Abstract

Background. Persistent sleep disturbances are one of the most common symptoms of Major Depressive Disorder (MDD) in adolescence. These are not typically targeted in psychological treatments and it is not known if psychological treatment for depression improves sleep.

Methods. Secondary analyses were conducted using data from a large, multi-centre, randomised controlled trial (Goodyer et al., 2017b). Young people aged 12-18 years (N = 465; 75% female) met diagnostic criteria for Major Depressive Disorder, based on the Kiddie-Schedule for Affective Disorders and Schizophrenia (K-SADS). They were randomised to one of three psychological treatments: Short-Term Psychoanalytic Psychotherapy; Cognitive Behavioural Therapy; and Brief Psychosocial Intervention. Sleep difficulties were assessed at baseline, post-treatment (36 weeks after randomization) and one year follow up (86 weeks after randomization) with the K-SADS, and the Mood and Feelings Questionnaire (MFQ).

Results. At baseline, 92% of young people exhibited clinically significant sleep difficulties. Exploratory analyses suggest that sleep difficulties significantly decreased from baseline to end of treatment on self-report and interview-based measures, and this decrease was maintained at follow up. Reduction in sleep difficulties did not differ between the psychological treatments. Approximately, half of young people reported residual sleep difficulties at the end of treatment and at follow-up.

Limitations. This paper reports secondary data analyses and findings are exploratory.

Conclusions. Tentative results suggest that psychological treatments for depression reduced sleep problems for some participants. However, young people with treatment-resistant sleep problems may benefit from adjunctive sleep interventions. Future work with a range of sleep measures is needed to determine those who have residual sleep problems at the end of treatment and post-treatment follow-up.

Keywords: Depression; Sleep; Psychotherapy; Adolescence

Introduction

Sleep problems are common in teenagers (Gradisar, Gardner, & Dohnt, 2011; Ohayon, Roberts, Zulley, Smirne, & Priest, 2000). These are believed to be due to a range of social factors and to changes in the circadian rhythm during puberty (Owens, 2014). Common sleep difficulties amongst the general adolescent population typically include insufficient sleep (Eaton et al., 2010) and weekday-to-weekend sleep variability (Wittmann, Dinich, Merrow, & Roenneberg, 2006). Sleep problems can become chronic and persistent during adolescence (Sivertsen, Harvey, Pallesen, & Hysing, 2017), and can be linked to poor emotional and physical health and impaired functioning at home and school (Shochat, Cohen-Zion, & Tzischinsky, 2014), as well as being distressing and disruptive for the whole family (Wake et al., 2006).

These persistent sleep problems are often experienced by individuals with worry and low mood (Gregory & O'Connor, 2002). As part of a diagnosis of Major Depressive Disorder (MDD), adolescents are required to present with at least one 'core' symptom, and a selection of further additional symptoms. One of the possible additional symptoms is 'sleep disturbance', and this has been found to be one of the most common symptoms of depression in adolescents (Orchard, Pass, Marshall, & Reynolds, 2017). The 'sleep disturbance' symptom is broken down into depression-specific sleep difficulties, including insomnia (not being able to get to sleep), hypersomnia (sleeping too much), circadian reversal (being active at night and sleeping during the day) and non-restorative sleep (not feeling rested after sleep) (APA, 2000). This range of difficulties are not typically experienced by the general adolescent population, or by adolescents with other common mental health problems such as anxiety, where individuals primarily experience difficulties with insomnia (Alfano, Ginsburg,

& Kingery, 2007). Furthermore, depressed young people who report problems with sleep have been found to be more likely to have severe depression (Liu et al., 2007) and to have thoughts about death and suicide (Urrila et al., 2012) than those who do not have problems sleeping. Insomnia in particular has also been found to be a common residual symptom among depressed adolescents (Kennard et al., 2006) and adults (Carney, Segal, Edinger, & Krystal, 2007; Nierenberg et al., 2010), and that residual insomnia may increase the risk of depression recurrence (Dombrovski et al., 2008).

In recent years there has been consideration as to whether sleep problems are not just a symptom or by-product of depression, but that they may contribute to the onset or maintenance of the disorder (Clarke & Harvey, 2012). Most of this work specifically examines insomnia. This is mainly due to insomnia being considered to be the most common sleep disorder in youth (e.g. Johnson, Roth, Schultz, & Breslau, 2006; Ohayon et al., 2000), and insomnia treatments being amongst the better-established sleep treatments i.e. Cognitive-Behavioural Therapy for Insomnia (CBTi). There is however evidence of a significant association between more general sleep disturbances and depression (Lovato & Gradisar, 2014), with good evidence that sleep difficulties in children and young people are likely to predict the risk of developing depression in later childhood or adulthood (de Bruin, Bögels, Oort, & Meijer, 2015; Gradisar et al., 2011; Roane & Taylor, 2008; Roberts & Duong, 2013). There may be different causal pathways or common factors associated with the onset of both sleep problems and depression, for example physical problems linked to chronic illnesses (e.g. pain, problems breathing), or negative life events or adverse socio-economic circumstances. This possible causal relationship, and the focus on insomnia, has led to the proposal of specific guidelines for treating ‘insomnia’ in this population, i.e. drawing on techniques from CBTi (Clarke & Harvey, 2012). It is also important to note that while sleep disturbance is a diagnostic criterion for MDD, sleep disorders (specifically insomnia disorder

and delayed sleep-wake phase disorder) are also commonly comorbid with depression (Buysse et al., 2008) and have been conceptualised by some as independent diagnostic entities (Staner, 2010).

Psychological interventions based on cognitive behavioural models of insomnia are an effective treatment for sleep problems in adults (Koffel, Koffel, & Gehrman, 2015; Trauer, Qian, Doyle, Rajaratnam, & Cunnington, 2015) and adolescents (de Bruin et al., 2015), including those with a range of mental health problems (van Straten et al., 2017; Wu, Appleman, Salazar, & Ong, 2015). Sleep interventions can be delivered in a variety of ways, including via computer (Cheng & Dizon, 2012), thus significantly increasing access to treatment. A recent meta-analysis of non-pharmacological sleep interventions also found that treatment for sleep problems significantly reduced symptoms of depression (Gee et al., 2018), even though the interventions did not address any other aspects of depression including core symptoms of e.g. low mood or anhedonia. The effect of sleep interventions on depression symptoms was larger in studies that recruited participants with current mental health difficulties (and hence more severe symptoms of depression). There was however limited research on the effect of treating sleep problems on depression in children and adolescents.

Just as treatment for sleep problems may improve symptoms of depression, so it is plausible that treatment for depression may improve sleep difficulties, particularly given that sleep difficulties are one of the symptoms used to diagnose Major Depressive Disorder (DSM-V; APA, 2013; ICD-10; WHO, 1992). Although sleep problems are extremely common amongst depressed young people (Orchard et al., 2017), most standardised manuals for psychological treatments for depression do not typically include sleep intervention techniques (e.g. Brent & Poling, 1997; Martell, Addis, & Jacobson, 2001; Ritschel, Ramirez, Jones, & Craighead, 2011). However, the improvement of other symptoms of depression may have an indirect benefit on sleep. For example, perseverative and ruminative thinking is

common in depression and is hypothesised to interfere with sleep onset (Lovato & Gradisar, 2014). Therefore, reducing perseverative and ruminative thinking may help make sleep onset more rapid. In addition, subjective report of specific sleep problems may be inflated by negative cognitive biases or self-critical thinking, which are pervasive in depression in adults (Joorman, Yoon, & Zetsche, 2007) and adolescents (Orchard, Pass, & Reynolds, 2016; Platt, Waters, Schulte-Koerne, Engelmann, & Salemink, 2017). Objective and subjective measurements of sleep are poorly correlated amongst depressed individuals (Bertocci et al., 2005; Dahl & Puig-Antich, 1990) and this may be explained by potential reporting biases, such as misperception of sleep deficit, cognitive inflexibility, and selective attention and monitoring (Hiller, Johnston, Dohnt, Lovato, & Gradisar, 2015), which are likely to be amplified by low mood and worries. Successful treatment for depression reduces the associated negative cognitive biases (Tang, DeRubeis, Beberman, & Pham, 2005) and this may lead to more favourable reports of sleep quality (Lofthouse, Gilchrist, & Splaingard, 2009).

It was recently found that cognitive-behavioural therapy for anxiety in children and adolescents improved self-reported sleep problems (Peterman et al., 2016). However, this was only found for parent report, not child report. The authors also note that child-reported sleep problems were low in this sample. Surprisingly, given the high prevalence of sleep problems in adolescents with depression, and the adverse impact of sleep difficulties, research has not yet examined whether treatments for adolescent depression improves sleep problems. Although there is evidence that CBT for insomnia reduces difficulties with insomnia, and may have an added benefit of reducing symptoms of depression, it is not known whether CBT for depression will improve sleep disturbances, or whether other psychological treatments will also improve sleep disturbances e.g. via reduction in other underlying common factors.

This paper reports on the exploratory post-hoc analyses of data from a randomised controlled trial for adolescent depression where there was high baseline prevalence of sleep symptoms (Goodyer et al., 2017b). Given the absence of evidence on this important subject, the aim of this study is twofold: first to describe the nature of sleep problems in a large sample of young people with depression, and second to examine the effect of three distinct psychological treatments for adolescent depression on sleep problems, measured by self-report depression questionnaires and diagnostic interviews.

Methods

Participants and Recruitment

Young people in this study were taking part in a large, multi-centre, randomised controlled trial; the HTA-funded IMPACT (Improving Mood through Psychoanalytic and Cognitive-Behavioural Therapy) Study (Goodyer et al., 2017b). IMPACT is a pragmatic superiority trial comparing the relative clinical and cost effectiveness of three psychological treatments for adolescent Major Depressive Disorder. Participants were recruited and treated at 15 National Health Service child and adolescent mental health service (CAMHS) clinics in three regions in England.

A total of 465 young people were randomised to one of three treatment approaches; Short-Term Psychoanalytic Psychotherapy (STPP); Cognitive Behavioural Therapy (CBT); and a manualised form of specialist clinical care termed Brief Psychosocial Intervention (BPI) which was chosen as the active control treatment. Lengths of treatment varied for the three approaches: 28 sessions over 30 weeks for STPP; 20 sessions over 30 weeks for CBT; and 12 sessions over 20 weeks for BPI. The three treatments were based on distinct theoretical models and were delivered in routine services by specialist clinicians according to treatment manuals (IMPACT Study CBT Sub-Group, 2010; IMPACT Study Child Psychotherapy Sub-Group, 2010; Kelvin et al., 2010). CBT and STPP manuals did not

specifically consider how to address problems with sleep. The BPI manual identified sleep hygiene as a routine intervention that clinicians could offer. Treatment manuals were developed for each modality and via examination of treatment adherence using the Comparative Psychotherapy Process Scale (Hilsenroth, Blagys, Ackerman, Bonge, & Blais, 2005), treatment fidelity was found to be good, (Midgley et al., 2018).

The mean age of participants was 15.6 years (SD = 1.4) and 348 (75%) were female. Most (85%) described their ethnicity as White British. Participants were recruited to the study and treated in 15 Child and Adolescent Mental Health services in the UK. All participants met diagnostic criteria for moderate to severe Major Depressive Disorder, based on the K-SADS (see below; Kaufman et al., 1997).

The study was approved by the Cambridgeshire 2 Research Ethics Committee (reference 09/H0308/137) and local NHS provider trusts. All patients and their parents gave written informed consent. See Goodyer et al. (2017a) for comprehensive information on the recruitment process and study design.

Measures and Procedure

Two measures were used to address the aims of this study: the interviewer administered Kiddie-Schedule for Affective Disorders and Schizophrenia (K-SADS-PL; Kaufman et al., 1997) and the adolescent version of the self-report Mood and Feelings Questionnaire (MFQ; Costello & Angold, 1988). The present analyses report data from three time points from the IMPACT trial: baseline, post-treatment (36 weeks after randomisation), and at one-year follow-up (86 weeks after randomisation). Time between randomization and first session varied as treatments were delivered in routine clinical practices. Median number of weeks across location and treatment ranged from 2.9 weeks to 7.3 weeks (Goodyer et al., 2017a).

The Kiddie-Schedule for Affective Disorder and Schizophrenia Present and Lifetime (K-SADS-PL) version is a semi-structured interview measure, which was used to establish the presence of DSM-IV diagnoses. Adolescents and parents/guardians completed the measure and both interviews were used to construct a diagnosis based on positive symptom reporting from either respondent. See Goodyer et al. (2017b) for information regarding inter-rater reliability. The K-SADS DSM-IV Major Depressive Disorder section examines the presence or absence of all symptoms of depression in the past two weeks. Six sleep disturbance symptoms are assessed in this section: 'initial insomnia', 'middle insomnia', 'terminal insomnia', 'hypersomnia', 'circadian-reversal' and 'non-restorative sleep'. Specific thresholds are provided by the manual to establish when participants meet criteria for each sleep symptom. For example, '*initial insomnia' = two hours or more to fall asleep, most nights*. The presence of individual disturbances, as well as the total number of disturbances, is reported in the analyses. The latter is used as in indication of severity of overall sleep disturbance.

The Mood and Feelings Questionnaire (MFQ) is a 33-item self-report measure completed by the adolescent, examining depressive symptoms present over the past two weeks. The instrument is designed to cover symptom areas specified in DSM-IV for an episode of MDD, e.g. sleep disturbance. To be consistent with other measures in the IMPACT study, the MFQ was scored on a four-point scale (0-3). Ratings of '2' and '3' were then combined to be in line with typical MFQ scoring procedures. The MFQ has good test–retest reliability (Pearson's $r = 0.78$), an α coefficient of 0.82 and discriminant validity for detecting an episode of Major Depressive Disorder in clinical adolescent samples. Of particular interest in this paper are the two sleep items: 'I didn't sleep as well as I usually sleep' and 'I slept a lot more than usual'. These items are designed to tap into the two key DSM Major Depressive Disorder sleep symptoms; respectively *insomnia* and *hypersomnia*

(APA, 2013). For the purpose of this report, these scores will be referred to as the 'insomnia' and 'hypersomnia' items.

Results

Data Preparation and Preliminary Analyses

Missing data at the end of treatment and follow-up was higher than expected (Goodyer et al., 2017b), with 392 (84%) participants with available data for the trial's primary outcome of MFQ scores by the end of follow-up. As such, multiple imputation (Rubin, 1987; Sinharay, Stern, & Russell, 2001) was utilised to create 20 complete data sets (N=465). The original and imputed data for repeated measures analyses were reanalysed using multilevel modelling and no difference in patterns of significance was found. The original data are reported for simplicity.

Continuous data were screened in relation to the assumptions of parametric tests (Tabachnick & Fidell, 2007). Where assumptions were violated, confirmatory analyses were conducted by running analyses with 1000 bootstrap samples or non-parametric alternatives. All results were consistent, suggesting that the original analyses were robust to the violations of assumptions, so results based on the original (non-bootstrapped) analyses are presented for simplicity.

All medication use was recorded at baseline, and participants were required to be on a stable dose of antidepressants to take part in the trial. Only one participant identified taking a sleeping medication (melatonin). Between-group differences for participants with and without medication for depression or anxiety were examined, no between-group differences were identified, and all participants were included in the following analyses. For further information on medication use and association with clinical characteristics in the present sample, see Cousins et al. (2016).

Descriptive Statistics of the Nature of Sleep Problems at Baseline

At baseline, 92% of young people described sleep difficulties that met threshold for a symptom on the K-SADS (Goodyer et al., 2017b). The most commonly reported problems at baseline were non-restorative sleep and initial insomnia, which were both reported by over half of the participants (Table 1). It was also common for adolescents to report more than one type of sleep disturbance (see Table 2), with 58% of young people reporting 1-2 distinct sleep problems, and 35% of young people reporting between three and six different sleep problems. There were no differences between treatment groups for frequency of baseline sleep problems, $F(2,465) = 0.53$, $p = 0.59$, $\eta_p^2 < 0.01$.

[Insert Table 1]

Pre- to Post-Treatment Change in Sleep Disturbance on Self-Report Questionnaire

First we examined subjective ratings of sleep problems (insomnia and hypersomnia) on the adolescent MFQ at baseline, post-treatment and follow-up. To test the effects of time and treatment modality, two mixed model ANOVAs were conducted with a between-subjects independent variable of treatment arm, with three levels (BPI, STPP, and CBT), and a within subjects independent variable of time, with three levels (baseline, end of treatment and follow-up), examining mean scores for the insomnia and hypersomnia items (see Figure 1).

For insomnia there was a significant main effect of time, $V = 0.43$, $F(2, 274) = 103.54$, $p < .001$, $\eta_p^2 = 0.43$, with ratings decreasing from baseline to follow up (Figure 1). Pairwise comparisons revealed a significant difference in insomnia between baseline and end of treatment ($p < .001$), and baseline and follow up ($p < .001$), but no difference between insomnia at the end of treatment and at follow-up ($p = .23$). There was no significant main effect of treatment modality on young people's ratings of insomnia, $F(2, 275) = 0.27$, $p = .77$,

η_p^2 < 0.01. There was also no significant interaction between time and treatment arm, V = .02, $F(4, 550) = 1.20$, $p = .31$, $\eta_p^2 < 0.01$.

Hypersomnia was reported infrequently on the MFQ (see Figure 1). There was no effect of time, $V = .01$, $F(2, 275) = 0.63$, $p = .53$, $\eta_p^2 = 0.01$, treatment, $F(2, 276) = 0.20$, $p = .82$, $\eta_p^2 < 0.01$, or time by treatment interaction $V < .01$, $F(4, 552) = 0.12$, $p = .98$, $\eta_p^2 < 0.01$, on ratings of hypersomnia.

[Insert Figure 1]

Pre- to Post-Treatment Change in Sleep Disturbance on Diagnostic Interview

Next, we examined symptom reports of sleep on the K-SADS clinical interview at baseline, post treatment and follow up (see Table 2). To assess if the number of sleep symptoms reduced over the course of psychological treatment for depression, we used a mixed model ANOVA with a between subjects independent variable of treatment with three levels (BPI, STPP, and CBT), and a within subjects independent variable of time with three levels (baseline, end of treatment and one year follow-up).

The number of sleep problems was significantly lower after treatment for depression $F(1.90, 390.61) = 106.70$, $p < .001$. Pairwise comparisons revealed a significant reduction in the number of sleep problems reported between baseline and end of treatment ($p < .001$), and baseline and follow up ($p < .001$), but not between end of treatment and follow-up ($p = .06$). There was not a significant main effect of treatment arm, $F(2, 206) = 0.43$, $p = .65$ on the number of sleep problems reported and no significant interaction between treatment arm and time, $F(3.79, 390.61) = 1.13$, $p = .34$.

[Insert Table 2]

Residual Sleep Problems

In the original trial, it is reported that 31-44% of adolescents were still experiencing Major Depressive Disorder at the end of treatment (across the treatment arms), with 26-29% still experiencing a diagnosis at one year follow-up (Goodyer et al., 2017b).

[Insert Table 3]

There was a marked reduction in the frequency of sleep problems after treatment (see Tables 1 and 2); however approximately half of young people reported residual sleep difficulties at the end of treatment and at follow-up. Furthermore, of the overall sample, 33% recovered from depression at 36 weeks but had residual problems with sleep.

Discussion

The results of this study show that sleep problems were virtually universal amongst a large sample of depressed adolescents and that, for some young people, psychological treatments for depression appeared to improve their sleep. Although a range of different sleep disturbances were reported as part of the diagnostic interview for depression, the most common difficulties were initial insomnia (difficulty falling asleep) and non-restorative sleep (not feeling rested after sleeping). No differences in recovery of sleep disturbance were found between the three psychological therapies (CBT, STPP and BPI). These data were collected from a clinically referred group of young people, with moderate to severe Major Depressive Disorder, who were referred to and treated in routine mental health services.

These exploratory analyses indicate that according to a clinician-delivered diagnostic interview for depression, approximately half of the young people did report a reduction in sleep disturbances after treatment for depression. An overall improvement was also reflected

in a self-report questionnaire item measuring difficulty getting to sleep. However, this means that a substantial proportion continued to meet threshold for sleep disturbances at the end of treatment and this proportion did not change over the year of follow up. Furthermore, one third of young people recovered from their Major Depressive Disorder diagnosis, but still reported residual sleep problems after treatment and at follow up.

Given that sleep difficulties have such adverse, lasting effects on physical and psychological well-being for the individual (Shochat et al., 2014), can be disruptive for the family (Wake et al., 2006) and may play a causal role in the development of depression (Lovato & Gradisar, 2014), residual sleep problems may constitute a significant on-going risk and may benefit from direct intervention. It is however also possible that for individuals that recover from depression but not from sleep disturbances, that sleep problems and depression may be distinct and not directly associated via causal relationships.

With regards to the individuals that recover from depression and sleep disturbance, the nature of the present data and analyses makes it difficult to identify psychological treatments improve sleep and depression independently, or whether improvement in sleep is simply a bi-product of depression recovery. It has been theorised that cognitive biases present in depression, such as perseverative and ruminative thinking, interfere with sleep onset (Lovato & Gradisar, 2014). Therefore, reducing perseverative and ruminative thinking may help make sleep onset more rapid. In addition, subjective report of specific sleep problems may be inflated by negative cognitive biases or self-critical thinking, which are pervasive in depression in adults (Joorman, Yoon, & Zetsche, 2007) and adolescents (Orchard, Pass, & Reynolds, 2016; Platt, Waters, Schulte-Koerne, Engelmann, & Salemink, 2017).

There is reasonable evidence that interventions for sleep problems are effective and cost effective (Koffel et al., 2015; Trauer et al., 2015), can be delivered in different ways and made accessible through online treatment programmes (Cheng & Dizon, 2012), and that they

significantly reduce symptoms of depression (Gee et al., 2018). This evidence has mostly been collected with adults, with much less evidence that similar interventions are effective for young people, or that they are acceptable to young people or their parents. However, feasibility and pilot work in this area is growing, with recent research reporting on case studies of CBT for insomnia (CBTi) with depressed adolescents (Orchard, Pass, Chessell, Moody, Ellis & Reynolds, in press) and the assessment of the feasibility of delivering web-based CBTi in child and adolescent mental health services in the UK (Cliffe, Croker, Denne, & Stallard, 2018). Further work is needed to establish the effectiveness of psychological interventions targeting sleep in depressed young people.

The young people in this study were randomised to receive one of three different psychological interventions for depression, Cognitive Behaviour Therapy (CBT), Short Term Psychodynamic Psychotherapy (STPP), or Brief Psychosocial Intervention (BPI). CBT and STPP did not target sleep problems but BPI did include advice about sleep hygiene. Young people in all three arms reported significantly fewer problems with sleep after treatment and one year later and there was no difference between the three treatments in the reduction of sleep problems. This equivalence between treatments in reducing sleep problems is consistent with the effects of the treatments for depression severity, for which there was no significant differences in effectiveness or cost effectiveness between the three treatments (Goodyer et al., 2017b). Although BPI included some material on sleep hygiene, it is unsurprising that this treatment did not result in greater reduction of sleep disturbance given that 1) the evidence suggests sleep hygiene is not one of the active components of CBT for insomnia, 2) there is no evidence of sleep hygiene improving insomnia, and 3) in many cases sleep hygiene is used as a control treatment (e.g. Stepanski & Wyatt, 2003; Taylor & Roane, 2010). It is however important to acknowledge that the conclusions regarding treatment are limited by the absence

of a waitlist control group. It is possible that sleep problems naturally improve over time for some individuals.

A major limitation of this study is the measurement of sleep. This limitation is somewhat attenuated by using two different measures of sleep – one based on a structured diagnostic interview by a trained assessor, the other a response to single items on a self-report questionnaire. However, because depression is associated with pervasive negative cognitive biases, perceived sleep quality may be a distortion of actual sleep quality and adolescents with depression may therefore significantly over-estimate their sleep problems. Similarly, reported improvements in sleep after treatment for depression may reflect a change in mood and hence perceptions of sleep, rather than any objective change in sleep quality or duration. Future work in this area should include validated questionnaire and diary measures of sleep (e.g. Insomnia Severity Index, Pittsburgh Sleep Quality Index, Consensus Sleep Diary) as well as measures that do not rely on self-report (e.g. actigraphy or polysomnography). The addition of a direct index of sleep duration, sleep variability, wakefulness, time to sleep and time awake, would help unpack the meaning of these data and untangle the subjective experience of depression and low mood, and perceived poor sleep quality.

It is also important to note that there are challenges with considering the role of sleep problems as both symptom and causal factor in depression, however, research addressing the exact nature of this relationship is still in it's infancy. There is a growing body of literature suggesting that sleep problems and depression may both be caused by a separate underlying factor e.g. rumination (Lovato & Gradisar, 2014), with a recent review considering a wide range of mechanisms which could result in associations between depression, anxiety and insomnia (Blake et al., 2018).

Relatedly, future work should also look to diagnose comorbid sleep disorders in addition to depressive disorders, as it is likely that some adolescents with MDD may be

experiencing comorbid insomnia disorder, or other sleep disorders. In the present study, it is possible that residual sleep symptoms may be indicative of untreated sleep disorders. With some emerging evidence that disorders of insomnia and depression may have distinct characteristics (Buysse et al., 2008), it is important that research addresses these comorbidities directly when examining treatment outcomes for depression and sleep.

This is the first study to examine recovery of sleep disturbance following psychological treatment for depression. Despite methodological shortcomings, we present important preliminary data that has clear treatment implications as well as highlighting important ways in which we can improve assessment of sleep problems in the context of depression. These clinical data highlight the almost universal and adverse impact of sleep difficulties amongst depressed young people. Findings suggest that approximately half of young people treated for depression will continue to experience residual sleep problems, and a third of young people will experience residual sleep problems even in absence of depression.

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Table 1. Frequency of K-SADS sleep disturbances at baseline, end of treatment and follow-up

%	Baseline (*n* = 465)	End of Treatment (36 weeks) (*n* = 270)	Follow-up (86 weeks) (*n* = 282)
Initial Insomnia	53.3	24.2	20.9
Middle Insomnia	32.5	11.9	11.5
Terminal Insomnia	17.6	14.4	10.2
Circadian Reversal	23.9	12.8	13.3
Non-Restorative	68.0	27.0	18.0
Hypersomnia	15.1	9.7	11.9
Any sleep problem	91.8	55.6	47.2

Table 2. Frequency of K-SADS sleep disturbances at baseline, end of treatment and follow-up (%)

No. Sleep Disturbances	**Baseline (*n* = 465)**	**End of Treatment (36 weeks) (*n* = 270)**	**Follow-up (86 weeks) (*n* = 282)**
0	8	44	53
1-2	58	44	37
3-4	32	11	10
5-6	3	0	0

Table 3. Frequencies of participants with and without a diagnosis of depression and residual sleep symptom at post-treatment (n = 270)

		Major Depressive Disorder	
		Yes	No
Sleep Symptom	Yes	62 (23.0%)	88 (32.6%)
	No	3 (1.1%)	117 (43.3%)

Table 4. Demographics and Characteristics of Individuals with and without Residual Sleep Disturbance at End of Treatment

		Sleep Disturbance	No Sleep Disturbance	Statistics
Gender (% female)		77.3	67.5	$\chi^2(1) = 4.24$, $p = .04$
Age (years)		15.6	15.7	$t(268) = -.02$, $p = .99$
Baseline Sleep	Initial Insomnia	55.7	52.1	$\chi^2(1) = 0.24$, $p = .62$
Disturbance (%)	Middle Insomnia	36.4	25.6	$\chi^2(1) = 5.99$, $p = .01$
	Terminal Insomnia	13.6	17.1	$\chi^2(1) = 0.32$, $p = .57$
	Circadian Reversal	26.1	20.5	$\chi^2(1) = 0.43$, $p = .51$
	Non-Restorative Sleep	71.6	63.2	$\chi^2(1) = 1.90$, $p = .17$
	Hypersomnia	18.2	11.1	$\chi^2(1) = 1.88$, $p = .17$
	Total (mean)	2.2	1.9	$t(268) = -2.66$, $p < .01$

NB. MDD: Major Depressive Disorder

Figure 1. Mean MFQ item ratings of insomnia and hypersomnia items at baseline (*n* = 462), end of treatment (*n* = 317) and 1-year follow-up (*n* = 349)