jansen, Beijers, Riksen-Walraven, & de Weerth, 2010). However, infants exposed to traumatic events may deviate from this expected blunting of cortisol reactivity (Hibel, Granger, Blair, & Cox, 2011; Saltzman, Holden, & Holahan, 2005). Thus, the development of the HPA axis is sensitive to early experience, and lasting alterations in the stress response may serve as a risk factor for the development of psychopathology. Research shows that different forms of psychopathology are associated with distinct patterns of HPA axis dysregulation, which are affected by early experience (Gunnar & Quevedo, 2007b; Pervanidou, 2008). Patterns of HPA axis dysregulation differ by the particular disorder and the developmental period studied. Depressed preschool children demonstrate higher levels of circulating basal cortisol and blunted reactivity to psychological stressors compared to nondepressed peers (Lopez-Duran, Kovacs, & George, 2009). In adults, depression is consistently associated with hypercortisolism (Southwick, Vythilingam, & Charney, 2005; Pariante & Lightman, 2008), while patients with PTSD often show low basal levels of cortisol but heightened cortisol reactivity to stress (Meewisse, Reitsma, De Vries, Gersons, & Olff, 2007). Because these patterns of HPA axis dysregulation are sensitive to early experience, it is important to study how characteristics of

children's early environments alter cortisol functioning and associated vulnerability to psychopathology (Bomyea, Risbrough, & Lang, 2012; Tarullo & Gunnar, 2006).

The effects of stress on HPA axis development may occur during the prenatal period. During pregnancy, the fetal environment is affected by maternal cortisol levels, which rise naturally during the course of the pregnancy but are also influenced by external stressors (O'Donnell, O'Connor, & Glover, 2009). In one proposed mechanism of prenatal programming, stress-induced elevations in a woman's cortisol levels can permeate the placenta, and linear correlations are sometimes found between cortisol levels of the mother and fetus in humans (Talge, Neal, & Glover, 2007). High levels of maternal stress during pregnancy may initiate the long-term programming of the HPA axis of the child (for a review, see Glover, O'Connor, & O'Donnell, 2010).

Prenatal stress is also associated with behavioral distress in infancy (Pleuss & Belsky, 2011). High levels of fussiness, irritability and emotional problems are found in infants and children of mothers who experienced anxiety or were exposed to stressful events during pregnancy, compared to non-exposed infants (Weinstock, 2008). For example, Davis and colleagues (2007) found that third trimester elevated maternal cortisol and maternal anxiety were associated with negative infant reactivity at eight weeks postpartum. Results suggest that prenatal stress affects behavior as well as the physiological stress response in infancy.

Hence, environmental factors during pregnancy and the first postnatal years likely contribute to the development of the HPA axis, emotional functioning, and later vulnerability to stress. However, little research examines both prenatal and postnatal stressors when considering infant biobehavioral outcomes. This line of research is important because the effect of prenatal stress on the HPA axis may interact with aspects of the postnatal environment to further shape

infant outcomes. Additionally, while both physiological and behavioral differences are found in infants exposed to stress prenatally, most studies of prenatal stress do not examine the coordination (or lack of) between expressed emotion and the physiological response.

Behavior and Cortisol in Infancy

Physiological and behavioral responses to stress do not always correspond. For example, infants who experience physiological hyperreactivity do not necessarily display heightened behavioral distress, and the regulation of distress may be associated with high cortisol reactivity (Lewis, 2011). Studies of behavior-cortisol relationships in infants often divide behavioral responses into emotion reactivity and emotion regulation. Emotion reactivity includes aspects of the infant's initial response to a stressor, including the intensity of response, latency to response, response threshold and overall intensity of response behaviors such as crying (Rothbart, 1989; Eisenberg, Fabes, Murphy, Maszk, Smith, & Karbon, 1995). Regulation involves the infant's ability to respond to this reactivity and modulate affective arousal (Calkins, Gill, Johnson, & Smith, 2001; Thompson, 1994). With development, infants increasingly employ internal and external strategies that function to initiate, extend, or alter their emotional experiences (Thompson, 1994). This regulation unfolds in the first year of life with help from caregivers who interpret and respond to the infant's affective cues, thereby serving as an external regulatory tool (Stifter, 2002). Because emotion reactivity and emotion regulation are implicated in behavior-cortisol relationships, it is important to assess both the intensity of the behavioral response (reactivity) as well as the pattern of modulation over time (regulation).

The relationship between emotion reactivity and stressed cortisol has been studied extensively in newborns, but few consistent biobehavioral associations have been found after the first few months of life (Lewis, 2011). For example, while the intensity of crying and cortisol

reactivity is linked in newborns exposed to mild physical stressors, this does not appear to be a consistent relationship across development (Gunnar & Donzella, 1999; Gunnar, Talge, & Herrera, 2009; Jansen et al., 2010). In another study, newborns' responses to a heel stick procedure showed that the HPA axis response habituates over the course of days, while crying remains stable (Gunnar, Hertsgaard, Larson, & Rigatuso, 2004).

Research with infants aged 6 months or older indicates that emotion reactivity and cortisol responses to psychological stressors are not necessarily linked (Ursache, Blair, Granger, Stifter, & Voegtline, 2013). van Bakel and Riksen-Walraven (2004) found that behavioral indicators of fear (e.g., crying, proximity seeking) were associated with cortisol reactivity during a robot-approach task in 15-month-olds. However, two studies of 24-month-olds did not find associations between expressed fear and cortisol responses during similar tasks (Buss, Davidson, Kalin, & Goldsmith, 2004; Fortunato, Dribin, Granger, & Buss, 2008). Research has also investigated infants' and toddlers' responses to tasks that elicit frustration. There is some indication that the intensity of behaviorally coded anger is associated with cortisol reactivity (Lewis & Ramsay, 2005; Schuetze, Lopez, Granger, & Eiden, 2008), but Ursache and colleagues (2013) found that cortisol-behavior relationships were more robust at 7 months than at 15 and 24 months of age in similar tasks.

In a review of cortisol-behavior relationships, Lewis (2011) notes that behavioral and cortisol reactivity are usually dysynchronous by 12 months of age. Stansbury and Gunnar (1994) note that, "While states of negative affect do appear to be associated with elevations in cortisol, it is not clear that they are sufficient to cause activation of the HPA stress circuits" (p. 120). Inconsistent findings on linear behavior-reactivity relationships have led researchers to consider whether emotion regulation, parenting, past stress and the complex relationships among these

factors are better predictors of the biobehavioral stress response (Gunnar & Donzella, 1999; 2002).

Stansbury and Gunnar (1994) propose that uncertainty about one's ability to control one's emotions, rather than the level of emotional reactivity itself, predicts heightened cortisol reactivity. Caregiver-supported emotion regulation increases during the first few years of life and is critical to healthy social-emotional functioning (Eisenberg, Spinrad, & Eggum, 2010). As emotion regulation develops in infancy, it may start to influence the relationship between cortisol and behavioral reactivity. Lewis and Ramsay (1995) distinguished between emotional reactivity and regulation and measured both in relation to cortisol reactivity in a longitudinal study with infants at two, four, six and 18 months of age. Interestingly, cortisol and behavior were not related at six months (Ramsay & Lewis, 2003). At 18 months, however, emotion regulation (measured as quieting after exposure to a stressful event), and not reactivity, was positively associated with cortisol reactivity. Additionally, Ursache and colleagues (2013) found that regulatory behaviors were associated with cortisol increases in response to a barrier task in 7-month-old infants. It appears that the infant's nascent ability to regulate emotion may activate the HPA axis response. In sum, initial behavioral reactivity, along with the modulation of that response, might differentially influence the cortisol response. As such, measures of both emotion reactivity and regulation are important to include in research with this age group to help resolve this debate.

Links between behavior and cortisol may also depend on trait-like individual differences in emerging emotion regulation. There is evidence to suggest that in early childhood, overcontrolled and undercontrolled regulatory responses are both associated with elevated cortisol reactivity to a stressful event. Studies with toddlers and preschoolers find that

behavioral inhibition and withdrawal behaviors can be associated with heightened basal and/or reactive cortisol levels (Fortunato, Dribin, Granger, & Buss, 2008; Granger, Stansbury, & Henker, 1994), although results are inconsistent across studies (Granger & Kivlighan, 2003). In work with preschoolers, Spinrad and colleagues (2009) examined HPA correlates of emotional expression in preschoolers and found that high levels of regulation, but not observed anger, were positively associated with cortisol reactivity. More work is needed to examine how a child's struggle to modulate emotion over time relates to emotional reactivity and HPA activation. Particularly sparse is research on regulation and HPA reactivity when infants are 12 months of age, a time when emotion regulation is emerging.

Environmental Stress

Environmental stressors also influence development of the biobehavioral stress response system, starting with fetal development. Exposure to stress *in utero* leads to changes in stress reactivity and behavior in infancy and beyond (Sandman, Davis, Buss, & Glynn, 2011; Van den Bergh, Van Calster, Smits, Van Huffel, & Lagae, 2008). In a review of the literature on prenatal stress and child HPA axis functioning, Glover and colleagues (2010) caution that interpreting results across studies is problematic due to methodological differences, such as the type of prenatal stress studied. Most extant research focuses on either discrete, rare stressful events (e.g., 9/11, ice storm – Yehuda, Engel, Brand, Seckl, Marcus, & Berkowitz, 2005; Laplante, Brunet, Schmitz, Ciampi, & King, 2008) or the effects of stress (e.g., maternal anxiety – Grant, McMahon, Austin, Reilly, Leader, & Ali, 2009; Van den Bergh, Mulder, Mennes, & Glover, 2005). More research is needed examining the effects of common stressors on child HPA axis functioning.

Intimate Partner Violence. IPV is a significant and prevalent stressor in the lives of pregnant and parenting women. A recent national survey of IPV found that 35.6% of women have reported physical violence, rape or stalking by an intimate partner in their lifetime (CDC, 2011). Women of childbearing age experience greater risk, as younger age is associated with increased rates of IPV (Abramsky et al., 2011). One review of 18 studies published from 1996 to 2010 found that rates of physical violence during pregnancy ranged from 1% to 30%, with most estimates ranging from 3% to 11% (Taillieu & Brownridge, 2010). Unfortunately, there is little research on how IPV during pregnancy affects the developing neurobiology of the human fetus (an exception is Radtke et al., 2011). While exposure to prenatal IPV is associated with externalizing symptoms in 12-month-old infants (Levendosky et al., 2006), questions remain about how infants' patterns of biobehavioral stress response contribute to behavior problems in young children exposed to violence *in utero*.

After birth, exposure to IPV in the home continues to influence infants' behavioral and physiological reactivity to stress. Rates of IPV exposure are high in young children, with one study finding that children six years of age and younger are disproportionately exposed to IPV compared to older age groups (Fantuzzo & Fusco, 2007). Exposure to IPV early in life is associated with altered HPA axis functioning (e.g., Hibel, Granger, Blair, & Cox, 2011; Pendry & Adam, 2007; Saltzman, Holden, & Holahan, 2005). Additionally, exposure to IPV in the first year of life is associated with heightened facial distress to simulated conflict (DeJonghe, Bogat, Levendosky, von Eye, & Davidson, 2005). Early physiological and emotional dysregulation may be crucial in explaining well-documented negative child outcomes of internalizing and externalizing symptoms in the aftermath of IPV exposure (e.g., Evans, Davies, & DiLillo, 2008; Kitzmann, Gaylord, Holt, & Kenny, 2003).

Towe-Goodman and colleagues (2012) used latent profile analysis to examine biobehavioral profiles of stress response in infants exposed to IPV. Basal cortisol levels, cortisol reactivity to a challenge task, peak negative behavioral reactivity, and global ratings of unhappiness and irritability were included in the model. A four-class model provided the best fit for the data, with over half of the sample characterized as "low reactors" (relatively low cortisol and behavioral reactivity). The study found that postnatal exposure to IPV increased the likelihood of membership in the profile that involved the greatest cortisol reactivity, but only moderate behavioral distress. These infants were also less likely to employ the regulatory strategy of orienting to a caregiver when stressed. The authors suggest that in this profile, infants do not communicate nor attempt to regulate distress despite pronounced physiological activation. This profile may indicate an attempt, on the part of the infant, to mask distress in a dangerous home context.

Although postnatal exposure to IPV may predispose infants to moderate behavioral reactivity with a high cortisol response, no extant studies have considered how *prenatal* exposure to IPV might contribute to behavioral and physiological patterns of reactivity in infancy. IPV during the prenatal period is especially important to explore because the dysregulating effects of fetal exposure to HPA axis activation can lead to physiological and behavioral alterations in offspring (Glover, O'Connor, & O'Donnell, 2010; Weinstock, 2008). Cortisol-behavior relationships likely rely on the environmental experiences of the child (Towe-Goodman, 2012) and prenatal influences may be especially pernicious due to the biological mechanisms underlying the prenatal programming of the stress response.

Parenting. Parenting is another environmental factor that can affect behavior and physiology in developing infants. Parental relationships that enhance infant coping are thought

to attenuate the relationship between behavioral distress and an elevated cortisol response to stress (Gunnar & Quevedo, 2007b). As noted earlier, before children are able to control their emotions, infants rely on caregivers to respond to their distress cues and meet their physical needs. As they develop, infants internalize responsive caregiving patterns and gain an increased sense of control in recognizing and modulating their own distress (Morris, Silk, Steinberg, Myers, & Robinson, 2007). This gradual shift from interdependent to independent regulation of affect engenders differences in affect reactivity and regulation (Gunnar & Donzella, 2002).

The relationship between behavioral distress and cortisol reactivity may also vary by the quality of the parenting or caretaking relationship. In one study, 9-month-olds who cried in response to being left with a babysitter showed elevations in cortisol reactivity only when the babysitter was minimally responsive (rather than nurturing), but caregiving quality was not related to the cortisol response in infants with no overt signs of distress (Gunnar, 1992). Similarly, studies of behavioral distress, parenting relationships, and cortisol in infants find elevated cortisol reactivity only in behaviorally dysregulated infants *with* insecure attachment relationships (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996; Schieche & Spangler, 2005). Nachmias and colleagues (1996) propose that insensitive parenting may have overwhelmed inhibited infants' ability to cope during the stress task in their study. This line of research suggests that infants who can reliably receive sensitive parenting in times of stress do not react physiologically even if they show signs of behavioral distress by crying, freezing or withdrawing. Gunnar (1998) emphasizes that responsive parenting is a buffer against the potential risk factor of behavioral inhibition in infants. Before the development of self-regulation, parents can enhance coping and the perceived controllability of the stressor. As such,

it is important to examine characteristics of parenting behavior when considering patterns of emotion reactivity, emotion regulation and cortisol reactivity.

While interpretations of the above studies view attachment status as an indicator of the ongoing quality of the parent-infant relationship (e.g., Gunnar & Donzella, 2002), there is also a need to determine how specific parenting behaviors relate to individual differences in the stress response system. Importantly, IPV in the home can disrupt parenting behavior (e.g., Levendosky & Graham-Bermann, 2000), which can then contribute to negative child outcomes (e.g., Levendosky, Leahy, Bogat, Davidson, & von Eye, 2006). A meta-analysis of the parenting literature found support for a negative relationship between interparental conflict and parenting, with the strongest effects emerging for associations between interparental hostility and high levels of harsh discipline and low levels of parental acceptance, including nurturing behaviors (Krishnakumer & Buehler, 2000). Individual differences in parenting behaviors related to experiences of IPV are conceptualized as influencing the relationship between IPV and biobehavioral infant outcomes (Sturge-Apple, Davies, Cicchetti, & Manning, 2010). For example, Hibel, Granger, Blair and Cox (2011) found that responsive parenting at seven months moderated the relationship between IPV and cortisol hyperreactivity in two-year-olds. Parenting relationships are therefore linked to the continued development of HPA axis functioning after birth.

Summary

In summary, linear relationships between infant emotional reactivity, emotion regulation, and cortisol reactivity are sometimes found, but results are inconsistent. Emotion reactivity and emotion regulation in response to stress are independently linked to subsequent cortisol reactivity. However, emotional and cortisol reactivity may start to dissociate from cortisol

reactivity as emotion regulation increases during the first year of life (Lewis, 2011; Stansbury & Gunnar, 1994). Few studies consider how both emotion reactivity and emotion regulation relate to cortisol reactivity in infants. Research suggests that biological and behavioral systems operate through complex relationships, and that multilevel measures are needed to understand the infant stress response (Granger & Kivlighan, 2003; Ramsay & Lewis, 2003). For this reason, person-centered analyses have been used successfully to create profiles of biobehavioral responses to stress with infants (Towe-Goodman et al., 2012) and older children (Zalewski et al., 2011).

Additionally, environmental factors such as prenatal and postnatal exposure to trauma and postnatal caregiving may influence the relationship between the physiological and behavioral components of the stress response. Unfortunately, most research does not include an assessment of both prenatal and postnatal stressors. The current study used a retrospective measure of prenatal IPV to explore whether *prenatal* as well as *postnatal* exposure to IPV stress influences the biobehavioral stress response system. Latent profile analysis was used to create biobehavioral profiles of behavioral and cortisol reactivity in a sample of 12-month-olds exposed to a mild laboratory stressor. While many infants were expected to demonstrate low behavioral and cortisol reactivity in combination, infants with varying degrees of cortisol reactivity were expected to decompose into groups demonstrating different levels of emotional responses. We expected to find one profile of infants who showed low to moderate behavioral distress and limited regulation in tandem with high cortisol reactivity. Following Towe-Goodman et al. (2012), this pattern may be associated with exposure to IPV. We predicted that infants without a history of exposure to IPV would display higher behavioral distress with less cortisol reactivity. Analyses examined how prenatal and postnatal factors, such as exposure to IPV and nurturing parenting, differentiated these profiles.

METHOD

Participants

Participants were 182 women and their 12-month-old infants recruited from mid-Michigan. Participants learned of the study through fliers posted in the community and online advertisements on Facebook and Craigslist. Locations for the physical fliers were chosen in an attempt to recruit women with young children and women exposed to IPV. Flier locations included pediatrician and OBGYN offices, preschools and daycare centers, local domestic violence shelters, and the Department of Human Services of Ingham and Wayne County. Women were required to be English-speaking, 18 to 34 years old, not pregnant, not lactating or willing to refrain from breastfeeding for 2 hours prior to the appointment and involved in a heterosexual relationship for at least 6 weeks during the pregnancy. Infants born prematurely (under 37 weeks gestation) were excluded, as premature birth is associated with alterations in HPA axis functioning (Grunau, Haley, Whitfield, Weinberg, Yu, & Thiessen, 2007). Additionally, women were screened out if they had endocrine or other disorders associated with abnormal glucocorticoid functioning (Cushings or Addison's Disease, cancer or cancer therapy; Golden, Malhotra, Wand, Brancati, Ford, & Horton, 2007). Efforts were made to ensure that women with IPV exposure matched women without IPV exposure on five key characteristics (age, race/ethnicity, income, relationship/cohabitation status and education status). However, chi-squared tests revealed a significant difference in mother's relationship status by IPV group (IPV endorsed or not endorsed), $\chi^2(2, N=182) = 24.54$, $p < .001$. Income level (below or above Medicaid cutoff) also differed by IPV group, $\chi^2(1, N=182) = 6.72$, $p = .010$. No significant differences in age, race/ethnicity or education status were found between groups.

Women's mean age was 24.5 years. The average monthly income was $1170 and 51% had a high school diploma or less. Fifty-one percent of participants were single and living alone. Women's ethnicity was 15% multiracial, 33% African American, 43% Caucasian and 9% Latino. Children were between the ages of 11 and 15 months, with a mean age of 11.7 months and a standard deviation of 2.44; 91 were female and 91 were male. Infants' ethnicity was 36% multiracial, 29% African-American, 28% Caucasian and 7% other race.

Measures

Cortisol. Research assistants collected cortisol samples from infants by placing hydrocellulose microsponges in their mouths to swab for saliva. Samples were refrigerated at 4°C following the interview and then frozen and stored at -80°C. To extract saliva, samples were thawed at 4°C and centrifuged for 15 minutes at 1300 rpm. Cortisol was assayed using a commercially available EIA assay kit manufactured by Salimetrics, LLC. Basal cortisol was calculated by taking the mean of the first saliva sample and the five-minute saliva sample. Cortisol reactivity was calculated by subtracting the basal cortisol level from the peak cortisol level sampled at either 20- or 40- minutes post task, depending on which sample was higher.

Emotional Reactivity and Emotion Regulation. A modified version of the Arm Restraint task from the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith & Rothbart, 1999) was implemented (see procedures below for more detail) and coded for emotional reactivity and emotion regulation. Videos of the task were divided into 12, ten-second intervals. Interval coding of facial, vocal and bodily indicators of emotion reactivity is a standard procedure (e.g., Ramsay & Lewis, 2003; Robinson & Acevedo, 2001). For each interval, reactivity codes were assigned that included observations of infants' facial, vocal and bodily distress. Infants were coded on a reactivity scale from zero to three (0 = No reactivity; 1 = Mild

reactivity; 2 = Moderate negative reactivity; 3 = High negative reactivity) for each interval. For example, a score of zero was given if infants showed no negative or facial affect and/or vocalizations. A score of two was given if infants exhibited a full cry, wide squared mouth, eyes open or partially open or negative body movements. The interval of peak distress was also coded. A second coder scored reactivity for each interval and the interval of peak distress for one-third of the sample. Weighted kappa was .76 in a 22% double coding of the data.

An emotion reactivity global score was calculated by taking the mean of the reactivity scores across all intervals. Emotion regulation was operationalized as the pattern of emotion reactivity over time. The variable 'upregulation' is the sum score of reactivity score increases from one interval to the next across the six intervals. The variable 'downregulation' is the sum score of decreases across the six intervals. An emotion regulation ratio score was calculated, defined as the ratio of upregulation to downregulation. A constant of one was added to both variables before the ratio was calculated. The emotion regulation ratio score ranges from 0 to 4. A ratio score of 1 corresponds to an equal up- to down-regulation score, which indicates stability, or an equal number of increases and decreases in reactivity over the task. This variable was labeled "stable lability."[1] Scores greater than 1 indicate more upregulation than downregulation; this variable was labeled "negative lability." Scores lower than 1 indicate more downregulation than upregulation; this variable was labeled "positive lability."

Intimate Partner Violence. The Severity of Violence Against Women Scale (SVAWS; Marshall, 1992) is a 46-item questionnaire including items that ask about threats of violence, physical violence and sexual violence from a male partner in the last year. Items include,

[1] Infants could also obtain an emotion regulation ratio equal to one by displaying high values of up- and downregulation that were even in proportion. However, this pattern did not occur in the stable lability group. Only three infants in this group made more than two changes across the six intervals.

"Pushed and shoved you," and, "Physically forced you to have sex." Women rate items on a four-point scale ranging from "Never " to "Many Times." Women in the study were asked to complete the SVAWS retrospectively about the pregnancy period. They also completed a separate SVAWS with their child's first year of life in mind. An event history calendar was employed to help women remember incidents of IPV in relation to other personally-relevant temporal landmarks (Belli, 1998). Coefficient alphas for the SVAWS items were .98 for the prenatal time period and .98 for the postnatal period. Two separate summed scores were created from these two questionnaires to represent frequency of prenatal IPV and postnatal IPV.

Nurturing Parenting. The Parenting Behavior Checklist (PBC) is a 100-item self-report measure of parenting behaviors and expectations used with mothers of infants from one to four years old (Fox, 1994). The nurturing subscale of 20 questions was derived from a component factor analysis that yielded three factors of Expectations, Discipline and Nurturing. Items that load on the nurturing subscale include "My child and I play together on the floor" and "If my child is overactive, I involve him/her in quiet activities." Higher scores indicate parent's greater use of positive nurturance behaviors. Cronbach's α for the Nurturing subscale was .83 for this sample.

Cumulative Risk. A cumulative risk score was created to account for maternal income, marital status, age, past negative life events and drug use. Five dichotomous variables were created and summed: income (below Medicaid cutoff = 1), marital status (single = 1), age (below 22 years = 1), negative life events using the Life Experiences Survey (Sarason, Johnson, & Siegel, 1978) (highest 25% percentile = 1), and drug use as measured with the Perinatal Risk Assessment Monitoring Survey (Gilbert, Shulman, Fischer, & Rogers, 1999) (any street drug use pre- or postnatal = 1). The cumulative risk score ranged from 0 to 5.

Infant Externalizing Symptoms. The Infant Social and Emotional Assessment (ITSEA; Carter, Briggs-Gowan, Jones, & Little, 2003) was administered to assess social and emotional problems and competencies. Mothers were given a list of statements about her child and rated their responses on a 3-point scale from "Not true/rarely" to "Very true/often." Sample items include "has temper tantrums" and "is restless and can't sit still. Cronbach's α for the externalizing scale in this sample was .89.

Procedures

Research visits were conducted at Michigan State University (MSU) and an MSU-Detroit Partnership site. Visits were scheduled as close as possible to the child's first birthday. Assessments were held between 1:00pm and 4:00pm to avoid time-based effects of the diurnal rhythm of cortisol. Mothers were instructed to refrain from feeding infants sugary or acidic foods one hour prior to the assessment. Upon arrival, maternal and infant health was assessed and visits were rescheduled if infant's temperature exceeded 101° F. The sequence of tasks then proceeded as follows: (1) primary saliva collection, (2) Lab-TAB Arm Restraint challenge task, (3) 5 minutes post-task saliva collection, (4) mother begins filling out questionnaire measures, (5) 20 minutes post-task saliva collection, (6) continued completion of questionnaires, (7) 40 minutes post-task saliva collection and (7) questionnaire completion.

A modified version of the Arm Restraint task from the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith & Rothbart, 1999) was used to elicit negative affect and cortisol reactivity. In this task, the infant was first placed in a high chair and a research assistant offered the infant an attractive toy and elicited interaction. After two minutes, the research assistant moved behind the infant and constrained the infant's arms by moving the arms to the side of the body. The research assistant continued to hold the arms in this position so that

the infant could not pull free until the end of the two-minute episode. If the infant cried hard for twenty seconds, the episode was terminated early. During the Arm Restraint task, the infant's mother was seated behind the infant so that she could monitor the task but could not be seen by the infant.

RESULTS

Data Preparation

First, data was cleaned and evaluated for missingness. Of the 182 infants, 39 did not have videos due to technical problems (N=26) or because the baby was too upset to undergo the procedure (N=13). Notably, the length of the arm restraint task varied by case because many of the procedures were cut short when the infant displayed a hard cry for 20 seconds. Of the 143 infants with video data, 30 infants terminated before completing the first minute and an additional 70 infants terminated during the second minute. ANOVA and chi-squared tests revealed that infants who terminated in the first half of the task did not differ from infants who terminated in the second half on cortisol reactivity ($t(124)=1.10$, $p > .05$), prenatal IPV exposure ($t(141) =-.66$, $p>.05$), postnatal IPV exposure ($t(140) =.63$, $p >.05$) or demographic variables of gender (χ^2 (1, N=143) = 1.76, $p > .05$), race (χ^2 (5, N=143) = 1.36, $p > .05$), maternal education (χ^2 (2, N=143) = 2.24, $p > .05$) or relationship status (χ^2 (2, N=143) = 1.51, $p > .05$). The only significant difference was that infants who terminated in the second half were more likely to come from homes with incomes above the Medicaid cutoff, χ^2 (1, N=143) = 4.31, $p = .04$. Data from the first six intervals of the arm restraint task were used to calculate emotion reactivity mean and emotion regulation ratio. Additionally, 26 infants were missing cortisol data from one or more of the collections. Data for cortisol values, emotion reactivity interval scores, and the other study variables (about 1% missing data) were imputed using the EM estimation method (SYSTAT 12). Across all of the variables imputed, 10 percent of the data were missing. Little's MCAR statistic was not significant (χ^2 (N=182, 600) = 654.51, $p = .06$).

Descriptive Statistics

Variable means, standard deviations, skewness and kurtosis are in Table 1. Bivariate correlations among variables are in Table 2. Some variables were highly correlated, such as pre- and postnatal IPV (r=.71). Pre- and postnatal IPV were also associated with the risk composite score as well as ITSEA externalizing scores. Of the two variables derived from the video coding of infant emotion, the emotion reactivity mean was only correlated with postnatal IPV (r=.21), and the emotion regulation ratio was not correlated with any other variables. Baseline cortisol and cortisol reactivity were correlated with each other, but not with other variables.

Latent Profile Analysis

Latent Profile Analysis (LPA) was conducted to examine patterns among the infant emotion and cortisol variables. Four variables [i.e., emotion reactivity mean, regulation ratio (upregulation/downregulation), baseline cortisol and cortisol reactivity] were entered into the LPA as indicators. Four covariates were also included: prenatal IPV, postnatal IPV, demographic risk, and nurturing parenting. These covariates were chosen because of their potential relevance to the infant stress response. In the first model, only two of the covariates, pre- and postnatal IPV, predicted LPA group membership. Therefore, risk and nurturing parenting were dropped in all subsequent LPA models. Reducing the number of covariates and including only pre- and postnatal IPV did not change group membership in the final model.

Fit indices for LPA models are listed in Table 3. A three-group solution emerged as the best fitting model. AIC, BIC and adjusted BIC decreased from the two-group to the three-group model. Entropy remained high. In contrast, BIC worsened from the three-group to the four-group model, and entropy dropped below .80. Importantly, the three-group model contained

groupings that were relevant to theoretically hypothesized groups. The three-group model was retained for further analysis.

Latent profile one (LP1, N=154), the largest group, is characterized by moderate values on all four variables (see Table 4). Latent profile two (LP2, N=9), the smallest group, is comprised of infants with moderate emotion reactivity, moderate emotion regulation, moderate baseline cortisol levels and the highest cortisol reactivity. Latent profile three (LP3, N=19) includes infants with low-moderate emotion reactivity, high-moderate emotion regulation, the highest levels of baseline cortisol, and the lowest levels of cortisol reactivity.

Pre- and postnatal IPV were the two covariates that predicted membership in the final model. Prenatal IPV was associated with a 4.6% increase in membership in LP2 compared to membership in LP1 (see Table 5). Postnatal IPV was associated with an 8% decrease in membership in LP2 compared to LP1. Pre and postnatal IPV did not differentiate LP3 from LP1.

Multinomial Logistic Regression was used to validate latent profiles on infant externalizing behavior. Odds ratios indicated the relative odds of membership in groups LP2 or LP3 compared to membership in LP1, which was the reference group. ITSEA externalizing scores predicted membership in LP2 and LP3. Each one-point increase on the ITSEA externalizing scale was associated with 8.75 times the odds of membership in the LP2 group (95% CI: 1.12 - 68.21, $p < .05$) compared to the reference group. A one-point increase on the ITSEA externalizing scale was also associated with 5.17 times the odds of membership in LP3 (95% CI: 1.13 - 23.69, $p < .05$).

Configural Frequency Analysis

A striking feature of all the LPA groups is that while there were differences on the cortisol reactivity variable, there was little variation on the emotional expression variables.

Hence, questions remained about how or whether infants' pattern of modulating emotion over time related to IPV exposure and cortisol reactivity. Configural Frequency Analysis (CFA) was conducted to explore configurations of IPV, emotion regulation and cortisol reactivity variables. CFA is a categorical statistical technique that creates cross-classifications of all values of all variables (von Eye, 2002). Configurations that occur more often than expected by chance constitute types. Configurations that occur less often than expected by chance constitute antitypes. The binomial test was used because of the possibility of small expected frequencies of some cells, and the Holland-Copenhaver procedure protected α. A median split was used to categorize values of prenatal IPV and postnatal IPV as low or high in frequency. Three groups were created based on the emotion regulation ratio to represent patterns of positive lability, negative lability, and stable lability. Under this classification, high regulation ratio scores (> 1, N=92) represented infants whose reactivity scores increased more than they decreased over time (positive lability). Low regulation ratio scores (< 1, N=27) represented infants whose reactivity scores decreased more than they increased (negative lability). Even regulation ratio scores (= 1, N=63) represented infants whose pattern of reactivity scores did not fit a directional pattern of positive or negative lability (stable lability). Infants' mean reactivity score across the six intervals did not differ by these regulation groups of positive, negative and stable lability, F (22,159) = 1.27, p>.05. For the cortisol reactivity variable, infants were categorized as demonstrating high or low reactivity based on a median split.

The base model was not a good fit for the pattern of cell frequencies, LR χ^2 (18, N=182) = 78.405, p < .001, thus types and antitypes were expected (see Table 6). One type was identified (i.e., a configuration that occurred more frequently than expected by chance). This type was characterized by high frequency of prenatal IPV, high frequency of postnatal IPV, an

even emotion regulation ratio score and high cortisol reactivity. Thus, in the configuration characterized by high cortisol reactivity, the combination of high pre- and postnatal IPV was associated with an emotion regulation ratio score of 1, which corresponds to stable lability in emotional reactivity scores over time.

DISCUSSION

Findings suggest that prenatal IPV was associated with a subgroup of infants characterized by heightened cortisol reactivity and a pattern of emotion expression that did not differ from that of the other groups. However, the occurrence of both pre- and postnatal IPV was associated with stable lability in addition to cortisol reactivity. Chronic exposure to IPV that starts in the prenatal period may be more likely to affect both HPA axis functioning and emotional expression in infants. While some past studies find synchrony between salivary cortisol and the display of emotions following exposure to stress, few consider how prenatal exposure may program the physiological stress response system independent from the effects on expressed emotion. Current findings suggest that exposure to IPV at different points in development may differentially influence an infant's physiological and behavioral reaction to stress.

Subgroups identified through Latent Profile Analysis revealed distinct biobehavioral profiles of the stress response in infants subjected to a laboratory stress task. Baseline and reactive cortisol levels helped distinguish these groups. High baseline or reactive cortisol levels were found in two LPA subgroups while average levels of baseline and reactive cortisol characterized the third, largest subgroup. Research with human infants finds that it is typical for infants to display a blunted cortisol response to stressors at 12 months of age, analogous to the stress hyporesponsive period in rat pups from 4 to 14 days (Gunnar & Quevedo, 2007b; Tarullo & Gunnar, 2006). During this stress hyporesponsive period, however, the HPA axis is still susceptible to social influence. Characteristics of pre- and postnatal environments are hypothesized to contribute to variations in the stress response system (see Hostinar, Sullivan, & Gunnar, 2013 for a review). High baseline cortisol or heightened cortisol reactivity to a stressor

may indicate that a high-stress environment has altered the stress response system such that the infant becomes hypervigilant to new stressors. As predicted, the LPA profile characterized by high cortisol reactivity was associated with prenatal exposure to IPV. Exposure to postnatal IPV, however, decreased the likelihood of membership in this group when prenatal IPV was accounted for.

Results also provide evidence for dysynchrony between emotional response and cortisol response within subgroups of infants. All three LPA groups showed moderate emotion reactivity, regardless of the pattern of cortisol levels (high baseline, high reactivity, or moderate on both). These findings support past research indicating that cortisol is not consistently associated with negative emotionality in 12-month-old infants (Jansen et al., 2010; Lewis, 2011). Similarly, emotion regulation did not distinguish LPA groups, as moderate regulation levels were found in all three groups. It is interesting that infants' emotional responses remained at average levels in the high cortisol group associated with prenatal IPV, as it did in all groups. Such findings could indicate that prenatal IPV exposure has the potential to alter HPA axis responsivity, while patterns of emotional expression are less affected.

Additional analyses revealed more about the patterns of co-occurrence between IPV, emotion regulation and cortisol reactivity. While mean emotion reactivity may be dissociated from physiological functioning in the LPA groups, post-hoc analyses examined the pattern of emotion reactivity over time (positive lability, negative lability, or stable lability) as it relates to IPV exposure and physiological reactivity. A configuration (type) of high frequency of pre- and postnatal IPV, cortisol reactivity and stable lability emerged. Infants in this group obtained an emotion regulation ratio of 1, indicating that there was no trend toward an increasing or decreasing pattern of reactivity scores. Therefore, infants exposed to stress in both the prenatal

and postnatal environment may be less likely to modulate their overt emotional response to a stressor, in a positive or negative direction, while experiencing a physiological reaction. Alternately, infants in environments without IPV may be more likely to express their physiological reactivity by intentionally increasing or decreasing overt signals of distress. A relatively stable emotional response in these high-risk groups may indicate that infants are less effective at matching an emotional response to the demands of the environment. If infants perceive stressful events as unchangeable, this sense of uncontrollability may subsequently influence the physiological stress response. The finding that stable lability is associated with increased cortisol from pre- to posttask is consistent with the hypothesis that uncertainty about one's ability to regulate emotion contributes to physiological reactivity (Stansbury & Gunnar, 1994).

Interestingly, this configuration comprised of high cortisol reactivity and stable lability was significant only when combined with high levels of both pre- and postnatal IPV. Thus, behavior-cortisol relationships may depend on environmental exposure to trauma. The Emotional Security Hypothesis provides a framework for understanding intrachild emotional processes that mediate the relationship between interparental conflict and the development of psychopathology (Davies & Cummings, 1994; Davies, Sturge-Apple, Winter, Cummings, & Farrell, 2006). In a recent reformulation of Emotional Security Theory, Davies and Martin (2013) propose four distinct strategies for how children attempt to increase their emotional security in response to characteristics of interparental relationships. Children with a 'secure' pattern are able to match emotional expression to the situation by transparently signaling a need for responsive caregiving. This profile is thought to develop in the context of non-violent home environments. Other profiles involve highly distressed emotional reactions or a dissociation of

emotional expression from subjective experience in children exposed to interparental violence. These profiles are characterized by exaggerated emotional responses such as hypervigilence or freezing. Of particular concern in these profiles is that children are less able to match their emotional responses to the demands of stressful situations. Instead, children develop rigid (unregulated) patterns of responding. CFA results indicate that cortisol reactivity in the context of pre- and postnatal IPV exposure is associated with the absence of either positive or negative emotional lability. This particular configuration of IPV exposure and biobehavioral stress response is consistent with profiles characterized by the rigid emotional expression predicted by Davies and colleagues (2013). Infants represented in this configuration may display an emotional response of freezing by consistently suppressing distress. A pattern of high stable reactivity is also captured in this configuration. Emotional Security Theory proposes that the use of such rigid responses can develop as an adaptation to interparental conflict as children learn how to maximize their own emotional security in the context of a dangerous home environment. Current findings indicate that high cortisol reactivity and stable lability are associated with chronic exposure to IPV. The combination of pre- and postnatal IPV may be particularly pernicious in contributing to such profiles of adaptation to violence in the home environment.

Biobehavioral profiles identified with Latent Profile Analysis were also related to infant outcomes. Externalizing behavior was associated with membership in the high cortisol, moderate emotion reactivity group. This same group was also associated with prenatal (not postnatal) IPV. These findings are consistent with past research on the association between prenatal IPV and externalizing symptoms in 12-month old infants (Levendosky, Leahy, Bogat, Davidson, & von Eye, 2006). Findings further indicate that alterations in HPA axis functioning may mediate the relationship between prenatal exposure to IPV and behavior problems in

infancy. Exposure to stress in pregnancy can disrupt the development of the fetal HPA axis and cause alterations such as cortisol hyperreactivity that persists after the birth of the infant (Glover, O'Connor & O'Donnell, 2010). The association between a profile with extreme hyperreactivity to a stress task and externalizing symptoms may indicate that chronic physiological dyregulation associated with IPV may overwhelm an infant's capacity to cope and contribute to mental health problems (Levendosky, Bogat, Lonstein, Martinez-Torteya, Muzik, & Granger, 2012). Current findings add to the literature in identifying a specific stress response profile that may mediate the relationship between prenatal IPV and externalizing symptoms in infancy and later childhood.

Taken together, the current research provides evidence that infants who were exposed to prenatal IPV displayed heightened cortisol reactivity, moderate emotion reactivity and moderate emotion regulation, a profile associated with externalizing behaviors per mother report. As well, infants exposed to both pre- and postnatal IPV, who also displayed high cortisol reactivity, demonstrated difficulty in regulating a predominant emotional response during the arm restraint task. Findings suggest that IPV exposure is associated with physiological reactivity and rigid emotional responses characterized by a limited ability to modify one's response in a consistent direction when stressed. Infants who are prone to cortisol reactivity and less able to regulate their emotions in the lab task may be at risk for the development of externalizing pathology. Normative emotion regulation at 12 months of age involves the co-regulation of emotional distress (Eisenberg, Spinrad, & Eggum, 2010). Because infants in the profile of interest may struggle with self-regulation and be less likely to rely on a caregiver to co-regulate emotional reactivity, a typical response to frustration may be to engage in externalizing behaviors. Hence, mothers may rate these infants as less successful overall in coping with daily stressors in part because of their unsuccessful regulation strategies.

Surprisingly, nurturing parenting was not related to biobehavioral profiles of stress response. Research suggests that nurturing parenting may buffer the HPA axis response to stress by enhancing infant coping (Gunnar & Donzella, 2002; Hostinar, Sullivan, & Gunnar, 2013). In the current study, prenatal IPV increased odds of membership in a dysregulated infant stress response profile, postnatal IPV decreased the likelihood of membership, and nurturing parenting and cumulative risk were not associated with infant stress response profiles. These findings suggest that prenatal IPV is an important predictor of individual differences in stress response, particularly the biological components of biobehavioral profiles. Parenting may not contribute to variation in such individual differences when prenatal IPV is accounted for.

Limitations to the current research should also be noted. Notably, the cross-sectional nature of this research makes it difficult to comment on the trajectory of the biobehavioral stress response from the prenatal period to infancy. All measures were administered when infants were 12 months of age, and mothers reported retrospectively about IPV that occurred before the birth of the child and in the first postnatal year. However, efforts were made to ensure accurate reporting of retrospective accounts of IPV. An event history calendar was included in the protocol to aid the mothers in remembering significant events during the pregnancy and past year. Mothers only reported on IPV after they recorded events in a physical calendar in order to orient them to the time period in question. Such techniques have the potential to improve the quality of retrospective reports (Belli, 1998). Regardless, longitudinal research is needed to investigate the proposed associations between IPV exposure *in utero* and subsequent development of physiological and behavioral responses to stress. Another limitation involves the operationalization of emotion regulation. While an emotion regulation ratio score of upregulation to downregulation was created to reflect global trajectories of emotion reactivity

over time, using one indicator of emotion regulation is incomplete. Characteristics of the emotional response such as latency to distress, rise time, and duration were not appropriate for the current analyses (Thompson, 1990). There is little consensus in the literature about how to represent the change in reactivity over time, but future research could incorporate multiple indicators of emotion regulation when modeling biobehavioral profiles of stress response.

In conclusion, these findings suggest that while exposure to prenatal IPV alone may be a risk factor for physiological reactivity, exposure to chronic IPV in the prenatal and postnatal period may be the most potent predictor of the regulation of emotional responses. These results extend past research indicating that infants with postnatal IPV exposure are more likely to exhibit a moderate emotional response in tandem with high physiological reactivity (Towe-Goodman et al., 2012). Furthermore, findings are consistent with components of the Emotional Security Hypothesis, which suggests that children exposed to IPV may find it difficult to regulate emotions sensitively because they are primed to either mask behavioral distress or maintain a hypervigilant state (Davies, Winter & Cicchetti, 2006). Lastly, findings support the hypothesis that differential relationships between behavior and physiology are associated with patterns of violence exposure in the pre- and postnatal period. To extend research on the timing of IPV and the biobehavioral stress response, it is important to conduct longitudinal studies that track behavior-cortisol relationships over time and consider the effect of maternal stress in the prenatal period.

APPENDIX

Table 1. *Descriptive Statistics*

	Emotion Reactivity	Regulation Ratio	Baseline Cortisol	Cortisol Reactivity	Prenatal IPV	Postnatal IPV	Nurturing Parenting	Risk	ITSEA ext.
Minimum	0.00	0.25	0.00	-0.40	0.00	0.00	33.00	0.00	0.03
Maximum	3.06	4.00	1.24	0.97	126.00	138.00	80.00	5.00	1.55
Mean	1.58	1.61	0.20	0.11	20.72	12.84	60.30	2.25	0.56
SD	0.89	0.91	0.24	0.19	28.34	21.96	8.94	1.25	0.30
Skewness	-0.14	0.91	2.35	1.47	1.83	2.91	-0.55	-0.08	0.72
Kurtosis	-1.00	0.23	5.51	4.15	3.02	10.43	0.13	-0.64	0.39

Table 2. *Bivariate Correlations*

	1	2	3	4	5	6	7	8	9
1. Emotion Reactivity Mean									
2. Emotion Regulation Ratio	-0.04								
3. Cortisol Baseline	-0.10	-0.13							
4. Cortisol Reactivity	0.08	0.01	-0.23*						
5. Prenatal IPV	0.12	-0.02	0.01	0.13					
6. Postnatal IPV	0.21*	-0.10	0.05	-0.07	0.71*				
7. Nurturing Parenting	0.13	-0.11	0.03	-0.05	0.09	0.02			
8. Risk	0.12	-0.13	0.08	0.07	0.44*	0.40*	-0.10		
9. ITSEA Externalizing	-0.04	0.06	0.12	0.05	0.34*	0.20*	-0.07	0.33*	

Note. * = $p < .01$

Table 3. *LPA Results*

	N	AIC	BIC	Adj BIC	Entropy
2 Latent Profiles	LP1 = 164 LP2 = 18	756.052	804.112	756.605	.983
3 Latent Profiles	**LP1 = 154** **LP2 = 9** **LP3 = 19**	**702.067**	**772.555**	**702.879**	**.973**
4 Latent Profiles	LP1 = 91 LP2 = 9 LP3 = 19 LP4 = 63	698.850	791.766	699.920	.780
5 Latent Profiles	LP1 = 9 LP2 = 9 LP3 = 98 LP4 = 21 LP5 = 45	657.152	772.496	658.479	.802

Table 4. *Standardized Mean Scores for the 3-group LPA Model*

	LP1	LP2	LP3
Emotion Reactivity Mean	0.06	-0.12	-0.42
Emotion Regulation Ratio	0.02	0.31	-0.32
Baseline Cortisol	-0.30	-0.18	2.52
Cortisol Reactivity	-0.09	2.98	-0.69

Table 5. *LPA Membership by Predictor Variable*

	LP2		LP3	
	O.R.	S.E.	O.R.	S.E.
Prenatal IPV	1.047*	.012	1.000	.014
Postnatal IPV	0.923*	.028	1.006	.018

Note. * = $p < .01$

Table 6. *CFA Model Results*

Prenatal IPV	Postnatal IPV	Emotion Reg. Ratio	Cortisol Reactivity	*fo*	*fe*	*p*	
L	L	<1	L	7	4	.072	
L	L	<1	H	7	4	.073	
L	L	1	L	10	9	.415	
L	L	1	H	8	9	.450	
L	L	>1	L	22	13	.008	
L	L	>1	H	21	13	.015	
L	H	<1	L	0	3	.054	
L	H	<1	H	1	3	.213	
L	H	1	L	3	7	.147	
L	H	1	H	3	7	.067	
L	H	>1	L	6	10	.057	
L	H	>1	H	3	10	.008	
H	L	<1	L	2	4	.122	
H	L	<1	H	2	4	.299	
H	L	1	L	5	9	.198	
H	L	1	H	2	9	.005	
H	L	>1	L	8	13	.110	
H	L	>1	H	7	13	.059	
H	H	<1	L	1	3	.213	
H	H	<1	H	7	3	.027	
H	H	1	L	14	7	.015	
H	H	1	H	18	7	.000	Type
H	H	>1	L	13	10	.215	
H	H	>1	H	12	10	.315	

Note. L and H correspond to low and high values based on a median split. For emotion regulation ratio scores, 1=stable lability, <1=negative lability, and >1=positive lability. LR χ^2 (18, N=182) = 78.405, $p < .001$; significance testing used binomial test with Holland-Copenhaver protection.

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