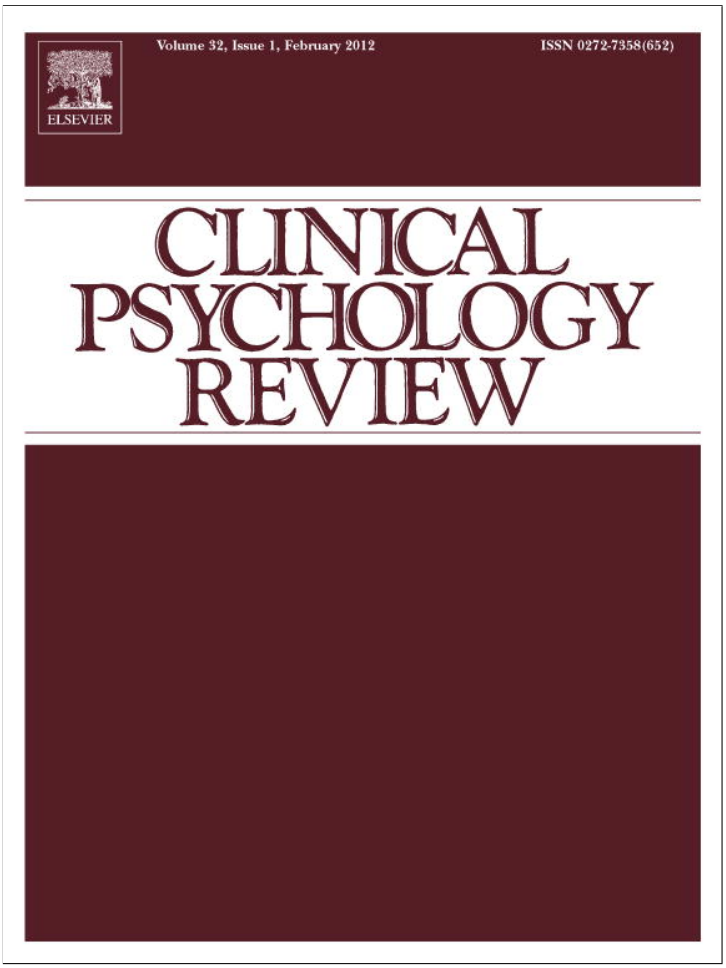

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Mediators of cognitive-behavioral therapy for insomnia: A review of randomized controlled trials and secondary analysis studies

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HIGHLIGHTS

- This study reviewed mediators of cognitive-behavioral therapy for insomnia.
- Randomized controlled trials and secondary analysis studies were investigated.
- 21 RCTs (39% of total) and 11 secondary analysis studies included mediator variables.
- Cognitive-behavioral therapy of insomnia appears to target proposed sleep processes.
- Future mediational analyses would allow for further refinement of treatment.

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ABSTRACT

The examination of treatment mechanisms in randomized controlled trials (RCTs) has considerable implications for research and clinical practice. Insomnia is a highly prevalent and distressing disorder, associated with many adverse outcomes. Although extensive work has focused on the cognitive-behavioral treatment of insomnia (CBT-I), few studies have directly examined the mechanisms of this intervention. CBT-I is a short-term, multi-component treatment that has demonstrated strong efficacy in treating insomnia. The purpose of the present study is: (a) to investigate if CBT-I works in accordance with its proposed mechanisms, and (b) to evaluate how the field is progressing in its understanding of these processes. This study comprehensively reviewed CBT-I RCTs for their inclusion of mediator variables. Secondary analysis studies were also surveyed for relevant mediator variables. Results demonstrated that 21 RCTs (39% of the total RCTs) and 11 secondary analysis studies examined at least one of the proposed mediators. Results of this review highlight that, although CBT-I appears to be targeting the hypothesized sleep processes, more research is needed to better understand whether CBT-I works in accordance with its theorized mechanisms. Inclusion of mediational analyses in future RCTs and secondary analysis studies would allow for further refinement of CBT-I and improved treatment outcomes.

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1. Introduction

The importance of examining mechanisms of treatment in randomized controlled trials (RCTs) has been emphasized throughout the literature (Kazdin, 2007; Kraemer, Wilson, Fairburn, & Agras, 2002). Treatment mechanism research may lead to a number of considerable outcomes, including a better understanding of the processes leading to therapeutic change, the refinement of current treatment protocols, and increased generalizability of treatment effects from research to practice. Understanding treatment mechanisms may also help to clarify the nature and etiology of disorders, thereby informing future research and clinical practice. According to Kraemer et al. (2002), mechanisms are causal links between treatment and outcomes and mediators explain why and how treatments have effects. Although mediators have less specificity than mechanisms, they identify possible mechanisms of treatment and are therefore an important first step in understanding the processes underlying therapeutic change (Kazdin, 2007).

In light of the implications of treatment mechanism research, there is a growing movement within the cognitive-behavioral therapy (CBT) literature to examine the processes of change throughout treatment. A number of research areas, including CBT for depression (e.g., Hollon, DeRubeis, & Evans, 1987; Quilty, McBride, & Bagby, 2008), anxiety disorders (e.g., Hofmann, 2004; Hofmann et al., 2007; Smits, Powers, Cho, & Telch, 2004), eating disorders (e.g., Murphy, Cooper, Hollon, & Fairburn, 2009; Spangler, Baldwin, & Agras, 2004), and medical illnesses (e.g., Knoop, van Kessel, & Moss-Morris, 2012; Turner, Holtzman, & Mancl, 2007; Wiborg, Knoop, Prins, & Bleijenberg, 2011) have made efforts to examine mediators of therapeutic change. To date, extensive research has focused on the cognitive-behavioral treatment of insomnia (CBT-I); however, few studies have examined the mechanisms of this intervention. An understanding of these processes is critical given the high prevalence of insomnia and the significant distress associated with it.

In a U.S. epidemiological study ($n = 10{,}094$), prevalence of insomnia varied depending on the criteria used. Prevalence estimates were 22.1% for DSM-IV-TR, 14.7% for Research Diagnostic Criteria/International Classification of Sleep Disorders, Second Edition (RDC/ICSD-2), and 3.9% for International Classification of Diseases, Tenth Revision (ICD-10) (Roth et al., 2011). Insomnia is present across a wide range of psychological illnesses, including depression and anxiety (Taylor, Lichstein, Durrence, Reidel, & Bush, 2005), as well as medical illnesses, including heart disease, cancer, high blood pressure, neurologic disease, breathing problems, urinary problems, chronic pain, and gastrointestinal problems (Taylor et al., 2007). It is associated with worsened quality of life, marked functional impairment (Katz & McHorney, 2002; Kyle, Morgan, & Espie, 2010; Ohayon, 2002), and considerable financial costs (Daley, Morin, LeBlanc, Grégoire, & Savard, 2009). A Canadian study examining the economic burden of insomnia estimated that the average annual per-person costs were $421 for good sleepers compared to $5010 for individuals with insomnia. Such elevated costs were a result of direct expenses (e.g., increased health-care utilization, prescription medications, and over-the-counter products) and indirect expenses (e.g., insomnia-related absenteeism, productivity losses/diminished job performance, and increased proneness to accidents) (Daley et al., 2009).

2. Theories of insomnia

In understanding the treatment mechanisms of CBT-I, an explanation of the processes underlying insomnia is first warranted. Spielman, Caruso, and Glovinsky (1987) proposed a theoretical model of insomnia based on three factors — predisposing factors, precipitating factors, and perpetuating factors. Predisposing factors precede the onset of sleep difficulties and increase individuals' vulnerability to insomnia; precipitating factors trigger the onset of insomnia and typically involve acute stressors; and perpetuating factors maintain insomnia after the initial sleep-disturbing factors have resolved (Spielman et al., 1987). Accordingly, although certain predisposing and precipitating factors may increase individuals' likelihood of developing sleep difficulties, it is the perpetuating factors that maintain sleep disturbances in the long-term. Theoretical models of insomnia have focused on the interplay of behaviors, cognitions, and hyperarousal as the primary perpetuating factors of insomnia.

2.1. Behavioral models of insomnia

Two separate biological systems regulate wakefulness and sleep — the homeostatic system, a process that arises during wakefulness and diminishes during sleep, and the circadian system, the body clock that determines the timing of sleep and wakefulness in the 24-hour day (Borbély, 1982; Webb, 1988). The homeostatic system operates based on the amount of time spent asleep or awake. Over the course of the day, during periods of wakefulness, individuals build up sleep drive. It is the amount of sleep drive accumulated throughout the day that determines the quantity and quality of sleep (Borbély, 1982; Webb, 1988). Certain behavioral patterns serve as perpetuating factors by disrupting homeostatic regulation and maintaining sleep difficulties. For example, although daytime napping may be perceived as an adaptive strategy of catching up on rest after a poor night's sleep, it may interfere with the body's homeostatic mechanisms by decreasing sleep drive. Spending excessive time in bed, sleeping in, going to bed early, and inactivity are also examples of sleep-related behaviors that negatively impact the homeostatic system by preventing the accumulation of sleep drive throughout the day (Carney, Edinger, Meyer, Lindman, & Istre, 2006; Edinger & Means, 2005).

The circadian system serves as a biological clock to regulate sleep-wake patterns while interacting with time cues in the environment (e.g., light–dark cycle, social interactions, work schedules) (Borbély, 1982; Webb, 1988). Physiological differences exist between individuals' circadian systems or chronotypes (i.e., propensity of

circadian rhythms to express themselves in distinct behavioral patterns) (McEnany & Lee, 2000). Although many individuals do not have a strong morning or evening chronotype, certain individuals have an advanced circadian clock and prefer morning hours (called "larks") and certain individuals have a delayed circadian clock and prefer evening hours (called "night owls") (McEnany & Lee, 2000). Individuals' chronotypes determine their sleep schedules and optimal sleep windows. Thus, a number of improper sleep scheduling behaviors may disrupt these processes and perpetuate sleep difficulties. These include waking up and going to sleep at different times each day and sleeping outside one's optimal window (e.g., a night owl getting up early in the morning).

2.2. Cognitive models of insomnia

The cognitive mechanisms underlying insomnia have also been a focus of the literature (Harvey, 2002, 2005; Kaplan, Talbot, & Harvey, 2009). According to the cognitive model of insomnia (Harvey, 2002), individuals with insomnia experience increased worry and rumination about a range of issues, including their inability to sleep and the impact of their sleep disturbance on daily functioning. As a result of this anxiety, their attention becomes focused on sleep-related threats, both internal (e.g., bodily sensations) and external (e.g., environmental noises). This selective attention further exacerbates worry and anxiety, as individuals become more attuned to minor cues that would otherwise be undetected. Additionally, individuals may develop erroneous beliefs about sleep and about the adaptive nature of worry, which will increase cognitive activity and arousal and decrease the likelihood of falling sleep (Harvey, 2002). Finally, the model states that individuals adopt safety behaviors to alleviate this arousal (e.g., napping, drinking alcohol, and avoiding challenging tasks); however, these behaviors ultimately exacerbate worry and prevent individuals from challenging their unhelpful sleep-related beliefs (Harvey, 2002).

In another cognitive model of insomnia, called the attention-intention-effort (A-I-E) pathway, Espie, Broomfield, MacMahon, Macphee, and Taylor (2006) argue that normal and automatic sleep processes become disrupted when individuals selectively attend to sleep (A), explicitly intend to sleep (I), and engage in efforts to produce sleep (E). According to this theory, these cognitive and behavioral disturbances to normal sleep (i.e., "sleep effort") are central to the sleep disruptions that occur in insomnia (Espie et al., 2006).

2.3. Hyperarousal models of insomnia

Past research has shown that individuals with insomnia appear hyperaroused across multiple biological systems (Bonnet & Arand, 1995, 1998). Hyperarousal models of insomnia (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997; Riemann et al., 2010) build upon behavioral and cognitive models of insomnia while integrating principles of learning theory. These models identify conditioned arousal as a key perpetuating factor in the maintenance of insomnia. Based on the principles of classical conditioning, it is proposed that repeated associations between poor sleep and the bed result in conditioned arousal, whereby the bed and sleep environment become stimuli for heightened arousal (Bootzin, 1972). On a cognitive level, individuals who ruminate about and selectively attend to their sleep difficulties may develop "learned sleep preventing associations", which are central to the development of chronic sleep disturbances (Riemann et al., 2010). Taken together, hyperarousal models of insomnia highlight the importance of conditioned arousal, both physiological and cognitive, as primary perpetuating factors of insomnia.

3. Cognitive-behavioral therapy for insomnia

Although CBT-I and pharmacotherapy demonstrate comparable efficacy in the treatment of insomnia (Morin, Colecchi, Stone, Sood, & Brink, 1999; Morin et al., 2009), CBT-I has been recommended as a first line treatment given its long-term efficacy (Espie, 2009). CBT-I was developed as a psychological intervention to target the perpetuating factors of insomnia. It is a short-term, multi-component treatment, comprised of behavioral and cognitive techniques (Morin & Espie, 2003; Perlis & Lichstein, 2003). CBT-I is typically conducted over the course of four to eight sessions (Morin et al., 2006), with a focus on psycho-education, behavioral and cognitive strategies (Edinger & Carney, 2008). Treatment often includes: stimulus control, sleep restriction, cognitive therapy, sleep hygiene, and relaxation training (see Edinger & Means, 2005 for a review). Each CBT-I component involves distinct skills and strategies intended to target specific mechanisms of insomnia.

3.1. Stimulus control

The purpose of stimulus control is to unlearn the negative associations between the bed/bedroom and sleep and re-associate the bed/bedroom with normal sleep (Bootzin, Epstein, & Wood, 1991). To achieve this, several techniques are proposed. These include: (a) only going to bed when sleepy, (b) setting a standard rise time, (c) leaving the bed/bedroom after long periods of wakefulness, avoiding sleep-incompatible behaviors in the bed or bedroom (e.g., reading, watching TV, eating), and (d) avoiding napping (Bootzin et al., 1991; Edinger & Carney, 2008; Edinger & Means, 2005).

3.2. Sleep restriction

This strategy is used to restore normal homeostatic sleep drive by restricting the amount of time spent in bed (Spielman et al., 1987). A specific set of instructions has been proposed in implementing sleep restriction (Edinger & Means, 2005; Wohlgemuth & Edinger, 2000). The first step in this process is to reduce the amount of time spent in bed to the estimated average total sleep time, which is calculated based on a sleep diary. An additional 30 minutes is added to this time to allow for normal nocturnal wakefulness time, and the initial time in bed prescription is typically not set below 5 hours. The second step is to adjust time in bed up or down in 15 to 30 minute increments based on individuals' sleep performance. By restricting the amount of time allotted for sleep each night, the goal is for the time in bed to eventually match individuals' sleep needs in order to improve sleep quality and efficiency (i.e., percentage of time asleep relative to time in bed) (Wohlgemuth & Edinger, 2000).

3.3. Cognitive therapy

Cognitive therapy for insomnia is designed to target the cognitive hyperarousal that perpetuates insomnia (Harvey, 2002, 2005; Harvey, Sharpley, Ree, Stinson, & Clark, 2007). Through psycho-education and cognitive restructuring, individuals are taught to correct their dysfunctional beliefs and attitudes about sleep. Similar to cognitive therapy for depression, techniques typically involve thought records to challenge maladaptive thoughts, behavioral experiments to test and disconfirm unrealistic expectations, and Socratic questioning to facilitate individuals' learning and self-efficacy (Harvey, 2002, 2005; Harvey et al., 2007).

3.4. Sleep hygiene

The goal of sleep hygiene is to correct certain behavioral patterns, environmental conditions, or other sleep-related factors that may be inhibiting sleep (Hauri, 1991; Stepanski & Wyatt, 2003). Individuals

are provided with psycho-education about normal and healthy sleep behaviors and sleep-promoting environmental conditions. For example, they may be encouraged to limit caffeine, alcohol, and nicotine intake, exercise on a regular basis, eat a light snack before bedtime, and maintain a cool, quiet, and dark sleeping environment (Hauri, 1991; Stepanski & Wyatt, 2003).

3.5. Relaxation training

Relaxation therapy, which has long been used as a treatment for insomnia, involves strategies targeting physiological and cognitive arousal in the context of sleep-related performance anxiety and bedtime arousal (Borkovec & Fowles, 1973; Nicassio & Bootzin, 1974). Formal relaxation techniques include progressive muscle relaxation, passive relaxation, autogenic training, biofeedback, imagery training, meditation, and hypnosis (Edinger & Carney, 2009). Regardless of the strategy, treatment involves learning the relaxation skill over several treatment sessions and practicing the techniques between sessions.

4. Hypothesized mediators of CBT-I

To date, no known studies have systematically reviewed the theorized mechanisms of treatment outcomes in CBT-I. Based on the aforementioned models of insomnia, a number of mediators are believed to account for therapeutic change in this intervention (see Table 1). First, spending excessive time in bed and napping have been proposed to disrupt the homeostatic systems by decreasing sleep drive (Bootzin et al., 1991; Spielman et al., 1987). Accordingly, restricted time in bed and reduced napping would be expected to target disturbances to the homeostatic system and thus improve sleep outcomes. Variability in bedtime and rise time has also been proposed as a maintenance factor in insomnia (Bootzin, 1972; Spielman et al., 1987); therefore, increased regularity in bedtime and rise time would be another hypothesized mediator of CBT-I. Details regarding time in bed, napping, and bedtime and rise time variability are available through sleep diaries.

In targeting cognitive mediators of insomnia, maladaptive beliefs and attitudes about sleep are considered primary perpetuating factors of sleep problems. As such, CBT-I would be expected to reduce or correct these dysfunctional beliefs, thereby improving sleep outcomes. The Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS; Morin, 1993; Morin, Stone, Trinkle, Mercer, & Remsberg, 1993) is a self-report measure that was developed to examine changes in sleep-related beliefs and attitudes. Abbreviated 10-item (DBAS-10; Espie, Inglis, Harvey, & Tessier, 2000) and 16-item (DBAS-16; Morin, Vallières, & Ivers, 2007) versions are also available. In addition to the DBAS, a number of other measures have been developed to measure cognitive processes in insomnia. Espie et al. (2006) proposed that sleep effort is a key perpetuating factor of insomnia; therefore reduced sleep effort would be expected to improve sleep outcomes. To measure sleep effort, the Glasgow Sleep Effort Scale (GSES; Broomfield & Espie, 2005) was developed to assess direct and voluntary attempts to control sleep. The Sleep Self-Efficacy Scale (Lacks, 1987) and the Sleep Locus of Control Scale (SLOC; Vincent, Sande, Read, & Giannuzzi, 2004) are other examples of measures examining individuals' sleep-related beliefs. The Sleep Self-Efficacy Scale measures individuals' level of confidence in achieving certain sleep-promoting behaviors, such as feeling physically relaxed in bed and controlling racing thoughts. The SLOC has two subscales — internal sleep locus of control (i.e., belief that one can control one's sleep) and chance sleep locus of control (i.e., belief that sleep is out of one's control). Research has demonstrated that, relative to a community sample, individuals with insomnia have a less internal sleep locus of control (Vincent et al., 2004). Thus, increased sleep-related self-efficacy and increased internal sleep locus of control would be presumed to improve sleep outcomes.

Given that physiological and cognitive hyperarousal have been cited as key maintenance factors of insomnia, CBT-I would be expected to reduce hyperarousal and subsequently improve sleep. Hyperarousal may be assessed using self-report and physiological measures. The Pre-Sleep Arousal Scale (PSAS; Nicassio, Mendlowitz, Fussell, & Petras, 1985) is a self-report measure that examines individuals' state of cognitive and physiological arousal prior to falling asleep. In terms of physiological measures, cortical arousal has been measured using electroencephalography (EEG), which measures electrical activity in the brain (e.g., Freedman, 1986; Merica, Blois, & Gaillard, 1998), and the Multiple Sleep Latency Test, which consists of a series of nap opportunities administered at 2-hour intervals throughout the day with EEG sleep monitoring (Carskadon et al., 1986). Neuroimaging methods are also available, including single photon emission computed tomography (Smith et al., 2002), positron emission tomography (Nofzinger et al., 2004), and functional magnetic resonance imaging (Altena et al., 2008). In addition, autonomic variables (i.e., heart rate, body temperature, whole-body metabolism, and galvanic skin response) and neuroendocrine variables (i.e., cortisol levels) (Adam, Tomeny, & Oswald, 1986) have been used to measure changes in arousal. For a comprehensive review of physiological measures of hyperarousal in insomnia, see Riemann et al. (2010).

Table 1
Summary of theoretical models of insomnia and proposed mediators.

Sleep regulating process	Proposed mediators
Behavioral	Decreased time in bed
	Decreased napping
	Decreased bedtime and rise time variability
Cognitive	Decreased maladaptive beliefs and attitudes about sleep
	Decreased sleep effort
	Increased sleep-related self-efficacy
	Increased internal sleep locus of control
Hyperarousal	Decreased physiological arousal
	Decreased cognitive arousal

5. The current study

Taken together, in spite of the strong theoretical and empirical support for CBT-I, it remains unknown whether CBT-I is in fact targeting its theorized mechanisms of insomnia. Accordingly, this paper has two primary goals: (a) to determine whether CBT-I is working in accordance with its theorized mechanisms; and (b) to investigate the field's progress in understanding the mechanisms of CBT-I.

6. Method

A thorough search of PsycINFO and PubMed was conducted, using general terms such as insomnia, sleep, CBT, and trial, and more specific terms such as randomized controlled trial, mediator, and mechanism. The search results were screened for their relevance to the review. Reference lists of relevant literature and papers citing the RCTs were searched and used to identify additional relevant studies. Studies were included based on the following inclusion criteria: (a) the study was a published RCT comparing CBT-I to another condition; (b) participants were adults diagnosed with insomnia, consistent with research diagnostic criteria (Edinger et al., 2004); (c) one treatment condition involved one or more of the CBT-I components; and (d) the dependent variables focused on sleep outcomes. Finally, given that many RCTs primarily focus on outcomes variables, we examined secondary analyses of the original RCT data. Therefore, we surveyed the literature to determine if any secondary analysis studies provided relevant information regarding mediators of CBT-I.

Table 2
Descriptions of mediator variables in CBT-I RCTs.

Study	Design	Insomnia sample	Mediator measures	Mediator findings
Bastien, Morin, Ouellet, Blais, and Bouchard (2004)	CBT vs. CBGT vs. phone CBT	45 adults	DBAS (French translation)	No group differences Main effect of time (DBAS scores improved with treatment)
Edinger, Wohlgemuth, Radtke, Marsh, and Quillian (2001a)	CBT vs. RT vs. PLA	75 adults	Rise time variability	CBT group had less variability than other groups during treatment (non-significant trends at 6-month FU)
			Sleep Self-Efficacy Scale	Higher scores in CBT vs. RT at 6-month FU
			TIB	CBT group spent less TIB during treatment and 6-month FU compared to other groups
Edinger and Sampson (2003)	CBT vs. SH	20 adults	DBAS	DBAS scores decreased in CBT group, not SH group
			Sleep Self-Efficacy Scale	Higher scores in CBT vs. SH at 3-month FU
Edinger, Wohlgemuth, Radtke, Coffman, and Carney (2007)	CBT (1, 2, 4, 8 sessions) vs. WL	86 adults	TIB variability	Actigraphy: all CBT groups had less TIB variability than WL group Sleep diary: all CBT groups except 1-session group had less TIB variability than WL group; trend toward lower TIB variability in 1-session group
			Sleep Self-Efficacy Scale	No group differences Scores increased from baseline to 6-month FU for CBT groups
Edinger et al. (2009)	CBT vs. SH	81 adults with mixed psychiatric disorders	DBAS	CBT group had greater improvements from pre- to post-treatment than SH
			Rise time variability	CBT group had less rise time variability than SH groups at post-treatment
			TIB variability	CBT group had less TIB variability than SH groups at post-treatment
Friedman et al. (2000)	SRT + SH vs. NSRT + SH vs. SH	39 older adults	Napping	SRT group: at baseline, 50% nappers; at 3-month FU: 75% of nappers stopped napping, 25% remained nappers, and 50% of total sample remained non-nappers
			TIB	Significant group main effect and group × time interaction for TIB For SRT + SH and NSRT + SH groups, TIB was lowest at post-treatment but closer to SH group at 3-month FU
Jansson and Linton (2005)	CBGT vs. self-help control	165 adults	DBAS-10	Greater reductions in CBT group vs. self-help control group at 1-year FU
Krystal and Edinger (2010)	CBT vs. PLA	30 adults	TIB	CBT group had greater reduction in TIB from pre- to post-treatment than PLA group
Lichstein, Wilson, and Johnson (2000)	RT + SC vs. no-treatment control	44 older adults with psychiatric or medical illness	Arousal	Arousal decreased in CBT group (based on ratings and pulse rate) post-exercises
			Napping	In RT + SC group, significant decrease in napping from baseline to post-treatment (ns at 3-month FU); greater improvements in RT + SC group compared to control
Lichstein, Riedel, Wilson, Lester, and Aguillard (2001)	RT vs. SRT vs. PLA	74 older adults	Arousal	Arousal decreased in RT group (based on ratings and pulse rate) post-exercises during treatment, post-treatment, and FU
			DBAS	Main effect of time and group × time interaction (DBAS scores decreased from baseline to FU only in RT group)
			Napping	Main effect of time (napping reduced from baseline to post-treatment)
			TIB	SRT group spent less TIB at post-treatment and 1-year FU than baseline; TIB was higher at 1-year FU than post-treatment
Means, Lichstein, Epperson, and Johnson (2000)	RT vs. no-treatment control	118 college students (57 with insomnia)	DBAS	Insomnia groups (receiving or not receiving treatment) had higher DBAS scores than no-insomnia group at baseline and post-treatment Main effect of time (DBAS scores improved from baseline to post-treatment)
Mimeault and Morin (1999)	CBT vs. CBT + consult vs. WL	54 adults	DBAS	Main effect of time (CBT and CBT + consult improved over treatment) Group × time interaction (CBT and CBT + consult had lower scores at post-treatment and FU; WL did not)
Perlis et al. (2004)	CBT + PLA vs. CBT + PHA vs. PHA + control	30 adults	Adherence to prescribed bedtime	80% of CBT + PHA group vs. 51% CBT + PLA adherent during treatment; CBT + PLA became more adherent over time
Rosen, Lewin, Goldberg, and Woolfolk (2000)	RT + PHA vs. PHA + imagery vs. PHA + SH	41 adults	PSAS	Decreased arousal from pre-treatment to 6-month FU across groups
			Sleep Self-Efficacy Scale	Increased scores from pre-treatment to 6-month FU across groups
Rybarczyk, Lopez, Benson, Alsten, and Stepanski (2002)	CBT vs. RT vs. delayed-treatment control	38 older adults with medical illness	DBAS	Group × time interaction (CBT group had lower scores than control at post-treatment and 4-month FU)
			TIB	Group × time interaction (CBT group had greater TIB change than control at post-treatment and 4-month FU; CBT group had greater TIB change than RT group at post-treatment only)
Rybarczyk et al. (2005a)	CBT vs. PLA (stress management/ wellness training)	92 older adults with medical illness	DBAS	Group × time interaction (CBT group had greater improvements from pre- to post-treatment than PLA)
			Napping	Group × time interaction (CBT group had greater reduction in number of weekly naps from pre- to post-treatment than PLA)
			TIB	Group × time interaction (CBT group had greater TIB change from pre- to post-treatment than PLA)
Ström, Petterson, and Andersson (2004)	Internet CBT vs. WL	109 adults	DBAS	Group × time interaction (CBT group had greater improvements from pre- to post-treatment than WL)
van Straten, Cuijpers, Smit, Spermon, and Verbeek (2009)	Self-help CBT vs. WL	247 adults	DBAS	CBT group had greater improvements from pre- to post-treatment than WL
Vincent and Lewycky (2009)	Computerized CBT vs. WL	118 adults	DBAS-10	Main effect of group, time, group × time interaction (CBT group had improvements from pre- to post-treatment and 4-week FU; WL did not)

Table 2 (*continued*)

Study	Design	Insomnia sample	Mediator measures	Mediator findings
			PSAS	Main effect of time, group × time interaction (CBT group had improvements from pre- to post-treatment and 4-week FU; WL did not)
Waters et al. (2003)	RT + CD vs. SRT + SC vs. PHA vs. SH	53 adults	Adherence to prescribed bedtime and rise time	For SR + SC group, participants reported going to bed at assigned time 81% of nights and rising at assigned time 73% of nights
Wu, Bao, Zhang, Deng and Long (2006)	CBT vs. PHA vs. CBT + PHA vs. PLA	71 adults	DBAS	CBT group had greater improvements than other groups at post-treatment, 3-month, and 8-month FU
			PSAS	CBT, PHA, and CBT + PHA groups had lower scores than PLA at post-treatment, 3-month FU, and 8-month FU CBT group had lowest scores at 3-month and 8-month FU

Note. CBGT = cognitive-behavioral group therapy; CD = cognitive distraction; DBAS = Dysfunctional Beliefs and Attitudes Scale; FU = follow-up; NSRT = nap-modified sleep restriction therapy; PHA = pharmacotherapy; PLA = placebo; PSAS = Pre-sleep Arousal Scale; RT = relaxation therapy; SC = stimulus control; SH = sleep hygiene; SRT = sleep restriction therapy; TIB = time in bed; WL = waitlist.

7. Results

7.1. Overview of mediators in CBT-I RCTs

Fifty-four CBT-I RCTs met the study's inclusion criteria. Of these RCTs, 21 studies (39%) included one or more of the hypothesized mediator variables. Table 2 presents an overview of these RCTs and their relevant mediator findings. A list of RCTs that met study criteria but did not include information on mediator variables is included in Appendix A.

Of the 21 RCTs that included mediator variables, 11 studies included information regarding time in bed, napping, and bedtime or rise time variability. Consistent with the expected findings, results demonstrated that individuals reported significant time in bed reductions from pre- to post-treatment, as well as greater time in bed reductions relative to comparison groups (Edinger et al., 2001a, 2007, 2009; Friedman et al., 2000; Krystal & Edinger, 2010; Lichstein et al., 2001; Rybarczyk, Lopez, Benson, Alsten, & Stepanski, 2002). A number of studies reported that time spent in bed increased at follow-up time points (Friedman et al., 2000; Lichstein et al., 2000); however, this is not surprising given that individuals' time in bed prescriptions are likely to be adjusted upwards following improved sleep performance (Wohlgemuth & Edinger, 2000). Findings related to napping were also consistent with expectations. Studies reported that individuals experienced decreases in napping from pre- to post-treatment and greater reductions in napping than comparison groups (Friedman et al., 2000; Lichstein et al., 2000, 2001; Rybarczyk, Stepanski, Fogg, Lopez, Barry, & Davis, 2005a). With regard to bedtime and rise time variability, two studies reported that individuals completing CBT demonstrated less rise time variability following treatment relative to comparison groups (Edinger et al., 2001a, 2009). Two other studies examined adherence to prescribed bedtime and rise time; however, no information was available regarding changes in these variables over the course of treatment (Perlis et al., 2004; Waters et al., 2003).

Thirteen of the reviewed RCTs included information on the DBAS (Bastien et al., 2004; Edinger & Sampson, 2003; Edinger et al., 2009; Jansson & Linton, 2005; Lichstein et al., 2001; Means et al., 2000; Mimeault & Morin, 1999; Rybarczyk, Lopez, Schelble, & Stepanski, 2005b; Rybarczyk et al., 2002; Ström, Petterson, & Andersson, 2004; van Straten, Cuijpers, Smit, Spermon, & Verbeek, 2009; Vincent & Lewycky, 2009; Wu, Bao, Zhang, Deng, & Long, 2006). Studies consistently reported that participants of CBT-I experienced significant improvements in their maladaptive beliefs from pre- to post-treatment and follow-up, as well as greater improvements in CBT groups relative to comparison groups.

In terms of other hypothesized cognitive processes, none of the reviewed RCTs included information on sleep effort or sleep locus of control. Four studies examined sleep-related self-efficacy (Edinger & Sampson, 2003; Edinger et al., 2001a, 2007; Rosen, Lewin, Goldberg, & Woolfolk, 2000). Two studies reported that, at follow-up assessment, individuals who had received CBT-I demonstrated higher self-efficacy relative to individuals who had received relaxation therapy (Edinger et al., 2001a) and sleep hygiene (Edinger & Sampson, 2003). In two additional studies, no group differences were found between CBT-I and comparison groups; however, individuals receiving CBT-I experienced an increase in sleep-related self-efficacy from pre- to post-treatment (Edinger et al., 2007; Rosen et al., 2000).

Minimal information was available on physiological arousal. Lichstein et al. (2001, 2000) reported that individuals completing CBT and relaxation therapy experienced significant reductions in subjective and objective arousal following relaxation exercises based on logs and pulse rate. However, these ratings were completed prior to and following relaxation inductions rather than pre- to post-treatment. For exclusively subjective measures, self-report ratings of arousal based on the PSAS were investigated in three studies (Rosen et al., 2000; Vincent & Lewycky, 2009; Wu et al., 2006). Studies consistently found that cognitive and physiological arousal decreased from pre- to post-treatment among individuals who completed CBT, and that, relative to comparison groups, individuals receiving CBT experienced greater reductions in arousal at post-treatment and follow-up (Rosen et al., 2000; Vincent & Lewycky, 2009; Wu et al., 2006).

7.2. Overview of mediators in CBT-I RCT secondary analysis studies

Of the surveyed literature, 11 RCT secondary analysis studies were found that included one or more of the hypothesized mediator variables. Table 3 presents an overview of these studies and their relevant mediator findings. Three studies included information on time in bed, napping, or bedtime and rise time variability (Rybarczyk, Lopez, Schelble, & Stepanski, 2005b; Tremblay et al., 2009; Vitiello et al., 2009). Results indicated that individuals receiving CBT demonstrated greater reductions in time in bed from pre- to post-treatment compared to controls (Rybarczyk et al., 2005b). As well, a higher adherence to prescribed bedtime and rise time was associated with improved sleep outcomes (Tremblay et al., 2009).

For napping, one study reported that higher avoidance of daytime napping was associated with sleep improvements in a sample of women with breast cancer (Tremblay et al., 2009). In another study of older adults experiencing osteoarthritis, no significant differences in napping were found from pre-treatment to post-treatment (Vitiello et al., 2009). However, this finding may be attributable to the fact that the mean napping time in the sample was low to begin with (i.e., 9.9 minutes per week at pre-treatment in CBT-I group). It is also important to mention that the duration and timing of daytime naps may impact the extent to which napping impairs subsequent nocturnal sleep (Feinberg et al., 1985; Lavie & Weler, 1989). Accumulation of delta wave activity, which occurs during deep sleep, is associated with decreased sleep drive (Borbély, 1982). Past research has found that an early afternoon nap contains more delta wave sleep than later naps (Lavie & Weler, 1989) and decreases the subsequent night's sleep quality (Feinberg et al., 1985). However, one study reported that brief 10-minute afternoon naps had positive effects

Table 3
Descriptions of mediator variables in RCT secondary analysis studies.

Study	Primary RCT	Design	Insomnia sample	Mediator measures	Mediator findings
Currie, Wilson, and Curran (2002)	Currie et al. (2000)	CBT vs. delayed treatment control	51 adults with chronic pain (29 improvers, 22 non-improvers)	Sleep Self-Efficacy Scale	Significant increases from pre- to post-treatment and 3-month FU Improvers had greater increases in SES over the course of treatment than non-improvers Lower SES at baseline predicted greater sleep improvements following CBT
Edinger, Wohlgemuth, Radtke, Marsh, and Quillian (2001b)	Edinger et al. (2001a)	CBT vs. RT vs. PLA	75 adults	DBAS-10	Group × time interactions for total score and sleep preoccupation scale CBT group had greater improvements from pre- to post-treatment than other groups CBT group had greater improvements than RT at 6-month FU Improvements in DBAS were related to improvements in subjective and objective sleep outcomes, particularly in CBT group
Edinger, Carney, and Wohlgemuth (2008)	Edinger et al. (2001a) & Edinger, Wohlgemuth, Radtke, Coffman, and Carney (2007)	CBT vs. RT vs. control	155 adults	Sleep Self-Efficacy Scale /DBAS	Individuals in CBT group with relatively high levels of pre-therapy unhelpful sleep-related beliefs (based on DBAS and SES scores) performed better on sleep outcomes in response to CBT
Espie, Inglis, and Harvey (2001)	Espie, Inglis, Tessier, and Harvey (2001)	CBT vs. control	109 adults	DBAS	Higher baseline scores on DBAS subscale (beliefs about negative long-term consequences of insomnia) were associated with improvements on sleep outcomes in response to CBT
Jansson-Fröjmark and Linton (2008)	Jansson and Linton (2005)	CBT vs. self-help control	64 adults	DBAS-10	High baseline scores were related to improvements in sleep efficiency and daytime sleepiness following CBT Pre- to post-treatment DBAS improvements were associated with greater improvement of sleep quality and daytime symptoms
Montserrat Sánches-Ortuno and Edinger (2010)	Edinger and Sampson (2003) & Edinger et al. (2001a, 2007)	N/A	385 adults	DBAS-10	4 subgroups of insomnia (worried and medication biased; worried and symptom-focused; mild sleep worries; low endorsement) based on DBAS subscale scores Groups differed on insomnia severity, medication use, depression and anxiety, and daytime sleepiness Groups benefitted differentially from CBT
Morin, Blais, and Savard (2002)	Morin, Colecchi, Stone, Sood, and Brink (1999)	CBT vs. PHA vs. CBT + PHA vs. PLA	78 adults	DBAS	CBT and CBT + PHA improved more than PHA and PLA at post-treatment Reductions on DBAS were associated with improvements in sleep efficiency Improved scores at post-treatment were associated with better long-term improvements at 3-, 12-, and 24-month FU
Rybarczyk, Lopez, Schelble, and Stepanski (2005b)	Rybarczyk et al. (2002)	CBT vs. home-based video CBT vs. control	12 older adults with medical illness	DBAS TIB	CBT and video CBT groups had greater improvements from pre- to post-treatment than control group No significant differences between CBT and video CBT at post-treatment CBT and video CBT groups had greater TIB reductions from pre- to post-treatment than control group
Tremblay, Savard, and Ivers (2009)	Savard, Simard, Ivers, and Morin (2005)	CBT vs. control	57 adult females with breast cancer	DBAS Napping Adherence to prescribed to bedtime and rise time	Main effect of time (CBT group improved from pre- to post-treatment and 6-month FU) Improved DBAS scores predicted sleep improvements Higher avoidance of daytime napping predicted sleep improvements Higher adherence to prescribed bedtime and rise time predicted sleep improvements
Vincent, Walsh, and Lewycky (2010)	Vincent and Lewycky (2009)	CBT vs. cCBT vs. WL	100 adults	SLOC/PSAS	SLOC mediated impact of cCBT on insomnia severity at FU; individuals with a more internal SLOC following cCBT reported less severe insomnia at 4-week FU cCBT group had higher internal SLOC compared to CBT and WL Trend toward association between internal SLOC and increased cognitive arousal (PSAS)
Vitiello, Rybarczyk, Von Korff, and Stepanski (2009)	Rybarczyk et al. (2005a)	CBT vs. attention control	51 older adults with osteo-arthritis	Napping	No significant changes in napping from pre- to post-treatment and 1-year FU in either group

Note. cCBT = computerized CBT; DBAS = Dysfunctional Beliefs and Attitudes Scale; FU = follow-up; PHA = pharmacotherapy; PLA = placebo; PSAS = Pre-sleep Arousal Scale; RT = relaxation therapy; SLOC = sleep locus of control; TIB = time in bed; WL = waitlist.

on alertness and performance and demonstrated only a small amount of delta wave activity, suggesting that brief afternoon naps may not interfere with sleep improvements (Brooks & Lack, 2006). Nevertheless, this study was conducted in a sample of good sleepers and may not be generalizable to individuals with insomnia. Further, given that individuals with insomnia may be more inclined to nap to compensate for poor sleep, there may be practical concerns regarding the scheduling of and adherence to brief naps within this population.

Eight secondary analysis papers examined individuals' dysfunctional beliefs and attitudes about sleep. Relative to the primary RCTs, these studies provided more detailed information regarding changes in cognitions

over the course of treatment, as well as associations between these changes and sleep-related outcomes. Findings demonstrated that (a) participants receiving CBT-I experienced pre- to post-treatment changes in DBAS scores (Edinger et al., 2001b; Jansson-Fröjmark & Linton, 2008; Morin et al., 2002; Rybarczyk et al., 2005b; Tremblay et al., 2009), (b) participants receiving CBT-I experienced greater reductions in dysfunctional attitudes about sleep compared to control groups (Edinger et al., 2001b; Morin et al., 2002; Rybarczyk et al., 2005b), and (c) at post-treatment and follow-up, reductions in DBAS scores were associated with a range of subjective and objective sleep outcomes (Edinger et al., 2001b; Jansson-Fröjmark & Linton, 2008; Morin et al., 2002; Tremblay et al., 2009).

Sleep-related self-efficacy was examined in two studies (Currie et al., 2002; Edinger et al., 2008). In the first study of adults with chronic pain, individuals experienced significant increases in self-efficacy from pre- to post-treatment and follow-up. As well, lower self-efficacy prior to treatment resulted in greater sleep improvements following treatment, suggesting a moderating effect (Currie et al., 2002). The second study examined self-efficacy along with maladaptive sleep beliefs as a measure of pre-therapy unhealthy beliefs. Results indicated that individuals with higher pre-treatment maladaptive beliefs performed better in CBT (Edinger et al., 2008).

Finally, one secondary analysis paper examined the mediating effect of sleep locus of control on treatment outcomes among individuals who completed a computerized CBT-I protocol (Vincent et al., 2010). Results indicated that individuals with a more internal sleep locus of control following CBT-I experienced less severe insomnia at follow-up. Further, compared to waitlist and in-person CBT-I, individuals who had completed computerized CBT had a higher internal sleep locus of control. The PSAS was also included in this study; however, no significant findings emerged. No other studies examined cognitive or physiological arousal. Sleep effort was not examined in any of the reviewed studies.

8. Discussion

The purpose of this paper was to determine whether CBT-I works in accordance with its proposed treatment mechanisms and to assess the field's progress in understanding the mediators of CBT-I. The reviewed studies provide some evidence to suggest that CBT-I leads to changes in the cognitive, behavioral, and hyperarousal precipitating factors of insomnia. In general, following CBT-I, individuals with insomnia demonstrated reductions in time in bed, napping, bedtime and rise time variability, and hyperarousal. Improvements in maladaptive beliefs and attitudes about sleep, sleep-related self-efficacy, and sleep locus of control were also reported. Further, significant interactions were found for many of these variables, indicating that individuals receiving CBT-I experienced greater improvements in these processes relative to comparison groups. Consistent with theoretical models of insomnia, these results suggest that CBT-I appears to be leading to changes in the processes that are proposed to perpetuate insomnia. Nevertheless, it cannot be concluded based on the reviewed studies that changes in these variables are accounting for improvements in sleep outcomes; more research is needed to clarify these relationships.

First, of the reviewed RCTs, 39% of the studies included one or more of the proposed mediator variables. However, in statistical analyses, these variables were examined as dependent variables, measures of adherence, or moderators. For example, the DBAS was included in a large number of RCTs as an outcome variable, in which reductions in maladaptive beliefs were regarded as a measure of improved sleep (Edinger & Sampson, 2003; Jansson & Linton, 2005; Means et al., 2000; Rybarczyk et al., 2005a; Rybarczyk et al., 2002). In several secondary analysis studies, the DBAS was examined as a moderator, in which authors investigated the impact of individuals' pre-treatment DBAS scores on treatment response (Edinger et al., 2008; Espie, Inglis, & Harvey, 2001; Jansson-Fröjmark & Linton, 2008; Montserrat Sánches-Ortuno & Edinger, 2010). Although these types of analyses provide important information, they do not indicate whether changes in sleep-related beliefs are mediators of treatment outcomes. Performing mediational analyses would further advance the field by clarifying whether sleep improvements are a result of changes in these theorized variables. A number of statistical techniques are available to test for mediation, including the causal steps approach (Baron & Kenny, 1986), the product of coefficients approach (Sobel, 1982), and bootstrapping (Hayes, 2009).

One of the reviewed secondary analysis papers utilized bootstrapping analyses to examine sleep locus of control as a mediator of treatment impact and insomnia severity (Vincent et al., 2010). Results indicated that individuals who developed a more internal sleep locus of control over the course of treatment experienced less severe insomnia at follow-up. These analyses provided detailed information regarding the nature of change in CBT-I. It is important to note, however, that this study examined sleep locus of control in the context of computerized CBT-I. Both this mediator and method of treatment delivery have received minimal theoretical and empirical support; thus, results provide only preliminary information regarding whether CBT-I works according to its theorized mediators. Nevertheless, this study serves as a model for future treatment mediator studies as bootstrapping is a powerful method of testing mediation (Hayes, 2009).

A second concern about the reviewed studies was the relative lack of focus on certain mediator variables. For example, none of the reviewed RCTs or secondary analysis studies examined sleep effort in their analyses. This was surprising, given that sleep effort has been proposed as a core perpetuating factor of insomnia that can be measured using the GSES (Broomfield & Espie, 2005; Espie et al., 2006). Recently, studies have shown significant reductions in sleep effort following sleep restriction therapy (Kyle, Morgan, Spiegelhalder, & Espie, 2011) and combined mindfulness meditation and CBT-I (Ong, Shapiro, & Manber, 2008, 2009). Accordingly, an examination of sleep effort as a mediator of CBT-I is warranted in future RCTs. In addition, many studies did not report on time in bed and bedtime and rise time variability, in spite of having this information from sleep diaries. With the exception of a few studies (Edinger et al., 2001a, 2007, 2009; Lichstein et al., 2001; Rybarczyk et al., 2002), changes in time in bed throughout CBT-I were not reported. Rather, time in bed was often included in the calculation of sleep efficiency (percentage of time asleep relative to time in bed), which was examined as an outcome variable. Several other RCTs (Perlis et al., 2004; Waters et al., 2003) reported on these behavioral variables, but included them as measures of adherence to CBT-I behavioral strategies. Although this information is helpful in gauging individuals' level of engagement with the CBT-I strategies, it does not indicate whether practice of these techniques accounted for changes in sleep outcomes.

Finally, in spite of the extensive literature citing hyperarousal as a key maintenance factor of insomnia, a small proportion of the reviewed studies examined changes in arousal over the course of treatment. The studies that did examine cognitive or physiological arousal reported decreases over the course of treatment, with no mediational analyses conducted. Of note, a large number of the RCTs included polysomnography (PSG) data, which can provide detailed information regarding individuals' physiological arousal. However, these studies included PSG as a measure of objective sleep and therefore reported primarily on sleep-related outcome variables. Given that there are many available self-report and psychophysiological measures of arousal, future research should make efforts to examine these arousal processes more thoroughly. Further, it is recommended that researchers who have used PSG in past studies consider re-analyzing these data, looking specifically at changes in arousal over the course of CBT-I. Rather than developing new studies to examine physiological hyperarousal as a mediator of CBT-I, secondary

analysis of PSG data may be a more cost-effective way to examine these processes.

9. How can treatment mechanisms be assessed?

The lack of distinction between mediators, moderators, and outcome variables in the CBT-I literature raises important questions regarding the nature of these relationships. Based on the theoretical models of insomnia, it is presumed that CBT-I targets the behavioral, cognitive, and hyperarousal processes underlying insomnia, and changes in these processes lead to improved sleep outcomes. However, without conducting appropriate mediational analyses in RCTs, it becomes difficult to assess the causality and/or temporality of these relationships. This is particularly important given that CBT-I involves a range of different techniques, which may be differentially related to outcomes. For example, Harvey, Inglis, and Espie (2002) found that stimulus control, sleep restriction, and cognitive restructuring predicted significant improvements on sleep outcomes, whereas relaxation training and sleep hygiene did not.

To provide an example, one can consider a patient receiving CBT-I who has been introduced to a number of cognitive and behavioral strategies, including sleep restriction, thought records, and relaxation training. Following treatment, the patient reports significant sleep improvements based on sleep diary measures. She reports that she has considerably reduced the amount of time spent in bed, experiences decreased subjective arousal, and demonstrates a significant reduction in maladaptive beliefs about sleep. Based on this information, one could posit that any one of these factors (reduced time in bed, decreased arousal, and reduced maladaptive beliefs) or a combination of these factors contributed to her sleep improvements. However, it is impossible to determine the temporal and/or causal relationship between these variables from this vignette. The patient may have found that practicing relaxation exercises prior to bed decreased her cognitive arousal and resulted in improved sleep. As a result of improved sleep, the patient may have become less concerned about the consequences of poor sleep and thus demonstrated reduced maladaptive beliefs about sleep. Alternatively, the patient may have found that challenging her maladaptive beliefs about sleep led her to become more relaxed and less aroused, thereby improving her sleep.

Evidently, it is difficult to establish causal and temporal links between treatment and outcomes, particularly given the multi-component nature of CBT-I. Nevertheless, initial efforts to uncover mechanisms of treatment outcomes by using mediational analyses will allow for a better understanding of the nature of these relationships and guide future research (Kazdin, 2007; Kraemer et al., 2002). With regard to the temporality of these relationships, future studies could measure mediators at multiple time points throughout treatment. For example, participants could complete self-report measures (e.g., DBAS, PSAS, Sleep Self-Efficacy Scale) on a daily or weekly basis, perhaps at the same time as their completion of sleep diaries. Statistical techniques for longitudinal data, such as cross-lagged correlations (Kenny, 1975) or generalized estimating equations (Zeger & Liang, 1986) could then be used to examine the temporal relationship between mediator variables and sleep outcome variables.

10. Limitations

Although the current paper provides a list of proposed treatment mediators derived from the theoretical insomnia literature, there are other examples of mediator variables that warrant further investigation. For example, cognitive models of insomnia have proposed that sleep-related attentional bias is an important perpetuating factor in insomnia (Espie et al., 2006; Harvey, 2002). These models suggest that individuals with insomnia develop a heightened awareness of sleep-related threats, resulting in selective attention to sleep-related cues. This sleep-related attentional bias serves to increase individuals' anxiety about sleep and disrupt normal sleep processes. A number of measures may be used to evaluate changes in attentional bias over the course of CBT-I, including the Sleep Associated Monitoring Index (SAMI; Neitzert Semler & Harvey, 2004), an emotional Stroop task (Taylor, Espie, & White, 2003), and a flicker paradigm (Jones, Macphee, Broomfield, Jones, & Espie, 2005). Daytime physical activity is another variable that has been found to impact sleep quality (Carney et al., 2006) and may be relevant in future mediational research. However, given that it is not a primary target in CBT-I, it was not examined in this paper.

A second limitation of this study is that it focused exclusively on RCTs and RCT secondary analysis papers. However, non-RCT studies may also provide important evidence to elucidate how this treatment works. For example, in Harvey et al.'s (2007) open trial of cognitive therapy for insomnia, five process variables were examined throughout the course of treatment: (a) unhelpful sleep-related beliefs; (b) sleep-related worry and rumination; (c) cognitive arousal; (d) monitoring of sleep-related threat; and (e) safety behaviors. Results demonstrated that participants experienced significant reductions following treatment and at 12-months post-treatment across all process variables. Although this study did not utilize a randomized controlled design or specific mediational analyses, it offered additional support for the cognitive mechanisms underlying sleep improvement. It is important to note, however, that this study did not examine CBT-I; rather, it focused exclusively on cognitive therapy for insomnia and the specific maintaining processes proposed by cognitive theories of insomnia.

Third, this study did not include an examination of moderators (i.e., variables identifying on whom and under what conditions treatments work), despite their importance in the progression of this field (Kraemer et al., 2002). A number of the reviewed secondary analysis studies examined the moderating effects of DBAS scores by investigating the relationship between individuals' pre-treatment levels of maladaptive sleep-related beliefs and sleep outcomes. These studies reported that individuals who experienced more dysfunctional sleep-related beliefs prior to receiving CBT were more responsive to treatment (Edinger et al., 2008; Espie, Inglis, & Harvey, 2001; Jansson-Fröjmark & Linton, 2008; Montserrat Sánches-Ortuno & Edinger, 2010). Future research that includes moderation analyses will help to establish which participants may be more or less suitable for treatment (Kraemer et al., 2002). Building on this information, researchers may also wish to conduct moderated mediational analyses to determine whether the mediators discussed in this paper may be differentially associated with sleep outcomes among different individuals. For example, a study might examine the extent to which changes in DBAS scores account for sleep improvements throughout CBT-I (mediator) depending on individuals' pre-treatment levels of unhelpful sleep-related beliefs (moderator).

Finally, given that the purpose of this study was to investigate the CBT-I treatment literature's inclusion of mediator variables, this study did not evaluate the rigor of each RCT and secondary analysis study.

11. How are we doing and where do we go from here?

Overall, in spite of the strong theoretical and empirical basis for CBT-I, there is considerable work to be done in better understanding the mechanisms of this treatment. Currently, there is some evidence to suggest that CBT-I works in accordance with its proposed mediators. However, little is known about which mediators account for the greatest changes in sleep improvements and to what extent these mediators overlap. By examining these research questions, subsequent treatment studies may focus specifically on the CBT-I strategies that have the most impact on sleep outcomes in order to refine treatment protocols and deliver CBT-I in a more time- and cost-effective manner.

Perhaps one of the largest obstacles preventing the progression of this field is that there is currently no consensus regarding which treatment mediators are most important to explore. In order to better understand the treatment mechanisms of CBT-I, a first step might be

to focus on reaching a consensus regarding which mediator variables are important to examine and, subsequently, to make recommendations regarding the measurement of these variables. Buysse, Ancoli-Israel, Edinger, Lichstein, and Morin (2006) developed consensus recommendations for standard measures of insomnia by obtaining feedback from a panel of insomnia researchers who critically and systematically reviewed the extant insomnia research measures. The authors proposed that these guidelines be periodically updated to accommodate progress within the field. In light of the current review, future researchers are encouraged to follow a similar procedure in identifying mediators of CBT-I. Once research has established that certain mediator variables help to account for improvements in insomnia over the course of CBT-I, future RCTs and component analysis studies may be conducted to determine which aspects of this treatment are most integral to producing change. This information may be used to advance the field's understanding of CBT-I's treatment mechanisms, further refine CBT-I, and optimize treatment outcomes in the future.

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Appendix A

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