Published in final edited form as:

Stress. 2019 March ; 22(2): 190–199. doi:10.1080/10253890.2018.1501019.

Maternal Witness to Intimate Partner Violence During Childhood and Prenatal Family Functioning Alter Newborn Cortisol Reactivity

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Abstract

Witnessing intimate partner violence (IPV) during childhood is a risk factor for mental health problems across the lifespan. Less is known about the intergenerational consequences of witnessing IPV, and if the current family climate buffers intergenerational effects of witnessing violence. The mother's experience of witnessing IPV against her own mother during childhood, prenatal family dysfunction, and prenatal perceived stress were examined as predictors of offspring cortisol in the first month of life (N = 218 mother-infant dyads). Mothers reported on witnessing IPV in their childhoods, prenatal family dysfunction, and prenatal perceived stress in pregnancy. At 2 days and again at 1 month postpartum, infants engaged in a neurobehavioral exam to assess infant cortisol reactivity. Infants whose mothers witnessed IPV in childhood exhibited alterations in their baseline cortisol and their cortisol reactivity at one month of age, whereas family dysfunction during pregnancy was associated with baseline cortisol and cortisol reactivity at 2 days of age. Prenatal perceived stress was not associated with infant cortisol at 2 days or 1 month. Prenatal family dysfunction and perceived stress did not moderate effects of the mother's experience of witnessing IPV. Results support the view that maternal experiences in childhood and during pregnancy exert intergenerational effects on the HPA stress response system.

Keywords

intimate partner violence; family functioning; intergenerational; HPA axis; cortisol; infant

Introduction

Intimate partner violence (IPV) is prevalent in the lives of children and families (Osofsky, 2003). Adults who witnessed IPV in childhood are more likely to exhibit mental illness (Russell, Springer, & Greenfield, 2010) and to be victims and perpetrators of IPV in their

own relationships (Kwong, Bartholomew, Henderson, & Trinke, 2003). Less is known about whether and how witnessing IPV in childhood exerts influence on the next generation. Given the importance of stress physiology in the intergenerational transmission of violence and psychopathology, we examined the possibility that the mother's experience of witnessing IPV against her own mother during childhood contributes to offspring cortisol in the first month of life. We also examined the contribution of prenatal stress and family functioning to offspring cortisol given the role of the prenatal environment in shaping infant physiology.

Parental early adversity has potential to exert intergenerational effects on offspring health and developmental outcomes (Bowers & Yehuda, 2016), and the hypothalamic-pituitary-adrenal (HPA) stress response system is a major intergenerational mechanism (Moisiadis & Matthews, 2014). Maternal childhood adversity contributes to dysregulated maternal HPA functioning in pregnancy (Bublitz & Stroud, 2012; Shea et al., 2007), and maternal HPA functioning in pregnancy "programs" infant HPA functioning (Davis, Glynn, Waffarn, & Sandman, 2011; de Weerth, Buitelaar, & Beijers, 2013). Prior work has demonstrated that infants whose mothers experienced sexual or physical abuse in childhood exhibit lower basal cortisol at 6 months of age than infants whose mothers were not abused (Brand et al., 2010). The maternal experience of witnessing IPV in her childhood may also contribute to infant HPA functioning, yet this has not been empirically demonstrated. However, evidence from an animal model supports this possibility. Male rats that were exposed to a non-social stressor early in life were aggressive towards their female partners later in life, as were their offspring who were not exposed to any parental aggression or stress (Cordero et al., 2012). Therefore, the first aim of the current study was to examine if the maternal experience of witnessing IPV in childhood is associated with infant HPA functioning. We focused on infant cortisol reactivity in the first month of life, during which the HPA system undergoes rapid change following birth and prior to a period of quiescence, to understand effects of preconception and prenatal stress over and above caregiving effects.

Although maternal childhood experiences contribute to offspring HPA functioning, maternal experiences in pregnancy are also salient. There is evidence that stress in pregnancy contributes to infant HPA functioning (Bowers & Yehuda, 2016; Gunnar & Donzella, 2002). Less is known about how prenatal family functioning and stress contribute to infant HPA functioning in conjunction with the mother witnessing IPV in childhood. A highly functioning family unit potentially buffers infants from effects of the mother witnessing IPV, whereas high levels of perceived stress potentially exacerbate these effects. Therefore, the second aim of the current study was to examine prenatal family functioning and perceived stress as moderators of the effect of the mother witnessing IPV on infant cortisol reactivity.

Methods

The sample was composed of 218 women who were drawn from two prospective longitudinal studies focused on the effects of maternal depression and smoking during pregnancy for fetal and infant development (Behavior and Mood in Babies and Mothers [BAM BAM]; Behavior and Mood in Mothers, Behavior in Infants [BAMBI]). Women were excluded from participating in the larger studies if they were less than 18 or more than 40 years of age and if they were pregnant with more than one infant. Women were also

excluded from the current report if they had chronic medical conditions including gestational diabetes, hypertension, hyperthyroidism, hypothyroidism, or medication use that has the potential to influence the fetus' HPA functioning including steroids and antidepressants in the third trimester of pregnancy. Among the 253 eligible women, 221 reported on their childhood experiences including witnessing intimate partner violence (the measure of childhood experiences was not included in the initial study protocol and was added following the first few months of data collection). Three of the 221 women did not complete postpartum infant assessments. Thus, the final sample for data analysis was 218 mother-infant dyads.

Measures

Maternal experience of witnessing IPV in childhood.—A modified version of the Adverse Childhood Experiences (ACES) scale (Dube et al., 2003) was completed during a prenatal assessment in the second or third trimester of pregnancy ($M = 26$ weeks gestational age, $SD = 5$ weeks) to assess the mother's experience of witnessing IPV against her own mother during childhood. Participants were asked "how often did your father, stepfather, or mother's boyfriend do any of these things to your mother" including "push, grab, slap, or throw something at her," "kick, bite, hit her with a fist, or hit her with something hard," "repeatedly hit her for at least a few minutes," and "threaten her with or hurt her with a gun or knife." Response options ranged from 0 (never) to 4 (very often). Consistent with the original conceptualization of the measurement of ACES (Bethell et al., 2017), and prior research utilizing the ACES scale (e.g. Steele et al., 2016), a dichotomous variable was created to indicate any experience of witnessing IPV in childhood. Mothers who endorsed any of these items at least once were considered to have witnessed IPV in childhood ($n = 57$).

Prenatal family functioning.—The Family Assessment Device (FAD; Epstein, Baldwin, & Bishop, 1983) was also completed at the prenatal assessment to assess family functioning. Participants reported on problem solving, communication, the appropriateness of roles, affective responsiveness, affective involvement, and behavioral control within the family during the past two months. Analyses utilized the General Functioning Scale, which is a continuous scale and includes 12 items to assess the overall health of the family unit (e.g. "Making decisions is a problem in our family"). Response options ranged from 1 (Strongly Agree) to 4 (Strongly Disagree), with higher scores indicating greater family dysfunction. A highly dysfunctional family unit may be characterized as having difficulty with problem solving and communication, and low levels of affective involvement and responsiveness among family members. The FAD has acceptable test-retest reliability, internal consistency reliability across many family types, and convergent validity with other self-reports and independent observer ratings of family functioning (Miller, Ryan, Keitner, Bishop, & Epstein, 2000). There was good internal consistency reliability of the general functioning scale in the current sample ($\alpha = .89$).

Prenatal perceived stress.—The Perceived Stress Scale (Cohen, Kamarck, & Mermelstein, 1983) was used to assess maternal perceptions of stress in pregnancy at the prenatal assessment. Participants reported their appraisals of stress in the past month on 14

items (e.g. “In the last month, how often have you felt nervous and “stressed”?”). Response options ranged from 0 (Never) to 4 (Very Often), with higher scores indicating greater perceived stress. The PSS has acceptable internal consistency reliability, and convergent validity with theoretically relevant indicators of mental, physical, and behavioral health (Cohen et al., 1983). There was good internal consistency reliability of the Perceived Stress Scale in the current sample ($\alpha = .84$).

Infant cortisol.—Salivary cortisol samples were collected from infants 2 days following delivery (M=40 hours following delivery, SD=24 hours) and 1 month (M=32 days, SD=3 days) postpartum to assess the infant cortisol response to the NICU Network Neurobehavioral Exam (NNNS), a mildly stressful task (Lester, Tronick, & Brazelton, 2004). The NNNS was designed to test neurobehavior in high-risk (such as drug-exposed or preterm) infants as well as normative infant neurobehavioral performance. Prior work from our lab (Stroud et al., 2014) has demonstrated that the NNNS elicits a cortisol stress response from infants in the first month of life. Trained research assistants collected four saliva samples from infants in the context of the NNNS exam using the sorbette sampling device (Salimetrics LLC, State College, Pennsylvania). The first sample was collected immediately prior to the NNNS exam (this was considered to be the “baseline” sample), the second sample was collected immediately following the NNNS exam, the third sample was collected 20 minutes following the exam, and the final sample was collected 40 minutes following the exam. Samples were frozen at –80 degrees Celsius until analysis by Salimetrics using a high-sensitive enzyme immunoassay. Cortisol values were log transformed prior to data analysis to adjust for skewed distributions.

Potential confounders.—A number of variables were considered as potential confounders. Demographic characteristics were collected from mothers during an interview in pregnancy to assess maternal age, marital status, education, income, race, and ethnicity. The Structured Clinical Interview for the DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 2002) was used to assess maternal depression in pregnancy. Mothers who met diagnostic criteria for Major or Minor Depressive Disorder in pregnancy were considered to be depressed in pregnancy (dichotomized for data analysis). Maternal smoking status during pregnancy was recorded via maternal report using the Timeline Follow-Back Interview (Robinson, Sobell, Sobell, & Leo, 1995). Infant birth outcomes, including Apgar scores at 5 minutes, birth weight, gestational age at delivery, and sex were recorded through medical chart review. Infant age at the time of each NNNS exam was also considered as a covariate. The ACE scale (Dube et al, 2003) was also used to assess physical abuse directed toward the mother in childhood (2 items, e.g., “How often did a parent, stepparent, or adult living in your home push, grab, slap, or throw something at you?”), sexual abuse directed toward the mother in childhood (4 items, e.g. “How often did an adult person at least 5 years older than you touch or fondle your body in a sexual way?”), and neglect experienced by the mother in childhood (3 items, e.g. “How often did you not have enough to eat?”).

Statistical Analysis

Descriptive statistics were conducted in SPSS 20. Latent growth curve modeling was conducted using Mplus 6.0 (L. K. Muthén & Muthén, 1998–2011) to test whether the

maternal experience of witnessing IPV in childhood, prenatal perceived stress, and prenatal family functioning predicted the infant cortisol stress response to the NNNS exam at 2 days and 1 month of life. Linear and quadratic slopes representing change in cortisol reactivity at 2 days and 1 month were estimated, then substantive variables of interest were modeled as predictors of the linear and quadratic slopes. The first set of latent growth curve models tested the main effects of witnessing IPV in childhood on infant cortisol at day 2. The second set of latent growth curve models tested the main effects of witnessing IPV in childhood on infant cortisol at 1 month. Each set of models first tested the maternal experience of witnessing IPV in childhood without prenatal family dysfunction or perceived stress included, then prenatal family dysfunction and perceived stress were included as covariates to understand if witnessing IPV in childhood was associated with infant HPA functioning beyond the functioning of the mother's own intimate relationships and perceived stress in pregnancy. Maternal depression in pregnancy (major or minor depression versus none) and smoking in pregnancy (any maternal smoking during pregnancy versus none) were included as covariates given that women who were depressed in pregnancy and women who smoked were oversampled for the larger studies. In addition to the a priori covariates included, maternal and infant socio-demographic characteristics were tested for association with predictors and outcomes to determine inclusion in the analytic models (described below).

Results

Sample characteristics

The sample included 218 mother-infant dyads. Mothers ranged in age from 18 to 40 years ($M=25.7$ years). The majority of mothers were of low socioeconomic status (44% of mothers had high school degree; 48% of mothers reported family incomes below \$30,000 per year) and were racially/ethnically diverse (16% Black, 24% Hispanic, 48% Caucasian, 6% more than one race, 6% other). Eighty percent of mothers were in a committed romantic partnership (37% married). Mean(SD) gravida was 2.4(1.5). Thirty-one percent of mothers experienced depression (major or minor) in pregnancy, and 29% of mothers smoked during pregnancy. Twenty-six percent of mothers ($n = 56$) experienced physical abuse, 22% ($n = 47$) experienced sexual abuse, and 29% ($n = 64$) experienced neglect in childhood. Fifty-seven women (26% of the sample) reported witnessing IPV in childhood whereas 161 women (74%) reported no experience of witnessing IPV in childhood. Ninety-nine mothers (45%) reported no adverse childhood experiences (ACES), 55 (25%) reported one ACE, 33 (15%) reported two ACES, 21 (10%) three ACES, and 10 (5%) reported experiencing all four ACES. All four ACES were significantly associated with each other (χ^2 p's all $.001$). Among the 57 women who witnessed IPV, 29 (51%) of the women also experienced physical abuse, 23 (40%) also experienced sexual abuse, and 28 (49%) also experienced neglect. Likewise, among the 56 women who experienced physical abuse, 25 (45%) also experienced sexual abuse and 28 (50%) also experienced neglect. Finally, among the 47 women who experienced sexual abuse, 23 (49%) of the women also experienced neglect.

Among the infants, 53% were male. Mean(SD) gestational age at birth was 39(1) weeks, mean(SD) growth percentile at birth at 45(25), and the mean Apgar score 5 minutes

following birth was 9(.34). The majority of infants were delivered vaginally (76%). Mean(SD) infant age at the day 2 NNNS was 40(24) hours. Mean(SD) age at the 1 month NNNS was 32(3) days.

Preliminary analyses

Witnessing IPV in childhood was not associated with prenatal family dysfunction ($t(216) = -1.66, p = .098$) or prenatal perceived stress ($t(216) = -1.46, p = .146$). Prenatal depression was an a priori covariate and was associated with witnessing IPV in childhood ($X^2(1) = 4.69, p = .030$). Maternal smoking in pregnancy was also an a priori covariate but was not associated with witnessing IPV in childhood ($X^2(1) = .34, p = .557$). As described above, maternal experiences of physical abuse ($X^2(1) = 25.65, p < .001$), sexual abuse ($X^2(1) = 16.12, p < .001$), and neglect ($X^2(1) = 14.54, p < .001$) in childhood were all associated with witnessing IPV. Maternal experiences of physical abuse and neglect were not associated with infant cortisol at any timepoint. Maternal experiences of sexual abuse were associated greater infant cortisol immediately following the NNNS exam at day 2 (sample 2; $r = .17, p = .04$), but not infant cortisol at any other timepoint. However, maternal experiences of physical abuse, sexual abuse, and neglect did not predict the intercept, linear slope or quadratic slope representing change in infant cortisol at day 2 or 1 month. Furthermore, sensitivity analyses were conducted including physical abuse, sexual abuse, and neglect in the latent growth curve models as covariates, and the results remained unchanged. Thus these alternate abuse experiences were considered no further. Additionally, a total ACES score that indicated the number of adversities experienced by the mother in childhood did not predict the intercept, linear slope or quadratic slope representing change in infant cortisol at day 2 or 1 month and thus was considered no further. None of the other maternal characteristics (age, race, marital status, education [high school degree], income, gravida) or infant characteristics (sex, delivery method, gestational age at birth, growth percentile at birth, Apgar score, or age at the observations) were associated with the mother witnessing IPV in childhood. Thus, only maternal depression and smoking in pregnancy were included in the models as covariates. Descriptive statistics and intercorrelations among the variables included in the analytic models are displayed in Table 1. Intercorrelations demonstrated significant over time associations of infant cortisol 40 minutes following the NNNS exam at day 2 (sample 4) with baseline cortisol, and cortisol 20 and 40 minutes following the NNNS exam at 1 month (samples 1, 3, and 4). Likewise, infant cortisol 20 minutes following the NNNS exam at day 2 (sample 3) was positively associated with infant cortisol 40 minutes following the NNNS exam at 1 month (sample 4).

Substantive analyses: Testing Hypothesis 1.

Predictors of infant cortisol at day 2.—Results of the latent growth curve models testing the mother's experience of witnessing IPV in childhood as a predictor of infant cortisol at day 2 are displayed in Models 1A, 1B, and 1C of Table 2. Witnessing IPV in childhood was not associated with the intercept, liner slope, or quadratic slope representing change in infant cortisol at day 2 (Model 1A). This pattern of results was consistent when prenatal family dysfunction was included in the model (Model 1B) and when prenatal perceived stress was included in the model (Model 1C). Examination of individual cortisol time points controlling for depression and smoking in pregnancy revealed that witnessing

IPV in childhood was not associated with any of the individual cortisol time points at day 2. In contrast, prenatal family functioning was significantly associated with the intercept, linear slope, and quadratic slope representing change in infant cortisol at day 2 (Model 1B). To understand the effect of family functioning on infant cortisol, we plotted change in cortisol at day 2 for the group of children above the median family dysfunction score (Median = 20) and below the median family dysfunction score. As illustrated in Figure 1, when prenatal family dysfunction was high (above the median), infants exhibited little response to the NNNS exam whereas when prenatal family dysfunction was low (below the median), infants demonstrated baseline cortisol that initially increased in response to the NNNS exam then eventually declined to lower levels. Examination of individual cortisol time points controlling for the mother's experience of witnessing IPV in childhood, and depression and smoking in pregnancy revealed that prenatal family dysfunction was significantly associated with sample 1 (baseline cortisol; $F(1) = 4.28, p = .040$) but not samples 2, 3, or 4. Perceived stress was not associated with the intercept, linear slope, or quadratic slope representing change in infant cortisol at day 2 (Model 1C), or any of the individual cortisol time points at day 2.

Predictors of infant cortisol at 1 month.—Results of the latent growth curve models testing the mother's experience of witnessing IPV in childhood as a predictor of infant cortisol at 1 month postpartum is displayed in Models 2A, 2B, and 2C of Table 2. Witnessing IPV in childhood was significantly associated with the intercept, linear slope, and quadratic slope representing change in infant cortisol at 1 month (Model 2A). This pattern of results was consistent when prenatal family dysfunction was included in the model (Model 2B) and when perceived stress was included in the model (Model 2C). As illustrated in Figure 2, infants whose mothers witnessed IPV exhibited elevated baseline cortisol that declined across the NNNS exam whereas infants whose mothers did not witness IPV demonstrated lower levels of baseline cortisol that initially increased in response to the NNNS exam then eventually declined to the initially low levels. Examination of individual cortisol time points controlling for depression and smoking in pregnancy revealed that the mother's experience of witnessing IPV in childhood was significantly associated with sample 1 (baseline cortisol; $F(1) = 5.57, p = .019$) but not samples 2, 3, or 4 at 1 month after delivery. In contrast to results for day 2, prenatal family functioning was not associated with the intercept, linear slope, or quadratic slope representing change in infant cortisol at 1 month (Model 2B), or any of the individual cortisol time points at 1 month. Likewise, perceived stress was not associated with the intercept, linear slope, or quadratic slope representing change in infant cortisol at 1 month (Model 2C), or any of the individual cortisol time points at 1 month.

Substantive analyses: Testing Hypothesis 2.

Family functioning and perceived stress were tested as moderators of the effect of the mother's experience of witnessing IPV in childhood on infant cortisol at 2 days and 1 month using procedures outlined by Aiken and West (1991). The interaction of witnessing IPV and prenatal family dysfunction was not a significant predictor of the intercept, linear slope, or quadratic slope representing change in infant cortisol at 2 days or 1 month postpartum. Likewise, the interaction of witnessing IPV and prenatal perceived stress was not a

significant predictor of the intercept, linear slope, or quadratic slope representing change in infant cortisol at 2 days or 1 month postpartum.

Post-hoc analyses: Maternal IPV victimization.

Although IPV victimization experienced by the mother was not measured in the studies from which data for this report was drawn, when physicians were aware of maternal IPV victimization it was noted in the medical charts reviewed for this research. Twenty-two of the mothers had reports of IPV victimization noted in their medical records. Preliminary analyses using these physician reports suggest that maternal IPV victimization determined through medical chart review mirrored the results we found for family dysfunction. Maternal IPV victimization was associated with the intercept ($B = -.43$, $SE = .20$, $p = .036$), linear slope ($B = .37$, $SE = .16$, $p = .020$), and quadratic slope ($B = -.05$, $SE = .03$, $p = .039$) at 2 days of age, but not 1 month of age. Furthermore, the mother witnessing IPV in childhood remained a significant predictor of the intercept ($B = .38$, $SE = .15$, $p = .012$), linear slope ($B = -.30$, $SE = .13$, $p = .024$), and quadratic slope ($B = .05$, $SE = .02$, $p = .028$) at 1 month when maternal IPV victimization was included as a covariate.

Discussion

Witnessing intimate partner violence in childhood is associated with increased mental health problems in children and adults, including alterations in HPA functioning (Cater, Miller, Howell, & Graham-Bermann, 2015; Salzman, Holden, & Holahan, 2005; Sturge-Apple, Davies, Cicchetti, & Manning, 2012). Less is known about the intergenerational consequences of witnessing IPV for HPA functioning in offspring. This is despite knowledge that exposure to conflict in the family of origin is linked with HPA functioning in young adulthood (Hagan, Roubinov, Purdom Marreiro & Luecken, 2014) and maternal HPA functioning in pregnancy contributes to alterations in fetal and infant biology (Davis et al, 2011; de Weerth et al., 2013). Furthermore, there is little knowledge of how the prenatal environment, including a highly functioning family unit, potentially buffers intergenerational effects of witnessing IPV. The current study builds upon this prior work to examine links between the mother's experience of witnessing IPV in childhood and her infant's cortisol in the first month of life, and examines the role of prenatal family functioning and perceived stress in these links. Results suggest that infants whose mothers witnessed IPV in their own childhood exhibit alterations in their baseline cortisol and their cortisol reactivity at one month of age, whereas family dysfunction during pregnancy is associated with infant cortisol in the first days of life. Family dysfunction and perceived stress in pregnancy did not moderate effects of the mother witnessing IPV in childhood on infant cortisol. These results were specific to the mother's experience of witnessing IPV in childhood. The mother's experience of physical abuse, sexual abuse, and neglect in childhood were not associated with baseline cortisol or infant cortisol reactivity in the first month.

To our knowledge, the current study is the first to demonstrate links between the mother's experience of witnessing IPV in childhood and her infant's HPA functioning. We found that infants whose mothers witnessed IPV exhibited elevated baseline cortisol at 1 month of age that declined in response to a temporary stressor. This is in contrast to infants whose mothers

did not witness IPV, who exhibited lower baseline cortisol and a more adaptive cortisol stress response characterized by temporary increases in cortisol that declined to initially low levels. These different patterns of infant cortisol reactivity may represent an early indicator of risk for psychopathology given that HPA functioning in early childhood is linked with children's biopsychosocial health and subsequent development (Alink et al., 2008; Lengua, Zalewski, Fisher, & Moran, 2013). Intergenerational patterns of violence are well documented (Kwong et al., 2003), it is possible that HPA functioning is a mechanism by which witnessing violence in the parent generation is associated with aggression and violent behavior among offspring. Supporting this possibility, basal cortisol at a year of age is associated with anger dysregulation at 3 years (Frost et al., 2017), and basal cortisol is concurrently associated with peer reports of aggression at 10 years (Oberle et al., 2017).

Another possibility is that alterations in infant HPA functioning associated with the mother witnessing IPV in childhood have origins in the grandmother's experience of the trauma of being a victim of IPV. Current conceptualization of intergenerational processes suggest that in addition to transmission through parental behavior, effects of stress and trauma are potentially transmitted through gametes and the uterine environment including maternal stress and neuroendocrine functioning in pregnancy (Bowers & Yehuda, 2016; Dias & Ressler, 2014). Supporting this possibility, offspring of rats who were aggressive to their female partners prior to conception were more aggressive to their own female partners even when they were not exposed to any parental aggression or stress (Cordero et al., 2012). Related, when mice were exposed to odor fear conditioning in the preconception period, both their children and grandchildren who were not exposed to any stress inherited the same sensitivity to the specific odor (Dias & Ressler, 2014). Further study using in-vitro fertilization and cross-fostering supported that this sensitivity was transmitted through parental gametes. Future work should carefully examine patterns of family violence, parenting behavior, and HPA function across generations to better understand potential transgenerational processes.

The mother's experience of witnessing IPV was associated with cortisol reactivity at one month postpartum but not infant cortisol immediately following delivery (day 2). In contrast, prenatal family dysfunction contributed to infant cortisol in the immediate postpartum period. Surprisingly, prenatal family dysfunction was not associated with infant cortisol at 1 month postpartum, and did not moderate links between witnessing IPV and infant cortisol. The birth of an infant is associated with substantial changes in family functioning and interpersonal relationships as the family integrates the newborn infant into the family system (Cox & Paley, 1997). Consequently, family dysfunction and maternal parenting behavior in the postpartum period may be more salient to infant cortisol at 1 month than is prenatal family dysfunction or perceptions of other stressors. Supporting this possibility, maternal sensitivity is linked with infant HPA functioning in the second year of life (Atkinson et al., 2013), and maternal sensitivity buffers children from effects of violence exposure (Hibel et al., 2011). Importantly, we considered infant HPA functioning in the first month following delivery, a period during which intergenerational factors may be more salient to infant stress responding than is the postpartum caregiving context which has yet to be fully established (Belsky, Gilstrap, & Rovine, 1984).

Interestingly, perceived stress in pregnancy was not associated with infant cortisol at either of our assessment timepoints in the first month postpartum. These non-significant effects are consistent with prior research using this same assessment of maternal perceptions of stress in pregnancy and infant cortisol 24 hours following birth (Davis et al., 2011). It is possible that discrete pregnancy stressors such as financial stressors, stressors associated with access to perinatal healthcare, or relationship stressors are more salient to the developing infant HPA system in the first month of life than are broader maternal perceptions of stress. It is also possible that the timing of the assessment of stress in pregnancy is relevant. Perceived stress earlier in pregnancy may be more salient to the development of infant HPA function. Future work should examine if maternal perceptions of stress in pregnancy are associated with change in infant HPA function over time to determine perceived stress becomes more salient to infant HPA function in the later postpartum period.

The mother's experience of witnessing IPV in childhood, not but her experience of physical abuse, sexual abuse, or neglect in childhood, was associated with infant cortisol reactivity. It is possible that witnessing IPV in childhood is associated with other psychological or social factors in pregnancy that contribute to offspring cortisol, including the mother's own experiences in her romantic relationship in pregnancy. Emotional Security Theory (Cummings & Davies, 2010) suggests that exposure to destructive forms of interparental conflict, including IPV, contributes to children's appraisals and representations of interparental relationships. Children may carry these representations forward into their own romantic relationships in adulthood, contributing to biopsychosocial processes during pregnancy and subsequently, the development of the infant HPA system. Future research should examine if psychosocial factors in pregnancy serve as mechanisms underlying the association of the mother's experience of witnessing IPV in childhood and offspring cortisol.

Prior studies considering cortisol reactivity in the first month following delivery are scant. Evidence that newborn cortisol reactivity is associated with temperamental reactivity at 6 months of age (Gunnar, Porter, Wolf, Rigatuso, & Larson, 1995), and that exposure to prenatal insults are associated with greater basal cortisol in the first week (Diego, Field, Hernandez-Reif, Cullen, Schanberg & Kuhn, 2004; Lundy et al., 1999) yet an attenuated cortisol stress response at 2 months (Ramsay, Bendersky, & Lewis, 1996), suggest that altered HPA functioning in the first month may be an early indicator of risk. Our observation that cortisol reactivity was attenuated at 1 month among infants whose mothers witnessed IPV in childhood is consistent with this prior work and suggests that efforts to support families who have an intergenerational history of trauma is warranted. The first month following delivery is a time during which the HPA system undergoes rapid change following the stress of birth (Stroud et al., 2014), and as discussed above, parental behavior has yet to become fully established given that parents are still adjusting to their infant's behavioral style and specific needs (Belsky et al., 1984). Thus, the first month of life may represent an important window of opportunity to support families with an intergenerational trauma history. Furthermore, the first month of life occurs prior to the stress "hyporesponsive period" during which the cortisol stress response dampens to protect the developing brain (Gunnar & Quevedo, 2007). Intergenerational consequences of the mother witnessing IPV in childhood on infant HPA functioning, and the effects of interventions to support families

with a trauma history on HPA functioning, may be more likely to be observed in the first month than later in infancy when it is more difficult to observe a cortisol stress response. Indeed, there is now evidence using prospective longitudinal methods that effects of early life stress on child HPA function extend into young adulthood (Kumsta et al., 2017), further underscoring the need for research and early intervention with infants and young children at risk. More work is needed in this domain to better understand these processes, and to deepen knowledge of the potential lasting impact of HPA functioning in the early infancy period for long term health and developmental outcomes

Results of the current study add to accumulating knowledge that stressors in both the preconception and prenatal periods have the potential to “program” the infant HPA system. Our findings that both the mother’s experience of witnessing IPV in childhood, and prenatal family dysfunction, alter the infant HPA stress response are consistent with prior work focusing on maternal exposure to violence in childhood and pregnancy, albeit in a different way. Prior work has demonstrated that infants whose mothers experienced sexual or physical abuse in childhood exhibit lower basal cortisol at 6 months of age than infants whose mothers were not abused (Brand et al., 2010), and maternal PTSD in response to the World Trade Center Attacks during pregnancy is associated with lower infant basal cortisol at 1 year (Yehuda et al., 2005). Further, intimate partner violence during pregnancy is associated with greater infant cortisol reactivity to laboratory challenge at 1 year (Levendosky et al., 2016). Our findings of greater baseline cortisol and a blunted cortisol stress response in relation to both family dysfunction and the mother’s experience of witnessing IPV in childhood suggest that developmental processes may shape the direction of the effect of prenatal exposures on infant physiology. We focused on infant cortisol during the first month whereas most prior work focused on infant cortisol in later infancy when it is more difficult to detect a cortisol stress response and a blunted cortisol response may be adaptive (Gunnar, Talge, & Herrera, 2009). Consistent with our results, maternal depression in pregnancy was associated with greater basal cortisol among infants in the first 24 hours and first week postpartum (Diego, Field, Hernandez-Reif, Cullen, Schanberg & Kuhn, 2004; Lundy et al., 1999). Furthermore, greater newborn cortisol reactivity was associated with lower temperamental reactivity at 6 months of age, suggesting that a more reactive HPA system in the neonatal period may be adaptive (Gunnar, Porter, Wolf, Rigatuso, & Larson, 1995).

The current study is characterized by a number of strengths including multiple assessments of infant HPA functioning across the first month, a socio-demographically diverse sample, and careful measurement of covariates and exclusionary variables that are known to influence infant HPA functioning. However, the study is also characterized by important limitations and directions for future research. This area of inquiry is exceedingly complicated given the involvement of maternal, placental, and fetal inputs and more research is needed to understand how maternal stress exposure before and during pregnancy influences the evolution of infant HPA development. First, we drew upon maternal retrospective reports of witnessing IPV in childhood rather than a prospective longitudinal design from the mother’s childhood through her pregnancy. Retrospective reports of childhood experience may be influenced by emotional dispositions and states of mind, including the current family environment and maternal mental health. However, that links between the mother’s experience of witnessing IPV in childhood and infant cortisol

reactivity remained significant even when controlling for the mother's experiences of depression, perceived stress, and family dysfunction in pregnancy, further support the perspective that the consequences of witnessing IPV may be passed down to offspring. Notably, women who experienced other forms of childhood trauma (physical abuse, sexual abuse, and neglect) may also have underreported these experiences and associations of these alternate abuse experiences and infant HPA function may have been underestimated. Prospective research designs or other more highly validated methods to assess child maltreatment and violence exposure (e.g. review of child protection records) would be advantageous for future work in this area. Related, although we considered family dysfunction and stress in the prenatal period as covariates and moderators of the impact of witnessing IPV in childhood, IPV experienced by the mother herself examined systematically would be the most conservative covariate and moderator to test. Preliminary analyses using physician reports of maternal IPV victimization determined through medical chart review mirrored the results we found for family dysfunction: maternal IPV victimization was associated with infant cortisol at 2 days of age, but not 1 month of age, and effects of witnessing IPV in childhood remained significant when maternal IPV victimization was included as a covariate. It is well established that women underreport IPV victimization to their medical providers, therefore future work should attempt to replicate these results using more careful measurement of IPV in pregnancy. Likewise, not all women in the larger study from which the current sample was drawn reported on their childhood experiences. Although the measure of maternal childhood experiences was added following the first few months of data collection, it is possible that some of the missing data was due to women who chose not to complete the measure to minimize or avoid reporting traumatic experiences. We did not ask women why they skipped questions and all aspects of the study were voluntary. For this reason, our results may have underestimated the association of witnessing IPV in childhood and infant HPA function. Likewise, associations of other alternate abuse experiences and infant HPA function may have been underestimated. Furthermore, our assessment of witnessing IPV focused exclusively on victimization of mothers by male partners. Although women are more likely to be victims of IPV than men (Black et al., 2011), future works should also consider male victimization by female partners, or IPV between same sex partners. Additionally, we utilized maternal depression status derived from DSM-IV criteria as a covariate. Future research should consider other indicators of maternal depressive symptoms and severity to potentially capture more subtle effects of maternal mood in the perinatal period as it relates to childhood exposure to IPV and infant HPA functioning. Related, the current sample was over-sampled for maternal depression and smoking. Although these effects were controlled statistically, it is possible that sample selection or interactions with smoking in particular may explain differences between the present study and prior literature. Finally, we do not know the association between childhood stress exposure and placental gene methylation that may protect the fetus from maternal glucocorticoid exposure. It is possible that placental gene methylation mediates or moderates associations of maternal early life stress, including witnessing IPV, and infant HPA function. Despite these limitations the current study is the first to document an intergenerational effect of witnessing IPV in childhood on offspring HPA functioning.

In conclusion, we found that the mother's experience of witnessing IPV in her childhood and prenatal family functioning were associated with infant cortisol reactivity in the first month postpartum, albeit in different ways. Witnessing IPV in childhood was associated with infant cortisol at 1 month postpartum, whereas prenatal family dysfunction was associated with infant cortisol at 2 days postpartum. Results support the view that parental early adversity has the potential to exert intergenerational effects on offspring. Interventions to support healthy familial relationships and prevent IPV may enhance health outcomes in children at risk for violence exposure, as well the health of their offspring.

Acknowledgments

Funding agencies:

This work was supported by the National Institutes of Health under grants R01 MH079153 and R01 DA019558.

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Figure 1.
Prenatal Family Functioning is Associated with Change in Infant Cortisol at Day 2

Figure 2.
Maternal Childhood Exposure to IPV is Associated with Change in Infant Cortisol at One Month Postpartum

Table 1.

Descriptive Statistics and Intercorrelations

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Witnessed IPV in Childhood	--												
2. Prenatal Family Functioning	.11	--											
3. Prenatal Perceived Stress	.09	.49**	--										
4. Depressed in Pregnancy	.15*	.26**	.49**	--									
5. Smoked in Pregnancy	-.04	.06	.09	-.02	--								
6. Cortisol (ug/dL): Sample 1, Day 2	-.10	.13	.06	.07	-.20**	--							
7. Cortisol (ug/dL): Sample 2, Day 2	.01	-.03	.08	.09	-.02	.47**	--						
8. Cortisol (ug/dL): Sample 3, Day 2	.04	-.14	-.10	-.02	.07	.15	.82**	--					
9. Cortisol (ug/dL): Sample 4, Day 2	-.01	-.12	-.10	-.01	.07	.16	.76**	.93**	--				
10. Cortisol (ug/dL): Sample 1, 1 Month	.15*	-.02	-.13	-.07	.00	.10	.00	-.08	-.19*	--			
11. Cortisol (ug/dL): Sample 2, 1 Month	-.02	-.11	-.07	.00	-.14	.06	.04	.08	.12	.34**	--		
12. Cortisol (ug/dL): Sample 3, 1 Month	-.06	-.07	-.06	.07	-.14	.03	.08	.17	.20*	.08	.80**	--	
13. Cortisol (ug/dL): Sample 4, 1 Month	.04	-.04	-.07	.08	-.14	.05	.09	.20*	.20*	.15*	.59**	.78**	--
Mean (SD) / %	26%	20.88 (5.75)	21.51 (8.38)	31%	29%	.78 (.78)	.98 (.86)	1.07 (.83)	.90 (.77)	.40 (.54)	.42 (.48)	.36 (.41)	.26 (.33)

Notes:

**
 $p < .01$.*
 $p < .05$.

Raw cortisol values presented for descriptive statistics (Mean and SD).

Cortisol values log transformed prior to hypothesis testing and for intercorrelations.

Correlations among dichotomous variables (Witnessed IPV in childhood, depressed in pregnancy, and smoked in pregnancy) are phi-coefficients.

Correlations of dichotomous variables (Witnessed IPV in childhood, depressed in pregnancy, and smoked in pregnancy) with continuous variables (prenatal family functioning, prenatal perceived stress, and all measures of infant cortisol) are point-biserial correlations.

Table 2.

Latent Growth Curve Models Predicting Change in Infant Cortisol

Predictor Variables	Infant Cortisol at 2 Days			Infant Cortisol at 1 Month		
	Model 1A	Model 1B	Model 1C	Model 2A	Model 2B	Model 2C
	B (SE)	B (SE)	B (SE)	B (SE)	B (SE)	B (SE)
<u>Intercept</u>						
Depression in Pregnancy	.151 (.133)	.054 (.136)	.032 (.159)	-.230 (.142)	-.255 (.146)	-.156 (.165)
Smoking in Pregnancy	-.373 (.133)**	-.418 (.132)**	-.381 (.134)**	.183 (.144)	.176 (.144)	.191 (.143)
Witnessing IPV in Childhood	-.188 (.141)	-.218 (.139)	-.197 (.142)	.398 (.153)**	.391 (.154)*	.359 (.153)*
Prenatal Family Functioning		.029 (.011)*		.008 (.012)		
Prenatal Perceived Stress			.010 (.009)			-.007 (.009)
<u>Slope</u>						
Depression in Pregnancy	-.096 (.106)	-.020 (.109)	-.003 (.126)	.175 (.123)	.209 (.126)	.143 (.144)
Smoking in Pregnancy	.247 (.106)*	.284 (.105)**	.253 (.107)*	-.216 (.124)	-.206 (.124)	-.213 (.125)
Witnessing IPV in Childhood	.122 (.122)	.145 (.110)	.132 (.112)	-.304 (.133)*	-.293 (.133)*	-.285 (.133)*
Prenatal Family Functioning		-.023 (.009)*			-.011 (.010)	
Prenatal Perceived Stress			-.008 (.007)			.002 (.008)
<u>Quadratic</u>						
Depression in Pregnancy	.013 (.017)	.002 (.018)	.000 (.021)	-.025 (.022)	-.032 (.023)	-.019 (.026)
Smoking in Pregnancy	-.031 (.017)	-.037 (.017)*	-.032 (.017)	.037 (.022)	.036 (.022)	.037 (.022)
Witnessing IPV in Childhood	-.016 (.018)	-.020 (.018)	-.018 (.018)	.053 (.024)*	.051 (.024)*	.051 (.024)*
Prenatal Family Functioning		.003 (.001)*			.002 (.002)	
Prenatal Perceived Stress			.001 (.001)			.000 (.001)

Note:

**
 $p < .01$.*
 $p < .05$.