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ORIGINAL ARTICLE

Sleep Quality Predicts Persistence of Parental Postpartum Depressive Symptoms and Transmission of Depressive Symptoms from Mothers to Fathers

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Abstract
Background Early parenthood is a time of chronic sleep disturbance and also of heightened depression risk. Poor sleep quality has been identified both as a predictor of postpartum depressive symptoms and as a consequence.
Purpose This study sought to clarify causal pathways linking sleep and postpartum depression via longitudinal path modeling. Sleep quality at 6 months postpartum was hypothesized to exacerbate depressive symptoms from 1 month through 1 year postpartum in both mothers and fathers. Within-couple associations between sleep and depression were also tested.
Methods Data were drawn from a low-income, racially and ethnically diverse sample of 711 couples recruited after the birth of a child. Depressive symptoms were assessed at 1, 6, and 12 months postpartum, and sleep was assessed at 6 months postpartum.
Results For both partnered mothers and fathers and for single mothers, depressive symptoms at 1 month postpartum predicted sleep quality at 6 months, which in turn predicted depressive symptoms at both 6 and 12 months. Results held when infant birth weight, breastfeeding status, and parents' race/ethnicity, poverty, education, and immigration status were controlled. Mothers' and fathers' sleep quality and depressive symptoms were correlated, and maternal sleep quality predicted paternal depressive symptoms both at 6 and at 12 months.
Conclusions Postpartum sleep difficulties may contribute to a vicious cycle between sleep and the persistence of depression after the birth of a child. Sleep problems may also contribute to the transmission of depression within a couple. Psychoeducation and behavioral treatments to improve sleep may benefit new parents.

Keywords Sleep · Sleep disturbance · Sleep duration · Postpartum depression · Couples · Parents

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Depression following the birth of a child occurs worldwide and has a wide range of both biological and psychosocial antecedents [1]. Prevalence rates of postpartum depression (PPD) range from 13 to 19 % in mothers over the first year postpartum, representing increased risk compared to non-postpartum women [2]. Postpartum depressive symptoms are known to affect not only parents' well-being but also

children's health and development. For example, women with postpartum depressive symptoms were less likely to place infants in safe sleeping positions, to bring infants in for routine health visits, and to have infants fully immunized [3]. Even mild maternal depressive symptoms appear to impair mother-infant bonding [4], suggesting that PPD may affect children even when depression is below clinical threshold. Men also suffer from increased risk of depression during the postpartum period [5], with new fathers twice as likely to report symptoms of depression as men in the general population [6]. PPD symptoms are positively correlated within couples [7], but the mechanisms of potential transmission between partners have not been explored in the research literature.

In addition to being a time of heightened depression risk for both mothers and fathers, the months following the birth of a child often include significant reductions in both sleep quantity and quality [8, 9]. Sleep problems are implicated in depression outside the postpartum period [10] and, more recently, have been linked specifically with postpartum depression [11]. Two reviews on postpartum sleep problems and depression appeared in 2015. One, by Bhati and Richards [12], examined 13 published studies and found a consistent, strong relationship between postpartum sleep disturbance and maternal PPD. Effect sizes ranged from 0.4 to 1.7, putting them in the medium-to-very large range (medium effect sizes are typically defined as effect sizes around .5 and very large effect sizes as >.8). In another review, Lawson and colleagues [13] incorporated 31 studies (including 12 of the 13 studies reviewed by Bhati and Richards) and also found consistent support for an association between sleep disruption and mood disorder risk in the postpartum period. However, the authors rated the overall quality of the reviewed studies as weak. In particular, almost all of the studies rated sleep and depressive symptoms concurrently, a significant limitation because disturbed sleep can be a symptom of depression.

Given this cross-sectional design of many published studies, the causal relationship between sleep and depressive symptoms warrants scrutiny. Sleep disturbance has been conceptualized as a cause, correlate, and consequence of depression. Researchers have been mixed on whether postpartum sleep problems precede and help to potentiate depression [14] or whether depression precedes and helps to potentiate sleep problems [15]. Alternatively, additional variables, such as breastfeeding or bedsharing, may contribute to changes in both sleep and depressive symptoms after the birth of a child. Understanding the temporal sequencing of relationships between sleep and depression is particularly important in the postpartum period, since an infant's sleep and feeding patterns may aggravate sleep problems.

Although fathers also experience increased postpartum depression risk, the literature to date has focused primarily on mothers. A meta-analysis of 43 studies from 16 countries reported a 10 % prevalence rate of paternal depression within the first year postpartum, more than double the 4.8 % 12-month prevalence rate observed among men in the general population [5]. This meta-analysis also found moderate correlations between paternal and maternal PPD (mean correlation coefficient of 0.31 across studies, 5). In another study, men whose partners experienced moderate-to-severe PPD had an eightfold increase in depression (odds ratio 8.44) in their own PPD risk [7]. Given that fathers as well as mothers may experience sleep disruption in early parenthood, sleep might be linked with paternal PPD and may even contribute to the transmission of PPD within couples. The two above-cited reviews of sleep and PPD [12, 13] did not include any studies on fathers. To our knowledge, no other studies have specifically examined the role of sleep in paternal PPD. This is a surprising omission because, unlike other potential correlates of maternal PPD such as post-birth changes in reproductive hormones [1], postpartum sleep disturbance can affect both parents.

The current study assessed postpartum depressive symptoms at 1, 6, and 12 months after a birth in an ethnically/racially diverse, predominately low-income sample of parents recruited as part of a larger five-site study [16]. Participants rated their sleep duration and quality (over the previous month) 6 months after the birth of their child. We tested a *concurrent* and two *longitudinal* path models. The concurrent model examined the links between depressive symptoms and sleep, with both constructs assessed at 6 months postpartum in both mothers and fathers. The 6-month longitudinal model tested associations in depressive symptoms from 1 to 6 months postpartum with parent sleep at 6 months as a prospective mediator, as well as associations between mothers and fathers and the cross-partner effects of mother sleep on father depressive symptoms and father sleep on mother symptoms. The 12-month longitudinal model tested depressive symptoms at 1 and 12 months postpartum with, again, sleep at 6 months as a prospective mediator. We also tested longitudinal associations between sleep and depression in single mothers separately, in order to explore whether sleep quality would mediate associations between 1-, 6-, and 12-month depressive symptoms among mothers who were not in a relationship with their baby's father.

Hypotheses

We hypothesized that parent sleep quality at 6 months would mediate the change in depressive symptoms from 1 to 6 months postpartum and from 1 to 12 months postpartum, for both partnered mothers and fathers and for single mothers. We also expected maternal and paternal depressive symptoms to be linked to each other concurrently and planned to test the exploratory hypothesis that each parent's sleep quality would mediate the association between the other parent's 1- and 6- or 12-month depressive symptoms.

Methods

Data were collected through the Child Community Health Network (CCHN), a large, five-site, multi-investigator community-based participatory research network funded by the *Eunice Kennedy Shriver* National Institute for Child Health and Human Development (NICHD). Recruitment, eligibility, and demographic characteristics for the CCHN study are described in other papers [16, 17]. Briefly, biological mothers were recruited at the birth of their first, second, or third child and followed at multiple timepoints over the first 2 years postpartum. Eligibility criteria included that mothers were between 18 and 40 years of age and self-identified as African-American, White, or Latina. Fathers were also invited to participate in the study with mothers' consent. Study sites included Baltimore, MD; Lake County, IL; Los Angeles, CA; eastern North Carolina; and Washington, D.C. The current paper includes data from the first three visits: at 1 month postpartum (time 1 or T1), at 6 months postpartum (T2), and at 12 months postpartum (T3). Within the full sample, 53 % of mothers were cohabiting with the baby's father at T1, 51 % at T2, and 50 % at T3; 70 % of mothers reported that they were in a romantic relationship with the baby's father at T1, 67 % at T2, and 68 % at T3. For current analyses, given our interest in cross-partner associations, we restricted our sample to include only couples that were in a romantic relationship (according to mother report) at both 1 and 6 months postpartum. We also included only couples in which both the mother and father provided complete data on sleep. Sample characteristics are shown in Table 1. Compared to national census data [18–20], our participants were more likely to be non-white (US census: 64 % of Americans are white, 16 % Latino, and 13 % African-American), had lower incomes (US census median income is $49,445), and were more likely to be in single-parent households (nationally, 27 % of young children live with one and 69 % with both parents).

The final sample comprised 711 couples. There were 1453 mothers who participated at T1 but were not included in this final sample: in 552 cases, the mother did not participate at T2; in 1267 cases, the partner did not participate at T2; and in 336 cases, both the mother and father did not participate at T2. Comparing mothers in the final sample to mothers who participated in the larger study but were not included in the present analyses (n = 1453) because they or their partner did not participate in T2 data collection, there were no significant differences in maternal sleep quality (t = 1.21, p = .22) or depressive symptoms at either T1 (t = 1.87, p = .06) or T3 (t = 1.42, p = .16). There were also no differences in the baby's birth weight (t = .14, p = .89), whether the baby slept alone or with parents (χ^2 = 3.67, p = .07), whether the mother was Latina

Table 1 Characteristics of the study sample

Variable	Mean	SD	Range
Sleep (abbreviated PSQI)			
Mothers 6 months postpartum	2.87	1.42	1–6
Fathers 6 months postpartum	2.79	1.44	1–6
Depressive symptoms (EPDS)			
Mothers	4.39	4.32	0–25
1 month postpartum	4.67	4.26	0–23
6 months postpartum	4.60	4.49	0–24
12 months postpartum			
Fathers			
1 month postpartum	3.57	3.57	0–21
6 months postpartum	4.33	3.88	0–25
12 months postpartum	4.03	3.98	0–25
Covariates			
Infant birth weight (in lbs)	7.08	1.47	3.48–11.14
Per capita household income	$14,405.88	$21,688.81	$0–36,657
Mother education	2.60	1.07	1–5
Father education	2.54	1.22	1–5
Mother BMI 6 months postpartum	29.57	7.92	16.36–61.50
Father BMI 6 months postpartum	28.87	5.06	18.96–48.69
Mother age (at study entry)	25.73	5.69	17.90–41.69
Father age (at study entry)	28.90	7.07	17.94–62.72

Per capita household income is adjusted for cost of living. Education is coded on a four-point scale (1 = less than high school, 2 = high school or GED, 3 = some college, 4 = completed 4-year college or higher)

EPDS Ediburgh Postnatal Depression Scale, *PSQI* Pittsburgh Sleep Quality Index, *BMI* body mass index

($\chi^2 = 1.35$, $p = .25$), or whether the mother was born in the USA or in a foreign country ($\chi^2 = 1.83$, $p = .19$). However, mothers not included in this sample reported higher depressive symptoms at T2 ($t = 2.62$, $p = .01$), had lower household income ($t = 2.69$, $p = .01$), were less educated ($t = 4.68$, $p = .001$), and were more likely to be African-American ($\chi^2 = 72.64$, $p = .001$). Comparing only within the smaller group of mothers who had complete data available at T2 but were not included in the sample because they were not in a relationship with their baby's father at 1 or 6 months postpartum ($n = 166$), there were no differences in maternal sleep quality ($t = 1.87$, $p = .06$), infant birth weight ($t = -0.43$, $p = .67$), or depressive symptoms at T1 ($t = 1.26$, $p = .21$) or at T2 ($t = 1.59$, $p = .11$). However, mothers who were not in a relationship with their baby's father reported higher depressive symptoms at T3 ($t = 2.02$, $p = .04$), had lower household income ($t = -2.67$, $p = .01$), and were less educated ($t = -5.32$, $p = .001$), less likely to be born in a foreign country ($\chi^2 = 15.10$, $p = .001$), more likely to be African-American ($\chi^2 = 47.84$, $p = .001$), less likely to be Latina ($\chi^2 = 20.30$, $p = .001$), and more likely to cosleep with their infants ($\chi^2 = 14.66$, $p = .001$).

Measures

Sleep

An abbreviated (three-item) version of the Pittsburgh Sleep Quality Index (PSQI), a well-validated and widely used sleep measure [21], was given at T2. Participants were asked to assess their sleep quantity and quality over the previous month. Specifically, they were asked about sleep duration (how many hours of actual sleep per night the participant had averaged over the past month) and sleep disturbance (how often s/he had trouble getting to sleep within 30 min and how often s/he had night or early morning wakings). The shortened version of this measure is similar to abbreviated measures of sleep duration and sleep disturbance that have been used in other large epidemiological studies, e.g., the Whitehall study [22]. Our use of a simple, dimensional measure of sleep quality is in keeping with recommendations for field trials from the DSM-5 Sleep-Wake Disorders Workgroup [23]. Responses were scored using the PSQI scoring criteria and then summed. Specifically, sleep duration estimates were recoded as 0 (>7 h per night), 1 (6–7 h per night), 2 (5–6 h per night), and 3 (<5 h per night); sleep disturbances were also rated on a 0–3 scale.

Depression

The Edinburgh Postnatal Depression Scale (EPDS; 24) is a well-validated and widely used scale that has also been validated in men [25]. It is a 10-item measure with a possible range of 0–30 regarding symptoms over the previous 7 days. Continuous scores were used in the present analyses because the current study used a community rather than high-risk sample and because of evidence that even mild depressed mood in the postpartum period can affect infant health and postpartum bonding [4].

The EPDS has one item assessing sleep ("I have been so unhappy that I have had difficulty sleeping"), so we dropped this item before calculating total scores in order to eliminate the possibility that this item was driving or contributing to our results. For both mothers and fathers and at T1, T2, and T3, the original EPDS was highly correlated with the rescored EPDS (all correlation coefficients >.985, all p values <.001). Analyses for this paper were repeated with the original EPDS scores, and results were unchanged.

Covariates

At study entry, medical records were used to obtain infant birth weight and parity, as well as C-section delivery. Within this sample, 45 % of mothers were first-time mothers, 40 % had one child, and 15 % had two children; 40 % of mothers had delivered the index child by C-section. At the T1 interview, demographic data were collected: maternal and paternal education (coded from 1 = less than high school, 2 = high school or GED, 3 = some college, 4 = completed 4-year college or higher); whether mother and father were US or foreign born (23 % of mothers and 25 % of fathers were foreign born); parent race and ethnicity (37 % of mothers were African-American and 27 % were Latina; 40 % of fathers were African-American and 26 % were Latino); poverty group (based on family income and household size, calculated as a percentage of the federal poverty level (FPL), scored as 1, ≤100 % FPL; 2, >100 to 200 % FPL; 3, >200 % FPL). Also at T1, mothers were asked where the baby sleeps—alone in a crib or in a shared bed with siblings, others, or parent(s). This variable was recoded as 0 = baby sleeps alone or with sibling/other and 1 = baby sleeps with parents. Within this sample, 68 % of mothers reported that the baby did not sleep with the parents and 32 % of mothers reported that the baby slept with the parents. At the T2 interview, mothers were asked if they were currently breastfeeding. In this sample, 26 % of mothers were still breastfeeding at 6 months and 74 % were not. To determine parent Body mass index (BMI), study personnel obtained measures of height and weight at T2. BMI was calculated by dividing weight in pounds by height in inches squared and multiplying by a conversion factor of 703 (BMI = weight (lb) / [height (in)]^2 × 703).

Analytic Approach

Associations between sleep and depressive symptoms were tested using structural equation modeling [26], which is well suited to examining nested longitudinal data. We used the MLR estimator which produces maximum likelihood

parameter estimates with standard errors that are robust to non-normality. We ran three models. First is the concurrent model, including sleep and PPD tested at the same timepoint (T2) for both mothers and fathers. Next, we ran two longitudinal models: first, a 6-month longitudinal model examining T1 to T2 depressive symptoms as mediated by T2 sleep; next, a longitudinal model with T1 to T3 depressive symptoms mediated by T2 sleep. Both models were run with and without additional covariates. We tested indirect effects, a test of mediation in SEM, by adding the "model indirect" command to our Mplus input file.

For the separate analysis of single mothers, we did not have adequate sample size to test mediation in SEM. Instead, we ran regression analyses in SPSS and used the resulting betas and standard errors to calculate the Sobel test statistic [27], which reflects whether the mediator variable exerts a significant indirect effect on the relationship between the independent variable and the dependent variable.

Results

Zero-order correlations between the sleep measure and other variables are shown in Table 2. Mothers' and fathers' depressive symptoms were correlated with each other at all three assessments, and mothers' and fathers' sleep problems were associated with more depressive symptoms for both partners at all three assessments. Being born outside the USA and being Latino were both associated with better sleep for both parents; being African-American was associated with worse sleep for both parents, and fathers' heavier BMI was associated with worse sleep for fathers. Mothers who were living in greater poverty and were less educated reported more depressive symptoms at multiple timepoints. Additionally, mothers' T1 depressive symptoms were linked with lower infant birth weight and fathers' T2 and T3 symptoms with the baby sharing a bed with parents. At T2, Latina mothers and African-American fathers reported more depressive symptoms.

Table 2 Bivariate correlations between study variables

	1	2	3	4	5	6	7	8
Sleep								
1. Mother 6 months postpartum	1						.	.
2. Father 6 months postpartum	.16***	1						
Depressive symptoms								
3. Mother 1 month postpartum	.30***	.12***	1					
4. Mother 6 months postpartum	.34***	.09**	.46***	1				
5. Mother 12 months postpartum	.27***	.08**	.43***	.50***	1			
6. Father 1 month postpartum	.11***	.20***	.25***	.19***	.20***	1		
7. Father 6 months postpartum	.15***	.29***	.14***	.27***	.18***	.41***	1	
8. Father 12 months postpartum	.17***	.24***	.21***	.24***	.26***	.47***	.52***	1
Demographics and other covariates								
Mother poverty group	−.05	−.07*	−.06*	−.10**	−.10**	−.11***	−.08**	−.04
Mother education	.02	−.01	−.02	−.11***	−.09*	−.08	−.05	−.01
Mother US vs. foreign birth	−.10***	−.13***	−.04	−.03	.04	−.06	−.05	−.02
Father poverty group	.01	.00	−.03	−.03	−.07	−.04	−.06	.04
Father education	.04	.01	.05	.02	.01	−.07*	.01	.02
Father US vs. foreign birth	−.11***	−.16***	−.01	−.02	.05	−.01	−.02	.01
Breastfeeding at 6 months postpartum	−.03	−.05	−.07*	−.07	−.05	−.07	−.01	.01
Infant birth weight	−.07	−.03	−.15***	−.02	−.02	−.10*	−.03	−.04
Mother body mass index	.05	.01	−.04	−.04	.01	.05	.06	−.02
Father body mass index	.03	.18***	.07	−.01	.04	.11	.07	.06
Baby sleeps with parents	.03	−.01	.08	.08	.08	.06	.13***	.11**
C-section delivery	.05	−.05	.02	−.02	−.08*	.06	.03	.02
Mother African American vs. not	.09**	.08**	.00	.01	−.03	.07	.06	.01
Father African American vs. not	.10***	.09**	.01	.01	−.04	.07	.07**	.02
Mother Latina vs. not Latina	−.08**	−.12***	−.02	.04	.08**	.01	.01	.04
Father Latino vs. not Latino	−.10***	−.14***	−.04	.03	.12***	−.03	−.01	.04
Mother parity	.01	−.02	.09**	.04	.07*	.01	.01	.002

*$p < .10$; ** $p < .05$; ***$p < .01$

At T1, 6.8 % of fathers and 13.4 % of mothers had clinically significant PPD (EPDS) score (reflecting total EPDS scores including the sleep item) >9 [28]. At T2, 9.7 % of fathers and 13.4 % of mothers had clinically significant PPD. At T3, 9 % of fathers and 13.5 % of mothers had clinically significant PPD. Of those mothers who reported clinically significant PPD at T1, 34 % also had clinically significant PPD at T2 and 36 % had clinically significant PPD at T3. Of those mothers who reported clinically significant PPD at T2, 37 % also had clinically significant PPD at T3. Of those fathers who reported clinically significant PPD at T1, 26 % also had clinically significant PPD at T2 and 50 % had clinically significant PPD at T3. Of those fathers who reported clinically significant PPD at T2, 39 % also had clinically significant PPD at T3. Together with moderate-to-high correlations in depressive symptoms across the three waves, these patterns point to stability but also heterogeneity in trajectories of participants' PPD across the first year postpartum.

Concurrent SEM Model: T2 Sleep Associated with T2 Depression

All parameter estimates for this model are shown in Fig. 1. For both mothers and fathers, T2 sleep quality ratings were associated with greater T2 depression. Maternal and paternal PPD symptoms were positively correlated at both T1 and T2, as were maternal and paternal sleep ratings. Additionally, mothers' sleep at T2 was associated with paternal depressive symptoms at T2. When we ran this model with all covariates (birth weight, T2 breastfeeding, infant sleep arrangement (in bed with parents/not with parents), C-section delivery, maternal and paternal education, poverty, race, ethnicity, parents' foreign birth, T2 BMI, and parity), key results were identical, but model fit was poor (CFI <0.90; RMSEA >.10), so we ran this model without covariates (CFI = 1; RMSEA = 0; model fit was perfect as this is a fully saturated model).

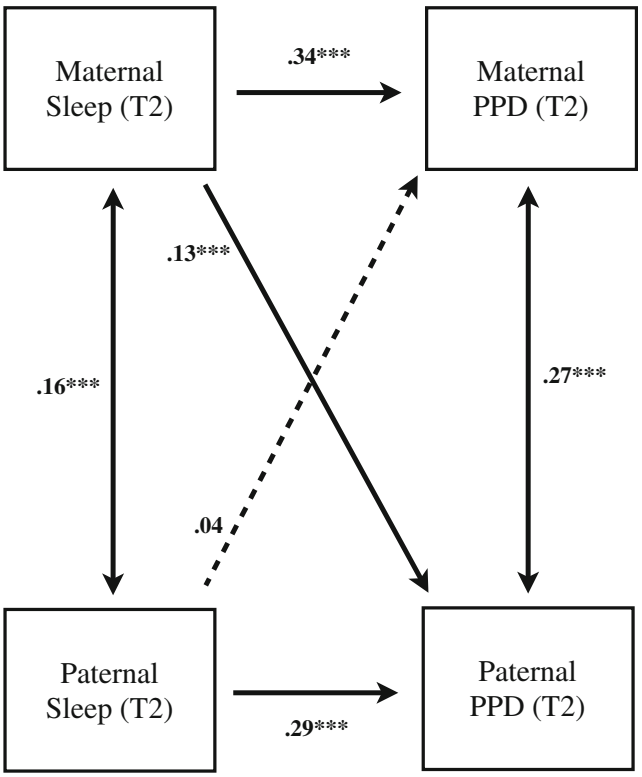

Fig. 1 Concurrent model depicting associations between 6-month (T2) depressive symptoms and 6-month (T2) sleep ratings. This figure was prepared using Mplus (Muthen and Muthen 2007). Non-significant paths are denoted with a *dotted line*, significant paths with a *solid line*. *$p < .05$; **$p < .01$

Six-Month Longitudinal SEM Model: T1 to T2 Depressive Symptoms with T2 Sleep

The complete model (Fig. 2) including T1 and T2 PPD, sleep, and all covariates (birth weight, T2 breastfeeding, infant sleep arrangement (with or not with parents), C-section delivery, maternal and paternal education, poverty, race, ethnicity, parents' foreign birth, T2 BMI, and parity) had very good model fit (scaled χ^2 = 59.21 [50]; CFI = 0.983; RMSEA = .016). For both mothers and fathers, T1 depression predicted worse sleep quality ratings at T2, and sleep quality in turn was associated with T2 depression. Maternal and paternal PPD symptoms were positively correlated at both T1 and T2, as were maternal and paternal sleep ratings. Additionally, mothers' sleep at T2 was associated with paternal depressive symptoms at T2. Figure 1 shows the model including sleep and depression but no additional covariates.

None of the added covariates were associated with depression, except for maternal education (negatively associated with maternal depression) and the baby cosleeping with parents (positively associated with paternal depression). Table 3 shows complete results. Tests of mediation (indirect effects) are presented in Table 5 and suggest that for each parent, sleep significantly mediated the association between T1 and T2 depression. Model fit was significantly improved when we added these indirect paths (T1 sleep ➔ T2 depression via sleep; $\Delta\chi^2$ = 38.18, p = .001).

Twelve-Month Longitudinal SEM Model: T1 to T3 Depressive Symptoms with T2 Sleep

The complete model including T1 and T3 EPDS, sleep, and all covariates had very good model fit (scaled χ^2 = 73.40 [50]; CFI = 0.947; RMSEA = .026). Results for the longitudinal model were almost identical to the concurrent model: T2 sleep mediated the association between T1 and T3 depressive symptoms for both mothers and fathers, and mothers' T2 sleep predicted fathers' T3

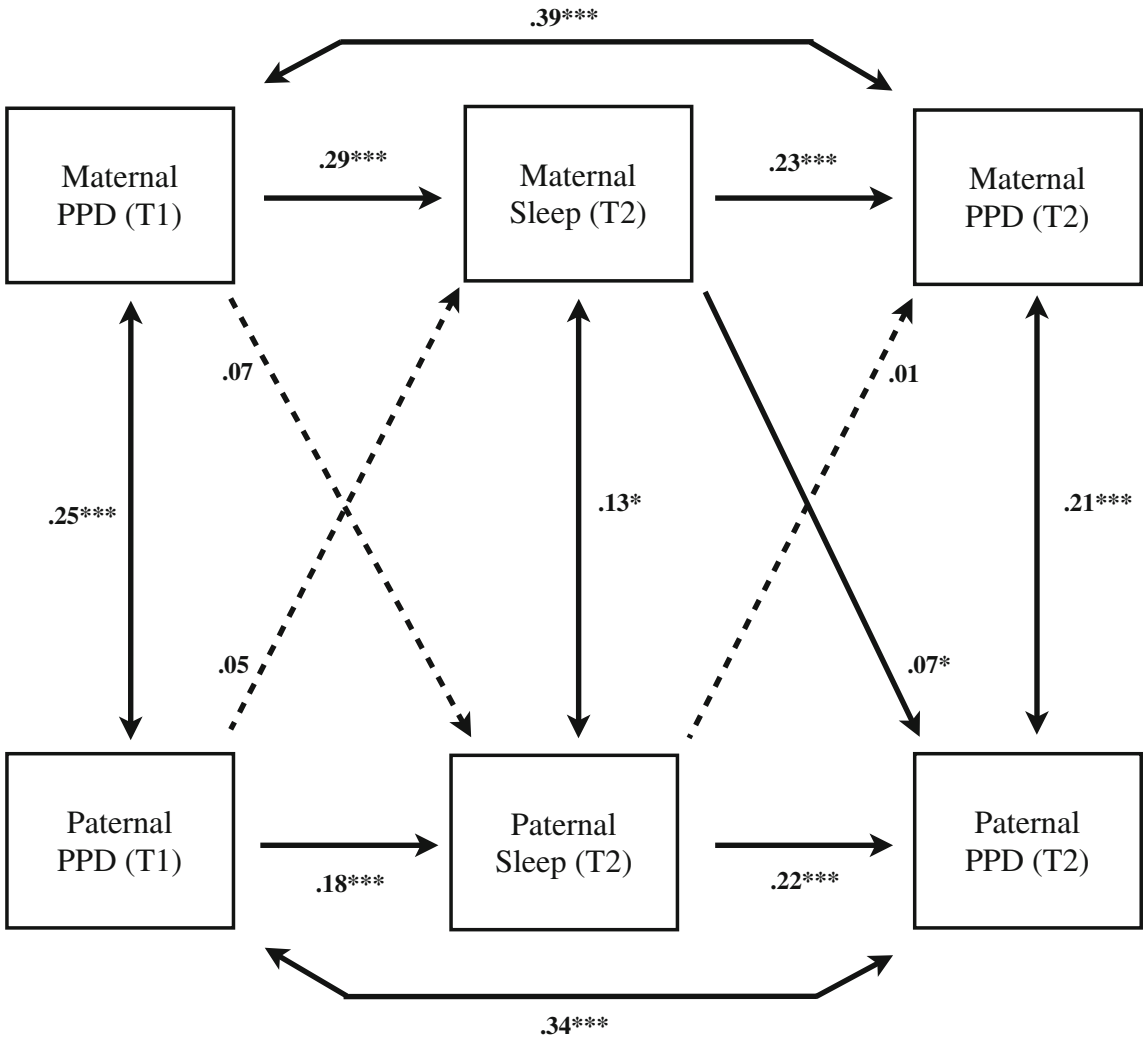

Fig. 2 Six-month longitudinal model depicting associations between one-month (T1) depressive symptoms and 6-month (T2) depressive symptoms, as mediated by 6-month (T2) sleep ratings. This figure was prepared using Mplus (Muthen and Muthen 2007). Non-significant paths are denoted with a *dotted line*, significant paths with a *solid line*. *$p < .05$; **$p < .01$

depressive symptoms. Mothers' and fathers' depressive symptoms were positively correlated at both T1 and T3, and their T2 sleep quality was also positively correlated. Only three covariates were associated with depression: C-section delivery was negatively associated with mothers' T3 depression; fathers' Latino ethnicity and the baby sleeping with parents were both positively associated with fathers' T3 depression.

Full results are shown in Table 4. Figure 3 depicts the model including sleep and depression but no additional covariates.

Our test of mediation found that sleep significantly mediated the association between T1 and T3 depression for each parent, and the path from mother T1 depression had a significant indirect effect on father T3 depression via (mediated by) mothers' T2 sleep. Table 5 shows the effects for the mediational model. Model fit was significantly improved when we added these indirect paths (T1 sleep ➔ T2 depression via sleep; $\Delta\chi^2 = 7.99$, $p = .02$).

Single Mother Analyses

Six-Month Longitudinal Model

T1 depression was a significant predictor of T2 sleep among single mothers, b(163) = .22, $p = .01$, and both T1 depression and T2 sleep were individually associated with T2 depression among single mothers, b(161) = .29, $p = .001$ for sleep and b(162) = .44, $p = .001$ for T1 depression. When we entered both sleep and T1 depression together as predictors of T2 depression, both remained significant but the coefficient for T1 depression became smaller (b(161) = .40, $p = .001$). The Sobel test statistic =2.27, $p = .01$, one-tailed, indicates significant mediation of the association between T1 and T2 depression by sleep.

Table 3 Parameter estimates for 6-month longitudinal SEM model predicting 1- to 6-month postpartum depressive symptoms (standardized (STDYX) coefficients shown)

	β	*S.E.*	*p* value
Direct effects: within- and between-partner depressive symptoms and sleep			
Mother depression (1 month) → mother sleep (6 months)	*.29*	*.04*	*.001*
Father depression (1 month) → mother sleep (6 months)	.05	.04	.18
Father depression (1 month) → father sleep (6 months)	*.18*	*.04*	*.001*
Mother depression (1 month) → father sleep (6 months)	.07	.04	.07+
Variables predicting mother depressive symptoms at 6 months postpartum			
Mother depression (1 month)	*.39*	*.03*	*.001*
Mother sleep (6 months)	*.23*	*.03*	*.001*
Father sleep (6 months)	.01	.03	.93
Race (African-American vs. not)	−.04	.04	.35
Ethnicity (Latina vs. not)	.07	.05	.18
Body mass index at 6 months	−.06	.04	.12
C-section delivery	.02	.03	.56
Infant birth weight	−.07	.04	.14
Cohabitation at 6 months	−.02	.03	.56
Mother poverty group	−.01	.04	.95
Education	*−.10*	*.04*	*.02**
Foreign birth	−.09	.05	.07+
Parity (number of children)	.01	.04	.90
Breastfeeding status at 6 months	−.03	.04	.39
Baby sleeping with parents	.05	.04	.24
Variables predicting father depressive symptoms at 6 months postpartum			
Father depression (1 month)	*.34*	*.04*	*.001*
Father sleep (6 months)	*.22*	*.03*	*.001*
Mother sleep (6 months)	*.07*	*.03*	*.05**
Race (African-American vs. not)	.04	.04	.39
Ethnicity (Latino vs. not)	.09	.06	.13
Six-month body mass index	.05	.06	.46
C-section delivery	.02	.04	.60
Infant birth weight	−.04	.05	.37
Cohabitation at 6 months	−.02	.04	.63
Father poverty group	−.05	.04	.17
Education	.06	.04	.12
Foreign birth	−.02	.05	.77
Mother parity (number of children)	−.03	.04	.49
Mother breastfeeding status at 6 months	.02	.04	.61
Baby sleeping with parents	*.11*	*.04*	*.01**

Statistically significant results are italicized

*$p < .05$; +$p \leq .10$

Twelve-Month Longitudinal Model

Both T1 depression and T2 sleep were individually associated with T3 depression among single mothers: b(164) = .19, $p = .01$ for sleep and b(163) = .34, $p = .001$ for T1 depression. When we entered both predictors together, both were significant, but the coefficient for T1 depression became smaller (b(160) = .33, $p = .001$). The Sobel test statistic =1.87, $p = .03$, one-tailed, indicates significant mediation of the association between T1 and T2 depression by sleep.

Table 4 Parameter estimates for 12-month longitudinal SEM model predicting 1- to 12-month depression (standardized (STDYX) coefficients shown)

	β	*S.E.*	*p* value
Direct effects: within- and between-partner depressive symptoms and sleep			
Mother depression (1 month) → mother sleep (6 months)	*.29*	*.04*	*.001***
Father depression (1 month) → mother sleep (6 months)	.05	.04	.19
Father depression (1 month) → father sleep (6 months)	*.17*	*.04*	*.001***
Mother depression (1 month) → father sleep (6 months)	.07	.04	.08+
Variables predicting mother depressive symptoms at 12 months postpartum			
Mother depression (1 month)	*.36*	*.04*	*.001***
Mother sleep (6 months)	*.17*	*.04*	*.001***
Father sleep (6 months)	.01	.04	.70
Race (African-American vs. not)	−.08	.05	.08+
Ethnicity (Latino/a vs. not)	.08	.06	.23
Body mass index at 6 months	.02	.04	.56
C-section delivery	*.09*	*.04*	*.02**
Infant birth weight	−.03	.04	.58
Cohabitation at 6 months	.01	.04	.99
Poverty group	−.05	.05	.34
Education	−.03	.05	.56
Foreign birth	−.02	.06	.68
Parity (number of children)	.03	.04	.53
Breastfeeding status at 6 months	−.02	.04	.67
Baby sleeping with parents	.06	.04	.17
Variables predicting father depressive symptoms at 12 months postpartum			
Father depression (1 month)	*.44*	*.04*	*.001***
Father sleep (6 months)	*.15*	*.04*	*.001***
Mother sleep (6 months)	*.10*	*.04*	*.006***
Race (African-American vs. not)	−.01	.05	.80
Ethnicity (Latino vs. not)	*.15*	*.06*	*.02**
Body mass index at 6 months	.01	.06	.97
C-section delivery	.01	.04	.36
Infant birth weight	.06	.05	.24
Cohabitation at 6 months	−.04	.04	.36
Poverty group	.06	.04	.18
Education	,06	.04	.16
Foreign birth	−.05	.06	.40
Mother parity (number of children)	.01	.04	.91
Breastfeeding status at 6 months	.04	.04	.34
Baby sleeping with parents	*.09*	*.04*	*.04**

Statistically significant results are italicized

*$p < .05$; ** $p < .01$; ***$p < .001$ +$p \leq .10$

Discussion

This study found that, for both partnered mothers and fathers and for single mothers, depressive symptoms at 1 month after the birth of a child was associated with poorer sleep at 6 months postpartum, which was in turn associated with more depressive symptoms at both 6 and 12 months postpartum. In other words, sleep appeared to mediate the associations between depressive symptoms measured shortly after birth and depressive symptoms measured 6 or 12 months later. Thus, sleep quantity and quality when caring for a newborn may contribute to a "vicious cycle" linked to the persistence or worsening of depression in the first year postpartum. Additionally, maternal sleep at 6 months was associated with father depression at both 6 and 12 months. Therefore, sleep may not only play a role in maintaining depression in early parenthood but also be involved in the transmission

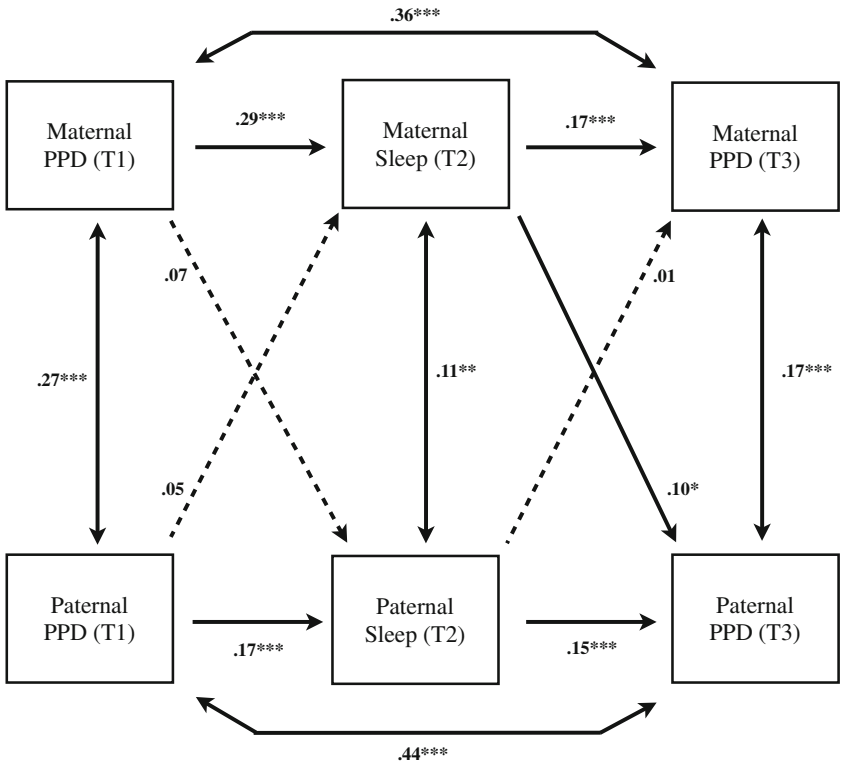

Fig. 3 Twelve-month longitudinal model depicting associations between 1-month (T1) depressive symptoms and 12-month (T3) depressive symptoms, as mediated by 6-month (T2) sleep ratings. This figure was prepared using Mplus (Muthen and Muthen 2007). Non-significant paths are denoted with a *dotted line*, significant paths with a *solid line*. *$p < .05$; **$p < .01$

of depression from mothers to fathers. Potentially confounding variables such as parental BMI, infant bedsharing, breastfeeding, and demographic characteristics (minority status, poverty, and education) did not explain or alter the results. When models were tested with all covariates, sleep was the strongest and most significant predictor of depressive symptoms at both 6 and 12 months, and sleep and depressive symptoms were strongly associated within and between partners in a "concurrent" or cross-sectional model including only measures taken at the 6-month visit. Although some parental sleep disturbance is expected after a new baby arrives, our results suggest that mood and affective problems in the early postpartum period can worsen parents' subsequent sleep quality, and parents who develop sleep problems may be at heightened risk of depressive symptoms [11].

This study is the first to assess parental sleep and postpartum depressive symptoms with longitudinal data measured over a year's time, using path modeling to clarify directional relationships among these variables. Given that previous papers on sleep and postpartum depression have used mostly cross-sectional designs, this research contributes to the literature by examining sleep as both preceding and following depressive symptoms. This is also the first study to assess the role of sleep in fathers' postpartum depressive symptoms and to examine both mothers and fathers together in a model examining within-couple

Table 5 Indirect estimates (tests of mediation) for longitudinal SEM models (standardized (STDYX) coefficients)

	β	*S.E.*	*p* value
Longitudinal model predicting depressive symptoms at 6 months postpartum			
Mother depression (1 month) → mother depression (6 months) via mother sleep	*.07*	*.01*	*.001***
Father depression (1 month) → mother depression (6 months) via father sleep	.01	.06	.93
Father depression (1 month) → father depression (6 months) via father sleep	*.04*	*.01*	*.001***
Mother depression (1 month) → father depression (6 months) via mother sleep	.02	.01	.06+
Longitudinal model predicting depressive symptoms at 12 months postpartum			
Mother depression (1 month) → mother depression (12 months) via mother sleep	*.05*	*.01*	*.001***
Father depression (1 month) → mother depression (12 months) via father sleep	.01	.06	.70
Father depression (1 month) → father depression (12 months) via father sleep	*.04*	*.01*	*.001***
Mother depression (1 month) → father depression (12 months) via mother sleep	*.03*	*.01*	*.01***

Statistically significant results are italicized. All sleep measures were assessed at the 6-month postpartum visit

** $p < .01$; *** $p < .001$; + $p \leq .10$

associations in sleep and depression. Taken together, these results suggest that the quantity and quality of sleep are associated with the course and stability of depressive symptoms over the first year postpartum. Interestingly, parenting practices and birth outcomes that might be expected to affect sleep—such as breastfeeding, cosleeping with the baby, C-section delivery, and infant low birth weight—were not correlated with sleep for either parent. Indeed, depressive symptoms were more strongly linked with sleep than any other covariate. These findings fit with recent research identifying sleep as a critical health behavior influencing inflammation, mood, and long-term health [29].

Not only do our findings highlight the importance of sleep in postpartum mood and depression for both parents, but they also point to an important and previously overlooked potential mechanism of transmission of depressive symptoms from mothers to fathers. Men whose partners have postpartum depression are known to be at higher risk of developing depression themselves [5]. In this study, we found that mothers with more symptoms of depression reported more sleep problems, which in turn contributed to fathers' depressive symptoms at 6 months postpartum. This suggests an important potential pathway linking new mothers' depressive symptoms to their partners. Considering that many new parents share a bed and participate jointly in nighttime parenting, it is not surprising that fathers' and mothers' sleep quality ratings were significantly associated or that cross-partner effects emerged. Postpartum depression in mothers has received considerable attention and may affect infant care and bonding, parental relationships, and the family as a whole [1]. Our findings propose a mechanism by which such effects take place, namely, via maternal sleep problems. While sleep disruption has been viewed as an unfortunate (and often short-lived) consequence of early infant care, these findings may spur recognition that it is actually a significant risk factor for families.

Our findings suggest that sleep problems in new parents warrant attention and may exacerbate depression. Unlike many other antecedents of depressive symptoms, problems with sleep duration and disturbance can be effectively treated behaviorally via approaches such as cognitive behavioral therapy for insomnia (CBT-I, 30) and stress reduction techniques including mindfulness meditation-based interventions [30]. Sleep medications may be contraindicated for breastfeeding mothers and for parents who bedshare with infants. However, behavioral and lifestyle-based treatments for sleep problems may be effective substitutes. For example, CBT-I has been shown to improve sleep quality when used either singly or in combination with medication, with lower remission rates when used without medication [31]. Adding a CBT-I component to depression treatment (in non-postpartum individuals) improved both depression and insomnia [32]. In other words, CBT-I can be used as an adjunct treatment even in the absence of a formal diagnosis of insomnia and when the primary treatment focus is another mental health disorder [33].

Sleep problems are likely more complex and multi-determined in new parents than in other populations, because of the presence of an "external stimulus" (the infant) to disrupt sleep. However, approaches such as mindfulness meditation and CBT-I are appropriate treatments even when sleep problems are fueled by outside sources. CBT-I has been used for secondary insomnia and comorbid insomnia (insomnia fueled by outside causes such as chronic pain and illness) as well as for primary insomnia [34]. CBT-I has also been used successfully within the postpartum period [35] and during pregnancy [36]. CBT-I targets cognitions and behaviors that exacerbate pre-existing sleep difficulties, such as parents becoming hypervigilant or over-responsive to their infants' night-time cues or catastrophizing about the potential consequences of sleep loss. These compensatory responses can heighten anxiety and make it more difficult for parents to get back to sleep after interruption. As with CBT-I, mindfulness approaches can also help reduce the anxiety that results from chronic sleep disturbance and facilitate more flexible sleep. Mindfulness has been used to treat secondary and comorbid insomnia, e.g., insomnia associated with cancer [37]. Mindful yoga has been adapted to treat sleep problems in pregnancy [38].Behavioral treatments can be successfully modified for the unique needs of new parents. The CBT-I treatment used with postpartum women [35] was adapted for this population, with modifications including that women could bring their infants to sessions, more flexibility with prescribed bed/wake times, and sleep hygiene education incorporating considerations related to infant care (e.g., reducing light exposure when waking up with the baby at night). Women were also encouraged to enlist their partners' participation in treatment. Similarly, the CBT-I protocol used with pregnant women [36] incorporated information about sleep-related changes that are common to pregnancy, such as needing to use the bathroom more often at night, and sleep restriction guidelines were modified so that women did not restrict their sleep window too severely. Psychoeducation about techniques to improve sleep and reduce stress might be made routinely available to all new parents, particularly parents at heightened risk of postpartum mood disorders, as a form of primary prevention. Parents might also be encouraged to test the compensatory value of short-term solutions, such as taking daytime naps when their infants sleep or another caregiver is present. Similarly, expectant parents should be educated about the importance of sleep and the reciprocal relationships between sleep and mood. Our results suggest that addressing sleep difficulties in new mothers may also reduce risk for their partners as well. Moreover, close relationship functioning may be associated with sleep quality: a recent study found that supportive social ties, including the relationship with one's partner, were associated with better sleep quality in a community sample of adults [39]. Further research can extend these findings by examining associations between relationship quality, sleep, and depression among new parents, who may be at risk for relationship strife in addition to sleep difficulties.

This paper has a number of limitations. Sleep was assessed only once at the 6-month visit and with a brief (three-item) self-

report measure that assessed only sleep duration and disturbance over the previous month, rather than a more comprehensive diagnostic measure of insomnia. Repeated assessments and objective measures via actigraphy or in vivo sleep monitoring might be used to improve precision in future studies, since a singly administered self-report measure may be subject to bias or error. The fact that sleep was only measured at the 6-month visit also limits interpretation of our 6-month longitudinal model, since sleep and depressive symptoms at 6 months postpartum were measured at the same study visit. An ideal test of mediation would involve temporal sequencing [40]. Thus, the 12-month longitudinal model results should be considered more definitive. Our sleep measure was shortened rather than using the full version of the PSQI because sleep was not a major focus of the larger study. Measures for the study were designed through community participatory methods involving community stakeholders and academic researchers with a guiding principle to limit participant burden and prioritize other goals. We also did not have data on naps or sleep medication use, which would have been useful covariates. Moreover, infant bedsharing was only assessed at T1, and other measures of infant sleep (e.g., frequency and duration of night-wakings) were not included in this study. Given that infant sleep patterns contribute to postpartum sleep disturbance, more comprehensive measures of infant sleep and cosleeping or bedsharing practices would enhance future work. Another limitation is that not all couples included in the study were cohabiting consistently throughout the first year of parenthood. We reasoned that couples who were parenting a child together and were romantically involved at both one and 6 months postpartum were likely to share the same sleep environment at least some of the time during the first 6–12 months of parenthood. Furthermore, cohabitation status did not emerge as a significant covariate in any of our analyses, suggesting that it did not confound the results. Related to this limitation, we focused on a sample that is low-income and had a greater percentage of minorities and non-cohabiting parents than the general population. As such, this sample might have been exposed to more stressors than a more representative sample, possibly affecting the pattern of results that we found.

These limitations are mitigated by the large and socioeconomically and racially diverse sample of both fathers and mothers from five US regions and by repeated assessments of parental depression at 1, 6, and 12 months, as well as inclusion of many potential covariates and confounds. It is striking that, even with a brief sleep measure, we found stronger effects for sleep and depression than any other covariate we tested. Despite its limitations, this study contributes to the literature as one of the largest and most thorough investigations of the role of sleep in postpartum depression. This paper is also unique in its inclusion of fathers, its modeling of associations between both parents, and its identification of sleep as a potential pathway linking mothers' depressive symptoms to fathers. Future studies should continue to include fathers along with mothers. Although fathers' depressive symptoms in the postpartum period have not been studied as extensively as mothers', fathers do appear to experience increases in depressive symptoms after the birth of a child that are linked with mothers' depression and that have consequences for children's health and development [41]. Therefore, addressing paternal as well as maternal depression is important to improving child and family outcomes over the early years of parenthood.

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Authors' Statement of Conflict of Interest and Adherence to Ethical Standards Authors Darby E. Saxbe, Christine Dunkel Schetter, Christine M. Guardino, Sharon L. Ramey, Madeleine U. Shalowitz, John Thorp, Maxine Vance, and Eunice Kennedy Shriver National Institute for Child Health and Human Development Community Child Health Network declare that they have no conflict of interest. All procedures, including the informed consent process, were conducted in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000.

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