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CLINICAL REVIEW

A meta-analysis of group cognitive behavioral therapy for insomnia

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SUMMARY

Insomnia is the most common sleep disorder among the general population. Although cognitive behavioral therapy for insomnia (CBT-I) is the psychological treatment of choice, the availability of individual therapy is often not sufficient to meet the demand for treatment. Group treatment can increase the efficiency of delivery, but its efficacy has not been well-established. Randomized controlled trials (RCTs) comparing group CBT-I to a control group in patients with insomnia were identified. A review of 670 unique citations resulted in eight studies that met criteria for analysis. Outcome variables included both qualitative (e.g., sleep quality) and quantitative (e.g., sleep diary) outcomes, as well as depression and pain severity, at both pre- to post-treatment and follow-up (3–12 mo post-treatment). Overall, we found medium to large effect sizes for sleep onset latency, sleep efficiency, and wake after sleep onset and small effect sizes for pain outcomes. Effect sizes remained significant at follow-up, suggesting that treatment gains persist over time. Other variables, including total sleep time, sleep quality, and depression, showed significant improvements, but these findings were limited to the within treatment group analyses. It is clear that group CBT-I is an efficacious treatment. Implications for stepped care models for insomnia are discussed.

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Background

Insomnia represents a serious public health concern. The estimated prevalence of insomnia among the general population ranges from 10 to 30%, and these numbers are even higher in patient populations, with an estimated prevalence of 69% among primary care patients [1–4]. In addition to the distress and impairment caused by insomnia, difficulties falling and staying asleep have been linked to the development of physical and psychological problems, including diabetes, cardiovascular disease, depression, and anxiety [5–9]. Both medication and behavioral therapies have been shown to be effective in treating insomnia [10,11]. Although medications tend to be more widely utilized, there are several risks associated with this treatment approach, including possible side-effects, dependence, and tolerance. Conversely, psychological treatments are less widely available but may provide more durable treatment gains without the associated risks of sleeping medications [11]. In addition, patients tend to prefer non-pharmacological treatments [12,13].

Cognitive behavioral therapy for insomnia (CBT-I) is a widely-used evidence-based treatment for insomnia. The basic components of CBT-I include: 1) sleep restriction, which involves limiting time in bed to consolidate sleep and increase the sleep drive; 2) stimulus control, which involves restricting the behaviors that occur in the bed/bedroom to sleep and sex and ensuring that protracted periods of wakefulness do not occur in the bed/bedroom so as to promote a strong association between sleep and sleep-related stimuli; and 3) cognitive restructuring, which addresses maladaptive thoughts and beliefs about sleep in order to decrease sleep-related anxiety. Systematic reviews have shown that CBT-I improves sleep as measured by diaries and polysomnography (e.g., shorter sleep latency, less time awake, higher sleep efficiency) and sleep as measured by questionnaires (e.g., more restful sleep, higher quality of sleep) [14–16]. Interestingly, there is also some evidence that CBT-I leads to modest improvements in physical and mental health symptoms, including reductions in depression, anxiety, and pain [14,17–19]. Although the exact mechanism driving this relationship is not known, it has been theorized that better sleep leads to improvements in emotional processing and affect [20,21], as well as an increased threshold for pain [19].

In the majority of CBT-I outcome studies, the treatment is delivered over the course of 5–8 sessions of individual therapy [10,11,14]. Unfortunately, this delivery method is untenable in many

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Glossary of terms

Cognitive behavioral therapy for insomnia (CBT-I)	therapy for insomnia utilizing behavioral and cognitive treatment components.
Analysis of Variance (ANOVA)	statistical test.
Randomized controlled trials (RCTs)	comparison of active treatment to control group.

settings, given the demands for treatment and the limited number of trained providers [11,22,23]. In recent years, a number of alternative methods of treatment delivery have been developed to make CBT-I more widely available. These include group therapy, self-administered therapy (including computerized CBT-I and applications for mobile devices), and delivery in classroom settings. Studies have also investigated reducing the number or duration of CBT-I sessions to increase access [24]. Brief behavioral sleep treatments, which have shorter treatment duration and focus on behavioral changes, have also been widely studied, particularly within general medical settings [25,26].

Often these alternative treatment modalities are placed within a framework of stepped care models. In these models, commonly conceptualized as a pyramid, the least intensive therapy (e.g., readily accessible, lowest cost, least personal inconvenience, least specialist time) is the entry step in the model, and progressively smaller volumes of patients move into more intensive treatment as needed [22,23]. As stepped care models are increasingly applied to insomnia, it will be essential to continue investigating the efficacy and durability of lower intensity interventions. It is important to note that within stepped care models, the efficacy of lower intensity interventions is not required or expected to equal more complex treatments; however, these interventions are required to provide health benefits to a considerable proportion of patients [22]. Ultimately, systematic reviews will be crucial for consolidating the findings from clinical outcomes studies to demonstrate that low intensity treatments provide significant improvement in sleep prior to their inclusion and implementation in stepped care models.

One of the most widely studied alterations of traditional CBT-I is group CBT-I, which has been proposed as a mid-level treatment in stepped care models [22]. Although a number of randomized controlled trials (RCTs) have been published comparing group CBT-I to control conditions, there is currently no systematic review summarizing the results from these trials. The goal of this paper is to utilize meta-analytic techniques to examine the efficacy of group CBT-I in patients with chronic insomnia. Insomnia has traditionally been classified as primary or secondary to a comorbid medical or mental health disorder, however, the utility and scientific basis of this distinction has been called into question [27]. Given that it is current practice to combine primary and secondary insomnia under the heading of insomnia disorder [28], this review includes insomnia diagnoses with and without co-existing medical and mental disorders. We did code for primary vs. secondary insomnia and included this distinction in the moderator analyses since it may be informative in regard to treatment efficacy.

To provide the most rigorous test of group CBT-I, we limited the review to RCTs and examined both sleep diary and questionnaire measures of sleep disturbance. Utilizing meta-analytic techniques provides a powerful estimate of the overall magnitude of treatment gains across patient populations and treatment conditions. In addition to sleep outcomes, we examined mental and physical health outcomes that were not directly targeted in the CBT-I treatment (e.g., depression, pain) when they were available. Follow-up data were included in our analyses to examine the durability of treatment gains over time. Finally, this meta-analytic approach allowed us to investigate potential moderators of treatment efficacy, including type of insomnia diagnosis (primary vs. secondary), location of recruitment (clinic vs. community), average duration of insomnia, use of sleeping medication, and length of treatment.

Method

Literature search

Relevant studies were obtained using several methods. First, literature searches were conducted on May 15th, 2013 in PsycINFO (Ovid Interface), PubMed and Scopus (including Embase citations) using the following keywords in various combinations: CBTI, CBT, cognitive behavioral therapy, insomnia, group. Full search strategies are presented in Appendix A. This search strategy yielded 231 citations from PubMed, 130 citations from PsycINFO, and 543 citations from Scopus. After removing duplicates, there were 670 unique citations, as shown in Fig. 1. The reference lists in the relevant empirical studies were reviewed to locate additional studies that may meet inclusion criteria; none were found.

Study selection and inclusion criteria

All abstracts obtained with the search described above were read to determine if they met inclusion criteria. Inclusion criteria included: 1) CBT-I treatment outcome study in which CBT-I was delivered in a group format of two or more patients, 2) randomized controlled trial in which CBT-I outcomes were compared to a control group, 3) reported outcomes for at least one measure of sleep with enough detail to calculate effect sizes, 4) written in English, 5) published by May 15th, 2013, 6) published in a peer-reviewed journal. In cases where there were multiple articles using data from the same sample, data from the most complete report were included. For this analysis, group CBT-I was defined as incorporating behavioral strategies (stimulus control, sleep restriction) and cognitive strategies (addressing dysfunctional beliefs about sleep) and involved more than one session. Our inclusion

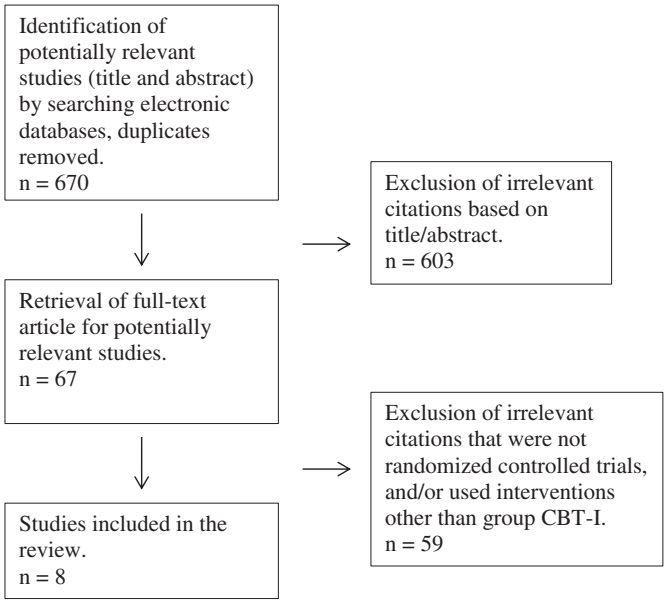

Fig. 1. Flowchart showing the process of selecting studies included in the review.

Table 1
Summary of study characteristics.

	Total N	Final N	Mean age (y)	% White	% Female	Sx criteria	Follow-up	Session number	Session length (min)	Tx manual	Control	Assess.	Quality score (%)
Currie et al. [30]	60	60*	45.0		55	Insom., chronic pain	3 mo	7	120	Yes	Diaries	Diary, PSQI, BDI, MPI-PS	23/26 (88.46)
Epstein & Dirksen [31]	81	72	58.2	85.2	100	Insom., cancer		4	60–120	Yes	Sleep edu.	Diary, sleep quality rating	23/26 (88.46)
Espie et al. [32]	201	201*	54.2		68.2	Insom.	6 mo	5	60	Yes	TAU	Diary, PSQI, SF PAIN	23/24 (95.83)
Jansson & Linton [29]	165	136	49.0		86.1	Insom.	12 mo	6	120	Yes	Sleep edu.	Sleep quality rating, HADS, pain rating	21/26 (80.77)
Miro et al. [33]	44	40	46.5		100	Insom., fibro.		6	90	Yes	Sleep edu.	PSQI, HADS, MPQ	23/26 (88.46)
Morin et al. [34]	24	24	67.1	91.6	70.8	Insom.	12 mo	8	90		WL	Diary	18/24 (75.00)
Rybarczyk et al. [35]	33	24	67.8	48.5	66.6	Insom., chronic illness	4 mo	8	90		WL	Diary, PSQI	22/24 (91.66)
Vitiello et al. [19]	51	51	67.7	64.7	88.2	Insom., arthritis	12 mo	8	120		Pain edu.	Diary, GDS, MPQ	21/26 (80.77)

*intent to treat. Assess = assessment. BDI = Beck depression inventory. Edu = education. Fibro = fibromyalgia. GDS = geriatric depression scale. HADS = hospital anxiety and depression scale. Insom = insomnia. MPI-PS = multidimensional pain inventory pain severity scale. MPQ = McGill pain questionnaire. SQI = Pittsburgh sleep quality index. SF-PAIN = SF-36 pain scale. Sx = symptom. TAU = treatment as usual. Tx = treatment. WL = wait list. . Blank spaces indicate that the information was not reported in the article.

criteria focused on the content of the session, requiring both behavioral and cognitive treatment elements, rather than treatment length. Length of treatment was examined as a potential moderator. The control group was defined as a condition that did not actively target the symptoms of insomnia (e.g., wait list, treatment as usual, placebo). Post-treatment was defined as data collected at two weeks or less following the last treatment session, and follow-up was defined as data collected after more than two weeks post-treatment. As shown in Fig. 1, 603 articles were deemed irrelevant after abstract review and 67 articles were obtained in full. Of these, 59 articles failed to meet the inclusion criteria due to not using an RCT design or not using group CBT-I as a treatment condition. The selection criteria resulted in a final set of eight randomized controlled studies (see Table 1) [19,29–35].

Study coding

The studies were coded for descriptive study information (title, authors, year), sample information (age, race, sex, inclusion criteria for the study, location of recruitment, type of insomnia diagnosis, average length of insomnia diagnosis, percentage of sample using sleeping medication), research design information (type of assignment to conditions, equivalence of groups, sample sizes, follow-up time period), treatment information (treatment duration, session duration, manual use, nature of comparison group), outcomes information (measures of sleep outcomes and non-sleep-related symptoms), and quantitative data necessary for calculating effect sizes (means and standard deviations for measures pre- and post-treatment in treatment and control groups).

All studies were coded independently by two study authors (EK & JK). This resulted in 91.3% agreement for study information and 99.5% agreement for outcomes information. Coding discrepancies were reviewed and agreed upon by consensus. Post-treatment data was reported for a total of 608 participants. For the majority of studies, post-treatment outcomes were limited to treatment completers. Two studies, Currie et al. [30] and Espie et al., [32] followed a more conservative intention-to-treat model and included outcome data for all participants who provided baseline data in their analyses.

The sleep inclusion criteria for all participants were derived from the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)* [36] and the *International Classification of Sleep Disorders (ICSD)* [37] criteria for insomnia, including both primary and secondary insomnia diagnoses. As these trials were conducted prior to the publication of the *DSM-5* [28], the primary/secondary distinction was still in use. Three studies required a primary insomnia diagnosis, whereas the remainder included insomnia comorbid with medical conditions or both primary and secondary insomnia diagnoses (62.5%). It is important to note that none of the RCTs included insomnia comorbid with a mental health condition, although half of the studies did include depression and anxiety outcome measures.

All the studies in this review were RCTs, which minimized bias; however, the control conditions did vary among studies, with some providing more robust tests of treatment efficacy than others. Five of the eight studies had an active control condition (e.g., education, monitoring sleep with diaries), whereas the remaining studies used wait list control and one used treatment as usual. None of the studies explicitly stated that they blinded therapists or patients; most likely the nature of the treatment precluded therapist blinding. One concern with treatment as usual is that patients may obtain active sleep treatments, attenuating group differences. In the treatment as usual control in Espie et al., [32] general practitioners continued to meet with patients and manage prescriptions; given that this study took place within a primary care setting, it is unlikely that these patients received behaviorally-based sleep treatments. In addition, the treatment group continued to receive

treatment as usual from their general practitioners, which makes it unlikely that group differences were attenuated. Only one study (Morin et al. [34]) excluded participants based on psychotropic medication usage. The remainder did not explicitly exclude participants who were on medications as long as dosages were stable and not above recommended guidelines.

All studies reported sleep outcome data; four studies included both sleep diaries and questionnaire measures of sleep outcome, whereas two were restricted to sleep diaries and two were restricted to sleep quality measures. In addition to measures of sleep, four studies included measures of depression or anxiety symptoms and five studies included measures of pain. Six studies collected data at follow-up. The follow-up time periods ranged from 3 to 12 mo. The average attrition from randomization to post-treatment in the seven studies that collected post-treatment data was 9.72%. The average attrition in the treatment condition was 10.32%.

Standardized quality scoring criteria for quantitative studies was applied in order to provide a comparative assessment of the risk of bias across the studies included in this review [38]. Fourteen items referring to study design and analyses (e.g., randomization, blinding, control of confounding variables) were summed to provide a total score for each study, and these scores, along with percentages of total scores, are listed in Table 1 (see Appendix B for a description of each item and scores for each study). In general, the RCTs included in this review had high global scores, with percentages ranging from 75.00 to 95.83.

Effect size calculation

Procedures and formulas for conducting the meta-analysis were based on recommendations by Lipsey and Wilson [39]. Within group effect sizes were calculated for pre-treatment vs. post-treatment and pre-treatment vs. follow-up using the following formula,

$$ES_{sg} = \frac{\overline{X}_{T2} - \overline{X}_{T1}}{s_p},$$

where $\overline{X}$ is the sample mean at each timepoint and the pooled standard deviation was defined as

$$s_p = \sqrt{(s_{T1}^2 + s_{T2}^2)/2},$$

where S^2 is the sample variance and the sample size bias adjustment was defined as

$$w_{sg} = \frac{2n}{4(1-r) + ES_{sg}^2},$$

and where sg denotes standardized gain, *T*2 denotes post-treatment values, *T*1 denotes pre-treatment values, and *r* is the correlation between Time 1 and Time 2 scores. Since none of the studies included the correlation between Time 1 and Time 2 scores, test–retest correlations from validation papers for each measure were used as is recommended by Lipsey and Wilson [39]. They recommend this practice since the mean effect size estimate is robust to modest variations in weights (and by extension, modest variations in the correlations that are used to calculate these weights) and conclude that reasonable estimates of this correlation, including test–retest correlations from validation papers, are appropriate.

Effect sizes were calculated for experimental vs. control groups at both post-treatment and follow-up time points, using the following formula for unbiased effect size estimates,

$$ES'_{sm} = \left[1 - \frac{3}{4N - 9}\right] ES_{sm},$$

where the uncorrected standardized mean difference was defined as

$$ES_{sm} = \frac{\overline{X}_{G1} - \overline{X}_{G2}}{s_p},$$

the pooled standard deviation was defined as

$$s_p = \sqrt{(n_{G1} - 1)s2_{G1} + (n_{G2} - 1)s2_{G2}/(n_{G1} - 1) + (n_{G2} - 1)},$$

and the sample size bias adjustment was defined as

$$w_{sm} = \frac{2n_{G1}n_{G2}(n_{G1} + n_{G2})}{2(n_{G1} + n_{G2})^2 + n_{G1}n_{G2}(ES'_{sm})^2},$$

where *N* denotes the total sample size, n_{G1} denotes the number of subjects in the treatment group and n_{G2} denotes the number of subjects in the control group.

The following constructs were included in this study: sleep onset latency, total sleep time, sleep efficiency, wake after sleep onset, sleep quality, depression severity and pain severity (anxiety measures were obtained in two studies and only one of these included follow-up; given the lack of data, this construct was not included). Each sample contributed only one effect size per construct to ensure statistical independence.

Data analysis

Mean effect sizes for each construct, 95% confidence intervals, and *z*-scores were computed using recommendations by Lipsey and Wilson [39]. The mean effect size for each construct was calculated by weighting each effect size by the inverse of its variance. Based on the guidelines provided by Lipsey and Wilson [39], effect sizes ranging from .20 to .49 were considered small, .50 to .79 were considered medium, and .80 and above were considered large. Homogeneity analyses were conducted using the *Q* statistic to determine if the variability of effect sizes around their means is no greater than what would be expected from sampling error alone.

Given that this meta-analysis was based solely on published journal articles that report significant findings, publication bias is a concern. Effect sizes may be inflated due to the exclusion of non-significant results that may be more likely to be reported in dissertations, abstracts, or unpublished manuscripts. We investigated potential upward bias of the mean effect size in this study by calculating fail-safe Ns, which indicate the number of studies with an effect size of zero needed to reduce the mean effect size to a negligible magnitude. Orwin's formula for fail-safe N [40] was used,

$$k_0 = k\left[\frac{\overline{ES}_k}{\overline{ES}_c} - 1\right],$$

where *k* is the number of studies contributing to the effect size, $\overline{ES}_k$ is the observed mean effect size, and $\overline{ES}_c$ is the criterion effect size. We used the recommended criterion effect sizes of .20. Publication bias was also examined through funnel plots of sample size by effect size. Follow-up moderator analyses were performed. Fixed effects models were used to evaluate the effect of treatment and sample characteristics on outcome measures. We used the between groups *Q*-statistic to examine moderating categorical variables, which is an analog to the analysis of variance (ANOVA). Two categorical moderating variables were examined: recruitment location (clinic recruitment, in which patients were recruited within a medical setting vs. community recruitment, in which patients were recruited using general advertisements within the community, such as newspaper ads) and insomnia diagnosis (primary vs. secondary diagnosis). Weighted least squares regression was used to examine continuous moderating

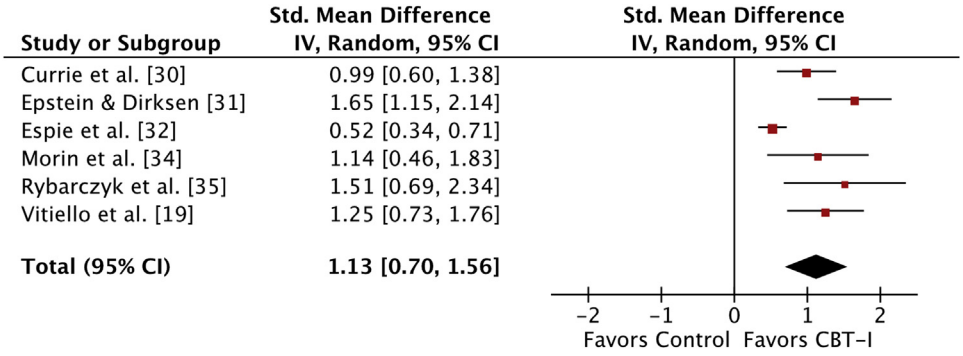

Fig. 2. Post-treatment effect size statistics for sleep efficiency. Individual study effect sizes are unweighted. Total value is based on the random effects model. Std = standardized. IV = inverse variance. Random = random effects model. CI = confidence interval. CBT-I = cognitive behavioral therapy for insomnia.

variables, including duration of insomnia, percentage of sample using sleeping medication, and length of treatment. It is important to note that these analyses are exploratory and should be interpreted with caution given the modest number of studies included in each analysis, with the number of studies ranging from 5 to 6. These analyses were conducted with the most commonly reported outcome variables, including sleep onset latency, sleep efficiency, wake after sleep onset, and total sleep time.

Results

For the within group analyses comparing pre- to post-treatment outcomes, six independent effect sizes were available for sleep diary variables. These outcomes variables included total sleep time, sleep onset latency, time awake after sleep onset, and sleep efficiency. There were five independent effect sizes for these outcome variables comparing pre-treatment to follow-up scores. In addition, there were five effect sizes for sleep quality pre- to post-treatment and three for pre-treatment to follow-up comparisons. There were three effect sizes for depression symptoms for both pre- to post-treatment and follow-up comparisons. Finally, there were four effect sizes for pain measures pre- to post-treatment and three effect sizes for follow-up comparisons. For the between group analyses comparing outcomes in the treatment and control groups, the same number of effect sizes for each outcome variable were available, however, there were fewer effect sizes for the comparison of groups at follow-up. The number of independent effect sizes ranged from two to three.

Homogeneity analyses indicated that there was significant heterogeneity in both the within- and between-group effect sizes. Specifically, for effect sizes based on pre- to post-treatment outcomes, $Q(5) = 27.84$, $p < .05$ for sleep efficiency, $Q(4) = 16.61$, $p < .05$ for sleep quality, and $Q(5) = 14.42$, $p < .05$ for wake after sleep onset. For effect sizes based on pre- to follow-up outcomes, $Q(4) = 23.04$, $p < .05$ for wake after sleep onset, $Q(4) = 23.01$, $p < .05$ for total sleep time, $Q(4) = 18.56$, $p < .05$ for sleep efficiency, and $Q(2) = 13.47$, $p < .05$ for depression symptoms. For effect sizes based on between group outcomes at post-treatment, $Q(4) = 22.41$, $p < .05$ for sleep quality, $Q(5) = 21.56$, $p < .05$ for sleep efficiency, $Q(5) = 15.69$, $p < .05$ for wake after sleep onset, and $Q(3) = 9.55$, $p < .05$ for pain. For effect sizes based on between group outcomes at follow-up, $Q(2) = 49.23$, $p < .05$ for sleep quality. These results suggest that variability among effect sizes is greater than what would be expected based on subject-level sampling error and led us to conclude that a fixed effects model is not justified. A random effects model was utilized in order to model random variability at the study- and subject-level.

Figs. 2–5 present the within group post-treatment effect size statistics for each of the studies included in the meta-analysis for several representative outcomes measures, including sleep efficiency, sleep quality, depression, and pain.

These figures also include the combined effect size based on the random effects models. It is important to note that the combined effect sizes are all significant. In particular, the sleep outcome variables show large effect sizes post-treatment (mean effect size of 1.13 for sleep efficiency and .85 for sleep quality).

Table 2 presents the mean change in sleep diary data from pre- to post-treatment and pre-treatment to follow-up in the CBT-I treatment groups. At baseline, the treatment groups were taking an average of 52.75 min to fall asleep and spending and average of 77.54 min awake after sleep onset. All variables improved in the expected directions, with reductions in sleep onset latency and wake after sleep onset and increases in sleep efficiency and total sleep time. The greatest improvements post-treatment were seen for sleep onset latency (51.66% improvement) and wake after sleep onset (53.37% improvement). This pattern was replicated at follow-up, although percentage of improvement was generally less.

Within group analyses are presented in Table 3, which includes the mean effect sizes for the treatment groups at both immediate post-treatment and follow-up for all outcomes measures. The mean effect sizes for all outcomes at both post-treatment and follow-up were statistically significant. At post-treatment, there were medium to large effect sizes for sleep onset latency, sleep efficiency, wake after sleep onset, and sleep quality and small effect sizes for total sleep time, depression, and pain. All effect sizes maintained at follow-up, with the exception of wake after sleep onset, which decreased from a large to medium effect size. Interestingly, several effect sizes were larger at follow-up compared to post-treatment, including total sleep time (mean effect size = .60 compared to .29), sleep quality (mean effect size = 1.26 compared to .85), depression (mean effect size = .32 compared to .26), and pain (mean effect size = .41 compared to .25). The remaining effect sizes decreased slightly in relative value, but continued to be within the medium to large range. The fail-safe N was within an acceptable range for most of these outcomes. The exceptions were total sleep time at post-treatment (fail-safe N = 3) and depression and pain at post-treatment and follow-up (fail-safe Ns range from <1 to 3). Publication bias was undetectable from funnel plots for each outcome.

Table 4 presents the mean effect sizes for the between groups analyses at post-treatment and follow-up. Three outcome measures reached significance at post treatment: sleep onset latency, sleep efficiency, and wake after sleep onset. The largest mean effect sizes were for sleep efficiency (mean effect size = .84) and wake after sleep onset post-treatment (mean effect size = .65). At follow-up, the effect sizes for these three outcome measures remained significant, although sleep efficiency was reduced from large to small, and wake after sleep onset was reduced from medium to small. Pain was a significant outcome variable at follow-up. The fail-safe N indicated that publication bias was unlikely to be a concern for most of these

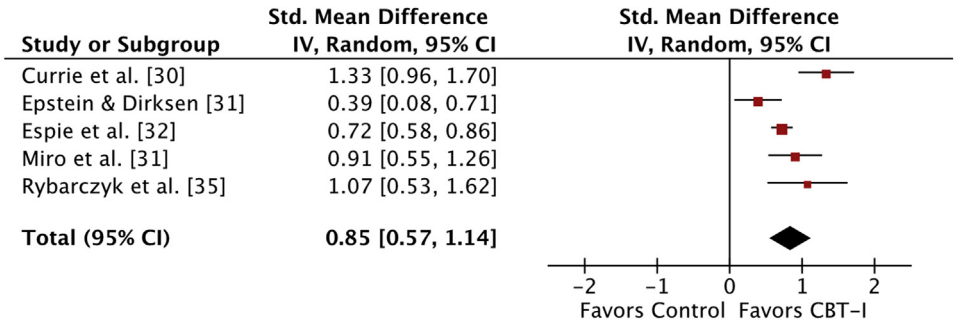

Fig. 3. Post-treatment effect size statistics for sleep quality. Individual study effect sizes are unweighted. Total value is based on the random effects model. Std = standardized. IV = inverse variance. Random = random effects model. CI = confidence interval. CBT-I = cognitive behavioral therapy for insomnia.

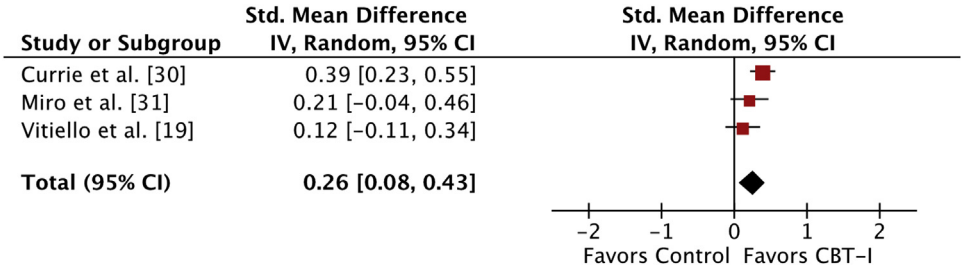

Fig. 4. Post-treatment effect size statistics for depression. Individual study effect sizes are unweighted. Total value is based on the random effects model. Std = standardized. IV = inverse variance. Random = random effects model. CI = confidence interval. CBT-I = cognitive behavioral therapy for insomnia.

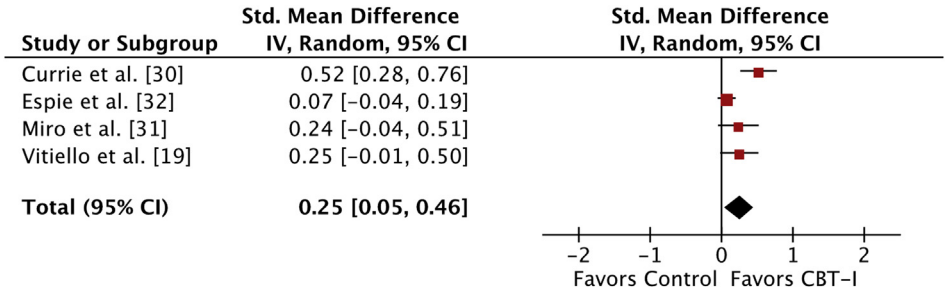

Fig. 5. Post-treatment effect size statistics for pain. Individual study effect sizes are unweighted. Total value is based on the random effects model. Std = standardized. IV = inverse variance. Random = random effects model. CI = confidence interval. CBT-I = cognitive behavioral therapy for insomnia.

Table 2
Pre- to post-treatment and follow-up change in sleep variables.

Outcome	Pre-treatment			Post-treatment			Pre-treatment			Follow-up		
	K	Mean	SD	Mean	SD	Change (%)	*K*	Mean	SD	Mean	SD	Change (%)
SOL	6	52.75	39.62	25.50	20.24	−27.25 (51.66)	5	49.66	37.29	29.98	24.94	−19.68 (39.63)
SE	6	69.51	14.87	83.32	9.16	13.81 (19.87)	5	68.77	14.33	79.96	11.82	11.19 (16.27)
WASO	6	77.54	60.52	36.16	29.28	−41.38 (53.37)	5	86.79	59.06	53.78	49.09	−33.01 (38.03)
TST	6	340.18	75.02	358.98	58.40	18.80 (5.53)	5	331.55	78.85	374.46	66.88	42.91 (12.94)

SOL = sleep onset latency. SE = sleep efficiency. WASO = wake after sleep onset. TST = total sleep time.

outcomes at post-treatment, although the values for follow-up were low (fail-safe Ns ranged from 2 to 4).

Table 5 presents the test statistics for the moderating variables. Significant moderating effects were found for recruitment location for all sleep outcome variables, with the exception of sleep onset latency, such that improvement in sleep was greater among samples recruited from the community. In addition, significant moderating effects were found for diagnosis, such that improvement in all sleep variables was greater among samples with secondary insomnia vs. primary insomnia. In examining continuous variables, years of insomnia diagnosis was inversely related to improvements in sleep efficiency, wake after sleep onset, and total sleep time. Similarly, percentage of the sample taking sleeping medication was inversely related to improvements in sleep efficiency and wake after sleep onset. As mentioned earlier, all but one study allowed concurrent treatment with psychotropic medications. Finally, minutes of treatment was positively related to improvements in sleep onset latency, sleep efficiency, and wake after sleep onset.

Discussion

It is well-documented that CBT-I is an effective treatment for insomnia that results in durable improvements in sleep. The high prevalence of insomnia and the limited number of trained practitioners has spurred the development of alternative, lower-intensity delivery methods for CBT-I. Although reducing resource intensity has the potential to dramatically increase access to sleep

Table 3
Mean effect sizes for within treatment group analyses.

Outcome	Post-treatment					Follow-up				
	K	ES	95% CI	*Z*	Fail-safe N	*K*	ES	95% CI	*Z*	Fail-safe N
SOL	6	.77	.54, .99	6.69**	17	5	.60	.39, .81	5.64**	10
SE	6	1.13	.70, 1.56	5.18**	28	5	.85	.51, 1.19	4.86**	16
WASO	6	.89	.60, 1.17	6.09**	21	5	.63	.28, .98	3.56**	11
TST	6	.29	.11, .46	3.26**	3	5	.60	.25, .95	3.40**	10
Quality	5	.85	.57, 1.13	5.90**	16	3	1.26	1.01, 1.53	9.63**	16
Depression	3	.26	.08, .43	2.90**	1	3	.32	.05, .60	2.31*	2
Pain	4	.25	.05, .46	2.41*	<1	3	.41	.24, .58	4.83**	3

*$p < .05$. **$p < .01$. SOL = sleep onset latency. SE = sleep efficiency. WASO = wake after sleep onset. TST = total sleep time. Quality = sleep quality. Depression = depression symptoms. Pain = pain severity. *K* = total number of effect sizes. ES = effect Size. CI = confidence interval.

Table 4
Mean effect sizes for between group analyses.

Outcome	Post-treatment					Follow-up				
	K	ES	95% CI	*Z*	Fail-safe N	*K*	ES	95% CI	*Z*	Fail-safe N
SOL	6	.47	.27, .66	4.75**	8	3	.45	.16, .74	3.02**	4
SE	6	.84	.38, 1.31	3.55**	19	3	.48	.14, .82	2.79**	4
WASO	6	.65	.26, 1.04	3.28**	14	3	.39	.05, .73	2.23*	3
TST	6	−.04	−.32, .23	−.31	–	3	.24	−.05, .53	1.61	–
Quality	5	.40	−.14, .93	1.46	–	3	.55	−.53, 1.64	1.00	–
Depression	3	.22	−.10, .55	1.31	–	2	.23	−.05, .51	1.58	–
Pain	4	.35	−.05, .75	1.70	–	2	.40	.12, .69	2.80**	2

*$p < .05$. **$p < .01$. SOL = sleep onset latency. SE = sleep efficiency. WASO = wake after sleep onset. TST = total sleep time. Quality = sleep quality. Depression = depression symptoms. Pain = pain severity. *K* = total number of effect sizes. ES = effect Size. CI = confidence interval. Dashes indicate that the Fail-safe N was not calculated due to non-significant findings.

treatments, it is important to demonstrate that these alternative treatments provide significant, lasting improvements in sleep before they are incorporated into stepped care models. A number of clinical outcome studies, including RCTs, have been published in the last several years for group CBT-I. The goal of this meta-analysis was to summarize the findings from RCTs involving group CBT-I. We report average treatment gains both pre- to post-treatment and relative to control groups.

Participants receiving group CBT-I showed substantial improvements across a range of sleep variables, including sleep onset latency, sleep efficiency, and wake after sleep onset (average effect sizes ranging from .77 to 1.13 pre- to post-treatment). A smaller effect was seen for total sleep time pre- to post-treatment (mean effect size = .29), which is consistent with the CBT-I literature [14–16]. Beyond these measures of sleep continuity and duration, it is also important to examine whether patients are reporting qualitatively better sleep; that is, if they feel that their sleep has improved. Across studies, patients in CBT-I groups report large improvements in sleep quality pre- to post-treatment (mean effect size = .85). Regarding non-sleep-related symptoms, group CBT-I resulted in a small but significant improvement in depression symptoms pre- to post-treatment (mean effect size = .26, $p < .01$) and pain pre- to post-treatment (mean effect size = .25, $p < .05$), even though these symptoms were not specifically targeted in treatment. This is consistent with theories that improvements in sleep may result in enhanced emotional processing and affect regulation [20,21], as well as an increase in the pain threshold [19]. However, given the relatively small effect sizes and the small fail-safe N values, these findings should be interpreted with caution.

An important consideration for behavioral sleep treatments, particularly those that are less intensive such as group CBT-I, is whether improvements in sleep are maintained when patients are no longer actively engaged in treatment. Durability of treatment gains is often described as a major advantage of behavioral treatments over pharmacological treatments of insomnia. Regarding group CBT-I, treatment outcomes for all variables continued to be significant at follow-up. The average effect sizes ranged from medium to large for sleep onset latency, sleep efficiency, wake after sleep onset, total sleep time, and sleep quality. As would be expected, effect sizes for most quantitative sleep variables were smaller at follow-up than at post-treatment. In particular, wake after sleep onset decreased from a large effect size (.89) to a medium effect size (.63). Conversely, total sleep time increased from a small effect size (.29) to a medium effect size (.60). Effect sizes also increased for sleep quality (mean effect size increased from .85 to 1.26), depression (mean effect size increased from .26 to .32), and pain (mean effect size increased from .25 to .41).

These findings of greater improvement at follow-up, which have been reported in other systematic reviews of individual CBT-I [14,15], suggest that some aspects of insomnia continue to show improvement over time following completion of group CBT-I. The continued improvement in total sleep time is most likely related to the techniques used in CBT-I, including sleep restriction and stimulus control. The goal of these interventions is to initially limit sleep opportunities to increase the drive for sleep and ultimately improve homeostatic regulation of sleep [10]. Sleep opportunity is increased only after sleep efficiency is improved and patients have consolidated their sleep during the night. As a result, one would expect more immediate improvements in the sleep variables linked to homeostatic regulation

Table 5
Variables potentially moderating the post-treatment sleep outcomes.

	Q_B/Q_R			
	SOL	SE	WASO	TST
Recruitment location (clinic vs. community)	1.05	17.08**	10.24**	4.60*
Diagnosis (primary vs. secondary)	5.93*	20.29**	8.11**	3.87*
Years of insomnia	3.50	18.31**	7.02**	4.70*
% Sleeping medication	.70	4.41*	6.63**	.30
Minutes of treatment	5.17*	8.14**	4.91*	.04

*$p < .05$. **$p < .01$. First two outcomes are categorical and moderating effect was calculated using the Q_B statistic, remaining outcomes are continuous and utilize the Q_R statistic. Q_B = between-groups homogeneity statistic for analog ANVOA. Q_R = Between-groups homogeneity statistic for the regression model. SOL = sleep onset latency. SE = sleep efficiency. WASO = wake after sleep onset. TST = total sleep time.

of sleep, including sleep onset latency, sleep efficiency and wake after sleep onset. Other variables, including total sleep time, may show greater improvements over time as participants continue to practice these skills and gradually increase their opportunity for sleep. Similarly, patients may experience greater reductions in daytime symptoms, including depression and pain, after they have experienced several months of a regularized sleep–wake cycle. This suggests that, while CBT-I has the advantage of being a brief treatment compared to most other applications of CBT, the short treatment duration may not be long enough to see the full benefit that patients get from using the techniques.

As a more rigorous test of CBT-I efficacy, we examined post-treatment and follow-up outcomes of the treatment group compared to the control group. Although effect sizes for these analyses tended to be smaller than the within group analyses, significant effect sizes were found for sleep onset latency, sleep efficiency, and wake after sleep onset at both post-treatment (mean effect sizes ranged from .47 to .84) and follow-up (mean effect sizes ranged from .39 to .48). Paralleling the within group analyses, effect sizes for these variables decreased at follow-up, in particular for sleep efficiency, which decreased from a large to a small effect size (.84–.48). The mean effect size for pain was small but significant at follow up. The mean effect sizes for the remaining variables did not reach significance at post-treatment or follow-up. This differs from the within group analyses and suggests that more research is needed regarding the degree of change in these outcomes following group CBT-I. Although the findings of significant improvements in sleep and pain are encouraging, the magnitude of these between group findings is modest and will need to be replicated, particularly given the potential concern of sampling bias at follow-up indicated by the fail-safe Ns.

Tests of moderation suggest that sample and treatment characteristics are related to the degree of improvement in sleep variables. Samples that were recruited from the community via advertisements, had secondary insomnia diagnoses, were taking less sleeping medication, and had shorter duration of insomnia diagnoses tended to show greater improvements in sleep variables post-treatment. Many of these variables are conceivably related to chronicity of sleep disturbance, suggesting that those entering treatment with less severe sleep disturbances tend to do better in treatment. It is less clear why samples with secondary insomnia diagnoses tended to do better, though it may be that their sleep disturbances were less severe than samples that were recruited on the basis of sleep problems alone. In addition, longer treatments tended to produce greater improvements in sleep. Many of these findings have been reported previously, particularly those indicating greater improvement with more therapy time and greater improvement among drug-free samples and those with shorter insomnia duration [15,16]. The significant moderation effects have implications for matching patients to treatment levels in stepped care models of behavioral sleep treatments. Although this review supports the interpretation that group CBT-I is generally an efficacious treatment, it does appear that this treatment modality provides the most benefit to patients with less complicated clinical presentations (i.e., patients recruited in the community who present with less chronic sleep complaints), although the same can be said for CBT-I delivered on an individual basis.

Overall, it is clear from these analyses that group CBT-I is an efficacious treatment for insomnia, particularly in regards to sleep diary variables such as sleep onset latency, sleep efficiency, and wake after sleep onset. There is also some suggestion that this treatment modality leads to improvements in total sleep time, sleep quality and symptoms of depression and pain, although these findings are not as robust. Given the benefits associated with psychological therapies for insomnia, including patient preference, reduced risk of side-effects and durability of treatment gains, it is encouraging that CBT-I remains efficacious when delivered in a group format. Group delivery may be necessary in settings where demand for this treatment is greater than can be accommodated in individual therapy. This review suggests that group CBT-I provides significant health gain, which is the primary criterion required to be included in stepped care models of insomnia [22,23]. Group CBT-I may also provide benefits not available in more intensive therapies, including social support at a time when patients are being asked to make challenging behavioral changes (e.g., moving bedtimes later, eliminating napping, getting out of bed when they can't sleep). Research has suggested that patients rate "meeting with other people with insomnia" as a helpful component of treatment [41].

Limitations and future directions

Although these analyses demonstrate that group CBT-I is an efficacious treatment, it was not possible to directly compare group CBT-I to individual CBT-I or to other treatment modalities (e.g., computerized CBT-I) given the limited number of published RCTs. Several meta-analyses have conducted moderator analyses examining the effect of treatment modality on outcomes, with mixed results. One meta-analysis of psychological treatments for insomnia suggests that individual, group and self-help treatments were equally effective, [16] whereas another indicates that individual treatment yields the greatest effect size, followed by group and then self-administered treatment [15]. Of the three studies that have directly compared individual and group CBT-I, two studies concluded that both forms of CBT-I are equally effective at improving sleep and found no group differences on qualitative or quantitative sleep outcomes [41,42]. The third study reported that individual CBT-I resulted in greater improvement on several sleep variables compared to group CBT-I, including sleep onset latency and overall sleep quality [43]. Table 6 compares effect sizes from this study with those from the Smith et al. (2002) [10] meta-analysis investigating behavioral therapy for insomnia. The majority of effect sizes in both studies, with the exception of total sleep time, fall within the medium to large range. As would be expected, group CBT-I does appear to have smaller effect sizes as a less intensive therapy; this is particularly evident with sleep onset latency, which demonstrated a large effect size (1.05) in the Smith et al. (2002) [10] review and a medium effect size in the current review (.77).

Perhaps a more important question is whether comparing the efficacy of less intensive and more intensive treatments is meaningful within a clinical, treatment implementation context. For the purposes of stepped care models, demonstrating relative efficacy may not be as helpful as examining who does and does not benefit from various levels of care; this type of research can be used to develop guidelines for allocating or transferring patients between levels of care based on individual characteristics (e.g., demographics, physical and mental comorbidities, treatment history, personality/temperament factors, motivation for treatment) [22].

There are several additional limitations to the current analyses that should be mentioned. First, we limited the studies included in this review to RCTs. Although this provides a rigorous test of group CBT-I, it does exclude a number of smaller, treatment outcome studies and results in a restricted range of participants. This makes it difficult to generalize the results across different patient populations. For example, participants in the trials included in this study were limited to those with primary insomnia or insomnia secondary to a medical condition (e.g., cancer, arthritis, chronic pain). None of the studies included participants with insomnia secondary to a psychiatric disorder. Previous studies have suggested that the treatment of sleep problems reduces symptoms of depression and anxiety [14,17,18,44], and the analyses in this study suggest some reduction pre- to post-treatment on depression

Table 6
Pre–post treatment mean effect size for behavioral therapy for insomnia (Smith et al., 2002) vs. group cognitive behavioral therapy for insomnia (current study).

Outcome	Smith et al. (2002)	Current study
SOL	1.05	.77
WASO	1.03	.89
TST	.46	.29
Quality	1.44	.85

SOL = sleep onset latency. WASO = wake after sleep onset. TST = total sleep time. Quality = sleep quality.

measures. Actively recruiting participants with psychiatric diagnoses will be important for future RCTs in order to determine if targeting sleep symptoms improves general mental health.

It is also important to note that the majority of participants included in the trials in this study were older Caucasian females (average age across trials ranged from 45 to 67.8). The findings will need to be replicated with non-white samples across a wider variety of age groups. Finally, although several studies in this review included objective sleep data, there were too few to calculate reliable mean effect sizes. Since outcome data was limited to subjective sleep variables, it may reflect some reporting bias, particularly among people suffering from insomnia [45,46]. Although some studies have failed to find improvement in subjective outcomes following CBT-I treatment, it is generally acknowledged that there are clinically significant improvements in objectively and subjectively measured sleep variables following sleep treatment [34]. One recent meta-analysis did find that objective sleep outcomes show significant although less robust improvements compared to subjective outcomes following behavioral treatments for sleep [14].

This study joins a growing body of literature suggesting that alternative modalities of CBT-I lead to significant improvements in sleep continuity and quality, and that these treatment gains are quite durable over time. Importantly, this study summarized data across RCTs in order to provide a robust test of the efficacy of group CBT-I. This delivery format may be particularly useful in settings in which individual therapy is not a viable treatment option due to limited providers or high patient demand. Within stepped care models, group CBT-I clearly meets the minimum criteria of providing benefit to a substantial portion of patients with insomnia and could be conceptualized as a mid-level treatment since it demands less resources than individual CBT-I but does require some practitioner involvement [22].

As healthcare systems continue to adapt insomnia treatment to meet the growing demand, it is crucial for researchers to continue investigating group CBT-I and for practitioners to consider incorporating it into clinical practice as a mid-level treatment option. It will be important for group CBT-I studies to continue investigating patient characteristics to determine who does and does not benefit from this particular form of treatment. Ultimately this information can be used to provide guidelines for entry into and movement between treatment levels.

Practice points

- Group CBT-I has a medium to large effect in improving sleep outcomes pre- to post-treatment, including sleep quality, sleep onset latency, sleep efficiency and wake after sleep onset.
- This treatment format leads to significant improvements in sleep outcomes compared to control conditions.
- Treatment gains are maintained, and in some cases augmented, over time.
- There is some indication that group CBT-I leads to improvement in non-sleep-related outcomes like depression and pain, but further research is needed.

Research agenda

- There is a need to conduct more studies to match patient characteristics to treatment modalities.
- Future studies should include non-sleep-related outcome measures, including depression, anxiety and pain as well as objective measures of sleep (e.g., actigraphy).
- It will be necessary to examine sleep and mental health outcomes in samples of patients with insomnia and comorbid mental health conditions who participate in group CBT-I.

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Appendix A

PubMed (1946 - May 15th, 2013):

(MeSH terms automatically mapped to keywords)

1). group
2). (cognitive behavioral therapy OR CBT) AND insomnia)
3). CBTI
4). English[lang]
5). 1 and 4 and (2 or 3)

PsycINFO (1806 - May Week 2, 2013):

1). CBTI.mp.
2). CBTI-I.mp.
3). 1 or 2
4). exp Cognitive Therapy/or exp Cognitive Behavior Therapy/
5). CBT.mp.
6). cognitive behavio?ral therapy.mp.
7). 4 or 5 or 6
8). insomnia.mp.
9). group.mp.
10). 3 or (7 and 8)
11). 9 and 10

Scopus (1960 - May 15th, 2013):

1). KEY(“cognitive therapy” OR “behavior therapy”)
2). TITLE-ABS-KEY(cbt)
3). TITLE-ABS-KEY(“cognitive behavioral therapy”)
4). 1 or 2 or 3
5). KEY(insomnia)
6). TITLE-ABS-KEY(insomnia)
7). 5 or 6
8). TITLE-ABS-KEY(cbti)
9). TITLE-ABS-KEY(group)
10). (4 and 7) or 8
11). 10 and 9

Appendix B

	1. Question or objective sufficiently described	2. Design evident and appropriate to answer study question	3. Method of subject selection described and appropriate	4. Subject characteristics sufficiently described	5. If random allocation to treatment group was possible, it is described	6. If blinding of investigators was possible, it is reported	7. If blinding of subjects was possible, it is reported	8. Outcome well defined and robust/ assessment reported	9. Sample size appropriate	10. Analysis described and appropriate	11. Estimate of variance reported for main outcomes	12. Controlled for confounding	13. Results reported in sufficient detail	14. Results support conclusions
Currie et al. [30]	Yes	Yes	Yes	Yes	Yes	NA	No	Yes	Yes	Yes	Partial	Yes	Yes	Yes
Epstein & Dirksen [31]	Yes	Yes	Yes	Yes	Yes	NA	No	Yes	Yes	Yes	Partial	Yes	Yes	Yes
Espie et al. [32]	Yes	Yes	Yes	Yes	Yes	NA	NA	Yes	Yes	Yes	Partial	Yes	Yes	Yes
Jansson & Linton [29]	Yes	Yes	Partial	Partial	Yes	NA	No	Yes	Yes	Yes	Partial	Yes	Yes	Yes
Miro et al. [33]	Yes	Yes	Yes	Yes	Yes	NA	No	Yes	Yes	Yes	Partial	Yes	Yes	Yes
Morin et al. [34]	Yes	Yes	Partial	Partial	Partial	NA	NA	Yes	Yes	Yes	Partial	Yes	Yes	Yes
Rybarczyk et al. [35]	Yes	Yes	Yes	Yes	Partial	NA	NA	Yes	Yes	Yes	Partial	Yes	Yes	Yes
Vitiello et al. [19]	Yes	Yes	Yes	Yes	Partial	NA	No	Yes	Yes	Yes	Partial	Partial	Yes	Yes

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