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Maternal Sleep Quality and Infant Cognitive Development: Potential Mediating Roles of Maternal Inflammatory Processes, Preterm Birth, and Infant Sleep Quality

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Maternal Sleep Quality and Infant Cognitive Development: Potential Mediating Roles of Maternal Inflammatory Processes, Preterm Birth, and Infant Sleep Quality

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Abstract

Maternal prenatal sleep quality has been implicated as a predictor of birth outcomes and child cognitive development, and African American women suffer from poor sleep quality more than women of other racial identities. Few studies have investigated the intergenerational effects of poor maternal sleep quality on childbirth and development. The current study sought to expand the conceptual framework by using a longitudinal design to investigate several potential mediators in the association between maternal prenatal sleep quality and infant cognitive development. Our sample of 142 African American mother-child pairs were assessed from pregnancy though 18 months postpartum. Hierarchical Linear Modeling (HLM) results revealed that maternal reports of disturbed sleep quality during the prenatal period were associated with poorer cognitive development in the social emotional and expressive language domains of the Bayley. None of the proposed mediators were significantly associated with maternal sleep quality or cognitive development. Further investigation of the mechanisms associated with the intergenerational impacts of maternal prenatal sleep quality on cognition in African American children is warranted.

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Introduction

Literature has shown that many factors contribute to a healthy pregnancy and birth, whether they be environmental or genetic (Kaiser, 2008; Varner et. al., 2005). Pregnancy is a time of increased risk for both mother and child; therefore, it is important to investigate how maternal prenatal experiences shape postpartum outcomes. Sleep disturbances have consistently been common complaints among pregnant women; thus, studying the detrimental effects they may have on pregnancy outcomes are of importance. Often negatively affected by day-to-day stressors, and as a stressor itself, sleep quality in mothers can become disordered and lead to a number of negative outcomes (Nodine & Matthews, 2013; Blair et. al., 2015; Li et. al., 2017; Okun et. al., 2011). Among those are an increase in risk of preterm birth, postpartum depression, caesarean birth, painful labor, and longer duration of labor (Chang et. al., 2010; Lee & Gay, 2004; Reichner, 2015; Zhou et. al., 2020). Disturbed sleep can affect system level processes in mothers, such as inflammation, nervous system activation, and neural circuitry responses as well (Buysse, 2014). Feinstein et al. (2020) found that African American women presented with a higher prevalence of sleep disorders than other racial groups. A review by Christian et. al., reported in 2019 that 45.8% of African American adults in the United States experience insufficient sleep duration independent of socioeconomic status. This percentage is higher than that of any other racial/ethnic group. Despite these alarming findings, relatively few studies have attempted to investigate this disparate affliction and create ways to mitigate the negative physical and psychological responses that ensue. Other risk factors for poor sleep quality in women include lower socioeconomic status, frequent experiences of discrimination, low self-perceived societal status, minority status, extended work hours, and living in an urban environment; all factors that African American women are more likely to experience (Chang et. al., 2010; Francis

et. al., 2017; Hoggard & Hill, 2018; Slopen et. al., 2016). These risk factors exist in addition to normal pregnancy symptoms such as fluctuating hormones, overall discomfort, nausea, etc. (*Pregnancy & Sleep*, 2020). However, despite the implications of sleep quality as an amplified stressor during pregnancy for African American women, few studies have investigated the effect of poor prenatal sleep quality in mothers of communities that may experience disparate levels of stress.

Infant cognitive development is an outcome often shaped by maternal experiences during pregnancy. The fetal programming hypothesis states that the environment in utero has the potential to alter the development of the fetus (Kwon & Kim, 2017). Prenatal impacts on infant cognitive development include maternal depressive symptoms, exposure to pathogens, drugs and alcohol, and heightened levels of maternal cortisol (Galler et. al., 2003; Petterson et. al., 2015; Ernst et. al, 2001; Bailey et. al., 2004; Bergman et. al., 2010). Several studies exist investigating maternal sleep quality and its effects on maternal health and immediate birth outcomes such as gestational age; however, no studies to date have examined whether there exists a developmental relationship between maternal sleep quality and infant cognitive functioning.

The present study will seek to fill these gaps in the literature, by investigating the association between poor prenatal maternal sleep quality on child cognitive development in a sample of African American women. Investigating problems early in pregnancy can inform ways to avoid such negative effects within these communities. For example, clinician efforts to recognize and adjust sleep habits during the prenatal period may contribute to decreased risk of negative health outcomes in high-risk children.

Given the data collected on maternal sleep quality at different times in pregnancy and child assessment of cognitive development in the postpartum, our study sample allowed us to

garner unique information about the potential intergenerational consequences of disturbed sleep in a high-risk sample. We hypothesized that poor sleep quality in the prenatal period has a detrimental effect on child cognitive development both directly, and indirectly through mediators such as infant sleep quality, preterm birth, and maternal inflammation. The overarching goal of this study was to increase our understanding about the associations between poor prenatal sleep quality and negative cognitive developmental outcomes for infants.

Preterm Birth

A full-term birth and healthy birth rate are vital for normal child brain development, as preterm children present with significantly lower cognitive ability than full term children (Cheong et. al., 2017; Woods et. al., 2010). Risk factors of preterm birth include access to quality prenatal health care, experiences of discrimination, exposure to stress, and poor nutrition, among others (Lewis et. al., 2013; Blair et. al., 2015). These are all risk factors more likely to be experienced by pregnant women of lower income communities and/or ethnic minority status.

As certain populations are more susceptible to negative birth outcomes than others, pregnant African American women and their children are an especially high-risk population with disproportionate preterm birth rates (Goldenberg et. al., 1996; Gortzak-Uzan et. al., 2001; Hobel et. al., 2008; Howell, 2018; March of Dimes Report Card, 2020; Raymond et. al., 2012). African American women exhibit significantly higher rates of preterm births than any other racial group. According to the literature, the reasons for this disparity have been associated with institutional racism, in addition to known risk factors of preterm birth, such as reduced access to quality prenatal care. Prior research that has explored risk factors in pregnant African American women often focuses on experiences of stress and define exposure to discrimination as a common and unique stressor within this population. Despite the ability to isolate unique experiences of

African American women, researchers have consistently been unable to come to a conclusive agreement as to why preterm birth occurs so disproportionally in this population; a statistic that has been stable across the years despite advances in healthcare and technology. Many additional factors contribute to increased risk during pregnancy; however, the literature addressing the glaring disparity in preterm birth of African American children is less than adequate. The present study incorporates gestational age in the statistical model with hopes to identify a prenatal risk factor for preterm birth.

Maternal Inflammatory Responses

Increases in inflammation have been found to mediate associations between sleep quality and adverse health outcomes, such as cardiovascular disease, diabetes, obesity, and even mortality (Dowd et. al., 2011). Inflammation, specifically in pregnancy, has been shown to have an influence on infant brain development, with a positive association between increase in inflammation and infant neurodevelopmental and psychiatric disorders (Rudolph et. al., 2018). Maternal immune activation, which is elicited by exposure to infectious stimuli, has been found to trigger inflammation for pregnant mothers. Exposure to infection stimulates an increase in cytokines, which act as a mediator between cells of nonimmune tissues, such as in the nervous system (Deverman & Patterson, 2009). This spike in inflammatory cytokines concurrently triggers an increase in maternal inflammation, which can cross the blood-brain barrier and create alterations in the fetal brain. Fetal brain alterations due to maternal inflammation in utero have been found to increase infant risk for schizophrenia, autism, and epilepsy (Estes & McAllister, 2016; Knuesel et. al., 2014). Although more research is necessary in the area of maternal immune activation in human models, it is clear that an increase in maternal cytokines directly

influences fetal development, and that prenatal exposures to inflammation have a negative effect on infant neurodevelopment (Giovanoli et. al., 2015; Smith et. al., 2007).

Disturbances in sleep have also been associated with increases in levels of inflammatory biomarkers, being most prevalent in women (Dzierzewski et. al., 2020). Poor sleep quality has been associated with significantly elevated levels of inflammatory biomarkers, with sleep deprivation episodes being linked to activation of proinflammatory responses. In African American women specifically, poor sleep quality has been associated with more robust inflammatory dysregulation and cytokine production than in White women (Carroll et. al., 2019). Blair et. al. (2015) found that inflammation was a significant mediator between sleep quality and gestational age at birth in African American women. Similar associations were not found for White women, despite rates of disturbed sleep being similar across racial groups in the sample. These findings have been replicated, suggesting that the association between inflammation and sleep quality may be amplified in African American women and, therefore, that maternal prenatal sleep quality may have an amplified effect in African American children as well.

Infant Sleep Quality

Infant sleep quality has been demonstrated to have an association with infant cognitive development. Several studies have found a link between higher cognitive achievement and sleep and wakefulness in newborns to school age children (Scher, 2005). The literature has found that differences in sleep quality and quantity are important in development of memory, language, and executive functioning. Normative maturation of structures in the central nervous system were found to underlie cognitive development and regulation of sleep/wake cycles (Tham, Schneider, & Broekman, 2017). A study by Pisch, Wiesemann, and Karmiloff-Smith found in a sample of 3-month-old female infants that the amount of sleep awakenings at night affected the

developmental trajectory of working memory. Those infants with an earlier maturation of working memory were found to spend less time awake during the night in the first months of life compared to those with a more delayed maturation of working memory processes. Despite these results, there is lack of consensus among researchers regarding the implication of infant sleep quality on normative cognitive development. Review studies have reported that although robust findings have not been found, it is of importance to further investigate the implications of the role of sleep in cognitive development; specifically, executive functioning, and reasoning and problem-solving abilities (Spruyt, 2019).

The majority of studies that have investigated infant sleep often examine infant sleep quality in relation to postpartum maternal sleep quality. It has been consistently found that maternal sleep quality postpartum is affected by infant sleep quality. However, researchers have not investigated whether there exists an intergenerational link between prenatal maternal sleep quality and postpartum infant sleep quality. It would be noteworthy to investigate whether a mother's prenatal sleep quality shapes her child's sleep quality early in life. The literature is also sparse when looking at effects of infant sleep on infant cognitive development concurrently, as most available studies have either investigated sleep only in school age children and adults or, if looking at infants, connecting infant sleep to cognition at a later developmental timepoint in childhood. In addition to the aforementioned gaps in the literature, no studies to date have investigated the effects of infant sleep quality on cognition in high-risk populations.

The Present Study

As an initial hypothesis, we proposed that maternal sleep quality in early pregnancy would be associated with infant cognitive development. Emphasis was placed on early pregnancy because research suggests that poor maternal sleep quality during the first trimester

may have particularly detrimental effects on birth outcomes, given that this is a critical period for fetal brain development. Negative effects on infant cognitive development may be more robust in African American women, who are already more susceptible to adverse birth outcomes as well as poor sleep quality. We theorized that poor sleep quality in the prenatal phase would negatively impact infant cognitive development postpartum. Because this intergenerational relationship has yet to be investigated and literature up to date studying pregnant African American women is sparse, we anticipated novel results from this study.

We incorporated mediators in the conceptual model that have been consistently investigated in both maternal sleep and infant cognitive functioning research. Inflammation was included as a mediator between maternal sleep quality and fetal brain development as previous research on infant cognitive development has established the negative effects of maternal immune response in utero within majority populations. Because our sample is exclusively African American mothers who are at statistically higher risk for preterm birth and because of the implications of low gestational age at birth on infant cognitive development, we investigated gestational age at birth as a mediator as well. Additionally, because the literature has established infant sleep quality as positively associating with cognitive development later in life, we included infant sleep quality as a mediator to examine whether these findings hold true for infant cognitive development as well.

Importantly, the current study utilized a longitudinal design to investigate the potential intergenerational impacts of maternal prenatal sleep quality on the development of cognitive abilities in infants. Evidence suggests that infant cognitive development may be strongly influenced by prenatal mechanisms; therefore, an investigation of these mechanisms may inform

early intervention research to increase positive outcomes for high-risk mothers and their children.

Ultimately, results from the current study attempt to explain the role that maternal sleep quality has on infant cognitive development through gestational age, infant sleep quality, and maternal pro-inflammatory biomarkers. Our use of an African American, longitudinal sample of mothers and children will hopefully provide new insights into the mechanisms driving infant cognitive development in this population. The specific hypotheses that were tested are as follows:

Aim 1: Determine whether prenatal maternal sleep quality predicts infant cognitive functioning in African American children. *Hypothesis 1*: Maternal sleep quality in the prenatal period will be positively related to infant cognitive development from 6 to 18 months of age.

Aim 2: Determine whether maternal sleep quality predicts infant cognitive functioning in African American children through the following mediators: infant sleep quality, maternal prenatal inflammation, and gestational age. *Hypothesis 2a*: A positive correlation will exist between maternal prenatal sleep quality and infant sleep quality; in turn, positively influencing infant cognitive development. *Hypothesis 2b*: Poor maternal sleep quality will trigger a pro-inflammatory response in mothers during the prenatal phase, increasing number of maternal cytokines and negatively affecting infant cognitive development in utero. Mothers in the sample with increased levels of inflammation will have children with relatively lower scores assessing cognition. *Hypothesis 2c*: Mothers in the sample with relatively poor maternal sleep quality will have an increased likelihood of having children born at an earlier gestational age. Children in the sample born at preterm will be more likely to have mothers with poor prenatal sleep quality and have relatively lower scores assessing cognition compared to those children born at full term.

Aim 3: Examine the associations between the mediators: infant sleep quality, maternal prenatal inflammation, and gestational age. As an exploratory aim, we hypothesized that there exists a relationship between infant sleep quality, inflammation, and preterm birth.

Methods

Participants

The study leveraged data collected from a longitudinal sample of n = 142 women and their children (Mean age = 25.29, SD = 5.11) experiencing no known chronic medical conditions or taking prescribed medications for chronic conditions. All the women recruited identified as African American and received prenatal care at the Emory University Hospital Midtown, a private hospital that services patients from a wide economic range, or Grady Memorial Hospital, a county-supported hospital that serves as a safety net for low-income patients, resulting in a socioeconomically diverse sample (Corwin et. al., 2017). Participants consented to participate in the Emory University Baby Microbiome Study (Brennan et al., 2019). Participants were included if they completed the maternal sleep quality measures and had at least one time point of infant cognitive data. The goal of the study was to assess these mother's sleep quality and inflammatory biomarkers in the prenatal phase and infant sleep quality, cognitive development, and gestational age at birth in the postpartum.

Procedure

Participants were given a health survey during the first trimester of their pregnancy at 8-14 weeks to gather information on current prenatal health and were followed through or beyond delivery. The first-trimester study visits included collection of blood and completion of sociodemographic health, nutrition, and stressor exposure questionnaires. Mothers and their infants who elected to participate in the follow up baby study completed a series of two-hour

home or in lab visits when infants were 1, 3, 6, 12 and 18 months old. Infant cognition was assessed at 3, 6, 12, and 18 months. Preterm birth information was collected through medical records obtained about the pregnancy and birth from the hospital.

Measures

*Mother's Prenatal Sleep Quality***.** Mother's prenatal sleep quality was assessed during early and late pregnancy using the PROMIS Sleep Disturbance Short Form. The PROMIS is an eight-item form that assesses a pure domain of sleep disturbance in individuals aged 18 and older. Each item on the measure is rated on a five-point Likert scale if during the past seven days each statement described their sleep quality (1 = Not at all, 2 = A little bit, 3 = Somewhat, 4 = Quite a bit, or 5 = Very much). Responses were summed to compute a total raw score with higher scores indicating sleep disturbance. The PROMIS short form correlates strongly with the longer forms and have been shown to be reliable and valid measure (Yu et. al., 2012). Internal consistency for the PROMIS is considered acceptable: Cronbach's $\alpha = 0.76$. PROMIS scores during early pregnancy were utilized in this study ($M = 21.75$, $SD = 8.03$).

Infant Cognitive Development. Infant neurocognitive development was assessed at 6, 12, and 18 months by trained and supervised test administrators. Recordings of all administrations were used for reliability. The Bayley Scales of Infant and Toddler Development III assessment was used to measure infant cognitive development. The Bayley is a standardized assessment for infants 1 to 42 months based on normative scores across the following five domains: motor, language, social emotional, cognitive, and adaptive functioning. In the current study, scores from the language, social emotional, cognitive, and adaptive functioning domains were utilized (Del Rosario et. al., 2020).

*Maternal Inflammation***.** Routine prenatal blood samples were collected between 24-30 weeks' gestation. Serum samples were analyzed for cytokines, interferon (IFN)-γ, IL-6, IL-10, and TNF-α, using the Meso Scale assay platform (Meso Scale Diagnostics Rockville, Maryland) according to the protocols supplied by the manufacturer. Fichorova et. al., found Meso Scale assay technology as having high inter-laboratory reproducibility, low matrix effects, and high reliability (Chowdhury et al., 2009). The current study used cytokine ratios of IL6:IL10 and TNF-α:IL6 as they have been implicated in adverse pregnancy outcomes in the literature and may provide an overall better picture of an immune response (Calleja-Agius et al., 2012). Inflammation variables, which exhibited high levels of skewness, were log adjusted for model fit.

Preterm Birth. Information regarding gestational age at birth was provided by medical records obtained from the hospitals regarding the pregnancy and birth.

Child's Sleep Quality Measures. Child sleep quality was evaluated postpartum using the Brief Infant Sleep Questionnaire (BISQ). The BISQ asks mothers to recall their child's sleep within the last week by self-reporting the number of hours and minutes. Variables of the BISQ include daytime duration, nighttime duration, number of nighttime awakenings, and nighttime sleep onset time and latency. The BISQ has been supported as a useful sleep screening tool for research and clinical purposes (Sadeh, 2004). In the current study sample, the internal consistency for sleep measures of the BISQ were acceptable: Cronbach's $\alpha = 0.73$. A variable for the average number of nighttime awakenings was created by averaging the total amount of minutes on this item over the 3- and 6-month visits ($M = 83.77$, $SD = 103.89$).

Covariates. In accordance with the standards in the field, we covaried for maternal education, breastfeeding, education, infant sleep location/co-sleeping, maternal depression,

infant sex, maternal age, marital status, prenatal tobacco use, and prenatal marijuana use in our analyses.

Results

We tested our first study hypotheses using HLM, a software for hierarchical linear modeling. HLM was chosen due to its ability to predict slope over time. HLM imputes missing values at level 1; if data were missing at level 2, the case was dropped from the analysis. To verify the assumptions of normality, we examined descriptive statistics and distribution fits for all included study variables. Mediation was tested by first examining whether the predictor predicted to the mediator and then if the mediator predicted to the outcome. If either was not true, analyses were discontinued for that mediator. If both were true, mediation analysis was planned in HLM. All analyses were performed using IBM SPSS (version 27.0; IBM Corp, 2019) and HLM (version 8.2). Statistical significance was set at a two-sided p-value of < 0.05.

Demographic and descriptive characteristics of our sample are presented in Table 1. Missing data for predictors and mediators resulted in variation of sample sizes for study analyses. In early pregnancy, all women (n = 142) completed the sleep measure. There was some missing data for the inflammatory measures at the second prenatal visit: n = 113 for CRP and n = 131 for IL6:IL10 and TNFα:IL6. At postpartum visits for the child aged 3 and 6 months, n = 110 mothers in the sample completed the child sleep questionnaire and n = 140 completed all child Bayley measures.

In HLM, all domains of the Bayley were tested with the empty model to assess for sufficient variance. The motor domain was dropped from analysis, as there was not adequate variance in the model to test for slope or intercept. The cognitive domain did not have adequate variance in the model to test for slope but had adequate variance for intercept, therefore it was

not dropped from analyses; instead, fixed effects were set for the slope. Table 2 displays the slope and intercept results for all HLM analysis. Maternal prenatal PROMIS scores were found to be negatively associated with child intercepts and slopes on the Expressive Language (Figure 1) and Social Emotional Functioning (Figure 2) domains of the Bayley from 6 to18 months postpartum.

We then assessed for mediating effects of child sleep, gestational age at birth, and maternal prenatal inflammation in the relationship between maternal sleep quality in pregnancy and her infant's cognitive development. Partial bivariate correlation analysis did not provide support for the hypothesis that infant night awakenings ($r = 0.80$, $p = 0.430$), gestational age ($r = 0.105$, $p = 0.303$), or maternal inflammatory markers (see Table 3) mediate the relationship between maternal sleep quality and cognitive development.

Discussion

To our knowledge, this study is the only known investigation in an exclusively African American sample examining the intergenerational relationship between maternal prenatal sleep quality and infant cognitive development. We found that the more sleep problems mothers experience early in pregnancy, the lower their child's cognitive development in the domains of social emotional functioning and expressive language across the first 18 months of life. Contrary to our literature review, we did not find significant results supporting our mediation analysis. However, findings from predominately White samples in this area of research may not directly generalize to African American populations, as this population exhibits unique experiences and disparities in postpartum outcomes.

Our overarching hypothesis was that maternal sleep problems in early pregnancy would predict lower cognitive development across the first 18 months of life both directly, and through

mediating factors that have been consistently supported in the literature. Our results provide partial support for this hypothesis, as we did find an intergenerational link between our predictor and outcome. This is the first study to date that has found evidence of a potential intergenerational relationship between maternal sleep and infant cognition. This significant finding suggests the importance of healthy sleep patterns in pregnant mothers to foster healthy brain development in their children.

Contrary to expectation, we did not find support for any type of relationship between the mediators (infant nighttime awakenings, inflammation, and gestational age) and maternal sleep quality. There exists lack of continuity in the literature regarding what type of infant sleep is most important to development (i.e., daytime awakenings, total duration of sleep, nighttime awakenings, etc.). Our lack of findings contributes to the uncertainty in the field of what the best measure of disrupted sleep during infancy is, with indications that variables other than nighttime awakenings may be more useful. It may also be that actigraphy or more direct measures of infant sleep would have been more reliable and may have shown associations with maternal sleep during pregnancy.

Regarding inflammation, the literature is strong connecting maternal inflammatory response during pregnancy to infant cognitive development, as cytokines may cross the blood-brain barrier of an infant in utero and disrupt brain development (Estes & McAllister, 2016; Knuesel et. al., 2014). Sleep deprivation is also commonly linked to a potential inflammatory response and therefore, surprising that this was not reflected in our results. It is possible that these findings were not replicated in our study, because African American women may experience inflammation differently than what has been observed in majority samples in the literature (Carroll et. al., 2019). Additionally, our study did not collect information on maternal

health at the time of study visits. A mother could have potentially been experiencing a cold when she came in for a blood draw, which may have confounded our results. Establishing a cytokine cut off point was difficult in this sample as well, as African American women experience higher rates of inflammation in general. Future research regarding how to measure and analyze cytokine levels, specifically for an African American sample may be useful in informing how to better assess this measure.

On the contrary, the null findings regarding gestational age at birth as a mediator was less surprising. The literature surrounding preterm birth in African American populations is both sparse and inconclusive. Pregnant women of this population exhibit lower gestational age across the board; however, it has been difficult for researchers to pinpoint why (Howell, 2018; March of Dimes Report Card, 2020). This study contributes to the literature that corroborates that there may be something else, unique to this population affecting the ability to carry full term. Future research should investigate the influence of unique environmental stressors (i.e., experiences of discrimination, neighborhood safety/noisiness, etc.) on gestational age.

This study has several limitations that we must acknowledge. The most striking being that we had a relatively small sample size. The small sample size may have caused an overall power issue, potentially driving our null findings. Additionally, as noted above, our measures of sleep were limited to self-report by the mothers. Having objective measures of sleep quality in addition to our subjective measures may have allowed us to find an intergenerational link between maternal sleep quality and infant sleep quality. This may also be why we did not discover a mediating effect with this variable, as it was the only subjective mediating variable used in the study. We encourage future research to examine child sleep quality in a more objective manner.

In the first known study to examine an intergenerational relationship between prenatal maternal sleep quality and postpartum cognitive development in an African American sample, we succeeded in identifying a relationship that has not previously been found. However, we were unable to pinpoint any mechanisms that explained relationship. This finding supports the need for more research surrounding sleep quality in an African American sample. With such limited literature dedicated to African American women, our failure to replicate corroborated findings in majority samples regarding inflammation, infant sleep quality, and gestational age calls for more within race research. We encourage future studies to further examine the unique experiences of these women with the goal of reducing the prevalence of negative outcomes for their children.

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Table 1

Sample Characteristics (N=142)

	n (% yes)	Mean (SD)	Range
Variable			
Maternal Age	142	25.29 (± 5.11)	18 - 40
Co-sleeping			
Bed Sharing at 3 or 6 months	110 (61.8%)		
Maternal Education			
Some High School	22 (15.5%)		
Graduate High School or GED	61 (43%)		
Some College or Technical School	37 (26.1%)		
Graduated College	15 (10.6%)		
Some Graduated Work or Degree	7 (4.9%)		
Infant Sleep Quality			
Nighttime Awakenings (total minutes)	110	83.77 (± 103.89)	0 - 750
Postpartum Depression			
Edinburgh Depression Scale	142	7.80 (± 5.70)	0 - 28
Prenatal Maternal Sleep			
PROMIS sleep quality score	142	21.58 (± 8.18)	8 - 40
Gestational Age	142	38.43 (± 2.24)	25 - 41.4
Infant Development Scores (average: 6, 12, 18 months)			
Bayley Cognitive	139	37.30 (± 6.95)	19 - 54
Bayley Receptive Language	139	11.97 (± 2.71)	7 – 20
Bayley Social Emotional	132	75.76 (± 13.85)	36 – 114
Bayley Expressive Language	137	12.41 (± 4.13)	4 – 28
Breastfeeding at 3 or 6 months	142 (25.7%)		
Cytokines			
CRP	113	0.78 (± 0.8)	0.03 - 4.95
IL6:IL10	131	2.95 (± 2.52)	0.10 - 14.16
TNFα:IL6	131	4.62 (± 4.21)	0.84 - 35.29
Tobacco	142 (15.5%)		
Sex (% male)	142 (53.5%)		
Marital Status	142		
Marijuana	142 (23.6%)		

Table 2

HLM Results Examining Maternal Sleep Quality and Bayley Domains of Cognitive Functioning

	Intercept: Age 18 months			Slope		
	β coefficient	t-ratio	*p* -value	β coefficient	t-ratio	*p* -value
Expressive Language	-0.119904	-2.245	**0.026***	-0.000367	-2.290	**0.024***
Receptive Language	-0.008701	-0.318	0.751	-0.000076	-0.871	0.385
Social Emotional	-0.356814	-1.993	**0.048***	-0.001857	-3.270	**0.001****
Cognitive	0.017363	0.321	0.749	-0.000032	-0.192	0.848

*$p < 0.05$; **$p < 0.01$

Table 3

Partial Correlation Results Examining Cytokines and Maternal Sleep Quality

Variable	M	SD	1	2	3
1. logCRP	-0.744	1.100			
2. logratio_IL6_IL10.1	0.745	0.873	0.315**		
3. logratio_TNFa_IL6.1	1.297	0.646	0.310**	-0.667**	
4. PROMIS	21.578	8.180	-0.005	-0.014	-0.016

*$p < 0.05$; **$p < 0.01$

Figure 1

Maternal Prenatal Sleep Quality Predicting the Slope of Performance on the Expressive Language domain of the Bayley at Age 6 to 18 Months

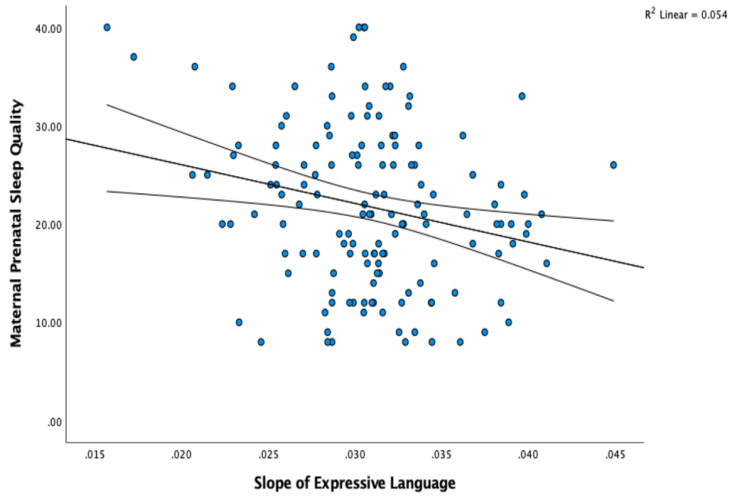

Figure 2

Maternal Prenatal Sleep Quality Predicting the Slope of Performance on the Social Emotional Functioning domain of the Bayley at Age 6 to 18 Months