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Systematic Review and Meta-analysis of Adolescent Cognitive–Behavioral Sleep Interventions

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Abstract This systematic review and meta-analysis examined the efficacy of adolescent cognitive–behavioral sleep interventions. Searches of PubMed, PsycINFO, CENTRAL, EMBASE, and MEDLINE were performed from inception to May 1, 2016, supplemented with manual screening. Nine trials were selected ($n = 357$, mean age = 14.97 years; female = 61.74%). Main outcomes were subjective (sleep diary/questionnaire) and objective (actigraphy) total sleep time (TST), sleep onset latency (SOL), sleep efficiency (SE), and wake after sleep onset (WASO). There were a small number of randomized controlled trials (RCTs; $n = 4$) and a high risk of bias across the RCTs; therefore, within sleep condition meta-analyses were examined ($n = 221$). At post-intervention, subjective TST improved by 29.47 min (95% CI 17.18, 41.75), SOL by 21.44 min (95% CI −30.78, −12.11), SE by 5.34% (95% CI 2.64, 8.04), and WASO by a medium effect size [$d = 0.59$ (95% CI 0.36, 0.82)]. Objective SOL improved by 16.15 min (95% CI −26.13, −6.17) and SE by 2.82% (95% CI 0.58, 5.07). Global sleep quality, daytime sleepiness, depression, and anxiety also improved. Gains were generally maintained over time. Preliminary evidence suggests that adolescent cognitive–behavioral sleep interventions are effective, but further high-quality RCTs are needed. Suggestions for further research are provided.

Keywords Adolescence · Sleep · Insomnia · Delayed sleep phase disorder · Anxiety · Depression · Intervention · Cognitive–behavioral therapy · Mindfulness · Systematic review · Meta-analysis

Introduction

There is growing recognition that many adolescents obtain insufficient and/or poor-quality sleep, which is increasingly being regarded as an epidemic of sleep deprivation among adolescents and an important public health problem

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(Adolescent Sleep Working Group 2014; American Medical Association 2010; Millman 2005; Office of Disease Prevention and Health Promotion 2011). The American Academy of Sleep Medicine, the National Sleep Foundation, and the American Center for Disease Control and Prevention have suggested that adolescents optimally require between 8 and 10 h of sleep per night (Hirshkowitz et al. 2015; Paruthi et al. 2016). For example, longitudinal studies have shown that adolescents sleep around 9.2 h per night under ad lib (unrestricted) sleep conditions (Carskadon and Acebo 2002). However, recent systematic reviews and meta-analyses have demonstrated that many adolescents obtain insufficient sleep (i.e., <8 h), especially on school nights (Gradisar et al. 2011b). Sixty-five percent of adolescents report sleep onset latencies exceeding 30 min (Hysing et al. 2013), more than half report the need for more sleep (Wolfson and Carskadon 1998), and most (59%) wake feeling unrefreshed at least a few times per week (National Sleep Foundation 2011).

Prevalence estimates of sleep disorders in adolescence vary considerably, but according to studies that have incorporated rigorous criteria, approximately 30% of adolescents suffer from a sleep disorder (Ohayon and Roberts 2001). Insomnia is the most prevalent sleep disorder among adolescents (Johnson et al. 2006; Roberts et al. 2009). Insomnia is defined as chronic dissatisfaction with sleep quantity and/or quality, despite adequate opportunity to sleep (American Psychiatric Association 2013). It is associated with difficulty initiating and/or maintaining sleep, early morning awakening, and unrefreshing sleep (Johnson et al. 2006). Approximately 8–11% of young people meet diagnostic criteria for insomnia at any one time (Dohnt et al. 2012), which tends to persist over time (Roberts et al. 2009).

Delayed sleep phase disorder (DSPD) is defined as normal sleep that is delayed in its timing with respect to the individual's sleep onset and rising times (American Psychiatric Association 2013). Seventeen percent of adolescents report difficulties falling asleep before 2 a.m. at least three times per week (Saxvig et al. 2012). Furthermore, between 1 and 7% of adolescent's meet diagnostic criteria for DSPD (Johnson et al. 2006; Ohayon et al. 2000; Pelayo et al. 1988), with the majority (51%) reporting at least one symptom (Lovato et al. 2013). This delay in sleep onset may lead to significant difficulty in rising for school in the morning, school non-attendance, daytime sleepiness, chronic sleep reduction, and poor school performance (Gradisar and Crowley 2013).

A number of factors interact to make sleep susceptible to disturbance in adolescence. First, children and adolescents are subject to the same physiological susceptibilities and psychological and environmental vulnerabilities that cause insomnia in adults (Keller & El-Sheikh 2011), such as predisposition to cognitive–emotional hyperarousal (Fernandez-Mendoza et al. 2011). Second, sleep during adolescence is affected by physiological development (Colrain and Baker 2011). Adolescence is associated with a progressive reduction in the accumulation of homeostatic sleep pressure during wakefulness, which leads to a reduction in sleep drive (Feinberg et al. 2006). Adolescence is also associated with a delay in the timing of sleep, which is related to a lengthening of the intrinsic period of the endogenous circadian oscillator (Carskadon et al. 2004). Melatonin is released later in the evening among adolescents than children, which also delays the onset of evening sleepiness (Carskadon et al. 1993). Third, parental control over bedtime lessens during adolescence (Short et al. 2011). Fourth, adolescents develop responsibilities and social interests (e.g., homework, employment, friendships) that encourage remaining awake later into the evening (Adam et al. 2007; Maume 2013). Finally, electronic devices have a deleterious impact on sleep in adolescence, including delaying sleep onset and reducing sleep duration (Bartel et al. 2015; Hale and Guan 2015). These physiological maturational processes and social and cultural factors have been described as a "perfect storm" of factors in adolescence (Carskadon 2011), so that reduced sleep propensity in the late evening becomes permissive of continued waking activities and delayed bedtimes (Carskadon 2011; Jenni and LeBourgeois 2006). This delay in sleep has two potential sleep-related consequences: (a) sleep restriction, because school starts early in the morning, and (b) reduced restorative value of sleep, because recovery sleep tends to occur at an inappropriate circadian phase (Carskadon et al. 2004).

Adolescent Sleep and Mental Health

Sleep disturbance is an important contributor to, and maybe even cause of, cycles of increasing vulnerability and risk among young people (Harvey 2015). Sleep problems are pervasive in psychiatric disorders and precipitate and maintain many emotional and behavioral problems (Dahl and Harvey 2007; Harvey et al. 2011). Indeed, there is emerging evidence that sleep may qualify as a transdiagnostic process (Harvey 2015; Harvey et al. 2011). Sleep problems are a highly relevant "bridge symptom" (Baglioni et al. 2014) and share the highest percentage of connected symptoms within all symptoms in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association 1994; Borsboom et al. 2011). In particular, there is a strong relationship between sleep disturbance and internalizing problems in adolescence. Recent evidence suggests that sleep problems, particularly wakefulness in bed (e.g., prolonged sleep onset latency and poor sleep efficiency), precede the development of anxiety and depression in

adolescence more than the reverse (Lovato and Gradisar 2014; McMakin and Alfano 2015).

Although the specific mechanisms are not fully understood, research suggests that shared biological, cognitive, and interpersonal risk factors may underlie the relationship between sleep disturbance and mental health problems in adolescence. In particular, several modifiable risk mechanisms appear to underlie the relationship between sleep disturbance and internalizing problems, including stress/arousal, emotion processing, and cognitive factors (Cowie et al. 2014). To the extent that sleep and mental health problems in youth share common etiological underpinnings, early treatment programs for sleep problems might reduce the risk of developing mental health problems and can be considered a helpful general preventive strategy (Baglioni et al. 2011; Dahl and Harvey 2007).

Cognitive–Behavioral Sleep Interventions

Cognitive–behavioral sleep interventions are short-term, multicomponent, goal-oriented psychotherapeutic treatments. They aim to modify the patterns of thinking and behavior that may be underlying an individuals sleep disturbance, such as poor sleep hygiene, irregular sleep–wake schedules, delayed bedtimes, pre-sleep hyperarousal, and maladaptive sleep-related cognitions. Before reviewing adolescent cognitive–behavioral sleep interventions, we will review adult cognitive–behavioral sleep interventions, as the adult literature is more extensive.

Research Among Adults

Cognitive–Behavioral Therapy for Insomnia Cognitive–behavioral therapy for insomnia (CBT-I) involves behavioral techniques such as sleep education, sleep hygiene instruction, stimulus control, sleep restriction, and relaxation training, but also addresses negative thought processes and unhelpful beliefs about sleep through a focus on bedtime worry and rumination (for a review, see Edinger and Means 2005). The components of CBT-I are described in Table 1. Empirical support for the effectiveness of CBT-I has grown steadily since the 1960s, especially over the past 15 years. CBT-I is recommended as an effective therapy for insomnia by the Standards of Practice Committee of the American Academy of Sleep Medicine (Morgenthaler et al. 2006). The American College of Physicians recently recommended that all adult patients should receive CBT-I as an initial treatment for chronic insomnia (Qaseem et al. 2016).

There is strong evidence from multiple systematic reviews and meta-analyses that CBT-I improves sleep in adults, usually with medium–large effect sizes (Koffel et al. 2015; Murtagh and Greenwood 1995; Morin et al. 1994; Trauer et al. 2015; van Straten et al. 2017). Effects tend to be strongest for wakefulness-in-bed variables compared to sleep duration variables and subjective sleep variables compared to objective sleep variables. Effects also tend to persist over time. For example, a recent systematic review and meta-analysis of 20 randomized controlled trials (RCTs) among adults diagnosed with insomnia [1162 patients (64% female; mean age = 56 years), range 20–201 patients] found that face-to-face CBT-I (delivered individually or in groups on at least two occasions) produced marked and statistically significant improvements in sleep diary measured sleep onset latency [SOL; 19.03 min (95% CI 14.12–23.93 min)], wake after sleep onset [WASO; 26.00 min (95% CI 15.48–36.52 min)], and sleep efficiency [SE; 9.91% (95% CI 8.09, 11.73%)], but not total sleep time

Table 1 Components of cognitive–behavioral therapy for insomnia (CBT-I)

Component	Description
Sleep hygiene	Psychoeducation about healthy sleep behaviors and sleep promoting environmental conditions. Specific instructions include establishing regular bedtimes and rise times, limiting napping during the day, avoiding lying in bed waiting to fall asleep, maintaining predictable pre-sleep activities and establishing a wind down routine before bed, creating a quick wake up routine, avoiding stimulating or emotionally laden activities before bedtime, avoiding consuming high-energy substances in the evening, creating a comfortable sleep environment, and only using the bed for sleep-related activities
Stimulus control	Behavioral instructions aimed at reducing conditioned arousal to the bed and bedroom and re-associating the bed and bedroom with normal sleep. Instructions include only going to bed when sleepy, setting a standard rise time, leaving the bed/bedroom after long periods of wakefulness, avoiding sleep-incompatible behaviors in the bed or bedroom, and avoiding napping
Sleep restriction	Behavioral instructions aimed at restricting time in bed as a way of increasing homeostatic sleep drive. Time allowed in bed is initially restricted to the average time perceived as sleep per night and then adjusted to ensure sleep efficiency remains above 85%
Relaxation	Any relaxation technique aimed at reducing physiological and cognitive arousal prior to sleep. Specific techniques might include progressive muscle relaxation, guided imagery, and/or breathing techniques
Cognitive therapy	Aims to addresses negative thought processes and unhelpful beliefs about sleep through a focus on bedtime worry and rumination. Specific techniques include identifying and challenging unrealistic expectations of sleep, fear of missing out on sleep, and overestimation of the consequences of poor sleep

[TST; 7.61 min (95% CI −0.51–15.74 min)] at post-treatment time points compared to control conditions (Trauer et al. 2015). Improvements were maintained at follow-up, suggesting that treatment gains persist over time. The effect sizes were similar in size to those seen in meta-analyses of hypnotics, such as benzodiazepines (Huedo-Medina et al. 2012; Nowell et al. 1997). However, different to hypnotics, CBT-I effects are likely to continue after treatment cessation (Sivertsen et al. 2006) and to have fewer side effects, such as adverse effects and rebound insomnia after discontinuation (Buscemi et al. 2007; Kales et al. 1991). For polysomnography-measured sleep, only five studies were available for meta-analysis, and effect sizes were similar to the sleep diary outcomes. For actigraphy-measured sleep (a device that measures the degree and intensity of motion), only three studies were analyzed, and the effect size estimates were markedly lower than for the sleep diary outcomes. However, a major limitation of the meta-analysis was that only one study included in the review had low risk of bias across all domains—while most studies were methodologically rigorous in regard to patient follow-up and outcome reporting, allocation concealment and blinding were rarely reported or were not undertaken. Furthermore, approximately half of the studies used a waiting list or placebo tablet comparator, rather than an active control condition. Wait list control groups do not account for treatment expectation effects, demand characteristics, and non-specific therapeutic factors, such as the quality of the therapeutic alliance and relationship. Studies with wait list control groups also tend to observe larger effect sizes than those using a psychological placebo (Furukawa et al. 2014).

Regarding the comparative efficacy of different modalities, three meta-analyses of psychological interventions for sleep problems have suggested that individual and group treatments are equally effective (Murtagh and Greenwood 1995; Trauer et al. 2015; van Straten et al. 2017), while another indicated that individual treatment is most effective (Morin et al. 1994). Three studies have directly compared individual and group CBT-I. Two of the studies found that both forms of CBT-I were equally effective at improving sleep (Bastien et al. 2004; Verbeek et al. 2006), while the third study found that individual CBT-I resulted in greater improvement on several sleep variables compared to group CBT-I, including SOL and sleep quality (Yamadera et al. 2013). However, emerging evidence suggests that face-to-face treatments delivered over at least four sessions are more effective than self-help interventions (either though books, audio, or Internet) or face-to-face interventions with fewer sessions (van Straten et al. 2017). For example, Lancee et al. (2016) recently conducted a randomized controlled non-inferiority trial comparing an online treatment program (dCBT-I) to standard CBT-I (6 weekly, 45-min sessions) and a wait list control among 90 (30 per group) individuals (mean age = 41.6 years) who were diagnosed with insomnia. Although dCBT-I and face-to-face CBT-I both significantly improved many features of sleep relative to wait list controls, face-to-face CBT-I treatment was found to be superior to online treatment. Patient adherence to online interventions also represents a major challenge, as indicated by the 50% completion rate for computerized CBT-I in two recent studies (Lancee et al. 2016; Christensen et al. 2016).

There is also emerging evidence that CBT-I leads to improvements in physical and mental health symptoms, including reductions in anxiety and depression, with small–medium effect sizes (Ballesio et al. 2017; Belleville et al. 2011; Taylor and Pruiksma 2014). For example, a recent systematic review of 16 RCTs that assessed the efficacy of in-person CBT-I among psychiatric populations [571 patients (aged 20–70 years), range 17–63 patients] found that CBT-I resulted in significant small-to-medium effects in depression symptomatology [mean effect size = 0.51 (95% CI 0.31–0.70)] and marginal improvements in anxiety symptomatology [mean effect size = 0.21 (95% CI −0.08, 0.50)] relative to control conditions (Taylor and Pruiksma 2014). However, again, more than half of the studies included in the review used wait list control conditions.

Other important findings from meta-analyses of adult CBT-I are that samples recruited from the community and that have shorter duration of insomnia diagnoses tend to show greater improvements in sleep variables post-treatment than do clinical samples (Koffel et al. 2015; Murtagh and Greenwood 1995). This is probably related to chronicity of sleep disturbance, suggesting that those beginning treatment with fewer sleep problems may do better in treatment (Koffel et al. 2015), highlighting the importance of early intervention. For example, several commentators have argued that future research is needed to determine whether cognitive–behavioral sleep interventions can be delivered prophylactically to at-risk, pre-insomniac psychiatric populations (Vitiello et al. 2013).

Mindfulness-Based Sleep Interventions Despite the effectiveness of CBT-I, many patients have not achieved clinically significant outcomes (Morin et al. 2006), suggesting that there is room for improvement among cognitive and behavioral approaches to sleep problems. There is emerging evidence that sleep problems can also be treated successfully using protocols that include a mindfulness component. Mindfulness can be defined as "the awareness that emerges through paying attention on purpose, in the present moment, and non-judgmentally to the unfolding of experience" and is considered a "third-wave" cognitive–behavioral therapy (Kabat-Zinn 2003, p. 145). Several RCTs have evaluated in person mindfulness-based

therapies in adults with sleep problems (Britton et al. 2010, 2012; Garland et al. 2014; Gross et al. 2011; Ong et al. 2014) and have found significant improvements within active treatment conditions for subjective and objective measures of sleep, especially SOL and SE, but not beyond active control conditions (Garland et al. 2014; Gross et al. 2011). However, many of these studies were limited by small sample sizes (mean = 49, range 23–111).

Cognitive–Behavioral Therapies for DSPD Finally, there is accumulating evidence that DSPD can be treated successfully using cognitive–behavioral therapies. Along with timed melatonin administration, timed light exposure and chronotherapy are recommended as effective therapies for DSPD by the Standards of Practice Committee of the American Academy of Sleep Medicine (Morgenthaler et al. 2006), based on a systematic review of the literature (Sack et al. 2007). Bright light therapy (Lewy and Sack 1996) consists of scheduled natural sunlight and/or broad-spectrum white light from a specialized lamp at usual wake time, while incrementally advancing light exposure each day. The goal of morning light exposure is to shift the circadian rhythm earlier and therefore correct the pathological phase delay. Chronotherapy (Czeisler et al. 1981) involves a prescribed progressive delay in the schedule of sleep time until the desired sleep schedule is reached. The treatment is based on the premise that patients with DSPD may have greater difficulty shifting their rhythm in an advance direction, and that the timing of sleep is the main synchronizer of the circadian system.

Research Among Adolescents

Despite the fact that (1) cognitive–behavioral therapies are firmly established as frontline treatments for many adult sleep problems/disorders, and (2) and there is high prevalence, high chronicity, and serious consequences associated with adolescent sleep problems/disorders, research on adolescent cognitive–behavioral sleep interventions is not as developed as the adult literature. No previous systematic reviews and meta-analyses have been conducted to provide a synthesis of this developing field, a lacuna in the literature that we address here.

The research on adolescent cognitive–behavioral sleep interventions may be less developed compared to the adult literature for a number of reasons. First, adolescent sleep intervention research has tended to focus on school-based sleep education programs, which have the potential to reach a large number of adolescents (i.e., whole school classes) and are relatively easy to implement (e.g., "train the teacher"). However, recent reviews and large-scale RCTs have found that while these psychoeducational programs are effective in increasing students' knowledge about sleep and insomnia, they are less effective in improving sleep behavior or mental health (Blunden et al. 2012; Blunden and Rigney 2015; Gruber 2016; Kira et al. 2014; Wing et al. 2015). These findings are consistent with recent reviews suggesting that targeted interventions are more effective than universal interventions in preventing child and adolescent depression (Horowitz and Garber 2006; Rohde 2015), and that simple sleep hygiene education does not guarantee positive outcomes in adults (Irish et al. 2015). In sum, these findings suggest that active sleep interventions that incorporate cognitive–behavioral principles demonstrated to be effective in adult populations (e.g., CBT-I, mindfulness-based sleep interventions, bright light therapy, and chronotherapy), and that are delivered to students who are already experiencing early signs of sleep and/or mental health problems, may be more effective (Wensing et al. 2010). Second, the later chronophase of adolescents may mask insomnia symptoms and/or reduce motivation to seek help. Many clinicians and researchers have reported an overlap between DSPD and sleep onset insomnia symptoms, especially on school nights (Gradisar and Crowley 2013). However, adolescents may experience delayed bedtimes and wakefulness in bed as egosyntonic, for both biological and social reasons. For example, delayed bedtimes and prolonged SOL may afford adolescents with opportunities to complete homework and/or use electronic devices in bed without parental supervision. Furthermore, adolescents may be reluctant to seek help for sleep inertia, daytime sleepiness, and fatigue, as they may see these experiences as normative parts of development and/or embarrassing/shameful. Finally, parents, teachers, and health professionals may focus on addressing the consequences of adolescent sleep disturbance, such as anxiety, depression, difficulty concentrating, and poor school performance, rather than the underlying causes (i.e., insomnia or DSPD).

Methods

A systematic review and meta-analysis was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher et al. 2015). The primary aim of the systematic review and meta-analysis was to answer the following research question: What is the efficacy of adolescent cognitive–behavioral sleep interventions for subjective (e.g., questionnaire/sleep diary) and objective (e.g., actigraphy) sleep outcomes, compared with baseline and control, for adolescents with self-identified sleep problems and/or a diagnosis of a sleep disorder? A secondary aim of the review was to examine the effects of the interventions on self-reported global sleep quality, daytime sleepiness, anxiety, and depression.

Study Selection

Eligible studies were trials of cognitive–behavioral sleep interventions among adolescents. Inclusion criteria were:

1. Participants were adolescents aged between 10 and 19 years. The age range was based on the definition of adolescence adopted by the World Health Organization (2015).
2. Studies were published in peer-review journals.
3. Studies included more than three participants (i.e., were not case studies).
4. The primary aim/focus was the use of a multimodal sleep intervention based on cognitive and behavioral principles.
5. Interventions incorporated at least two behavioral components (e.g., sleep hygiene, stimulus control, sleep restriction, and/or bright light therapy) and at least one cognitive component (e.g., identifying and challenging dysfunctional beliefs and attitudes about sleep and/or pre-sleep worry). A previous systematic review and meta-analysis evaluated *behavioral* interventions for pediatric insomnia (Meltzer and Mindell 2014), but did not include *cognitive*–behavioral interventions, and focused on both children and adolescents (the review identified 16 controlled trials and 12 within-subject studies, but only one study was conducted among adolescents). Furthermore, the package of CBT-I has been shown to be more effective than separate delivery of the behavioral or cognitive components for improving insomnia among adults. Harvey et al. (2014) found that cognitive therapy resulted in slower, but more long-lasting, improvements in insomnia symptoms compared with behavior therapy. However, the full complement of CBT-I produced the largest short- and long-term effects on insomnia symptoms.
6. Interventions were delivered on at least four occasions. Adult CBT-I is usually conducted over the course of four to eight sessions (Edinger and Means 2005), and a recent meta-analysis found that adult CBT-I delivered on at least four occasions is more effective than CBT-I delivered on fewer occasions (van Straten et al. 2017).
7. Interventions were delivered individually or in small groups. School-based sleep education programs were excluded because they are delivered to entire school classes without regard to individual risk factors or symptoms, are didactic rather than therapeutic, and do not include cognitive restructuring.
8. Interventions were delivered in person. Interventions delivered online were excluded because the aim of the review was to evaluate the efficacy of face-to-face interventions, and recent evidence suggests that face-to-face CBT-I treatment is superior to online treatment (e.g., Lancee et al. 2016; van Straten et al. 2017).
9. Participants were identified as having self-reported symptoms of sleep disturbance (e.g., high scores on a screening questionnaire) or a diagnosis of a sleep disorder (e.g., insomnia or DSPD).
10. To increase the generalizability of findings, studies evaluating participants with comorbid medical or psychiatric problems were not excluded, as adolescents with sleep problems and disorders often have related physical and mental health problems.
11. Finally, studies that combined traditional CBT-I therapies with "third-wave" therapies (e.g., mindfulness) or other treatment modules (e.g., anxiety-/depression-specific components, bright light therapy), were not excluded, as adult studies suggest the efficacy of such combined approaches (Britton et al. 2010, 2012; Garland et al. 2014; Gross et al. 2011; Ong et al. 2014), and the aim of the review was to evaluate the broad-based and transdiagnostic effects of adolescent cognitive–behavioral sleep interventions.

Data Sources and Searches

Online searches of the PubMed (United States Library of Medicine), PsycINFO (Wolters Kluwer Health OvidSP), CENTRAL (Cochrane Central Register of Controlled Trials), EMBASE, and MEDLINE (Thomson Reuters Web of Knowledge) databases were performed on May 1, 2016, by the first author using several relevant search terms [e.g., (sleep OR insomnia) AND (intervention OR therapy OR treatment) AND (cognitive–behavior OR cognitive–behaviour OR CBT-I OR mindfulness OR multimodal OR multicomponent)] and search limits (e.g., "Ages: Adolescent"). Full data sources and search terms are provided in Online Resource 1. Abstracts were examined for references to cognitive–behavioral sleep interventions and adolescence, and if the study appeared relevant, then the full text was retrieved. "Pearling," the process of manually scanning the reference list of identified articles, was used for additional relevant studies not identified in the electronic database search. Examination of abstracts and reference lists for selection of appropriate articles was conducted independently by two authors (MB, LS).

Data Extraction

Pertinent details from each study were recorded using standardized forms, including methodological information regarding study design, sample characteristics, intervention framework, program delivery, and outcome measures. These details are described in the study characteristics section and Table 3. MB and LS independently extracted the data.

Quality Assessment

To conduct an appraisal of the studies' methodological quality, each of the trials was evaluated according to the quality index for randomized and non-randomized studies proposed by Downs and Black (1998). The index was adapted to be consistent with a procedure used by Blunden et al. (2012) in their review of school-based sleep education programs. The original quality index is a 27-item checklist assessing five subscales: (1) reporting whether the information provided in the paper is sufficient to allow the reader to make an unbiased assessment of the findings; (2) external validity: the extent to which the findings from the study could be generalized to the population from which the study subjects were derived; (3) bias: the extent of bias in the measurement of the program and the outcome; (4) confounding: addressing the bias in the selection of study subjects, and (5) power: whether the negative findings from a study could be due to chance. Item descriptions are provided in Table 2. Answers are scored 0 or 1, except for one item in the reporting subscale that scored 0–2, and the single item on power, which scored 0–5. The total maximum score for quality is 32. However, because most studies included in the review did not report statistical power, and there was not enough data to calculate power, the power score was excluded. Therefore, 26 of the possible 27 items were evaluated, giving a maximum score of 27 points.

The Downs and Black (1998) quality index has demonstrated high internal consistency (Kuder–Richardson 20: 0.89), good test–retest ($r = 0.88$) and inter-rater ($r = 0.75$) reliability, and high correlations ($r = 0.90$) with other validated quality assessment instruments ($r = 0.90$) for non-RCTs. It has been recommended as a strong instrument for assessing the methodological quality of interventions (Deeks et al. 2003; Higgins and Green 2011).

Additionally, all controlled trials were reviewed for risk of bias using the recommended Cochrane guidelines (Higgins and Green 2011) with ratings for randomization, allocation concealment (selection bias), blinding of outcome assessment (detection bias), and selective reporting (reporting bias).

LS and MR independently completed the study quality assessments and risk-of-bias ratings. Given that one of these raters (MR) was an author on one of the studies, an independent rater (AP; see Acknowledgements) performed an additional rating of the study by Blake et al. (2016).

Data Synthesis and Analysis

The main outcomes of interest were subjective (e.g., sleep diary/questionnaire) and objective (e.g., actigraphy) measures of TST, SOL, SE, and WASO. Secondary outcomes of interest were questionnaire-based ratings of global sleep quality, daytime sleepiness, anxiety, and depression. Primary and secondary endpoints were assessed at two time points: immediately after the treatment and at follow-up.

All analyses used random-effects models. Heterogeneity was assessed using the I^2 statistic (25% = low, 50% = moderate, 75% = high; Higgins et al. 2003). Additionally, all trials were assessed for publication bias using funnel plots and the Egger test (Egger et al. 1997; Higgins et al. 2003). Publication bias was only assessed when there were three or more studies for the relevant analysis. When variables were defined consistently across studies (i.e., objective and subjective SOL, TST, SE), effects were based on unstandardized raw mean differences. When variables were not defined consistently across studies (i.e., objective and subjective WASO), or when different questionnaires were used to assess the same construct (i.e., global sleep, daytime sleepiness, anxiety, and depression), data were pooled using standardized mean differences using change score standardization. For all analyses, we calculated effects based on an assumed correlation between measurements over time of $r = 0.5$, which was based on results obtained in Blake et al. (2016). Effect sizes were based on Cohen's d and interpreted with the following: 0.2 = small, 0.5 = medium, 0.8 = large (Cohen 1992).

Four primary sensitivity analyses were performed. First, we restricted analyses to studies with high methodological quality, defined as a score of 17 or above on the Downs and Black (1998) instrument. We chose 17 as the cutoff because all RCTs scored above 16, while all uncontrolled trials scored below 17 (see Table 4). Second, we limited analyses to studies that identified adolescents through clinical diagnosis, rather than based on self-report symptoms, as adolescents with more severe problems may benefit more from intervention. Third, we restricted analyses to studies that evaluated adolescents with "pure" sleep problems, rather than adolescents with concomitant sleep and psychiatric problems, as concomitant problems may interfere with sleep treatment. Fourth, we limited analyses to studies that incorporated sleep restriction because this may be among the most effective component

Table 2 Downs and Black (1998) criteria used to appraise the methodological quality of the reviewed studies

1.	Is the hypothesis/aim/objective of the study clearly described?
2.	Are the main outcomes to be measured clearly described in the Introduction and Methods section?
3.	Are the characteristics of the patients included in the study clearly described?
4.	Are the interventions of interest clearly described?
5.	Are the distributions of principal confounders in each group of subjects to be compared clearly described?
6.	Are the main findings of the study clearly described?
7.	Does the study provide estimates of the random variability in the data for the main outcomes?
8.	Have all important adverse events that may be a consequence of the intervention been reported?
9.	Have the characteristics of patients lost to follow-up been described?
10.	Have actual probability values been reported for the main outcomes except where the probability value is less than 0.001?
11.	Were the subjects asked to participate in the study representative of the entire population from which they were recruited?
12.	Were those subjects who were prepared to participate representative of the entire population from which they were recruited?
13.	Were the staff, places, and facilities where the patients were treated, representative of the treatment the majority of patients receive?
14.	Was an attempt made to blind study subjects to the intervention they have received?
15.	Was an attempt made to blind those measuring the main outcomes of the intervention?
16.	If any of the results of the study were based on "data dredging," was this made clear?
17.	In trials and cohort studies, do the analyses adjust for divergent lengths of follow-up of patients, or in case–control studies, is the time period between the intervention and outcome the same for cases and controls?
18.	Were the statistical tests used to assess the main outcomes appropriate?
19.	Was compliance with the intervention/s reliable?
20.	Were the main outcome measures used accurate (valid and reliable)?
21.	Were the patients in divergent intervention groups (trials and cohort studies) or were the cases and controls (case–control studies) recruited from the same population?
22.	Were study subjects in divergent intervention groups (trials and cohort studies) or were the cases and controls (case–control studies) recruited over the same period of time?
23.	Were study subjects randomized to intervention groups?
24.	Was the randomized intervention assignment concealed from both patients and health care staff until recruitment was complete and irrevocable?
25.	Was there adequate adjustment for confounding in the analyses from which the main findings were drawn?
26.	Were losses of patients to follow-up taken into account?

All items are scored 0 or 1, except item 5, which is scored 0, 1 or 2

of CBT-I (Fernando et al. 2013). Sensitivity analyses were only conducted at the post-intervention time point, as few trials included follow-up time points. Finally, we conducted a separate set of sensitivity analyses to explore whether increasing or decreasing the assumed correlation between measurements over time (from the assumed $r = 0.5$) influenced the results.

We did not conduct sensitivity analyses on length of treatment (i.e., short vs. long), type of facilitator (i.e., psychologist vs. experienced CBT-I sleep therapist), type of delivery (i.e., individual vs. group), or type of intervention (i.e., pure CBT-I vs. added components), as there was little variation across trials. Several authors have recommended limiting the number of sensitivity analyses in meta-analyses with relatively few studies (Cuijpers 2016).

All statistical tests were two-tailed, with p values <0.05 considered statistically significant. For effect size and I^2 estimates, 95% confidence intervals were calculated. Meta-analyses were performed using the metafor package v1.9.8 (Viechtbauer 2010), in R software v 3.3.1 (R Core Team 2015).

Results

Study Selection

One hundred and eighty-nine articles were retrieved from the database searches, of which 10 met inclusion criteria. The study flow diagram is presented in Fig. 1. Two articles (Bootzin and Stevens 2005; Britton et al. 2010) reported data from the same trial (pre- and post-intervention and follow-up). Therefore, nine trials were included in the final analysis. One trial (Clarke et al. 2015) included adolescents aged 12–20 years. However, because only 5% of the participants ($n = 2$) were aged over 19 years, this study was included in the final analysis.

Study Characteristics

Table 3 shows the general characteristic of the nine selected trials. Three trials were large-scale RCT's (Blake et al. 2016; De Bruin et al. 2015; Gradisar et al. 2011a), one was a prospective RCT (Clarke et al. 2015), and five were uncontrolled feasibility trials (Bei et al. 2013; Bootzin and Stevens 2005; De Bruin et al. 2014; Roeser et al. 2016; Schlarb et al. 2011). Two RCTs used active controls (Blake et al. 2016; Clarke et al. 2015), and two used wait list comparators (De Bruin et al. 2015; Gradisar et al. 2011a). Three trials were conducted in Australia, two in the USA, two in the Netherlands, and two in Germany. Two hundred and twenty-one participants (mean age = 14.89 years; female = 62.27%; range 11–20 years) completed the sleep intervention conditions (average per study = 25 participants, range 9–63 participants), and three hundred and fifty-seven participants (mean age = 14.97 years; female = 61.74%; range 11–20 years) completed the trials overall (average per study = 40 participants, range 9–123 participants). Seven out of nine interventions were delivered in groups (ranging from 2 to 9 participants), two individually, and two included parents. Psychologists delivered seven out of the nine interventions and experienced CBT-I sleep therapists the other two. Eight out of nine interventions were delivered over six/seven weekly 90- to 100-min sessions and one in 10 weekly sessions over 12 weeks. Three trials evaluated adolescents with insomnia disorder, one DSPD, and four with sleep/insomnia complaints. Four trials included participants with comorbid mental health disorders/symptoms (substance abuse, depression, anxiety), and three trials excluded participants with non-sleep disorders. Six trials included longitudinal follow-up, ranging from 2 to 12 months.

The content of the interventions reflects the range of choices facing clinicians (Harvey 2015). Only two studies evaluated manualized CBT-I (De Bruin et al. 2014, 2015); the other interventions included added treatment components. All nine interventions incorporated cognitive restructuring, eight sleep education, eight sleep hygiene, eight stimulus control, five sleep restriction, four relaxation techniques, two bright light therapy, two mindfulness-based cognitive therapy, one mindfulness-based stress reduction, two savoring, two hypnotherapy, two anxiety-specific modules, and one depression-specific modules. The mean number of treatment components was 6 (range 4–7).

Study Quality

The methodological quality of the reviewed studies is presented in Table 4. The average total score for the nine reviewed trials was 16 out of a possible 27 (59%), ranging from 11 to 19 (41–70%). All of the trials scored poorly on the external validity subscale, and most of the trials scored poorly on the confounding subscale, suggesting that the trials had low generalizability. Furthermore, few trials attempted to blind those measuring the main outcomes of the intervention (item 15) or measured compliance with the intervention (item 19).

Risk of Bias

The risk of bias in the controlled studies is presented in Table 5. Only two RCTs were universally assessed as having low risk of bias across all domains. Sequence generation and allocation concealment generally followed accepted methods. However, many RCTs did not report blinding techniques in detail. No RCTs described blinding of participants and personnel, possibly because of the difficulty of doing so with behavioral interventions. Moreover, two RCTs did not describe blinding of outcome assessors. Additionally, one RCT included incomplete outcome data (i.e., did not use intent-to-treat analysis and/or appropriate data imputation methods). Furthermore, selective outcome reporting was generally unclear, because most RCTs failed to publish a methods protocol paper prior to the commencement of the interventions. In sum, there was a high risk of bias across the RCTs. That is, the proportion of information from the RCTs at a high risk of bias was sufficient to affect the interpretation of the results (Higgins and Green 2011).

Meta-analyses

Because a small number of RCTs were available for meta-analyses ($n = 4$), and there was a high risk of bias across the RCTs, meta-analyses comparing sleep and control condition were considered unreliable. Nevertheless, these results are presented in Online Resource 2, but should be interpreted with caution. Within sleep condition meta-analyses were examined instead (Table 6).

Primary Endpoints

All nine trials measured subjective sleep using sleep diaries or self-report questionnaires, six measured objective sleep using actigraphy, and five included a follow-up time point. While most trials reported TST, not all reported SOL, SE, or WASO.

There were marked and statistically significant improvements in subjective TST [29.47 min (95% CI 17.18–41.75 min; $I^2 = 26.62$)], SOL [−21.44 min (95% CI −30.78 to −12.11 min; $I^2 = 85.94$)], SE [5.34% (95% CI 2.65, 8.04%; $I^2 = 69.60$)], and WASO [$d = 0.59$ (95% CI 0.36, 0.82; $I^2 = 28.64$)], at the post-intervention time

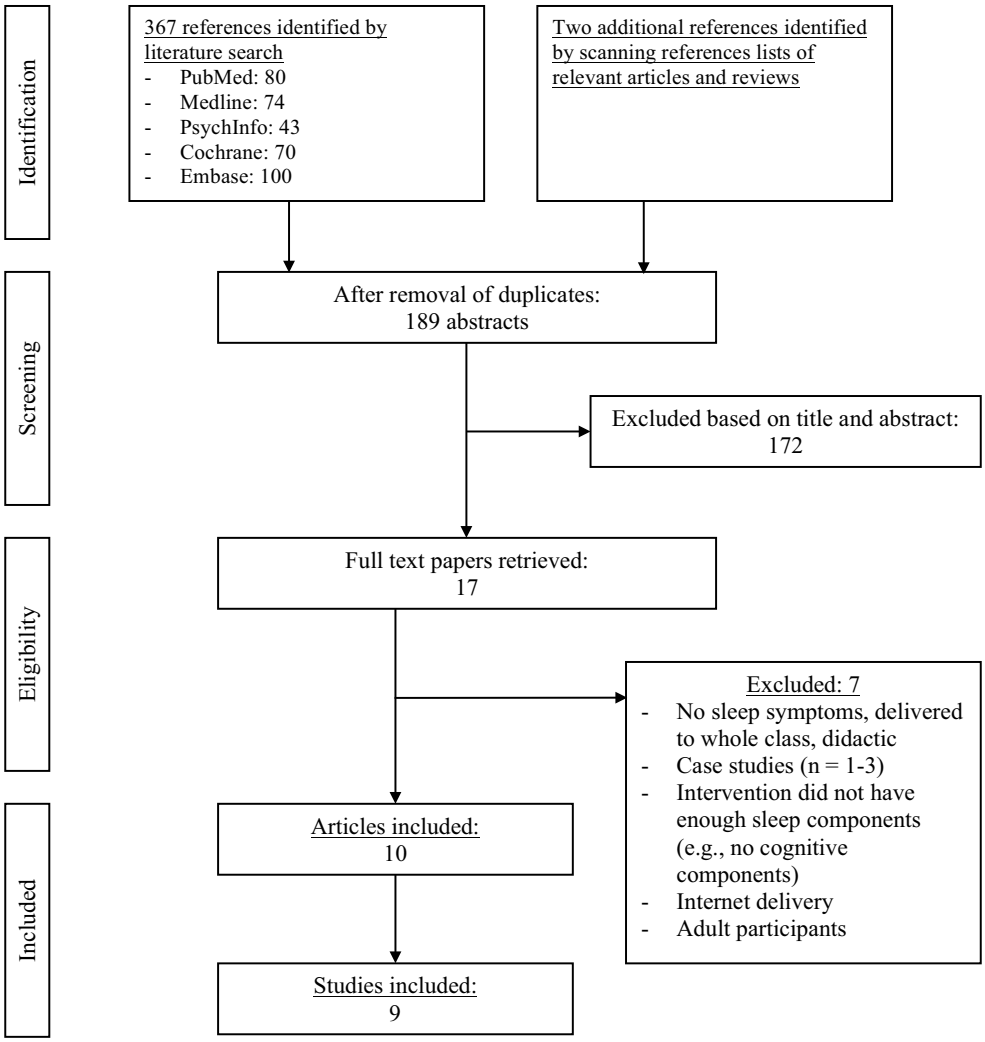

Fig. 1 Flowchart of inclusion of studies

point. Improvements were generally maintained at the follow-up time point and increased for subjective TST and SOL. Moreover, there were marked and statistically significant improvements in objective SOL [−16.15 min (95% CI −26.13 to −6.17 min; $I^2 = 72.87$)] and SE [2.82% (95% CI 0.58, 5.07%; $I^2 = 66.06$)], at the post-intervention time point. Improvements were maintained at follow-up and increased for SE, but decreased for SOL. There was also a marginal improvement in objective TST [9.19 min (95% CI −0.65 to 19.04 min; $I^2 = 10.40$)] at the post-intervention time point although there was only weak evidence to reject the null hypothesis of no effect ($p = 0.07$), and there was no statistically significant support that this improvement was maintained at follow-up, possibly because fewer studies were available for this meta-analysis (i.e., $n = 3$). Objective WASO did not improve at the post-intervention or follow-up time points.

It should be noted, however, that there was evidence of moderate–high heterogeneity between trials in SOL and SE outcomes, as well as considerable uncertainty in the heterogeneity estimates (shown by the wide I^2 confidence intervals).

Secondary Endpoints

Five trials measured global sleep quality. There was a significant improvement at the post-intervention time point, with large effect sizes [$d = -0.92$ (95% CI −1.52, −0.32; $I^2 = 86.89$)]. Improvements were maintained at follow-up. Four trials measured daytime sleepiness. There was a significant improvement at the post-intervention time point, with a moderate effect size [$d = -0.63$ (95% CI −1.07, −0.18; $I^2 = 79.34$)]. Improvements increased at follow-up. Three trials measured depression symptoms. There were marginal improvements at post-intervention, with a large effect size [$d = -1.22$ (95% CI −2.47, 0.01; $I^2 = 94.56$)]. Improvements increased at follow-up. Two studies measured anxiety symptoms. There was a marginal improvement at the post-intervention time point, with a small effect

Table 3 General characteristics of the cognitive–behavioral interventions to treat adolescent sleeping difficulties

Reference (Country)	*n*	Sample	Framework	Design	Program	Primary outcome
		1. Age, %F 2. Inclusion 3. Exclusion	1. CBT-I 2. Mindfulness	1. Control 2. Assessments	1. Delivery 2. Facilitator 3. Duration	1. Sleep—subj. 2. Sleep—obj. 3. Mental health
Bei et al. (2013) (Australia)	9	1. 13–15, 100 %F	1. Sleep education Sleep hygiene Stimulus control Cognitive restructuring Anxiety specific	1. None	1. Group (n = 9)	1. PSQI
		2. Sleep and anxiety symptoms	2. MBCT	2. Pre-intervention Post-intervention	2. Psychologist	2. Actigraphy
		3. None			3. 6 × 90 min 6 weeks	3. SCAS
Blake et al. (2016) (Australia)	123	1. 12–16, 59 %F	1. Sleep education Sleep hygiene Stimulus control Cognitive restructuring Anxiety specific Savoring and switching	1. Active (Study Skills)	1. Group (n = 4–9)	1. PSQI PDSS
		2. Sleep and anxiety symptoms	2. MBCT	2. Pre-intervention Post-intervention	2. Psychologist	2. Actigraphy
		3. MDD			3. 7 × 90 min 7 weeks	3. SCAS CESD
Bootzin and Stevens (2005) and Britton et al. (2010) (USA)	17	1. 13–19, 43 %F	1. Sleep hygiene Stimulus control Bright light therapy Cognitive restructuring	1. None	1. Group (n = 2–6)	1. Sleep diary ESS
		2. Substance abuse program	2. MBSR	2. Pre-intervention Post-intervention 3 months 12 months	2. Psychologist	2. Actigraphy
		3. None			3. 6 × 90 min 7 weeks	3. GAINS (GMDI)
Clarke et al. (2015) (USA)	41	1. 12–20, 63 %F	1. Sleep education Stimulus control Sleep restriction Cognitive restructuring Savoring CBT-D	1. Active (CBT-D + SH)	1. Individual	1. Sleep diary ISI
		2. Insomnia/unipolar depression diagnoses	2. None	2. Pre-intervention Post-intervention 14 weeks	2. Psychologist	2. Actigraphy
		3. Other disorder			3. 10 sessions 12 weeks	3. CDRS-R

Table 3 continued

Reference (Country)	*n*	Sample		Framework		Design		Program		Primary outcome	
		1.	Age, %F	1.	CBT-I	1.	Control	1.	Delivery	1.	Sleep—subj.
		2.	Inclusion	2.	Mindfulness	2.	Assessments	2.	Facilitator	2.	Sleep—obj.
		3.	Exclusion					3.	Duration	3.	Mental health
De Bruin et al. (2014) (Netherlands)	13	1.	13–19, 77 %F	1.	Sleep education Sleep hygiene Stimulus control Sleep restriction Cognitive restructuring Relaxation therapy	1.	None	1.	Group[a] (n = 6–7)	1.	Sleep diary[b] HSDQ-I CSRQ
		2.	Insomnia symptoms	2.	None	2.	Pre-intervention Post-intervention 2 months	2.	Sleep therapist	2.	Actigraphy[b]
		3.	Other disorder					3.	6 × 90 min 6 weeks	3.	–
De Bruin et al. (2015) (Netherlands)	77	1.	12–19, 70 %F	1.	Sleep education Sleep hygiene Stimulus control Sleep restriction Cognitive restructuring Relaxation therapy	1.	Wait list	1.	Group[a] (n = 6–8)	1.	Sleep diary HSDQ-I CSRQ
		2.	Insomnia diagnosis	2.	None	2.	Pre-intervention Post-intervention 2 months	2.	Sleep therapist	2.	Actigraphy
		3.	Other disorder					3.	6 × 90 min 6 weeks	3.	–
Gradisar et al. (2011a) (Australia)	40	1.	11–18, 47 %F	1.	Sleep education Sleep hygiene Bright light therapy Cognitive restructuring	1.	Wait list	1.	Individual with parent	1.	Sleep diary[b] PDSS
		2.	DSPD	2.	None	2.	Pre-intervention Post-intervention 6 months (treatment arm only)	2.	Psychologist	2.	–
		3.	None					3.	6 × 90 min 8 weeks	3.	MFQ
Schlarb et al. (2011) (Germany)	18	1.	11–16, 56 %F	1.	Sleep education Sleep hygiene Stimulus control Sleep restriction Cognitive restructuring Relaxation therapy Hypnotherapy	1.	None	1.	Groups (n = 4–8)	1.	Sleep diary
		2.	Insomnia diagnosis	2.	None	2.	Pre-intervention Post-intervention	2.	Psychologist	2.	–
		3.	Other disorder					3.	6 × 100 min 6 weeks		YSR

Table 3 continued

Reference (Country)	*n*	Sample		Framework		Design		Program		Primary outcome	
		1.	Age, %F	1.	CBT-I	1.	Control	1.	Delivery	1.	Sleep—subj.
		2.	Inclusion	2.	Mindfulness	2.	Assessments	2.	Facilitator	2.	Sleep—obj.
		3.	Exclusion					3.	Duration	3.	Mental health
Roeser et al. (2016) (Germany)	19	1.	11–16, 68 %F	1.	Sleep education Sleep hygiene Stimulus control Sleep restriction Cognitive restructuring Relaxation therapy Hypnotherapy	1.	None	1.	Group (n = 4–8)	1.	Sleep diary
		2.	Insomnia symptoms	2.	None	2.	Pre-intervention	2.	Psychologist	2.	–
		3.	None				Post-intervention	3.	6 × 100 min 6 weeks	3.	–
							3 months				
							6 months				
							12 months[c]				

DSPD delayed sleep phase disorder, *MBCT* mindfulness-based cognitive therapy, *MBSR* mindfulness-based stress reduction, *CBT-D* cognitive–behavioral therapy for depression, *PSQI* Pittsburgh Sleep Quality Index, *PDSS* Pediatric Daytime Sleepiness Scale, *SCAS* Spence Children's Anxiety Scale, *CESD* Center for Epidemiologic Studies Depression Scale, *ESS* Epworth Sleepiness Scale, *GAINS (GMDI)* Global Appraisal of Individual Needs Interview General Mental Distress Index, *ISI* Insomnia Severity Index, *CDRS-R* Children's Depression Rating Scale-Revised, *CSHI* Clinical Sleep History Interview, *HSDQ-I* Holland Sleep Disorder Questionnaire-Insomnia Scale, *CSRQ* Chronic Sleep Reduction Questionnaire, *MFQ* Short Mood and Feelings Questionnaire, *YSR* Youth Self-Report

[a] Also included Internet condition (not reported)

[b] School day statistics reported

[c] 12 months = primary follow-up

Table 4 Study quality assessments[a]

Reference	Reporting (/11)												External validity (/3)			Internal validity bias (/7)							Internal validity confounding (/6)						Total (/27)
	1	2	3	4	5	6	7	8	9	10	=	11	12	13	=	14	15	16	17	18	19	20	=	21	22	23	24	25	26	=	=
Bei et al. (2013)	1	1	1	1	1	1	1	0	0	0	**7**	0	0	0	**0**	0	0	1	1	1	0	1	**4**	0	0	0	0	0	0	**0**	**11**
Blake et al. (2016)	1	1	1	1	1	1	1	0	1	1	**9**	0	0	0	**0**	0	1	1	1	1	1	1	**6**	1	0	1	0	1	1	**4**	**19**
Bootzin and Stevens (2005)	1	1	1	1	1	1	1	1	1	1	**10**	0	0	0	**0**	0	0	1	1	1	0	1	**4**	0	0	0	0	1	1	**2**	**16**
Clarke et al. (2015)	1	1	1	1	1	1	1	0	0	0	**7**	0	0	0	**0**	0	1	1	1	1	0	1	**5**	1	0	1	1	1	1	**5**	**17**
De Bruin et al. (2014)	1	1	1	1	1	1	1	0	1	1	**9**	0	0	1	**1**	0	0	1	1	1	0	1	**4**	0	0	0	0	1	1	**2**	**16**
De Bruin et al. (2015)	1	1	1	1	1	1	1	0	1	1	**9**	0	0	1	**1**	0	0	1	1	1	1	1	**5**	1	0	1	0	1	1	**4**	**19**
Gradisaret al. (2011a)	1	1	1	1	1	1	1	0	1	1	**9**	0	0	1	**1**	0	0	1	1	1	0	1	**4**	1	1	1	0	1	0	**4**	**18**
Schlarb et al. (2011)	1	1	1	1	0	1	1	0	1	1	**8**	0	0	0	**0**	0	0	1	1	1	0	1	**4**	1	0	0	0	0	1	**2**	**14**
Roeser et al. (2016)	1	1	1	1	1	1	1	0	1	1	**9**	0	0	0	**0**	0	0	1	1	1	0	1	**4**	1	0	0	0	0	0	**1**	**14**
Average											**9**				**0**								**4**							**3**	**16**

[a] Assessed using the Downs and Black (1998) study quality index. All items are scored 0 or 1, except item 5, which is scored 0, 1, or 2

size [$d = -0.33$ (95% CI -0.67, -0.00; $I^2 = 23.93$)]. Only one trial measured anxiety at the follow-up time point, so a meta-analysis was not conducted on this outcome. Here again, there was evidence of moderate–high heterogeneity between trials in outcomes and/or considerable uncertainty in heterogeneity estimates.

Sensitivity Analyses and Publication Bias

None of the sensitivity analyses described earlier showed consistent differences in effect sizes across subjective or objective outcomes (shown in Online Resources 3–6). This suggests that there were no consistent differences in efficacy between studies with high- and low-quality, clinical diagnoses versus self-report symptoms, pure versus comorbid sleep problems, or sleep restriction components. To assess for publication bias, funnel plots were constructed and Egger tests were performed for each outcome variable. No statistically significant results were found, apart from objective TST and depression at the post-intervention time point. However, the publication bias results should be interpreted with caution, as funnel plots require a considerable number of studies, generally at least 30 (Cuijpers 2016; Lau et al. 2006). Additionally, the size of the effects was largely robust to the decision to use an estimated within-participant correlation of $r = 0.5$ between measurements over time. This was evidenced by consistency in findings when sensitivity analyses were conducted with a stronger ($r = 0.7$) and weaker ($r = 0.3$) assumed correlation between measurement time points. The only changes to interpretation were that the effect for objective TST ($p = 0.049$) and anxiety symptoms ($p = 0.01$) became statistically significant from baseline to post-intervention, when using a stronger and weaker correlation, respectively.

Program Acceptability

Five studies reported data on sleep condition program acceptability (Bei et al. 2013; Blake et al. 2016; Bootzin and Stevens 2005; De Bruin et al. 2014; Schlarb et al. 2011). In general, the sleep programs were well accepted. Average rating of the programs and components was above 70%. Sleep education (Bei et al. 2013; Blake et al. 2016; Schlarb et al. 2011), sleep scheduling (Bei et al. 2013; Blake et al. 2016), personalized bedtimes (De Bruin et al. 2014), relaxation (De Bruin et al. 2014), and mindfulness (Blake et al. 2016) were rated as the most helpful components.

Discussion

Despite the fact that (1) cognitive–behavioral therapies are firmly established as frontline treatments for many adult sleep problems/disorders; (2) there is high prevalence, high

Table 5 Risk-of-bias assessments for RCTs

Reference	Sequence generation	Allocation concealment	Blinding of outcome assessors	Incomplete outcome data	Selective outcome reporting	Other issues	Summary assessment
Blake et al. (2016)	Low	Low	Low	Low	Low	Low	Low
Clarke et al. (2015)	Low	Low	Low	Low	Unclear	Low	Low
De Bruin et al. (2015)	Low	Low	High	Low	Unclear	Low	High
Gradisar et al. (2011a)	Low	Low	High	High	Unclear	Low	High
Summary assessment	Low	Low	High	Low	Unclear	Low	High

Assessed using the Cochrane risk-of-bias instrument

chronicity, and serious consequences associated with adolescent sleep problems/disorders; and (3) there is a growing body of research on adolescent cognitive–behavioral sleep interventions, this was the first systematic review and meta-analysis to provide a synthesis of this emerging field. From the data presented here, it is not possible to make firm conclusions about the efficacy of adolescent cognitive–behavioral sleep interventions. There was evidence of considerable heterogeneity between the trials, which might have been due to the small number of trials, the small number of participants per trial (Cuijpers 2016), and/or the differences in the approaches to delivering the interventions, including variation in participants, treatment components, and comparators. Moreover, the methodological quality of the trials varied, and there was high risk of bias across the RCTs.

The within sleep condition results were promising. There was evidence that adolescent cognitive–behavioral sleep interventions produce meaningful improvements in subjectively and objectively measured TST, SOL, and SE. As with adults, improvements tended to be stronger for wakefulness-in-bed variables compared to sleep duration variables and subjective sleep variables compared to objective sleep variables. Improvements also tended to persist over time, particularly for wakefulness-in-bed variables. Furthermore, the interventions were associated with meaningful improvements in perceived sleep quality and functional outcomes (daytime sleepiness, depression, and anxiety). The programs were also well-accepted, particularly the stimulus control, relaxation, and mindfulness components.

As described by Koffel et al. (2015), the smaller improvements in TST compared with wakefulness-in-bed variables were most likely related to the techniques used in cognitive–behavioral sleep interventions. The goals of stimulus control and sleep restriction are to initially limit sleep opportunities to increase the sleep drive. As a result, one would expect more immediate improvements in SOL and SE, and delayed improvements in TST, as participants gradually increase their opportunity for sleep. The findings are also consistent with large epidemiologic studies showing that in adolescents, evening chronotype and increased daytime sleepiness are associated with deficits in self-regulation, while short sleep duration is not (Anderson et al. 2009; Owens et al. 2016; Perez-Lloret et al. 2013). These findings suggest that circadian misalignment, wakefulness in bed, and daytime sleepiness, more than sleep duration, may increase the risk of mental health problems during adolescence.

In general, the effects sizes were smaller in magnitude than those reported in meta-analyses of CBT-I among adults with chronic insomnia (Koffel et al. 2015; Trauer et al. 2015; van Straten et al. 2017). However, the baseline deficits were also smaller than those reported in adult trials. For example, at baseline, the mean subjective TST, SOL, and SE in the included trials were 448.25 min, 46.05 min, and 83.72%, respectively, whereas they were 334.1 min, 57.6 min, and 71.8%, respectively, in the meta-analysis by Trauer et al. (2015). Other studies have found that patients with insomnia diverge from healthy control participants in subjectively assessed TST, SOL, and SE by around 95 min, 23 min, and 16%, respectively (Buysse et al. 2007), and Scholle et al. (2011) found that normal polysomnography assessed TST, SOL, and SE among adolescents are around 473 min, 24 min, and 89%, respectively. Therefore, improvements in subjective TST, SOL, and SE of 29.47 min, 21.44 min, and 5.34%, respectively, and objective TST, SOL, and SE of 9.19 min, 16.15 min, and 2.82%, respectively, represent major increases in these variables, especially SOL.

These results are particularly promising for three reasons. First, there are numerous barriers to sleep intervention in adolescence, including powerful physiological and social/cultural factors that combine to delay sleep onset and restrict sleep opportunities (Adam et al. 2007; Carskadon et al. 2004; Feinberg and Campbell 2010; Hale and Guan 2015; Maume 2013; Short et al. 2011). Second, school-based sleep education programs have been shown to be largely ineffective for improving objective and subjective sleep among adolescents (Blunden et al. 2012; Blunden and Rigney 2015; Gruber 2016; Kira et al. 2014; Wing et al. 2015). Third, wakefulness in bed during adolescence is a

consistent indicator of current and future internalizing problems (Alfano et al. 2013; Forbes et al. 2009; Lovato and Gradisar 2014; McMakin and Alfano 2015).

Suggestions for Further Research

Trials of adolescent cognitive–behavioral sleep interventions have been limited in several ways. First, more than half of the trials were uncontrolled feasibility trials evaluating a small number of participants (Bei et al. 2013; Bootzin and Stevens 2005; De Bruin et al. 2014; Roeser et al. 2016; Schlarb et al. 2011). Second, two of the RCTs used wait list controls (De Bruin et al. 2015; Gradisar et al. 2011a). It is generally accepted that for a trial to achieve a high standard of evidence, it must include adequate statistical power to detect significant effects and an active control condition or psychological placebo (Furukawa et al. 2014). The two RCTs that used active control conditions (Blake et al. 2016; Clarke et al. 2015) found smaller effects. Future adolescent cognitive–behavioral sleep intervention trials should recruit larger sample sizes and use high-quality active control conditions (Morin et al. 2006; Riemann and Perlis 2009; Vitiello et al. 2013).

Third, two RCTs did not describe blinding of outcome assessors (De Bruin et al. 2015; Gradisar et al. 2011a). Empirical evidence shows that lack of reporting of blinding is associated with biased estimates of treatment effect and more positive outcomes (Boutron et al. 2008; Higgins and Green 2011; Moher et al. 2012). Blinding is an especially important issue for non-pharmacologic trials, where outcome assessors can have an important influence of the treatment effect (Boutron et al. 2008). Studies are also needed that measure treatment expectancy and credibility. Beliefs and expectations about behavioral and psychological treatments play an important role in determining experiences and outcomes of treatment (Devilly and Borkovec 2000).

Fourth, most of the studies did not include trial protocol papers. Reporting and publishing trial protocol papers prior to the commencement of interventions is an important step in increasing the transparency of research and the reliability of outcome papers (Cybulski et al. 2016). Furthermore, although all of the RCTs were registered as clinical trials, only one was registered prospectively (Clarke et al. 2015). Retrospective registration makes it impossible to determine whether aims and methods were determined prior to data analyses being conducted. Additionally, while most of the studies included in the meta-analysis reported TST, not all reported SOL, SE, and WASO, and few studies controlled for familywise false rejection error rates. Although there is no consensus about optimal outcomes when treating sleep problems, most adult CBT-I trials include TST, SOL, SE, and WASO as dependent variables. Morin (2003) has described recommendations for measuring outcomes in insomnia trials.

Fifth, most of the studies failed to measure compliance with the intervention. When evaluating new interventions, if significant results are found, but treatment fidelity is not monitored and optimized, the outcome could be due to an effective treatment or to unknown factors unintentionally added or omitted from the treatment (Bellg et al. 2004). On the other hand, if nonsignificant results are found, the outcome could be due to an ineffective treatment or a lack of treatment fidelity (Bellg et al. 2004). Improving treatment fidelity can also increase statistical power, by reducing random and unintended variability (i.e., drift in interventionist adherence). Furthermore, translating clinical interventions from research to practice can be improved when treatments are well defined and there are guidelines for implementing them (Bellg et al. 2004).

Sixth, all of the studies were limited by low generalizability. For example, the RCT by De Bruin et al. (2015) included adolescents diagnosed with insomnia but excluded adolescents diagnosed with other psychiatric disorders. Between 40 and 92% of all cases of insomnia occur in the context of psychiatric disorders (Breslau et al. 1996; Kessler et al. 2012). The RCT by Gradisar et al. (2011a) evaluated adolescents with DSPD, which is a low prevalence disorder (between 1 and 7%; Johnson et al. 2006; Ohayon et al. 2000; Pelayo et al. 1988). The RCTs by Blake et al. (2016) and Clarke et al. (2015) evaluated adolescents with specific comorbidities. Trials among adolescents experiencing subclinical and generalized levels of sleep and mental health problems are needed (Chase and Pincus 2011; Johnson et al. 2000; Moore et al. 2009; Warner et al. 2008).

Seventh, a diverse range of methods has been used to assess sleep. Adolescent sleep interventions have tended to use subjective sleep measures more consistently than objective sleep measures. Most studies have used sleep diaries to measure subjective sleep, but other studies have relied on retrospective questionnaires (Bei et al. 2013; Blake et al. 2016). Sleep diaries are considered the gold standard of subjective self-report (Buysse et al. 2006; Morin and Espie 2003) but are vulnerable to poor compliance (missing data and entry errors), and all subjective measures of sleep are subject to the limits of introspection and memory bias. In general, there have been discrepancies in results between studies comparing subjective and objective sleep. This may be due to self-report bias, the limits of introspection, and poor compliance, but also to the limits of currently available objective measures of sleep. While wrist actigraphy shows high sensitivity (to detect sleep), it demonstrates lower specificity (to detect wakefulness in bed; Meltzer et al. 2012). Therefore, differences between subjective and objective SE may be due to

Table 6 Summary of findings for within sleep condition meta-analyses

Variable			No. of studies	Effect size			I^2 (CI)	Eggers test	
				Estimate (CI)	SE	*p*		Estimate	*p*
Pre–post-intervention	Subjective sleep	TST (min)	8	29.47 (17.18, 41.75)	6.27	<0.001	26.62 (0.00, 78.39)	−0.25	0.80
		SOL (min)	9	−21.44 (−30.78, −12.11)	4.76	<0.001	85.94 (65.06, 96.51)	−1.36	0.17
		SE (%)	7	5.34 (2.65, 8.04)	1.38	0.00	69.60 (27.10, 95.37)	0.81	0.42
		WASO	6	−0.59 (−0.82, −0.36)	0.11	<0.001	28.64 (0.00, 89.48)	−1.96	0.05
	Objective sleep	TST (min)	6	9.19 (−0.65, 19.04)	5.02	0.07	10.40 (0.00, 53.73)	2.28	0.02
		SOL (min)	4	−16.15 (−26.13, −6.17)	5.09	0.00	72.87 (20.11, 96.41)	−1.30	0.19
		SE (%)	5	2.82 (0.58, 5.07)	1.15	0.01	66.06 (13.64, 94.60)	0.48	0.63
		WASO	5	0.05 (−0.18, 0.27)	0.11	0.68	37.36 (0.00, 88.71)	−0.16	0.87
	Secondary endpoints	Global sleep	5	−0.92 (−1.52, −0.32)	0.31	0.00	86.89 (56.39, 98.80)	−0.99	0.32
		Sleepiness	4	−0.63 (−1.07, −0.18)	0.22	0.00	79.34 (16.97, 98.86)	−1.01	0.31
		Anxiety	2	−0.33 (−0.67, 0.00)	0.17	0.05	23.93 (0.00, 99.92)	NA	NA
		Depression	3	−1.22 (−2.47, 0.01)	0.63	0.05	94.56 (78.04, 99.87)	−5.29	<0.001
Pre-follow-up	Subjective sleep	TST (min)	4	37.30 (20.97, 53.62)	8.33	<0.001	0.00 (0.00, 25.89)	−0.41	0.68
		SOL (min)	5	−30.25 (−39.12, −21.39)	4.52	<0.001	46.13 (0.00, 89.54)	−0.70	0.48
		SE (%)	4	5.53 (−2.93, 14.00)	4.32	0.20	92.65 (76.29, 99.21)	0.46	0.65
		WASO	5	−0.52 (−0.71, −0.32)	0.10	<0.001	0.00 (0.00, 87.26)	−1.12	0.26
	Objective sleep	TST (min)	3	15.97 (−8.93, 40.87)	12.7	0.21	63.86 (0.00, 94.64)	−0.15	0.88
		SOL (min)	2	4.82 (−0.22, 9.88)	2.57	0.06	0.00 (0.00, 85.25)	NA	NA
		SE (%)	3	4.87 (1.68, 8.05)	1.62	0.00	65.91 (0.00, 97.39)	−0.26	0.79
		WASO	3	−0.05 (−0.51, 0.42)	0.23	0.84	70.53 (0.00, 99.31)	−0.14	0.89
	Secondary endpoints	Global sleep	3	−1.54 (−2.48, −0.60)	0.48	0.00	85.06 (38.76, 99.65)	−0.79	0.43
		Sleepiness	3	−1.18 (−1.81, −0.54)	0.32	0.00	75.02 (0.00, 99.46)	−0.40	0.69
		Anxiety	1	NA	NA	NA	NA	NA	NA
		Depression	2	−2.21 (−4.47, 0.06)	1.16	0.05	92.62 (62.90, 99.80)	NA	NA

A meta-analysis was not conducted if the total number of associations available <2. Effect size estimates = Cohen's D unless otherwise noted (i.e., TST & SOL = minutes, SE = %)

NA not applicable, *N* total number, *CI* confidence interval, I^2 indicator of heterogeneity in percentages, *TST* total sleep time, *SOL* sleep onset latency, *SE* sleep efficiency, *WASO* wake after sleep onset

actigraphy algorithms scoring epochs as sleep, whereas the individual perceives it as wake. Removal of the actigraph, technical failures, and other measurement errors, can also distort sleep behaviors. Human scoring of actigraphy, using collateral information (e.g., light and event markers), may improve its accuracy. Web or mobile platforms, with built-in reminders and error checking, may improve sleep diary accuracy. However, no adolescent sleep intervention studies have measured sleep with polysomnography, which is widely considered to be the gold standard of objective sleep measurement. Overall, a combination of subjective and objective assessments should be used to assess outcomes in trials of adolescent cognitive–behavioral sleep interventions.

Eighth, cognitive–behavioral sleep intervention studies conducted among adolescents have not comprehensively assessed functional outcomes, such as anxiety and depression. Studies that have assessed mental health have tended to use generic measures of emotional distress (Bootzin and Stevens 2005; Schlarb et al. 2011) or have not examined heterogeneous anxiety and depressive symptoms (Blake et al. 2016; Clarke et al. 2015; Gradisar et al. 2011a), limiting specificity of findings for any symptom cluster. Morin (2003) has suggested that it is critical to document efficacy beyond the simple reduction of insomnia symptoms in CBT-I trials and to incorporate additional indicators of efficacy, such as daytime functioning, fatigue, mood, anxiety, quality of life, school attendance, academic performance, and physical health.

Ninth, most of the studies included in the meta-analysis did not report whether interventions and assessments were conducted during school term or school holiday periods. If pre-intervention assessments were conducted during school term and post-intervention assessments during school

holiday periods, effect sizes could have been overestimated for TST and underestimated for wakefulness in bed. There is strong evidence that adolescent sleep is characterized by sleep restriction and high sleep drive during the school week and sleep saturation and lower sleep drive during vacation periods (Bei et al. 2014). Studies are also needed that differentiate between outcomes on school days and weekend/vacations. Adolescent sleep is strongly affected by school schedules (Bei et al. 2014), and outcomes of interventions may differ across the week.

Tenth, adolescent cognitive–behavioral sleep intervention studies have rarely considered intra-individual variability of sleep. Implementing different techniques to reduce sleep variability, such as establishing regular sleep–wake times, sleep hygiene instruction, stimulus control therapy, and cognitive–behavioral therapy, is a common goal of sleep interventions, and regularity and variability of sleep behaviors have been recommended as measures of treatment adherence and non-adherence, respectively, in behavioral insomnia treatments (Buysse et al. 2010). A systematic review of daily sleep variability among adults found that higher variability in sleep is associated with sleep and mental health problems (including insomnia, depression, anxiety), and that CBT-I reduces such variability (Bei et al. 2016). With respect to adolescents, a number of studies have found that variability in sleep schedules is associated with poor functional outcomes among adolescents, including anxiety, depression, daytime sleepiness, behavioral problems, and poor school performance (Biggs et al. 2011; Fuligni and Hardway 2006; Pasch et al. 2010; Wolfson and Carskadon 1998), as well as altered brain development (Telzer et al. 2015). In sum, these findings suggest that variability in sleep patterns should be considered as an additional dimension of sleep in trials of adolescent cognitive–behavioral sleep interventions.

Eleventh, studies are needed that examine mechanisms of change during adolescent cognitive–behavioral sleep interventions (Morin et al. 2006; Schwartz and Carney 2012; Vitiello et al. 2013). In order for such an intervention approach to reach the highest standards of evidence, studies must not only demonstrate that it is effective, but also show that the intervention works via its purportedly active mechanism (Garratt et al. 2007; Insel 2015). Treatment mechanism research can lead to a number of positive outcomes, including clarifying the nature and etiology of disorders, elucidating the processes leading to therapeutic change, identifying active treatment components, and refining current treatment protocols (Kazdin 2007; Kraemer et al. 2002). A number of mediators may account for therapeutic change in adolescent cognitive–behavioral sleep interventions, including improvements in sleep–wake variability, pre-sleep hyperarousal, and maladaptive beliefs and attitudes about sleep (Schwartz and Carney 2012). Studies are also needed that examine moderators of change (e.g., age and gender), predictors of treatment adherence (e.g., baseline symptoms (short sleep duration, anxiety, depression), self-efficacy, and attitude to treatment; Matthews et al. 2013), dose–response relationships (i.e., high- vs. low-intensity treatments), and temporal relations between variables (Morin et al. 2014).

Twelfth, studies are needed that examine specificity of effects of treatment components. Across studies included in the meta-analysis, adolescents rated behavioral treatments as more helpful than cognitive treatments, consistent with results from the adult literature—a recent meta-analysis found that relaxation treatments are particularly effective for reducing SOL in adults (van Straten et al. 2017). However, no adolescent studies have compared the efficacy of different treatment components using dismantling designs (Harvey et al. 2014). In particular, studies are needed that examine the therapeutic efficacy and specificity of different treatment components for adolescents with different symptom profiles. A personalized approach to adolescent sleep interventions (i.e., tailoring the interventions based on biopsychosocial symptom profiles) may improve their therapeutic efficacy and cost-effectiveness. On the other hand, it is also possible that transdiagnostic approaches may solve the "too many empirically supported treatments problem" that can impede the dissemination and uptake of treatments (Weisz et al. 2014, p. 68). Because comorbidity is the norm (Kessler et al. 2005, 2007), clinicians are often faced with the dilemma of which disorder/disorders to prioritize for treatment. Treating transdiagnostic processes may reduce the burden on clinicians, who are often required to learn multiple disorder-focused protocols (e.g., CBT-I, mindfulness-based sleep interventions, bright light therapy, and chronotherapy), with shared theoretical underpinnings (Harvey 2004, 2015; Mansell et al. 2009). Harvey (2015) recently proposed a transdiagnostic intervention for youth sleep and circadian problems (TranS-C-Youth), which is informed by basic sleep/circadian principles, and aims to address the broad range of sleep disturbances that adolescent's experience, including pre-sleep arousal, maladaptive sleep-related cognitions, prolonged SOL, poor SE, delayed bedtimes, inadequate opportunity to sleep, irregular sleep–wake schedules, difficulty waking up/getting out of bed in the morning, and daytime sleepiness. However, RCTs evaluating TranS-C-Youth are yet to be published.

Thirteenth, many of the studies included in the meta-analysis did not include follow-ups (Bei et al. 2013; Blake et al. 2016; Schlarb et al. 2011) or used short follow-ups (i.e., up to 14 weeks; Clarke et al. 2015; De Bruin et al. 2014, 2015). Studies are needed that investigate durability and progression of change over time. Finally, studies are

needed that evaluate the broad-based effectiveness and acceptance of adolescent cognitive–behavioral sleep interventions in the community (e.g., primary care, mental health, adolescent health, sleep medicine, and schools), using different modes of delivery (e.g., non-specialist practitioners, group settings, individual settings, Internet, and other e-health modes of delivery). As described by Gradisar and Richardson (2015), "the list of research questions is extensive and highlights that our field has only begun to evaluate treatment for adolescent sleep disturbance, which may or may not present and respond to treatment like adult insomnia" (p. 1841).

Conclusion

Our meta-analysis provides preliminary evidence that adolescent cognitive–behavioral sleep interventions are an efficacious treatment for sleep and mental health problems, producing clinically meaningful responses within active treatment conditions. Their efficacy seems to be maintained over time and result in significant alleviation of sleep problems and improvement in functional outcomes. However, it is not possible to make definitive conclusions about the efficacy of adolescent cognitive–behavioral sleep interventions from the data presented here. Further large-scale controlled treatment outcome studies are needed to confirm these findings.

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Compliance with Ethical Standards

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

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