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Infant adrenocortical reactivity and behavioral functioning: Relation to early exposure to maternal intimate partner violence

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Abstract

Prenatal stress negatively affects fetal development, which in turn may affect infant hypothalamic-pituitary adrenal (HPA) axis regulation and behavioral functioning. We examined effects of exposure to a traumatic stressor in families [intimate partner violence (IPV)] on both infants' HPA axis reactivity to stress and their internalizing and externalizing behaviors. Infants (n= 182, 50% girls, $\bar{x}$ age = 11.77 months) were exposed to a laboratory challenge task designed to induce frustration and anger (i.e., arm restraint). Saliva samples were taken pre-task and 20 and 40

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Declaration of Interest
In the interest of full disclosure, Douglas Granger is founder and chief scientific and strategy advisor at Salimetrics LLC (State College, PA), and this relationship is managed by the policies of the committee on conflict of interest at Arizona State University.

minutes post-task and then assayed for cortisol. Mothers reported on their pregnancy and postpartum IPV history, current mental health, substance use, and their infants' behaviors. Structural equation modeling revealed that prenatal, but not postnatal, IPV was independently associated with infant cortisol reactivity *and* problem behavior. Maternal mental health predicted infant behavioral functioning but not infant HPA axis reactivity. These findings are consistent with the prenatal programming hypothesis; that is, early life stress affects later risk and vulnerability for altered physiological and behavioral regulation.

Keywords

cortisol; HPA axis; prenatal; stress; trauma; intimate partner violence

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Prenatal and postnatal stress can produce long-lasting changes in young children's stress-related physiology and behavior (Glover et al., 2010; Howell & Sanchez, 2011). Research on this topic has often focused on individual differences in activation of environmentally sensitive biological systems, such as the hypothalamic-pituitary-adrenal (HPA) axis, which generates a critical homeostatic response to stressors (Gunnar & Quevedo, 2007). The purpose of the current research was to examine intimate partner violence (IPV), a common stressor for pregnant women (Taillieu & Brownridge, 2010), as a model to begin testing the relative effects of prenatal and postnatal stress on infant HPA axis reactivity and behavioral problems.

Women can experience IPV pre- and/or postpartum, so IPV's unique effects at each time period on neurobehavioral development can be determined. The prenatal period is of particular interest because the fetal brain is less developed than the infant's and undergoes periods of rapid change, making it especially vulnerable to insults (Talge et al., 2007). For example, the hippocampus (vital for glucocorticoid negative feedback loop), shows peak cell proliferation during gestation, suggesting it may be a sensitive period for the effects of stress on later infant HPA axis regulation (Noorlander et al., 2006; Seress et al., 2001). Furthermore, the amygdala (mediates emotional and behavioral responses to stress) differentiates during mid-to-late gestation, again pinpointing prenatal life as particularly susceptible (Ulfig et al., 2003). Additionally, communication between the amygdala and prefrontal cortex eventually mediates the behavioral response to stress (Gee et al., 2013) and frontal cortical expression of glucocorticoids also begins during gestation (Kitraki, et al., 1996). Maternal pregnancy stress produces its negative effects by "programming" fetal physiological systems, including the HPA axis (O'Donnell et al., 2009), and some human research supports this hypothesis (Yehuda et al., 2005; Davis et al., 2011). Other fetal neural systems involved in later attention, memory, and emotion are also adversely affected by prenatal stress (Eichenbaum, 2000), thereby derailing infant and child regulatory behaviors, putting children at risk for difficult temperament (Bergman et al., 2007) and mood and anxiety symptoms (Huizink et al., 2007; Van den Bergh et al.,2008).

Early postnatal stress can also uniquely alter infants' HPA axis (Sturge-Apple et al., 2012) and is associated with infant behavior problems (Appleyard et al., DeJonghe et al., 2011). The HPA axis of infants and toddlers is reactive to stressors (Jansen, et al., 2010), is responsive to social regulation (Flinn et al.,2011), and shows malleability to environmental risk (e.g., IPV: Hibel et al., 2011; Towe-Goodman, et al., 2012). Research examining postnatal stress often does not assess prenatal stress (Davies, et al., 2007). Indeed, many prenatal stress studies examine atypical, one-time stressors (Yehuda et al., 2005; Laplante et al., 2008) that cannot occur both pre- and postnatally. Additionally, prenatal stress is sometimes defined as maternal mental health symptoms (Davis et al., 2011), which may result from stress (Kendler et al., 1997) but are not themselves stressors. Finally, the relationship between women's mental health and HPA axis functioning is equivocal (see Burke et al., 2005), so mental health symptoms are not a proxy for HPA axis activity. In sum, IPV is an excellent model for studying prenatal stress effects because it is a frequent stressor that can occur pre- and/or postnatally and is associated with HPA axis dysregulation (Valladeres et al., 2009). The current study hypothesized that prenatal and postnatal IPV and poor postnatal maternal mental health would negatively affect these infant HPA axis reactivity and behavior, while maternal mental health was predicted to be a mediator of these effects.

Methods

Participants

Participants were 182 mother-infant dyads recruited from mid- and southeastern Michigan. Participants were recruited based on the presence or absence of prenatal and postnatal IPV. Women in the study met eight inclusion criteria: (1) a child who was 11–13 months old, (2) English-speaking, (3) 18–34 yrs old, (4) not pregnant, (5) not lactating or were willing to not breast feed their child for 2 hours prior to assessment, (6) without endocrine or other disorders affecting glucocorticoid release, (7) in a heterosexual romantic relationship for at least 6 weeks during the pregnancy; and (8) no premature delivery.

Women were recruited with fliers posted in local businesses, organizations that served families with young children, and organizations that served women experiencing IPV. Electronic media including Craigslist™ and Facebook™ were used. All of the women who met the inclusion criteria and who had experienced IPV pre- and/or postpartum were enrolled in the study. Control women who had not experienced any IPV pre or postpartum were recruited and included in the study based on group matching procedures using race/ethnicity, income, marital status, age, and education. Women were considered to have IPV pre- and/or postpartum if they endorsed experiences of threats of moderate physical violence or more serious violence on a measure of IPV. The number of women recruited for each IPV status was as follows: no IPV n= 58; pre-partum IPV only n=34; postpartum IPV only n=12; pre and post-partum IPV n=78. The demographics of the sample by the four *a priori* groups are presented in Table 1.

Procedures

Interested women telephoned the project office to complete an intake determining participation eligibility. After consenting to the telephone screening, women provided information about their (and their children's) physical health. Based on eligibility and consent, women were scheduled for interviews with their children. Research visits occurred when children were one-year-old (range: 11–13 months).

Data were collected in project offices on the university campus. All assessments began between 12:30 and 1:30 pm and were completed by 4 pm to ensure that differences in challenged cortisol were not confounded by time of sampling. To further ensure the quality of cortisol samples, mothers were instructed as follows: no alcohol consumption within 12 hours of the interview, and no teeth brushing, food consumption, or use of salivary stimulants (e.g., gum, cough drops) 1 hour before the interview. The three-hour interview was administered by two trained graduate and/or undergraduate students. Mothers signed informed consent for themselves and their infants. Mothers were financially compensated for their participation, and the infants were given a small stuffed animal. The study was approved by the university IRB.

Measures

Intimate Partner Violence—Women's experiences of IPV were assessed with the *Severity of Violence against Women Scales* (Marshall, 1992). This measure consists of 46 items rated on a 4-point scale -("Never" to "Many Times") ranging from threats of violence, to physical and sexual violence. Women completed the measure twice: once for pregnancy and once for first year postpartum [prenatal IPV ($\bar{x}$ = 20.72, *SD* = 28.74; postnatal IPV ($\bar{x}$ = 12.57, *SD* = 21.82)]. Notably, an event history calendar was used to obtain the IPV assessment (Belli, 1998; Kessler & Wethington, 1991). This method increases the accuracy of reporting of IPV compared with a standard interview about violent events (Yoshihama, Gillespie, Hammock, Belli, & Tolman, 2005).

Maternal Mental Health—The *Edinburgh Postnatal Depression Scale (EPDS;* Cox, Holden & Sagovsky, 1987), a 10-item self-report questionnaire, was used to assess depressive symptoms for the past week. Each item has 4 responses ranging from 0 – 3, which are summed to create a full-scale score ranging from 0–30 ($\bar{x}$ = 13.57, *SD* = 4.80); higher scores indicate greater symptom severity. Fifty-six percent of women scored in the "probable" depression range, with scores > 12 (Cox et al., 1987). Women were assessed for PTSD symptoms with the *Modified PTSD Symptom Scale – Self Report (MPSS-SR;* Falsetti, Resnick, Resick & Kilpatrick, 1993), which measures the frequency (0–3 scale) of symptoms present for the past two weeks and is summed to create a total score ($\bar{x}$ = 10.04, *SD* = 11.06). Eighteen percent displayed "probable" PTSD, as indicated by the recommended cut-off score of >13 (Coffey, Gudmundsdottir, Beck, Palyo, & Miller, 2006). A six-item subscale of the *Brief Symptom Inventory* (BSI; Derogatis & Melisarotis, 1983) (each with a five-point response), was used to assess women's anxiety symptoms during the last week. Responses are summed for a total score ($\bar{x}$ = 7.93, *SD* = 6.06). Twenty-three percent had scores in the clinical range of anxiety, a T-score >70.

Cumulative Risk—This variable was created to control for demographic/environmental risk factors that affect child outcomes (Sameroff, Seifer, Baldwin, & Baldwin, 1993). This approach, recommended when a large number of risk factors are assessed in a relatively small sample (Burchinal, Roberts, Hooper, & Zeisel, 2000), was the sum of 5 binary variables: income (below Medicaid poverty cut-off = 1; above Medicaid poverty cut-off = 0), marital status (single = 1; living with a partner = 0), age (younger than 22 years = 1; equal to older than 22 years = 0), negative life events [highest 25% percentile during pregnancy or postpartum year = 1; lowest 75% percentile at both pregnancy and postpartum year = 0, based on scores on the *Life Experiences Survey* (Sarason, Johnson, & Siegel, 1978)], and street drug use [any during pregnancy or postpartum year = 1; none during pregnancy or postpartum year = 0, based on the *Perinatal Risk Assessment Monitoring Survey* (Gilbert, Shulman, Fischer, & Rogers, 1999)]. The cumulative risk score ranged from 0 to 5 ($\bar{x}$ = 2.25; SD = 1.26).

Challenge Task—Infant HPA-axis response was assessed by measuring cortisol in saliva samples obtained during a stress task, the *Modified Lab-TAB Arm Restraint* procedure (Goldsmith & Rothbart, 1996). The infants were placed in a highchair and given a fun toy to play with for two minutes (Eiden et al., 2011). The toy was then placed out of reach of the infant, and the interviewer restrained the infant by standing behind him or her and gently placing hands on the infant's forearms and holding firmly for 2 minutes while the mother watched from a chair placed out of view of the infant. If the child cried hard for 20 consecutive seconds, the restraint was terminated early. Early termination was not associated with amount of prenatal IPV ($F_{1, 142}$ = 1.63, p = .20), postnatal IPV ($F_{1, 142}$ = .51, p = .48), or the dyads' *a priori* IPV group membership (Pearson's r = 4.93, p = .18). After the restraint, the infant was removed from the chair and returned to the mother for a short recovery period. Ninety-six percent of children showed visible distress during the task, and 70% had early terminations.

Saliva Collection and Cortisol Determination—Following methods used by Granger and colleagues (2007), saliva was collected from infants using hydrocellulose microsponges placed in their mouths before the challenge task and then again 20 and 40 minutes after its conclusion. Samples were frozen and stored at −80 °C. Microsponges were later thawed at 4°C and centrifuged for 15 minutes at 1300 rpm to extract saliva. Saliva samples were assayed for cortisol using a commercially available enzyme immunoassay kit specifically designed for use with saliva using the manufacturer's recommended protocol. The assay is 510K cleared (US FDA) as a diagnostic measure of adrenal function; the range of detection is from 0.003 to 3.0 μg/dl. All assays were completed in the Lonstein lab at Michigan State University and the Vasquez lab at University of Michigan Hospital, with 10% of the samples randomly assayed in duplicate for measures of intra-assay variability. Consistent with common practices, cortisol measures were log-transformed to improve the distribution of the data. The intra- and inter-assay coefficients of variation in this study were 7.9% and 9.8%, respectively. Mean log-transformed cortisol levels were .20 ug/dL (SD = .26) at baseline, .23 ug/dL (SD = .24) 20 minutes after the arm-restraint procedure, and .27 ug/dL (SD = .26) 40 minutes after the arm-restraint procedure. Cortisol levels were not associated with variations in the one hour time window during the afternoon when saliva was collected, time from the infant's last meal, or time since the infant slept; thus, these factors were not controlled in the analyses.

Infant Social and Emotional Assessment (ITSEA; Briggs-Cowan & Carter, 2001)—This mother-report instrument uses a three-point scale (“not true” to “very true”) to assess externalizing and internalizing behaviors. The alpha reliability coefficients were .89 for externalizing and .78 for internalizing, similar to the published reliability estimates of .87 and .80, respectively (Carter, Briggs-Gowan, Jones, & Little, 2003).

Results

Missing Data

Twelve percent of infant salivary cortisol data were missing. This was primarily due to the infant not providing enough saliva for the assay. In addition, raw cortisol scores that were outside of the range of detection (.003 to 3) were considered an error. We deleted them from the database and treated them as missing data. Scores within the range of detection were screened for outliers using (mean + 3SDs) as the upper limit. Six baseline, five 20-min post-challenge, and three 40-min post-challenge cortisol scores were identified as outliers. Outliers were windsorized to preserve rank order.

Little's MCAR test revealed the data were not missing completely at random, $\chi^2 = 172.494$, $df = 133$, $p = .01$. Thus, t-tests were used to compare subjects with and without missing data. Those infants with complete salivary cortisol data did not differ from those with missing samples across all the variables used in hypothesis testing ($p > .10$) and were only significantly different in that their mothers were more likely to be single, a variable accounted for by the cumulative risk score. Thus, there is no indication of biased missing data for the variables used in this study, and the final dataset used in analyses included estimates obtained using Expectation-Maximization (EM) Estimation for the outcome variables. All questionnaire data were complete except for one item-level score for one subject. Following Parent’s (2012) findings that simple methods are as appropriate as more complex imputation strategies when missingness is item-level and very small, mean imputation was used for that item score.

Cortisol Reactivity

Overall, the infants showed a significant HPA axis response to the arm restraint task. Using repeated-measures ANOVA, there was a significant main effect of time for log-transformed cortisol values, $F(2, 362) = 10.53$, $p = .00$, with infants increasing from baseline across the 3 time points (baseline, peak and recovery). Contrast tests indicated that the effect was linear, $F(1, 181) = 21.28$, $p = .00$, rather than quadratic.

Infant cortisol reactivity (peak – baseline) was related to the amount of prenatal IPV ($r = .15$, $p < .05$) but was not related to postnatal IPV ($r = .00$, $p = .97$). However, rather than using a continuous measure of the infants’ cortisol to assess change, we chose to use the presence versus absence of a cortisol response. This is consistent with the literature that indicates that the presence (versus absence) of a cortisol response of 12-month-old children is affected by early-life stress (Tarullo & Gunnar, 2006), rather than a dose-response relationship between early life stress and degree of cortisol reactivity. Thus, children were grouped into Reactors and Non-Reactors using their log-transformed cortisol data. Following Schuetze, Lopez, Granger, and Eiden (2008), and based on the inter- and intra-assay coefficients of variation, infants who displayed an increase in cortisol from baseline that was > 16% (greater than twice the intra-assay variability) at 20- or 40-minutes post-stress were classified as "Reactors" (n = 119), while infants who did not show an increase of at least 16% were classified as "Non-Reactors" (n = 63). Within Reactors, 40% of infants peaked at 20 minutes post-stressor, and 60% peaked at 40 minutes post-stress. Within Reactors, the mean baseline-to-peak change in log-transformed cortisol levels was .20 ug/dL (SD = .20). Difference scores > 16% were used because twice the intra-assay reliability represents an increase that is unlikely to be due by chance, and thus meaningful/interpretable and is consistent with the literature (e.g. Fortunato, Dribin, Granger, & Buss, 2008). This classification captures adrenocortical reactivity more accurately than the widely used "Area under the Curve" score, which prevents one from distinguishing between different patterns of cortisol secretion (e.g., Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). Finally, dichotomization of metric response variables reduces measurement error, providing more unbiased results as well as more statistical power (Shentu & Xie, 2010).

Descriptive Statistics

Bivariate correlations (Pearson product-moment or point biserial) were used for preliminary examination of the associations between variables (See Table 2).

Hypothesis Testing

Structural equation modeling (SEM) was used to test the hypotheses that prenatal and postnatal IPV exposure would affect infant behavioral and physiological functioning, mediated by maternal mental health. Cumulative risk exposure was also included in the model to control for these effects. All SEM models were fitted using Mplus 5.2 (Muthén & Muthén, 2007) with Full Information Maximum Likelihood estimation, a method that is robust to non-normally distributed data (Muthén & Muthén, 2007). Global model fit was evaluated using the overall χ^2 test of model fit ($p > .05$), the root mean square error of approximation (RMSEA < 0.08; Browne & Cudeck, 1993), and the comparative fit index (CFI > 0.90). Based on the widely used guideline of 10 cases per parameter, the present sample size was appropriate for model testing.

The test of the final hypothesized model is shown in Figure 1. Notably, the correlation estimate between infant cortisol reactivity and infant behavior outcomes was set to zero (due to a non-significant association in a prior model with the following fit indices: $\chi^2 = 27.41$, $df = 16$, $p = .04$; CFI = .94, RMSEA = .06). Global fit indices for this model indicated that the constrained model provided a good fit for the data based on all the assessed indices, $\chi^2 = 25.58$, $df = 17$, $p = .08$; CFI = .96, RMSEA = .05. The chi-square difference test revealed that the more parsimonious (constrained) model did not have significantly worse fit than the model in which the path between infant cortisol and behavior was not constrained, $\chi^2 = 1.83$, $df = 1$, $p =$ *ns*.

In addition, the indirect effect of prenatal IPV and cumulative risk, as mediated via maternal mental health, was evaluated using the Sobel test obtained through the command "MODEL INDIRECT" in Mplus. Results indicated significant mediation for both prenatal IPV and cumulative risk (*std b* = .09, *p* = .02, and *std b* = .10, *p* = .01, respectively). There was complete mediation for the effects of cumulative risk on infant behavioral functioning and partial mediation for the effects of prenatal IPV on infant behavioral functioning, as shown in Figure 1. The estimated model predicted 35% of the variance in maternal mental health, 34% of the variance in infant behavioral/emotional outcomes, and 13% of the variance of the likelihood of infant cortisol reactivity (see Figure 1).

In order to test whether the effects of prenatal and postnatal IPV on maternal mental health and infant outcomes were equivalent, given the correlation of .70 between prenatal and postnatal IPV, another model was estimated. In this model, the paths between prenatal IPV and maternal mental health, and between postnatal IPV and maternal mental health were constrained to be equal. In addition, the paths between prenatal IPV and infant outcomes, and postnatal IPV and infant outcomes, were constrained to be equal. This model did not fit the data adequately, $\chi^2 = 41.10$, $df = 19$, $p = .00$; CFI = .89, RMSEA = .08, and was a worse fit than the prior model. Thus, the effects of prenatal and postnatal IPV were not equivalent and the model represented in Figure 1 was considered the best and final model for the data.

Discussion

Prenatal stress is well known to affect infant outcomes. In the present study, prenatal exposure to IPV was associated with infant HPA axis reactivity to a laboratory challenge, as well as higher levels of mother-reported internalizing and externalizing problems. Typically developing one-year old infants do not show a cortisol reactivity response to lab stress tasks, thus the cortisol reactivity shown in this study likely indicates HPA axis dysregulation. Importantly, the effect of postnatal exposure to IPV on infant behavior was not significant when prenatal IPV was included in the model. The lack of a relationship between postnatal IPV and child behavior appears inconsistent with recent meta-analyses of IPV and children's functioning (e.g., Evans, Davies, & DiLillo, 2008), although it is critical to note that these studies do not include infants or assess prenatal IPV. Thus, extant research may be overestimating the independent effects of postnatal stressors on a host of infant physiological and behavioral outcomes.

Two theoretical models may explain why we found that prenatal IPV stress, but not postnatal IPV stress, affected later infant HPA reactivity and behavior problems. McEwen's (2000) allostatic model proposes that over time, chronic exposure to complex-chaotic circumstances, such as IPV, changes the set-point or threshold of the sensitivity of the HPA axis to environmental challenge. Exposure to adrenal hormones and other chemical signals released during these continual stressful experiences results in accumulated "wear and tear" on numerous physiological systems, thereby lowering the threshold for future HPA axis activation (Danese & McEwen, 2012). Alternatively, the adaptive calibration model (Del Guidice, Ellis, & Shirtcliff, 2011) proposes that individual differences in stress responsivity are due to the ability of the individual to change his/her phenotypic developmental trajectory in response to environmental conditions. In other words, adaptive, rather than pathological, processes change the biobehavioral phenotype of an individual. Exposure to the prenatal stress of IPV may be "adaptively calibrating" the infant to a harsh environment so that the child is better prepared to cope with significant challenges later in life by being more physiologically or behaviorally responsive to the environment. Postpartum maternal mental health problems are another environmental exposure and consistent with this model. We found that they partially mediated the effects of prenatal IPV on infant behavior problems, suggesting that maternal affect dysregulation in response to IPV exposure may influence the infant's developing affective regulation through epigenetic mechanisms and/or learning. Either the allostatic model or the adaptive calibration model is consistent with the idea that prenatal exposure to IPV may set the stage for a trajectory of long-term changes in offspring HPA axis and behavioral response to stressors.

Infant HPA axis reactivity and behavioral dysregulation were not themselves associated in this study, supporting the idea that physiological stress responses and behavior are functionally different aspects of environmental adaptation (e.g. Haley & Stansbury, 2003; Ramsay & Lewis, 2003; Towe-Goodman et al., 2012). This lack of coordination between infant physiology and behavior suggests three distinct pathways to future mental health problems in children exposed to prenatal IPV (see Figure 1): (1) a pathway to depression, anxiety, or oppositional/conduct problems starting with insults to the neural system (e.g., amygdala and prefrontal cortex), which occur through fetal exposure to elevated cortisol from maternal IPV experiences, (2) a pathway to depression or PTSD via a sensitized HPA axis, possibly due to altered hippocampal development and irregular negative feedback to cortisol, that becomes increasingly overloaded in response to life stressors that include postnatal exposure to IPV (e.g., Yehuda & Seckl, 2011), and (3) a pathway that is maternally-mediated by poor maternal mental health leading to infant behavior problems. These may be distinct pathways or they may transact over time, but either way, they lead to greater congruence between HPA axis reactivity and mental health problems as children become adolescents and adults. A recent review suggests that altered HPA axis regulation precedes the development of depressive symptoms in adolescents and that congruence between HPA axis dyregulation and mood disorders increases across development (Guerry & Hastings, 2011). The lack of coordination that we found between HPA axis reactivity and behavior problems in infants may become more coordinated over the course of development and may result in specific mental health problems later in life.

Several limitations of the study should be noted. First, the research was cross-sectional and used maternal retrospective report to assess pregnancy IPV. Thus, IPV during specific time periods within pregnancy was not assessed. Future research using a prospective design might be helpful, especially to detect possible sensitive periods within the broader pregnancy period. However, we have confidence in our assessment of IPV for the two broad time periods, pregnancy and post-partum. IPV is a highly salient event that is likely to be remembered more easily than less salient events. In addition, we used a retrospective reporting technique that has been shown to reliably measure women's past IPV events (Yoshihama et al., 2005). Second, our research relied on maternal report for measurement of mental health and child behavior; there is a possibility that this may have introduced method bias into the findings. Future research should include observational assessment of child behavior. Third, we did not assess whether children witnessed the IPV. It would be valuable for future research to do so, as the deleterious effects of IPV on young children may be stronger for those who witness (as compared to live with) IPV (DeJonghe et al., 2011). Fourth, and finally, although we were not able to find a sufficient number of women who experienced postpartum IPV to analyze the four *a priori* groups, future researchers might be able to do so. We believe that there are few women who engage in a new, violent relationship after the birth of their children, so ever finding such a group in sufficient numbers could continue to prove particularly difficult.

In conclusion, our findings are consistent with a prenatal programming hypothesis for the negative effects of IPV exposure and suggest that future prospective, longitudinal research on this topic would be fruitful. Understanding the mechanisms through which prenatal IPV affects the developing fetal HPA axis is crucial as well as delineating the neural source(s) contributing to HPA axis impairment and behavior problems in infants.

Abbreviations

IPV	Intimate partner violence

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Key Points

1. This is the first study to demonstrate that prenatal IPV affects infant HPA axis reactivity.
2. Prenatal IPV, but not postnatal IPV, affects infant behavioral functioning directly, as well as indirectly through maternal mental health.
3. Infant HPA axis reactivity and behavioral functioning were not related, supporting the idea that these are functionally different aspects of our capacity to adapt to our environment during early development.

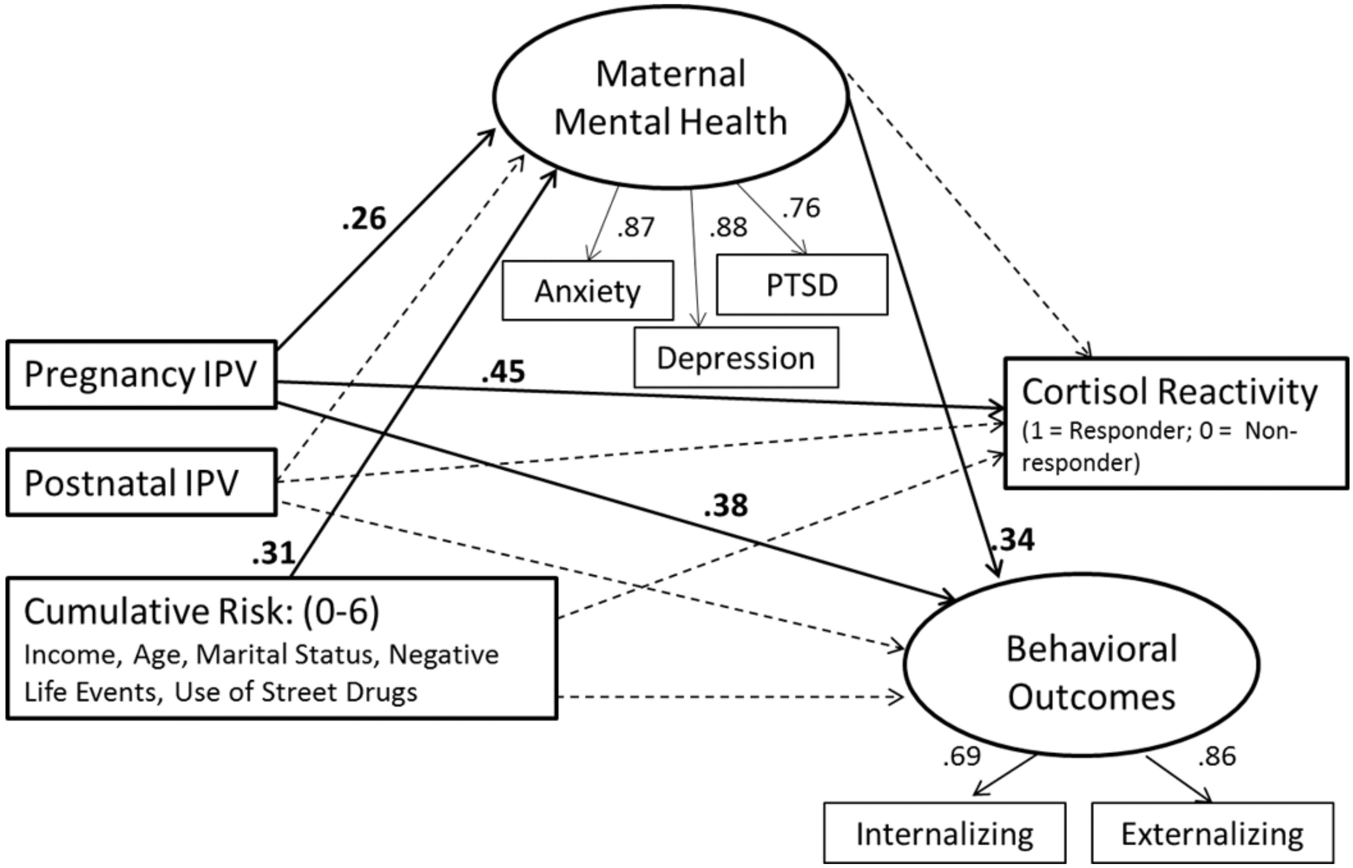

Figure 1.

Structural equation model for infant cortisol reactivity and behavioral outcomes.

Table 1

Descriptive statistics for demographics and study variables for the 4 *a priori* IPV groups.

	No IPV	Prenatal IPV	Postnatal IPV	Pre- & Post-IPV
N's				
Maternal ethnicity				
African-American	16	11	4	29
Caucasian	29	16	5	18
Multiracial	5	5	1	16
Other	8	2	2	5
Maternal education				
Less than high school	8	10	5	13
High school	18	11	1	27
Post-high school	32	13	6	38
Maternal marital status				
Not living with Partner	14	17	7	54
Living with Partner	23	11	5	12
Married	21	6	0	12
Infant gender				
Female	39	26	5	38
Male	19	8	7	40
Infant ethnicity				
African-American	13	10	4	26
Caucasian	24	8	1	17
Multiracial	16	14	7	29
Other	5	1	0	6
Means (standard deviations)				
Maternal age	25.48 (4.90)	23.24 (4.72)	22.75 (4.09)	24.51 (4.81)
Prenatal IPV	0 (0)	19.75 (26.17)	0 (0)	28.33 (30.38)
Postnatal IPV	0 (0)	0 (0)	5.43 (6.45)	20.83 (25.14)
Depression	10.03 (3.98)	12.69 (3.85)	13.43 (5.53)	14.89 (4.72)
Anxiety	4.52 (4.53)	6.36 (5.50)	6.14 (6.09)	9.56 (6.11)
PTSD	3.35 (7.34)	7.61 (9.34)	6.00 (8.00)	13.03 (11.61)
Cumulative Risk	1.35 (.92)	1.92 (1.30)	2.57 (.98)	2.59 (1.20)
Infant Internalizing	.37 (.18)	.40 (.19)	.49 (.30)	.44 (.19)
Infant Externalizing	.42 (.24)	.48 (.27)	.68 (.40)	.61 (.30)
Cortisol – Baseline (raw)	.24 (.40)	.17 (.19)	.16 (.15)	.29 (.52)
Cortisol – 20-min post (raw)	.24 (.26)	.17 (.26)	.19 (.25)	.35 (.48)
Cortisol – 40-min post (raw)	.36 (.30)	.31 (.47)	.20 (.24)	.40 (.47)

Table 2

Bivariate correlations among the study variables.

	2	3	4	5	6	7	8	9[a]
1. Cumulative Risk	.47*	.43*	.56*	.57*	.54*	.31*	.40*	.12
2. Prenatal IPV		.70*	.37*	.34*	.49*	.37*	.34*	.21*
3. Postnatal IPV			.36*	.31*	.43*	.19*	.19*	.10
4. Maternal Depression				.74*	.67*	.28*	.41*	.03
5. Maternal Anxiety					.65*	.28*	.41*	.01
6. Maternal PTSD						.20*	.30*	.03
7. Infant Internalizing							.59*	.04
8. Infant Externalizing								.12
9. Infant Cortisol Reactivity (1= Reactive, 0 = Non-reactive)								

Note.

* $p < .05$

[a] Point-biserial correlations