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Infant sleep and negative reactivity: The role of maternal adversity and perinatal sleep

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Abstract

Sleep during infancy contributes to the development and maintenance of infant regulatory functioning and may be an early risk marker for more difficult temperamental traits like negative reactivity. Further, maternal adverse childhood experiences (ACEs) may predispose individuals to greater sleep disturbances in adulthood and have been linked with sleep disturbances in both mothers and infants. Thus, examining maternal history of ACEs and maternal sleep difficulties during pregnancy and postpartum may provide insight into underlying risk factors affecting infant sleep difficulties and early temperament development. Fifty-nine mothers from a diverse, community sample (44% white) completed questionnaires on ACEs, maternal sleep, infant sleep, and infant temperament at 30-weeks gestation, 6-weeks postpartum, and 16-weeks postpartum. Results indicated that maternal ACES and sleep problems during pregnancy have long term implications for infant negative reactivity at 16-weeks, with significant indirect effects through maternal and infant sleep problems at 6-weeks. Addressing psychosocial functioning and prenatal sleep during pregnancy, particularly among women with high ACEs, may be a target of intervention to improve maternal and infant sleep health during the postpartum, and reduce the risk for difficult infant temperament.

Keywords

Sleep; Pregnancy; Infancy; Temperament; Adversity

1. Introduction

Sleep during infancy contributes to the development and maintenance of infant regulatory functioning and may be an early risk marker for later behavioral, emotional, and physical

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health challenges (Field, 2017; Mindell et al., 2017), including more difficult temperamental traits like negative reactivity (Sidor et al., 2017). There is evidence that sleep-wake patterns and infant temperament may reflect similar aspects of infant biological maturity and organization, especially during early infancy (Ednick et al., 2009), and that there are likely transactional associations between infant sleep and intrinsic infant factors like temperament (Camerota et al., 2019), suggesting that sleep-wake patterns may shape infant temperament and behavior over time (Morales-Munoz et al., 2020; Spruyt et al., 2008). Further, research suggests that maternal and infant sleep difficulties are highly positively correlated (Sadeh et al., 2010), and that maternal adverse childhood experiences (ACEs) may predispose individuals to greater sleep disturbances in adulthood and have been linked with sleep disturbances in both mothers and infants (Kajeepeta et al., 2015; Hairston et al., 2011). Thus, examining maternal history of ACEs and maternal sleep difficulties during pregnancy and postpartum may provide insight into underlying risk factors affecting infant sleep difficulties and early temperament development.

1.1 Maternal prenatal sleep problems

It is common for birthing persons (referred to as women and mothers in this study based on our sample demographics) to experience a wide range of changes and stressors during pregnancy, including changes in sleep quality (Schetter & Tanner, 2012; Pien & Schwab, 2004). According to the National Sleep Foundation, more than 79% of women reported that their sleep had changed during pregnancy and approximately 28-38% of women report experiencing inadequate sleep and a decline in sleep quality during pregnancy (Okun et al., 2013). Sleep problems such as incremental nocturnal awakenings, difficulties in falling asleep, too-early awakenings, sleep restriction, disordered breathing, and parasomnias are commonly reported during pregnancy (Mindell et al., 2015), and tend to worsen into the third trimester (Hayase et al., 2014). Although changes to sleep are to be expected during pregnancy given the extensive biological and psychophysiological changes associated with gestation (Palagini et al., 2014; Yanıkkerem et al., 2006), chronic sleep problems during the prenatal period are associated with poor maternal and infant health outcomes, including maternal and infant sleep difficulties (Sadeh et al., 2010).

Several factors may increase the risk for sleep disturbances during pregnancy, and there is increasing evidence that early life exposure to adversity like ACEs may play a role in chronic disturbances in sleep. ACEs are often chronic and repeated stressors that occur during early childhood, including experiences of abuse, neglect, and household dysfunction (Felitti et al., 1998) that have been shown to be associated with numerous health outcomes across the life course, including sleep disorders (Chapman et al., 2011; Kajeepeta et al., 2015). Several studies have found that adults with a history of ACEs are more likely to report sleep problems compared to those without a history of ACEs, and recent studies have shown similar findings in perinatal populations (Chapman et al., 2011; Kajeepeta et al., 2015; Miller-Graff & Cheng, 2017). For example, Paulson and Miller-Graff (2019) found that ACE scores were associated with sleep problems during pregnancy, and in the context of ACEs and intimate partner violence, women with sleep impairments had worsening trajectories of posttraumatic stress symptoms across the perinatal period. Additionally, exposure to ACEs is associated with increased risk for psychopathology, including prenatal

and postpartum depressive symptoms (Racine et al., 2021), as well as increased risk for adverse health behaviors (e.g., substance use, smoking, obesity), which can negatively impact sleep (Anda et al., 2006).

It has been hypothesized that experiencing a heightened stress response for a prolonged period of time, as often occurs with ACEs, initiates allostasis, or physiological "wear and tear" on the stress system as it adapts to acute stress, altering neuroendocrine, immune, and cardiovascular functioning, and placing individuals at greater risk for psychological and physical health problems (Danese et al., 2009; Danese & McEwen, 2012; Jaffee & Christian, 2014; Nurius et al., 2015). Previous studies have suggested a bidirectional relationship between sleep and the stress response system, and chronic sleep problems have been characterized as both a consequence of stress as well as a stressor in itself (Levenson et al., 2015; McEwen, 2006; Palagini et al., 2014). In line with the allostatic load hypothesis (McEwen & Stellar, 1993), disrupted sleep, like other stressors, may activate allostasis, and in turn, the allostatic reactions may lead to neuroendocrine alterations that contribute to an ongoing cycle of chronic sleep problems (McEwen, 2006; Palagini et al., 2014). The cumulative effects of chronic sleep problems have been associated with increased risk of hypertension, diabetes, obesity, depression, heart attack, and stroke (McEwen, 2006; Spiegel, 2005), and have been associated with similar health complications that onset during pregnancy, including prenatal depression, gestational diabetes, and preeclampsia (Chang et al., 2010; Okun et al., 2009). Sleep problems during pregnancy have also been shown to have lasting impacts for birth outcomes and infant health, with the most frequently reported complications being extended duration of labor, type of delivery, intrauterine growth restriction, and preterm birth (Palagini et al., 2014).

1.2 Prenatal exposure and infant temperament

Importantly, alterations in the maternal stress response system during pregnancy have been hypothesized as a mechanism for the development of adverse infant outcomes, including adverse birth outcomes (e.g., length of labor, type of delivery, preterm birth, and intrauterine fetal growth) (Bublitz et al., 2018; Palagini et al., 2014) and long-term neurodevelopmental difficulties among offspring (Bergman et al., 2007; Weinstock, 2005). According to prenatal programming or the Developmental Origins of Health and Disease (DOHaD) hypothesis (Barker, 1998; Gillman, 2005), *in utero* exposure to adversity, including exposure to maternal stress hormones and sleep disturbances, may result in permanent biological and neurodevelopmental changes in offspring with implications for long-term development, behavior, and disease risk (Talge et al., 2007; Weinstock, 2005). For instance, research has shown that prenatal exposure to maternal stress and symptoms of depression and anxiety is associated with delayed fetal maturation, impaired cognitive development, increased fearful behavior in the infant, diminished gray matter volume in children, altered developmental trajectory of the fetal HPA axis, and infant stress regulation and sleep (Bergman et al., 2007; Davis et al., 2004, 2007; Kim et al., 2020; Monk et al., 2019; O'Connor et al., 2007; Weinstock, 2005).

Notably, biological and neurodevelopmental alterations resulting from prenatal exposures may shape the infant's behavioral and physiological responses to stressors in the postnatal

environment, thereby playing a role in shaping infant temperament (Werner et al., 2007). Infant temperament is broadly conceptualized as relatively stable, individual variation in behavioral dispositions rooted in distinct neurophysiological differences in reactivity to stress (Fox, 2004; Rothbart & Ahadi, 1994; Werner et al., 2007), thought to be influenced by both a child's genetics as well as the environment (Goldsmith et al., 1997; Shiner et al., 2012). Although there is not a singular model or definition of temperament, it is commonly believed that there is a distinct category of temperament known as "difficult temperament" (Goldsmith et al., 1987).

In Rothbart's model of temperament, difficult temperament is characterized by negative reactivity or emotionality, which refers to an infant's negative mood, low soothability, and irritability (Rothbart & Putnam, 2002; Putnam, et al., 2008). Negative reactivity represents a child's tendency to react to environmental stimuli with higher degrees of negative emotion, such as fear, sadness, anger, or irritability (Gartstein & Rothbart, 2003), which can be observed early in infancy at two to three months of age, increases throughout infancy (Razza et al., 2012), and has been associated with an increased risk for internalizing and externalizing symptoms during childhood (Janson & Mathiesen, 2008; Nielsen & Olino, 2019) as well as difficulties with sleep (Sorondo & Reeb-Sutherland, 2015; Spruyt et al. 2008). Rothbart's model of temperament also includes factors of surgency and orienting (Rothbart & Putnam, 2002), which have also been associated with infant sleep (Jian & Teti, 2016; Scher et al., 1998). More specifically, surgency has been associated with increased sleep duration and orienting has been associated with fewer night awakenings and higher quality sleep (Jian & Teti, 2016; Scher et al., 1998). However, individual differences in surgency and orienting emerge somewhat later in infancy compared to negative emotionality (approximately 6 to 8 months of age (Rothbart & Putnam, 2002), making these factors more difficult to examine with younger infants (Razza et al., 2012).

1.3 Negative reactivity and infant sleep problems

Broadly, research examining temperament and sleep during infancy has shown that temperament is associated with daytime, nighttime, and total sleep duration (Kaley et al., 2012; Jian & Teti, 2016; Scher et al., 1998; Sorondo & Reeb-Sutherland, 2015; Spruyt et al., 2008). Negative reactivity has been specifically associated with infant and childhood sleep difficulties, including frequent nighttime awakenings and disorganized sleep patterns (Kaley et al., 2012; Sorondo & Reeb-Sutherland, 2015; Spruyt et al. 2008). It is posited that difficult temperamental traits can contribute to sleep problems relative to an infant's inability to self-soothe, interfering with the ability to fall or stay asleep (Burnham et al., 2002; De Marcas et al., 2015). However, research also suggests the relationship between difficult temperament and infant sleep may be bidirectional (Spruyt et al., 2008), as there is evidence that infants with irregular sleep patterns and shorter sleep durations are more likely to display difficult temperaments (Sadeh et al., 1994; Spruyt et al., 2008). That is, infants who get insufficient sleep may be prone to negative mood, irritability, and difficulty soothing, and as such, chronic sleep problems can foster or exacerbate difficult temperamental traits (Spruyt et al., 2008).

1.4 Negative reactivity and maternal sleep problems

Additionally, maternal sleep difficulties and related stress can also exacerbate both infant sleep problems and difficult temperament alike (Tikotzky & Sadeh, 2009; Tikotzky et al., 2015). Specifically, maternal sleep difficulties in the early postnatal period have been found to predict infant sleep difficulties later in infancy (Tikotzky et al., 2015), as well as behaviors comprising difficult temperaments (Tikotzky et al., 2010). Moreover, maternal stress has been found to have indirect effects on infant sleep and difficult temperament by negatively influencing mothers' sleeping behaviors and the way they interact with their infants (McQuillan et al., 2019). Notably, the majority of research on maternal sleep behaviors and their influence on infant sleep and development has primarily focused on the postnatal period, suggesting a need to also examine the influence of prenatal maternal sleep on postnatal infant development (Newland et al., 2016). Research that has focused on the prenatal period has found that maternal psychological stress, depression, and negative life events, which are often associated with significant sleep disturbance, have been linked to infant fussiness and crying at 3 and 6 months postpartum (Davis et al., 2004; Wurmser et al., 2006) and irregular sleep behaviors (Armitage et al., 2009; Dias & Figueiredo, 2020; O'Connor et al., 2007; van den Huevel et al., 2021), and more recent research has found significant associations between maternal prenatal sleep and infant sleep (Burdayron et al., 2021a; Chen et al., 2020). Even though prenatal risk factors such as psychological stress and negative life events can negatively influence maternal sleep behaviors during pregnancy (Chang et al., 2010), prenatal maternal sleep itself has not been frequently examined (Burdayron et al., 2021b; Chen et al., 2020).

Given the evidence that postnatal sleep-wake patterns may shape infant temperament and behavior over time (Morales-Munoz et al., 2020) and the research highlighting the positive associations between maternal and infant sleep difficulties (Sharkey et al., 2016; Tikotzky & Sadeh, 2009), there is a need to further examine the relationship between infant sleep problems and infant negative reactivity in the context of prenatal and postnatal maternal sleep problems. The literature is lacking in studies that examine infant sleep problems during early infancy as a risk factor for the emergence of difficult temperament, as well as studies that consider the influence of maternal early life adversity and prenatal and postnatal sleep problems on infant sleep problems and difficult temperament.

1.5 Current Study

The current study examines maternal history of ACEs and maternal sleep difficulties during pregnancy and postpartum as underlying risk factors for infant sleep difficulties and early temperament development, specifically, negative reactivity. We hypothesized that greater exposure to ACEs would be associated with prenatal and postnatal maternal sleep difficulties, and in turn, greater infant sleep problems and negative reactivity.

2. Methods

2.1 Participants and Study Procedures

The study examined a diverse community sample of 59 pregnant women and their infants who had complete survey data on prenatal and postpartum demographic variables including

education and infant sex. Participants were recruited from prenatal clinics serving racially diverse and low-income populations (n= 56) and a community baby shower serving low-income families (n=3). The same inclusion criteria (1. Over 18 years old and 2. English-speaking) were used in both recruitment settings. Mothers were assessed at three time points: 30-weeks gestation, 6-weeks postpartum, and 16-weeks postpartum. The study received approval from the university Institutional Review Board, and all participants provided informed consent and were compensated $10 for each survey.

Participants were 25.8 years old on average and almost half of the sample reported having a high school education or less (54.2%). A majority of the sample reported being married or cohabitating (63.8%) and earning an annual income of $20,000 or less (65.5%). The racial breakdown of the sample included 44.1% white, 20.3% Black or African American, 15.3% Native American or Alaskan Native, 1.7% Asian, 15.3% Biracial or Multiracial and 3.4% Other. Almost a third (27.1%) of mothers reported this pregnancy as their first. Complete demographic data for the entire sample are provided in Table 1.

2.2 Attrition

Approximately 33% of the total sample (n = 20) was lost to attrition at 6-weeks postpartum (retained at 6-weeks, n = 39), and an additional 8% of the sample was lost to attrition at 16-weeks postpartum (retained at 16-weeks, n = 35). This rate of attrition is consistent with other research studying similar populations of mothers in the early postpartum (e.g., Cicchetti et al., 2006; McDonnell & Valentino, 2016). Participants missing at 6-weeks postpartum did not differ from retained participants on any demographic variables, including types of ACEs, but reported statistically significantly more sleep problems during pregnancy on average (M = 13.5 v. M = 9.4), $t(53) = 3.39$, p = .002. Participants missing at 16-weeks postpartum did not differ from retained participants on any baseline variables. These analyses indicate that women with more sleep problems during pregnancy were less likely to remain in the study, and thus, potential bias in the model estimates associated with missingness could result in underestimates of the effects.

2.3 Measures

Demographics.—Participants completed a demographic questionnaire and provided information about age, race, and education level.

Maternal Adverse Childhood Experiences.—During pregnancy, participants completed the Adverse Childhood Experiences Short-Form (ACES-SF; Felitti et al., 1998), an instrument that retrospectively assesses for traumatic childhood events prior to the age of 18. The scale assesses 10 domains of ACEs including emotional, sexual, and physical abuse, neglect, and household dysfunction. Participants provided a yes/no (1 = yes, 0 = no) response to adversity, summed for a total score (possible range 0-10), with higher scores indicating greater exposure to adverse events.

Maternal Sleep Problems.—During pregnancy and at 6-weeks postpartum, participants completed the Women's Health Initiative Insomnia Rating Scale (WHIIRS), a brief, 5-item scale that evaluates symptoms of insomnia (Levine et al., 2003). Participants self-reported

their sleep patterns within the last month using a 5-point Likert scale (0=problem has not been experienced in the past 4 weeks; 4=problem occurs at least 5 times a week). Items were totaled (possible range 1-20), with higher scores indicating more frequency of sleep difficulties. The WHIIRS has shown good internal consistency and construct validity (Levine et al., 2003). Cronbach's coefficient alpha showed acceptable internal reliability during pregnancy (α = .81), at 6-weeks (α =.72) and 16-weeks (α =.83).

Infant Sleep Problems.—At 6- and 16-weeks postpartum, participants completed the Infant Sleep Questionnaire (ISQ; Morrell, 1999), 10-item scale used to assess infant's sleeping habits. Mothers were asked to report on their infant's sleep behaviors including frequency of waking up at night and ability to settle and soothe. Items were totaled (possible range 0-38), with higher scores indicating more infant sleep difficulties. Cronbach's coefficient alpha showed acceptable internal reliability at 6-weeks (α =.75) and 16-weeks (α =.72).

Infant Temperament.—Participants completed the Infant Behavior Questionnaire-Revised-Short Form (IBQ-R), a 91-item scale to assess their infant's behavior and temperament at 16-weeks postpartum. (Gartstein & Rothbart, 2003). The IBQ-R is normed to assess infant temperament as early as 4 months of age, and includes 14 subscales: approach, vocal reactivity, high intensity pleasure, smiling and laughter, activity level, perceptual sensitivity, sadness, distress to limitations, fear, falling reactivity, low intensity pleasure, cuddliness, duration of orienting, and soothability. Infant negative reactivity was scored according to IBQ-R instructions using the mean of the sadness, distress to limitations, fear, and falling reactivity (reversed) subscales. The IBQ-R has shown good reliability and validity (Gartstein & Rothbart, 2003). Cronbach's coefficient alpha showed acceptable internal reliability at 16-weeks (α =.71).

Maternal Depressive Symptoms.—During pregnancy and at 6-weeks postpartum, participants completed the Edinburgh Postnatal Depression Scale (EPDS), a brief, 10-item scale that evaluates symptoms of depression on a 4-point scale (Cox et al., 1987). Items were totaled (possible range 0-30), with higher scores indicating higher depressive symptoms. Prenatal scores greater or equal to 15, and postpartum scores greater or equal to 13 were used as accepted cut-off scores indicating clinically significant symptomology (Matthey et al., 2006). Cronbach's coefficient alpha showed acceptable internal reliability during pregnancy (α = .91), at 6-weeks (α =.86) and 16-weeks (α =.94). It should be noted that there is substantial overlap between depressive symptoms and sleep problems (Feng et al., 2019), and as such, depressive symptoms were examined as a covariate.

2.3 Data Analytic Plan

Descriptive statistics and bivariate associations were calculated for all variables. A path model using full information maximum likelihood (FIML) estimation in Mplus 8.0 (Muthén & Muthén, 1998-2017) was used to examine the hypothesized model (Figure 1). The model included maternal race, level of education, infant sex (0 = female; 1 = male), and infant sleep problems at 16-weeks as covariates predicting infant negative reactivity at 16-weeks. Prenatal, 6-week, and 16-week maternal depressive symptoms were examined as predictors

of maternal sleep difficulties at 6- and 16-weeks postpartum, infant sleep problems at 6-weeks, and infant reactivity at 16-weeks. Missing data was handled in Mplus using Full Information Maximum Likelihood (FIML) estimation with bootstrapping procedures to correct for standard error bias associated with missing data and to derive confidence intervals for the mediating effect (Enders, 2010; MacKinnon et al., 2004).

3. Results

3.1 Descriptive statistics and correlations

Descriptive statistics on all study variables are presented in Table 1. For the current study, participant race was re-coded into a dichotomous variable for analyses (0 = Black, Native American/Alaska Native, Hispanic, Asian, Other (46%); 1 = white (44%). The decision to collapse all individuals identifying as persons of color into one category was made due to inadequate representation in each of the racial groups, thus inhibiting more culturally appropriate and meaningful analyses. Because links between ACEs and psychosocial functioning tend to follow a graded dose-response relationship and to remain consistent with previous literature (Felitti et al., 1998), participants' scores were coded into categories according to low= 0-1 ACEs, moderate= 2-3 ACEs, high= 4+ ACEs. On average, participants reported experiencing 3 ACEs (M = 3.02, SD = 2.62) prior to adulthood, and total scores ranged from 0 to 9. Examination by type of ACEs showed that 46% of participants reported experiencing at least one instance of abuse, 40% reported experiencing at least one instance of neglect, and 78% reported experiencing at least one instance of household dysfunction. Results from Analysis of Variance (ANOVA) examining mean differences in study variables according to ACEs categories indicated that participants reporting High ACEs (4+) had greater sleep difficulties at 6- and 16-weeks postpartum, reported more infant sleep problems at 6-weeks postpartum, and reported more depressive symptoms during pregnancy and at 6- and 16-weeks postpartum (Table 2). Further, when examined by ACEs type (i.e., abuse, neglect, household dysfunction), ANOVA results (not reported) showed mothers with specific histories of abuse, and less consistently neglect, were more likely to report maternal and infant sleep difficulties and depressive symptoms, relative to participants without such exposure. No statistically significant differences across study variables were found between those with and without household dysfunction ACEs.

Mean scores over time suggested that maternal sleep disturbances and depressive symptoms were slightly higher for the majority of study participants at 30-weeks gestation, but that sleep problems and depressive symptoms declined during the postpartum for participants with Low and Moderate ACEs, whereas participants with High ACEs maintained elevated symptoms over time. Overall, the study sample reported low to moderate symptoms of depression on average. Approximately 10.9% of the sample reported clinically significant symptoms of depression during pregnancy (scores ≥ 15), 14.6% reported clinically significant symptoms of depression at 6-weeks postpartum (scores ≥ 13), and 22.2%. reported clinically significant symptoms of depression at 16-weeks postpartum (scores ≥ 13). Participants with High ACEs accounted for two-thirds of those with clinically elevated prenatal symptoms, and 100% of clinically elevated symptoms at 6- and 16-weeks postpartum.

Simple correlations among the variables appear in Table 3. Sleep difficulties were positively correlated over time for both mothers and infants, and there were significant positive correlations between infant sleep difficulties at 6-weeks and maternal sleep difficulties at each measurement point. Infant sleep problems at 6-weeks were positively correlated with infant negative reactivity at 16-weeks. Infant orienting had a small, negative correlation with infant sleep at 6-weeks. Prenatal and 6-week postpartum depressive symptoms were highly correlated ($r = .71$), and both similarly correlated with maternal and infant sleep variables. Total scores for abuse- and neglect-type ACEs were positively associated with both maternal and infant sleep difficulties and depressive symptoms at 6-weeks, whereas abuse-type ACEs were also associated with maternal sleep and depressive symptoms during pregnancy. Total score for household dysfunction only had small positive associations with maternal postpartum sleep problems (see Table 3). Due to small cell sizes for types of ACEs, total ACE scores were used in subsequent analyses.

3.2 Path analysis

Path analysis using Mplus 8.0 (Muthén & Muthén, 1998-2017) was used to examine the mediating roles of maternal prenatal sleep difficulties, maternal sleep difficulties at 6-weeks postpartum, and infant sleep at 6-weeks postpartum on the relationship between maternal ACEs and infant negative reactivity at 16-weeks postpartum. Maternal race (white = reference), level of education, infant sex (0 = female; 1 = male), and infant sleep problems at 16-weeks were included as covariates in the model as well as correlations between infant sleep problems at 6- and 16-weeks, and among the exogenous predictors (ACEs, race, education, 16-week infant sleep problems). Preliminary analyses examined prenatal and 6-week postpartum depressive symptoms as predictors of maternal sleep difficulties at 6- and 16-weeks postpartum, infant sleep problems at 6-weeks, and infant negative reactivity at 16-weeks, as well as 16-week postpartum depressive symptoms as a predictor of infant negative reactivity at 16-weeks. Across all preliminary analyses examining maternal depressive symptoms, the only significant path coefficient was the association between 6-week maternal depressive symptoms and concurrent 6-week maternal sleep problems (b = .37, SE = .14, p < .01). However, excluding 6-week maternal depressive symptoms slightly improved model fit and made model interpretation more parsimonious, and thus maternal depressive symptoms was dropped in the final model.

Overall, the fit indices for the final model presented in Figure 1 indicated acceptable fit (Hooper et al., 2008; Asparouhov & Muthén, 2018), $\chi^2 = 15.7$ (16), $p = .47$; RMSEA = .00 (90% CI[.00, .11]; CFI = 1.0; and SRMR = .09. The model predicted 80% of the variance in infant negative reactivity, 20% of the variance in infant sleep problems at 6-weeks, 44% of the variance in maternal sleep problems at 6-weeks, and 6% of the variance in maternal sleep problems during pregnancy, according to R^2. See Table 4 for complete model results.

Maternal sleep problems at 6-weeks and infant sleep problems at 6-weeks were found to be significant mediators in the association between maternal ACEs and infant negativity reactivity, indirect effect = 0.06, 95% CI = [.002, .15] (Table 4). Results also indicated a second mediated pathway, with maternal sleep problems at 6-weeks and infant sleep problems at 6-weeks mediating the association between maternal sleep problems during

pregnancy and infant negativity reactivity, indirect effect = 0.06, 95% CI = [.003, .13] (Table 4). Finally, infant sleep problems at 6-weeks mediated the association between maternal sleep problems at 6-weeks and infant negativity reactivity, indirect effect = 0.10, 95% CI = [.01, .20] (Table 4).

4. Discussion

Findings from the current study indicated that maternal adverse childhood experiences and prenatal sleep problems were independently associated with the emergence of infant negative reactivity at four months of age, with significant indirect effects through maternal and infant sleep problems at 6-weeks postpartum. That is, infants were more likely to develop sleep difficulties and show more negative reactivity if their mother had a history of ACEs, and/or experienced sleep problems during pregnancy and postpartum. These findings provide evidence for early infant sleep as an indicator of and a risk factor for emerging difficult temperament and for maternal sleep as a potential mechanism that influences infant neurodevelopment.

4.1 Infant sleep and negative reactivity

Infant sleep problems at 6-weeks appear to be a highly predictive precursor to infant negative reactivity at 16-weeks, which is consistent with previous research linking early sleep regulation to later neurobehavioral outcomes like irritable and negative temperament (Sadeh et al., 1994; Spruyt et al., 2008). Interestingly, although infant sleep problems were highly correlated between 6- and 16-weeks, infant sleep problems at 16-weeks (concurrent) only had a small but nonsignificant association with infant negative reactivity. At 6-weeks, infants do not yet have an established circadian rhythm and tend to only sleep for short periods of time (0.5-2 hours on average; Mindell et al., 2016), whereas consolidation of sleep often begins between 3-4 months, with distinct consolidation of nighttime sleep and a circadian pattern by six months (Mindell et al., 2016; Henderson et al., 2010). In our sample, mother-reported infant sleep problems declined significantly from 6- to 16-weeks, which may reflect this emerging sleep consolidation and typical neurobiological maturation.

With mothers reporting general improvements in infant sleep overall, even among infants with persistently higher sleep problems, it seems reasonable that sleep would be less associated with maternal perceptions of negative reactivity as infants age, which is consistent with research examining early regulatory problems in infants older than three months of age (Sidor et al., 2017). Thus, although infant sleep in the first 2-3 months is highly variable and characterized by short durations, it seems that relative elevations in sleep problems during this period may be an early marker of neurobiological dysregulation that reflects an innate predisposition to negative reactivity or may foster negative reactivity through a cycle of insufficient sleep. With evidence that sleep-wake patterns and infant temperament may reflect similar aspects of infant biological maturity and organization (Morales-Muñoz et al., 2020; Spruyt et al., 2008), infant sleep problems warrant further investigation as a predictor of negative affect and the emergence of more difficult temperamental traits. More frequent measurement of infant sleep and sleep trajectories across infancy and toddlerhood, as well as studies with sleep interventions, may help to

disentangle environmentally-driven sleep-related regulatory problems from neurobiological predisposition.

4.2 Effects of maternal sleep problems

For the majority of the sample, sleep problems were elevated during pregnancy and declined to relatively stable levels from 6- to 16-weeks, suggesting that pregnancy-related discomfort and associated disruptions in sleep were likely common in the third-trimester (Hayase et al., 2014). Although the majority of women had fewer sleep problems postpartum, possibly returning closer to their typical baseline levels, it is notable that elevated prenatal sleep problems that persisted at 6-weeks postpartum were associated with infant sleep problems and negative reactivity. Notably, there was a prospective, indirect path from prenatal sleep problems to mother and infant sleep problems at 6-weeks and infant negative reactivity at 16-weeks, suggesting maternal prenatal sleep may be an important factor that contributes to early sleep regulation and emergence of negative reactivity, with implications for prenatal programming research. That is, chronic sleep disruption during pregnancy may reflect underlying neurobiological dysregulation (Buss et al., 2017; Levenson et al., 2015; McEwen, 2006), and may be an important pathway for prenatal programming.

At 6-weeks postpartum, our data showed that infant sleep problems were strongly associated with maternal sleep problems, which may reflect a shared genetic or neurobiological predisposition to sleep dysregulation (Kocevska et al., 2021), or may also reflect the influence of the shared postnatal environment and maternal behavior, including maternal sleep behavior, maternal attunement, and emotionality (Field, 2017). For example, there is evidence that maternal characteristics may be influencing infant sleep patterns, with reports of elevated maternal stress being associated with concurrent infant sleep problems (van den Huevel et al., 2021; McQuillan et al., 2019), and data showing that by 2-weeks postpartum, infants of mothers with depression had greater sleep difficulties that persisted across six months (Armitage et al., 2009). Additionally, Burdayron and colleagues (2021b) reported that by six months postpartum, high levels of maternal depression in the context of high levels of infant negative affectivity contributed to mothers' perceptions of infant sleep problems, independent of objective measures of infant sleep duration and awakenings, suggesting that maternal mental health may be particularly important to consider when examining infant sleep problems and difficult temperament. However, it should be noted that these previous studies did not include independent reports of maternal sleep difficulties, and it may be possible that the negative influence of maternal depression or stress is driven in part by disruptions in maternal sleep.

Our preliminary analyses included maternal depressive symptoms as a covariate that was ultimately excluded from the final model due to reduced model fit and a limited impact on primary study variables. In the preliminary analyses, maternal depressive symptoms at 6-weeks (but not prenatally) and prenatal sleep problems both had unique associations with 6-week maternal sleep problems, suggesting that although sleep dysregulation and depressive symptoms likely share underlying etiology, they may reflect independent pathways for postpartum sleep disruption, at least in our limited data.

4.3 Effects of maternal adversity

Consistent with previous work on the intergenerational transmission of adversity, our findings suggest that maternal experiences, such as those that occur during childhood and pregnancy, can have downstream consequences for offspring via biological and neurodevelopmental mechanisms (Buss et al., 2017). Interestingly, mothers with high ACEs reported significantly more sleep problems at 6- and 16-weeks postpartum compared to mothers with low to moderate ACEs who had notable improvements in sleep postpartum, suggesting that mothers with high ACEs exposure may be at particular risk for chronic sleep problems (as well as depressive symptoms, which followed a similar pattern in our data) (Miller-Graff & Cheng, 2017; Paulson & Miller-Graff, 2019).

There is considerable evidence that ACEs are associated with increased rates of stress and stress-related difficulties and that early life experiences are relevant for pregnancy-related outcomes and perinatal well-being (Buss et al., 2017). Several studies document altered HPA-axis and immune functioning associated with ACEs exposure (Danese & McEwen, 2012; Danese et al., 2009; Jaffee & Christian, 2014; Nurius et al., 2015), and the current study suggests that associated alterations in sleep regulation may also reflect ACEs-related neurobiological dysregulation for both mothers and infants (Hairston et al., 2011; Kajeepeta et al., 2015;). This may be particularly true for those who experience abuse-type, or threat-based, ACEs, as there is research showing that exposure to abuse, relative to neglect or household dysfunction, is associated with heightened physiological reactivity, dysregulated emotion regulation, and enhanced fear learning (Grasso et al., 2020; Milojevich et al., 2019), which are factors also associated with sleep disturbances (Gruber & Cassoff, 2014). Moreover, several studies report other psychological and behavioral factors associated with ACEs that may also contribute to the emergence or maintenance of sleep problems. For instance, risky health behaviors, such as substance use and smoking can influence sleep health (Anda et al., 2006). Importantly, the current results specify interrelated pathways of maternal childhood adversity and perinatal sleep disturbances that may help inform future research on neurobehavioral dysregulation in early infancy and emerging difficult temperament from a prenatal programming perspective (Barker 1998; Buss et al., 2017; Gillman, 2005; Talge et al., 2007).

In utero exposure to dysregulated maternal stress reactivity, which has been associated with both history of ACEs and chronic disruptions in sleep, is hypothesized to program or alter infant neurodevelopment and subsequent physiological stress reactivity (Buss et al., 2017; Monk et al., 2019), which may contribute to sleep difficulties in early infancy as well as predispose infants to negative reactivity and difficult temperament (Sidor et al., 2017; Talge et al., 2007). Research on maternal prenatal stress and depression has pointed to infant neurobehavioral reactivity, including physiological stress reactivity and temperament, as a key mechanism or underlying risk factor associated with prenatal programming and the emergence of infant sleep problems (Field, 2017; Dias & Figueiredo, 2020; Kim et al., 2020; O'Connor et al., 2007). In our data, there is evidence for continuity in sleep problems over time for both mothers (starting in pregnancy) and their infants, which may reflect this programmed underlying neurobehavioral dysregulation, or in turn, is also consistent with research that characterizes sleep problems as chronic stressors that can activate allostasis,

leading to persistent cycles of disrupted sleep and stress activation (Levenson et al., 2015; McEwen, 2006; Palagini et al., 2014). Together, these findings provide supportive evidence to examine neurobiological correlates of stress reactivity and sleep in future research to determine whether sleep dysregulation during pregnancy and postpartum may be a marker of, or trigger for, underlying physiological dysregulation, including HPA axis functioning (Bublitz et al., 2018; Davis et al., 2007).

Of course, there are many other stressors that could prospectively or concurrently contribute to the onset of sleep problems, leading to a cycle of hyperarousal and chronic sleep disturbance, including stressful life events, interpersonal conflict, and depression or anxiety (Chapman et. al., 2011; Drake et al., 2014). Our sample included a racially diverse, economically disadvantaged demographic that may have higher exposure to psychosocial stressors linked to sleep disturbances, like racism or financial instability (Hicken et al., 2013; Okun et al., 2014). Even though data from our small sample showed that maternal race and education were not associated with maternal ACEs, maternal or infant sleep, or infant temperament, previous studies suggest that many types of adversity are disproportionately distributed across race/ethnicity and income, with families of color and those living in poverty facing an unbalanced burden of adversity, as well as notably higher prevalence of ACEs in among racially marginalized groups like non-Hispanic Black Americans and Native Americans/Alaska Natives (Goldstein et al., 2020; Nurius et al., 2012). As such, the relationship between pregnancy, sleep, and race/ethnicity is complex, and recent evidence suggests the association between pregnancy and sleep may differ by race/ethnicity (Feinstein et al., 2020). Further, adverse environmental conditions, such as living in poverty, are associated with reduced opportunities to obtain sufficient sleep and may compromise sleep quality (Okun et al., 2014). Thus, although our sample did not indicate differences by race or social class, it is important to consider the interrelatedness of racial and social health disparities and chronic sleep disturbances and further examine these associations in larger, more diverse populations.

4.4 Strengths, Limitation, and Future Directions

The current study had several strengths including being a prospective longitudinal study that examined ACEs and sleep difficulties among a sample of racially diverse, economically disadvantaged, and medically underserved mothers and infants. Notably, our study included multiple assessment points from pregnancy into postpartum, documenting sleep through the transition to motherhood and in early infancy, which is currently lacking within the literature.

Although the present study has notable strengths, it is not without limitations. Our small sample size and attrition over time resulted in low power to detect effects, particularly of smaller estimates like indirect effects. Confidence in our reported results is bolstered by several factors despite limited power: the hypothesized model was based on previously described, empirically-supported associations among all study variables; the data were collected using validated scales with good internal reliability; participants were measured at multiple time points within a narrow perinatal timeframe; effect sizes and associations were consistent across analyses (i.e., correlation, ANOVA, path analysis); and in follow-up

analyses, the primary and indirect effects reported in Fig. 1 were replicated using PROCESS in SPSS. Further, research on sample size requirements for common types of SEMs showed that a sample size of $n = 50$ had sufficient power (86%) to analyze primary single-indicator direct relationships of interest showing moderate to large effects (Wolf, Harrington, Clark, & Miller, 2013), which provides some support for examining primary relationships of interest in our data. Of course, caution should be used when interpreting or generalizing our results in light of the issue of low power, and the findings examined in the context of previous research. These findings provide important preliminary evidence in understanding the role of maternal adversity and perinatal sleep disturbance in the intergenerational transmission of adversity, and these estimates will be helpful for researchers planning similar intergenerational studies so that they may have adequately powered study designs. Future research would benefit from larger sample sizes with variability in ACE scores that would allow a more detailed analysis according to types of ACE exposure (Milojevich et al., 2019), and extra efforts for participant engagement should be made to minimize attrition.

Additionally, data were self-reported, which may have contributed to recall bias or lack of comfortability with reporting past experiences and be may be confounded by mood. Self-report data specific to maternal and infant sleep difficulties may be particularly vulnerable to biased reporting, as there is evidence that maternal perceptions of infant behavior and sleep are influenced by maternal mood symptoms (Burdayron et al., 2021b). Thus, future research would benefit from assessing sleep using objective measures of sleep, like actigraphy, wearable biosensors, or video recordings, in addition to self-report, to provide a less biased and comprehensive examination of infant and maternal sleep patterns. Further, infant sleep was not measured earlier than 6-weeks, and data on infant sleep, temperament, and behavior was not examined beyond 16-weeks, capturing only a limited timeframe of development that may miss important patterns in infant regulation that may emerge earlier in infancy or later during and beyond the first year. Importantly, to support the hypothesized theory that sleep problems and negative reactivity may reflect underlying neurobiological dysregulation and that sleep may play a role in prenatal programming of stress reactivity, future research should include biomarkers of stress reactivity, including HPA-axis functioning (Bublitz et al., 2018; Davis et al., 2007).

Lastly, our final model did not include a measure of maternal psychopathology. Preliminary analyses examining prenatal and 6-week maternal depressive symptoms showed overlapping variance among maternal ACEs, depressive symptoms, and sleep problems that complicated model interpretation and reduced the fit. However, it seemed that depressive symptomology pulled variance from ACEs more so than sleep, suggesting that depressive symptoms may be closely tied to ACEs-related alterations in neurobiological functioning (Danese et al., 2009; Teicher et al., 2013). In turn, sleep disturbances during pregnancy/postpartum may likely stem from multiple factors, and reflect a somewhat independent pathway that is important for infant sleep and social-emotional development (Chang, 2010; McQuillan et al., 2019). Given that prenatal stress and depression have implications for neurobehavioral reactivity (Field, 2011), as well as sleep regulation (McQuillan et al., 2019), future research would benefit from a more thorough examination of maternal stress, anxiety, and depression in relation to infant sleep problems and negative reactivity. Future studies should also collect data on complications associated with pregnancy and childbirth, as risk for perinatal

complications have been associated with maternal ACEs (Ciciolla et al., 2021) and linked with disruptions in sleep and neurobiological dysregulation (Chang et al., 2010).

4.5 Practical Implications

Our findings underscore the importance of perinatal screening for sleep problems and known risk factors, such as ACEs, to help to identify mothers who may benefit from targeted support and intervention. Our data supports the literature suggesting the importance of screening for ACEs during prenatal care visits and aligns with findings from Flanagan and colleagues (2018) on the feasibility and acceptability of this practice. Further, screening for sleep problems into the postpartum for both mothers and their infants is especially important, given that chronic sleep problems can be seen as "normative" and several maternal health care needs go unmet during the first few months following birth or into the "fourth trimester" (Tully et al., 2017).

Our findings also demonstrate the importance of characterizing prenatal sleep problems as a unique risk factor that may influence infant sleep and infant temperament. Addressing prenatal sleep problems may also prevent subsequent difficulties such as the emergence of infant sleep problems and more difficult temperamental traits. For example, there is evidence that interventions like the Sleep Enhancement Training System for Pregnancy, an evidence-based cognitive-behavioral training program, may help to reduce chronic sleep disturbance by improving duration and quality of sleep during late pregnancy and into early postpartum (Lee et al., 2016). Addressing psychosocial functioning and maternal sleep during pregnancy, particularly among women with high ACEs, may be a target of intervention to improve maternal and infant sleep health during the postpartum, and reduce the risk for difficult temperament among infants.

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Highlights

- Infant sleep problems at 6-weeks appear to be a highly predictive precursor to infant negative reactivity at 16-weeks.
- Mothers with high ACEs exposure may be at particular risk for chronic perinatal sleep problems
- Maternal ACEs and perinatal sleep are associated with infant sleep dysregulation and emerging difficult temperament.
- Results suggest perinatal sleep disturbance may be a key factor for intergenerational outcomes.

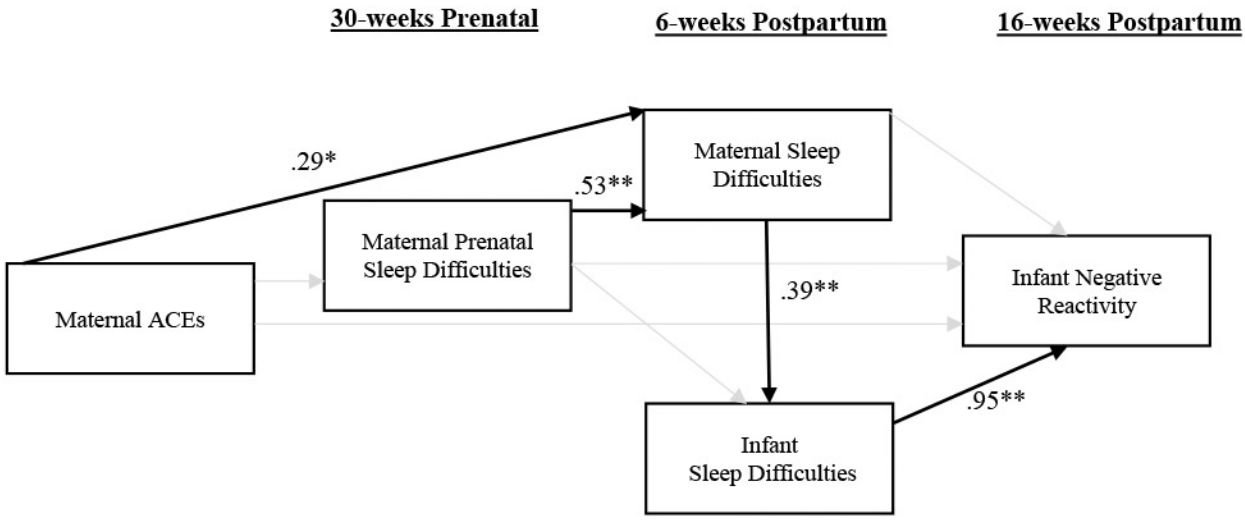

Fig. 1.

Path model examining maternal and infant sleep at 6 weeks postpartum as mediators. Analyses controlled for race, education, infant sex, and 16-week infant sleep problems. Statistically significant standardized path coefficients are reported. ACEs = Adverse childhood experiences. *$p < 05$; **$p < .01$.

Table 1.

Sample demographic characteristics.

Variable	*n* (%)
Age	
19-30	41 (69.5)
31-41	18 (30.5)
Marital Status	
Married	21 (36.2)
Cohabitating	16 (27.6)
Single/separated/divorced/widowed	21 (36.1)
Annual Income ($US)	
< $20,000	38 (65.5)
$21,000 – $60,000	15 (25.8)
> $61,000	5 (8.6)
Education Level	
High school or less	32 (54.2)
Some college/associates degrees	23 (39.1)
Bachelor's degree or more	4 (6.8)
Race	
White	26 (44.1)
Black or African American	12 (20.3)
Native American or Alaska Native	9 (15.3)
Asian	1 (1.7)
Biracial or Multiracial	9 (15.3)
Other	2 (3.4)
First pregnancy	16 (27.1)

Table 2

Descriptive statistics and mean comparisons of key study variables according to ACEs category

	Total Sample	0-1 ACE	2-3 ACEs	4+ ACEs	ANOVA
	n **= 59**	***n*** **= 20**	***n*** **= 14**	***n*** **= 21**	
Variable	***M (SD)***	***M (SD)***	***M (SD)***	***M (SD)***	F_{ACES}
Prenatal WHII	10.76 (4.91)	8.45 (4.12)	11.43 (6.03)	11.94 (3.99)	3.07
6-week WHII	8.36 (5.23)	5.33 (3.60)	8.43 (5.29)	11.13 (5.35)	5.84**
16-week WHII	8.89 (5.35)	5.67 (4.72)	6.00 (4.74)	13.07 (3.01)	13.36**
6-week ISQ	21.34 (10.72)	14.07 (7.10)	23.00 (11.55)	27.44 (9.44)	8.67**
16-week ISQ	14.43 (8.85)	12.67 (7.95)	14.80 (6.02)	17.20 (9.89)	0.90
16-week Infant Negative Reactivity	3.92 (1.11)	3.43 (.99)	3.64 (.53)	4.13 (1.11)	1.59
ACEs total[a]	3.02 (2.62)	-	-	-	-
Prenatal EPDS	7.09 (6.22)	4.9 (4.58)	5.92 (6.8)	10.26 (6.22)	4.48*
6-week EPDS	5.85 (5.3)	2.93 (3.55)	6.25 (4.33)	8.24 (6.1)	4.65*
16-week EPDS	6.58 (7.34)	3.42 (4.32)	2.8 (1.48)	10.13 (8.63)	4.42*

Note. WHII = Women's Health Initiative Insomnia Total Score; ISQ = Infant Sleep Questionnaire; ACEs = Adverse Childhood Experiences; EPDS = Edinburgh Postnatal Depression Scale

[a]Total ACE scores ranged from 0 to 9.

*$p < .05$

**$p < .01$.

Table 3.

Correlation between demographic variables and key study variables.

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Prenatal WHII	-														
2. 6-week WHII	.62**	-													
3. 16-week WHII	.42*	.56**	-												
4. 6-week ISQ	.37*	.46**	.49**	-											
5. 16-week ISQ	−.07	−.02	.42*	.65**	-										
6. 16-week Infant Neg Reactivity	.31	.15	.26	.69**	.24	-									
7. 16-week Infant Orienting	.03	−.17	−.15	−.36*	−.25	−.33	-								
8. 16-week Infant Surgency	.28	.28	−.05	−.13	−.33	.11	.40*	-							
9. ACEs categories[a]	.31*	.50**	.65**	.57**	.24	.32	.01	−.11	-						
10. ACEs total score[b]	.27	.51**	.60**	.39*	.07	.11	−.02	−.09	.88**	-					
11. Abuse ACE	.35*	.50**	.58**	.32*	.11	.13	−.14	−.10	.75**	.84**	-				
12. Neglect ACE	.16	.46**	.48**	.39*	.04	−.11	−.10	−.11	.69**	.85**	.69**	-			
13. Household dysfunction ACE	.18	.33*	.39*	.17	−.02	−.06	.18	−.07	.73**	.85**	.53**	.65**	-		
14. Prenatal EPDS	.37**	.45**	.43*	.35*	.14	.12	−.17	.16	.37**	.34*	.39**	.25	.17	-	
15. 6-week EPDS	.38*	.63**	.42*	.48**	.16*	.42	−.44*	.23	.44**	.43**	.47**	.33*	.18	.71**	-
15. 16-week EPDS	.20	.53**	.45**	.39*	.04	.37*	−.37*	.14	.44*	.48**	.48**	.41*	.20	.69**	.86**

Note. WHII=Women's Health Initiative Insomnia Total Score; ISQ = Infant Sleep Questionnaire; ACEs = Adverse Childhood Experiences; EPDS = Edinburgh Postnatal Depression Scale

[a] High (4+), Moderate (2-3), and Low (0-1) ACEs

[b] ACEs total scores ranged from 0 to 9. No statistically significant correlations were found for Infant sex (0 = female, 1 = male), Maternal Education, or Maternal Race (1=white; 0=Black, Native American/Alaska Native, Hispanic, Asian, Other).

* $p < .05$

** $p < .01$.

Table 4.

Model coefficients for path analysis.

	β	b(SE)	95% CI
Direct Paths			
ACEs → Prenatal WHII	.25	.46 (.28)	[−0.08, 1.02]
ACEs → 6wk WHII	.29*	.55 (.22)	[.15, 1.03]
ACEs → Negative Reactivity	−0.22	−0.11 (.08)	[−0.24, .06]
Prenatal WHII → 6wk WHII	.53**	.55 (.18)	[.17, .83]
Prenatal WHII → 6wk ISQ	.09	.19 (.37)	[−0.62, .88]
Prenatal WHII → Negative Reactivity	.42	.12 (.08)	[−0.04, .26]
6wk WHII → 6wk ISQ	.39**	.79 (.32)	[.22, 1.5]
6wk WHII → Negative Reactivity	−0.38	−0.11 (.07)	[−0.26, .05]
6wk ISQ → Negative Reactivity	.95**	.13 (.04)	[.04, .18]
Covariates			
Maternal Race (white) → Negative Reactivity	.04	.11 (.38)	[−0.61, .87]
Maternal Education→ Negative Reactivity	−0.15	−0.20 (.19)	[−0.47, .29]
16-week ISQ → Negative Reactivity	−0.22	−0.04 (.03)	[−0.09, .03]
Infant sex	.07	.18 (.47)	[−0.75, 1.13]
Correlations			
Maternal Race (white) with Maternal Education	.16	.08 (.06)	[−0.04, .20]
Maternal Race (white) with ACEs	.11	.15 (.17)	[−0.22, .46]
Maternal Race (white) with 16wk ISQ	−0.09	−0.38 (.61)	[→1.53, .84]
Maternal Education with ACEs	−0.06	−0.16 (.41)	[−1.09, .55]
Maternal Education with 16wk ISQ	.03	.25 (1.2)	[−2.12, 2.57]
ACEs with 16wk ISQ	−0.03	−0.62 (3.4)	[−8.06, 5.61]
6wk ISQ with 16wk ISQ	.65**	52.25 (18.8)	[17.07, 91.38]

Mediated Paths	***Indirect Effects***	
ACEs → 6wk WHII →6wk ISQ →Negative Reactivity	.06 (.04)	[.002, .15]
Prenatal WHII→ 6wk WHII →6wk ISQ →Negative Reactivity	.06 (.04)	[.003, .13]
6wk WHII →6wk ISQ →Negative Reactivity	.10 (.07)	[.01, .20]

Note. WHII=Women's Health Initiative Insomnia Total Score; ISQ = Infant Sleep Questionnaire; ACEs = Adverse Childhood Experiences total score (range 0-9). Maternal Race (1=white; 0=Black, Native American/Alaska Native, Hispanic, Asian, Other). Infant sex (0=female; 1=male).

* $p < .05$

** $p < .01$.