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Changes in Dysfunctional Beliefs about Sleep after Cognitive Behavioral Therapy for Insomnia: A Systematic Literature Review and Meta-analysis

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Summary

Cognitive behavioral therapy for insomnia (CBT-I) is the preferred treatment for chronic insomnia and sleep-related cognitions are one target of treatment. There has been little systematic investigation of how sleep-related cognitions are being assessed in CBT-I trials and no meta-analysis of the impact of CBT-I on dysfunctional beliefs about sleep, a core cognitive component of treatment. Academic Search Complete, Medline, CINAHL and PsychInfo from 1990–2018 were searched to identify randomized controlled trials of CBT-I in adults (≥ 18 years) reporting some measure of sleep-related cognitions. Sixteen randomized controlled trials were identified comparing 1134 CBT-I and 830 control subjects. The Dysfunctional Beliefs and Attitudes about Sleep Scale was utilized almost exclusively to assess sleep-related cognitions in these trials. Hedge's g at 95% confidence interval (CI) was calculated to assess CBT-I effect size at post-treatment compared to controls. CBT-I significantly reduced dysfunctional beliefs about sleep ($g = -0.90$, 95% CI $-1.19, -0.62$) at post-treatment. Three trials contributed data to estimate effect size for long-term effects ($g = -1.04$ (95% CI $-2.07, -0.02$) with follow up time ranging from 3 – 18 months. We concluded that cognitive behavioral therapy for insomnia has moderate to large effects on dysfunctional beliefs about sleep.

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Keywords

Dysfunctional Beliefs about Sleep; DBAS; Cognitive Behavioral Therapy; Insomnia; CBT-I; Sleep-related cognitions

Numerous systematic reviews and meta-analyses have established the efficacy of cognitive behavioral therapy for insomnia (CBT-I) for improving several sleep-wake parameters and insomnia symptoms (1–10). Research across CBT-I trials has primarily focused on the behavioral components of treatment, mainly sleep restriction and stimulus control. Both strategies have been shown to be effective for insomnia even if delivered as standalone treatments (11–16). Less attention has been given to the cognitive components of CBT-I. The objective of cognitive strategies in CBT-I is to break the cycle of dysfunctional beliefs about sleep that can lead to, maintain, or worsen insomnia. Cognitive restructuring, which involves identifying and challenging dysfunctional cognitions and unrealistic expectations that can perpetuate insomnia, along with Socratic questioning to facilitate learning (17,18), is one widely used cognitive strategy. There have been limited systematic reviews of the effectiveness of cognitive components of CBT-I and measurement of sleep-related cognitions in randomized trials.

Several theoretical models of insomnia recognize sleep-related cognitions that perpetuate insomnia symptoms. Increased worry and rumination about lack of sleep can increase cognitive arousal and decrease likelihood of falling asleep (19,20). Lack of sleep can lead individuals to engage in failed explicit attempts to produce sleep further increasing cognitive arousal (21). Individuals may also develop erroneous beliefs about sleep that lead to maladaptive behaviors such as napping or avoiding challenging tasks (22). The measurable concepts associated with these mechanisms could potentially include, but are not limited to: sleep locus of control (23), dysfunctional beliefs and attitudes about sleep (22), sleep effort (24), sleep self-efficacy (25), sleep-related worry (26), anxiety and preoccupation about sleep (27), and insomnia symptom rumination (28). However, it is unclear whether changes in sleep-related cognitions are being reported and which measures, if any, are being collected in CBT-I trials.

We thus conduct a literature review and meta-analysis to answer the following research questions:

- a. How are sleep-related cognitions assessed and reported across CBT-I trials?
- b. What is the effect of CBT-I on sleep-related cognitions?

METHODS

We performed a systematic review and meta-analysis in accordance with the PRISMA (Preferred Reporting Items for Systematic reviews and Meta-Analyses) guidelines (29). We assessed for risk of bias using the Revised Cochrane risk-of-bias tool for randomized trials (RoB 2) from the Cochrane Handbook for Systematic Reviews (30). The predetermined methods were registered online with PROSPERO (Reg. CRD42018112173).

Data Sources and Searches

We searched Academic Search Complete, Medline, CINAHL, and PsychInfo with the terms “cognitive behavioral therapy for insomnia,” “sleep restriction,” “cognitive restructuring,” and “sleep behavior therapy” for publications from 1990–2018 in peer-reviewed journals and written or translated in English. We also reviewed the reference lists of 5 review articles (1,4,31,32).

Study Selection

We sought to identify randomized controlled trials (RCT) of CBT-I in adults (aged 18 years) reporting some measure of sleep-related cognitions. We identified the following treatments as being part of CBTI: relaxation, sleep restriction, stimulus control, and cognitive restructuring, including identifying and challenging dysfunctional thoughts about sleep. Acceptable control groups included wait list, no treatment/usual care, sleep hygiene or general health education, or alternative inactive control condition. We did not specify mode of delivery or length of treatment as inclusion criteria to maximize data capture. All measures targeting sleep-related cognitions as identified in our search were allowed, including the Dysfunctional Beliefs and Attitudes Scale (DBAS) (22), Sleep Locus of Control Scale (23), Sleep Effort Scale (24), Sleep Self-efficacy Scale (25), Sleep-related Worry Questionnaire (33), Anxiety and Preoccupation about Sleep Questionnaire (19), Sleep Disturbance Questionnaire (34) and Daytime Insomnia Symptom Response Scale (28). We excluded trials with only measures of hyperarousal or testing cognitive performance or impairment without any measure of sleep-related cognitions.

Data Extraction

For each CBT-I trial, the first author completed all data collection, extraction and coding. Trials in which the effect on sleep-related cognitions were reported, were also investigated for protocol papers and other primary or secondary results papers if necessary, for extracting relevant data for the meta-analysis and summary of evidence tables. Trials testing multiple treatments or modes of delivery were included but only data for arms testing CBT-I and an acceptable control condition were extracted for meta-analysis. When necessary data were missing, the authors of the articles were contacted and requested to provide these data. No inter-rater reliability ratings were collected because all data were extracted and coded by the first author.

Data Synthesis and Analysis

We examined the effects reported immediately after treatment (post-test) for the intervention versus control groups using the available published statistics or obtained from the corresponding author. Hedges’s g , a variation of Cohen’s d correcting for possible bias due to small sample size was used as the standardized effect size (35). Effect sizes were computed using post-intervention means and their standard deviations or standard errors. Pooled effect sizes were weighted by the inverse standard error, considering the precision of each study. A random effects model was chosen for all analyses. A negative value was chosen to indicate an effect size in the expected direction, e.g., less dysfunctional beliefs. If necessary, independence of results was ensured by averaging effect sizes across all

subgroups so that only one result per study was used for each quantitative data synthesis. Effect sizes of 0.56–1.2 can be assumed to be large, while effect sizes of 0.33–0.55 are moderate, and effect sizes of 0–0.32 are small (36).

To calculate the individual effect sizes as well as the pooled mean effect size we used the computer program Comprehensive meta-analysis (CMA) version 3.3.070 for Windows, developed for support in meta-analysis (www.metaanalysis.com). Long-term effect sizes were estimated for studies with DBAS assessments beyond post-treatment for intervention and control groups using the time point furthest from baseline. Publication bias was assessed with funnel plots, Trim and Fill method (37) and the Egger test (38).

RESULTS

Our search strategy identified 1475 references for review of the title and abstract after removal of duplicates. Of these, 150 articles were considered potentially appropriate for inclusion and the full text was obtained and reviewed for 51 studies. Sixteen studies met the criteria for inclusion and contributed data to the pooled estimates presented (39–54). The study flow diagram with reasons for exclusion is presented in Figure 1.

Study Characteristics

Table 1 reports descriptive data for the 16 included studies, which involved a total of 1964 participants (range 34 to 312 participants per study): 1134 in CBT-I, and 830 in control. Most study populations were of late or middle age (mean age 49.7 years). Sex was predominantly female (68.1%), and all trials were performed in developed countries. Three trials recruited from health centers either by direct referral from clinicians (39,41,42), one from identification of electronic health records (45), and the remaining recruited individuals in the general community through print and online advertisements. Eleven trials defined insomnia using criteria from the Diagnostic and Statistical Manual of Mental Disorders (55) (40–44,47,48,50–53) while three referred to research diagnostic criteria defined by the American Academy of Sleep Medicine (56) (45,46,49). The remaining two trials used the Insomnia Severity Index score indicating mild chronic insomnia for eligibility (39,54). Several trials targeted populations with specific comorbidities including cancer (39,42), heart disease (54), and osteoarthritis (45). Only 2 trials excluded individuals on the basis of sedative or hypnotic medication use (42,51) and one trial included information about a cessation program for participants wishing to discontinue sedative/hypnotic use under medical supervision as part of the CBT-I intervention (39).

CBT-I was delivered over a minimum of 6 and maximum of 10 weeks either in-person group (n=4), self-paced in print or online (n=6), in-person individual (n=3), or telephone (n=4) (non-mutually exclusive groups). Comparator control groups consisted of a waiting list or usual treatment (n=11), self-management education (n=3), alternative behavioral intervention (n=1), and medication placebo (n=1). Of the 16 included trials, four trials had more than one intervention group; in three out of these four trials a self-help format of CBT-I was compared to a self-help format with telephone support from a designated intervention provider (46,49,52) and the remaining trial tested individual in-person to pre-packaged DVD CBT-I program (42). All intervention providers were either trainee or licensed mental health

professional. Six trials reported that either audio or video recordings of CBT-I sessions were reviewed to assess the interventionists' fidelity to the treatment protocol (42,44,47,51,54,57) while four trials utilized debrief and consultation sessions between interventionists and professionals experienced with CBT-I (40,41,45,50), and the remaining provided no details about fidelity.

The DBAS was employed in all trials assessing sleep-related cognitions. The majority of trials selected the 16-item DBAS as the primary sleep-cognition instrument (7 out of 16 trials) (40,42,44,48,50,51,53), five trials used the 30 or 28-item (43,49,52–54), three trials used 10-item (45–47), and one trial used 13-item (39). One trial included two additional sleep-cognition measures: Sleep Locus of Control and Sleep Self-efficacy (40), and another trial included Sleep Disturbance Questionnaire (54). The primary outcome for 13 out of 16 trials was the Insomnia Severity Index (58); two trials used measures of sleep-wake time (43,52); and the remaining trial used the Fatigue Severity Scale (59) (41).

In referring to the cognitive component of therapy, 13 of 16 trials reported cognitive restructuring as the primary technique. Two trials did not include any cognitive components of CBT-I: Vitiello et al. (2013) combined behavioral components of CBT-I with cognitive behavioral therapy for pain, and Horsch et al. (2017) included only relaxation, sleep restriction, and sleep hygiene (45,48). The remaining trial made reference to techniques to improve sleep and cope with worry with no mention of cognitive restructuring, specifically (41). Additionally, one trial reported using the Automatic Thought Record technique where participants are asked to explore the situation and intensities of emotions associated with automatic thoughts about sleep (46), and another reported using the Constructive Worry technique in which the time, place and method of worry is clearly prescribed such that the process is not sleep disruptive (41).

Study Quality

Methods for randomization were generally reported in detail with a few exceptions (Table 2). Bias due to deviations from intended interventions were avoided by a majority of the studies by using intent-to-treat analyses resulting in a low risk assessment for that domain. Trials used single and multiple imputation methods to adjust for differential retention rates across groups and address missing outcome data, resulting in generally low risk assessment. Most studies did not use blinding techniques for participants because in many cases it wasn't feasible due to the nature of the intervention and a wait list or no treatment control group. Per the Cochrane risk of bias tool (30), knowledge of intervention rises to the level "Some concerns" for outcomes where it is unlikely that knowledge of intervention could influence reporting of that outcome, i.e. bed time or rise time. "High" risk assessment is indicated for outcomes where it is likely that knowledge of intervention could influence reporting of that outcome, as in the DBAS. For example, "...insomnia is ruining my ability to enjoy my life..." is a DBAS item where it is likely that participant reporting could be influenced by knowledge of the intervention and thus judged as "High". As a result, 73.3% of included trials with either waitlist or no treatment control groups were judged as "High" risk for bias in measurement of outcome domain.

Post-Treatment Effects

As seen in Figure 2 under the random effects model the point estimate and 95% confidence interval for the combined studies is $g = -0.90$ ($-1.19, -0.62$). Using Trim and Fill method, these values are unchanged. The significant effect sizes ranged from large (-2.09) to moderate ($-.42$). Effect sizes for three trials did not reach statistical significance; two of those three trials included only behavioral components of CBT-I and no cognitive components. Egger's test of asymmetry in the distribution of effect sizes did not reach statistical significance ($p = 0.09$). Three studies contributed data to estimate effect size for long-term effects ($g = -1.04$, 95% CI $-2.07, -0.02$) with follow up time ranging from 3 – 18 months.

DISCUSSION

Our first aim was to understand how sleep-related cognitions were being assessed across CBT-I trials. We found that the DBAS was the primary sleep-related cognitions instrument for all 16 included trials. One reason for the popularity of the DBAS could be that several validated CBT-I programs specifically mention identification and challenging of dysfunctional beliefs about sleep as a core component (18,60,61). This component is meant to facilitate identification of maladaptive sleep cognitions, challenging their validity, and reframing them as more adaptive thoughts. The principal beliefs and attitudes addressed are based on the Dysfunctional Beliefs and Attitudes Scale (DBAS) 30-items which include five subscales (a) misattribution or amplification of the consequences of insomnia (e.g. "I cannot function without adequate sleep"), (b) control and predictability of sleep (e.g. "Insomnia is destroying my entire life"), (c) unrealistic sleep expectations (e.g. "I should sleep as well as my bed partner"), (d) misconceptions about the causes of insomnia (e.g. "Insomnia is due to a chemical imbalance") and (e) faulty beliefs about sleep-promoting practices (e.g. "If I try harder, I will eventually fall asleep") (62). We found that 6 of the 16 included trials utilized the 16-item version, three utilized the 10-item, one utilized the 13-item, and the remaining the 30 or 28-item versions. The psychometric properties of the 10, 16 and 30-item DBAS have been compared and a significantly greater reduction in total DBAS scores was found in CBT-I vs waitlist for all 3 versions (63).

Some protocols suggested that participants complete the DBAS as part of the intervention which allows the interventionist to focus attention on strongly held dysfunctional beliefs. As such, the DBAS could be considered both a process and intervention outcome for the trials that included DBAS scoring within the intervention. However, the level of detail provided about the cognitive components of treatment varied, from trials reporting fully outlined sessions to little explanation other than "cognitive restructuring." Therefore, it was not always clear whether the DBAS was utilized within the intervention as a scored assessment or whether the individual items were used as examples to illustrate dysfunctional beliefs. This difference in trial procedures could result in some participants being exposed to the DBAS pre and post-treatment and others having an added exposure during the intervention. This potential difference could introduce bias that we were not able to fully assess. Future trials should provide more detail about cognitive components of CBT-I and employ placebo

behavioral control groups to address potential sources of bias from knowledge of the intervention.

We were surprised to find that the DBAS was used almost universally in trials assessing sleep-related cognitions. One possible reason is that researchers may not see the addition of other sleep-related cognition measures as contributing unique information beyond the DBAS because of the significant moderate to high positive correlation between the DBAS and other measures, i.e. Glasgow Sleep Effort Scale ($r = 0.50$, $p < .0001$) (24), the Anxiety and Preoccupation about Sleep Questionnaire (APSQ) ($r = .50$ – $.61$, $p < .01$) (64), and the Sleep Disturbance Questionnaire (SDQ) ($r = 0.56$, $p < .0001$) (54). Only two included trials employed alternative measures of sleep-related cognition along with the DBAS. Chow et al. (2018) tested an online format of CBT-I and found that those receiving the intervention had significantly higher Internal Sleep Locus ($p = .03$, indicating greater control over sleep problems) as well as significantly lower levels of Chance Sleep Locus ($p < 0.01$, indicating less attribution of sleep-problems to external forces) (40). Chow (2018) also found no significant difference between groups in Sleep Self-efficacy at post-intervention (40). Redeker et al. (2017) collected data on the Sleep Disturbance Questionnaire and found intervention group score at post-intervention was not significantly different compared to the attention control group (54). We found a single trial that met all inclusion criteria and used the Anxiety and Preoccupation about Sleep Questionnaire (APSQ) as the primary sleep-cognition instrument without DBAS data (64). This trial could not contribute to the meta-analysis but reported significant differences between intervention and control in APSQ scores pre to post-treatment ($p < .01$). These results underscore the need to focus future research on sleep-related cognition measures other than the DBAS.

In our meta-analysis we found that the CBT-I interventions reduced dysfunctional beliefs about sleep. The overall moderate to large effect sizes of CBT-I on dysfunctional beliefs were in line with CBT-I effects on insomnia severity ($g = .98$, 95% CI 0.82, 1.15) (65). We also found moderate to large long-term effect sizes ($g = -1.04$, 95% CI -2.07 , -0.02) with follow up time ranging from 3 – 18 months. Few trials contributed data on long-term effects, in part because waitlist control groups were often not followed beyond post-treatment. Our findings are important because improvements in dysfunctional beliefs about sleep among people with insomnia are associated with improvements in sleep quality (66), daytime symptoms (67), depressive symptoms (33), fatigue (41,68) and better maintenance of treatment gains (53,69). How and to what extent changes in dysfunctional beliefs are associated with improvement in insomnia and related symptoms such as mental health warrant future investigation (70). Our data was limited to published trials therefore we were unable to discern differences in DBAS subscale scores. More detailed reporting of subscale scores of the DBAS could help elucidate which elements of dysfunctional beliefs are most sensitive to change.

Despite the various modes of intervention delivery employed, improvement in sleep-related cognitions was consistently observed. For example, fully automated online CBT-I interventions reduced DBAS scores which suggests that self-administered exercises can yield benefits for dysfunctional cognitions without interventionist facilitation (40,43,46,48,50). Duration of treatment and average age of participants did not vary widely

across studies. Eleven out of 16 trials delivered CBT-I over 6 weeks and only two trials had an average participant age of less than 40 years which suggests some standardization of the intervention in the field and targeting of middle and older age groups with higher prevalence of insomnia than younger age groups. Other preliminary research suggests individuals with low sleep self-efficacy and high level of dysfunctional beliefs regarding the consequences of poor sleep and helplessness related to insomnia may be particularly good candidates for CBT-I (71).

There were three trials with non-significant effects of CBT-I on DBAS(45,48,54). One trial was a small study exclusively among individuals with heart failure whose experience of insomnia may not be representative of individuals without life threatening medical comorbidity (54). This trial among individuals with heart failure reported statistically significant group $\times$ time interaction effects on DBAS in the CBT-I group after controlling for age and comorbidity across multiple time points (baseline, post-treatment, 6 mos) compared to the control group where there was no decrease (54). The remaining two trials included only behavioral components of CBT-I (sleep restriction and stimulus control) and showed no significant effects on DBAS(45,48). Few studies have investigated the effect of different components of CBT-I on dysfunctional beliefs. Eidelman et al. (2016) found that behavioral components alone (sleep restriction and stimulus control) had a significant and sustained improvement in dysfunctional beliefs about sleep, and that cognitive components (whether alone or in combination with behavioral components) had a stronger effect than the behavioral components alone (72). Jansson-Frojmark et al. (2012) found that constructive worry added to the effect of sleep restriction and stimulus control, which resulted in greater reductions in sleep-related worry (73). Further study is needed to confirm the added benefit of the different strategies within the cognitive component of CBT-I in reducing dysfunctional beliefs about sleep and determine whether it is cost-effective when translated into practice.

In conclusion, we found that the DBAS was employed almost universally to assess sleep-related cognitions in randomized trials evaluating CBT-I and that CBT-I was effective in reducing dysfunctional beliefs about sleep with moderate to large effects.

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Glossary

Cognitive behavioral therapy for insomnia (CBT)

a structured program to treat insomnia by eliminating common sleep-disruptive behaviors and correcting the beliefs/attitudes that support such practices.

Dysfunctional beliefs about sleep

faulty beliefs about consequences of poor sleep, uncontrollability and helplessness related to sleep, sleep-promoting behaviors, and the causes of insomnia that are instrumental in exacerbating sleep disturbances

Stimulus control

CBT strategy is based on the assumption that both the timing (bedtime) and sleep setting (bed/bedroom) are associated with repeated unsuccessful sleep attempts and, over time, become conditioned cues for arousal that perpetuate insomnia. As a result, the goal of this strategy is that of re-associating the bed and bedroom with successful sleep attempts.

Sleep restriction

goal of this CBT strategy is to reduce nocturnal sleep disturbance primarily by restricting the time allotted for sleep each night so that, eventually, the time spent in bed closely matches the individual's presumed sleep requirement.

Relaxation therapy

goal of this CBT strategy is to reduce or eliminate sleep-disruptive physiological (e.g., muscle tension) and/or cognitive (e.g., racing thoughts) arousal.

Cognitive restructuring

process of challenging expectations for good sleep, ability to tolerate a poor night of sleep, and misattribution of causes and consequences of insomnia

Automatic Thought Record

goal of this CBT strategy is to explore the situations and intensities of emotions associated with the automatic thoughts about sleep

Constructive Worry

strategy where time, place and method of worry is clearly prescribed such that the process is not sleep disruptive

Placebo Behavioral Control

a psychological placebo for behavioral treatment that occupies hours of patient-therapist interaction

Blinding

concealment of group allocation from one or more individuals involved in a clinical research study

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Practice Points

1. Cognitive behavioral therapy for insomnia is effective with moderate to large overall effects on dysfunctional beliefs about sleep.
2. Fully automated online cognitive behavioral therapy for insomnia can change dysfunctional beliefs about sleep without interventionist facilitation.

Research Agenda

1. There is a dearth of research on the impact of cognitive behavioral therapy for insomnia on measures of sleep-related cognitions other than Dysfunctional Beliefs and Attitudes Scale. Future studies including more detailed reporting of subscale scores of the DBAS could help elucidate which elements of dysfunctional beliefs or sleep-related cognitions are most sensitive to change.
2. Future randomized controlled trials can address potential sources of bias from participants' knowledge of group assignment by utilizing placebo behavioral control groups.
3. Further study of the cognitive component of cognitive behavioral therapy for insomnia could elucidate which specific cognitive strategies provide enough benefit to be cost-effective relative to behavioral components alone.
4. How and to what extent changes in dysfunctional beliefs are associated with improvement in insomnia and related symptoms such as mental health warrant future investigation.

Figure 1.
Flowchart of Literature Search Results

Figure 2.

Forest Plot of Included Trials and Effect Sizes, No Tx – No treatment, Placebo – placebo behavioral control, WL – waitlist, Rx Placebo – medication placebo, Combined – Effect size is averaged between 2 comparisons of intervention and control (3 arm trials).

Table 1.**Characteristics of Randomized Trials of CBT-I**

Study, year	N total	Diagnosis	Mean Age (SD)	Location	% Women	CBT-I protocol when/how often	Mode of Delivery	Comparator	DBAS Item #
Casault, 2015 ³⁹	38	Nonmetastatic cancer and insomnia	56.9	Canada	92.1	9 sessions over 6 weeks	Self-help book/telephone reinforcement	Usual care	13
Chow, 2018 ⁴⁰	303	Insomnia	43.3 (11.6)	USA	72.0	1 module released 7 days after completion of the previous, total 6 modules	Online fully automated	Selfmanagement education	16*
Edinger, 2001 ⁴⁷	70	Insomnia	55.3 (10.5)	USA	46.7	6 sessions over 6 weeks	Individual inperson, first session audiotape	Behavioral placebo	10
Horsch, 2017 ⁴⁸	153	Insomnia	41.0 (13.9)	Netherlands	64.0	6–7 weeks self-paced	Online fully automated	Waitlist	16
Jernelov, 2012 ⁴⁹	135	Insomnia	47.9 (13.9)	Sweden	82.0	Self-help book or book with 6 weekly telephone calls	Telephone	Waitlist	30
Lancee, 2015 ⁵⁰	63	Insomnia	50.0 (13.7)	Netherlands	83.3	6 modules over 6 weeks	Online with individual email feedback	Waitlist	16
Lovato, 2014 ⁵¹	118	Insomnia	64.0	Australia	50.0	4 sessions over 4 weeks	In-person group	Waitlist	16
Mimealt, 1999 ⁵²	54	Insomnia	50.8 (12.6)	Canada	59.0	Self-paced over 6 weeks with telephone support, materials mailed once per week	Self-paced with and without weekly telephone reinforcement	Waitlist	30
Morin, 2002 ⁵³	38	Insomnia	65.0 (6.5)	USA	64.1	8 sessions over 8 weeks	In-person group	Medication placebo	28
Redeker, 2017 ⁵⁴	48	Heart failure and insomnia	59.2 (14.8)	USA	52.1	8 sessions over 8 weeks	In-person group alternating telephone support	Selfmanagement education	28*
Sandlund, 2018 ⁴¹	129	Insomnia	55 (17.1)	Sweden	71.7	7 sessions over 10 weeks	In-person group	Usual care	16
Savard, 2014 ⁴²	241	Breast cancer and insomnia	54.4 (8.8)	Canada	100	6 sessions over 6 weeks or 6 videos	In-person individual or video	Usual care	16
Strom, 2004 ⁴³	81	Insomnia	44.1 (12.0)	Sweden	71.0	5 sessions over 5 weeks	Online with individual email feedback	Waitlist	28
Taylor, 2014 ⁴⁴	34	Insomnia	20.0 (2.5)	USA	41.2	6 sessions over 6 weeks	In-person Individual	Waitlist	16
Vitiello, 2013 ⁴⁵	245	Osteoarthritis and insomnia	73.0 (8.4)	USA	80.3	6 sessions over 6 weeks	In-person group	Selfmanagement education	10

Study, year	N total	Diagnosis	Mean Age (SD)	Location	% Women	CBT-I protocol when/how often Self-paced over 6 weeks, materials delivered once per week	Mode of Delivery	Comparator	DBAS Item #
Yan-Yee Ho, 2014 ⁴⁶	312	Insomnia	38.5 (12.5)	China	71.1		Online	Waitlist	10

* Chow et al. 2018 included 2 additional sleep-cognition measures: Sleep Locus of Control and Sleep Self-efficacy (40).

* Redeker et al. 2017 included the 1 additional sleep-cognition measure: Sleep Disturbance Questionnaire (54).

Table 2.

Risk-of-bias assessment

Author, year	Bias arising from the randomization process	Bias due to deviations from intended interventions	Bias due to missing outcome data	Bias in measurement of the outcome	Bias in selection of the reported result	Overall assessment
Casault, 2015 ³⁹	Low	Low	Low	High	Low	Some concerns
Chow, 2018 ⁴⁰	Low	Low	Low	Low	Low	Low
Edinger, 2001 ⁴⁷	Low	Low	High	Low	Low	Some concerns
Horsch, 2017 ⁴⁸	Low	Low	Low	High	Low	Some concerns
Jernelov, 2012 ⁴⁹	Low	Low	Low	High	Low	Some concerns
Lancee, 2015 ⁵⁰	Some concerns	Low	Low	High	Low	Some concerns
Lovato, 2014 ⁵¹	Some concerns	Low	Low	High	Some concerns	Some concerns
Minealt, 1999 ⁵²	Some concerns	Low	Low	High	Low	Some concerns
Morin, 2002 ⁵³	Low	Low	Low	Some concerns	Some concerns	Some concerns
Redeker, 2017 ⁵⁴	Some concerns	Some concerns	Low	Low	Low	Some concerns
Sandlund, 2018 ⁴¹	Low	Low	Low	High	Low	Some concerns
Savard, 2014 ⁴²	Low	Some concerns	Some concerns	High	Low	Some concerns
Strom, 2004 ⁴³	Some concerns	Some concerns	High	High	Low	Some concerns
Taylor, 2014 ⁴⁴	Low	Low	Low	High	Low	Some concerns
Vitiello, 2013 ⁴⁵	Low	Low	Low	Low	Low	Low
Yan-Yee Ho, 2014 ⁴⁶	Low	Low	Low	High	Low	Some concerns