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CLINICAL REVIEW

The impact of cognitive behavioural therapy for insomnia on objective sleep parameters: A meta-analysis and systematic review

Laura J. Mitchell [a], Lampros Bisdounis [a, b], Andrea Ballesio [c], Ximena Omlin [a], Simon D. Kyle [a, *]

[a] Sleep and Circadian Neuroscience Institute, Nuffield Department of Clinical Neurosciences, University of Oxford, Oxford, UK
[b] Department of Clinical Psychology, University of Amsterdam, Amsterdam, the Netherlands
[c] Department of Psychology, Sapienza University of Rome, Italy

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SUMMARY

It is well-established that cognitive behavioural therapy for insomnia (CBT-I) improves self-reported sleep disturbance, however the impact on objective sleep is less clear. This meta-analysis aimed to quantify the impact of multi-component CBT-I on objective measures of sleep, indexed via polysomnography (PSG) and actigraphy. Fifteen studies met inclusion criteria. Following appraisal for risk of bias, extracted data were meta-analysed using random-effects models. The quality of the literature was generally high, although reporting of methodological detail varied markedly between studies. Meta-analyses found no evidence that CBT-I reliably improves PSG-defined sleep parameters. Actigraphy evidence was more mixed; with a small effect for reduction in sleep onset latency (Hedge's $g = -0.28$ [95% confidence interval (CI) −0.51 to −0.05], $p = 0.018$) and a moderate effect for reduction in total sleep time (TST) (Hedge's $g = -0.51$ [95% CI −0.75 to −0.26], $p < 0.001$). In contrast, and consistent with recent meta-analyses, CBT-I was associated with robust improvements in diary measures of sleep initiation and maintenance (Hedge's g range = 0.50 to 0.79) but not TST. While the literature is small and still developing, the sleep benefits of CBT-I are more clearly expressed in the subjective versus objective domain.

Introduction

Insomnia is characterised by perceived difficulty initiating or maintaining sleep, which in turn has a negative influence on daytime functioning [1]. Reports on the prevalence of insomnia are variable [2], with one estimate suggesting that one third of the adult population displays at least one symptom of insomnia, while 6% meet diagnostic criteria [3]. It is estimated that the prevalence of insomnia is steadily increasing [4]. Chronic insomnia is associated with substantial impairment to quality of life, loss of functioning and increased health risk [5,6]. It can also be a risk factor or trigger for mental health difficulties such as depression [7], psychotic experiences [8,9] and episodes of mania or depression in bipolar disorder [10]. Treatment of insomnia is important, both as a condition in its own right [11] and as a comorbid condition in those with psychiatric or medical diagnoses [12].

The most common clinical response to insomnia is the prescription of hypnotic drugs [13], despite cognitive behavioural therapy for insomnia (CBT-I) being recommended as the first-line treatment in clinical guidelines [14–16]. CBT-I is considered to have well-established efficacy [17–19] and its effects may be more long-lasting than medication [20]. A number of previous reviews demonstrate the efficacy of CBT-I when measured by sleep diaries [11,19,21,22], however relatively few studies collect or report on objective sleep outcomes, measured by polysomnography (PSG) and actigraphy. As such, the impact of CBT-I on objective sleep has been less thoroughly investigated [18]. This is in contrast to literature on the pharmacological management of insomnia [23–25] or treatments for other common sleep disorders, like obstructive sleep apnoea, where PSG measurement is routine in intervention trials [26].

While contemporary diagnostic criteria [1,27] clearly consider insomnia to be a disorder based on the perception of sleep, research guidelines emphasise the assessment of *both* objective and subjective sleep [28]. Consistent with this, meta-analyses show that

* Corresponding author. Sleep and Circadian Neuroscience Institute (SCNi), Nuffield Department of Clinical Neurosciences, Sir William Dunn School of Pathology, University of Oxford, South Parks Road, Oxford, OX1 3RE, United Kingdom.
E-mail address: simon.kyle@ndcn.ox.ac.uk (S.D. Kyle).

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List of abbreviations

ACT	Actigraphy
BBTI	brief Behavioural Treatment for Insomnia
CBT-I	cognitive Behavioural Therapy for Insomnia
CC	control condition
CI	confidence interval
CMI	comorbid insomnia
CR	cognitive restructuring
CT	cognitive therapy
DSM(IV-TR)	Diagnostic and Statistical Manual of Mental Disorders (IV-Text Revision)
f	female
f/up	follow up
IC	information control
ICBT-I	internet cognitive behavioural therapy for insomnia
ICSD	International Classification of Sleep Disorders
ISR	intensive sleep retraining
MCI	multi-component intervention
Min	minutes
Mo	month(s)
PE	psychoeducation
PI	primary insomnia
PSG	polysomnography
PT	placebo therapy (psychological)
RCTs	randomised controlled trials
RDC	research diagnostic criteria
REL	relaxation
RP	relapse prevention
RT	relaxation therapy
SC	sleep compression
SCT	stimulus control therapy
SD	standard deviation
SE	sleep efficiency
SH	sleep hygiene
SOL	sleep onset latency
SRT	sleep restriction therapy
SS	sleep seminar
TCC	Tai Chi Chih
TST	total sleep time
TWT	total wake time at night
WASO	wake after sleep onset
Wk(s)	week(s)
Yrs	year(s)

insomnia is characterised by objective sleep impairment relative to normal sleeping controls (e.g., in PSG-defined SOL, SE, and TST) [29]. Assessment of objective sleep is also important because insomnia with objective short sleep may be particularly linked to poorer health outcomes and impaired cognitive performance [30], as well as blunted response to CBT-I [31].

Assuming that objective sleep disturbance is a reliable feature of insomnia disorder, a logical extension of the above literature is that 1) if CBT-I truly "treats" insomnia we should observe amelioration of objective sleep impairment; and 2) CBT-I is likely to confer greater health benefits if it can improve objective indices of sleep (particularly for those who exhibit pronounced baseline sleep deficits). One previous meta-analysis, published in 2011, reported on changes in objectively-defined sleep (PSG and/or actigraphy). This review observed medium effect sizes at post-treatment for wake-time after sleep onset and sleep efficiency, with no evidence of improvement in objective total sleep time [18]. However, the authors combined actigraphy and PSG when computing effect size estimates - limiting precision - and did not perform quality appraisal of studies. Moreover, since 2011, several published studies have included objective measures of sleep [32–38]. Clearly the field requires a contemporary assessment of the effects of CBT-I on objective sleep to advance understanding and guide both future studies and treatment refinement. We set out to undertake a systematic search of the insomnia treatment literature with the aim of quantitatively and qualitatively appraising randomised controlled trials (RCTs) of multi-component CBT-I that report on objective measures of night-time sleep.

Methods

Search strategy

This systematic review and meta-analysis was conducted in line with PRISMA guidelines [39]. Published studies were identified via medical and allied health online databases EMBASE, PsycINFO, MEDLINE, CINAHL and AMED. Databases were searched from inception to 13th March 2018 using terms for cognitive behavioural therapy, insomnia and objective measures of sleep (PSG and actigraphy). The following search terms were used with the OVID database platform: "cognitive behav* therap*" or "cognitive behav* intervention" or "cognitive therap*" or "behav* therap*" or "behav* modification" or "CBT" or "CBT-I" or "ICBT" or "sleep hygiene" or "stimulus control" or "relaxation" or "sleep restriction" or "psychotherap*" AND "actigraph*" or "actimetry" or "ACT" or "polysomnograph*" or "PSG" or "objective measures" or "objective sleep" or "sleep parameters" AND "insomnia" or "chronic insomnia" or "sleeplessness" or "sleep disorder*" or "sleep initiation" or "sleep maintenance" or "poor sleep" or "sleep problem" or "sleep disturbance".

In addition, bibliographies from previous reviews and retrieved articles were hand searched [11]. Grey literature was not included. All titles and abstracts were screened by the first author to identify studies providing a CBT-I intervention to adults with insomnia disorder. If the abstract indicated that the study would potentially meet the inclusion/exclusion criteria outlined, the full-text version was obtained for data extraction. Where there was uncertainty, an inclusion decision was reached by author consensus.

Study selection

Included papers were peer reviewed and written in English. Research trials were included in the review if they met the following criteria: 1) a randomised controlled trial (RCT) design, 2) the population sampled were adults (≥18) who had insomnia disorder, as diagnosed using either research [40] or clinical diagnostic criteria (the diagnostic and statistical manual of mental disorders (DSM) [1] or the international classification of sleep disorders (ICSD) [27] criteria) 3) the intervention was CBT-I, defined as multimodal therapy delivered on at least two occasions and incorporating at least two of the five most widely accepted components of CBT-I: cognitive therapy, stimulus control, sleep restriction, sleep hygiene (psychoeducation), and relaxation therapy. All delivery modalities (e.g., face-to-face, web-based) were eligible for inclusion and there was no requirement for the treatment to combine cognitive and behavioural components, 4) outcomes included mean differences in sleep parameters measured using validated objective measures of sleep (PSG or

acitgraphy), and 5) studies had at least one passive control arm (i.e., wait list control, sleep hygiene education, or psychological placebo condition).

Studies were excluded if co-morbid psychiatric, medical or sleep disorders besides insomnia were listed as the *main focus* of the study, or as a participant inclusion criterion. However, studies were not excluded if the frequency of comorbid conditions in the sample were reported. Patients with insomnia seen in clinical practice are likely to have a number of co-morbid conditions, therefore it was not considered pragmatic to exclude studies based on this parameter. Studies were also excluded if CBT-I was combined with alternative treatment for sleep disorders (e.g., CBT-I combined with medication) and when the control condition was focussed on pharmacologic intervention (i.e., medication withdrawal/placebo).

Data extraction

Details of the included studies were recorded by the first author (LM) within a standardised data-extraction form. The form was constructed to extract the following study characteristics: 1) citation of the publication, 2) geographic location, 3) total number of participants and the number of female participants, 4) mean age (and standard deviation when applicable), 5) social and cultural characteristics of the population, 6) method of recruitment, 7) diagnostic criteria and mean duration of insomnia, 8) medication status, 9) co-morbidity, 10) inclusion and exclusion criteria for participation - including screening for objective sleep problems -, 11) components, format -individual or group, face to face or phone-frequency and duration of the treatment intervention and who delivered it, 12) type, frequency and duration of the control condition and who delivered it, 13) objective and subjective sleep measurements used, 14) the duration of PSG and actigraphy when applicable and 15) the format and timing of the follow-up.

Findings regarding the effect of CBT-I on objective measures at post-treatment were extracted and coded for data analysis. Although some studies reported differences within groups before and after treatment, this meta-analysis only considered differences between treatment and control groups (i.e. between-group differences) as robust evidence to assess the *effect* of CBT-I on objective sleep. Additionally, the meta-analysis was interested in the main, super-ordinate features of objective sleep. As such, we extracted outcomes regarding the following commonly reported sleep parameters: sleep onset latency (SOL), wake-time after sleep onset (WASO), sleep efficiency (SE) and total sleep time (TST). As a secondary aim, and to permit comparison with objective dependent variables, we also extracted and calculated effects sizes for diary-reported variables (SOL, WASO, SE, TST) where available. Note, reporting of sleep diary variables was not considered a criterion for study inclusion.

Quality assessment

This meta-analysis considered whether the conclusions made were reliable and valid based on the methodological quality of the summarised research. Therefore, all included studies were independently appraised by the first and second authors using the Cochrane Collaboration's tool for assessing risk of bias [41]. This involves appraising bias in selection, performance, detection, attrition and reporting domains. In each domain, studies were given a rating of low, high or unclear risk. In the case of a discrepancy in appraisal of risk of bias between the two authors', a consensus was reached after consultation with the remaining authors.

Data synthesis and analysis

All analyses were conducted in R, version 3.5.1 [42], using the *tidyverse* [43] and *metafor* [44] packages. Effect sizes and 95% confidence intervals (CI) were calculated for between-group comparisons at post-treatment. The effect size statistic selected for this meta-analysis was Hedge's g [45], which is a measure of standardised mean difference. Hedge's g is similar to Cohen's d in the sense that both metrics operate on the assumption of equal population variance. However, Cohen's d can be positively biased especially in the context of smaller sample sizes [45]. Hedge's g reduces this upward bias by weighting each group's standard deviation (SD) by its sample size and therefore using pooled weighted standard deviations (SDs). This makes Hedge's g more appropriate for studies including smaller sample sizes [46], a common characteristic for studies in clinical psychology. In this meta-analysis Hedge's g was the quotient of the difference between the mean of the experimental group against the mean of the control group, divided by the pooled weight standard deviation incorporating Bessel's correction:

$$g = \frac{\mu_{exp} - \mu_{ctrl}}{s}$$

where μ_{exp} and μ_{ctrl} denote the mean values of the experimental and control group respectively, whilst s denotes the pooled weighted standard deviation, calculated as follows:

$$s = \frac{\sqrt{(n_{exp} - 1)SD_{exp}^2 + (n_{ctrl} - 1)SD_{ctrl}^2}}{n_{exp} + n_{ctrl} - 2}$$

where n_{exp} and n_{ctrl} indicate the number of participants and SD_{exp} and SD_{ctrl} the standard deviation points for the experimental and control group respectively. Effect sizes of 0–0.32 are interpreted as small, effect sizes of 0.33–0.55 are moderate and effect sizes of 0.56–1.2 are assumed to be large [47]. For the effect size calculations, we used the available means and SDs as published in the included papers. Some studies reported other measures of dispersion, such as CIs [36] and standard errors [33,34,37,38,48,49]. These metrics were transformed to SDs using the following formulae:

$$SD = ES * \sqrt{n}$$

$$SD = \frac{(\mu - CI.lb) * \sqrt{n}}{1.96}$$

where *CI.lb* denotes the lower bound of the confidence intervals.

Two papers [37,50] reported on two controlled post-treatment analyses. One of them [50] divided participants according to insomnia profile (i.e., primary or co-morbid insomnia) and assigned them to either the treatment or control arm. The other study [37] classified participants across both trial arms into long (≥6 hrs) and short (<6 h) sleepers based on one night of baseline PSG. These four comparisons were independently coded in the dataset and entered in the main analysis.

For each measurement and outcome, heterogeneity analyses were conducted to test for the implementation of a fixed-effects model. Cochran's Q-statistic was utilised for the heterogeneity tests. Cochran's Q is calculated as the weighted sum of squared differences between individual study effects and the pooled effect across studies, with the weights being those used in the pooling method. According to the associated Q-statistic, variance in a considerable amount of effect size estimates was larger than what would be predicted based on subject-level sampling variability. Therefore, a fixed-effects model was implemented in the sensitivity

analysis for the appropriate outcomes and random effects modelling was utilised for the main analysis. Furthermore, to account for small sample sizes and achieve nominal coverage probabilities, heterogeneity was also assessed based on I^2 and the accompanying CIs as calculated using the Q-profile method [51]. I^2 indicates the percentage of total variability in effect sizes that reflects heterogeneity among the true effects and not sampling error. An I^2 value of 0 signifies 0% heterogeneity; the greater the I^2 value the greater the observed heterogeneity.

Sensitivity analysis was performed to assess the effect of model choice (i.e., fixed vs random modelling) on the effect size estimations. This was achieved by implementing fixed effects models to the applicable outcomes and measurements and comparing the results with those provided from random effects models. Additionally, sensitivity analysis was conducted to investigate the effect of double comparisons within a single study [37,50]. This was achieved by re-calculating the main analysis each time including only one comparison per outcome and measurement from each study.

Publication bias was assessed via visual inspection of the funnel plots and Egger's weighted regression test for funnel plot asymmetry [52]. Wherever publication bias was detected, the Duval and Tweedie trim-and-fill method [53] was implemented to produce a model after accounting for any publication bias. This method also yields a numerical estimate of the missing studies due to the congregation of the most extreme results on one side of the funnel plots [43].

Results

After removing duplicate records (n = 628), titles and abstracts of 1701 records were screened for inclusion. This procedure resulted in the exclusion of 1638 references and a full-text inspection of 63 citations followed. A total of 15 published studies met the inclusion criteria for this meta-analysis (see Fig. 1).

Study characteristics

Table 1 presents study characteristics for all included studies. The earliest study was published in 1993 and the latest in 2017, with one study published before 2000 (6.67%), five studies published between 2000 and 2010 (33.33%), and nine studies published from 2011 onwards (60%). Three studies solely analysed PSG outcomes, 11 studies solely analysed actigraphy outcomes and one study [49] analysed both PSG and actigraphy outcomes. Out of the 15 studies, eleven were conducted in the USA, one in the UK and three in Australia. These studies recruited a total of 1541 participants, with sample sizes ranging between 24 and 201. Studies recruited adults and older adults from the community (n = 10), patients attending a medical practice (n = 1), military veterans (n = 2), active army personnel (n = 1) and college students (n = 1) with a mean age across studies of 57 yrs. A total of 47% of the sample was female; the relatively low percentage of females can be explained by the predominantly male veteran and army population in three studies.

In all studies, insomnia criteria (DSM or ICSD) was ascertained via clinical interview and/or sleep diaries. Ten studies excluded sleep disorders other than insomnia using clinical interview and PSG screening [32,34,35,37,48–50,54,58], three studies used other in-home monitoring devices [36,55,56], whereas two studies used clinical interview only [38,57]. No studies used objective sleep criteria for study entry, although Edinger and colleagues excluded individuals who met clinical and PSG criteria for sleep state misperception in one trial [48] and in another study used data from one-night diagnostic PSG to exclude individuals for whom objective sleep time was ≥2 times higher than their subjectively estimated sleep time [54]. One trial [37] used baseline PSG to classify participants into short and long sleep duration groups. Hypnotic medication was allowed in five studies [36,38,49,50,57]. All studies, with one exception [32], used sleep restriction therapy, alongside an educational element of either sleep hygiene (n = 5), psychoeducation (n = 6), or both (n = 4). Other components included cognitive techniques (n = 8), relaxation (n = 3) and relapse prevention (n = 3). Only three RCTs did not include SCT [34,37,55].

The RCTs delivered weekly treatment, with the exception of one study, which delivered sessions bi-weekly [50]. A range of methods were used to deliver the intervention; face-to-face individual (n = 8) and group sessions (n = 6) or a comparison of both (n = 1); face-to-face group sessions with individual follow up phone calls (n = 1), face-to-face individual sessions with follow up phone calls (n = 1) and a comparison of both face-to-face and online individual formats (n = 1). In all but two studies treatment was four-to-six sessions in length. One study [33] included 16 wkly CBT sessions compared with an equal amount of Tai Chi Chi and a sleep education control group, whilst another study [54] compared one, two, four and eight sessions of CBT-I with a wait-list control. All studies indicated the use of a structured protocol, with the majority of studies specifically referencing a manualised approach or describing methods to ensure treatment fidelity such as training, supervision, evaluation of recordings and checklists. Only two studies did not reference the use of a treatment manual or measures to ensure treatment fidelity [37,55]. The control arms included wait-list (n = 6), treatment as usual at a GP practice (n = 1), minimal contact control (n = 1), relaxation therapy (n = 1), psychological placebo (n = 1) and educational control (n = 5).

Regarding PSG outcomes, two studies conducted PSG in a sleep lab and reported mean outcomes over two nights at each assessment time point [33,58], with one study including an additional night for adaptation [33]. The other two studies conducted ambulatory PSG in the home environment for one night [48] or two nights [49] at each assessment point. Sleep stages were scored using standardised criteria. Only one study [58] reported that reliability checks were performed, with inter-rater agreement being greater than 85% for sleep-stage scoring.

The actigraphy outcomes were typically calculated using the mean score of one or two weeks of actigraphy data at each assessment time point. In and out of bed times were recorded via an event marker on the actigraph hardware, or via concurrent use of sleep diaries. Studies reported using scoring algorithms associated with their chosen software, however it was unclear how these may have been programmed differently. Only one study reported manually adjusting sensitivity criteria [32]. The majority of studies did not report the frequency at which the actigraph records movement, although the studies that did report this indicated either 30-s or one-min epochs. Comprehensiveness of the description of actigraph recordings varied widely between studies, which may be expected considering objective measures were often reported as secondary outcomes.

Quality assessment

Appraisal of risk of bias (see Table S1) yielded a mixed picture with regards to how comprehensively methodological details were reported, with only two studies reporting sufficient detail to perform a complete appraisal. However, in general, the studies were judged to have low risk of bias in most domains.

Almost all studies (n = 14; 93%) generated an adequate randomisation sequence and in only one study (7%) the randomisation protocol was unclear. In five studies the allocation was adequately concealed (33%), whereas in the rest of the studies (n = 10; 67%) the

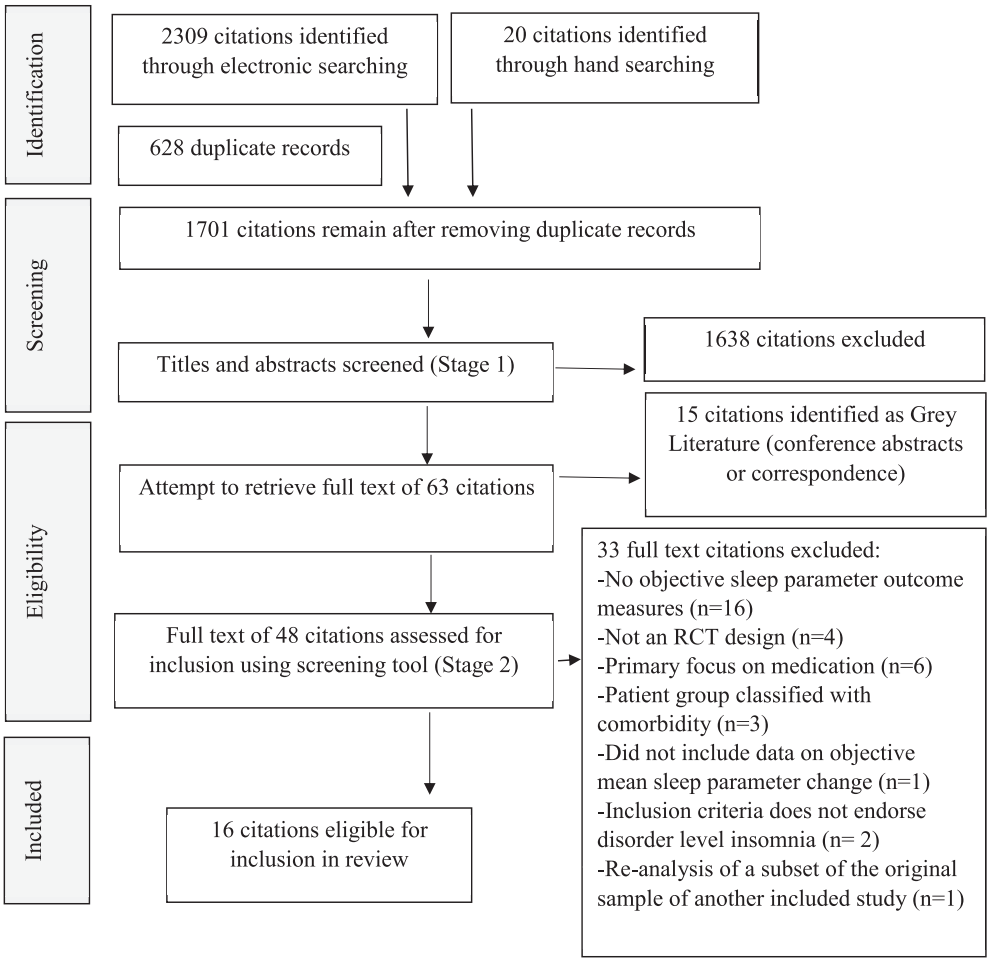


Fig. 1. PRISMA flow chart of included studies.

concealment procedure was unclear. The inherent risk of bias in psychotherapy research due to difficulty blinding participants and researchers was considered unavoidable by some authors. However, three out of the 15 studies (20%) attempted to minimise this bias by using minimally active control conditions and informing all participants that they would receive a behavioural treatment for insomnia [36,48,50]. The remaining studies either reported that blinding was impossible (n = 6; 40%) or did not provide enough details to reach a conclusion (n = 6; 40%).

Regarding blinding of outcome assessors scoring objective sleep, eleven studies (73%) did not provide enough details, three studies (20%) reported a sufficient blinding with one study (7%) reporting that blinding of assessors was not performed. Both actigraphy and PSG require analysis and interpretation [59], therefore blinding of outcome assessors is important to report. Since objective measures features as secondary outcomes it may be that full reporting on these details was overlooked. In total, 14 studies (93%) described the completeness of the outcome data and provided a good amount of details concerning their attrition rates. Only one study (7%) was unclear regarding the handling of incomplete data points. In 14 studies (93%) there was no evidence for selective reporting. However, there was evidence for selective reporting in one study (7%). In this particular study [36], although individual and group CBT-I protocols were independently administered, they were merged together in the analysis with sleep measurements reported collectively from both protocols. Selective reporting was only assessed on the basis of objective measurements, meaning that selective reporting in relation to diary measurements was not accounted for.

Heterogeneity analyses

Heterogeneity analyses revealed that approximately one half of effect size estimates showed pronounced heterogeneity, while the other half were more homogeneous. Specifically, for PSG variables: SOL [$Q(3) = 1.59$, $p = 0.661$], WASO [$Q(3) = 3.08$, $p = 0.379$], SE [$Q(4) = 10.534$, $p = 0.032$] and TST [$Q(4) = 8.726$, $p = 0.068$]. For actigraphy variables: SOL [$Q(10) = 22.241$, $p = 0.014$], WASO [$Q(11) = 16.817$, $p = 0.113$], SE [$Q(13) = 22.103$, $p = 0.054$] and TST [$Q(11) = 24.385$, $p = 0.011$]. For sleep diary variables: SOL [$Q(15) = 33.132$, $p = 0.005$], WASO [$Q(15) = 37.313$, $p = 0.001$], SE [$Q(16) = 49.764$, $p < 0.001$] and TST [$Q(15) = 24.345$, $p = 0.060$]. Due to the pattern of heterogeneity in effect size estimates across sleep parameters, the more conservative random effects model was implemented for the main analysis. When appropriate, fixed effects modelling was also implemented as part of the sensitivity analysis.

Effects on PSG-defined sleep variables

See Table 2 for effect size summary and Fig. 2a–d for forest plots grouped by sleep parameter. For PSG, effect sizes were predominantly small and non-significant [SE ($N_c = 5$; $g = 0.33$ [95% CI = −0.14 to 0.80], $p = 0.169$); WASO ($N_c = 4$; $g = -0.10$ [95% CI = −0.39 to 0.19, $p = 0.509$);

Table 1
Randomised controlled trials of cognitive behavioural therapy for insomnia which report post treatment changes in objective night time sleep.

Author (year) *Location*	Measure	Sample size (f), mean age (SD) Recruitment population	Group Allocation N: randomised (completed)	Diagnosis/Definition of Insomnia (*determined by*) Mean duration (SD)	Components of the CBT-I treatment arm assessed in the meta-analysis	Hypnotic medication status	Therapy Delivery Therapist Qualifications	Number of nights' data at each assessment (*Time points*)
Morin et al. (1993) [58] *USA*	PSG	N = 24 (17 f) 67.1 yrs (5.3) Older adult (60+) community sample	CBT-I: 12 WLC/delayed treatment: 12	Persistent Psychophysiological Insomnia - ICSD WASO > 30 min, ≥ 3n/wk, ≥ 6 mo + daytime imp. *(Clinical interview and sleep log at baseline).* 13 yrs (14.1)	PE, SRT, SCT, CR	Medicationc free	8 sessions; face-to-face group (n = 4−6); weekly; 90 min clinical psychologist	Average of two nights (*Pre-post*)
Friedman et al. (2000) [55] *USA*	Actigraphy Voluntary PSG (no analysis)	N = 39 (26 f) 64.2 y (7.4) Older adult (55+) community sample	SRT*: 12 (11) NSRT: 16 (15) SH*: 11 (11)	ICSD diagnosis SE < 80%, SOL >30 min, TST < 6hr, WASO < 30 min, ≥ 5 n/2wk *(Sleep log at baseline and ICSD diagnosis assessment).*	SRT, SH	Medication free	5 sessions: face-to-face individual; weekly.	Actigraphy: 4 d (*Pre-post*, 3 mo) PSG: 2 nights (*pre-post*, 3 mo)
Edinger et al. (2001) [48] *USA*	PSG	N = 75 (35 f) 55.3 yrs Adult (40-80) community sample	CBT-I*: 25 (23) RT: 25 (23) PT*: 25 (24)	Persistent Primary Insomnia (DSM) Mean WASO ≥ 60 min, > 6 mo + ≥1 sleep disruptive practice e.g., napping. *(Clinical interview and sleep log at baseline)* 13.6 yrs	SCT, PE, SRT	Medication free	6 sessions; face-to-face individual; weekly; 30-60 mins. "Beginning-level" clinical psychologists	1 night (*Pre-post*)
Edinger et al. (2007) [54] *USA*	Actigraphy	N = 86 (43 f) 55.4 yrs (9.7) Middle age and older adult community sample	WLC*:11 (9) CBT-I (No. of Sessions) (1): 16 (16) (2): 18 (17) (4)*: 24 (21) (8): 17 (15)	Primary Insomnia (DSM-IV) WASO >60 min, > 6 mo with onset after age 10, ≥1 sleep disruptive practice e.g., napping (*Clinical interview and sleep log at baseline*).	PE, SCT, SRT	Medication free	4 bi-weekly sessions; face-to-face individual; first session 45−60 mins, follow up sessions 15−30 min. Clinical psychologists	2 wks (*Pre-post*, 3 mo, 6 mo)
Espie et al. (2007) [57] *Scotland, United Kingdom*	Actigraphy	N = 201 (137 f) 54 yrs Patients attending General Medical Practice	CBT-I: 107 (107) TAU: 94 (94)	Insomnia (ICSD-R/DSM-IV) SOL ≥ 30 min and/or WASO ≥ 30 min, ≥3 nights per week, ≥6 mo and negative impact. (*Clinical interview and sleep log at baseline)* CBT: 11.6 yrs (9.79) TAU: 10.6 yrs (12.2)	PE, SH, SCT, SRT, CT	Medication allowed	5 sessions; face-to-face group (n = 4−6) weekly, 1 h. Trained and supervised health visitors	2 wks (*Pre- post*)
Edinger et al. (2009) [50] *USA*	Actigraphy	N = 81 (11 f) 54.2 yrs (13.7) Veteran outpatients with a history of sleep disturbance	CBT-I CMI: 21 (20) CBT-I PI: 20 (16) SH CMI: 20 (15) SH PI: 20 (18)	Insomnia Disorder (RDC) and/or DSM-IV-TR Primary Insomnia SOL + WASO ≥ 60 min per night *(sleep log at baseline and diagnostic interview)* 10.0 yrs (11.0)	PE, SCT, SRT	Medication allowed	4 sessions; bi-weekly; individual; 30−60 min. Clinical psychologists	2 wks (*Pre- post*, 6 mo)
Buysse et al. (2011) [49] *USA*	PSG and Actigraphy	N = 82 (54 f) 71.7 yrs Older adult community sample and 1 primary care clinic.	BBT-I: 42 (39) IC: 40 (40)	Primary insomnia (DSM-IV-TR) minus comorbidity criteria and general insomnia (ICSD-2) Sleep complaint ≥ 1 mo + daytime impairment. (*Self-report questionnaire and clinical interview*).	PE, SRT, SCT	Medication allowed	4 contacts; weekly; two individual face-to-face (initial 45−60 mins, 30 min f/up at 2 wk) 2 20 min telephone calls (wk 1 and 3).	PSG: Average of two nights Actigraphy: 2 wks (*Pre-post*)

(*continued on next page*)

Table 1 (*continued*)

Author (year) *Location*	Measure	Sample size (f), mean age (SD) Recruitment population	Group Allocation N: randomised (completed)	Diagnosis/Definition of Insomnia (*determined by*) Mean duration (SD)	Components of the CBT-I treatment arm assessed in the meta-analysis	Hypnotic medication status	Therapy Delivery Therapist Qualifications	Number of nights' data at each assessment (*Time points*)
Epstein et al. (2012) [56] *USA*	Actigraphy	N = 179 (115 f) 68.9 yrs Older adult (55+) community sample	SCT: 44 (38) SRT: 44 (39) MCI*: 41 (39) WLC*: 50 (38)	Chronic primary insomnia SOL ≥45 min for ≥3 nights/wk, ≥6 mo + daytime impairment. (*Clinical interview and sleep log at baseline*). SCT: 8.23 yrs SRT: 9.82 yrs MCI: 9.94 yrs WLC: 11.7 yrs	PE, SH, SCT, SRT	Medication free	Masters level nurse practitioner 6 sessions; wk 1–4 group (n = 4–6), wk 5–6 telephone; weekly; 60–120 min groups, 15 min telephone. Mental health clinical nurse specialist (Masters Level)	2 wks (*Pre-post*, 3 mo, 1yr)
Harris et al. (2012) [32] *Australia*	Actigraphy	N = 79 (56 f) 40.9 yrs (13.8) Community sample	ISR: 19 (16) SCT*: 20 (17) SCT + ISR: 20(20) SH*: 20 (18)	Sleep onset insomnia (+/− sleep maintenance difficulties) SOL > 30 min, ≥3 nights/week, ≥6 mo + daytime impairment. (*Clinical interview and sleep log at baseline*). 16 yrs (13.6)	SCT, SH, RP	Medication free	5 sessions; Individual face-to-face; weekly, 30 min. Clinical psychologist	2 wks (*Pre-post*, 2 mo, 6 mo)
Irwin et al. (2014) [33] *USA*	PSG	N = 123 (88 f) 65.7 yrs Older adult (55+) community sample	CBT-I*: 50 (48) TCC: 48 (40) SS*: 25 (24)	Primary insomnia (DSM-IV-TR) and general insomnia (ICSD-2) Difficulty initiating/maintaining sleep ≥1 mo + Daytime impairment. (*Clinical interview*).	PE, CR, SCT, SRT, REL, RP, mood intervention	Medication free	16 sessions: face-to-face group (n = 7–10) 120 min class weekly, 4 mo Clinical psychologist supervised by PhD specialist	Average of two nights after 1 night adaptation (*Pre-post*)
Lovato et al. (2014) [34] *Australia*	Actigraphy	N = 188 (63 f) 63.76 yrs (6.45) Older adult community sample	CBT-I: 86 (78) WLC: 32 (31)	Sleep maintenance insomnia WASO <30 min, for >3 night/wk, ≥6mo + daytime impairment: (*Clinical interview and sleep log at baseline*) Mean duration not reported (73% >5 yrs)	SRT, CT, PE, SH, RP	Medication free	4 sessions, weekly; group (n = 4–5); 60 min. Five trainee psychologists	1 wk (*Pre-post*, 3 mo)
Taylor et al. (2014) [35] *USA*	Actigraphy	N = 34 (14 f) 19.71 yrs (2.10) College students (age 18–24 yrs) from University of North Texas	CBT-I: 17 (16) WLC: 17 (13)	Insomnia (DSM-5) SOL or WASO ≥30 mins, 3 nights/wk, ≥3mo + daytime impairment: (*Clinical interview and sleep log at baseline*)	SCT, SRT, SH, REL, CR	Medication free	6 sessions; face-to-face individual; weekly Three doctoral level graduate students	1 wk (*Pre-post*)
Alessi et al. (2016) [36] *USA*	Actigraphy	N = 159 (5 f) 72.2 yrs (7.7) Veteran (age 60+) community sample	CBT-I total: 106(97) (Individual: 54 Group: 52) PE: 53 (53)	Chronic Insomnia Disorder (ICSD-2) ≥3 mo (*Postal survey: ICSD-2 insomnia disorder)*	SH, SCT, SRT, CT	Medication allowed	5 sessions; face-to-face individual or group (n = 3–5); weekly; 60 min	1 wk (*Pre-post*, 6 mo, 12 mo)

Lovato et al. (2016) [37] *Australia*	Actigraphy	N = 91 (48 f) 63.34 yrs (6.41) Community "young" older adults (65–74) targeted but no age exclusion criteria used	CBT-I: 63 (63) (Sleep duration: Short: 30; Long: 32) WLC: 28 (28) (Short: 9; Long: 19)	Mean duration not reported (90.6% ≥ 12 mo) Sleep maintenance insomnia WASO <30 min, for >3 night/wk, ≥6 mo + daytime impairment. *Clinical interview* Not reported (76.92% <5 y)	SRT, CT, PE	Medication free	Masters level Sleep-coaches 4 sessions, weekly; group (n = 4–5); 60 min.	1 wk (*Pre-post*, 3 mo)
Taylor et al. (2017) [38] *USA*	Actigraphy	N = 100 (17 f) 32.73 yrs (7.73) Active duty US Army personnel	ICBT-I: 34 (27) CBT-I*: 33 (30) CC*: 33 (29)	Chronic Insomnia (DSM-5) Diagnosis + SE <85%. *Clinical interview*	SCT, SRT, SH, REL, CR	Medication allowed (if stable use for 4wk)	6 sessions; weekly: 60 mins; face-to-face Clinical psychologist, postdoctoral fellows, social worker.	1 wk (*Pre-post*, 6mo)

* Note that in trials with more than two active treatment arms the asterisk denotes the experimental and control conditions that were compared in the meta-analysis. Abbreviations: BBTI = Brief Behavioural Treatment for Insomnia; CBT-I = Cognitive Behavioural Therapy for Insomnia; CC = Contact control condition; CMI = comorbid insomnia; CR = Cognitive Restructuring; CT = Cognitive Therapy; DSM(IV-TR) = Diagnostic and Statistical Manual of Mental Disorders (IV-Text Revision); f. = Female; f/up = follow up; IC = information control; ICBT-I = Internet Cognitive Behavioural Therapy for Insomnia; ICSD = International Classification of Sleep Disorders; ISR = Intensive Sleep Retraining; ITT = Intention To Treat; MCI = multi-component intervention; Min. = minutes; Mo = Month; n = Nights; PE = Psychoeducation; PI: primary insomnia; PSG = Polysomnography; PT: placebo therapy (psychological); RDC = research diagnostic criteria; REL = Relaxation; RT = relaxation therapy; RP = Relapse Prevention; SC = Sleep Compression; SCT = Stimulus Control Therapy; SD = Standard Deviation; SE = Sleep Efficiency; SH = Sleep Hygiene; SOL = Sleep Onset Latency; SRT = Sleep Restriction Therapy; SS = Sleep Seminar; TCC = Tai Chi Chih; TST = Total Sleep Time; TWT = total wake time at night; WASO = Wake After Sleep Onset; WLC = Wait List Control; Wk(s) = Week(s) Yrs = Year.

SOL ($N_c = 4$; $g = -0.07$ [95% CI = −0.36 to 0.22], $p = 0.641$; and TST ($N_c = 5$; $g = 0.04$ [95% CI = −0.37 to 0.45], $p = 0.848$)]. It is noteworthy that the highest number of available comparisons (N_c) was just five.

Effects on actigraphy-defined sleep variables

A small, reliable effect was observed for a decrease in actigraphy-defined SOL ($N_c = 11$; $g = -0.28$ [95% CI = −0.51 to −0.05], $p = 0.018$), while a negative effect of moderate size was observed for TST ($N_c = 12$; $g = -0.51$ [95% CI = −0.75 to −0.26], $p < 0.001$), indicating a *reduction* of sleep time in the experimental versus control arm. There was no significant effect for WASO ($N_c = 12$; $g = -0.14$ [95% CI = −0.33 to 0.05], $p = 0.138$) or SE ($N_c = 14$; $g = 0.14$ [95% CI = −0.04 to 0.31], $p = 0.137$).

Effects on sleep diary variables

Moderate-to-large effects were found for SOL ($N_c = 16$; $g = -0.50$ [95% CI = −0.70 to −0.30], $p < 0.001$), SE ($N_c = 17$; $g = 0.79$ [95% CI = 0.57 to 1.02], $p < 0.001$) and WASO ($N_c = 16$; $g = 0.75$ [95% CI = −0.96 to −0.55], $p < 0.001$), while there was no reliable difference for TST ($N_c = 16$; $g = 0.06$ [95% CI = −0.12 to 0.23], $p = 0.537$).

Sensitivity analyses

Sensitivity analysis employing fixed-effect modelling was conducted for the post-treatment outcome measures where no significant heterogeneity was detected, yielding comparable results (see Table S2). Sensitivity analysis was also conducted to control for the effect of double comparisons within a study for the same outcome (as quantified by the same measurement). The re-calculations of the random effects model resulted in highly comparable values as those reported in the main analysis.

Publication bias analysis

Visual inspection of the funnel plots indicated that there was no considerable evidence of publication bias across all outcomes and measurements for post-treatment. Egger's regression tests confirmed the results from the visual inspections, with no study yielding a z-score larger than 1.50 and a p-value of less than 0.135.

Discussion

This meta-analysis sought to evaluate the extent to which multi-component CBT-I improves objective measures of sleep in insomnia disorder, whilst simultaneously appraising the quality of the evidence-base. Our results find no evidence that CBT-I reliably improves PSG sleep parameters relative to control groups at post-treatment, although it must be noted that very few studies incorporated PSG measurement (n = 5). For actigraphy, where we had more comparisons, there was a small reduction in effect for SOL ($g = -0.28$) and a moderate (negative) effect for TST ($g = -0.51$). In contrast, and while not a primary aim of our study, there was clear evidence that CBT-I results in robust improvements in diary measures of SOL ($g = -0.50$), WASO ($g = -0.75$) and SE ($g = 0.79$), but not TST ($g = 0.06$), which is consistent with other recent meta-analyses [11] reporting effect sizes of $g = 0.57$ (SOL), $g = 0.63$ (WASO), $g = 0.71$ (SE) and $g = 0.16$ (TST).

The most robust effects for objective sleep in this review were found for TST, reflecting a *reduction* in actigraphy-defined sleep time of approximately 30 min relative to the control group. This decrease may reflect an *initial* reduction in sleep time driven by sleep restriction therapy and/or stimulus control in order to consolidate sleep [60], however it is noteworthy that we did not observe reliable reductions

Table 2
Effect sizes for post-treatment sleep differences between patients assigned to CBT-I versus control.

Outcome	Polysomnography				Actigraphy				Sleep Diary			
	N_c	g (95% CI)	I^2 (95% CI)	p	N_c	g (95% CI)	I^2 (95% CI)	p	N_c	g (95% CI)	I^2 (95% CI)	p
Sleep Onset Latency (SOL)	4	−0.07 (−0.36 to 0.22)	0 (0–86)	0.641	11	**−0.28 (−0.51 to −0.05)**	**52 (5–86)**	**0.018**	16	**−0.50 (−0.70 to −0.30)**	**56 (17–84)**	**<0.001**
Wake After Sleep Onset (WASO)	4	−0.10 (−0.39 to 0.19)	0 (0–97)	0.509	12	−0.14 (−0.33 to 0.05)	38 (0–69)	0.138	16	**−0.75 (-0.96 to −0.55)**	**56 (18–81)**	**<0.001**
Sleep Efficiency (SE)	5	0.33 (−0.14 to 0.80)	63 (0–97)	0.169	14	0.14 (−0.04 to 0.31)	42 (0–69)	0.137	17	**0.79 (0.57 to 1.02)**	**66 (36–85)**	**<0.001**
Total Sleep Time (TST)	5	0.04 (−0.37 to 0.45)	53 (0–97)	0.848	12	**−0.51 (−0.75 to −0.26)**	**54 (9–84)**	**<0.001**	16	0.06 (−0.12 to 0.23)	41 (0–72)	0.537

95% CI = 95% confidence interval; N_c = number of comparisons. Bold text denotes effect size differences significant at p<.05.

in WASO or improvements in SE (for either actigraphy or PSG) and only a small relative reduction of approximately 6 min for actigraphy-defined sleep latency. The assumption is that any reduction in TST is transient, however we conducted exploratory (and by extension, cautionary) meta-analyses of studies with follow-up data at three months and found a sustained effect for reduced (actigraphy-defined) total sleep time (Nc = 4; g = −0.56, $p < 0.001$ see Table S3). Future studies, particularly those employing both PSG and actigraphy, are needed to systematically track changes in sleep *during* CBT treatment and over long-term follow-up.

While design differences between psychotherapy and pharmacotherapy trials make it difficult to directly compare treatment effect size estimates (across meta-analyses) it appears that hypnotics have more pronounced effects on PSG-defined sleep than CBT. A meta-analysis of both published and unpublished trials of *z* drugs reported a small, but reliable effect size (SMD) of −0.36 (CI: −0.57 to −0.16) for PSG-defined SOL relative to placebo [23]. This effect, of course, is greater than what we observed in the present analysis (g = −0.07 CI: −0.36 to 0.22). A recent Cochrane review of eszopiclone versus placebo also showed reliable reductions in PSG-defined SOL and WASO, and increased TST, although number of comparisons was small [25]. In contrast to our findings, hypnotics seem to have similar effects across both objective and subjective sleep parameters, while we see pronounced effects on subjective but not PSG-defined variables. However, results must be interpreted cautiously because our review clearly shows that objective sleep measurement is rarely applied in trials of CBT-I and sample sizes are typically small (contrasting with studies of hypnotics).

A recent comprehensive meta-analysis reported on sleep diary data from 87 studies [11]. While our meta-analysis, in comparison, included a much smaller number of studies with sleep diary data, results show a similar pattern and magnitude of effects, supporting the view that CBT-I principally modifies subjective perceptions of sleep (see Fig. 2a–d). Indeed, previous research indicates that CBT-I "corrects" the discrepancy between self-reported sleep and objective sleep, while having no significant impact on PSG-defined sleep at post-treatment [61]. Conceivably, CBT may modify key psychobiological processes that maintain *perceptions* of poor sleep and daytime impairment in insomnia, such as dysfunctional arousal, sleep incompatible behaviours and key cognitive processes, without having pronounced therapeutic effects on *gross* measures of objective sleep [62,63]. However, it should be noted that we did not assess number of awakenings, sleep architecture or microstructure in the present review, instead focussing on more commonly reported sleep continuity measures. It is possible that CBT modifies EEG indices of arousal (e.g., beta and gamma spectral power) and sleep intensity (delta power) but given the paucity of PSG studies, the evidence-base for these variables is assumed to be limited. Future, adequately-powered and pre-registered studies, are needed to 1) test the effects of CBT on sleep physiology and 2) investigate associations with important clinical outcomes.

Ability to show change in objective sleep likely depends on sample characteristics. Studies in our review recruited participants based on self-report data - consistent with diagnostic criteria - and not on objective sleep. In contrast, trials of hypnotics often recruit participants on the basis of PSG-confirmed sleep disruption [64,65]. It is possible that in the studies we reviewed there was relatively little scope to change objective sleep. One recent, uncontrolled study of inpatient CBT-I [66], found that patients with objective sleep impairment (TST<6hrs) at baseline were more likely to show PSG improvements in WASO at 6 mo follow-up, compared to those with relatively normal sleep (defined as ≥6 hrs sleep duration). Interestingly, those with normal sleep duration were more likely to report a post-treatment *decrease* in PSG-defined total sleep time, consistent with actigraphy observations in the present review, as well as results from a recent secondary analysis of a randomised trial [31]. To fully understand the impact of CBT-I on objective sleep, studies may need to sub-group based on baseline sleep profiles and assess differential change at post-treatment and follow-up.

Study quality

Overall, reviewed studies were judged to have low risk of bias in most domains, lending credibility to our review findings. However, our review revealed a mixed picture regarding reporting of methodological details, which made it more difficult to establish potential bias in objective measurement. Comprehensiveness of the description of PSG and actigraphy recordings and scoring algorithms varied widely between studies. This may be expected considering objective measures were often reported as secondary outcomes. For example, only two RCTs reported their definition of PSG SOL, which were clearly different from one another (latency to stage 2 sleep [58], and the first 10 min of sleep containing no more than 2 min of wake time, stage 1, or movement time [48]). Moreover, studies rarely reported details on blinding status of outcome assessors in relation to objective measurement, which is a potentially important source of bias because actigraphy and PSG require analysis and interpretation [59]. In order to facilitate consistency between trials and to appraise sources of bias, studies should more fully describe procedures for the set-up and recording of sleep (e.g., how the recording period was defined in PSG studies), as well as the analysis and interpretation of objective data, including blinding status of objective data.

Limitations

The narrow inclusion criteria of this review may be considered a limitation, since this led to the exclusion of two recent, potentially relevant studies, as a result of their insufficiently stringent diagnostic criteria [67,68]. Findings may not generalise to insomnia complaints in the general population, where a significant proportion experience symptoms of insomnia but do not meet diagnostic criteria and/or experience significant comorbid medical or psychiatric disorders [3]. Most studies included in this review used a highly selected sample via the exclusion of other sleep disorders, dementia/mild cognitive impairment and uncontrolled physical or psychiatric difficulties. Although this reduces the influence of potentially confounding factors, it also limits the generalisability of review findings. Future work should aim to synthesise evidence on the objective sleep effects of

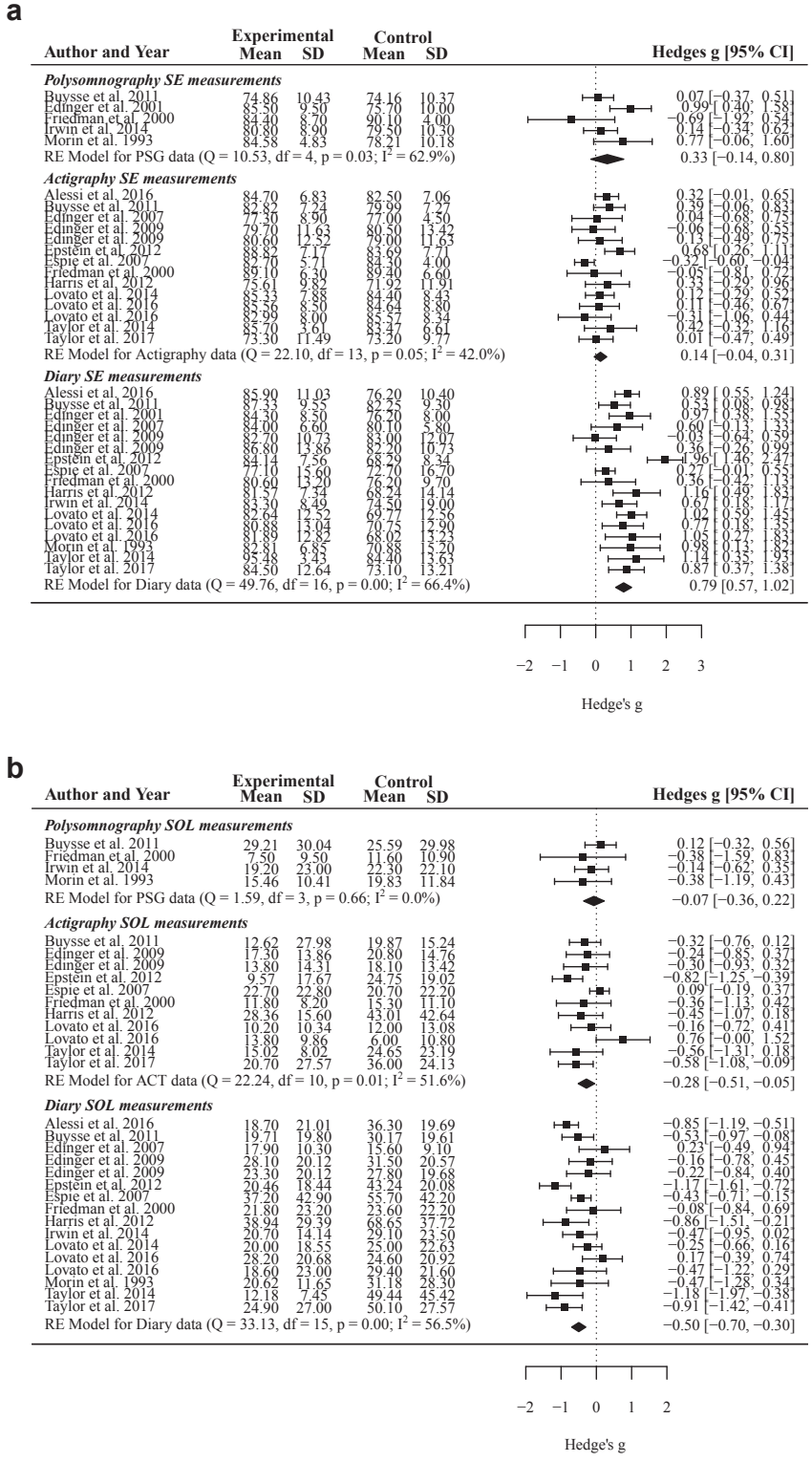
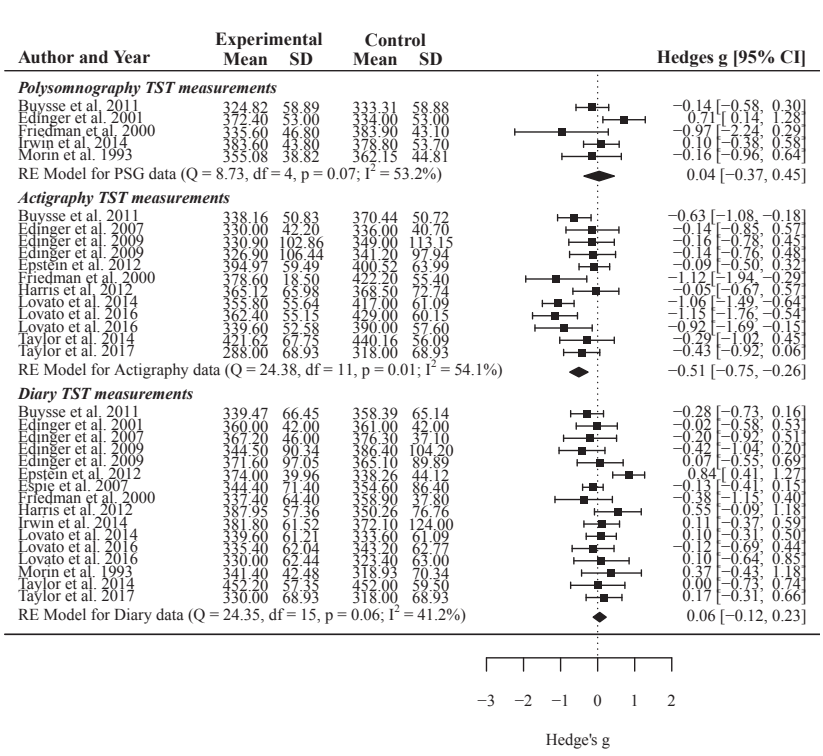


Fig. 2. Forest plots showing individual study and global effect size estimates for comparisons between CBT-I and control groups on (a) sleep efficiency; (b) sleep onset latency; (c) total sleep time; and (d) wake-time after sleep onset as measured by polysomnography, actigraphy and sleep diary.



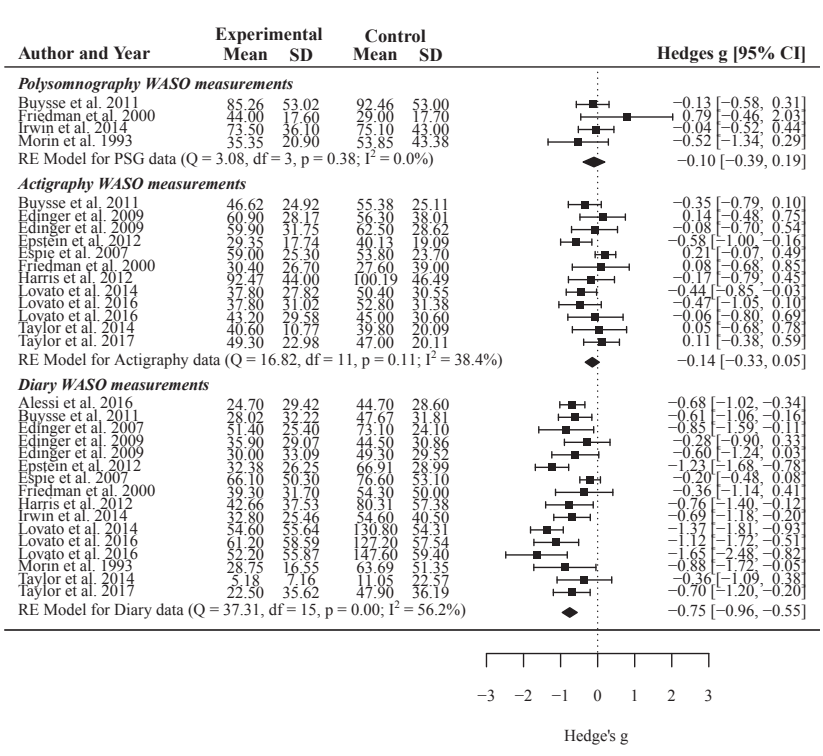


Fig. 2. (*continued*).

CBT-I in comorbid insomnia samples. This review sought to make conclusions based on a relatively homogeneous set of studies; therefore, narrow inclusion criteria were considered appropriate. Despite this, differences between the studies included in this review should also be considered, particularly given observed heterogeneity for some of the treatment effect estimates. First, it must be noted that studies with very small samples (for example in the case of Friedman et al., n = 8 for CBT vs n = 4 for control) can strongly influence estimates from random-effects models and contribute to heterogeneity. Second, treatment protocols included in this meta-analysis differed with respect to component composition, treatment duration and format, and therapist background. While this grouping is common in

CBT meta-analyses, in part because such variables appear to have limited impact on clinical outcomes [11], the effect on objective measures should be systematically interrogated in pre-defined sub-group analyses as the field matures. A final point is that hypnotic medication was *permitted* in five studies [36,38,49,50,57], which may impact objective sleep outcomes, particularly if there are between-group differences in the proportion taking medication at post-treatment. We found that only one of these studies reported hypnotic medication use by study arm at post-treatment; future studies should report such information in order to aid interpretation of effects and permit sub-group analyses when synthesising across studies.

Conclusion

Insomnia is a highly prevalent condition that has a considerable negative impact on daytime functioning and health. Objective measurement of sleep is relevant since insomnia is characterised by reliable impairments in objective sleep, and such impairments may confer risk for adverse medical and cognitive outcomes. While the literature is small and still developing, CBT-I appears to have limited impact on objective sleep continuity, a small effect for decreasing actigraphy-defined sleep latency and a robust effect (of moderate strength) for decreasing actigraphy-defined sleep duration. Future, well-designed studies, with low risk of bias and transparent reporting are needed to specifically test the impact of CBT-I on objective sleep, including sleep microstructure.

Author contributions

LM and SDK conceived the original idea and designed the data selection criteria. LM conducted the literature search, paper screening and extraction of study details. LB and AB extracted and cross-validated data for the meta-analysis under the supervision of SDK. LB planned and performed the statistical analysis under the supervision of SDK. AB cross-checked the analysis scripts. LM, LB and SDK drafted the manuscript. All authors contributed to and approved the final manuscript.

Practice points

1) Objective sleep outcomes are rarely investigated in trials of CBT-I
2) While the literature is small, this meta-analysis finds no evidence that CBT-I modifies PSG measures of sleep initiation, maintenance or duration. Conversely, actigraphy shows a small reduction in sleep onset latency and moderate reduction in total sleep time.
3) The sleep benefits of CBT-I are more clearly expressed in the subjective versus objective domain.

Research agenda

1) Studies should clearly report methodological details of actigraphic and polysomnographic assessments, as well as information on use of prescription sleep aids during these assessments
2) Studies should include objective parameters (including SOL, WASO, SE, TST, number of awakenings and arousals), alongside subjective sleep outcomes, which should be tracked *throughout treatment* and assessed at both post-treatment and at follow-up.
3) Prospective randomised trials, stratifying participants by objective sleep profiles (e.g., normal and short sleep duration) are needed to understand effects on post-treatment objective sleep.
4) Studies should determine whether degree of change in objective sleep, pre-to-post CBT-I, mediates improvement in relevant health and disease outcomes.
5) Objective measures beyond gross sleep architecture or continuity, such as quantitative EEG metrics and sleep microstructure, should be examined in future RCTs (and potentially through registered secondary analyses of completed studies).

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.smrv.2019.06.002.

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* The most important references are denoted by an asterisk.

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