Journal of Pediatric Psychology, 45(1), 2020, 1–11
doi: 10.1093/jpepsy/jsz079
Advance Access Publication Date: 21 October 2019
Original Research Article

Higher Rates of Sleep Disturbance Among Offspring of Parents With Recurrent Depression Compared to Offspring of Nondepressed Parents

Jessica L. Hamilton [1] PhD, Cecile D. Ladouceur,[1,2] PhD, Jennifer S. Silk,[1,2] PhD, Peter L. Franzen,[1] PhD, and Lauren M. Bylsma,[1,2] PhD

[1]Department of Psychiatry, University of Pittsburgh and [2]Department of Psychology, University of Pittsburgh

All correspondence concerning this article should be addressed to Lauren M. Bylsma, PhD, 3811 O'Hara Street, Pittsburgh, PA 15213, USA. E-mail: BylsmaL@pitt.edu

Received May 23, 2019; revisions received September 14, 2019; accepted September 17, 2019

Abstract

Objective Youth who have a parent with recurrent depression are at high risk for mental health problems. There is a need to identify transdiagnostic and clinically actionable mechanisms that explain higher rates of psychopathology among high-risk youth. The present study sought to examine whether offspring of depressed parents exhibit greater parent- and self-reported sleep disturbance, shorter sleep duration, and later sleep midpoint compared to youth without any parental psychopathology. **Method** Participants included 82 youth, including 41 youth (ages 9–13; mean age = 11.07 years; 46% female) deemed to be at high-risk based on having a parent with a recurrent depression history, and 41 (mean age = 11.16 years; 49% female) at low-risk based on having parents without any history of psychopathology. Youth and their parents completed measures of youth sleep disturbance, and youth completed measures of sleep duration and midpoint using a daily sleep diary for 9 days. **Results** Offspring of parents with depression exhibited more sleep disturbance (e.g., problematic nighttime behaviors and daytime sleepiness) than low-risk youth as reported by both parents and youth. For parent-reported sleep disturbance, there were also sex differences. High-risk girls had more sleep disturbance than high-risk boys or low-risk girls. There were no group differences for daily sleep duration and midpoint. **Conclusion** Sleep disturbance may be an important area for assessment among offspring of parents with depression. Our findings highlight one potential transdiagnostic risk factor that may emerge among high-risk youth, and sex-specific differences in sleep disturbance, which have implications for prevention and intervention.

Key words: depression; high-risk; sleep disturbance; sleep duration; sleep midpoint; offspring.

Introduction

Adolescence is a developmental period of heightened risk for first onset of psychopathology, including depression and suicidal thoughts and behaviors (Hankin et al., 1998). Parental depression is one of the most robust risk factors for developing depression in childhood and adolescence (Weissman et al., 2016), with a three- to fourfold increased risk for depression and suicidality in offspring of depressed parents than youth without depressed parents (Kovacs & Lopez-Duran, 2010; Weissman et al., 2016). Parental depression not only confers risk for offspring in developing

depression, but also increases the likelihood of youth experiencing other disorders, such as anxiety, behavioral problems, and substance use (Lieb, Isensee, Hofler, Pfister, & Wittchen, 2002). Although a range of factors have been identified as potential mechanisms linking parental depression to offspring psychopathology (Goodman & Gotlib, 1999), including genetic heritability, impaired emotion regulation (Silk, Shaw, Skuban, Oland, & Kovacs, 2006), and family functioning (Daches, Vine, Layendecker, George, & Kovacs, 2018), there remains a need to identify risk factors that are developmentally informed, transdiagnostic, and modifiable to aid in early prevention and intervention efforts among high-risk offspring.

Sleep disturbances have received increasing attention as a promising target of prevention and intervention for psychiatric disorders (Harvey, 2009; Harvey et al., 2016). This is particularly relevant during adolescence when sleep undergoes significant developmental changes (Carskadon, 2011). Biological shifts in sleep, including changes in the circadian system that delay onset of melatonin, and slower build-up of the homeostatic "sleep" drive, contribute to an increased biological preference toward eveningness among adolescents (Hagenauer, Perryman, Lee, & Carskadon, 2009). Increased psychosocial demands, such as academic pressure, extracurricular activities, and late-night socializing, all combine to push sleep toward both biological and behaviorally induced later bedtimes and sleep midpoints (Carskadon, 2011; Hagenauer et al., 2009; Wong, Hasler, Kamarck, Muldoon, & Manuck, 2015). School start times remain early and typically shift even earlier as youth transition to middle school and again to high school. This combination of biological and environmental factors results in the majority of youth receiving less than 8 hrs of sleep per night and falling short of the recommended 9–11 hrs for youth ages 10–13 and 8–10 hrs of sleep for youth ages 14–17 (Hirshkowitz et al., 2015). Insomnia and sleep disturbance also increase across adolescence, with girls more likely to develop insomnia (Johnson, Roth, Schultz, & Breslau, 2006). Given developmental changes related to sleep duration (e.g., total time asleep), sleep midpoint (i.e., middle clock time of the sleep cycle), and sleep disturbance (e.g., difficulty falling and staying asleep, problematic nighttime behaviors, and daytime sleepiness), targeting these aspects of poor sleep may be critical for mental health prevention. Indeed, studies indicate that poor sleep precedes and predicts the onset of psychopathology (Hertenstein et al., 2019; Zhang et al., 2017). In particular, short sleep duration, later sleep midpoint, and sleep disturbance predict depression (Clarke & Harvey, 2012; Roberts & Duong, 2014), anxiety (McMakin & Alfano, 2015), and suicidality (Liu et al., 2019).

Offspring of parents with depression have higher rates of psychiatric disorders. Poor sleep may be a candidate mechanism that explains the development of higher rates across psychiatric disorders among these vulnerable youth. Specifically, parents with depression may directly or indirectly contribute to poor sleep in their offspring through genetic risk, parenting practices (e.g., bedtime routine; modeling behavior), and/or associated environmental factors (e.g., stress, inadequate sleeping environment) that disrupt or impede healthy sleep (Goodman & Gotlib, 1999; Hall & Nethery, 2018). High-risk youth (i.e., offspring of depressed parents) may not only be more likely to develop sleep problems, but also more vulnerable to the cognitive and affective consequences of poor sleep (Palmer & Alfano, 2017). Indeed, several studies identify differences in sleep between offspring of parents with and without depression (Chen, Burley, & Gotlib, 2012) and risk for later disorder (Silk et al., 2007). Using actigraphy, questionnaires, and diary reports in 44 girls (10–16 years old) with and without maternal depression, high-risk girls reported poorer subjective sleep quality than low-risk girls, but there were no differences between actigraphy-derived or diary-reported sleep measures. This finding suggests that there only may be a subjective, but not objective, difference in sleep (Chen et al., 2012), reflecting a potential cognitive bias among high-risk offspring (Gobin, Banks, Fins, & Tartar, 2015). However, objective indices of sleep may differentiate risk for psychopathology over time among offspring of parents with mood disorders (Silk et al., 2007; Soehner et al., 2019). A longitudinal study of 14 high-risk girls found that those with longer sleep onset latency (derived from electroencephalography records) were more likely to have onset of major depression in young adulthood (Silk et al., 2007). A second longitudinal study of offspring of parents with bipolar disorders found that changes in sleep patterns (e.g., shorter sleep duration, later sleep timing, longer sleep latency, nighttime awakenings, and greater daytime sleepiness) also experienced increases in psychiatric symptoms over time (Soehner et al., 2019).

Together, these studies highlight the importance of examining sleep among offspring of parents with depression. Notably, however, research is needed to examine offspring *prior* to the onset of psychopathology to determine whether sleep problems are present prior to disorder rather than simply as a correlate. Given differences in development and psychiatric risk between adolescent girls and boys, with girls more at risk (Zahn-Waxler, Shirtcliff, & Marceau, 2008), it is also important to examine sex differences in sleep characteristics between high and low risk youth. Identifying potential sex differences between female and male high-risk youth may reveal unique or shared risk pathways and targets for prevention. Thus, the

current study sought to test the hypothesis that high-risk youth (based on parents with recurrent depression) would exhibit impaired sleep patterns, including shorter duration, later midpoint, and more disturbance using questionnaires and sleep diaries, which may provide better estimates of sleep patterns than single items at one point in time. As an exploratory aim, we also explored potential sex differences in these processes. Building upon past research, the current study offers a unique contribution by examining both female and male offspring of parents with recurrent depression (including both biological mothers and fathers), younger offspring than prior studies, and assessment of multiple sleep characteristics by both sleep diary and parent- and youth-reported self-report measures.

Method

Study Recruitment and Participants

Participants included 82 youth (aged 9–13, 46% female) determined to be at high or low risk based on having at least one biological parent with recurrent depression (2 or more episodes; 88% were mothers). Participants were recruited as part of a larger study on neurobehavioral indices of emotional functioning and depression risk (K01MH104325). Exclusion criteria for all participants included parental history of bipolar disorder, mania, or psychosis. Low-risk youth had parents without a lifetime history of any psychopathology. Youth exclusion criteria included a lifetime diagnosis of a depressive disorder, pervasive developmental disorder, intellectual disability, history of substance abuse/dependence, or a serious head injury or neurological condition. High- and low-risk youth and parents were recruited through local advertising methods online and in print (e.g., websites, research registries, psychiatric clinics, and email listservs). All procedures were approved by the local Institutional Review Board. Parents and youth gave consent and assent prior to the study procedures and received compensation for their time and participation.

Parents completed a screening by phone, which included questions about parental and child mental health history. Participants (parent and child) were then scheduled for their first laboratory visit to participate in a clinical diagnostic assessment with the child and one of his or her parents by trained clinical interviewers (Kiddie Schedule for Affective Disorders and Schizophrenia [K-SADS-PL]; Kaufman et al., 1997 and Structured Clinical Interview for DSM-IV [SCID]; Spitzer, Williams, Gibbon, & First, 1992). In the current study, interviewer reliability for depression diagnosis was excellent (kappa > .9). See Supplementary Table 1 for a summary of sample recruitment and procedures. The final sample included 41 (46% female) high-risk and 41 (49% female) low-risk youth with at least one sleep diary completed and valid data for sleep onset and offset times. Table I includes demographic information for the overall sample and by youth high- and low-risk status. There were no significant differences between participants included in the present study and those in the larger sample.

Procedure

Following the clinical interview to confirm eligibility, parents and youth completed questionnaires, including youth sleep disturbance (completed by parent and child) and youth internalizing symptoms. Youth then completed a 9-day ecological momentary assessment battery for five weekdays and four weekend days, which included a morning sleep diary. The protocol was administered using a custom app installed on study-provided Android smartphones. Participants were instructed on how to use the devices and the study application. Sleep items were completed at the morning battery, which youth were instructed to complete within 1 hr of waking on weekdays (mean completion time = 7: 39 a.m.) and weekend days (mean = 8: 49 a.m.). On average, youth completed 7.65 sleep diaries (85%) out of 9 possible diaries, of which 4.58 days (out of 5) were weekdays. Total number of sleep diaries was not correlated with study outcomes (r's < .15).

Measures

Sleep Disturbance

Parents and youth each completed measures assessing youth sleep disturbance. Parents completed the abbreviated version of the Children's Sleep Habits Questionnaire (CSHQ-A; Bonuck, Goodlin-Jones, Schechter, & Owens, 2017; Owens, Spirito, & McGuinn, 2000), which is a 22-item questionnaire assessing behavioral sleep problems. It includes a total score, which is comprised of 6 subscales: bedtime resistance, sleep duration, sleep anxiety, sleep onset latency, daytime sleepiness, and behaviors around sleep and night awakenings. The modified short-form version has been validated and demonstrated excellent psychometric properties (Bonuck et al., 2017). Item responses range from 1 (never) to 5 (always), with total scores ranging in the current study from 25 to 61. Youth completed the Sleep Self Report (SSR; Owens, Spirito, McGuinn, & Nobile, 2000). Similar to the CSHQ, the SSR includes 26-items assessing common behavioral sleep problems, including bedtime sleep behaviors, nighttime sleep behaviors, and daytime sleepiness. Item responses range from 1 (rarely) to 3 (usually), with total scores ranging in the current study from 27 to 64. In the current study, we used the total score of the CSHQ and SSR to assess overall sleep disturbance rather than individual subscales due to lower, inadequate reliability among several subscales (α =.42–.76). Higher total scores indicate more sleep

Table I. *Descriptives of Primary Study Variables for the Overall Sample and by Risk Status*

Measure	Overall sample ($N = 82$)		High-risk ($N = 41$)		Low-risk ($N = 41$)		Statistical test
	M (N)	*SD (%)*	*M (N)*	*SD (%)*	*M (N)*	*SD (%)*	*t* (χ^2)
Demographic							
Sex (Female)	39	48%	19	46%	20	49%	−.01
Race							1.74
White	41	50%	23	56%	17	41%	
Black/African American	33	40%	16	39%	15	37%	
Age	11.12	1.45	11.07	1.52	11.16	1.40	−.28
SES	22	27%	15	37%	7	17%	5.79*
PDS	2.82	1.10	2.95	1.08	2.70	1.12	1.06
Sleep							
Duration	9.08	.95	9.15	.92	9.02	.92	.64
Midpoint	3.60	1.25	3.96	1.15	4.03	1.26	1.68
SSR	38.25	7.55	41.21	8.15	35.65	5.85	3.63***
CSHQ	37.80	8.58	41.36	9.27	35.05	6.62	3.41***
Internalizing symptoms							
SCARED	14.76	11.44	18.00	12.90	11.71	8.65	2.65**
MFQ	8.38	10.45	9.81	10.48	6.95	10.42	.21
Clinical cutoffs							
CSHQ	21	26%	15	37%	6	15%	4.80
SCARED	12	15%	9	22%	3	7%	1.71
MFQ	7	9%	5	12%	2	5%	1.03

Note. *$p < .05$, **$p < .01$, *** $p < .001$. Youth who did not identify as White or Black/African American identified as biracial ($N = 3$), Asian American ($N = 1$), Pacific Islander ($N = 2$), or Native American ($N = 2$). SES = Socioeconomic status, which was assessed based on Receipt of Public Assistance (Yes = 1; No = 0); PDS = Pubertal Development Scale; SSR = Sleep Self Report (youth report of sleep); CSHQ = Children's Sleep Habits Questionnaire (parent report of youth sleep); SCARED = Screen for Child Anxiety-Related Emotional Disorders; MFQ = Mood and Feelings Questionnaire. Risk Status is coded 0 (low risk) and 1 (high risk). Clinical cutoff scores reflect scores that are: CSHQ ≥ 41, SCARED ≥ 30, MFQ ≥ 27 (there is no validated clinical cutoff score for the SSR). Sleep duration and midpoint were averages over the 9-day stud period calculated using sleep onset and offset times from sleep diaries.

disturbance for both measures. Internal reliability for the total scale for both measures was adequate ($\alpha >$ .80).

Sleep Duration and Midpoint

On morning sleep diaries, youth reported the timing of sleep onset ("About what time did you go to sleep last night?") and sleep offset ("About what time did you wake up this morning?"), which is standard for sleep diaries. These scores were used to calculate a proxy of sleep duration (e.g., difference between sleep offset and onset times). The middle clock time between sleep on and offset times was used to defined the midpoint of sleep cycle, which correlates with chronotype and dim light melatonin onset (Kantermann, Sung, & Burgess, 2015). Self-reported sleep has demonstrated modest to strong correlations with actigraphy-derived sleep parameters among youth (Wolfson et al., 2003).

Youth Internalizing Symptoms

Youth reported their depression symptoms using the 33-item Mood and Feelings Questionnaire (MFQ)-Long Form (Angold et al., 1995) and anxiety symptoms using the 41-item Screen for Child Anxiety-Related Emotional Disorders (SCARED; Birmaher et al., 1997, 1999). The sleep items were removed from the total scores of both measures to avoid confounds in assessing their association. Item responses range from 0 to 2. Scores ranged from 0 to 47 for the SCARED and 0 to 61 for the MFQ, with higher scores indicating higher levels of anxiety and depressive symptoms. Though these measures were treated as continuous variables in the current study, we included the percentages of clinical cutoffs for the SCARED (≥ 30; Birmaher et al., 1999) and MFQ (≥ 27; Daviss et al., 2006). Internal reliability for the MFQ ($\alpha = .96$) and SCARED scales with the sleep items removed was excellent ($\alpha = .92$).

Puberty

The Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988) assessed youth pubertal maturation, which is a well-validated measure of self-reported pubertal development. Scores were calculated using a coding system that parallels the Tanner Stages and converts the PDS to a 5-point scale (Shirtcliff, Dahl, & Pollak, 2009), with higher scores indicative of more pubertal maturation. Pubertal development is linked with sleep (Colrain & Baker, 2011), thus, pubertal maturation was covaried in analyses.

Parent-Set Bedtime

Given links between parent-set bedtime and youth sleep patterns (Short et al., 2011), we covaried for parent-set bedtime. Using a single-item on the youth sleep measure (SSR; "Who in your family sets the rules about when you go to bed?"), responses were dichotomized to reflect parent-set or child-set bedtimes (Short et al., 2011).

Statistical Analyses

First, descriptive statistics and bivariate correlations between the primary variables were examined, and *t*-tests were conducted to examine demographic differences by risk status for sex, age, and race. We also examined sleep variables by timing of when they were assessed (school-break/school year), and conducted paired samples *t*-tests to examine weekday-weekend differences for sleep duration and midpoint. In analyses, we covaried pubertal development, age, sex, SES (indicated by receipt of public assistance), youth depression and anxiety symptoms, and whether parents set youth bedtimes, which are biological and environmental factors that can impact youth sleep (Hagenauer et al., 2009; Short et al., 2011). We also covaried completion of the study during the school-break or school-year for analyses predicting sleep duration and midpoint. For analyses that examined whether sleep disturbance was associated with youth risk status, we conducted path analysis with parent and child-reported sleep disturbance as the primary outcomes in Mplus 7.0 (Muthén & Muthén, 2007). Sleep disturbance reported by parents and youth were endogenous variables, allowed to covary to determine correlations.

To determine whether youth risk status predicted sleep midpoint and duration reported on the sleep diary, we conducted two-level multilevel modeling in Mplus 7.0 with Full Information Maximum Likelihood to estimate parameters for missing data to maximize data. Sleep was reported daily (within-person data) and predictors were between-person. Fixed effects were entered for all covariates and risk status (group) predicting sleep parameters. For a parsimonious model, sleep midpoint and duration were estimated simultaneously and allowed to covary. Random effects were included for the intercept of sleep parameters. Supplementary analyses were conducted for weekdays and weekends (results provided in Supplementary Tables 2 and 3). For exploratory analyses examining sex differences, we added an interaction term between risk status and sex to path and multilevel models. When there was evidence of a significant interaction, we probed the interaction for girls and boys by risk status and plotted the results.

Results

Table I presents descriptive statistics of primary study variables for the overall sample and by risk status, as well as percentage of youth above clinical cutoffs for sleep disturbance and internalizing symptoms. Bivariate correlations are provided in Supplementary Table 1. Both parent and child reports of sleep disturbance were correlated, signifying convergence of reports. On average, participants reported 9.08 (SD = .10; range: 6.9–12.56) hours of sleep per night. However, 37% of youth under 13 reported less than the recommended 9 hrs of sleep on weekdays, 64% of all youth reported at least one night less than 8 hrs, and 35% reported at least one night less than 7 hrs. The average sleep midpoint was 3: 38 a.m., with youth ranging from 1: 58 a.m. to 8: 11 a.m. Sleep midpoint was later on weekends than weekdays ($t(81) = 8.87$, $p < .001$) and sleep duration was longer ($t(81) = 2.55$, $p < .001$). Youth who completed the study during a school-break had later sleep midpoints ($B = 1.56$; $SE = .26$; $p < .001$), but there was no effect on sleep duration or disturbance. Most youth (66%) completed the study during the school year; high-risk youth were more likely to complete the study during the school-break. Ten youth reported that they set their own bedtimes (7 low-risk youth and 3 high-risk). There were no significant differences by sex on primary demographic variables (age, puberty, sleep, or internalizing symptoms, sleep; p's $> .05$); however, high-risk youth had lower SES.

Child Risk Status Predicting Sleep Disturbance, Duration, and Midpoint

Our model fit for path analysis was saturated. Consistent with hypotheses, we found a significant main effect of risk status for sleep disturbance as reported by *both* high-risk youth and their parents (Table II). Of note, these results simultaneously examined parent- and child-reported sleep disturbance, and covaried for youth age, sex, SES, puberty, parent-set bedtimes and internalizing symptoms. Our model accounted for 24.5% of the total variance in parent-reported sleep disturbance and 44.9% of child-reported sleep disturbance; risk status accounted for 6.5% and 5.3%, respectively. Several covariates (age, depression) predicted youth sleep disturbance, with younger youth and those with elevated symptoms reporting more sleep disturbance, whereas SES (receipt of public assistance) predicted parent-reported sleep disturbance.

For the daily sleep variables, sleep duration and sleep midpoint were significantly correlated at the within-person level ($B = -.28$; $SE = .07$; $p < .001$) and at the between-person level ($B = -.44$; $SE = .14$; $p = .002$). There were no main effects of risk status on the intercept of sleep midpoint or duration over the 9-day study period (Table III), controlling for age, sex, pubertal status, SES, school break, and youth internalizing symptoms. Similarly, there were no significant

Table II. *Parent and Youth Reports of Sleep Disturbance for High- and Low-Risk Youth: Main Effects of Risk Status and Interaction of Risk Status and Sex*

Variable	Parent-report (CSHQ)				Child-report (SSR)			
	β	CI	*SE*	*B/SE*	*β*	CI	*SE*	*B/SE*
Intercept	3.67	1.66–5.68	1.03	3.57***	5.55	3.90–7.20	.08	6.58***
Sex	−.17	−.38 to .05	.11	−1.49	−.04	−.21 to .14	.09	−.39
Age	−<.01	−.27 to .26	.13	−.02	−.22	−.42 to −.01	.11	−2.06*
SES	.26	.03–.48	.11	2.24*	.06	−.12 to .24	.09	.65
PDS	.15	−.23 to .41	.14	1.09	.16	−.05 to .37	.11	1.50
MFQ	−.02	−.28 to .23	.13	−.17	.29	.08–.50	.11	2.70**
SCARED	<.01	−.27 to .28	.14	.02	.22	−.01 to .46	.12	1.89
Bedtime	.06	−.15 to .27	.11	.52	.02	−.16 to .19	.09	.19
Risk status	.28	.06–.50	.11	2.54*	.26	.08–.44	.09	2.79**
Interaction								
Sex	−.40	−12.45 to −1.53	.15	−2.57*	−.12	−5.37 to 1.76	.12	−.99
Risk status	1.04	3.56–14.71	.30	3.44**	.15	−1.67 to 6.40	.13	1.15
Risk × sex	−.36	−14.46 to −.15	.18	2.02*	.16	−2.37 to 8.04	.15	1.07

Note. $*p < .05$, $**p < .01$, $***p < .001$. *β* represents the standardized parameter estimates and *SE* represents the standard error from path analyses. CI = Confidence Interval (95%); SSR = Sleep Self Report (youth report of sleep); CSHQ = Children's Sleep Habits Questionnaire (parent report of youth sleep); SES = Socioeconomic status (receipt of public assistance); PDS = Pubertal Development Scale; MFQ = Mood and Feelings Questionnaire; SCARED = Screen for Child Anxiety-Related Emotional Disorders; Bedtime = Parent-set bedtime. Risk status is coded 0 (low risk) and 1 (high risk); Sex is coded as 0 = female, 1 = male.

effects when separately examined by weekday or weekend days (Supplementary Tables 2 and 3). This finding is in contrast to our hypothesis that high-risk youth would have shorter sleep duration and later sleep timing. Several covariates, school break and SES, significantly predicted later sleep midpoint. There was significant variability observed within-individual (level 1) and between-individual (level 2) in sleep duration and midpoint (Table III), indicating that other factors may explain individual variability in sleep duration and midpoint.

Sex Differences in Risk Status Predicting Sleep Domains

For exploratory analyses examining whether high and low risk youth demonstrated distinct sleep disturbances by sex, we only found significant interactions of sex and risk status for parent-reported sleep disturbance (Table II). Probing this interaction (Figure 1), high-risk girls had more parent-reported sleep disturbance than low-risk girls ($B = .36$; $B/SE = 3.35$; $p = .001$) and high-risk boys ($B = .40$; $B/SE = 2.57$; $p = .01$). There were no significant interactions for sleep duration or midpoint (Table III). There were still significant individual variability and fluctuations from sleep midpoint and sleep duration (i.e., random intercept).

Discussion

Offspring of parents with recurrent depression are at heightened risk for mental health problems (Weissman et al., 2016), including earlier onset, poorer course, and more severe depression (Lieb et al., 2002). Using a multimethod approach of parent and child-reported sleep disturbance and 9-day sleep diary-based measures of sleep duration and sleep midpoint, our study examined differences between offspring of parents with recurrent depression and offspring of parents without psychopathology. Our findings indicate that per both self- and parent-report, high-risk youth had more sleep disturbance than low-risk youth, controlling for demographic characteristics (age, sex, pubertal status, SES, and timing of study completion) and youth internalizing symptoms. In contrast, there were no significant differences between high- and low-risk youth on sleep duration and midpoint assessed via sleep diary. Further, our study is the first to include both male and female offspring of parents with recurrent depression and examine potential sex differences. Our exploratory analyses found that high-risk female offspring had more parent-reported sleep disturbance compared to high-risk boys and low-risk girls. There were no sex differences for child-reported sleep disturbance, duration, or midpoint.

Overall, our findings support prior research that subjective reports of sleep disturbance but not sleep duration or midpoint, differentiate high- and low-risk youth based on parent history of depression, even prior to youth onset of psychopathology (Chen et al., 2012). These findings suggest that there may be specific differences between high and low risk youth on sleep disturbance, which broadly encompasses prebedtime and night-time behaviors (e.g., difficulty falling/staying asleep, nightmares) and daytime sleepiness. High-risk youth may be more likely to have sleep

Table III. *Multilevel Models of Daily Sleep Duration and Sleep Midpoint for High- and Low-Risk Youth: Main Effects of Risk Status and Interaction of Risk Status and Sex*

Variable	Sleep duration			Sleep midpoint		
Between-person fixed effects	*B*	*SE*	*B/SE*	*B*	*SE*	*B/SE*
Intercept	9.60	1.04	9.25***	1.17	1.04	1.12
Age	−.04	.11	−.36	.07	.11	.71
SES	.10	.29	.34	.57	.29	1.98*
School break	−.15	.27	−.54	1.40	.27	5.12***
PDS	.03	.14	.19	.21	.14	1.54
MFQ	.01	.01	.87	−.02	.01	−1.03
SCARED	<.01	.01	−.01	.02	.01	1.60
Parent-set bedtime	−.19	.14	−1.39	−.04	.14	−.27
Sex	−.09	.26	−.36	.21	.26	.84
Risk status	.03	.26	.11	.13	.27	.48
Interaction						
Sex	−.56	.38	1.50	.28	.39	.72
Risk status	.42	.35	1.20	.18	.36	.51
Risk × sex	−.80	.48	1.65	.113	.50	.23
Random effects						
Within-Person (σ^2)	2.57	.16	15.91***	1.04	.07	15.89***
Between-Person (τ_{00})	.65	.16	4.05***	.87	.16	5.27***

Note. $^*p < .05$, $^{**}p < .01$, $^{***}p < .001$. *B* represents the unstandardized parameter estimates and *SE* represents the standard error estimated from multilevel models. Random intercepts were included in the model. PDS = Pubertal Development Scale; MFQ = Mood and Feelings Questionnaire; SCARED = Screen for Child Anxiety-Related Emotional Disorders; Risk Status is coded 0 (low risk) and 1 (high risk). Sex coded as Girls (0) and Boys (1). School break is coded 0 (school) and 1 (school break).

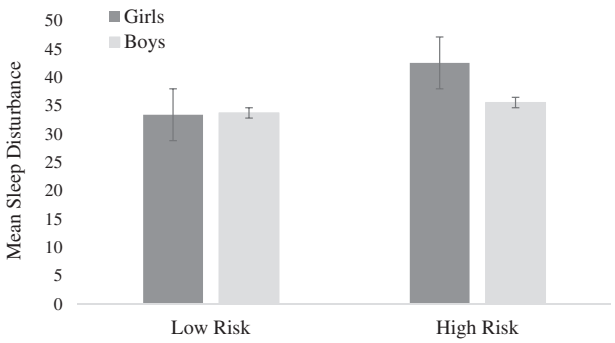

Figure 1. Average amount of parent-reported sleep disturbance by risk status and sex.
Note. Means depicted are the average amount of sleep disturbance reported on the Children's Sleep Habits Questionnaire (CSHQ).

disturbance due to genetic risk (as the parents were biologically related in the current study), parenting styles (Kopala-Sibley et al., 2017), emotion regulation impairments (Silk et al., 2006), and other environmental factors (e.g., stress, neighborhood environment) associated with parental depression (Marco, Wolfson, Sparling, & Azuaje, 2011). Indeed, in the current study, high-risk youth were more likely to receive public assistance, an indicator of SES, which may be associated with environmental factors that disrupt sleep such as inconsistent schedules, noise, and air pollution (Marco et al., 2011). These factors, along with vulnerability for emotion dysregulation, may also make high-risk youth more susceptible to both experience more sleep disturbance and the cognitive-affective impact of disturbed sleep (Karazsia & Berlin, 2018), thereby increasing risk for subsequent psychopathology.

Consistent with prior studies (Chen et al., 2012), our findings were specific to subjective measures of sleep quality, and extend these findings to both youth and parent-reported sleep disturbances. There may be several reasons for our study results. First, our findings may suggest that high-risk offspring have more biased cognitive information processes and attend more to negative information (Sfärlea et al., 2019). Although depressed parents also reported more offspring sleep disturbance, particularly for females, this finding may reflect shared cognitive and affective biases among parents and offspring that contribute to perception of poor sleep quality. Further, depressed parents are more likely to have disturbed sleep, which may contribute to higher levels of youth sleep disturbance or overestimation by parents of their child's sleep disturbance (Ronnlund, Elovainio, Virtanen, Matomaki, & Lapinleimu, 2016). Second, it could be that our sleep diary did not capture more habitual sleep patterns that reflect differences in sleep duration and midpoint between high- and low-risk youth. Our study was conducted continuously throughout the year, which may not fully capture critical school-year versus school-break differences in sleep and underestimate potential group differences. For instance, nearly 40% of the

sample had short weekday sleep (defined as < 9 hrs for youth under 13 years old), which is comparable to national averages (Wheaton, Jones, Cooper, & Croft, 2018), though sleep duration did not differ between youth who were studied in the school year versus school break. Importantly, there also was considerable variability within individuals on sleep duration and midpoint, suggesting other factors may be more important than risk status in determining these sleep characteristics. Third, it is also possible that sleep disturbances may be more closely linked to risk for psychopathology than sleep duration and midpoint. For instance, sleep disruptions such as delayed sleep onset latency or nighttime awakenings may be more problematic or indicative of future psychopathology among high-risk youth, whereas shorter sleep duration and midpoint may reflect more developmental patterns in sleep that affect most youth.

Importantly, our study examined sex differences in sleep among female *and* male offspring of parents with recurrent depression, which represents a unique contribution to the field. Our findings indicate that depressed parents reported higher levels of sleep disturbance specifically among female offspring, but there were no self-reported sleep differences by sex. While these findings may reflect sex-specific mechanisms of risk for high-risk girls compared to high-risk boys, there are other possible explanations for these findings. There may be sex differences in parental awareness of sleep disturbances for high-risk females due to more observable or verbalized disturbances (e.g., parents awakened in nighttime) or specific bias in parental perception of sleep among female offspring. For instance, it could be that parents (especially mothers given the study composition) may be more attune to the health behaviors of their female child given their own history of recurrent depression, and be more likely to notice specific difficulties in sleep behaviors or daytime sleepiness among female offspring. Given limitations in power and a relatively small sample size, it will be important for future studies to replicate these findings and estimate potential mechanisms through which high-risk girls may or may not have more sleep disturbances.

Our study has notable strengths, such as the use of both parent and youth report, sleep diary design, and high-risk youth of parental recurrent depression in a developmental stage of heightened risk. However, several methodological limitations should be noted. First, we did not record the timing of parents' most recent depressive episode, which limits the ability to determine whether youth were exposed to parental depression during the youth's lifetime. Second, paternal depression was relatively rare in our sample, which limited examination of differences between maternal and paternal depression and pathways of risk for offspring to sleep disturbances. Our sample also was relatively small, and we had limited power to detect associations for between-person effects of multilevel models, particularly sex differences. Interaction analyses should be considered exploratory, and future studies should be designed with sufficient power to test these hypotheses. This study also did not have objective measures of sleep to evaluate specificity of findings to subjective estimates of sleep. Several subscales of sleep measures had low internal reliability in our sample, which limited examination of specific areas of sleep disturbance. In our sample, younger youth were more likely to report sleep disturbance than older youth, which may reflect developmental differences or perceptions of sleep. Further, our sleep measures domains are not independent and may capture overlapping sleep characteristics (e.g., sleep disturbance includes sleep duration). It will be important for future research to examine specific aspects of sleep using multiple measures and methods, including actigraphy and the gold-standard of polysomnography to inform intervention targets.

Our findings identify differences in sleep disturbance between offspring of parents with depression, which may reflect a potential transdiagnostic risk factor for subsequent psychopathology (Harvey, 2009; Harvey et al., 2016). However, the current study did not evaluate this directly, which remains an important area of future investigation. Despite advances in the field and understanding of potential mechanisms of risk among high-risk youth, existing interventions are still limited in their prevention of disorder. Existing treatments primarily focus on cognitive and emotional skills (Loechner et al., 2018). Sleep disturbance impairs emotion regulation (Palmer & Alfano, 2017) and executive functioning skills that facilitate skill acquisition, consolidation, and recall (Walker, 2009), which may impair the ability of youth to learn and implement these cognitive-behavioral strategies. Thus, it is possible that targeting sleep may enhance existing intervention and prevention programs (Harvey, 2009; Harvey et al., 2016). Although existing literature of sleep within high-risk youth is emerging, a body of research indicates that sleep disturbance predicts a range of psychopathology (Dolsen, Asarnow, & Harvey, 2014), including risk for depression and suicidality (Liu et al., 2019). Thus, sleep disturbance may be an actionable target for prevention and intervention among high-risk youth because: (a) it is a risk factor associated with physical and mental health that is modifiable compared to other known factors (e.g., genetic risk), (b) high-risk youth may be even more susceptible to the impact of sleep, particularly given that these youth are more likely to have emotion dysregulation (Silk et al., 2006), and (c) there are well-validated and publicly available self-report sleep measures, such

as the CSHQ (Owens, Spirito, McGuinn, et al., 2000), that can be efficiently administered to identify sleep disturbance. Promoting healthy sleep practices and intervening on nighttime disturbances may be valuable targets among these vulnerable youth, with the long-term potential to improve early prevention and intervention efforts. Future research is needed to examine whether differences in sleep disturbance between high- and low-risk youth and sex differences within high-risk youth persist over time and confer risk for subsequent psychopathology. It will also be important to determine which aspects of sleep disturbance are present among high-risk youth and should be prioritized in treatments. Future studies are needed to examine whether targeting sleep disturbance among high-risk youth serves as a critical early intervention to prevent the intergenerational transmission of psychopathology (Soehner et al., 2019).

Acknowledgments

We would like to thank Amanda Adams and Lorraine Scott for their assistance with data collection for this project.

Supplementary Data

Supplementary data can be found at: https://academic.oup.com/jpepsy.

Funding

This work was supported by NIH grants to Jessica L. Hamilton (T32HL082610) and Lauren M. Bylsma (K01MH104325). This project was also supported by a NIH grant to the University of Pittsburgh Clinical and Translational Science Institute (UL1TR001857).

Conflict of interest: The authors have no conflicts of interest to declare.

References

Angold, A., Costello, E. J., Messer, S. C., Pickles, A., Winder, F., & Silver, D. (1995). The development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *International Journal of Methods in Psychiatric Research*, *5*, 237– 249.

Birmaher, B., Brent, D. A., Chiappetta, L., Bridge, J., Monga, S., & Baugher, M. (1999). Psychometric properties of the Screen for Child Anxiety Related Emotional Disorders (SCARED): A replication study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *38*, 1230–1236.

Birmaher, B., Khetarpal, S., Brent, D., Cully, M., Balach, L., Kaufman, J., & Neer, S. M. (1997). The Screen for Child Anxiety Related Emotional Disorders (SCARED): Scale construction and psychometric characteristics. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 545–553.

Bonuck, K. A., Goodlin-Jones, B. L., Schechter, C., & Owens, J. (2017). Modified children's sleep habits questionnaire for behavioral sleep problems: A validation study. *Sleep Health*, *3*, 136–141.

Carskadon, M. A. (2011). Sleep in adolescents: The perfect storm. *Pediatric Clinics of North America*, *58*, 637–647.

Chen, M. C., Burley, H. W., & Gotlib, I. H. (2012). Reduced sleep quality in healthy girls at risk for depression. *Journal of Sleep Research*, *21*, 68–72.

Clarke, G., & Harvey, A. G. (2012). The complex role of sleep in adolescent depression. *Child and Adolescent Psychiatric Clinics of North America*, *21*, 385–400.

Colrain, I. M., & Baker, F. C. (2011). Changes in sleep as a function of adolescent development. *Neuropsychology Review*, *21*, 5–21.

Daches, S., Vine, V., Layendecker, K. M., George, C. J., & Kovacs, M. (2018). Family functioning as perceived by parents and young offspring at high and low risk for depression. *Journal of Affective Disorders*, *226*, 355–360.

Daviss, W. B., Birmaher, B., Melhem, N. A., Axelson, D. A., Michaels, S. M., & Brent, D. A. (2006). Criterion validity of the Mood and Feelings Questionnaire for depressive episodes in clinic and non-clinic subjects. *Journal of Child Psychology and Psychiatry*, *47*, 927–934.

Dolsen, M. R., Asarnow, L. D., & Harvey, A. G. (2014). Insomnia as a transdiagnostic process in psychiatric disorders. *Current psychiatry reports, 16,* 471

Gobin, C. M., Banks, J. B., Fins, A. I., & Tartar, J. L. (2015). Poor sleep quality is associated with a negative cognitive bias and decreased sustained attention. *Journal of Sleep Research*, *24*, 535–542.

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review*, *106*, 458–490.

Hagenauer, M. H., Perryman, J. I., Lee, T. M., & Carskadon, M. A. (2009). Adolescent changes in the homeostatic and circadian regulation of sleep. *Developmental Neuroscience*, *31*, 276–284.

Hall, W. A., & Nethery, E. (2018). What does sleep hygiene have to offer children's sleep problems? *Paediatric Respiratory Reviews*, 64–74, doi:10.1016/j.prrv.2018.10.005.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology*, *107*, 128–140.

Harvey, A. G. (2009). A transdiagnostic approach to treating sleep disturbance in psychiatric disorders. *Cognitive Behaviour Therapy*, *38 (Suppl 1)*, 35–42.

Harvey, A. G., Hein, K., Dong, L., Smith, F. L., Lisman, M., Yu, S., . . . Buysse, D. J. (2016). A transdiagnostic sleep and circadian treatment to improve severe mental illness outcomes in a community setting: Study protocol for a randomized controlled trial. *Trials*, *17*, 606.

Hertenstein, E., Feige, B., Gmeiner, T., Kienzler, C., Spiegelhalder, K., Johann, A., . . . Baglioni, C. (2019). Insomnia as a predictor of mental disorders: A systematic review and meta-analysis. *Sleep Medicine Reviews*, *43*, 96–105.

Hirshkowitz, M., Whiton, K., Albert, S. M., Alessi, C., Bruni, O., DonCarlos, L., ... Ware, J. C. (2015). National Sleep Foundation's updated sleep duration recommendations: Final report. *Sleep Health*, *1*, 233–243.

Johnson, E. O., Roth, T., Schultz, L., & Breslau, N. (2006). Epidemiology of DSM-IV insomnia in adolescence: Lifetime prevalence, chronicity, and an emergent gender difference. *Pediatrics*, *117*, e247–256.

Kantermann, T., Sung, H., & Burgess, H. J. (2015). Comparing the Morningness-Eveningness Questionnaire and Munich ChronoType Questionnaire to the dim light melatonin onset. *Journal of Biological Rhythms*, *30*, 449–453.

Karazsia, B. T., & Berlin, K. S. (2018). Can a mediator moderate? Considering the role of time and change in the mediator-moderator distinction. *Behaviour Therapy*, *49*, 12–20.

Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., ... Ryan, N. (1997). Schedule for affective disorders and schizophrenia for school-age children-resent and lifetime version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*, 980–988.

Kopala-Sibley, D. C., Jelinek, C., Kessel, E. M., Frost, A., Allmann, A. E. S., & Klein, D. N. (2017). Parental depressive history, parenting styles, and child psychopathology over 6 years: The contribution of each parent's depressive history to the other's parenting styles. *Development and Psychopathology*, *29*, 1469–1482.

Kovacs, M., & Lopez-Duran, N. (2010). Prodromal symptoms and atypical affectivity as predictors of major depression in juveniles: Implications for prevention. *Journal of Child Psychology and Psychiatry*, *51*, 472–496.

Lieb, R., Isensee, B., Hofler, M., Pfister, H., & Wittchen, H.U. (2002). Parental major depression and the risk of depression and other mental disorders in offspring: A prospective-longitudinal community study. *Archives of Geneneral Psychiatry*, *59*, 365–374.

Liu, J.-W., Tu, Y.-K., Lai, Y.-F., Lee, H.-C., Tsai, P.-S., Chen, T.-J., ... Chiu, H.-Y. (2019). Associations between sleep disturbances and suicidal ideation, plans, and attempts in adolescents: A systematic review and meta-analysis. *Sleep*, *42*, zsz054.

Loechner, J., Starman, K., Galuschka, K., Tamm, J., Schulte-Korne, G., Rubel, J., & Platt, B. (2018). Preventing depression in the offspring of parents with depression: A systematic review and meta-analysis of randomized controlled trials. *Clinical Psychology Review*, *60*, 1–14.

Marco, C. A., Wolfson, A. R., Sparling, M., & Azuaje, A. (2011). Family socioeconomic status and sleep patterns of young adolescents. *Behavioral Sleep Medicine*, *10*, 70–80.

McMakin, D. L., & Alfano, C. A. (2015). Sleep and anxiety in late childhood and early adolescence. *Current Opinion in Psychiatry*, *28*, 483–489.

Muthén, L. K., & Muthén, B. O. (1998–2012). Mplus User's Guide: Statistical Analysis with Latent Variables (7th ed.). Los Angeles, CA: Muthén & Muthén.

Owens, J. A., Spirito, A., & McGuinn, M. (2000). The Children's Sleep Habits Questionnaire (CSHQ): Psychometric properties of a survey instrument for school-aged children. *Sleep*, *23*, 1–1051.

Owens, J. A., Spirito, A., McGuinn, M., & Nobile, C. (2000). Sleep habits and sleep disturbance in elementary school-aged children. *Journal of Developmental & Behavioural Pediatrics*, *21*, 27–36.

Palmer, C. A., & Alfano, C. A. (2017). Sleep and emotion regulation: An organizing, integrative review. *Sleep Medicine Reviews*, *31*, 6–16.

Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence*, *17*, 117–133.

Roberts, R. E., & Duong, H. T. (2014). The prospective association between sleep deprivation and depression among adolescents. *Sleep*, *37*, 239–244.

Ronnlund, H., Elovainio, M., Virtanen, I., Matomaki, J., & Lapinleimu, H. (2016). Poor parental sleep and the reported sleep quality of their children. *Pediatrics*, *137*, e20153425.

Sfärlea, A., Löchner, J., Neumüller, J., Asperud Thomsen, L., Starman, K., Salemink, E., ... Platt, B. (2019). Passing on the half-empty glass: A transgenerational study of interpretation biases in children at risk for depression and their parents with depression. *Journal of Abnormal Psychology*, *128*, 151–161.

Shirtcliff, E. A., Dahl, R. E., & Pollak, S. D. (2009). Pubertal development: Correspondence between hormonal and physical development. *Child Development*, *80*, 327–337.

Short, M. A., Gradisar, M., Wright, H., Lack, L. C., Dohnt, H., & Carskadon, M. A. (2011). Time for bed: parent-set bedtimes associated with improved sleep and daytime functioning in adolescents. *Sleep*, *34*, 797–800.

Silk, J. S., Shaw, D. S., Skuban, E. M., Oland, A. A., & Kovacs, M. (2006). Emotion regulation strategies in offspring of childhood-onset depressed mothers. *Journal of Child Psychology and Psychiatry*, *47*, 69–78.

Silk, J. S., Vanderbilt-Adriance, E., Shaw, D. S., Forbes, E. E., Whalen, D. J., Ryan, N. D., & Dahl, R. E. (2007). Resilience among children and adolescents at risk for depression: Mediation and moderation across social and neurobiological contexts. *Dev Psychopathol*, *19*, 841–865.

Soehner, A. M., Bertocci, M. A., Levenson, J. C., Goldstein, T. R., Rooks, B., Merranko, J., ... Birmaher, B. (2019). Longitudinal associations between sleep patterns and psychiatric symptom severity in high-risk and community comparison youth. *Journal of American Academy of Child & Adolescent Psychiatry*, *58*, 608–617.

Spitzer, R. L., Williams, J. B., Gibbon, M., & First, M. B. (1992). The structured clinical interview for DSM-III-R (SCID): I: History, rationale, and description. *Archives of General Psychiatry*, *49*, 624–629.

Walker, M. (2009). The role of sleep in cognition and emotion. *Annals of the New York Academy of Sciences*, *1156*, 168–197.

Weissman, M. M., Berry, O. O., Warner, V., Gameroff, M. J., Skipper, J., Talati, A., ... Wickramaratne, P. (2016). A 30-year study of 3 generations at high risk and low risk for depression. *JAMA Psychiatry*, *73*, 970–977.

Wheaton, A. G., Jones, S. E., Cooper, A. C., & Croft, J. B. (2018). Short sleep duration among middle school and

high school students - United States, 2015. *Morbidity and Mortality Weekly Report*, *67*, 85–90.

Wolfson, A. R., Carskadon, M. A., Acebo, C., Seifer, R., Fallone, G., Labyak, S. E., & Martin, J. L. (2003). Evidence for the validity of a sleep habits survey for adolescents. *Sleep*, *26*, 213–216.

Wong, P. M., Hasler, B. P., Kamarck, T. W., Muldoon, M., & Manuck, S. B. (2015). Social jetlag, chronotype, and cardiometabolic risk. *The Journal of Clinical Endocrinology & Metabolism*, *100*, 4612–4620.

Zahn-Waxler, C., Shirtcliff, E. A., & Marceau, K. (2008). Disorders of childhood and adolescence: Gender and psychopathology. *Annual Review of Clinical Psychology*, *4*, 275–303.

Zhang, J., Paksarian, D., Lamers, F., Hickie, I. B., He, J., & Merikangas, K. R. (2017). Sleep patterns and mental health correlates in US adolescents. *The Journal of Pediatrics*, *182*, 137–143.