Rapid #: -25979910

CROSS REF ID:	**1196437**
LENDER:	**COF (Colorado State University) :: Morgan Library**
BORROWER:	**WTU (Washington University in St. Louis) :: Olin Library**
TYPE:	Article CC:CCG
JOURNAL TITLE:	European child & adolescent psychiatry
USER JOURNAL TITLE:	European child & adolescent psychiatry
ARTICLE TITLE:	The association between maternal sleep and circadian rhythms during pregnancy and infant sleep and socioemotional outcomes.
ARTICLE AUTHOR:	Hoyniak, Caroline P
VOLUME:	34
ISSUE:	4
MONTH:	4
YEAR:	2025
PAGES:	1365-1377
ISSN:	1018-8827
OCLC #:	

Processed by RapidX: 1/20/2026 9:41:18 AM

European Child & Adolescent Psychiatry (2025) 34:1365–1377
https://doi.org/10.1007/s00787-024-02571-y

ORIGINAL CONTRIBUTION

The association between maternal sleep and circadian rhythms during pregnancy and infant sleep and socioemotional outcomes

Caroline P. Hoyniak[1] · Meghan R. Donohue[1] · Joan L. Luby[1] · Deanna M. Barch[1,2,3,4] · Peinan Zhao[5] · Christopher D. Smyser[4,6,7] · Barbara Warner[7] · Cynthia E. Rogers[1,7] · Erik D. Herzog[8] · Sarah K. England[5]

Received: 7 February 2024 / Accepted: 19 August 2024 / Published online: 24 August 2024

Abstract

Studies have established that maternal sleep and circadian rhythm disturbances during pregnancy are associated with poor prenatal and perinatal outcomes for mothers and offspring. However, little work has explored its effects on infant sleep or socioemotional outcomes. The current study examined the relationship between maternal sleep and circadian rhythm disturbances during pregnancy and infant sleep and socioemotional outcomes in a diverse sample of $N=193$ mothers and their infants (51% White; 52% Female; $M_{age}=11.95$ months). Maternal sleep and circadian rhythms during pregnancy were assessed using self-reports and actigraphy. Mothers reported on infants' sleep and socioemotional outcomes when infants were one year old. When controlling for infant sex, age, gestational age at birth, family income-to-needs ratios, and maternal depression, mothers who reported more sleep problems during pregnancy had infants with more sleep disturbances when they were one year old. Moreover, mothers who had later sleep timing (i.e., went to bed and woke up later, measured via actigraphy) during pregnancy had infants with more dysregulation (e.g., increased feeding difficulties, sensory sensitivities) and externalizing problems, and mothers with increased intra-daily variability in rest-activity rhythms (as measured via actigraphy) had infants with more externalizing problems. Findings suggest that maternal sleep and circadian rhythm disturbances during pregnancy may be a risk factor for infant sleep problems and socioemotional difficulties.

Keywords Maternal sleep · Maternal circadian rhythms · Pregnancy · Infant sleep disturbance · Infant socioemotional outcomes

✉ Caroline P. Hoyniak
choyniak@wustl.edu

1 Department of Psychiatry, Washington University School of Medicine in St. Louis, 4444 Forest Park Ave, Suite 2100, St. Louis, MO 63108, USA

2 The Program in Neuroscience, Washington University in St. Louis, St. Louis, MO, USA

3 Department of Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, MO, USA

4 Department of Radiology, Washington University School of Medicine in St. Louis, St. Louis, MO, USA

5 Department of Obstetrics and Gynecology, Washington University School of Medicine in St. Louis, St. Louis, MO, USA

6 Department of Neurology, Washington University School of Medicine in St. Louis, St. Louis, MO, USA

7 Department of Pediatrics, Washington University School of Medicine in St. Louis, St. Louis, MO, USA

8 Department of Biology, Washington University in St. Louis, St. Louis, MO, USA

Introduction

Sleep disturbances during pregnancy are common, impacting up to 62% of pregnant individuals [1]. These sleep disturbances are likely due to a combination of physical factors (e.g., discomfort, frequent urination) as well as hormonal and inflammatory changes that occur throughout pregnancy [2]. During pregnancy, individuals experience normative shifts in their sleep behaviors, including sleep onset advances (i.e., going to bed earlier), increases in daytime sleepiness, and changes in sleep architecture [3]. Sleep disturbances may be the most pronounced in the first and third trimester [2, 3], but findings have been inconsistent [4, 5], making it difficult to identify the "typical" progression of sleep disturbances across gestation. Despite this, research generally suggests that sleep disturbances during pregnancy are associated with poorer maternal and fetal outcomes, including increased risk of gestational diabetes, pre-term birth, restricted fetal growth, and increased labor length [3, 6]. These findings parallel a body of literature that has established sleep and circadian rhythm disturbances as a risk factor for a variety of maladaptive health and behavioral outcomes across the lifespan, including increased risk for cardiovascular disease [7], poorer immune functioning [8], and psychopathology, such as depression [9]. Sleep disturbances include both diagnosable sleep disorders (e.g., insomnia, parasomnias, obstructive sleep apnea), as well as variability in sleep parameters including short sleep durations, late sleep timing, and variability in sleep schedules from day-to-day. The sleep–wake schedule is governed, in part, by circadian rhythms, the approximately 24-h biological rhythms generated by the circadian clock (governed by the central pacemaker, the suprachiasmatic nucleus) that optimize the timing of various biochemical and physiological processes. Circadian disruption (often referred to as chronodisruption or circadian misalignment) can occur when an individual's circadian rhythm becomes misaligned with the local environment (as determined by light/dark cycles). Circadian disruptions can be quantified in a number of ways, including examining the rhythmicity of core temperature, circadian-controlled hormones (e.g., melatonin and cortisol), or by examining daily variability in activity patterns and sleep midpoints (i.e., the point halfway between sleep onset and offset) across days [10, 11]. Although a fairly robust literature has explored sleep disturbances during pregnancy and the effects on birth outcomes, substantially less research has focused on the relationship of sleep disturbances during pregnancy to infant outcomes. In addition, to our knowledge, none of this research has focused explicitly on the effects of circadian rhythm disruption during pregnancy on offspring. To fill this gap in the literature, the current study examined the effects of sleep and circadian rhythm disturbances during pregnancy on infant outcomes, focusing specifically on sleep and socioemotional development at age one.

Evidence from both animal models and human studies suggests that circadian rhythms synchronize between mother and fetus, likely through hormonal signaling via melatonin, dopamine, and corticosterone, which can cross the placenta and bind to receptors on fetal tissue [12–14]. Such synchronization of maternal and fetal circadian rhythms may underlie the negative effects of irregular maternal sleep–wake cycles on fetal development [15]. In rodents, in utero exposure to sleep and circadian disruption programs a range of endocrine, circadian, behavioral, and metabolic effects in offspring that have been observed to last into adulthood [16]. Sleep disruption in pregnant dams, including REM sleep deprivation or sleep restriction, resulted in brain immaturity in pups at birth [17, 18] and altered behavioral development in offspring [19]. Specifically, Peng et al. [19] identified that adult offspring of pregnant dams that were exposed to sleep deprivation demonstrated increased depression and anxiety-related behaviors.

Although relatively little human research exists, available data suggest an association between prenatal sleep disturbances and subsequent infant/child sleep disturbances [20–24]. In a relatively small study, Ciciolla et al. [20] demonstrated an association between mother-reported insomnia symptoms during the third trimester and infant sleep difficulties 6-weeks postpartum. Similarly, in a large epidemiological study, mother-reported sleep disturbances (e.g., poor sleep quality, late bedtimes) and short sleep durations during pregnancy were associated with increased infant sleep disturbances at one month of age (e.g., frequent nighttime awakenings, sleeping more during the day than at night) [22] and at one year of age (e.g., late bedtimes, frequent nighttime awakenings, and shorter sleep durations) [21]. Lahti-Pulkkinen et al. [23] demonstrated that mothers' self-reported sleepiness during the second trimester was associated with increased sleep problems in 2-to 6-year-old children. Similarly, Lugo-Candelas [24] found that maternal, self-reported sleep disturbances in second trimester (but not in first or third trimester) were associated with increased parent-reported child sleep problems at age four years. Longer maternal sleep during pregnancy has also been associated with infant sleep problems: Newland et al. [25] found that mothers who reported longer sleep durations during pregnancy had infants with poorer sleep efficiency and more sleep variability at 8 months (measured via actigraphy). These early findings highlight a potential association between optimal prenatal maternal sleep durations (i.e., neither too short nor too long) and infant sleep outcomes.

Even less research has explored whether human maternal sleep and circadian rhythm disturbances during pregnancy relate to child socioemotional outcomes. Emerging evidence indicates that maternal sleep and circadian disturbances during pregnancy are associated with structural brain alterations in neonates [26, 27], and these brain alterations may be a possible pathway through which later socioemotional development is affected. In addition, infants of mothers who reported having poor quality sleep and short sleep durations during pregnancy demonstrated increased irritability at one month of age [22]. Adler et al. [1] found that mothers who reported insomnia during pregnancy were more likely to report their children had deficits in self-regulation and social interaction at age two. In this study, the association between prenatal maternal insomnia and child socioemotional development was mediated by both postnatal maternal insomnia and depression [1], suggesting a pathway through which prenatal sleep problems have downstream effects on child development via continued maternal sleep and/or mood disturbances postpartum. Lahti-Pulkkinen et al. [23] demonstrated that mother-reported sleepiness during pregnancy was associated with increased mother-reported child externalizing and internalizing problems in their 2-to 6-year-old children. Similarly, shorter sleep durations and poorer sleep quality, as reported by mothers during their second trimester of pregnancy, and increased overall sleep disturbances reported in the first trimester of pregnancy were found to be associated with elevated, parent-reported attention deficit hyperactivity disorder symptoms in offspring at age four [24]. Notably, none of these previous studies use an objective measure of maternal sleep such as actigraphy or measure circadian rhythm disruption.

Research suggests a strong influence of poverty on maternal sleep during pregnancy [28], infant sleep [29], and infant socioemotional development [30]. There are multiple studies examining sleep health disparities across populations, including pregnant individuals and infants. Research suggests that pregnant individuals living in poverty are at increased risk for sleep disturbances [28, 31], potentially through a combination of environmental (e.g., noise and light pollution, non-ideal work schedules) and psychosocial (e.g., increased anxiety, role overload) factors. Similarly, infants and young children living in poverty are more likely to experience sleep disturbances, including shorter sleep durations and more nighttime awakenings [32–34]. Finally, a long tradition of research has demonstrated an effect of poverty on socioemotional development in early childhood [35, 36]. However, despite well-established associations between these constructs, prior research exploring the effect of maternal prenatal sleep on infant sleep and socioemotional development has not considered the effect of poverty.

In sum, relatively little research has explored potential associations between maternal sleep during pregnancy and infant sleep and socioemotional functioning. Moreover, despite that circadian rhythm disruption has been found to be associated with mood disorders [37] and that maternal and fetal circadian rhythms have been found to synchronize during pregnancy [12, 13], no study to date has examined effects of maternal prenatal circadian rhythm disruption on infants' sleep or socioemotional outcomes. To address these gaps in the literature, the current study examined how maternal sleep and circadian rhythm disruption during pregnancy was associated with infant sleep and socioemotional outcomes using objective (i.e., actigraphy) and subjective (i.e., self-report) measures of maternal sleep, allowing us to explore both sleep and circadian rhythms. We tested the hypothesis that mothers with sleep and circadian rhythm disturbances during pregnancy would have infants with more sleep problems and more socioemotional difficulties (e.g., behavior problems, dysregulation) at one year of age. To address the possibility that poverty accounted for any observed associations between prenatal maternal sleep and infant outcomes, we re-examined associations controlling for family income-to-needs ratios. Given some inconsistencies in the literature examining the progression of sleep disturbances across pregnancy and that few previous studies have explored trimester-specific effects of maternal prenatal sleep on offspring outcomes, we did not have a specific hypothesis about how associations between maternal sleep and offspring outcomes may differ across trimesters. As such, we focused our analysis on average sleep across pregnancy and examined trimester-specific associations in an exploratory analysis.

Methods

Participants

Participants in the current study included mothers-infant dyads enrolled in two large studies conducted at Washington University School of Medicine: the 1000 Women Cohort (1KWC) and the Early Life Adversity and Biological Embedding (eLABE) study. In the 1KWC, 1220 women with a live birth were assessed throughout their pregnancy, providing detailed survey data, sleep and circadian data, and birth outcome data [38]. Mothers were recruited during the first trimester of pregnancy and followed throughout pregnancy and delivery. Exclusion criteria for the 1KWC included: multiple gestations, diagnosed infections known to cause congenital disease (e.g., syphilis), and maternal alcohol or drug use during pregnancy (excluding tobacco and marijuana). In their third trimester, a subsample ($n = 399$) of the participants in the 1KWC were recruited to participate in the eLABE study, an ongoing, longitudinal study focused on child development from birth through early childhood.

Of the 399 mother-infant dyads who participated in the eLABE study, 244 mothers provided usable actigraphy data at some point during their pregnancy. Of the 244 dyads with usable actigraphy data, 193 dyads participated in sleep and socioemotional data collection at the Year 1 assessment and met our inclusion criteria: birth weight > 2000 g and no Neonatal Intensive Care Unit admission > 7 days. For the 206 mother-infant dyads who could *not* be included in the current analysis (e.g., because they did not provide maternal actigraphy or infant socioemotional data), mothers were more likely to be Black ($t = -7.06$, $p < 0.001$), to have a lower income-to-needs ratio ($t = 7.82$, $p < 0.001$), to be younger at the time of delivery ($t = 5.15$, $p < 0.001$), to have a higher BMI ($t = -2.45$, $p < 0.001$), to have a higher parity ($t = -3.40$, $p < 0.001$), and to be experiencing more depression symptoms during pregnancy ($-2.42 < t\text{'s} < -1.99$, $p < 0.05$). There were no differences between included and excluded dyads in terms of the child's sex, the child's birthweight, the child's gestational age (GA) at delivery, or mother's depression symptoms at the age one assessment.

In the final sample of 193 mother-infant dyads, infants were delivered at a mean GA of 38.01 weeks and mothers ranged in age from 18.77 to 41.80 years ($M_{age} = 29.83$ years; $SD_{age} = 5.47$ years) at delivery. Infants (52% female) were

51% White, 47% Black or African American, 2% other, and families reported an average income-to-needs ratio of 3.84 ($SD = 3.09$, range = 0.44–12.04). At the Year 1 assessment, infants ranged in age from 11 to 19-months ($M_{age} = 11.95$ months; $SD_{age} = 1.21$ months; some variability in age at the year 1 assessment was due to delayed visits caused by the COVID-19 pandemic). The 1KWC and eLABE studies were approved by the Washington University Human Research Protection Office. Informed consent was obtained for each mother and subsequent parental informed consent was obtained for each infant prior to participation. Data were collected with consent forms that allow data sharing. De-identified data have been deposited in the NIMH Data Archive.

Procedures

Mothers were asked to wear the actigraphs continuously for at least two weeks during each trimester of pregnancy. Mother reports about the child's socioemotional development and sleep patterns were collected the year 1 assessment.

Measures

Maternal sleep and circadian rhythms during pregnancy

Actigraphy data. Maternal sleep and circadian rhythms were measured using actigraphs, worn for two weeks during the first, second, and third trimesters of pregnancy. Actigraphs contain an accelerometer that measures minute-by-minute motor activity that can be used to estimate sleep and wake patterns. Actigraphy watches, the MotionWatch 8 (CamNtech Ltd., Cambridge, U.K.), were worn by mothers on their non-dominant wrist. The MotionWatch 8 contains a piezoelectric accelerometer with a sensitivity greater than 0.05 g, a sampling frequency of 32 Hz, and bandpass filter of 3–11 Hz. The actigraphy data were collected in 1-min epochs, processed using MotionWare software 2.5 (CamNtech Ltd), and analyzed using a high-throughout, automated method to derive the sleeping time indexes [39].

In our analysis, we focused on two sleep metrics and three circadian metrics derived from the actigraphy data. The two sleep metrics of interest included: (1) sleep duration –length of time between sleep onset and offset and (2) sleep midpoint—the time equidistant between sleep onset and offset (often used as a proxy measure of chronotype). The three circadian metrics included: (1) composite phase deviation (CPD), (2) interdaily stability (IS), and (3) intradaily variability (IV). CPD is a metric of circadian misalignment that examines sleep midpoints, relative to habitual sleep midpoints (i.e., the individual's chronotype) and sleep midpoints from the prior day [11], with higher scores indicating increased misalignment. IS and IV are common, nonparametric measures of circadian disruption derived from activity data that provide information about the stability and fragmentation of an individual's 24-h activity rhythms. Activity rhythms are often used as indicators of circadian organization, with the added benefit of enabling the examination of circadian patterns across multiple days or weeks of data. IS provides an index of how stable activity rhythms are over multiple days, quantifying similarities in activity profiles across multiple days [40, 41]. IV provides an index of how fragmented an individual's rhythm is within a day, quantifying the rate of shifts between rest and activity, with higher scores reflecting increased fragmentation [40].

Although all mothers were asked to provide actigraphy data during each trimester, the number of data points collected from this sample varied, with 83 mothers providing data during the first trimester, 127 mothers during the second trimester, and 134 mothers during the third trimester, with 244 mothers providing actigraphy data during at least one trimester. Our primary analyses used the mean of each sleep and circadian metric collected over all three trimesters. However, as some previous research has identified trimester-specific effects of maternal sleep disturbances on offspring outcomes [19, 24, 27], so we have opted to include a trimester-specific, exploratory analysis in Supplemental Appendix A (Supplemental Tables S1 and S2).

Self-reported maternal sleep problems. Maternal sleep problems were measured using the Pittsburgh Sleep Quality Index (PSQI [42]) collected during each trimester of pregnancy [43]. The PSQI is a well-validated, self-report measure for adults that assesses a variety of domains of sleep disturbance, including sleep quality, sleep duration, use of sleeping medication, etc., which are summed to yield one global score, with higher scores reflecting the presence of more sleep problems. Cronbach's α values for the PSQI global score were 0.68, 0.65, and 0.67 for the first, second, and third trimester, respectively. Analyses used a single index of self-reported maternal sleep problems created by averaging PSQI global scores across trimesters.

Infant socioemotional outcomes

Parent-reported socioemotional outcomes. Infant socioemotional outcomes were measured using the Infant–Toddler Social and Emotional Assessment (ITSEA), a validated, parent-report of child behavior for children ages 12-to 36-months [44]. The ITSEA contains 166 items rated on a 3-point scale, from (0) *not true/rarely*, (1) *somewhat true/sometimes*, and (2) *very true/often*. Analyses examined three domains, including (1) externalizing problems—comprised of the activity/impulsivity, aggression/defiance, and peer aggression subscales, (2) internalizing problems—comprised of the depression/withdrawal, general anxiety, separation distress, and inhibition to novelty subscales, and (3) dysregulation—comprised of the sleep, negative emotionality, eating problems, and sensory sensitivity subscales. Items in each subscale were averaged to form the subscale score, and subscale scores were averaged to form the overall domain score. T-scores were used for the externalizing and internalizing problems domains. As the dysregulation domain score also included the sleep subscale, we opted to create a *modified dysregulation score* that did not include the sleep subscale. Higher scores on each domain indicate greater externalizing and internalizing problems and dysregulation.

Infant sleep disturbances

Infant sleep disturbances were measured using the sleep subscale on the ITSEA. The sleep subscale contains five items assessing general sleep disturbances, including (1) "wakes up at night and needs help to fall asleep again", (2) "has trouble falling asleep or staying asleep", (3) "sleeps through the night", (4) "must be held to go to sleep", and (5) "wants to sleep in someone else's room/bed." Scores on these five items were averaged to reflect an index of general infant sleep disturbances, with higher scores indicating greater sleep disturbances.

Analytic plan

We began by using bivariate correlations to examine whether there were significant associations between study variables of interest. Next, we used linear regression to test our hypotheses about associations between maternal prenatal sleep and circadian rhythms and infant sleep disturbances when accounting for covariates, including infant sex, GA at birth, and child age at the Year 1 assessment.

Next, we used linear regression to examine the association between maternal prenatal sleep and circadian rhythms and child socioemotional outcomes (i.e., internalizing problems, externalizing problems, and dysregulation). As we used t-scores to measure internalizing and externalizing problems (standardized based on age and sex), only GA was included as a covariate in these models. Models examining the dysregulation scale included all three covariates, as this score was a modified scale, not a T-score. Family-wise error was corrected for using the Benjamini–Hochberg False Discovery Rate correction [45].

Finally, to explore if these associations remained significant when controlling for additional, potentially relevant covariates, including family poverty and maternal depression, we re-examined significant models controlling for maternal depression and family income-to-needs ratios at the Year 1 assessment. Maternal depression was measured using the total score on the Edinburgh Postnatal Depression Scale [46], a self-report measure of maternal depression, where higher scores indicate increased depression symptoms. All analyses were conducted in R Version 4.3.1 [47].

Results

Sample descriptive statistics for the maternal sleep and circadian metrics, overall and by trimester, are presented in Table 1. In this cohort of 193 pregnant women, the actigraphy-based sleep and circadian metrics did not vary by trimester (one-way ANOVAs, $0.15 < F$'s < 2.41, p's > 0.05). However, we did see differences between subjectively-reported sleep differences across trimesters (one-way ANOVA, $F = 5.54$, $p < 0.01$). Post-hoc analysis using Tukey's multiple comparisons of means indicated a significant difference between subjective sleep disturbances in trimester 1 ($M = 6.56$) and subjective sleep disturbances in trimester 2 ($M = 5.50$). Sample descriptive statistics for the infant outcome variables are also presented in Table 1. The infants in our sample showed a range of sleep, externalizing, internalizing and dysregulation problems, similar to levels expected in a community sample.

Association between prenatal maternal sleep and infant sleep

Bivariate correlations between prenatal maternal sleep and circadian rhythms and infant sleep are presented in Table 2. There were significant correlations between prenatal maternal sleep midpoints, CPD, and sleep problems and infant sleep problems, such that mothers who had later sleep midpoints, more circadian misalignment (i.e., higher CPD values), and reported experiencing more sleep problems had infants with more sleep problems at Year 1. Each of these associations remained significant when controlling for infant sex, GA at birth, and age at the Year 1 assessment (Table 3).

When re-examining significant associations controlling for maternal depression symptoms at the Year 1 assessment, all of the significant findings remained (see Supplemental Table S3). Next, when re-examining significant associations controlling for family income-to-needs ratio, only the association between maternal self-reported sleep problems and infant sleep remained significant (see Supplemental Table S3), such that mothers who reported experiencing more prenatal sleep problems had infants with more sleep disturbances.

Association between prenatal maternal sleep and infant socioemotional outcomes

Bivariate correlations between prenatal maternal sleep and circadian rhythms and infant socioemotional development

Table 1 Descriptive statistics for variables included in analysis

	Variable	Mean	*SD*	Minimum	Maximum
Maternal prenatal sleep (average across trimesters)	Sleep duration (hours)	8.89	1.10	5.10	12.08
	Sleep midpoint (HH:MM)	03:03	1:12	00:12	06:35
	Composite phase deviation	3.83	2.55	0.48	12.47
	Intradaily variability	0.61	0.11	0.32	1.39
	Interdaily stability	0.43	0.12	0.13	0.75
	Sleep problems (PSQI)	6.41	2.79	1.00	16.00
Maternal prenatal sleep trimester 1	Sleep duration (hours)	9.12	1.18	5.65	12.08
	Sleep midpoint (HH:MM)	02:49	01:13	11:22	06:43
	Composite phase deviation	3.80	2.87	0.40	13.68
	Intradaily variability	0.59	0.10	0.40	0.95
	Interdaily stability	0.44	0.13	0.11	0.67
	Sleep problems (PSQI)	6.70	3.38	1.00	17.00
Maternal prenatal sleep trimester 2	Sleep duration (hours)	8.86	1.19	5.10	13.00
	Sleep midpoint (HH:MM)	03:07	01:14	22:56	06:35
	Composite phase deviation	3.89	3.44	0.49	18.80
	Intradaily variability	0.61	0.16	0.35	1.75
	Interdaily stability	0.44	0.15	0.08	0.76
	Sleep problems (PSQI)	5.63	2.88	1.00	17.00
Maternal prenatal sleep trimester 3	Sleep duration (hours)	8.92	1.03	6.01	12.13
	Sleep midpoint (HH:MM)	03:00	01:13	00:52	06:58
	Composite phase deviation	3.58	3.09	0.46	16.20
	Intradaily variability	0.61	0.15	0.29	1.80
	Interdaily stability	0.44	0.14	0.06	0.76
	Sleep problems (PSQI)	6.54	3.09	1.00	18.00
Infant outcome variables (year 1)	Sleep disturbances	0.49	0.42	0.00	2.00
	Externalizing problems (T-score)	51.97	10.16	36.00	83.00
	Internalizing problems (T-score)	46.77	10.04	28.00	79.00
	Dysregulation	0.38	0.20	0.00	1.08
Covariates	Gestational age at birth	38.01	2.00	28.00	41.00
	Family income-to-needs ratio	3.62	3.09	0.38	12.15
	Maternal depression (year 1)	5.94	4.53	0.00	19.00

Except for the measure of Maternal Sleep Problems on the PSQI, all other maternal sleep and circadian metrics were derived from actigraphy data

HH:MM 24-h time, *PSQI* Pittsburgh Sleep Quality Index

are presented in Table 2. Broadly, mothers who had later sleep midpoints and more circadian rhythm disturbances (i.e., heightened circadian misalignment [higher CPD values], more variability in their rest-activity rhythms throughout the day [higher IV values], and decreased stability of rest-activity rhythms between days [lower IS values]) during pregnancy had infants with greater externalizing problems. Mothers with more variability in their rest-activity rhythms throughout the day (i.e., higher IV values) had infants with greater internalizing problems. Mothers with later sleep midpoints, increased circadian misalignment (i.e., higher CPD values), and who reported more sleep problems had infants with more dysregulation.

Results of the regression analysis examining the association between maternal prenatal sleep and circadian rhythms and parent-reported socioemotional outcomes, controlling for relevant covariates, are presented in Table 4. Findings indicated that maternal sleep midpoints, CPD, and IV were associated with infant externalizing problems, such that pregnant mothers who had later sleep schedules, evidenced circadian misalignment (i.e., higher CPD values), and evidenced more variability in their rest-activity rhythms throughout the day (i.e., higher IV values), had infants with greater externalizing problems at age one. In addition, maternal sleep midpoints and self-reported sleep problems were associated with infant

Table 2 Bivariate correlations between variables included in analysis[a]

Variables	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Maternal sleep duration	1.00												
2. Maternal sleep midpoint	– 0.14*	1.00											
3. Maternal CPD	– 0.20*	0.35**	1.00										
4. Maternal IV	– 0.04	0.06	0.34**	1.00									
5. Maternal IS	0.22**	– 0.34**	– 0.61**	– 0.36**	1.00								
6. Maternal sleep problems (PSQI)	– 0.04	0.28**	0.25**	0.11	– 0.23**	1.00							
7. Infant sleep disturbances	0.08	0.20**	0.18*	0.05	– 0.11	0.25**	1.00						
8. Infant externalizing problems	– 0.02	0.25**	0.26**	0.24**	– 0.15*	0.11	0.27**	1.00					
9. Infant internalizing problems	0.12	0.13	0.14	0.16*	– 0.10	0.14	0.19*	0.44**	1.00				
10. Infant dysregulation	0.08	0.22**	0.17*	0.11	– 0.10	0.19**	0.34**	0.52**	0.62**	1.00			
11. Infant age	– 0.03	0.03	0.06	– 0.02	– 0.03	0.09	– 0.07	0.06	0.09	0.08	1.00		
12. Infant sex	– 0.05	– 0.06	0.02	0.02	0.11	0.04	– 0.05	– 0.16*	– 0.22**	– 0.14*	– 0.03	1.00	
13. Family income-to-needs ratio	0.14	– 0.45**	– 0.52**	– 0.16*	0.45**	– 0.41**	– 0.21**	– 0.26**	– 0.14	– 0.13	0.004	0.001	1.00
14. Maternal depression	0.001	0.16*	0.07	– 0.06	– 0.04	0.28**	0.18*	0.11	0.26**	0.27**	0.13	– 0.09	– 0.06

CPD Composite Phase Deviation, *IV* Intradaily Variability, *IS* Interdaily Stability, *PSQI* Pittsburgh Sleep Quality Index

[a]Maternal sleep variables reflect average values across trimesters

*$p < .05$

**$p < .01$

Table 3 Linear regressions between infant sleep and average prenatal maternal sleep and circadian metrics controlling for infant sex, gestational age at birth, and child age at the year 1 assessment

Infant outcome	Maternal sleep predictor	β	t	p	FDR-p
Infant sleep disturbances	Sleep duration	0.09	1.19	0.24	0.28
Infant sleep disturbances	Sleep midpoint	0.19	2.66	0.01	0.02
Infant sleep disturbances	CPD	0.18	2.48	0.01	0.02
Infant sleep disturbances	IV	0.05	0.63	0.53	0.53
Infant sleep disturbances	IS	− 0.10	− 1.33	0.18	0.27
Infant sleep disturbances	Sleep problems	0.25	3.45	< 0.001	< 0.001

CPD composite phase deviation, *IV* intradaily variability, *IS* interdaily stability

Table 4 Linear regressions between infant socioemotional outcomes and average prenatal maternal sleep and circadian metrics controlling for infant sex, age at the Year 1 assessment, and gestational age at birth

Infant outcome	Maternal sleep predictor	β	t	p	FDR-p
Infant externalizing problems	Sleep duration	– 0.01	– 0.08	0.93	0.93
	Sleep midpoint	0.24	3.41	< 0.001	< 0.001
	CPD	0.25	3.50	< 0.001	< 0.001
	IV	0.23	3.20	< 0.001	< 0.001
	IS	– 0.14	– 1.89	0.06	0.09
	Sleep problems	0.10	1.36	0.18	0.22
Infant internalizing problems	Sleep duration	0.13	1.78	0.08	0.11
	Sleep midpoint	0.12	1.70	0.09	0.11
	CPD	0.13	1.78	0.08	0.11
	IV	0.15	2.00	0.05	0.11
	IS	– 0.09	– 1.17	0.24	0.24
	Sleep problems	0.13	1.74	0.08	0.11
Infant dysregulation (modified)	Sleep duration	0.09	1.26	0.21	0.25
	Sleep midpoint	0.20	2.80	0.01	0.03
	CPD	0.16	2.23	0.03	0.06
	IV	0.10	1.39	0.17	0.25
	IS	– 0.07	– 0.91	0.36	0.36
	Sleep problems	0.20	2.80	0.01	0.03

The modified dysregulation outcome is a raw score, so these regression models additionally controlled for child age and sex

CPD composite phase deviation, *IV* intradaily variability, *IS* interdaily stability, externalizing and internalizing problems are t-scored, accounting for child age and sex

dysregulation, such that mothers who went to sleep later and reported more sleep problems had infants who evidenced increased dysregulation.

When re-examining significant associations controlling for maternal depression symptoms at the Year 1 assessment, all of the significant findings remained, except for the association between maternal self-reported sleep problems and infant dysregulation (see Supplemental Table S4), which was likely attributable to the large association between maternal depression and infant dysregulation ($r = 0.27$, $p < 0.001$). Next, when controlling for family income-to-needs ratios, all significant associations held, except for the associations between maternal CPD and externalizing problems and between maternal sleep problems and dysregulation (Supplemental Table S4).

Exploratory analysis: trimester-specific effects of maternal sleep

The exploratory, trimester-specific analysis is included Supplemental Appendix A (Supplemental Tables S1 and S2). When we examined maternal sleep in each trimester separately, we found little evidence of consistent trimester-specific effects of exposure to maternal sleep and circadian rhythm disruption on infant sleep and socioemotional outcomes (Supplemental Tables S1 and S2).

Discussion

Using both objective and subjective measures of sleep in mothers, the current study is the first to examine the association between maternal sleep and circadian rhythms during pregnancy and sleep and socioemotional outcomes in one-year-old infants. We found that mothers who exhibited more prenatal sleep and circadian rhythm disturbances had infants with more sleep problems and poorer socioemotional outcomes. Beyond the association between maternal prenatal sleep problems and infant dysregulation at age one, all our findings held when controlling for maternal depression symptoms, suggesting that the association between mother and infant sleep and socioemotional development exists above and beyond the effects of concurrent maternal mood symptoms on reports of infant sleep and socioemotional development. When controlling for family income-to-needs ratios, several findings were no longer significant, including the association between maternal sleep midpoints and infant sleep, the association between maternal circadian misalignment and infant sleep and externalizing problems, and the association between maternal sleep problems and infant dysregulation. This highlights the complex relationship between poverty—which has a strong effect on maternal sleep during pregnancy [26], infant sleep [27], and infant socioemotional development [28]— and the pathway from maternal sleep disturbances and offspring outcomes.

Later sleep midpoints during pregnancy, as measured using actigraphy, were associated with greater infant externalizing problems and dysregulation (e.g., greater feeding difficulties, sensory sensitivities, and negative emotionality). Sleep midpoints reflect the timing of an individual's daily sleep schedule, providing a proxy measure of their chronotype. Later sleep midpoints reflect a later ("evening") chronotype. Research suggests that evening chronotypes experience increased risk for mood disorders, impulsivity, and substance use [48, 49]. An evening chronotype may be especially problematic during the first trimester when pregnant individuals tend to shift to an earlier chronotype, potentially due to changes in hormones [50]. The maintenance of a late chronotype during pregnancy may reflect highly disturbed sleep, which might have downstream consequences for offspring. Although there has been some research on the impact of chronotype on pregnancy outcomes (e.g. [51],) additional research on the association between chronotype during pregnancy and offspring outcomes is clearly needed.

Additionally, we found that increased intradaily variability (IV) in rest-activity rhythms, as measured via actigraphy, was associated with greater infant externalizing problems. IV is index of fragmentation of rest-activity rhythms within a day, quantifying the rate of switches between rest and activity. Higher IV values can reflect less consolidated activity and sleep bouts, and more daytime naps or nighttime activity. Fragmentation of activity rhythms has been associated with mortality and increased risk for depression, obesity, and cognitive decline in aging populations [52, 53]. However, little research has explicitly focused on circadian rhythm fragmentation during pregnancy, but our findings indicate potential consequences of fragmentation of activity rhythms during pregnancy and offspring outcomes.

We also found an association between subjective, prenatal sleep problems, based on mother report, and infant sleep and dysregulated behaviors. Mothers who reported more prenatal sleep problems had infants with more sleep disturbances and more dysregulated behavior at one year of age. Several additional significant associations between prenatal maternal sleep and infant sleep and socioemotional outcomes emerged but did not survive once we controlled for family poverty. The fact that poverty accounted for some of the association between maternal sleep and infant outcomes was unsurprising given demonstrated robust associations between poverty and maternal prenatal sleep, infant sleep, and infant socioemotional development [28, 29, 31–36]. Poverty has been shown to impact both maternal and child sleep in a variety of ways, through the effects of both environmental (e.g., noise and light pollution, increased neighborhood crime, and non-ideal work schedules) and psychosocial (e.g., increased anxiety, role overload) factors. These factors likely have both direct (e.g., household crowding leads to directly to disturbed sleep) and indirect (e.g., increased neighborhood crime leads to increased pre-sleep anxiety which in turn disturbs sleep) effects on child sleep. Because of this robust association, family poverty likely plays an important role in the association between prenatal maternal sleep and infant outcomes, especially infant sleep outcomes, and additional research exploring the nature of this association is warranted.

Our findings add to a small, but growing literature linking maternal prenatal sleep disturbances to infant sleep disturbances [20–23] and child socioemotional outcomes [1, 21, 23] and extend these findings in important ways. Perhaps most critically, our study is the first to document associations between maternal prenatal sleep disturbances and child sleep and socioemotional outcomes using an objective measure of sleep disturbance (actigraphy) and including measures of circadian rhythm disruption. Next, whereas many prior studies have demonstrated effects of maternal prenatal sleep disturbances on infant outcomes in the early postnatal period (e.g., a few weeks to months after birth), the current study extends these findings to more distal outcomes when infants were approximately one-year-old, when daily sleep–wake rhythms have become more stabilized [4]. Second, with the exception of Lahti-Pulkkinen et al. [23] and Lugo-Candelas et al. [24], our study is one of the only investigations to assess a variety of socioemotional domains that map onto symptoms of later psychiatric disorders. Finally, our study demonstrates associations between maternal prenatal sleep disturbances and infant outcomes in a larger and more racially and socioeconomically diverse sample than is typically studied, increasing the generalizability of findings.

There are several possible mechanisms through which maternal prenatal sleep might be associated with infant sleep and socioemotional disturbances. Circadian rhythms have been found to synchronize between mother and fetus [12–14], and irregular maternal sleep–wake cycles may disrupt the synchronization of circadian rhythms between mother and fetus, in turn negatively impacting fetal neurodevelopment [15]. Altered brain development may also be a mechanism through which maternal prenatal sleep is associated with poorer infant outcomes; indeed, emerging evidence indicates that maternal sleep and circadian disturbances during pregnancy are associated with structural brain alterations in neonates [26, 27]. Environmental influences such as maternal stress, depression, and/or maternal postnatal sleep disturbances may also help explain these associations [54]. Researchers have proposed a bidirectional relationship between sleep problems and the stress response system, whereby stress exposure can lead to sleep disturbances, and sleep disturbances can, in turn, increase stress reactivity [55]. Increased maternal stress during pregnancy, which might be both caused and exacerbated by sleep disruptions, is a possible mechanism for long-term developmental effects in offspring, including sleep disruptions [56] and psychopathology [57]. It is also possible that maternal sleep problems during pregnancy continue postpartum and impact parenting and the mother–child relationship [58, 59]. Mothers with sleep problems may show increased levels of exhaustion and subsequently be less emotionally available to their child, provide lower levels of linguistic and cognitive stimulation, or be less likely to institute healthy child sleep habits, leading to poorer offspring socioemotional development. In addition, prenatal sleep disturbances have been previously associated with shorter GA [6], which is also associated with infant sleep disturbances [60], suggesting a pathway from maternal to infant sleep disturbances through early birth. Future research should further elucidate the extent to which the current findings reflect prenatal programming of the developing fetal nervous system versus the influence of features of the postnatal environment.

Our findings add to mounting evidence that prenatal maternal sleep disturbances are associated with a host of poor fetal and infant outcomes, also including restricted fetal growth [6], preterm birth [3], and altered neonatal brain volumes [26, 27]. This literature suggests that improving maternal sleep during pregnancy could be a useful target for interventions aimed at reducing maternal and fetal risk for negative outcomes. Interventions, such as providing psychoeducation about the importance of sleep during pregnancy and connecting pregnant individuals with sleep disturbances with evidence-based sleep interventions, could mitigate maternal and infant risks. Finally, our findings suggest multiple, specific sleep characteristics—including sleep and circadian timing—to target in such interventions. For example, chronotherapies that directly affect circadian rhythms, such as bright light therapy, wake therapy, and social rhythms therapy are effective behavioral interventions with few to no side effects [61] that could be delivered to pregnant individuals experiencing sleep disturbances.

This study also had limitations. Our measure of infant sleep relied on a relatively brief parent-report, rather than an objective measure of infant sleep. Although in the future we hope to use more objective measures of sleep during infancy and childhood to further characterize the association between prenatal sleep and circadian disruptions and child sleep, the current study is an important first step to establishing this association. Moreover, although the sample size in this study was larger than many published studies of associations between maternal prenatal sleep and infant outcomes, only a subset of women with actigraphy data were recruited into the eLABE study that examined infant outcomes. A major limitation of the current study was the large amount of missingness in the actigraphy data collected at each trimester. Although we did not see evidence for consistent and coherent trimester-specific effects across sleep measures in our exploratory analysis, this could be attributed to limitations in the sample size of our actigraphy data rather than a true lack of trimester-specific effects. Given that prior research has found evidence of varied effects of sleep disturbances across trimesters on offspring outcomes [19, 24, 27], future larger studies examining trimester-specific effects of prenatal maternal sleep disruptions on offspring sleep and socio-emotional outcomes will be critical. In addition, despite large efforts to retain subjects in the study, mothers who were not able to be included in our analysis due to missing actigraphy data during pregnancy or missing infant data at age one year were more likely to be living in poverty, to be younger, to be Black, and to be experiencing prenatal depression symptoms. However, this missingness likely reduced our detectable effect size, rather than causing or inflating spurious correlations. Our significant findings, in the presence of this missingness, underscore the strength of the association between maternal prenatal sleep and infant outcomes. Finally, we did not assess mother sleep in the first year of life—and thus it is possible that maternal postnatal sleep disturbances are a primary driver of observed effects. Future research should consider how maternal postnatal sleep affects the association between maternal prenatal sleep and infant outcomes.

Overall, we found that maternal sleep and circadian rhythm disturbances during pregnancy were associated with infant sleep disturbances and were also associated with infant socioemotional disturbances even when controlling for infant sleep disturbances. Our findings suggests that maternal prenatal sleep and circadian rhythm disturbances may be a risk factor for infant sleep problems and socioemotional disturbances and may be an important target of preventive interventions during pregnancy.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00787-024-02571-y.

Author contributions J.L., D.B., C.D., B.W., C.R., E.H., and S.E. were involved in conceptualization, supervision, and funding acquisition. C.H. and P.Z. were involved in formal analysis and visualization. C.H. and M.D. wrote the main manuscript text. All authors reviewed the manuscript.

Funding This work was supported by a research grant from the March of Dimes Foundation (to E.D.H, S.K.E), NIH grant: R01 MH11388 (PIs: C.D.S., B.W., and J.L.L.), and institutional support from St. Louis Children's Hospital, Barnes-Jewish Hospital, and the Washington University in St. Louis Department of Obstetrics and Gynecology. C.P.H.'s work was supported by NIH grant: K23 MH127305. M.R.D.'s work was supported by NIH grant: K23 MH125023-03.

Data availability De-identified data have been deposited in the NIMH Data Archive.

Declarations

Conflict of interest The authors have no conflicts of interest to report

References

1. Adler I, Weidner K, Eberhard-Gran M, Garthus-Niegel S (2020) The impact of maternal symptoms of perinatal insomnia on social-emotional child development: a population-based, 2-year follow-up study. Behav Sleep Med 19(3):1–15. https://doi.org/10.1080/15402002.2020.1746661
2. Sedov ID, Cameron EE, Madigan S, Tomfohr-Madsen LM (2018) Sleep quality during pregnancy: a meta-analysis. Sleep Med Rev 38:168–176. https://doi.org/10.1016/j.smrv.2017.06.005
3. Meers JM, Nowakowski S (2022) Sleep during pregnancy. Curr Psychiatry Rep 24(8):353–357. https://doi.org/10.1007/s11920-022-01343-2
4. Mindell JA, Cook RA, Nikolovski J (2015) Sleep patterns and sleep disturbances across pregnancy. Sleep Med 16(4):483–488. https://doi.org/10.1016/j.sleep.2014.12.006
5. Guo Y, Xu Q, Dutt N, Kehoe P, Qu A (2023) Longitudinal changes in objective sleep parameters during pregnancy. Women's Heal 19:17455057231190952. https://doi.org/10.1177/17455057231190952
6. Warland J, Dorrian J, Morrison JL, O'Brien LM (2018) Maternal sleep during pregnancy and poor fetal outcomes: a scoping review of the literature with meta-analysis. Sleep Med Rev 41:197–219. https://doi.org/10.1016/j.smrv.2018.03.004
7. Fan M, Sun D, Zhou T et al (2020) Sleep patterns, genetic susceptibility, and incident cardiovascular disease: a prospective study of 385 292 UK biobank participants. Eur Hear J 41(11):1182–1189. https://doi.org/10.1093/eurheartj/ehz849
8. Meerlo P, Sgoifo A, Suchecki D (2008) Restricted and disrupted sleep: effects on autonomic function, neuroendocrine stress systems and stress responsivity. Sleep Med Rev 12(3):197–210. https://doi.org/10.1016/j.smrv.2007.07.007
9. Baglioni C, Battagliese G, Feige B et al (2011) Insomnia as a predictor of depression: a meta-analytic evaluation of longitudinal epidemiological studies. J Affect Disord 135:10–19. https://doi.org/10.1016/j.jad.2011.01.011
10. Vetter C (2020) Circadian disruption: what do we actually mean? Eur J Neurosci 51(1):531–550. https://doi.org/10.1111/ejn.14255
11. Fischer D, Vetter C, Roenneberg T (2016) A novel method to visualise and quantify circadian misalignment. Sci Rep 6(1):38601. https://doi.org/10.1038/srep38601
12. Bates K, Herzog ED (2020) Maternal-fetal circadian communication during pregnancy. Front Endocrinol 11:198. https://doi.org/10.3389/fendo.2020.00198
13. Mark PJ, Crew RC, Wharfe MD, Waddell BJ (2017) Rhythmic three-part harmony: the complex interaction of maternal, placental and fetal circadian systems. j Biol Rhythms 32(6):534–549. https://doi.org/10.1177/0748730417728671
14. Serón-Ferré M, Mendez N, Abarzua-Catalan L et al (2012) Circadian rhythms in the fetus. Mol Cell Endocrinol 349(1):68–75. https://doi.org/10.1016/j.mce.2011.07.039
15. Pires GN, Benedetto L, Cortese R et al (2021) Effects of sleep modulation during pregnancy in the mother and offspring: Evidences from preclinical research. J Sleep Res 30(3):e13135. https://doi.org/10.1111/jsr.13135
16. Smarr BL, Grant AD, Perez L, Zucker I, Kriegsfeld LJ (2017) Maternal and early-life circadian disruption have long-lasting negative consequences on offspring development and adult behavior in mice. Sci Rep 7(1):3326. https://doi.org/10.1038/s41598-017-03406-4
17. Aswathy BS, Kumar VM, Gulia KK (2018) Immature sleep pattern in newborn rats when dams encountered sleep restriction during pregnancy. Int J Dev Neurosci 69(1):60–67. https://doi.org/10.1016/j.ijdevneu.2018.06.007
18. Zhao Q, Peng C, Wu X, Chen Y, Wang C, You Z (2014) Maternal sleep deprivation inhibits hippocampal neurogenesis associated with inflammatory response in young offspring rats. Neurobiol Dis 68:57–65. https://doi.org/10.1016/j.nbd.2014.04.008
19. Peng Y, Wang W, Tan T et al (2016) Maternal sleep deprivation at different stages of pregnancy impairs the emotional and cognitive functions, and suppresses hippocampal long-term potentiation in the offspring rats. Mol Brain 9(1):17. https://doi.org/10.1186/s13041-016-0197-3
20. Ciciolla L, Addante S, Quigley A, Erato G, Fields K (2022) Infant sleep and negative reactivity: the role of maternal adversity and perinatal sleep. Infant Behav Dev 66:101664. https://doi.org/10.1016/j.infbeh.2021.101664
21. Nakahara K, Michikawa T, Morokuma S et al (2021) Association of maternal sleep before and during pregnancy with sleep and developmental problems in 1-year-old infants. Sci Rep 11(1):11834. https://doi.org/10.1038/s41598-021-91271-7
22. Nakahara K, Michikawa T, Morokuma S et al (2020) Association of maternal sleep before and during pregnancy with preterm birth and early infant sleep and temperament. Sci Rep 10(1):11084. https://doi.org/10.1038/s41598-020-67852-3
23. Lahti-Pulkkinen M, Mina TH, Riha RL et al (2019) Maternal antenatal daytime sleepiness and child neuropsychiatric and neurocognitive development. Psychol Med 49(12):2081–2090. https://doi.org/10.1017/s003329171800291x
24. Lugo-Candelas C, Hwei T, Lee S et al (2023) Prenatal sleep health and risk of offspring ADHD symptomatology and associated phenotypes: a prospective analysis of timing and sex differences in

the ECHO cohort. Lancet Reg Heal - Am 27:100609. https://doi.org/10.1016/j.lana.2023.100609

25. Newland RP, Parade SH, Dickstein S, Seifer R (2016) Goodness of fit between prenatal maternal sleep and infant sleep: associations with maternal depression and attachment security. Infant Behav Dev 44:179–188. https://doi.org/10.1016/j.infbeh.2016.06.010
26. Hoyniak CP, Whalen DJ, Luby JL et al (2023) Sleep and circadian rhythms during pregnancy, social disadvantage, and alterations in brain development in neonates. Dev Sci. https://doi.org/10.1111/desc.13456
27. Nevarez-Brewster M, Demers CH, Mejia A et al (2022) Longitudinal and prospective assessment of prenatal maternal sleep quality and associations with newborn hippocampal and amygdala volume. Dev Cogn Neurosci 58:101174. https://doi.org/10.1016/j.dcn.2022.101174
28. Okun ML, Tolge M, Hall M (2014) Low socioeconomic status negatively affects sleep in pregnant women. J Obstet Gynecol Neonatal Nurs 43(2):160–167. https://doi.org/10.1111/1552-6909.12295
29. MacKinnon AL, Tomfohr-Madsen L, Tough S (2021) Neighborhood socio-economic factors and associations with infant sleep health. Behav Sleep Med 19(4):458–470. https://doi.org/10.1080/15402002.2020.1778478
30. Engle PL, Black MM (2008) The effect of poverty on child development and educational outcomes. Ann N York Acad Sci 1136(1):243–256. https://doi.org/10.1196/annals.1425.023
31. Kalmbach DA, Cheng P, Sangha R et al (2019) Insomnia, short sleep, and snoring in mid-to-late pregnancy: disparities related to poverty, race, and obesity. Nat Sci Sleep 11:301–315. https://doi.org/10.2147/nss.s226291
32. Williamson AA, Gould R, Leichman ES, Walters RM, Mindell JA (2021) Socioeconomic disadvantage and sleep in early childhood: Real-world data from a mobile health application. Sleep Heal 7(2):143–152. https://doi.org/10.1016/j.sleh.2021.01.002
33. Lobermeier M, Staples AD, Peterson C et al (2022) Cumulative risk, infant sleep, and infant social-emotional development. Infant Behav Dev 67:101713. https://doi.org/10.1016/j.infbeh.2022.101713
34. Williamson AA, Mindell JA (2020) Cumulative socio-demographic risk factors and sleep outcomes in early childhood. Sleep 389:77–13. https://doi.org/10.1093/sleep/zsz233
35. Evans GW (2004) The environment of childhood poverty. Am Psychol 59(2):77–92. https://doi.org/10.1037/0003-066x.59.2.77
36. Brooks-Gunn J, Duncan GJ (1997) The effects of poverty on children. Future Child 7(2):55. https://doi.org/10.2307/1602387
37. Walker WH, Walton JC, DeVries AC, Nelson RJ (2020) Circadian rhythm disruption and mental health. Transl Psychiatry 10(1):28. https://doi.org/10.1038/s41398-020-0694-0
38. Stout MJ, Chubiz J, Raghuraman N et al (2022) A multidisciplinary prematurity research cohort study. PLoS ONE 17:e0272155. https://doi.org/10.1371/journal.pone.0272155
39. Zhao P, Bedrick BS, Brown KE et al (2022) Sleep behavior and chronotype before and throughout pregnancy. Sleep Med 94:54–62. https://doi.org/10.1016/j.sleep.2022.04.002
40. Witting W, Kwa IH, Eikelenboom P, Mirmiran M, Swaab DF (1990) Alterations in the circadian rest-activity rhythm in aging and Alzheimer's disease. Biol Psychiat 27(6):563–572. https://doi.org/10.1016/0006-3223(90)90523-5
41. Fischer D, Klerman EB, Phillips AJK (2021) Measuring sleep regularity: theoretical properties and practical usage of existing metrics. Sleep. 44(10):zsab103. https://doi.org/10.1093/sleep/zsab103
42. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ (1989) The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 28:193–213. https://doi.org/10.1016/0165-1781(89)90047-4
43. Sutcliffe S, Zhao P, Pilz LK et al (2023) Risk of pre-term birth as a function of sleep quality and obesity: prospective analysis in a large prematurity research cohort. Sleep Adv. 4(1):043. https://doi.org/10.1093/sleepadvances/zpad043
44. Carter AS, Briggs-Gowan MJ, Jones SM, Little TD (2003) The Infant-Toddler Social and Emotional Assessment (ITSEA): factor structure, reliability, and validity. J Abnorm Child Psychol 31(5):495–514. https://doi.org/10.1023/a:1025449031360
45. Benjamini Y, Hochberg Y (1995) Controlling the false discovery rate: a practical and powerful approach to multiple testing. J R Stat Soc Ser B Methodol 57:289–300
46. Cox JL, Holden JM, Sagovsky R (1987) Detection of postnatal depression: development of the 10-item edinburgh postnatal depression scale. Br J Psychiatry 6:782–786. https://doi.org/10.1192/bjp.150.6.782
47. R Core Team (2023) R: a language and environment for statistical computing. https://www.R-project.org/. Accessed 16 June 2023
48. Taylor BJ, Hasler BP (2018) Chronotype and mental health: recent advances. Curr Psychiatry Rep 20(8):59. https://doi.org/10.1007/s11920-018-0925-8
49. Gillett G, Watson G, Saunders KEA, McGowan NM (2021) Sleep and circadian rhythm actigraphy measures, mood instability and impulsivity: A systematic review. J Psychiatr Res 144:66–79. https://doi.org/10.1016/j.jpsychires.2021.09.043
50. Martin-Fairey CA, Zhao P, Wan L et al (2019) Pregnancy induces an earlier chronotype in both mice and women. J Biol Rhythm 34(3):323–331. https://doi.org/10.1177/0748730419844650
51. Takmaz T, Unal B, Ozcan P et al (2022) Are chronotype and subjective sleep quality associated with preeclampsia and preterm birth? Biol Rhythm Res 53(1):159–171. https://doi.org/10.1080/09291016.2020.1730617
52. Luik AI, Zuurbier LA, Hofman A, Someren EJWV, Tiemeier H (2013) Stability and fragmentation of the activity rhythm across the sleep-wake cycle: the importance of age, lifestyle, and mental health. Chronobiol Int 30(10):1223–1230. https://doi.org/10.3109/07420528.2013.813528
53. Paudel ML, Taylor BC, Ancoli-Israel S et al (2010) Rest/activity rhythms and mortality rates in older men: MROS sleep study. Chronobiol Int 27(2):363–377. https://doi.org/10.3109/07420520903419157
54. Heuval MVD, Hect JL, Smarr BL et al (2021) Maternal stress during pregnancy alters fetal cortico-cerebellar connectivity in utero and increases child sleep problems after birth. Sci Rep 11(1):2228. https://doi.org/10.1038/s41598-021-81681-y
55. Martire VL, Caruso D, Palagini L, Zoccoli G, Bastianini S (2020) Stress & sleep: a relationship lasting a lifetime. Neurosci Biobehav Rev 117:65–77. https://doi.org/10.1016/j.neubiorev.2019.08.024
56. O'Connor TG, Caprariello P, Blackmore ER, Gregory AM, Glover V, Fleming P (2007) Prenatal mood disturbance predicts sleep problems in infancy and toddlerhood. Early Human Dev 83(7):451–458. https://doi.org/10.1016/j.earlhumdev.2006.08.006
57. Weinstock M (2005) The potential influence of maternal stress hormones on development and mental health of the offspring. Brain, Behav, Immun 19(4):296–308. https://doi.org/10.1016/j.bbi.2004.09.006
58. Gregory AM, Moffitt TE, Ambler A, Arseneault L, Houts RM, Caspi A (2012) Maternal insomnia and children's family socialization environments. Sleep 35(4):579–582. https://doi.org/10.5665/sleep.1750
59. Okun ML (2016) Disturbed sleep and postpartum depression. Curr Psychiatry Rep 18(7):66. https://doi.org/10.1007/s11920-016-0705-2
60. Lyu J, Groeger JA, Barnett AL et al (2022) Associations between gestational age and childhood sleep: a national retrospective

cohort study. BMC Med 20(1):253. https://doi.org/10.1186/s12916-022-02443-9

61. Germain A, Kupfer DJ (2008) Circadian rhythm disturbances in depression. Hum Psychopharmacol Clin Exp 23(7):571–585. https://doi.org/10.1002/hup.964