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The impact of cognitive-behavior therapy for anxiety disorders on concomitant sleep disturbances: A meta-analysis

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ABSTRACT

Introduction: Sleep disturbances are present in approximately 70% of individuals with an anxiety disorder (AD). Treatments for AD may alleviate associated sleep problems, but empirical support for this view is sparse.
Objective: To assess state of knowledge about the impact of CBT for AD on sleep disturbances.
Method: Systematic search for clinical trials of CBT for any AD in PsycINFO, MedLine, and Proquest Dissertations and Theses.
Results: Of 1205 studies, only 25 (2.07%) reported sleep data. The combined ES of CBT for AD on sleep was 0.527 [95%CI 0.306–0.748], indicating a moderate effect of anxiety treatment on concomitant sleep difficulties. The impact did not significantly differ according to study design, sleep variable or anxiety disorder.
Conclusions: Although substantial amounts of research documented the efficacy of CBT for AD, very few reported its effect on concomitant sleep problems. Current state of knowledge does not permit definitive conclusions and future research is needed.

1. Introduction

Individuals with mental health problems often present with sleep difficulties; this problem is particularly common in patients with anxiety disorders (AD). Nightmares and difficulty falling or staying asleep are included in the DSM-IV-TR (APA, 2000) diagnostic criteria for post-traumatic stress disorder (PTSD). Approximately 70% of individuals with PTSD report symptoms of insomnia (Ohayon & Shapiro, 2000), and 19% (Ohayon & Shapiro, 2000) to 71% (Leskin, Woodward, Young, & Sheikh, 2002) report frequent or disturbing nightmares. Insomnia is also included in the DSM-IV diagnostic criteria for Generalized Anxiety Disorder (GAD), and 60% to 70% of individuals with GAD report sleep disturbances (Papadimitriou & Linkowski, 2005). Sleep-maintenance insomnia is one of the most common complaints in individuals with GAD (Monti & Monti, 2000)

Although not listed in the DSM-IV diagnostic criteria, sleep disturbances are frequently associated with panic disorder with or without agoraphobia (PD/A), obsessive-compulsive disorder (OCD), social phobia or social anxiety disorder (SAD), and specific phobia (SP). Approximately two thirds of individuals with PD/A report sleep disturbances (Overbeek, van Diest, Schruers, Kruizinga, & Griez, 2005; Stein, Chartier, & Walker, 1993a), including difficulty falling or staying asleep, fatigue, and daytime sleepiness (Kessler et al., 1994; Myers et al., 1984). Individuals with PD/A also report nocturnal panic attacks, that is, abrupt waking (usually from slow-wave sleep) in a state of panic. Over one half (58%) of individuals with PD/A have experienced at least one nocturnal panic attack, and 30% to 45% experience repeated nocturnal panic (Craske et al., 2002).

In individuals with OCD, obsessions and compulsions can interfere with the normal sleep onset process. Early polysomnographic (PSG) data revealed decreased sleep efficiency and increased total wake time in OCD patients, in comparison to controls (Hohagen et al., 1994; Insel et al., 1982). One study indicated that patients with SAD frequently reported insomnia when directly questioned about sleep problems (Uhde, 2000). Other studies found a greater frequency of sleep disturbances in individuals with SAD than in

Abbreviations: Ad, anxiety disorders; CBT, cognitive-behavior therapy; DSM-IV-TR, Diagnostic and statistical manual of mental disorders, 4th edition, text revised; ES, effect size; GAD, generalized anxiety disorder; OCD, obsessive-compulsive disorder; PD/A, panic disorder with or without agoraphobia; PSG, polysomnography; PTSD, post-traumatic stress disorder; SAD, social anxiety disorder (or social phobia); SP, specific phobia.

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healthy controls (Stein, Kroft, & Walker, 1993b), but no difference in PSG data between the two groups (Brown, Black, & Uhde, 1994). Both studies used fairly small samples and neither has been replicated to date. Finally, sleep disturbances do not appear to be a typical feature of SP, although some sleep problems could develop in relation to the phobic stimulus (e.g., difficulty falling asleep due to fear that there is a spider in the bedroom) (Uhde, 2000). To our knowledge, sleep in individuals with SP has not been empirically documented.

In sum, empirical evidence suggests that sleep disturbances are part of the clinical portrait for a significant portion of patients with any AD. The strength of the empirical support varies among the different disorders. One recent report found that PD/A, OCD, SAD, GAD, and SP were all associated with poorer sleep and that, with the exception of OCD, the associations remained significant after adjustment for mood and substance use disorders (Ramsawh, Stein, Belik, Jacobi, & Sareen, 2009). Whereas sleep is a state of relaxation and "letting go," anxiety implies arousal, activation, hypervigilance, and alarm. Sleep disturbances can reasonably be expected in any condition characterized by excessive anxiety, but surprisingly little research documenting this association exists. Some evidence suggests a bidirectional relationship between daytime anxiety symptoms and sleep disturbances (Harvey, Hairston, Gruber, & Gershon, 2009). On the one hand, worry and physiological arousal are core features of anxiety and can disrupt sleep (Akerstedt, Kecklund, & Axelsson, 2007). On the other hand, sleep loss, sleep deprivation, and other sleep problems can elevate the risk of daytime anxiety and panic (Babson, Feldner, Trainor, & Smith, 2009).

In the context of treatment for AD, sleep disturbances warrant further clinical and research attention. First, sleep disturbances are a core feature of PTSD and GAD, and are frequently associated with PD/A, OCD, SAD, and SP. Second, AD are the most common diagnoses in patients who complain of insomnia symptoms (Ohayon, 2002). Third, sleep problems can produce anxiety or exacerbate existing AD symptoms. For example, sleep deprivation and insomnia have been associated with increased anxiety levels the following day among healthy individuals (Fernández-Mendoza et al., 2009; Sagaspe et al., 2006), as well as with increased anxious reactivity (Babson et al., 2009) and panic attacks (Roy-Byrne, Uhde, & Post, 1986) the following day among individuals with PD/A. Poor sleep quality has also been associated with more severe post-traumatic symptoms among individuals with PTSD (Belleville, Guay, & Marchand, 2009a) and could predict the development of PTSD after a trauma (Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2010). Finally, sleep problems often linger after successful treatment for anxiety (Belleville, Guay, & Marchand, 2009b; Zayfert & DeViva, 2004) and constitute a risk factor for relapse (Ohayon & Roth, 2003).

Despite the numerous theoretical and empirical associations between AD and sleep disturbances, many cognitive-behavior treatments (CBT) for AD do not include strategies for managing sleep problems (exceptions include imagery rehearsal to decrease the frequency of nightmares in PTSD (Krakow et al., 2001) and an adapted CBT for PD/A designed specifically to treat nocturnal panic attacks (Craske, Lang, Aikins, & Mystkowski, 2005)). One rationale for not addressing sleep management in CBT for AD may be the belief that successful treatment for AD alleviates concomitant sleep disturbances. Indeed, the author of the chapter about anxiety disorders in *Principles and Practice of Sleep Medicine* concluded that, "Effective treatment of the core symptoms of most anxiety disorders will almost always result in a corresponding improvement in associated sleep disturbances" (Uhde, 2000). However, recent opinions suggest anxiety and sleep are intrinsically related (Harvey et al., 2009), could be symptoms of one underlying pathology, and that sleep-specific interventions in the AD treatment are likely to be necessary (Uhde, Cortese, & Vedeniapin, 2009).

To date, no empirical review of the effect of CBT for AD on associated sleep difficulties has been published. The primary objective of the present meta-analysis was to assess the state of knowledge about the impact of CBT for AD on concomitant sleep problems. The second objective was to explore the characteristics of studies that could influence the effects of CBT for AD on sleep.

2. Method

2.1. Search

To collect all studies that evaluated the efficacy of a cognitive and/or behavior treatment for any AD, we conducted an extensive search of three online bibliographic databases, that is, PsycINFO, MedLine via NCBI (PubMed), and Proquest Dissertations and Theses. The following search command was used: (anxiety OR panic OR phobia OR "stress disorder" OR obsessive OR compulsive OR insomnia) AND (cognitive OR behavior* OR "CBT" OR exposure) AND (treatment OR therapy) AND (results OR findings). We further reviewed the reference lists of recent meta-analyses of the efficacy of CBT for specific AD. Finally, the reference list of each study included in the present review was examined.

2.2. Inclusion criteria

The inclusion criteria for the studies in the present review were the following: (a) the study constituted a report of treatment outcome; (b) at least one group received a psychological treatment with a behavioral or cognitive component; (c) the study either had a group protocol or was a case study of at least three participants; (d) the participants were adults; (e) the participants had at least one AD (i.e., GAD, PD/A, Agoraphobia, PTSD, OCD, SAD, or SP); (f) the study was published between 1980 and 2009; and (g) the study was published in English or French.

2.3. Data extraction

All of the eligible studies were independently reviewed by at least two judges, who extracted the relevant data. A data coding form was developed to ensure accuracy and consistency in the data collection process. The following information was extracted from each study: (a) study identification, i.e., authors, date, and source; (b) study description, i.e., protocol, sampling, assignment of participants, comparison group; (c) sample characteristics, i.e., size, gender, age (mean and standard deviation), AD diagnosis, psychiatric or medical comorbidity, explicit reports of sleep problems, and rate of attrition; (d) treatment characteristics, i.e., theoretical orientation, number and duration of therapy sessions, mode of treatment delivery (e.g., individual, group, remote), use of a therapy manual, type of mental health professional who delivered the treatment, and inclusion of specific sleep management strategies; (e) sleep measures (PSG, actigraphic measures, clinical interview, validated sleep questionnaires, sleep diaries, isolated items from instruments validated for other problems, and survey measures); (f) quantitative data for calculating effect sizes (ES), i.e., means and standard deviations of all sleep measures pre- and post-treatment for experimental and control groups, where available.

2.4. Inter-rater agreement

The raters for this study were one postdoctoral fellow and three graduate students in psychology. The judges pre-tested the coding form; all four judges independently rated one selected article, and the results were discussed as a group.

Table 1
Percentage of inter-rater agreement for coding form variables.

Variable	Percentage agreement
Study characteristics	**92.1**
Type of protocol	94.7
Sampling method	94.7
Randomization	100
Comparison group	78.9
Sample characteristics	**82.2**
Sample size	73.7
Percent female	73.7
Age (mean)	84.2
Age (SD)	78.9
Anxiety Disorder	100
Medical/Psychiatric condition	94.7
Sleep difficulties explicitly reported	94.7
Percent attrition	57.9
Treatment characteristics	**77.4**
Type of treatment	68.4
Number of treatment sessions	63.2
Duration of treatment sessions	94.7
Treatment modality	84.2
Use of a treatment manual	73.7
Therapist profession	68.4
Sleep difficulties addressed in treatment	89.5
Sleep measures	**87.7**
Type of sleep measure	94.7
Number of reported sleep variables (usable in the analysis)	73.7
Aspect of sleep targeted by the measure	94.7
Effect sizes	**98.1**

Studies were randomly divided and assigned to two independent judges. Data from each study were coded systematically. The results were compared in biweekly meetings of all four raters and discrepancies were resolved through discussion.

Overall agreement between raters was good, fluctuating between 77.4% and 98.1% (see Table 1). Inter-rater agreement for the ES of the impact of CBT on sleep (the main variable of interest) was very strong (98.1%). The weakest levels of inter-rater agreement were observed for treatment characteristics, particularly type of treatment; number of treatment sessions; and type of professional delivering therapy. These discrepancies may reflect a lack of detail in the original reports.

2.5. Data analyses

Analyses were performed with Comprehensive Meta-Analysis (version 2.2.050; Biostat, Englewood, NJ, USA). Hedge's *g* was calculated for each sleep variable (the primary outcome variables). In studies including a control group, between-group ES (i.e., the difference between the experimental and control groups post-treatment) were computed. In studies reporting only the data from one (or more) treatment group, within-group ES (i.e., the difference between pre- and post-treatment data within the same group) were computed. One study did not report means and standard deviations; ES were estimated from the reported *p* value for each sleep variable. All ES that reflected improved sleep after treatment (i.e., positive outcome in the experimental group or better sleep post-treatment) were expressed by a positive ES. All ES that indicated poorer sleep after treatment (i.e., positive outcome in the control group or better sleep pre-treatment) were expressed by a negative ES. ES were collected for four categories of sleep variables: (a) objective sleep data (PSG) (including measures of sleep continuity or fragmentation, such as total wake/sleep time, sleep-onset latency, sleep efficiency, etc. Measures of sleep architecture, i.e., percentage of time spent in each sleep stage, were not included); (b) self-reported sleep data (including estimates of sleep parameters, obtained with sleep diaries or retrospective questions, and estimates of sleep quality, obtained with questionnaires or single questions); (c) self-reported insomnia data; and (d) self-reported nightmare data. For studies that reported more than one ES per variable, a combined ES was used for the analyses. For studies that reported more than one relevant treatment group, a combined ES was retained for the analyses. Moderator variables included type of sleep outcome (PSG, subjective sleep, insomnia or nightmares), type of comparison (within-group or between-group), and type of anxiety disorder (PTSD, GAD, TP/A, OCD, SAD or SP).

Summary ES were obtained using a random-effects model. In the random-effects model, it is assumed that studies reflect populations of studies that differ from each other systematically (e.g., the true effect size for GAD patients is assumed to be different from the true effect size of patients with other AD). Presence of heterogeneity was assessed with Q, and the proportion of true variance was assessed with I^2. The effects of moderator variables were assessed with Q. Publication bias was tested by inspecting the funnel plot of the relationship between effect size and standard error. The Orwin fail-safe N was also calculated in order to estimate the number of studies with no effect necessary to reduce the combined effect size to a clinically non significant value (0.20).

3. Results

3.1. Search results and characteristics of studies

The search results are summarized in a flow chart in Fig. 1. Using the keywords reported above; a preliminary shortlist of 1,112 eligible studies was created from PsycINFO, MedLine via NCBI (PubMed), and Proquest Dissertations and Theses databases (we included in that list one study conducted by our research team that examined the effect of CBT for PD/A on sleep, but was still in its preparation stage by the time the meta-analysis was conducted; Belleville, Marchand, Lessard, Pelland & Poirier-Bisson, in preparation). A second list of 79 studies was produced from the reference lists of recent meta-analyses, and a third list of 14 studies of CBT for AD was drawn from the reference lists of the articles that were eventually included in the present study. Altogether, 1205 trials of CBT for AD were reviewed. Of this sample, 27 (mostly unpublished theses) were not accessible for consultation and 1153 did not report sleep data. Of the 25 that published sleep data, five studies reported data that were not conducive to calculating ES and two studies presented findings from the same set of data (only one was retained for the present review). The final sample included 19 studies of CBT for AD.

The characteristics of the studies included in the present review are presented in Table 2. All were group studies and used samples of volunteers. Eleven studies used random assignment to treatment conditions; nine studies included a comparison group that did not receive treatment. The number of participants included in the present review (the sum of all of the study samples) was 1201; the average attrition rate was 20.16%. Ten studies focused on participants with PTSD, five studies used participants with GAD, three studies used participants with PD/A, and one study included a mixed sample of participants with various AD. None of the studies reported on participants with OCD, SAD, or SP. Only one study included participants who explicitly reported sleep problems. Most of the treatments included both cognitive and behavioral (CBT) components, but none directly addressed sleep difficulties. The majority of the studies involved individual and group therapies, and one treatment was delivered remotely via Internet. Many of the studies measured sleep with items extracted from broader measures (e.g., a question about nightmares from a diagnostic interview

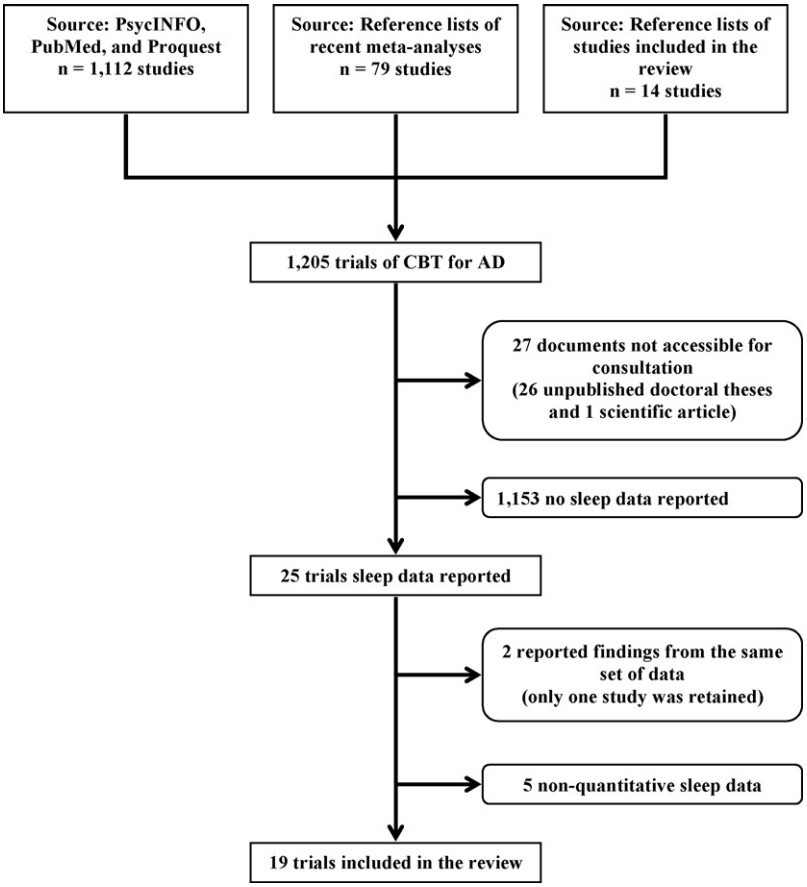

Fig. 1. Flow chart of search results.

validated to assess PTSD), but sleep questionnaires, sleep diaries, and PSG data were also used.

3.2. ES summary data

The summary data for ES are presented in Fig. 2. ES varied considerably, from negative and very small (-0.05) to positive and very large (1.41). Under the random-effects model, the combined ES was 0.527 [95%CI 0.306–0.748], indicating a significant moderate effect of CBT for AD on sleep ($Z = 4.669$, $p < .001$).

There was significant heterogeneity in the sample of ES ($Q(18) = 126.741$, $p < .001$). Most of the observed heterogeneity (86%) could be attributed to true variance across studies ($I2 = 85.798$). Three moderator variables were assessed. There was no significant heterogeneity according to "type of comparison" (within-group or between-group; $Q(1) = 1.228$, $p = .268$) nor according to "type of sleep outcome" (PSG, subjective sleep, insomnia or nightmares; $Q(3) = 4.269$, $p = .234$). Combined ES appeared to be different according to the different anxiety disorders: combined ES was 0.843 [95%CI 0.149–1.536] for GAD, 0.404 [95%CI 0.233–0.575] for PTSD, and 0.216 [95%CI 0.035–0.398] for PD/A. However, the heterogeneity indicator did not reach the significance criterion ($Q(2) = 4.323$, $p = .115$). Given the small number of studies included in each subgroup (GAD = 5; PTSD = 10; PD/A = 3), it is plausible that this could be due to a lack of statistical power.

3.3. Publication bias

Observation of the funnel plot indicated a possible publication bias, suggesting that the combined effect size was overestimated. The Orwin fail-safe test indicated that the number of studies with a zero effect necessary to reduce the combined effect size to 0.2 was 26. It is plausible that, among the 1153 trials of CBT for AD that did not report sleep data, 26 (or more) could have found a zero effect.

4. Discussion

The primary objective of the present review was to assess the state of knowledge about the impact of CBT for AD on concomitant sleep disturbances. Of 1205 clinical trials that evaluated the efficacy of CBT for various AD, only 25 (2.07%) assessed the impact of the treatment on sleep. Nineteen studies (1.6%) provided original and interpretable quantitative sleep data. Although experts in the field have suggested that effective treatment for AD results in a concomitant improvement in sleep (Uhde, 2000), very few empirical data are available to support this conclusion for CBT. Recent opinions suggest that treatment for AD has a positive impact on sleep only if the treatment is initiated in the early phase of the AD. Given that patients with anxiety and sleep problems often wait months or years before seeking treatment, sleep-specific interventions in the AD treatment are likely to be necessary (Uhde et al., 2009).

In this very limited sample of studies, the combined ES of CBT for AD on sleep was 0.527, indicating a moderate effect of anxiety treatment on concomitant sleep difficulties. ES did not differ whether the effect was estimated from post-treatment data comparing a treatment group and a control condition or whether it was obtained from a pre- to post-treatment comparison in a single treatment group. Magnitude of ES were similar across different sleep variables, including subjective sleep parameters, self-reported severity of insomnia, self-reported presence of nightmares or objective sleep data obtained with PSG. Although findings from primary studies have previously been interpreted as indi-

Table 2
Characteristics and effect sizes of studies included in the review.

	Basoglu, Salcioglu, & Livanou (2007)	Bélanger, Morin, Langlois, & Ladouceur (2004)	Belleville et al. (2009b)	Belleville et al. (in preparation)	Bouvard, Mollard, Guerin, & Cottraux (1997)	Cervena, Matousek, Prasko, Brunovsky, & Paskova (2005)	Cooper & Clum (1989)	Ford, Fisher, & Larson (1997)	Frueh, Turner, Beidel, & Mirabella (1996)	Gaines (2002)	Gersons, Carlier, Lamberts, & van der Kolk (2000)	Gosselin, Ladouceur, Morin, Dugas, & Baillargeon (2006)	Lange et al. (2003)	Murphy (2009)	Nishith et al. (2003)	Power, Simpson, Swanson, & Wallace (1990)	Reiner, 2008	Salcioglu et al., 2007	Vaughan et al., 1994
Study																			
Type of protocol	Group	Group	Group	Group	Group	Group	Group	Group	Group	Group	Group	Group	Group	Group	Group	Group	Group	Group	Group
Sample of volunteers	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Randomization	Yes	Yes	No	Yes	No	No	Yes	No	No	No	Yes	Yes	Yes	Yes	No	Yes	No	Yes	Yes
Comparison	BET	BET	WIT	BET	WIT	WIT	BET	WIT	WIT	WIT	BET	BET	BET	WIT	WIT	BET	WIT	BET	WIT
Sample																			
Sample size	100	52	94	42	77	20	22	75	15	96	42	61	184	79	10	113	24	59	36
Percent female	87.00	72.73	69.00	38.01	–	70.00	0.00	0.00	0.00	67.71	11.90	59.02	80.00	70.00	100.0	71.29	50.00	85.00	63.89
Age (mean)	35.50	40.50	40.73	41.76	–	32.90	37.00	48.00	47.90	37.04	36.43	50.33	39.00	–	30.70	40.79	–	36.30	32.00
Age (SD)	11.00	9.40	12.81	12.37	–	10.00	–	5.90	2.10	7.01	6.48	9.84	10.50	–	9.80	–	–	11.50	14.70
Anxiety disorder	PTSD	GAD	PTSD	PD/A	PD/A	PD/A	PTSD	PTSD	PTSD	GAD	PTSD	GAD	PTSD	GAD	PTSD	GAD	AD	PTSD	PTSD
Medical/psychiatric condition	No	No	No	No	No	No	No	No	No	No	No	No	No	No	No	No	No	No	No
Sleep difficulties explicitly reported	No	No	No	No	No	No	Yes	No	No	No	No	No	No	No	No	No	No	No	No
Percent attrition	14.00	15.38	43.88	–	37.00	–	27.27	0.00	26.67	0.00	0.00	8.19	23.91	43.04	40.00	26.55	16.67	–	0.00
Treatment																			
Type of treatment	BEH	CBT	CBT	CBT	CBT	CBT	BEH	CBT	BEH	CBT	CBT	CBT	CBT	CBT	CBT	CBT	CBT	CBT	BEH
Number of treatment sessions	1	14	19	7	–	18	–	–	29	–	16	12	10	8	9	7	–	1	4.30
Duration of sessions (min.)	33	120	–	60	–	90	90	–	90	–	60	90	45	90	90	40	–	60	50
Treatment modality	–	Group	IND	IND	–	Group	IND + group	IND + group	IND + group	–	IND	IND	WEB	Group	IND	IND	–	–	IND
Treatment manual	Yes	Yes	No	Yes	No	No	No	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	No	No	no
Type of therapist	PSY	PSY	PSY	STU	–	–	PSY	–	–	–	PSY	PSY	STU	PSY+ STU+ other	PSY	PSY	–	PSY	–
Sleep difficulties addressed	No	No	No	No	No	No	No	No	No	–	No	No	No	No	No	No	No	No	no
Sleep measures																			
Type of sleep measure	ITEM	ITEM SQ	SQ	SQ diary DX	ITEM	PSG diary	ITEM diary	SQ	diary	ITEM	ITEM	SQ	SQ	SQ	PSG SQ	ITEM	SQ	ITEM	ITEM
Number of sleep variables	2	2	1	5	1	7	3	1	2	1	1	1	1	1	5	1	1	2	1
Aspect(s) targeted	Sleep NIGH	Sleep INS	Sleep	Sleep INS	Sleep	Sleep	Sleep NIGH	Sleep	Sleep NIGH	INS	Sleep	INS	Sleep	Sleep	Sleep	Ins	Sleep	INS NIGH	NIGH
Effect sizes																			
Hedge's *g*	0.43	1.30	0.48	0.41	0.17	0.23	1.27	0.22	0.70	1.41	1.10	0.48	0.11	−0.05	0.56	1.12	0.42	0.19	0.17

Note. AD = any anxiety disorder; BEH = behavior therapy; BET = between-group comparison; CBT = cognitive-behavior therapy; DX = diagnostic interview; GAD = generalized anxiety disorder; IND = individual; INS = insomnia; ITEM = single item from a broader measure; NIGH = nightmares; PD/A = panic disorder with or without agoraphobia; PSG = polysomnography; PSY = psychologist; PTSD = post-traumatic stress disorder; SQ = sleep questionnaire; SD = standard deviation; STU = graduate student in psychology; WEB = delivered via Internet; WIT = within-group comparison.

cating that improvements of nightmares were more likely to be observed following CBT for PTSD than improvements of insomnia problems (Zayfert & DeViva, 2004), the present combined findings did not support this interpretation. However, the absence of differences regarding study designs or sleep outcomes may also reflect a lack of statistical power in the present analyses due to the small number of studies included in each subgroup and should thus be interpreted with caution.

The obtained ES was large for samples of individuals with GAD, moderate for individuals with PTSD and small for individuals with PD/A, although the statistical analysis indicated the absence of a significant difference (which might or might not be attributable to a lack a statistical power). The finding that CBT for GAD might lead to more positive outcome on sleep is consistent with other observations regarding insomnia and GAD. First, some treatment strategies used in CBT for GAD are very similar to those used in CBT for insomnia (e.g., relaxation, cognitive restructuring of dysfunctional beliefs such as catastrophic thoughts and dramatization). Thus, it is possible that the strategies learned in CBT for GAD could generalize and be used by patients to overcome their sleep problems. Second, GAD and insomnia could both be signs of a same underlying condition involving cognitive and somatic hyperarousal (Uhde et al., 2009). CBT for GAD could help manage this hyperarousal condition, resulting in improvements in both anxiety and insomnia symptoms.

Despite some limitations, the present findings have certain clinical implications. First, many sleep problems constitute sleep disorders such as insomnia, nightmares, or nocturnal panic attacks. Clinicians treating patients with AD should be aware that certain sleep disorders are more strongly associated with certain AD (e.g., insomnia and GAD; nightmares and PTSD; nocturnal panic attacks and PD/A). Clinicians working with anxious patients should not assume that sleep will necessarily improve as a function of successful treatment for anxiety. Although moderate improvement in sleep can be expected following treatment for GAD, PTSD or PD/A, no data concerning the impact of CBT for OCD, SAD, or SP on sleep were found. Second, clinicians can expect residual sleep difficulties after treatment in AD patients who present associated sleep problems. If they are not addressed, sleep disturbances in the early phase of any AD can develop into a bona fide sleep disorder (Uhde et al., 2009). Third, clinicians may wish to consider integrating sleep management strategies into AD treatment. Preliminary findings with PTSD and PD/A patients suggest that interventions that directly target sleep may have a positive impact on anxiety symptoms, in addition to improving sleep (Craske et al., 2005; DeViva, Zayfert, Pigeon, & Mellman, 2005; Germain, Shear, Hall, & Buysse, 2007; Krakow et al., 2002).

Although statistically significant, the present findings were based on data from less than 2% of all trials of CBT for AD, and must be interpreted with caution. The very few trials that fit our inclusion criteria cannot be considered representative of all trials of CBT for AD. Further, authors of trials that included sleep outcome measures but found negative results may have omitted the sleep data from the report. Is it plausible that the present review suffer from a significant publication bias since only 26 studies with no effect on sleep would have been necessary to render the effect size clinically non significant. The only unequivocal conclusion that can be drawn from the present study is that the impact of CBT for AD on concomitant sleep problems is currently unknown. While this may not be a very popular addition to the literature on anxiety and sleep, we felt it important to challenge the widespread clinical opinion that sleep problems necessarily improve during treatment for anxiety. Current empirical data do not provide sufficient support for this statement.

Further limitations of the present study include potential biases in the review process. None of the four judges who completed the data extraction coding forms were blind to the study's objectives. However, we made an effort to minimize this threat to validity by having two judges independently rate each study. Although inter-rater agreement varied according to type of data, it was fairly high for the key variables, that is, the quantitative information used to compute ES.

Further research on this topic is clearly necessary. First, additional empirical data on the impact of CBT for AD on concomitant sleep problems are required. Existing sleep self-report instruments, such as the Insomnia Severity Index (Bastien, Vallieres, & Morin, 2001) or the Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), are simple and valid, and are available in many languages; sleep could easily be included as an outcome variable in future trials of CBT for any AD. PSG assessments, daily sleep diaries, and actigraphic recordings could also document the evolution of sleep problems in individuals receiving

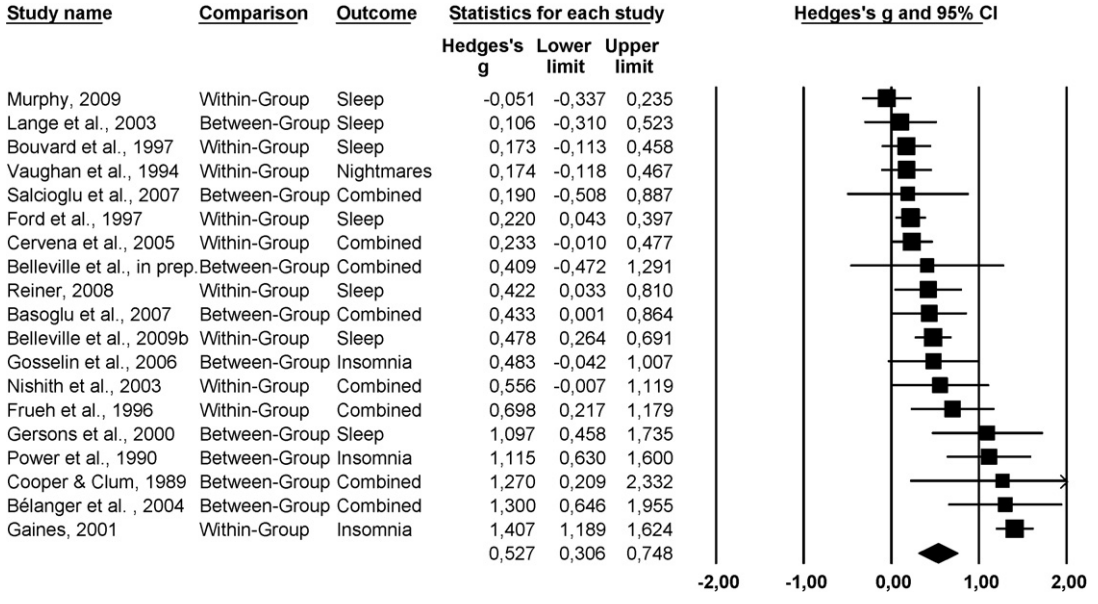

Fig. 2. Results of Meta-analysis under the random-effects model.

cognitive-behavioral treatment for AD. Second, we need to evaluate the impact of including sleep management strategies in treatment for AD on sleep, anxiety symptoms, and general daytime functioning. The feasibility of integrating sleep-specific components to CBT for AD must be examined. Possible integration methods need to be considered (e.g., presentation of sleep strategies before, during, after, or in place of, anxiety management strategies), as well as the possibility of developing sleep management strategies that are specific to different AD. Although considerable research confirms that individuals with AD report poor sleep, there is a distinct lack of empirical evidence to guide clinicians in effectively addressing concurrent anxiety and sleep problems.

5. Conclusions

In conclusion, the available data suggest that CBT for AD has a positive impact on associated sleep difficulties. However, the current body of research is insufficient to support the commonly held view that successful treatment for AD alleviates associated sleep disturbances. Of 1205 studies that evaluated the efficacy of CBT for AD, only 25, that is, 2%, evaluated the impact of the treatment on some aspects of sleep. In addition to an overall lack of research, when sleep was assessed, it was often inadequately measured, highlighting the need for the inclusion of valid and reliable sleep measures in trials of CBT for AD. The true magnitude and clinical significance of the impact of CBT for AD on sleep are yet to be determined. Further research on treatment for sleep difficulties related to AD is required; in particular, we recommend the inclusion of sleep as an outcome variable in future clinical trials of the efficacy of CBT for AD.

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6. Conflict of interest

The authors have no conflict of interest to disclose.

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