European Child & Adolescent Psychiatry
https://doi.org/10.1007/s00787-020-01641-1

ORIGINAL CONTRIBUTION

Sleep problems in preschool-onset major depressive disorder: the effect of treatment with parent–child interaction therapy-emotion development

Caroline P. Hoyniak[1] · Diana J. Whalen[1] · Deanna Barch[1,2,3] · Joan L. Luby[1]

Received: 6 April 2020 / Accepted: 2 September 2020

Abstract

In school-aged children, adolescents, and adults, more than 72% of individuals diagnosed with major depression report co-occurring sleep problems, but little is known about sleep problems in the context of preschool-onset major depressive disorder (PO-MDD). The current study examined the prevalence of various sleep problems in a sample of young children diagnosed with PO-MDD and explored how the treatment of depression, using a modification of parent–child interaction therapy focused on emotional development (PCIT-ED), affects sleep problems. Participants included 229 preschoolers (ages 3–6 years) who met criteria for PO-MDD and participated a single-blind, randomized control trial comparing PCIT-ED to a waitlist control condition. Children were randomly assigned to either PCIT-ED ($n = 114$) or the waitlist condition ($n = 115$). Children were assessed at baseline, immediately after PCIT-ED, and 3 months after treatment completion for parent-reported sleep problems across the domains of insomnia, hypersomnia, daytime fatigue, and a total sleep problem index. In our sample, 45% of children had at least one subthreshold sleep problem, 38.4% had at least one threshold sleep problem, and 72.5% had at least one sleep problem (either threshold or subthreshold). Treatment with PCIT-ED significantly reduced sleep problems, including insomnia, daytime fatigue, and total sleep problems, compared to a waitlist condition, even when controlling for child depression. This reduction was maintained at a 3-month follow-up. Sleep problems are a prevalent co-occurring condition with PO-MDD. Interventions such as PCIT-ED that also effectively reduce sleep problems may be particularly beneficial for recovery from PO-MDD.
Clinical trial registration information: a randomized control trial of PCIT-ED for preschool depression; https://clinicaltrials.gov/NCT02076425.

Keywords Sleep · Preschool-onset major depressive disorder · Early childhood · PCIT

Introduction

Nearly 90% of adults [1] and 73% of school-aged children/adolescents [2] diagnosed with major depression report experiencing sleep problems. Sleep problems accompanying major depression frequently take the form of insomnia (difficulty initiating or maintaining sleep), hypersomnia (prolonged sleep episodes), or co-morbid insomnia and hypersomnia. The presence of sleep disturbances is one of the nine symptoms of major depression outlined in the diagnostic and statistical manual of mental health disorders. Fifth Edition (DSM-5) [3]. In school-aged children/adolescents, co-occurring major depression and sleep problems are associated with more severe depressive symptoms [2]. Additionally, sleep problems have been found to predict the onset of a depressive episode in adults suffering from recurrent

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s00787-020-01641-1) contains supplementary material, which is available to authorized users.

✉ Caroline P. Hoyniak
choyniak@wustl.edu

[1] Department of Psychiatry, Washington University School of Medicine, 4444 Forest Park Avenue, St. Louis, MO 63108, USA

[2] Department of Psychology, Washington University, St. Louis, MO, USA

[3] Department of Radiology, Washington University, St. Louis, MO, USA

Published online: 15 September 2020

depression [4]. Across the lifespan, there is evidence of a strong bi-directional linkage between sleep and depression [5], as well as common neurobiological mechanisms underlying both sleep disturbances and depressive symptoms [6].

Corresponding with the bi-directional association between sleep problems and depression, research suggests that sleep problems affect the treatment of depression. In adults and adolescents, individuals with co-occurring depression and sleep problems are less likely to respond to treatment for depression [7, 8], and sleep problems are the most commonly reported residual symptom in incompletely-remitted depression [9, 10]. Additionally, the treatment of insomnia, as an adjunctive to more traditional treatments for depression, leads to improvements in depressive symptoms [11–13]. In some research, the treatment of depression may even alleviate sleep problems [10], though not uniformly so [7, 12, 14].

Despite a robust literature on the association between sleep disturbances, depression, and the treatment of depression in adults and adolescents, substantially less research has focused on these constructs in early childhood. Both sleep problems and depression have been found to emerge early in life and show relative stability across development [15, 16]. Therefore, additional research on these problems in early childhood is crucial to better understand how to both prevent and intervene upon sleep and depression in young children. The current study examined the prevalence of various sleep problems in a sample of young children diagnosed with preschool-onset major depressive disorder (PO-MDD) and explored how the treatment of depression, using a modification of parent–child interaction therapy (PCIT-ED), affects sleep problems.

Sleep, internalizing symptoms, and depression in early childhood

Reports of the overall prevalence of sleep problems in preschoolers range from 0.8% for hypersomnia to 60% for significant night waking [17, 18]. Clinical sleep problems are thought to be relatively stable across childhood [19] and have been shown to be associated with a range of psychopathology [20]. Research suggests a concurrent association between higher sleep problems and higher internalizing symptoms in early childhood [21, 22]. Additional findings suggest an association between sleep problems in early childhood and depression in both community [18] and clinical samples [23]. Findings from longitudinal research also suggest increased risk for later internalizing problems in children with higher levels of parent-reported sleep problems. Parent-reported sleep problems in preschool have been found to be associated with increased risk for internalizing problems in mid-adolescence [15]. Similarly, parent-reported sleep problems in preschool are associated with increased severity in the trajectory of MDD across childhood into early adolescence [24]. Longitudinal findings from Quach et al. [25] indicate that sleep disturbances across childhood predict the subsequent onset of internalizing problems, but not the reverse. Conversely, Steinsbekk and Wichstrøm [20] suggests a bi-directional association between insomnia and depression in early childhood.

Much of this research has focused on internalizing problems on a whole, though, and research is needed to explore the specific association between depression and sleep problems in early childhood. Given increasing understanding of the effect that sleep loss has on neurodevelopment [26, 27], and the possibility that these effects are even more pronounced in early childhood [26, 28], it is crucial to better understand sleep problems in early childhood and how these sleep problems may be affected by psychopathology treatments.

Depression in early childhood

The lack of research focused on sleep and PO-MDD is likely attributable to a general lack of research focused on major depression in early childhood. PO-MDD has been validated across multiple samples and shown to have a chronic, relapsing course, with significant functional impairment [16, 29, 30]. Community prevalence rates hover around 2% with significant psychiatric co-morbidity [31, 32]. Two recent reviews have highlighted the developmental correlates and negative outcomes among children with PO-MDD that span across emotional, social, neural, and cognitive domains [33, 34]. This growing literature confirms the continuity of PO-MDD with school-age and adolescent MDD as well as the clinical significance of symptoms such as maladaptive guilt, concentration difficulties, and suicidal thoughts that although infrequently displayed, represent a significant risk.

Effective psychotherapeutic treatments for PO-MDD still lag behind treatment options for older children, adolescents and adults [35]. Given evidence highlighting the important role that the parent–child relationship plays in the maintenance of depressive symptoms in young children [36], recent efforts have centered on adapting parent–child interaction therapy (PCIT) for the treatment of PO-MDD. PCIT is a widely-tested, empirically-supported dyadic parent–child treatment originally designed to treat externalizing behaviors in young children [37, 38]. It utilizes live, in vivo parent coaching during stressful parent–child interactions to teach the caregiver to become "the arm" of the therapist. Standard PCIT consists of two treatment components, the "child-directed interaction (CDI)," which teaches parents to interact positively with their children without criticizing, and the "parent-directed interaction (PDI)," which teaches parents how to use nurturing yet firm limit-setting techniques.

Adapting PCIT in the first randomized control trial (RCT) for treatment of PO-MDD, Luby et al. [35] found promising evidence for the benefits of PCIT-ED in treating PO-MDD. PCIT-ED expands PCIT by adding a novel emotion development (ED) module following the standard modules. The emotion development module teaches caregivers to enhance their child's emotional competence and regulation [39], specifically by validating the child's expression of distressing emotions, decreasing reactivity to negative stimuli, and increasing reactivity to positive stimuli. Preschoolers evidenced enormous benefit from the treatment including remission from depression, lower impairment, and enhanced emotional functioning [35], and many of these benefits were sustained at a 3-month follow-up [40].

There are several mechanisms through which PCIT-ED might affect sleep problems in the context of PO-MDD. First, as the treatment of depression has been associated with decreases in sleep problems in adolescents (e.g., [10]), it is plausible that a similar process occurs in early childhood. As PCIT-ED has been shown to be an effective treatment for depression, treatment may lead to a corresponding decrease in sleep problems, even if sleep problems are not addressed specifically in the course of treatment. Next, the parent–child relationship has been shown to be an important risk and protective factor for sleep problems in early childhood [41, 42]. Parents play a critical role in helping young children settle to sleep, both through facilitating adequate sleep hygiene (i.e., bedtime routines) as well as through promoting children's sense of well-being and security enabling children to down regulate their vigilance to effectively settle to sleep. Additionally, positive parenting practices, including warmth, sensitivity, responsivity, and involvement, are thought to promote good child sleep [41, 43–45]. PCIT-ED has a specific focus on increasing such positive parenting skills and improving the parent–child relationship on a whole, as such, it is possible that more positive parenting is one mechanism through which child sleep is improved in the context of PCIT-ED.

The current study

The current study had three aims. First, we sought to provide normative data to establish the prevalence of sleep problems in children with PO-MDD. Given research with adults and older children suggesting differences in the prevalence of insomnia and hypersomnia symptoms in the context of depression [2, 46], we examined the prevalence of several major domains of sleep problems, including insomnia, hypersomnia, and fatigue, separately. Next, to examine whether the treatment of depression in early childhood with PCIT-ED also impacted child sleep problems, we examined changes in child sleep problems (as assessed using parent-reports on both a clinical interview and a questionnaire) following treatment with PCIT-ED. We expected that, compared to the waitlist group, children in the PCIT-ED treatment group would have fewer sleep problems post-treatment, and that this improvement in sleep problems would be maintained at the 3-month follow-up assessment.

Finally, research in older children/adolescents suggests that youth with co-occurring depression and sleep problems often show a poorer response to treatment for depression [7, 8]. Researchers have theorized that youth with sleep disturbances may be less likely to benefit from therapeutic techniques targeting depression due to a variety of interrelated neurobiological factors, including increased hypothalamic-pituitary-adrenocortical and sympathoadrenal activity in individuals with sleep problems that may increase dysphoric cognitions and cause downstream negative effects on cognitive abilities [8]. To test this possibility in our sample, we examined whether post-treatment depression severity (quantified by the number of symptoms of depression endorsed by parents, not including any of the sleep items) was associated with sleep problems at baseline. Based on the prior literature, we expected that children with more sleep problems at baseline would also have increased depression severity post-treatment.

Materials and methods

Treatment overview

Data were drawn from a single-blind, RCT comparing PCIT-ED to a waitlist control condition (see [35] for further details; Fig. 1). To summarize, a modified version of the empirically tested parent–child interaction therapy with a novel "emotion development" module was compared with a waitlist condition in a large sample of preschoolers with depression, with those receiving the active treatment followed up again 3 months after treatment completion. All study materials and procedures were approved in advance by the Washington University School of Medicine institutional review board. Written informed consent was obtained from all caregivers and verbal assent from children. The trial was registered with ClinicalTrials.gov (NCT02076425). Caregiver-child dyads participated in a comprehensive baseline assessment, and children who met criteria for PO-MDD were randomly assigned to either PCIT-ED ($n = 114$) or the waitlist condition ($n = 115$), with randomization stratified by gender and comorbid externalizing disorders. Children and their caregivers in the PCIT-ED group completed 6 sessions of CDI and 6 sessions of PDI, for a total of 12 sessions of traditional PCIT, as well as an additional 8 sessions of the ED module. Children and caregivers in the waitlist group completed 18 weeks of "watchful waiting" before completing the 12

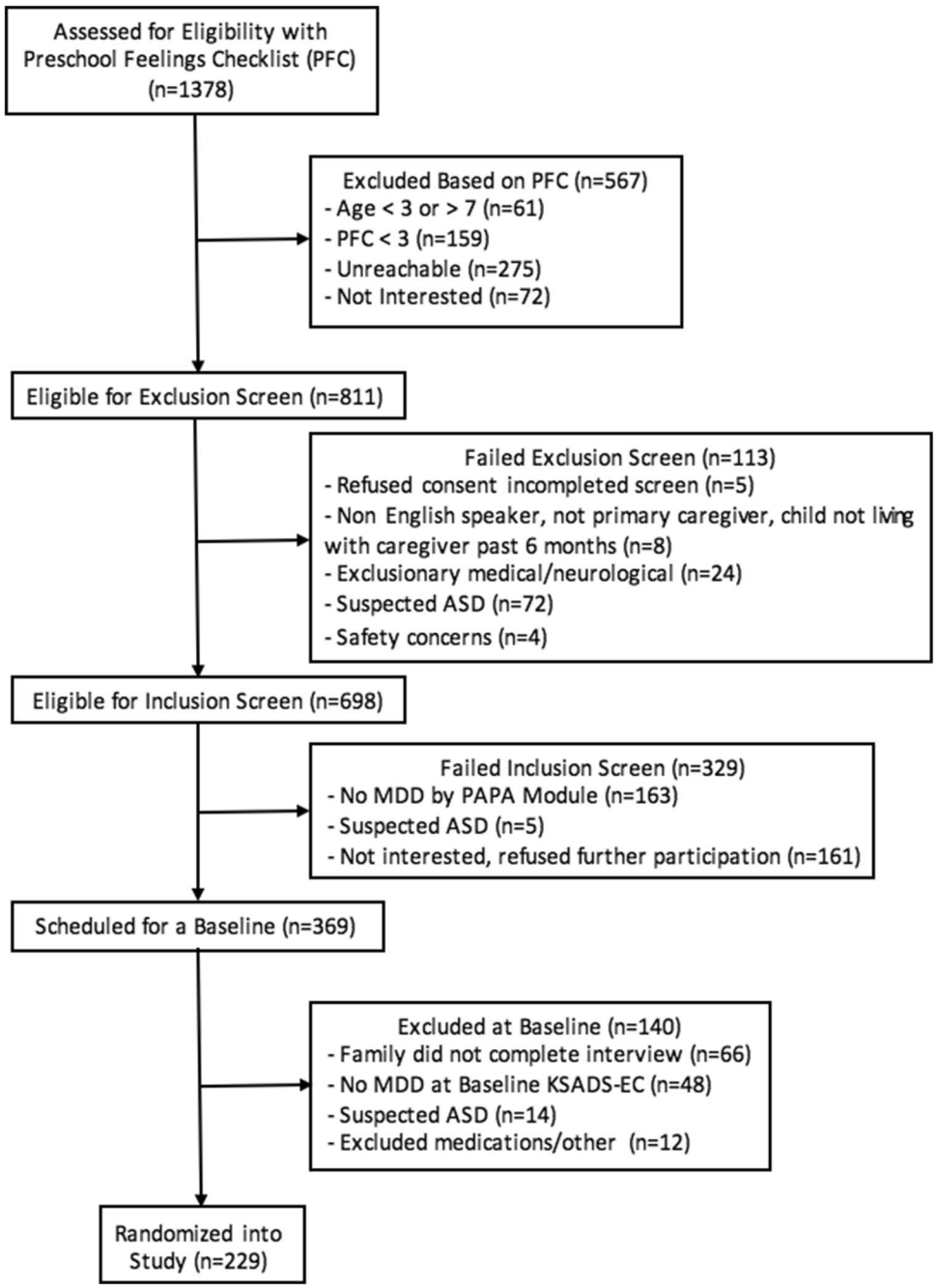

Fig. 1 Consort diagram of PCIT-ED study

sessions of traditional PCIT and 8 sessions of ED. All caregiver-child dyads participated in three assessments: (1) a baseline assessment (prior to any treatment), (2) a "post-treatment" assessment (~ 18 weeks after the baseline assessment, post PCIT-ED in the treatment first group, and post "watchful waiting" in the waitlist group), and (3) a 3-month follow-up assessment (~ 18 weeks after the post-treatment assessment; 3 months after treatment for the treatment first group and post PCIT-ED for the waitlist group). Of the 114 subjects randomized to PCIT-ED, 6 did not complete any therapy sessions, and 93 completed all 20 therapy sessions. The mean (SD) number of sessions completed was 17.4 (6.0).

During the baseline assessment, caregivers were interviewed using the schedule for affective disorders and schizophrenia–early childhood (K-SADS-EC) [47] to assess the child's psychiatric symptoms and assign DSM-5 diagnoses. K-SADS-EC interviews were conducted by masters-level clinicians, videotaped, reviewed for reliability, and calibrated for accuracy. Satisfactory interrater reliability was established prior to the start of the study, and kappa values during the study were computed on a monthly basis

(kappa = 0.74 for depression and 0.88 for all diagnoses; see [35, 48] for details of reliability coding procedures). All independent raters were blind to the treatment group and otherwise uninvolved in the study (see [35] for details about maintaining the blind). Caregivers also completed a battery of psychosocial questionnaires. During the post-treatment assessment and the follow-up assessment, the above procedures were repeated.

Participants

Preschoolers from the St. Louis metropolitan area, ages 3.0–6.92 years, were recruited from community sources and mental health facilities. The preschoolers were screened for depression using the Preschool Feelings Checklist [49], a validated screening measure for early childhood depression. All children who met symptom criteria for early childhood depression, and who did not have an autism spectrum disorder, a serious neurological or chronic medical disorder, or a significant developmental delay, were invited for an in-person assessment. The sample included 229 preschoolers and their caregivers, who were either randomized to the active treatment with PCIT-ED condition ($n = 114$) or randomized to the waitlist condition ($n = 114$). Demographic information for each group is provided in Table 1. From the original sample of 115 caregiver-child dyads randomized to a waitlist, 24 did not complete a "post-treatment" assessment. From the 114 caregiver-child dyads randomized to PCIT-ED treatment, 14 did not complete a post-treatment assessment. Therefore, data from 91 dyads from the waitlist group and 100 dyads from the treatment group had data at both assessments.

Table 1 Descriptive statistics, mean (SD) or % frequency, for treatment and waitlist groups

	PCIT-ED	Waitlist	*p*-value
Baseline			
Age (years)	5.14 (0.97)	5.28 (1.13)	0.32
Sex (% female)[a]	33.3%	36.5%	0.61
Race (%)			
White	82.4%	71.3%	0.05
Black	7.9%	15.6%	0.07
Asian	0.1%	0.0%	0.32
Biracial	8.8%	13.0%	0.30
Ethnicity (% Latino)	13.2%	8.7%	0.28
Income-to-needs ratio	3.13 (1.31)	2.85 (1.35)	0.12
K-SADS-EC sleep problems	1.59 (1.46)	1.94 (1.76)	0.10
CBCL sleep problems	5.18 (3.51)	5.23 (3.55)	0.89
Core depression symp	4.49 (1.25)	4.70 (1.31)	0.23
Post-treatment			
K-SADS-EC sleep problems	0.43 (0.79)	1.21 (1.38)	< 0.001
CBCL sleep problems	2.40 (2.65)	3.96 (3.00)	< 0.001
Core depression symp	1.44 (1.50)	3.43 (1.71)	< 0.001
3-Month follow-up[b]			
K-SADS-EC sleep problems	0.54 (1.18)	0.33 (0.68)	0.16
CBCL sleep problems	2.37 (2.49)	2.36 (2.62)	0.99
Core depression symp	1.52 (1.48)	1.75 (1.71)	0.37

K-SADS-EC schedule for affective disorders and schizophrenia–early childhood, *CBCL* child behavior checklist, *Symp* symptoms

[a]The higher percentage of males in the sample reflects the recruited population, not any specific attempt to overrecruit male children

[b]The 3-month follow-up assessment occurred 3 months after treatment for the PCIT-ED treatment first group, and immediately post-treatment with PCIT-ED for the waitlist group. Core depression symptoms includes the number of core depression symptoms endorsed on the K-SADS-EC, excluding the two sleep-related symptoms (insomnia/hypersomnia and fatigue)

Measures

Sleep problems

Child sleep problems were assessed using parent reports on the K-SADS-EC and the child behavioral checklist (CBCL) [50, 51].

K-SADS-EC sleep problems Within the mood disorders module, the K-SADS-EC probes the presence of child sleep problems in the context of mood symptoms across several domains, including insomnia (e.g., difficulty getting to sleep or staying asleep), hypersomnia (e.g., increased need for sleep or sleeping more than usual), and daytime fatigue (e.g., feeling fatigued, tiredness, or lack of energy). The insomnia domain is further broken down to examine initial insomnia (e.g., difficulty falling asleep at night), middle insomnia (e.g., waking up in the middle of the night and having difficulty returning to sleep), and terminal insomnia (e.g., waking up earlier than desired in the morning). For each sleep problem domain, parent-responses were coded as either 0 (not present), 1 (subthreshold), or 2 (threshold), based upon adherence to DSM-5 criteria. Threshold symptoms of insomnia include: taking more than 1.5 h to fall asleep at night nearly every night, awakening for more than 30 min in the middle of the sleep period nearly every night, and waking 30 min earlier nearly every day, for initial, middle, and terminal insomnia, respectively. Threshold symptoms of hypersomnia include: sleeping 2 h or more than usual nearly every day, or, if left to wake on his or her own, regularly sleeping more than 11–12 h per night. Threshold symptoms of fatigue include: feeling tired, without energy, most of the day, nearly every day. Interrater reliability, as measured using interclass correlations (ICC), across all of the K-SADS-EC sleep items was 0.83 (95% CI 0.76, 0.88).

CBCL sleep problems The sleep problems subscale of the CBCL provides a summary index of child sleep problems

across a variety of sleep domains. Depending on the child's age, s/he was administered either the CBCL 1.5–5 years or 6–18 years. The sleep problems subscales computed from these two versions differ slightly but assess largely comparable constructs in developmentally-appropriate ways. On the CBCL 6–18, the questions assessing sleep problems are not typically assessed as a single subscale (and most of the questions are a part of other subscales, including somatic problems and anxiety/depression problems). However, the limited number of measures assessing sleep in the current study, and the fact that children in the sample received either the CBCL 1.5–5 or the CBCL 6–18 depending on their age, we opted to include the derived sleep problems subscale from the CBCL 6–18 despite lower levels of internal consistency.

On both versions of the CBCL, primary caregivers indicate whether items are true of their child on a 3-point scale from 0 (not true) to 2 (very true or often true). The sleep problems subscale from the CBCL 1.5–5 contained 7 items, including "doesn't want to sleep alone", "has trouble getting to sleep", "nightmares", "resists going to bed at night", "sleeps less than most kids during day and/or night", "talks or cries out in sleep", and "wakes up often at night". The Cronbach's alpha values for this composite were 0.78, 0.79, and 0.79, at baseline, post-treatment, and follow-up, respectively. The sleep problems subscale derived from the CBCL 6–18 contained 6 items, including "nightmares", "overtired without good reason", "sleeps less than most kids", "sleeps more than most kids during the day and/or night", "talks or walks in sleep", "trouble sleeping" (one item, "wets the bed" was excluded from the scale as it is conceptually distinct from the other sleep items and its inclusion led to poor levels of internal consistency). The Cronbach's alpha values for this composite were 0.58, 0.57, and 0.54, at baseline, post-treatment, and follow-up, respectively. These relatively modest internal consistency values could be due to the conceptual distinctness of the various forms of sleep disturbance assessed by the CBCL 6–18.

Depression symptoms

Core depression symptoms from the K-SADS-EC was included as a control variable in the analysis. As the sleep problems assessed as outcomes in the current study are included in the core symptoms of depression, we created a depression symptom index from the K-SADS-EC that included all of the core depression symptoms except for insomnia/hypersomnia and fatigue. This index included seven items assessing child depressed and/or irritated mood, anhedonia, cognitive disturbances (e.g., difficulty concentrating), appetite disturbances, psychomotor disturbances (e.g., increased agitation or retardation), feelings of worthlessness and/or excess and inappropriate guilt, and recurrent thoughts of death and/or suicidal ideation and/or suicide attempts. For each core symptom, parent-responses on the K-SADS-EC interview are coded as either 0 (not present) or 1 (present) based upon adherence to criteria for each symptom described in the DSM-5. The core depression symptoms composite was calculated as the sum of scores on these seven items. Interrater reliability across the items included in this core depression symptoms score, as measured using ICC, was 0.89 (95% CI 0.84, 0.92). The Cronbach's alpha values for this composite was 0.66, 0.69, and 0.61, for baseline, post-treatment, and follow-up, respectively.

Data analysis

Prevalence rates for each of the domains of sleep problems were calculated across the entire sample at baseline. Next, to be able to include all subjects who underwent randomization in analysis, even those missing post-treatment or 3-month follow-up assessments, we used multiple imputation to create and analyze 25 multiply imputed datasets. Multiple imputation is regarded as a state-of-the-art technique for handling missing data as it improves accuracy and statistical power relative to techniques. Incomplete variables were imputed under fully conditional specification, using the default settings of the mice 3.0 package [52] in R [53]. Statistical parameters of interest (described below) were estimated in each imputed dataset separately and combined using Rubin's rules. For comparison, we also performed the analysis on the subset of complete cases.

First, generalized linear models (GLMs) were used to examine whether PCIT-ED reduced child sleep problems when compared to a waitlist control condition. Each domain of sleep problem assessed using the K-SADS-EC was examined using logistic regression in separate models, allowing for the comparison of effects across the sleep problem domain. If a significant result in the insomnia domain emerged, this effect would be further probed to examine specific effects across initial, middle, and terminal insomnia. The effect of treatment on the total sleep problems index from the CBCL was examined using multiple regression. All fitted GLMs controlled for sleep problems in the relevant domain at baseline, core depression symptoms on the K-SADS-EC (without sleep problems) at baseline, child age at baseline, and child sex, in order to adhere to best practices in clinical trial evaluation and improve our power to detect treatment effects [54]. Treatment effect sizes were quantified using standardized regression coefficients.

Next, to examine if changes in sleep problems were maintained from the post-treatment assessment to the 3-month follow-up assessment in children who were randomized to the treatment first condition, paired-samples t-tests were used to examine if there were significant differences in each type of sleep problem between these two assessments.

Finally, to examine whether pre-treatment sleep problem severity was associated with post-treatment depression severity, we used a multiple regression approach, examining how sleep problems at baseline predicted core depression symptoms post-treatment in the treatment first group when controlling for core depression symptoms at baseline.

Results

Descriptive statistics for the variables included in the analysis are presented in Table 1. Analyses comparing dyads who did and did not complete a post-treatment assessment are included in Supplemental Table 1. Correlations between each of the variables at baseline are presented in Supplemental Table 2.

Sleep problems in PO-MDD

The prevalence rates of sleep problems across both treatment conditions at baseline are presented in Fig. 2. In this sample, 45% of had at least one subthreshold sleep problem, 38.4% had at least one threshold sleep problem, and 72.5% had at least one sleep problem (either threshold or subthreshold). Insomnia was significantly more prevalent than hypersomnia (t [228] = 10.85, $p < 0.001$). Of the types of insomnia, initial and middle insomnia were more prevalent than terminal insomnia ($3.16 < t < 4.00$, $p < 0.05$), with no significant difference in the prevalence of initial and middle insomnia (t [228] = 1.14, $p > 0.05$). As prior research has identified sex-related differences in the prevalence of sleep problems in the context of major depression, our prevalence rates of threshold sleep problems by sex are presented in Fig. 3. Our findings generally suggest that,

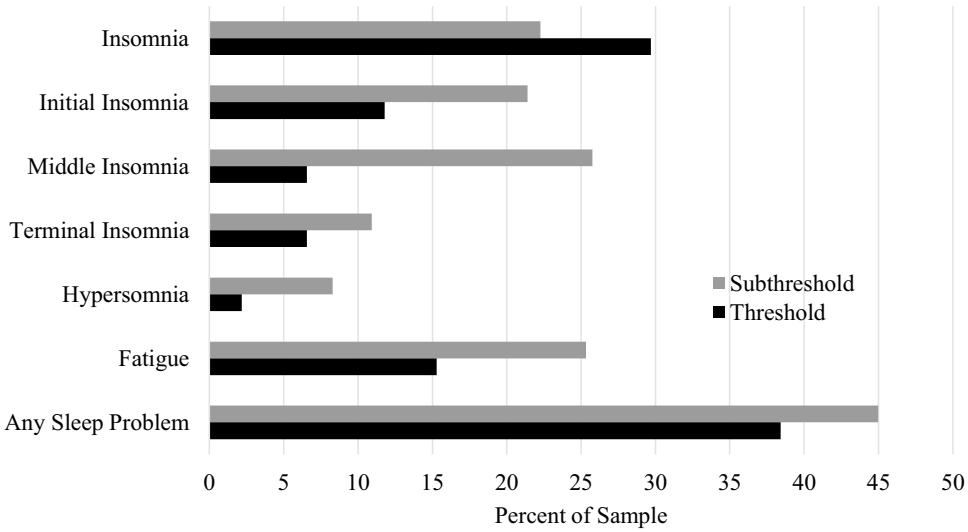

Fig. 2 Prevalence of sleep problems at baseline assessment ($N = 229$)

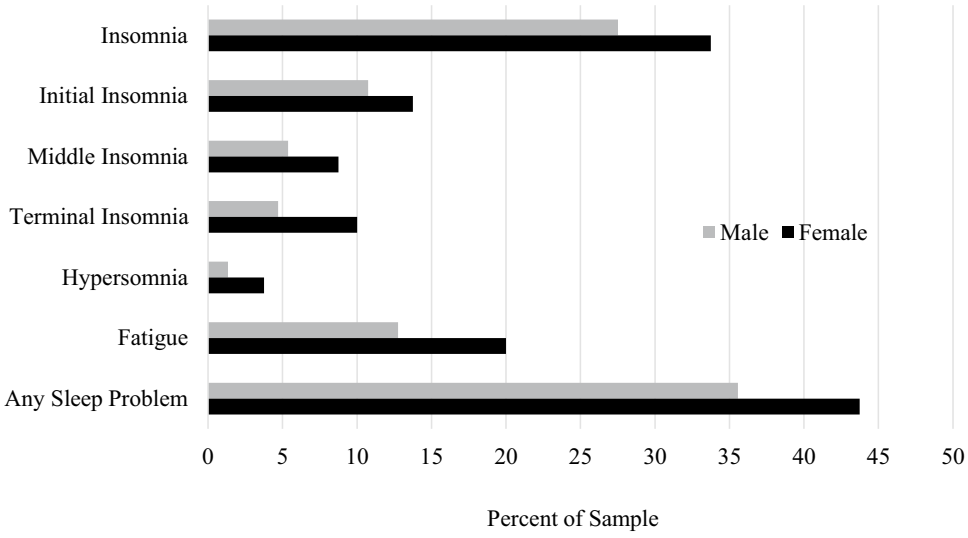

Fig. 3 Prevalence of threshold sleep problems at baseline assessment by males ($n = 149$) and females ($n = 80$)

across types of sleep problems, females were more likely to have disturbed sleep; however, these sex differences were not significant ($0.16 < F\,[1,227] < 2.97$, p's > 0.05).

The effect of PCIT-ED on sleep problems

The substantive pattern of findings when using multiple imputations and complete case analyses were similar, so only findings using the multiply imputed dataset are presented and discussed further. When compared to the waitlist control condition, children who received PCIT-ED treatment showed a significant reduction in insomnia (Table 2A) and daytime fatigue (Table 2C) but did not show a significant reduction in hypersomnia (Table 2B). Within the insomnia domain, PCIT-ED was effective at reducing initial, middle, and terminal insomnia symptoms when compared to the waitlist control condition (Table 2D–F). Findings were similar for the total sleep problems index from the CBCL: when compared to the waitlist control condition, children who received PCIT-ED treatment showed a significant reduction in total sleep problems (Table 2G). Findings remained significant when controlling for child age, child sex, the relevant sleep problem, and core depressive symptoms at baseline. Given the significant differences between the treatment and waitlist groups in terms of racial composition, we also ran all analyses controlling for child race (see Supplemental Table S3). The pattern of significant findings for the effects of treatment group was generally unchanged when controlling for race, however, the effects of group in the models exploring middle and terminal insomnia were only trending in significance when controlling for race. It is likely, though, that this is due to the increased number of predictors included in the models controlling for race (thus decreasing power).

Follow-up analyses of treatment effects in the active, treatment-first group indicated no significant difference between the post-treatment and follow-up assessments for any of the sleep domains examined ($-1.6 < t$'s < 0.15, p's > 0.05). These findings suggest that the reduction in sleep problems that children who received treatment with PCIT-ED experienced was maintained, at least across the 3-month follow-up duration.

Findings examining if sleep problems at baseline predicted core depression symptoms at post-treatment in the treatment first group are included in Supplemental Table S3. Pre-treatment sleep problem severity was not associated with post-treatment depression severity when controlling for baseline depression severity across any of the domains of sleep problems (e.g., insomnia, hypersomnia, fatigue, or total sleep problems on the CBCL).

Table 2 Comparison of K-SADS-EC sleep problems at the post-treatment assessment in treatment (group = 1) vs. waitlist (group = 0)

	β	SE	p value
(A) Insomnia (post-tx)			
Group	− 0.25	0.07	< 0.001
Insomnia (baseline)	0.23	0.13	0.13
Age (baseline)	0.04	0.07	0.57
Sex	0.03	0.06	0.64
Dep (baseline)	− 0.09	0.10	0.38
(B) Hypersomnia (post-tx)			
Group	− 0.11	0.08	0.20
Hypersomnia (baseline)	0.17	0.09	0.08
Age (baseline)	0.05	0.10	0.61
Sex	0.10	0.09	0.26
Dep (baseline)	0.00	0.09	0.97
(C) Daytime fatigue (post-tx)			
Group	− 0.22	0.08	0.01
Daytime fatigue (baseline)	0.15	0.07	0.03
Age (baseline)	0.05	0.07	0.51
Sex	0.00	0.08	0.97
Dep (baseline)	0.09	0.09	0.33
(D) Initial insomnia (post-tx)			
Group	− 0.16	0.07	0.02
Initial insomnia (baseline)	0.34	0.12	0.03
Age (baseline)	− 0.01	0.08	0.95
Sex	0.10	0.08	0.19
Dep (baseline)	− 0.02	0.10	0.87
(E) Middle insomnia (post-tx)			
Group	− 0.16	0.08	0.05
Middle insomnia (baseline)	0.33	0.15	0.08
Age (baseline)	− 0.01	0.07	0.84
Sex	− 0.02	0.07	0.77
Dep (baseline)	− 0.02	0.09	0.81
(F) Terminal insomnia (post-tx)			
Group	− 0.17	0.06	0.02
Terminal insomnia (baseline)	0.17	0.06	0.02
Age (baseline)	− 0.10	0.03	0.16
Sex	− 0.10	0.06	0.17
Dep (baseline)	0.15	0.03	0.05
(G) CBCL sleep problems (post-tx)			
Group	− 0.27	0.33	< 0.001
CBCL sleep problems (bseline)	0.55	0.05	< 0.001
Age (baseline)	− 0.11	0.17	0.07
Sex	− 0.02	0.35	0.78
Dep (baseline)	0.00	0.14	0.97

K-SADS-EC schedule for affective disorders and schizophrenia–early childhood, *tx* treatment, *Dep* core depression symptoms endorsed on the schedule for affective disorders and schizophrenia–early childhood (without the sleep symptoms)

Discussion

The current study examined sleep problems in the context of PO-MDD, exploring how treatment with PCIT-ED affects child sleep problems. Sleep problems were found to affect 72.5% of preschoolers with major depression. Findings suggest that, despite no specific sleep-related interventions throughout the course of treatment, PCIT-ED significantly reduced sleep problems, including insomnia, daytime fatigue, and total sleep problems, compared to a waitlist condition. The only domain of sleep problem not effectively reduced by PCIT-ED was hypersomnia. Additionally, the reduction in sleep problems across the domains of insomnia, fatigue, and a total sleep problems index was maintained at a 3-month follow-up. Finally, pre-treatment sleep problem severity was not associated with post-treatment depression severity across any of the sleep problem domains.

The first aim was to examine the prevalence rates of sleep problems in the context of PO-MDD. Findings indicated that 45% of the sample had at least one subthreshold sleep problem and 38.4% had at least one threshold sleep problem. Our finding that 72.5% of children with PO-MDD had some sort of sleep problem mirrors previous epidemiological findings with children/adolescents (72.7% in [2]), indicating continuity in the prevalence of sleep problems in the context of depression across childhood. Insomnia was significantly more prevalent than hypersomnia, with both initial and middle insomnia more prevalent than terminal insomnia. Interestingly, there were no significant sex differences in the prevalence of sleep problems, despite notable sex differences found in both the prevalence of sleep problems and sleep microarchitecture in adolescents and adults (e.g., [55]). This corresponds with findings that there are fewer sex-related differences in depression in early childhood [30]. It is plausible that significant sex-related differences in the prevalence of sleep problems in major depression may not emerge until puberty, as hormones are thought to influence risk for both sleep problems and mood disorders [56].

Next, we examined how PCIT-ED, a novel treatment for PO-MDD that focuses on improving the parent–child relationship and children's emotional development, affects sleep problems. Insomnia (including initial, middle and terminal insomnia), daytime fatigue, and total sleep problems all showed a significant reduction with treatment with PCIT-ED compared to a waitlist control condition, even when controlling for child age, child sex, the relevant sleep index, and core depressive symptoms at baseline. Unlike the other domains of sleep problems, hypersomnia did not significantly decrease during treatment. The low prevalence rate of hypersomnia (2.1% at baseline) may account for this lack of effect.

Treatment effect sizes, based on the standardized regression coefficients from each GLM, ranged from small to medium, with the largest effect sizes noted for overall insomnia, daytime fatigue, and CBCL total sleep problems. It is difficult to compare these effect sizes with previous research given how few studies have examined depression treatment effects on sleep disturbances in children, and of the few that have, many found no effect on sleep disturbances [7, 12, 14]. Perhaps unsurprisingly, our findings of small to medium effect sizes are smaller than effect sizes often reported for behavioral interventions directly targeting child sleep problems [57] but are similar in size to findings of the downstream effects of cognitive-behavioral treatments used to treat anxiety on co-occurring sleep problems [58].

There is mounting evidence that sleep problems are not "just a symptom" of depression and are in fact a separable co-morbid condition, which precede the emergence of depression and signify increased risk for depression [4, 59]. Although our previous research suggests that depression overall in this sample was reduced by PCIT-ED [35, 48], these findings did not clarify which symptoms of depression were specifically reduced by PCIT-ED. We believe that there is considerable value, not only in gaining further understanding of what aspects of the cluster of symptoms that make up the complex diagnosis of depression are impacted by treatment, but also to consider the nature of sleep disturbances in the context of depression in their own right. There is a precedent of researchers exploring the effects of treatment on specific symptoms of mood disorders, not just the disorder on a whole (e.g., suicidality, hopelessness; [60, 61]). Our study joins a number of other studies examining the effect of treatment for depression on sleep disturbances (e.g., [7, 9, 10]), as well as studies examining the effects of treatment of various disorders (e.g., anxiety) on co-morbid sleep disturbances [58].

Our findings also add to a growing, but contradictory, literature focused on how the treatment of depression affects youth/child sleep problems. For example, Manglick et al. [10] found that sleep problems decreased in depressed adolescents treated with either cognitive behavioral therapy (CBT), Sertraline, or CBT and Sertraline (equally across groups), but this study lacked a control condition. In contrast, neither Emslie et al. [7], which included treatment with fluoxetine in a sample of depressed child/adolescents, nor McGlinchey et al. [14], which included treatment with Interpersonal Therapy in a sample of depressed adolescents, found that treatment for depression significantly reduced sleep problems. These results suggest a complex relationship between the treatment of depression in youth and sleep problems, whereby it is not always the case that youth who respond to treatment for depression show corresponding decreases in sleep problems. In the current study, sleep problem symptoms significantly decreased with treatment,

suggesting that sleep problems in the context of early childhood may be more malleable and less entrenched, and thus may respond better to treatment. Additionally, the specific focus on the parent–child relationship in PCIT-ED may be responsible for the high-level of the response of sleep problem symptoms to treatment. This corresponds with theoretical interpretations that the parent–child relationship plays an important role in conferring risk for sleep problems in young children, with positive parenting practices, such as warmth and sensitivity, promoting good child sleep [62] and family conflict and negative parenting practices associated with worse child sleep [41].

In contrast to prior research suggesting that youth with higher levels of pre-treatment sleep disturbances are more likely to show higher levels of post-treatment depression [7, 8], we found no association between pre-treatment sleep problems and post-treatment depression across any of the domains of sleep problems examined. However, Emslie et al. [7] identified developmental differences in the effect of pre-treatment sleep problems on depressive symptoms across treatment, such that adolescents with sleep problems were more likely to show worse outcomes during treatment for depression, while children with sleep problems were more likely to show better depression outcomes during treatment for depression. Taken together, these results suggest developmental differences in the effect of co-occurring sleep problems on responsiveness to treatment for depression, and additional research to evaluate this developmental effect is clearly needed.

The current study has a number of important strengths. It included a large sample of young children all meeting criteria for PO-MDD, collected across a narrow age window. This allowed us to provide crucial information about the prevalence of sleep disturbances in PO-MDD. Additionally, it included a novel, efficacious intervention, PCIT-ED, that improves depressive symptoms through intervening on family relationships. Such a focus makes PCIT-ED especially well-poised to improve child sleep outcomes. There are also several limitations worth noting. First, this RCT used a waitlist as opposed to an active control treatment, and therefore, results may have been different if parents on the waitlist group were also receiving a control intervention. Findings also need to be interpreted in light of the larger treatment study inclusion/exclusion criteria. Although sampling ensured equal distribution of specific characteristics across the two-groups, the final sample was relatively high in socioeconomic status and primarily white. An additional limitation is a reliance on parent-reports of young children's sleep via clinical interview and questionnaire measures. These measures may be prone to reporter bias, and additional work using objective indicators of children's sleep is needed. Finally, some of the combined scales we used, including the sleep problems subscale of the CBCL and the core depression symptom index excluding insomnia/hypersomnia and fatigue items from the K-SADS-EC, showed relatively modest levels of internal consistency. Although unsurprising given the small number of items included in these scales (ranging between 5 and 7 items), these modest levels of internal consistency may indicate a lack of cohesion in the constructs assessed by these scales. However, given robust previous research validating the symptoms of early childhood depression [33, 34] and our relatively limited pool of sleep assessments to choose from in the current study, we opted to utilize these scales in our analysis. Future research on this topic should include longer and more reliable measures of child sleep problems.

The current study provides important information about the prevalence of sleep problems in the context of early-emerging depression, and the capacity for treatments targeting PO-MDD to reduce sleep problems. The prevalence of sleep problems in PO-MDD is similar to that of older children and adolescents, suggesting continuity in the comorbidity of sleep and mood disorders across childhood. Treatment with PCIT-ED significantly reduced sleep problems, suggesting that this treatment is efficacious in targeting sleep problems in the context of major depression. Finally, children with hypersomnia symptoms at baseline showed a poorer response to treatment for PO-MDD. Given the importance of early intervention efforts in reducing the impact of PO-MDD, sleep problems may be important to consider during treatment. Choosing an intervention, like PCIT-ED, that also effectively reduces sleep problems may be beneficial for recovery from PO-MDD.

Acknowledgements This work was supported by the National Institute of Mental Health, Grant #s 5R01MH098454-04 (PI: Luby), K23MH22325028202-01 (PI: Whalen); L30 MH108015 (PI: Whalen); T32 MH0100019-06.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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