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Maternal Lifetime Trauma Exposure, Prenatal Cortisol, and Infant Negative Affectivity

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Abstract

Little research has examined the impact of maternal lifetime trauma exposure on infant temperament. We examined associations between maternal trauma history and infant negative affectivity and modification by prenatal cortisol exposure in a sociodemographically diverse sample of mother–infant dyads. During pregnancy, mothers completed measures of lifetime trauma exposure and current stressors. Third-trimester cortisol output was assessed from maternal hair. When infants were 6 months old, mothers completed the Infant Behavior Questionnaire-Revised. In analyses that controlled for infant sex and maternal age, education, race/ethnicity, and stress during pregnancy, greater maternal trauma exposure was associated with increased infant distress to limitations and sadness. Higher and lower prenatal cortisol exposure modified the magnitude and direction of association between maternal trauma history and infant rate of recovery from arousal. The association between maternal trauma history and infant distress to limitations was somewhat stronger among infants exposed to higher levels of prenatal cortisol. The analyses suggested that maternal lifetime trauma exposure is associated with several domains of infant negative affectivity independently of maternal stress exposures during pregnancy and that some of these associations may be modified by prenatal cortisol exposure. The findings have

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implications for understanding the intergenerational impact of trauma exposure on child developmental outcomes.

Research indicates that infant temperament has significant long-term consequences for development, including influencing later personality and social development and risk for emotional and behavioral problems (Gartstein & Rothbart, 2003). Domains of negative affectivity (fear, sadness, distress reactivity and recovery) may have particular import for developmental outcomes. Measures of infant negative affectivity show similarity to child measures of negative affectivity and to the adult personality factor of Neuroticism (Gartstein & Rothbart, 2003), suggesting that negative affectivity may be relatively stable across the life course. Moreover, elevated negative affectivity appears to heighten sensitivity to environmental influences, increasing the likelihood of negative outcomes (e.g., externalizing behaviors) under poor environmental conditions and positive outcomes (e.g., self-control, morality, academic, and social competence) under enriched conditions (Belsky & Pluess, 2009; Feldman, Greenbaum, & Yirmiya, 1999; Kochanska, Aksan, & Joy, 2007; Pluess & Belsky, 2009; Van Aken, Junger, Verhoeven, van Aken, & Dekovic, 2007). Thus, explicating factors that shape negative affectivity may inform our understanding of the earliest origins of mental health risk, resilience, and other developmental outcomes.

Prenatal stress appears to influence infant negative affectivity (Davis et al., 2007). The fetal brain develops rapidly and is particularly vulnerable to stress effects (Davis et al., 2007). Maternal stress and trauma exposures during pregnancy are associated with maternal report of increased infant distress in response to novelty, poor recovery from distress, fearfulness, and difficult temperament (Brand, Engel, Canfield, & Yehuda, 2006; Davis et al., 2007; Huizink, de Medina, Mulder, Visser, & Buitelaar, 2002; Jansen et al., 2009). Notably, almost no studies have reported testing associations between maternal stress and infant sadness. Moreover, one study found that low prenatal socioeconomic status, a potential proxy for stress, was linked to elevated infant fear and distress and poorer distress recovery but to lower infant sadness (Jansen et al., 2009).

Some posit that heightened negative affectivity is the behavioral manifestation of extreme physiological responses to stress (Belsky & Pluess, 2009). Prenatal cortisol exposure is hypothesized to program the fetus's hypothalamic–pituitary–adrenal axis (HPAA) and other neural systems involved in the stress response (e.g., amygdala); thus, functioning of the maternal HPAA in pregnancy may have a critical influence on infant negative affectivity (Davis et al., 2007). Studies have linked maternal stress during pregnancy to disruptions in prenatal HPAA functioning, including a blunted morning response and flatter waking to bedtime rhythm in early to mid-pregnancy and elevated evening cortisol in late pregnancy (Obel et al., 2005; Suglia et al., 2010). Moreover, greater maternal HPAA reactivity in pregnancy is associated with increased fear, distress, irritability, and difficult behaviors in infants and toddlers (Davis et al., 2005, 2007; De Weerth, van Hees, & Buitelaar, 2003; Field & Diego, 2008).

Although several studies document positive associations among maternal stress exposures, prenatal cortisol output, and infant negative affectivity, inconsistencies have been reported. For example, some studies link maternal stress in pregnancy to lowered prenatal cortisol

levels (Suglia et al., 2010); others report weak or no associations (Baibazarova et al., 2013; Bergman, Glover, Sarkar, Abbott, & O'Connor, 2010; Davis et al., 2007; DiPietro, 2012; Doyle et al., 2015; Zijlmans, Riksen-Walraven, & de Weerth, 2015). Meta-analysis findings indicate that stress exposures are associated with both hyper- and hypocortisolemia, with the direction of effects dependent on stressor and person characteristics, including the nature and timing of the stress exposure and the emergence of psychopathology (Luo et al., 2012; Miller, Chen, & Zhou, 2007). Further, some studies failed to find associations between maternal pregnancy cortisol levels and infant temperament or only found indirect relations through mediating factors (Baibazarova et al., 2013; Bergman et al., 2010). These complex relationships have complicated efforts to specify associations among maternal stress, prenatal cortisol, and infant temperament and likely contribute to inconsistent findings in the literature.

To date, studies assessing links between maternal HPAA disruption in pregnancy and infant temperament have relied on salivary or serum measures of cortisol. Such measures represent relatively short-term assessments of HPAA functioning and thus may not provide an accurate depiction of the fetus's overall cortisol exposure. The effects of prenatal HPAA disruption on infant temperament may be better quantified by methods that characterize longer term HPAA activity during pregnancy (D'Anna-Hernandez, Ross, Natvig, & Laudenslager, 2011; Kalra, Einarson, Karaskov, Van Uum, & Koren, 2007; Kirschbaum, Tietze, Skoluda, & Dettenborn, 2009; Russell, Koren, Rieder, & Van Uum, 2012). Measures of concentration of cortisol in hair are emerging as promising markers of long-term HPAA activity (Braig et al., 2015; Kalmakis, Meyer, Chiodo, & Leung, 2015; Russell et al., 2012; Staufenbiel, Penninx, Spijker, Elzinga, & van Rossum, 2013; Wosu, Valdimarsdottir, Shields, Williams, & Williams, 2013). Furthermore, hair cortisol levels show expected increases over the course of pregnancy and correlate with prenatal measures of diurnal salivary cortisol (e.g., mean daily salivary concentrations; area under the curve for diurnal salivary cortisol; 24-h urinary cortisol concentrations), stress during pregnancy, and child hair cortisol up to at least age 3 years (D'Anna-Hernandez et al., 2011; Kalra et al., 2007; Karlen, Frostell, Theodorsson, Faresjo, & Ludvigsson, 2013; Kirschbaum et al., 2009; Wosu et al., 2013). As with salivary cortisol, there is evidence that both elevated and reduced hair cortisol concentrations are associated with stress, depending on stressor characteristics (Wells et al., 2014). Importantly, hair cortisol measures are not affected by factors that influence salivary and serum cortisol protocols, including participant nonadherence, circadian patterns, invasiveness, and situational characteristics (e.g., stress of sampling, eating before sampling) (Braig et al., 2015; Stalder & Kirschbaum, 2012; Wosu et al., 2013). Together, these data suggest that hair cortisol measures may provide a more valid method of assessing the impact of maternal prenatal HPAA functioning on infant temperament.

The extant literature has a number of limitations that hamper efforts to specify the nature of associations among maternal stress, prenatal cortisol exposure, and infant negative affectivity. First, studies have been inconsistent in their operationalization of prenatal stress, using measures of stress exposures during pregnancy, overall perceptions of stress, symptoms of depression and anxiety, and/or pregnancy-specific anxiety. Furthermore, these studies have largely ignored the potential impact of maternal lifetime stress exposures on infant temperament, although such an approach may be necessary. Maternal exposures to

stressors throughout life, particularly during childhood, can result in permanent disruptions to maternal physiological systems that affect prenatal health (e.g., neuroendocrine, autonomic nervous system [ANS], immune) and, consequently, programming of the fetal brain and stress regulatory systems involved in infant temperament (Anda et al., 2006; Enlow et al., 2009; Sternthal et al., 2009; Yehuda & Bierer, 2008). Exposure to traumatic events, that is, events involving threat of serious harm/death to self/others (American Psychiatric Association, 2013), may have particular import given their increased likelihood of effecting permanent and extreme changes in relevant psychobiological systems (Anda et al., 2006; Enlow et al., 2009). Moreover, consideration of accumulation of exposures may be necessary, as evidence suggests that chronic and multiple forms of trauma are most impactful (Anda et al., 2006). A limited number of studies report associations between maternal childhood or lifetime trauma history and infant negative affectivity (Bosquet Enlow et al., 2011; Lang, Gartstein, Rodgers, & Lebeck, 2010). Others have linked trauma exposure to both elevated and depressed hair cortisol concentrations (Luo et al., 2012; Steudte et al., 2011, 2013). Notably, maternal childhood and lifetime trauma exposures have been associated with greater maternal hair cortisol levels reflecting the course of pregnancy (Schreier, Enlow, Ritz, Gennings, & Wright, 2015; Schreier et al., 2016).

Second, prenatal cortisol exposure has primarily been theorized as a mediator of maternal stress effects on infant temperament (D'Anna-Hernandez et al., 2011; Davis et al., 2007; Field & Diego, 2008), with strong support from animal studies (Bergman et al., 2010). However, evidence in human studies is weak (Bergman et al., 2010; Davis et al., 2007; Doyle et al., 2015), leading some to suggest that an HPAA-mediated link between prenatal stress and infant temperament may be more complicated than assumed (Bergman et al., 2010). Therefore, other types of relations among maternal stress, prenatal cortisol exposure, and infant temperament should be explored. Also, most studies presuppose that prenatal cortisol levels have a positive linear relationship with both maternal stress exposures and infant negative affectivity despite evidence that hypocortisolemia may result from stress exposure and predict difficulties with child emotion regulation (Kalmakis et al., 2015; Miller et al., 2007; Smider et al., 2002; Tout, de Haan, Campbell, & Gunnar, 1998). Thus, studies should explore whether prenatal exposure to elevated or depressed cortisol levels influence associations between maternal stress exposures and infant negative affectivity.

Finally, within studies, only one or two infant temperamental traits (e.g., fearfulness) are typically examined. Consequently, the literature is unclear as to whether prenatal stress has distinct effects on different domains of infant negative affectivity or broad effects across domains. This lack of a comprehensive approach to assessing infant negative affectivity may contribute to inconsistencies in the literature. Studies that examine whether associations among maternal stress, prenatal cortisol exposure, and infant negative affectivity vary by specific negative affectivity domain are needed.

Given these limitations, the goals of this study were to (1) examine associations between maternal lifetime trauma exposure history and several domains of infant negative affectivity (distress to limitations; rate of recovery from arousal; fearfulness; sadness) in 6-month-old infants and (2) test the modifying effects of maternal prenatal HPAA activity on the relationship between maternal trauma history and infant negative affectivity. To the best of

our knowledge, this is the first study to test the impact of maternal lifetime trauma exposure on infant temperament independent of prenatal stress exposures, to examine the roles of both elevated and reduced levels of prenatal cortisol exposure as modifiers of associations between maternal trauma history and infant temperament, and to relate cortisol measured in maternal hair to infant temperament.

In addition to the literature described above, our hypotheses were influenced by studies that have linked infant fear with later internalizing problems, child internalizing problems (anxiety, social wariness) with hypercortisolemia, infant distress to limitations and poor arousal recovery with later externalizing problems, and externalizing problems with hyper- and hypocortisolemia (Jansen et al., 2009; Miller et al., 2007; Smider et al., 2002; Tout et al., 1998). Specifically, we hypothesized that maternal trauma history is positively associated with infant fearfulness and distress, negatively associated with arousal recovery, and not associated with sadness. We further hypothesized that the association of maternal trauma history with infant fear is strengthened under conditions of elevated prenatal cortisol exposure and that the associations of maternal trauma history with infant distress and arousal recovery are strengthened under conditions of elevated or reduced prenatal cortisol exposure. Because there are no established cut-off levels at which prenatal cortisol influences infant behavior, we utilized a continuous measure of prenatal cortisol in analyses.

METHOD

Participants

Participants were mothers and their 6-month-old infants enrolled in the PRogramming of Intergenerational Stress Mechanisms (PRISM) study, a prospective pregnancy cohort designed to examine the role of perinatal stress exposures on child development. Between March 2011 and December 2013, pregnant women were recruited from prenatal clinics in a Boston area hospital and a community health center. Recruitment sites were chosen given desired heterogeneity in sociodemographic and racial/ethnic characteristics. Eligibility criteria included (1) English- or Spanish-speaking, (2) age ≥ 18 years at enrollment, and (3) single gestation birth. Exclusion criteria included (1) maternal endorsement of drinking ≥ 7 alcoholic drinks/week prior to pregnancy recognition or any alcohol following pregnancy recognition, as usage above these thresholds has been associated with increased risk for numerous health problems (Patra et al., 2011; Testa, Quigley, & Eiden, 2003), and (2) maternal or child chronic health conditions that would impede study participation. Among eligible women, 319 (71% of those approached) agreed to participate and continued follow-up after delivery. Based on screening data, there were no significant differences on race/ethnicity, education, or income between women who enrolled and those who declined. Mother–infant dyads with data on maternal lifetime trauma exposure history, pregnancy stressors, and infant temperament were included in the current analyses, resulting in a sample size of $N = 289$ dyads (149 male infants).

Approximately 6 months after study initiation, additional funding was obtained to collect hair for cortisol assessment. All women subsequently enrolled ($n = 229$) consented to hair collection. Of these, 35 women did not provide a sufficient hair sample (≥ 6 cm) within 1 week of delivery or were excluded from the hair cortisol analyses due to shift work or

exogenous steroid use in the past 6 months, as these factors may influence cortisol levels (Granger, Hibel, Fortunato, & Kapelewski, 2009). The resulting 194 women who provided usable hair samples differed from the 95 who did not on maternal race/ethnicity, $p = .002$, with cortisol data provided by 60% of White participants, 58% of Black participants, 82% of Hispanic participants, and 65% of participants who self-identified as another race/ethnicity (primarily Asian or multiracial). Mothers who did and did not provide cortisol data did not differ on age, education, lifetime trauma exposures, prenatal stress exposures, or their infant's sex, $ps > .05$.

Measures

Maternal lifetime trauma exposure—Maternal lifetime exposure to traumatic events was measured via interview using the Life Stressor Checklist-Revised (LSC-R) (Wolfe & Kimerling, 1997). The LSC-R assesses exposure to 30 potentially traumatic events (e.g., experiencing or witnessing a serious accident or natural disaster, childhood maltreatment), including experiences particularly relevant to women (e.g., sexual assault, interpersonal violence). For each endorsed event, the LSC-R includes a follow-up question regarding whether the respondent thought that she or someone else could be killed or seriously harmed during the event; this qualifier allows events to be scored as to whether they met the Diagnostic and Statistical Manual of Mental Disorders—5th Edition (DSM-V) post-traumatic stress disorder (PTSD) Criterion A for classifying an event as traumatic (American Psychiatric Association, 2013). A maternal lifetime trauma exposure score was derived from the number of endorsed events during which the mother reported thinking that she or someone else could be killed or seriously harmed. The LSC-R has established test–retest reliability and validity in diverse populations (McHugo et al., 2005; Wolfe & Kimerling, 1997).

Infant negative affectivity—Infant negative affectivity was ascertained using the Infant Behavior Questionnaire-Revised (IBQ-R) (Gartstein & Rothbart, 2003). The 191-item IBQ-R has demonstrated good reliability and validity in 6-month-olds (Parade & Leerkes, 2008) and has been used in English- and Spanish-speaking populations to assess infant temperament (Carnicero, Perez-Lopez, Salinas, & Martinez-Fuentes, 2000; Gartstein et al., 2006). The IBQ-R was administered as an interview, with the mother shown a card with response choices. Mothers rated the frequency that their infant engaged in specific day-to-day behaviors in the prior week using a seven-point scale, with responses ranging from 1 (*never*) to 7 (*always*). Scores were summed across items according to IBQ-R scoring criteria to create 14 scales assessing different behavioral domains.

Research with the IBQ-R has identified a negative affectivity factor comprised of four scales: Distress to Limitations/Frustration, Falling Reactivity/Rate of Recovery (hereafter called “Falling Reactivity”), Fear, and Sadness (Gartstein & Rothbart, 2003). The 16-item Distress to Limitations scale assesses propensity to fussing, crying, or showing other indicators of distress during caretaking activities or when confined or unable to perform a desired action; higher scores indicate greater tendency toward distress. The 13-item Falling Reactivity scale assesses rate of recovery from peak distress, excitement, or general arousal; higher scores indicate more rapid recovery from arousal. The 16-item Fear scale assesses

inhibited approach to novelty as well as the tendency to startle or show distress to sudden changes in stimulation or to novel objects or social stimuli; higher scores indicate greater fearfulness. The 14-item Sadness scale assesses general low mood and lowered mood and activity related to personal suffering, physical state, object loss, or inability to perform a desired action; higher scores indicate greater propensity to sadness. Analyses focused on these four scales and the overarching Negative Affectivity Factor, which was calculated by averaging the mean scores of the four scales (Falling Reactivity reverse scored); higher scores indicate greater negative affectivity.

Hair cortisol—Hair cortisol during the third trimester of pregnancy was assessed from participants' scalp hair, collected within 1 week postdelivery. A hair sample 3 mm in diameter was cut close to the scalp at the posterior vertex, the suggested position given that this region has the most uniform growth rate, lowest interindividual variability, and lowest proportion of telogen (Sauve, Koren, Walsh, Tokmakejian, & Van Uum, 2007; Stalder & Kirschbaum, 2012). Given a hair growth rate of approximately 1 cm/month (Wennig, 2000), the 3-cm segment closest to the scalp was used to reflect cumulative cortisol secretion during the third trimester, as previously described (Braig et al., 2015; Kirschbaum et al., 2009). Data support the use of cortisol measured from hair collected postpartum as an indicator of maternal prenatal HPA activity (Braig et al., 2015; D'Anna-Hernandez et al., 2011; Kirschbaum et al., 2009).

Hair samples were analyzed in the Kirschbaum laboratory at the Technical University of Dresden, Germany. Washing and steroid extraction followed an established protocol (Stalder et al., 2013). Hair was washed in isopropanol, and cortisol was extracted from 7.5 mg of whole nonpulverized hair using methanol in the presence of internal standards. Samples were centrifuged at $15,200 \times g$, and the supernatant was collected; alcohol was evaporated under a stream of nitrogen and reconstituted with double-distilled water and then injected into a Shimadzu HPLC–tandem mass spectrometry system (Shimadzu, Canby, Oregon) coupled to an AB Sciex API 5000 Turboion-spray triple quadrupole tandem mass spectrometer (AB Sciex, Foster City, CA, USA), with purification by online solid-phase extraction (Gao et al., 2013). Lower limits of quantification were 0.1 pg/mg; inter- and intra-assay variabilities were 3.7–8.8%.

Cortisol output during the third trimester was used because studies suggest that cortisol in late pregnancy may have the greatest influence on infant negative affectivity (e.g., Baibazarova et al., 2013; Davis et al., 2007; Yehuda et al., 2005; Zijlmans et al., 2015). Additionally, because the segment of hair representing the third trimester was that closest to the scalp, all consenting women were able to provide samples, with fewer having hair long enough to measure cortisol earlier in pregnancy (~50% had sufficient hair to assess first trimester levels). Previous reports on this cohort showed that, among women with cortisol levels assessed from hair samples representing multiple trimesters, the correlation coefficients across trimesters ranged from .86 to .94; thus, mothers in this sample generally maintained their relative cortisol level ranking across trimesters (Schreier et al., 2015, 2016).

Data are inconsistent as to whether hair characteristics or hair care routines are associated with hair cortisol levels (Braig et al., 2015; Kirschbaum et al., 2009; Russell et al., 2012;

Wosu et al., 2013). In this sample, hair cortisol levels were not associated with natural hair color, with whether the hair was artificially colored, chemically straightened, or curled in the past year, or with the use of gels, oils, or sprays on the day of sampling, $ps > .25$. Therefore, these variables were not considered further.

Maternal stress exposures during pregnancy—Maternal stress exposures during pregnancy were assessed using the Crisis in Family Systems-Revised (CRISYS-R) survey, which inquires about exposure to negative life events during the prior 6 months (Berry, Shalowitz, Quinn, & Wolf, 2001). The CRISYS-R is suitable for sociodemographically diverse populations, has good test/retest reliability, has been validated in English and Spanish in samples of parents, and has been utilized in several studies as a measure of prenatal stress (Berry, Quinn, Portillo, & Shalowitz, 2006; Cowell et al., 2015; Suglia et al., 2010; Tse, Rich-Edwards, Koenen, & Wright, 2012). The survey encompasses 11 domains (financial, legal, career, stability in relationships, medical issues pertaining to self, medical issues pertaining to others, safety in the community, safety in the home, housing problems, difficulty with authority, discrimination), with multiple items assessing each domain. Women rated endorsed items as positive, negative, or neutral. Research suggests increased vulnerability when exposed to negative events across multiple domains (Myers, 2009). Thus, the number of domains with one or more negative events endorsed was summed to create a Negative Life Events Domain Score (NDS; possible range 0–11), as performed in prior research (Cowell et al., 2015). Higher scores indicate greater stress exposures.

Sociodemographic covariates—Mothers self-reported their age, race/ethnicity, and highest level of education. Race/ethnicity was categorized into White, Black/Haitian, Hispanic, and other. Education was dichotomized into “completion of high school or less education” or “education beyond high school degree.” Infant sex was also included as a covariate in analyses.

Perinatal risk variables—Factors previously associated with maternal prenatal stress/trauma exposure, hair cortisol in pregnancy, and/or poor infant outcomes were assessed as potential confounders, including maternal prenatal cigarette smoking, maternal prenatal body mass index (BMI), delivery method (vaginal, Cesarean section), and infant gestational age and birthweight (Baibazarova et al., 2013; Braig et al., 2015; Karlen et al., 2013). Prenatal smoking was categorized yes/no based on maternal self-report as to whether she smoked during pregnancy, a validated method for classifying prenatal smoking status (Pickett, Kasza, Biesecker, Wright, & Wakschlag, 2009). Mothers self-reported pre-pregnancy weight and height; BMI was calculated by dividing weight (kg) by height squared (meter). A previously reported internal validation analysis demonstrated high agreement between height and weight measured <10 weeks into pregnancy and self-reported values (Wright et al., 2013). Delivery method and infant gestational age and birthweight were extracted from medical records.

Procedure

Participant sociodemographics were assessed shortly following recruitment ($M = 26.9$ weeks gestation, $SD = 8.1$ weeks gestation). Within 2 weeks of enrollment, trained research

assistants administered the LSC-R and CRISYS-R. Within 1 week postdelivery, staff collected maternal hair samples. When the infants were approximately 6 months of age ($M = 28.5$ weeks, $SD = 2.2$ weeks), mothers completed the IBQ-R. Study procedures were approved by the relevant institutions' human studies ethics committees. Mothers provided written informed consent in their preferred language.

Data analytic plan

Data analyses proceeded in several steps. First, descriptive statistics were calculated. Third-trimester hair cortisol scores were log-transformed to reduce skewness. *T*-test analyses tested whether male and female infants differed on the IBQ-R variables. ANOVAs followed by Tukey's HSD post hoc tests analyzed differences by race/ethnicity on maternal lifetime trauma exposure (i.e., LSC-R score), stress exposures during pregnancy (i.e., NDS score from CRISYS-R), and third-trimester hair cortisol levels. Spearman bivariate correlations tested associations of the perinatal risk factors with the predictor and outcome variables. Spearman bivariate correlation coefficients were also calculated among the main variables.

Next, linear regression models assessed the association between maternal lifetime trauma exposure and the infant IBQ-R scores (Negative Affectivity Factor, Distress to Limitations, Falling Reactivity, Fear, Sadness). Each IBQ-R score was modeled separately. All models controlled for maternal stress exposures during pregnancy as well as infant sex and maternal age, education, and race/ethnicity, as these sociodemographic factors have been linked to maternal stress/trauma and child behavioral outcomes (Nomaguchi & House, 2013; Palmer et al., 2013).

Finally, analyses extended each linear regression model to assess modification of maternal lifetime trauma history associations with infant negative affectivity scores by prenatal cortisol level. Because the literature suggests that associations between maternal trauma exposures and infant negative affectivity may be more extreme under conditions of elevated or reduced prenatal cortisol exposure, the modification hypothesis was tested using varying coefficient models that allowed the interaction between continuous IBQ-R and prenatal cortisol scores to be nonlinear as a function of prenatal cortisol level. The varying coefficient model took the form $LSC-R_i = \beta_0 + \beta_1 IBQ-R_i + \beta_2(c) + \beta_3(c) IBQ-R_i + \beta_4 \chi_i + \epsilon_i$, where $\beta_2(c)$ is the main, potentially nonlinear smooth association between IBQ-R score and prenatal cortisol level; $\beta_3(c)$ is the association between maternal lifetime trauma exposure and IBQ-R score, as a potentially nonlinear function of prenatal cortisol; and χ_i is a vector containing the additional covariates (maternal stress exposures during pregnancy, maternal age, race/ethnicity, and education, and infant sex). Figures were created to demonstrate estimates and corresponding confidence intervals for the association of maternal lifetime trauma exposure with each IBQ-R score as a function of prenatal cortisol levels. To test modification across all cortisol levels, global *p*-values for modification from the varying coefficient models were calculated. These global *p*-values tested the null hypothesis that $H_0: \beta_3(c) = 0$ (i.e., the null hypothesis being that the smooth varying coefficient term is horizontal).

All tests were based on two-sided significance tests conducted using R software (R Core Team, 2015). For all analyses, $p < .05$ was considered statistically significant.

RESULTS

Descriptive data

Table 1 details sample characteristics. Male and female infants did not differ on any of the negative affectivity scores. Black women endorsed higher levels of stress exposures during pregnancy [3.07 (2.09)] than White [1.61 (1.70)] and Hispanic [1.96 (1.98)] women, $F(3, 285) = 8.59, p < .001$, and greater lifetime trauma exposures [2.32 (2.79)] than White women [1.04 (1.30)], $F(3, 285) = 5.66, p = .001$. Black [1.44 (0.72)] and Hispanic [1.37 (0.66)] women demonstrated greater third-trimester hair cortisol levels than White women [0.91 (0.69)], $F(3, 190) = 6.71, p < .001$. None of the perinatal risk factors were associated with maternal lifetime trauma exposure, stress exposures during pregnancy, third-trimester hair cortisol, or the infant negative affectivity variables with the exception of a very modest association between maternal prenatal BMI and cortisol ($r = .15, p = .028$). Therefore, the perinatal risk variables were not considered further in analyses. Table 2 provides the Spearman bivariate correlation coefficients among the main variables.

Associations between maternal lifetime trauma exposure history and infant negative affectivity and modification by prenatal cortisol exposure

In the full sample ($N = 289$), main effects regression models, adjusted for infant sex and maternal age, race/ethnicity, education, and stress exposures during pregnancy, showed that increased maternal lifetime trauma exposure was associated with greater infant Distress to Limitations, $\beta = 0.078, p = .004$, and Sadness, $\beta = 0.061, p = .030$ (Table 3; Figure 1). The association between maternal trauma history and the infant Negative Affectivity Factor approached significance, $\beta = 0.035, p = .078$. Maternal trauma history was not associated with infant Falling Reactivity, $\beta = 0.028, p = .312$, or Fear, $\beta = 0.026, p = .403$.

For the subsample with third-trimester hair cortisol data ($N = 194$ dyads; 95 male infants), Figure 2 depicts the association between maternal lifetime trauma exposures and each infant negative affectivity variable as a smooth function of cortisol (log-transformed), as estimated from the varying coefficient model. Each model controlled for infant sex and maternal age, race/ethnicity, education, and stress exposures during pregnancy. In tests of modification across all third-trimester cortisol levels [i.e., testing $H_0: \beta_3(c) = 0$], the analyses provided evidence for overall effect modification of maternal lifetime trauma history by cortisol level for Falling Reactivity, $p = .026$. Specifically, infants exposed to lower cortisol levels (log cortisol < 0.22) exhibited a positive association between maternal lifetime trauma exposures and Falling Reactivity, whereas infants exposed to higher cortisol levels (log cortisol > 2.16) exhibited a negative association between these variables (Figure 2). Results also suggested that the association between maternal trauma exposure history and infant Distress to Limitations was strongest for infants exposed to higher levels of cortisol ($1.34 < \log \text{cortisol} < 2.60$; Figure 2), although the overall test for effect modification did not reach significance, $p = .142$. No significant interactions between maternal trauma exposure history and third-trimester cortisol were found for infant Fear, Sadness, or the Negative Affectivity Factor, p s $> .15$.

DISCUSSION

The goals of this study were to examine associations between maternal lifetime trauma exposure history and infant negative affectivity and to test the modifying effects of maternal prenatal HPA functioning on these associations. Because data have linked trauma exposure and child emotional/behavioral problems to both hyper- and hypoarousal of the HPA, analyses considered the modifying impact of a continuous measure of prenatal cortisol exposure on the association between maternal trauma history and infant negative affectivity. Analyses controlled for sociodemographic factors as well as maternal stress exposures during pregnancy. To date, the majority of studies examining links between maternal stress and infant outcomes have focused narrowly on exposures experienced during pregnancy and ignored potential lifetime cumulative exposure effects.

Three of the four infant negative affectivity scales were associated with maternal trauma history although the overall Negative Affectivity Factor was not. This pattern suggests that a nuanced approach to understanding links between maternal trauma history and infant negative affectivity is required and that discrete facets of this temperamental domain may be differentially influenced by perinatal risk factors. Specifically, greater maternal trauma exposure was associated with increased infant sadness and distress to frustration or confinement (i.e., Distress to Limitations scale scores), with some evidence that the latter association was stronger when third-trimester cortisol levels were elevated. An association between maternal trauma history and infant rate of recovery from arousal (i.e., Falling Reactivity scale scores) emerged only when prenatal cortisol exposure was considered: At higher levels of prenatal cortisol, greater maternal trauma exposure was associated with lower infant Falling Reactivity scale scores; at lower levels, greater maternal trauma exposure was associated with higher infant Falling Reactivity scale scores.

Associations of maternal trauma history with infant Distress to Limitations and Falling Reactivity scale scores are consistent with other studies (e.g., Bosquet Enlow et al., 2011; Lang et al., 2010). Contrary to our hypotheses, maternal trauma history was associated with infant Sadness but not Fear scale scores. Because studies testing associations between prenatal stress and infant sadness are essentially nonexistent, we do not know whether the association found here is specific to maternal trauma history or may extend to other forms of stress. Trauma may exert a unique impact on both maternal psychobiological functioning and caregiving behaviors (e.g., withdrawal, dissociation) that increase risk for infant sadness. More research is needed to replicate the findings and specify underlying mechanisms. There are several potential explanations for the lack of an association between maternal trauma and infant fear. The Fear scale mean was relatively low in this sample; associations may emerge when there is greater variability in fear behaviors. Also, expressions of fear undergo substantial changes during infancy, with considerable increases between 6 and 12 months (Gartstein & Rothbart, 2003); consequently, associations between maternal trauma history and infant fear may vary by infant age. Finally, the IBQ-R Fear scale combines expressions of different types of fear/anxiety (e.g., heightened startle, social fears; Gartstein & Rothbart, 2003); possibly, as fear becomes differentiated into more distinct phenotypes in later development, specific relations with maternal trauma history and prenatal cortisol exposure will emerge.

That Falling Reactivity only showed an association with maternal trauma history when prenatal cortisol exposure levels were considered is noteworthy. The Falling Reactivity scale has been conceptualized as a measure of infant stress regulatory capacities (Bosquet Enlow et al., 2011), and the HPA axis has an integral role in regulating the stress response. Maternal trauma history was associated with slower recovery from arousal only among infants exposed to higher prenatal cortisol levels. One interpretation of this finding is that elevated cortisol levels programmed the fetal HPA axis to be more stress sensitive. Notably, research suggests that trauma-exposed mothers may be at increased risk for engaging in insensitive caregiving behaviors (Liotti, 2004). The combination of a prenatally sensitized HPA axis and exposure to insensitive caregiving may compromise the infant's developing stress regulatory abilities, reflected in diminished arousal recovery. Interestingly, maternal trauma history was associated with faster arousal recovery among infants exposed to lower prenatal cortisol levels. Perhaps among these infants, relatively low prenatal cortisol exposure prepared the HPA axis to regulate stress responses more rapidly. These findings are consonant with theories that pre- and postnatal stress can be both detrimental and facilitative to fetal/infant development (DiPietro, 2012). The extent of prenatal cortisol exposure may be one factor influencing the direction of stress effects on infant outcomes. These interpretations are highly speculative and in need of further exploration.

This study has a number of strengths, including the prospective epidemiological design and the use of a relatively large, sociodemographically diverse, community sample, which broadens the generalizability of the findings. Care should be taken in applying the results to clinically at-risk populations (e.g., extreme temperament), for whom developmental processes may differ (Perez-Edgar, Schmidt, Henderson, Schulkin, & Fox, 2008). The study is unique in its comprehensive assessment of maternal trauma and stress exposures. This is one of the only studies to examine associations between maternal lifetime trauma exposure history and infant temperament; the majority of research has focused on stress during pregnancy, operationalized inconsistently across studies. The current findings suggest that maternal trauma exposures prior to conception may have intergenerational effects on infant temperament, particularly given that the analyses controlled for stress exposures during pregnancy. Thus, future studies may benefit from employing a lifetime approach when operationalizing maternal stress exposures. Using hair to assess prenatal cortisol exposure is unique in the infant temperament literature. Hair cortisol measures may be more useful than salivary or serum measures in quantifying the child's prenatal environment (D'Anna-Hernandez et al., 2011). This study is also unusual in its consideration of prenatal HPA axis functioning as a modifier of associations between maternal trauma exposure and infant temperament. Finally, this is one of the only studies to consider the potential impact of a range of prenatal cortisol levels on infant negative affectivity. Currently, the specific levels at which prenatal cortisol exposure impacts infant behavior are not known and likely vary across behavioral domains. More studies are needed to specify cortisol exposure levels that influence different domains of infant negative affectivity.

This study also has limitations. Maternal report was used to assess maternal trauma history, pregnancy stress exposures, and infant negative affectivity. Studies have demonstrated that self-reports of trauma history are relatively accurate, with inaccuracies appearing to be in the direction of underestimating exposures, which would lead to an underestimation of the

magnitude of associations among study variables (Hardt & Rutter, 2004; McHugo et al., 2005; Wolfe & Kimerling, 1997). Maternal report of infant temperament leverages the mother's ability to observe her infant's behavior over a range of contexts; however, maternal psychopathology or personality may influence reporting accuracy (Davis et al., 2007). The IBQ-R was designed to reduce the influence of such biases by inquiring about multiple examples of concrete infant behaviors for each domain rather than asking for abstract judgments (Gartstein & Rothbart, 2003). Furthermore, maternal ratings of infant negative affectivity on the IBQ-R correlate with laboratory observations (Gartstein & Marmion, 2008; Goldsmith & Campos, 1990; Parade & Leerkes, 2008), although some find discrepancies (Stifter, Willoughby, & Towe-Goodman, 2008). Future studies should consider independent assessments of infant negative affectivity to confirm that associations between maternal trauma history and infant temperament are not due to maternal reporting biases influenced by trauma history, associated psychopathology, or other factors.

This study did not consider pre- or postnatal maternal psychopathology or caregiving quality, which have been associated with infant emotional reactivity. Notably, data suggest that prenatal exposures exert effects on infant emotional functioning independent of postnatal factors (Davis, 2004; Davis et al., 2005, 2007). Additionally, studies have shown that trauma history is associated with hair cortisol levels regardless of the presence of psychopathology and that stress exposures have larger associations with hair cortisol than stress-related psychopathology, including among pregnant women (Hinkelmann et al., 2013; Schreier et al., 2015, 2016; Staufienbiel et al., 2013; Steudte et al., 2013; Van Voorhees, Dennis, Calhoun, & Beckham, 2014). Nevertheless, studies should consider whether the variables assessed here demonstrate associations with infant negative affectivity independently of pre- and postnatal maternal mental state and other relevant postnatal factors (Bergman, Sarkar, Glover, & O'Connor, 2008; Kaplan, Evans, & Monk, 2008).

Hair growth rates may have varied across participants, influencing the accuracy of the timing of the cortisol assessment. Notably, studies suggest small individual differences in hair growth rates, including between individuals from different racial/ethnic backgrounds (Loussouarn, 2001; Loussouarn, El Rawadi, & Genain, 2005). Because hair was collected postdelivery, delivery experiences may have affected cortisol levels. Also, the cortisol measurement may reflect different developmental periods for infants born preterm versus term. However, hair cortisol level was not associated with delivery method or gestational age, arguing against such influences. Moreover, few participants were born prematurely (92% born 37 weeks and 97% 36 weeks).

Analyses focused on third-trimester cortisol output to maximize available samples. Furthermore, evidence suggests that maternal cortisol levels during late pregnancy may be most influential on infant negative affectivity (Davis et al., 2007; Yehuda et al., 2005; Zijlmans et al., 2015). In the subsample with sufficient samples, cortisol levels were highly correlated across trimesters, suggesting that third-trimester levels may represent a reasonable proxy for relative cortisol levels across pregnancy. However, we cannot conclude that third-trimester levels are a representation of prenatal cortisol exposure levels during earlier trimesters. Future studies should consider whether maternal HPA functioning during each trimester has differential or additive effects (Davis et al., 2005). Notably, fetal cortisol

exposure changes over pregnancy due to variations in cortisol output and placental permeability that support the needs of the developing fetus (Zijlmans et al., 2015). Thus, understanding prenatal cortisol effects may require consideration of the interaction between level and timing of exposure.

Additional variables may be worthy of consideration. Although studies demonstrate prenatal stress effects on child emotional development independent of genetic influences (Baibazarova et al., 2013), genetic factors may contribute to associations between prenatal cortisol exposure and infant temperament. Male and female infants did not differ on the negative affectivity variable mean scores; however, sex should be explored as a potential moderator of associations among the study constructs. Male and female fetuses experience different patterns of cortisol exposure and differential rates of neurobiological development; further, HPA functioning predicts different emotional behaviors in male and female children (DiPietro, Costigan, Kivlighan, Chen, & Laudenslager, 2011; Doyle et al., 2015; Perez-Edgar et al., 2008; Smider et al., 2002; Tout et al., 1998). Race/ethnicity may also modify the observed associations. Consistent with prior work (Cohen et al., 2006; Hatch & Dohrenwend, 2007; Roberts, Gilman, Breslau, Breslau, & Koenen, 2011; Schreier et al., 2015, 2016), minority women, particularly Black women, experienced greater stress and trauma exposures and greater prenatal hair cortisol levels in this sample. Studies suggest that some racial/ethnic minority groups may be more vulnerable to stress-elicited health effects due to increased exposure to negative life events, reduced access to buffering resources, ongoing discrimination stress, and differences in physiological stress responses (Brunst et al., 2014; Cohen et al., 2006; Hatch & Dohrenwend, 2007; Roberts et al., 2011; Suglia et al., 2010; Thoits, 2010; Tse et al., 2012). These variations may differentially influence prenatal HPA reactivity and stress exposure effects on infant temperament (Brunst et al., 2014; Schreier et al., 2015, 2016). Finally, the current analyses focused on infant negative affectivity domains given evidence of their critical influence on numerous developmental outcomes and associations among these traits and maternal stress and HPA functioning. Examination of other temperamental factors (Surgency/Extraversion, Orienting/Regulation) was beyond the scope of this study but may be worthwhile.

To date, research has focused on the potential mediating role of prenatal HPA functioning on infant outcomes, with limited success (Bergman et al., 2010). The current study suggests that modifying effects may be possible and that both elevated and reduced levels of prenatal cortisol exposure may have impact. Going forward, models that test mediating and modifying effects of varying levels of prenatal cortisol exposure may provide the most comprehensive examination of prenatal HPA effects on infant temperament in the context of maternal stress exposure history. Potential mechanisms of action of maternal trauma/stress need to be explored further, including dysregulation of maternal–fetal HPA and ANS reactivity, glucocorticoid sensitivity, and/or genetic expression in stress response systems as well as bidirectional influences between fetal behaviors and maternal physiological reactivity and psychological state (Bergman et al., 2010; DiPietro, 2012; Enlow et al., 2009).

In sum, our findings suggest associations among maternal lifetime trauma exposure history, prenatal HPA functioning, and several domains of infant negative affectivity. These findings have developmental and clinical implications, as infants who express frequent,

intense negative emotions may be more challenging to parent. For mothers with trauma histories and consequent difficulties with stress regulation, such infant behaviors may provoke angry or fearful caregiving behaviors (Liotti, 2004; Stovall-McClough & Cloitre, 2006). Exposure to such caregiving behaviors may be particularly harmful for infants high on negative affectivity given their increased reactivity to their environment, thus setting up a cascade of adverse parent–child interactions that lead to poor developmental trajectories and increased risk for psychopathology. Traumatized mothers and their children may benefit from pre- and postnatal interventions that influence these processes. Studies are needed to determine the exact nature of treatments that would be most effective in maximizing positive child developmental outcomes in these populations.

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Figure 1.

Associations between maternal lifetime trauma exposure history and infant negative affectivity ($N = 289$). Maternal lifetime trauma exposure scores assessed via continuous ratings from the Life Stressor Checklist-Revised (LSC-R). Infant negative affectivity scores assessed via the Infant Behavior Questionnaire-Revised (IBQ-R). Effect estimates represent the estimated mean difference in the IBQ-R score per one unit change in the LSC-R score. Analyses adjusted for maternal stress exposures during pregnancy, assessed via the Crisis in Family Systems-Revised (CRISYS-R) survey, and for infant sex and maternal age, education, and race/ethnicity. * $p < .05$. ** $p < .01$.

Figure 2.

Associations between maternal lifetime trauma exposure history and infant negative affectivity: Effect modification by prenatal cortisol exposure ($N = 194$). Maternal lifetime trauma exposure scores assessed via continuous ratings from the Life Stressor Checklist-Revised (LSC-R). Infant negative affectivity scores assessed via the Infant Behavior Questionnaire-Revised (IBQ-R). Prenatal cortisol (continuous ratings) assessed from maternal hair representing the third trimester of pregnancy. Panels depict the association between maternal lifetime trauma exposure and each infant negative affectivity variable as a smooth function of prenatal cortisol (log transformed) estimated from the varying coefficient model (solid line). Dotted bands represent the 95% pointwise confidence bounds for the change in a given infant negative affectivity mean score associated with a one-unit increase in maternal trauma exposure at each value of prenatal cortisol exposure. Areas in which both dotted bands (upper and lower) fall above or below the zero line indicate intervals of prenatal cortisol values with a significant association between maternal lifetime trauma exposure and the specified IBQ-R scale. Analyses adjusted for maternal stress during pregnancy, assessed via the Crisis in Family Systems-Revised (CRISYS-R) survey, and for infant sex and maternal age, education, and race/ethnicity. Global p -values for modification from the varying coefficient models were calculated to test modification across all cortisol levels (i.e., the null hypothesis being that the smooth varying coefficient term is horizontal). The overall effect modification of maternal lifetime trauma history by prenatal cortisol level was significant for Falling Reactivity. * $p < .05$ for test of effect modification.

TABLE 1Sample Characteristics and Distributions of Main Study Variables (*N* = 289)

	<i>n</i> (%)	<i>M</i>	<i>SD</i>
Maternal education: High school diploma	89 (31)		
Maternal race/ethnicity			
White	98 (34)		
Black/Haitian	73 (25)		
Hispanic	98 (34)		
Other ^a	20 (7)		
Maternal age (years)		31.21	5.51
Maternal lifetime trauma exposures: LSC-R ^b		1.57	2.08
Infant negative affectivity variables (IBQ-R) ^c			
Negative Affectivity Factor		3.01	0.68
Distress to Limitations Scale		3.53	0.89
Falling Reactivity Scale		5.22	0.90
Fear Scale		2.45	1.10
Sadness Scale		3.26	0.94
Prenatal hair cortisol, log transformed ^d		1.23	0.71
Prenatal stress exposures: CRISYS-R: NDS ^e		2.15	1.99
Maternal prenatal body mass index (BMI), kg/m ²		28.08	5.87
Maternal prenatal cigarette smoking	47 (16)		
Delivery method ^f			
Vaginal	203 (70)		
Cesarean section	74 (26)		
Infant birthweight (g)		3345	513
Infant gestational age (weeks)		39.12	1.68

Note.

^aThe majority of mothers categorized as “other” race/ethnicity self-identified as Asian or multiracial.^bMaternal lifetime trauma exposures assessed as number of endorsed events meeting DSM-V criteria via the Life Stressor Checklist-Revised (LSC-R).^cInfant negative affectivity scores derived from the Infant Behavior Questionnaire-Revised (IBQ-R).^dThird-trimester hair cortisol data available for *N* = 194 of the total sample.^ePrenatal stress exposures assessed via the Negative Life Events Domain Score (NDS) from the Crisis in Family Systems-Revised (CRISYS-R) survey.^fData missing for 12 (4%) participants.

TABLE 2

Spearman Correlation Coefficients Among Main Study Variables ($N = 289$)

Variable	1	2	3	4	5	6	7
1. Maternal lifetime trauma exposures (LSC-R ^a)	–						
2. Prenatal hair cortisol, log transformed ^b	.07	–					
3. Prenatal stress exposures (CRISYS-R; NDS ^c)	.34***	.07	–				
4. Negative Affectivity Factor (IBQ-R)	.10	.09	.14*	–			
5. Distress to Limitations Scale (IBQ-R)	.12*	.09	.10 [†]	.81***	–		
6. Falling Reactivity Scale (IBQ-R)	–.07	–.14*	–.09	–.61***	–.44***	–	
7. Fear Scale (IBQ-R)	.02	.02	.04	.60***	.31***	–.10 [†]	–
8. Sadness Scale (IBQ-R)	.06	.01	.13*	.75***	.60***	–.30***	.25***

Note. IBQ-R = Infant Behavior Questionnaire-Revised.

^aMaternal lifetime trauma exposures assessed as number of endorsed events meeting DSM-V criteria via the Life Stressor Checklist-Revised (LSC-R).

^bThird-trimester hair cortisol data available for $N = 194$ of the total sample.

^cPrenatal stress exposures assessed via the Negative Life Events Domain Score (NDS) from the Crisis in Family Systems-Revised (CRISYS-R) survey.

[†] $p < .10$;

* $p < .05$;

*** $p < .001$.

TABLE 3

Results of Linear Regression Models Testing Associations Between Maternal Lifetime Trauma Exposures and Infant Negative Affectivity Temperament Domains

<i>Variable</i>	<i>Beta estimate</i>	<i>CI lower</i>	<i>CI upper</i>	<i>p-Value</i>
Negative Affectivity Factor				
Maternal lifetime trauma exposures: LSC-R ^a	0.035	−0.004	0.074	.078
Prenatal stress exposures: CRISYS-R: NDS ^b	0.023	−0.019	0.064	.289
Maternal race/ethnicity: Black/Haitian ^c	−0.039	−0.256	0.177	.722
Maternal race/ethnicity: Hispanic ^c	0.259	0.038	0.480	.022
Maternal race/ethnicity: Other ^c	0.312	0	0.624	.051
Maternal education: >High school diploma ^d	−0.311	−0.514	−0.108	.003
Maternal age	−0.002	−0.016	0.013	.830
Infant sex: Female ^e	−0.001	−0.153	0.151	.989
Distress to Limitations Scale				
Maternal lifetime trauma exposures: LSC-R ^a	0.078	0.026	0.131	.004
Prenatal stress exposures: CRISYS-R: NDS ^b	−0.001	−0.057	0.055	.976
Maternal race/ethnicity: Black/Haitian ^c	−0.040	−0.331	0.251	.787
Maternal race/ethnicity: Hispanic ^c	0.335	0.038	0.631	.028
Maternal race/ethnicity: Other ^c	0.376	−0.043	0.795	.080
Maternal education: >High school diploma ^d	−0.170	−0.443	0.103	.223
Maternal age	−0.003	−0.023	0.016	.750
Infant sex: Female ^e	−0.071	−0.276	0.134	.496
Falling Reactivity Scale				
Maternal lifetime trauma exposures: LSC-R ^a	−0.028	−0.081	0.026	.312
Prenatal stress exposures: CRISYS-R: NDS ^b	−0.013	−0.071	0.044	.652
Maternal race/ethnicity: Black/Haitian ^c	−0.227	−0.525	0.070	.135
Maternal race/ethnicity: Hispanic ^c	−0.209	−0.512	0.095	.179
Maternal race/ethnicity: Other ^c	−0.428	−0.857	0	.051
Maternal education: >High school diploma ^d	0.205	−0.075	0.484	.152
Maternal age	0.006	−0.014	0.026	.555
Infant sex: Female ^e	0.098	−0.111	0.308	.358
Fear Scale				
Maternal lifetime trauma exposures: LSC-R ^a	−0.026	−0.088	0.035	.403
Prenatal stress exposures: CRISYS-R: NDS ^b	0.019	−0.047	0.084	.580
Maternal race/ethnicity: Black/Haitian ^c	0.153	−0.187	0.493	.378
Maternal race/ethnicity: Hispanic ^c	0.539	0.192	0.885	.003
Maternal race/ethnicity: Other ^c	0.271	−0.218	0.761	.278

<i>Variable</i>	<i>Beta estimate</i>	<i>CI lower</i>	<i>CI upper</i>	<i>p-Value</i>
Maternal education: >High school diploma ^d	−0.613	−0.931	−0.294	<.001
Maternal age	0	−0.023	0.023	.983
Infant sex: Female ^e	0.134	−0.106	0.373	.274
Sadness Scale				
Maternal lifetime trauma exposures: LSC-R ^a	0.061	0.006	0.116	.030
Prenatal stress exposures: CRISYS-R: NDS ^b	0.060	0.001	0.119	.048
Maternal race/ethnicity: Black/Haitian ^c	−0.498	−0.802	−0.193	.002
Maternal race/ethnicity: Hispanic ^c	−0.047	−0.358	0.264	.769
Maternal race/ethnicity: Other ^c	0.173	−0.266	0.613	.440
Maternal education: >High school diploma ^d	−0.258	−0.544	0.028	.079
Maternal age	0.003	−0.018	0.024	.769
Infant sex: Female ^e	0.031	−0.183	0.246	.774

Note.

^aMaternal lifetime trauma exposures assessed as number of endorsed events meeting DSM-V criteria via the Life Stressor Checklist-Revised (LSC-R).

^bPrenatal stress exposures assessed via the Negative Life Events Domain Score (NDS) from the Crisis in Family Systems-Revised (CRISYS-R) survey.

^cReference group is non-Hispanic White maternal race/ethnicity.

^dReference group is maternal education = high school diploma.

^eReference group is male infants.