Published in final edited form as:
Sleep Med Clin. 2014 June 1; 9(2): 261–274. doi:10.1016/j.jsmc.2014.02.007.

Cognitive-Behavioral Therapy for Comorbid Insomnia and Chronic Pain

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Abstract

This article summarizes the literature on cognitive-behavioral therapy for insomnia (CBT-I) in patients with comorbid insomnia and chronic pain. An empirical rationale for the development of CBT-I in chronic pain is provided. The six randomized controlled trials in this area are described and contrasted. The data suggest that CBT-I for patients with comorbid insomnia and chronic pain produces clinically meaningful improvements in sleep symptoms. Effects on pain are inconsistent, but tend to favor functional measures over pain severity. Hybrid interventions for insomnia and pain have demonstrated feasibility, but larger trials must be conducted to determine efficacy relative to CBT-I alone. Future efforts should employ more comprehensive assessments of pain and psychosocial factors.

Keywords

Insomnia; Chronic Pain; Comorbid; CBT-I

Introduction

The majority of patients with chronic pain report poor sleep quality. Insomnia, the most prevalent form of sleep disturbance in chronic pain may directly contribute to poor long-term outcomes by impacting multiple dimensions of chronic pain pathophysiolgy and psychosocial functioning. Sleep loss impairs immune function (1, 2), emotion regulation (3, 4), cognitive function (5, 6), and heightens pain sensitivity (7). These processes are considered both vulnerabilities to and consequences of chronic pain (8-11). As such, there is a growing interest in the development and evaluation of sleep-related interventions among patients with chronic pain as well as individuals at risk for developing chronic pain.

Treating sleep disturbance in patients with chronic pain is of interest for several reasons. First, a recent systematic review indicates that psychological and pharmacological strategies for chronic pain management evidence only modest effects in reducing pain and pain-related

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disability (12). Targeting additional symptom clusters that may actively contribute to chronic pain severity and persistence, such as sleep disturbance, may be necessary to improve pain management outcomes. Furthermore, and perhaps somewhat surprisingly, recent prospective studies using rigorous statistical methodologies have suggested that in some idiopathic pain conditions, such as temporomandibular joint disorder (13), sleep disturbance might in fact be a more robust predictor of subsequent pain than vice versa. A recent laboratory study demonstrated that extension of sleep among individuals with mild chronic sleep loss was associated with reduced pain sensitivity (14). Thus, it is reasonable to hypothesize that interventions that target sleep in patients with chronic pain may produce improvements in pain symptoms as insomnia dissipates.

Cognitive behavioral therapy for insomnia (CBT-I) is the standard of care for treating chronic primary insomnia. The efficacy of CBT-I in primary insomnia is comparable to modern sedative hypnotics, with the advantages of long-term sustainability of treatment effects (15,16) and a favorable side-effect profile (17). CBT-I is standardized and can be implemented in a wide range of outpatient settings and formats (18-21). In the past decade, CBT-I has increasingly been investigated for the treatment of insomnia occurring in the context of medical and psychiatric comorbidities with promising results (22). The majority of these studies however, limited their primary outcomes to sleep parameters. A handful of researchers, however, have recently begun to study the effects of CBT-I on sleep and pain-related outcomes. The theoretical frameworks and some of the coping skills taught in CBT-I overlap with the skills taught in cognitive behavioral therapy for pain (CBT-P), but the two treatments also retain many distinct pain- and sleep-related components. There is a potential for skills taught in CBT-I to improve pain-related outcomes despite a primary focus on insomnia. Additionally, it is reasonable to hypothesize that the components of CBT-I and CBT-P may be blended into a hybrid intervention format that works synergistically on sleep and pain symptoms. Both of these possibilities have been tested in preliminary randomized controlled trials (RCTs), which are the focus of the present review. The objectives of this review are to: 1) summarize the findings of this “first wave” of published clinical trials, 2) discuss the limitations, and 3) identify promising directions for the “next wave” of studies.

CBT-I for Comorbid Insomnia and Chronic Pain

CBT-I is most commonly practiced as a multicomponent treatment that utilizes a variety of behavioral interventions and cognitive restructuring approaches. It is a short-term intervention that is typically conducted over 4-8 individual or group sessions. The most commonly employed therapeutic elements include: (a) general sleep education; (b) the application of operant and classical conditioning principles via stimulus control instructions; (c) the replacement of sleep-interfering behaviors with sleep-promoting behaviors through sleep hygiene education and behavior change counseling; (d) relaxation training (e.g., deep breathing; guided imagery); (e) the alteration of circadian regularity and alignment; (f) the manipulation of homeostatic sleep drive to consolidate sleep via sleep restriction (23); and (g) the use of cognitive therapy techniques to modify maladaptive sleep-related cognitions. Much of the therapy is data-driven, relying on the patient’s self-monitoring of daily sleeping patterns, including time in bed (TIB), sleep onset latency (SOL), wake after sleep onset (WASO), and total sleep time (TST).

Currie et al. (24)

Primary Findings—The first RCT of CBT-I for patients with comorbid insomnia and chronic pain (N = 60) compared seven, 2-hour CBT-I group sessions held weekly to a self-monitoring/wait list control (24). The components of therapy followed traditional CBT-I guidelines (see: Table 1), with one notable modification: the contribution of pain to sleep problems was specifically addressed through guided readings and group discussion. The authors found that patients in the CBT-I condition demonstrated significant improvements in daily diary-measured SOL, sleep efficiency (SE), WASO, and sleep quality. These effects were maintained at the three month follow up assessment. Actigraphic measures of nocturnal physical activity were also significantly reduced in the CBT-I condition. Notably, however, pain severity ratings did not significantly improve.

Analysis—This study established that CBT-I may produce clinically significant changes in self-reported and actigraphy-measured sleep for patients with insomnia and comorbid chronic pain. The use of a wait list control condition, however, significantly tempers the strength of this conclusion, because of the strong expectation for minimal improvement created by randomization to a wait list. That pain was not significantly reduced in the CBT-I group raises the possibility that clinical improvements in sleep do not directly lead to reductions in pain severity (24). However, methodological constraints may limit this interpretation. First, participants were selected on the basis of having insomnia symptoms that developed secondary to chronic pain, and information about their pain status prior to the development of insomnia was not available. Since it is not possible to determine if and to what extent the development of insomnia symptoms altered pre-existing pain symptoms in this sample, one cannot conclude that the lack of pain changes following treatment of insomnia necessarily indicate that CBT-I is inefficacious for pain symptoms. Rather, it could be that the pain symptoms, by virtue of having preceded the development of insomnia symptoms, were not being maintained by insomnia and therefore were not influenced by improvements in insomnia symptoms. This possibility could be evaluated in future studies by assessing time-contingent dependencies between insomnia and pain symptoms through daily diaries before and after treatment. A second limitation is that pain severity was the only pain-related outcome measured, and was considered a secondary outcome; a wider range of pain-related outcomes that measure the multidimensional nature of pain (e.g., pain interference; pain catastrophizing) would be necessary to adequately evaluate the broader influence of CBT-I on pain-related outcomes. Third, although groups did not significantly differ from pre- to post-treatment on changes in pain, the means trended in that direction. The between-groups effect size (Cohen's *d* (25)) of CBT-I vs. Control on pain was 0.51 at post-treatment and 0.64 at the three month follow-up. These are considered medium effect sizes (25). As a point of comparison, a recent meta-analysis of the effect of CBT for pain (CBT-P) shows small to medium effect sizes on pain severity, disability, and catastrophizing at post-treatment, with even smaller effects at follow-up (12, 26). The fact that reductions in pain severity were observed (though not to the point of statistical significance) at three months for the CBT-I group in the Currie et al. study might be considered clinically relevant, and raises the issue of whether longer-term follow up periods are needed to realize the full effect of CBT-I on pain and pain-related outcomes.

Rybarczyk et al. (27)

Primary Findings—Expanding upon the Currie et al. study, Rybarczyk et al. (27) compared CBT-I (N = 23) to a stress management and wellness control (N = 28) in a subsample of patients with comorbid insomnia and osteoarthritis (OA). The CBT-I intervention (see: Table 1) did not specifically address pain interference with sleep, and the OA patients participated in group sessions along with patients with other non-painful medical conditions. We focus here on the data for the OA/pain subsample only. Results indicated that, relative to the stress management control, CBT-I significantly improved daily diary-reported SE, WASO, and SOL (28). Pain on the McGill Pain Questionnaire (MPQ; 29), which measures both sensory and affective components of pain, was not significantly reduced by the CBT-I intervention. However, a follow-up analysis (28) revealed that CBT-I patients reported significantly less pain on the SF-36 Bodily Pain scale (30) at post-treatment, and tended to maintain those benefits at 1-year follow-up (p =.08).

Analysis—By including an active control condition, this study permitted a more rigorous test of CBT-I against a stress management control with similar cognitive demand and expectancy characteristics. Overall, the findings from this study confirmed the efficacy of CBT-I for self-reported sleep-related outcomes in patients with comorbid chronic pain and insomnia. Findings on pain-related measures were equivocal, perhaps owing to differences in measurement. The MPQ assesses sensory and affective characteristics of the pain experience, whereas the SF-36 is a measure that includes items measuring both pain severity and interference in function (30). Similar to the Currie et al. study, this study was primarily focused on sleep-related outcomes, and lacked a comprehensive pain assessment strategy. Nonetheless, the superiority of CBT-I over an active control on primary sleep outcomes offers compelling support for its efficacy in treating sleep among patients with insomnia and comorbid chronic pain.

Edinger et al. (31)

Primary Findings—Similar CBT-I benefits were observed in a small RCT involving patients with comorbid fibromyalgia and insomnia (31). Clinically significant improvements in daily diary SE and TST were observed in 43% (6/14) of CBT-I patients compared to 7% (1/15) in a control condition receiving only sleep hygiene education and 0/7 in a usual care condition. Objective actigraphic measures of sleep yielded comparable results, including shorter mean SOL and lower variability across days in both SOL and TST, suggesting that sleep became more reliable following CBT-I, an underappreciated, but common outcome associated with CBT-I. Across most sleep outcomes, treatment gains were maintained at 6-month follow-up. Significant differences between the CBT-I and control groups were not observed for pain outcomes (i.e., severity and sensory/affective pain), although means trended in that direction and effect sizes between the CBT-I and usual care groups at post-intervention were medium to large (Cohen's d was 0.51 and 1.74 for pain severity (32) and sensory/affective pain (29), respectively). Interestingly, patients in the sleep hygiene group were significantly different than usual care controls on both pain severity and sensory/affective pain measures. Subgroup analyses indicated that the association of sleep hygiene treatment and pain-related improvements was driven by a portion of patients in the sleep hygiene group who self-initiated the behavioral strategy of standardizing their sleep times

and achieved a 25% or greater reduction in time-in-bed variability. This subgroup evidenced a significantly greater reduction in self-reported sensory/affective pain than usual care controls.

Analysis—Overall, the results of this study suggest that CBT-I may be an effective intervention for patients with fibromyalgia, as it provided favorable results on both subjective and objective sleep criteria. However, as with prior studies in other chronic non-malignant pain populations, the results were equivocal with respect to the efficacy of CBT-I for reducing pain and pain-related symptoms. Fibromyalgia pain may be substantively different from pain associated with osteoarthritis and other types of degenerative musculoskeletal disorders, so it is difficult to compare general pain measures across studies. Future efforts with this population could expand outcome measures to include fibromyalgia-specific measures, such as a body map or the Fibromyalgia Impact Questionnaire (33). Further, because non-restorative sleep is such a prevalent complaint among fibromyalgia patients (34), it would be important to know if CBT-I improvements in SOL, SE, and TST are associated with improvements in the perception of restorative sleep.

Jungquist et al. (35)

Primary Findings—Another RCT (35) of patients with chronic non-malignant back and/or neck pain and comorbid insomnia found that, relative to a contact control (N = 9), CBT-I (N = 19) produced significant posttreatment improvements in daily diary-reported SOL, SE, WASO, number of awakenings, insomnia severity, and pain interference measured with the Multidimensional Pain Inventory (36). CBT-I did not significantly improve posttreatment daily diary-reported pain severity or pain disability measured with the Pain Disability Index (37). Although TST was not significantly enhanced at posttreatment--which is commonly the case among interventions that include sleep restriction--clinically and statistically significant gains in TST (23 minutes) were observed at 6 month follow-up (38). In addition, post-treatment reductions in pain interference were maintained at both 3 and 6 month follow-up periods, providing preliminary evidence for the sustainability of CBT-I effects on a key functional pain-related outcome.

Analysis—The multimodal (i.e., diary and single-occasion measurement), multidimensional (i.e., severity vs. disability and interference) assessment of pain is a particular strength of this study, and an important advancement in the literature. Whereas the self-reported perception of pain severity was essentially unaltered by CBT-I, the extent to which pain reportedly interfered with daily functioning was significantly reduced. One implication of this finding is that CBT-I may be more efficacious for improving the ability to cope with pain rather than perceptions of pain severity/intensity itself. It seems that, despite minimal changes in the severity of pain, patients are less inclined to perceive pain-related functional limitations following CBT-I. Although the reason for this distinction at present is unclear, we speculate that as the perception of pain interference declines, patients may be less likely to experience disability and impairment of social functioning (39). In turn, this may promote an increase in healthy pain coping behaviors. Such effects may not translate into pain severity changes in the short term, and the failure of CBT-I to produce short-term changes in pain severity, despite changes in pain interference, is consistent with

effects observed in trials of CBT-P, in which pain coping is directly targeted (26). Thus, longer-term follow ups in the range of one to two years may be warranted for these effects to be fully manifested. Such a study would be reasonable given the durability of CBT-I over these longer-term periods, and the general intractability of many chronic pain conditions.

Hybrid CBT-I for Comorbid Insomnia and Chronic Pain

If the inconsistency in pain severity changes across CBT-I trials in patients with comorbid insomnia and chronic pain is due to a narrow treatment focus on sleep behaviors and cognitions, it is possible that a hybrid intervention targeting both sleep and pain may produce more favorable results. Given the similarity in theoretical grounding of cognitive and behavioral skills taught in both CBT-I and CBT-P, it is reasonable to hypothesize that a hybrid of the two may be feasibly delivered and more efficiently treat both insomnia and pain symptoms. Two preliminary pilot studies have examined the effects of a hybrid intervention of CBT-I/P on pain and insomnia symptoms.

Pigeon et al. (40)

Primary Findings—A 10-week hybrid intervention was delivered to patients with chronic non-cancer pain and comorbid insomnia. The efficacy of the hybrid intervention (N = 6) was compared to CBT-P alone (N = 5), CBT-I alone (N = 6), and a wait-list control (N = 4) at post-intervention, but no follow-up time points. The hybrid intervention contained all components of both the CBT-I and CBT-P conditions (see: Table 1 for complete treatment components). The pain treatment components in both the CBT-P and hybrid conditions included pain psychophysiology education, relaxation training, pacing, pain –specific cognitive therapy, activity planning, problem solving, communication skills, pain flare-up planning, and relapse prevention (40). The timing and sequence of specific intervention components was not explicitly described. Results indicated that the largest effect for pain severity was observed in the CBT-P group, whereas the CBT-I and hybrid groups did not evidence improvements in pain severity (see: Table 2). CBT-P also produced the largest reduction in pain disability, but modest reductions on pain disability were also observed for CBT-I and the hybrid intervention (between-group Cohen's d = 0.28 and 0.35, respectively). With respect to insomnia severity, however, the largest effect was observed for the hybrid treatment, followed by CBT-I, and a smaller, non-significant effect for CBT-P. The hybrid and CBT-I interventions evidenced similar gains in diary-measured sleep continuity, including approximately 50 minute increases in TST from baseline, and mean SE above 90% (from baseline mean <70%) at post-intervention, both clinically significant (but not statistically significant) marks.

Analysis—In sum, the findings of this study establish the feasibility of a hybrid insomnia/pain intervention. Across both sleep and pain outcomes, the hybrid intervention was comparable to CBT-I. Interestingly, the hybrid intervention was not efficacious for pain severity and was not significantly different than control for pain disability, despite a modest reduction. The small sample size, however, precludes any firm conclusions. One interesting aspect of this study is the use of a CBT-P arm, which demonstrated benefits for chronic pain symptoms but failed to alleviate insomnia symptoms. As with the studies reviewed above,

the findings of Pigeon et al. (40) should be cautiously interpreted until a larger study is conducted.

Tang et al. (41)

Primary Findings—One other pilot study investigated the effects of a 4-week hybrid intervention (N = 10; see Table 1 for treatment component details) compared to a symptom monitoring control (N = 10) in patients with chronic heterogenous, non-malignant pain and comorbid insomnia (41). The sleep interventions included stimulus control and sleep restriction therapies. The pain treatment components in the hybrid intervention included pain education, behavioral activation, and cognitive therapy focused on reducing pain catastrophizing, safety-seeking behavior, and increasing positive reappraisal and growth strategies. Sessions lasted 2 hours, but the sequencing of individual components was not explicitly described. Significant improvements at post-intervention were observed for the hybrid intervention relative to the control in insomnia severity, diary-reported SOL, WASO, SE, and TST (see: Table 2 for between-group effect sizes), as well as actigraphically-assessed SOL, WASO, TST, and TIB. The hybrid intervention was associated with significant post-intervention improvements in pain interference, but not pain severity. Primary diary and actigraphy measures were not obtained on follow-up, but 1- and 6-month follow-up data were available for retrospective questionnaire data, which included insomnia severity, pain severity and interference. Significant pre-post gains in insomnia severity and pain interference were maintained at 1- and 6-month follow-up.

Analysis—Together, the data from the Tang et al. and Pigeon et al. studies confirm the feasibility of a hybrid intervention. Further, they demonstrate that brief (i.e., 4 sessions) and longer (i.e., 10 sessions) hybrid intervention formats produce comparable results on sleep outcomes. There was overlap in the content of both the Tang et al. and Pigeon et al. hybrid interventions (see: Table 1 for comparisons). Relative to the Pigeon et al. study, the Tang et al. intervention notably omitted relaxation and relapse prevention modules for both insomnia and pain. Pigeon et al. reported ‘pain-specific cognitive therapy’ as a therapeutic module, but did not provide additional details about the content of the cognitive therapy. In contrast, Tang et al. reported incorporating specific modules on positive reappraisal and growth, as well cognitive therapy to reduce pain catastrophizing. It is notable that, within the Pigeon et al. intervention, the hybrid condition included twice as many components in the same number of sessions as both the CBT-I and CBT-P comparison conditions. Although speculative, it is possible that the absence of hybrid intervention effects on pain severity in that study was due to patient difficulty in managing the demands of simultaneously learning a large number of pain coping skills and sleep habit changes. It would be interesting to see in future studies if reducing the number of skills taught improves pain-related outcomes in hybrid interventions (although Pigeon et al. noted that patients and therapists anecdotally remarked that the number of sessions could actually be reduced and still accommodate the material (40)). Future hybrid interventions may be better evaluated by measuring process and adherence measures in addition to discreet outcomes. For example, it would be useful to know if patients adhered to sleep and pain home practice assignments differently in the hybrid intervention compared to the CBT-I and CBT-P interventions.

Integrated Analysis and Future Directions

The available evidence consistently indicates that cognitive-behavioral interventions targeting sleep in chronic pain patients, including both stand-alone CBT-I and hybrid CBT-I/P interventions, are efficacious for a range of sleep diary and actigraphy derived outcomes, including sleep duration, continuity, and perceived quality. Further, CBT-I and hybrid interventions may improve pain-related outcomes, with greater effects on pain interference and disability relative to pain severity. The two preliminary hybrid interventions incorporating both pain and sleep elements seem to primarily benefit sleep, with modest effects on pain interference and disability. Despite the promise of these initial findings, they are limited by several factors that must be addressed in future research.

1. Sample Size—Small sample sizes in the treatment and control conditions of all of the RCTs to date threaten the reliability and validity of the data. Increasing sample size is the most straightforward way to improve reliability and validity in the outcome data available from existing trials.

2. Optimization of Sleep Outcome Measurement—A more complex issue than sample size is how to optimize outcome measurement. All RCTs reviewed used daily diaries to assess primary sleep outcomes, and 4 studies (24, 31, 35, 41) additionally employed actigraphic measurements. In general, the diary procedures are minimally described across studies, and only very limited data pertaining to participant adherence are available across studies. In several studies, it is not clear whether the diaries were paper/pencil or electronic, the latter of which can be objectively verified with time stamps and, therefore, may produce more reliable data. Thus, it is unclear whether diary entries were reliably made within specified windows, and how much retrospective bias may have interfered with self-report estimations. Further, no empirically validated methods are available to guide the analysis of actigraphy data for patients with comorbid insomnia and chronic pain. It is plausible, for example, that patients with certain chronic pain disorders may evidence greater WASO on actigraphy by virtue of heightened motor activity related to the pain source (e.g., leg movements due to knee osteoarthritis). This pattern was observed in the Jungquist et al. (38) study, in which actigraphy-assessed WASO was significantly greater than daily diary-assessed WASO. In contrast, Edinger et al. (31) did not report any systematic differences between diary and actigraphy-based outcomes. Future research should examine the validity of actigraphy as an outcome measure by determining if actigraphically-assessed sleep parameters are different in patients with certain types of chronic pain. As ambulatory polysomnography (PSG) becomes increasingly available and affordable, the reliability and validity of actigraphy data may be readily compared to that of PSG.

3. Optimization of Pain Outcome Measurement—Though interpreted with caution due to small sample sizes, the available data suggest that pain severity may not be robustly altered by CBT-I and hybrid CBT-I/P, and therefore may be a poor primary endpoint, at least when measured within 6 months. Other more functionally relevant outcomes, such as pain interference appear to be more strongly influenced by CBT-I and hybrid interventions (35, 41). The minimal effects on pain severity contrast with the experimental literature because they suggest that changes in sleep do not precipitate changes in pain. However, an

alternative explanation is that the pain assessments in clinical trials to date have not been sufficiently broad to capture the true variance or complexities underpinning the association of insomnia and chronic pain. For example, in future studies it may prove important to incorporate a broader range of diary-based pain-related outcomes and to evaluate time-variant contingencies in pain, sleep, functional disability, mood, and stress from day-to-day (42, 43). Such an assessment strategy would yield potentially important information about the psychosocial antecedents and sequelae of changes in pain and sleep from one day to the next, and whether those contingent daily associations change following treatment. In addition, it may be useful to investigate individual differences in response to quantitative sensory testing (QST). QST includes a range of nociceptive stimuli (e.g., thermal, pressure, electrodermal, etc.) that evoke a variable range of pain responses in individuals with and without chronic pain (8). Experimental studies have demonstrated that response to QST, including pain threshold (44-47) and endogenous pain inhibition (48), are altered by sleep deprivation. The evaluation of changes in QST responses throughout treatment may shed light on the ability of CBT-I to effect change on neurobiologically-mediated processes that may contribute to the maintenance of chronic pain, such as central sensitization (49, 50) and endogenous pain inhibition (51). The mechanisms by which such changes may take place are not clear at present, though sleep has been identified as a potential source of variance in these endogenous pain modulatory processes (48, 52). Additionally, physical function and psychosocial factors are associated with dysfunctional endogenous pain modulation (49, 53). If CBT-I and hybrid interventions are shown to reliably improve sleep and functional pain-related outcomes, it is reasonable to postulate that those effects may be associated with improved endogenous pain modulation, which might be hypothesized to precede changes in clinical pain report. The potential for such changes to affect clinical pain severity might be best determined over long-term follow-up (e.g., 1-3 years), and therefore may not be reflected in the results of the studies reviewed here.

4. Examination of Demographic Moderators—The reviewed studies were not large enough to investigate or explore the possibility that key moderators known to influence pain sensitivity and sleep, such as sex, age, and ethnicity (54-59), may have obscured effects. Future studies should be appropriately powered to examine these moderation models. It is possible, for example, that demographic subgroups vary in the magnitude or time course of response to CBT-I and/or a hybrid intervention.

5. Evaluation of Secondary Sleep-Related Phenomena—Another potentially important issue that creates complexity within this literature is the possibility that other sleep-related phenomena, such as central or obstructive apneas and periodic limb movements (PLMs), may influence some of the pain-related outcomes. Although all studies reviewed here attempted to identify many of these intrinsic sleep disorders for the purpose of exclusion through self-report (e.g., Structured Interview for Sleep Disorders), two studies (24, 41) did not use polysomnography to rule out sleep disorders other than insomnia, and the remaining studies varied in the thresholds used to exclude subjects based upon apnea-hypopnea index (AHI) and PLM index (see: Table 1). It is likely that severe cases of sleep apnea and periodic limb movement disorder were excluded from the studies, but it is possible that variability among individuals with mild apnea (e.g., AHI = 5-15) and PLMs

(e.g., PLM index < 15) may have influenced results. None of the studies reviewed attempted to control or assess the effect of these indices on outcomes. However, emerging data suggest, for example, that sleep-related hypoxemia may actually reduce pain sensitivity in chronic pain patients (60, 61). Other data suggest that nocturnal hypoxemia may increase pain sensitivity, especially in healthy subjects (62). Thus, future studies should covary continuous measures of hypoxemia and other relevant nocturnal phenomena to partial out any potential influence on primary outcomes.

6. Process-Oriented Evaluation of Hybrid Intervention Components— Theoretically, hybrid interventions may offer advantages over CBT-I for pain outcomes by incorporating strategies to behaviorally manage both sleep and pain (63). Larger hybrid studies may yet bear this out, as the effect sizes of CBT-I on pain severity are comparable to the effects of CBT-P on pain severity. But at present it seems that findings in the experimental (7,14, 44, 47, 48, 64) and longitudinal literature (13,13, 52, 65, 65, 66) that support a causal association of sleep and pain severity may not easily translate into the treatment context in this first wave of studies. That said, it may simply be the case that a more systematic refinement of hybrid treatment protocols may be needed to yield treatment gains. Issues related to the sequencing of pain- and sleep-related treatment components may be critical and have yet to be investigated. It may additionally be necessary to evaluate which sleep and pain components are the most compatible, and whether specific pairings of sleep and pain components enhances or detracts from outcomes. It is possible that flooding patients with too many skills over too short a period of time may have contributed to the relatively disappointing preliminary findings, particularly with respect to the Pigeon et al study. The Tang et al. intervention incorporated fewer treatment components, but needs to be tested against CBT-I and CBT- P with long-term follow-up to understand its true promise.

Larger scale investigations of CBT-I in patients with comorbid insomnia and chronic pain are currently under way. An ongoing RCT in our laboratory is comparing the effects of CBT-I to an active placebo (behavioral desensitization) on clinical pain and QST in patients with knee osteoarthritis and comorbid insomnia. Another ongoing RCT was described by Von Korff et al. (67), but data are not yet available. That study aims to provide the first large-scale test of a hybrid intervention for patients with comorbid insomnia and osteoarthritis. By comparing a hybrid CBT-I/P intervention to a CBT-P arm and an education control, the authors expect to observe improvements in both insomnia and pain symptoms by increasing sleep, promoting behavioral activation, and engendering positive emotions (67).

Conclusion

Insomnia and chronic pain evidence a high rate of comorbidity and have been regarded as reciprocally related conditions. Four RCTs have investigated the efficacy of CBT-I and two RCTs have investigated the efficacy of hybrid CBT-I/P interventions in reducing insomnia and pain symptoms. In general, these interventions demonstrate clinically meaningful improvements in sleep symptoms. Improvements in pain-related outcomes have been observed in functional domains, such as pain interference and disability, with limited

evidence supporting the short-term efficacy of CBT-I or hybrid interventions for pain severity. Hybrid interventions are feasible, but small sample sizes to date prohibit firm conclusions from being drawn about their efficacy relative to CBT-I or CBT-P. Future clinical trials should consider employing more comprehensive pain assessments with longer-term follow up assessment of at least one year or more. Measurement strategies that permit the analysis of time-variant contingencies in sleep, pain and associated psychosocial variables may also be particularly informative. Larger scale investigations are needed to clarify the limited efficacy data from the small pilot RCTs on hybrid interventions. Future studies aimed at developing hybrid interventions should be designed to investigate issues related to the sequencing of pain and sleep components and address the trade-off between the number of new skills patients are expected to master versus the quality and mastery of critical components. Hybrid approaches that combine sleep and pain intervention components continue to hold promise for improving the treatment of chronic pain among patients with comorbid insomnia.

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Key Points

- CBT-I for comorbid insomnia and chronic pain demonstrates clinically meaningful improvements in sleep symptoms, particularly sleep continuity.
- CBT-I for comorbid insomnia and chronic pain does not consistently improve pain severity, but appears to reduce pain interference and disability in several studies.
- Hybrid interventions that target both pain and insomnia have demonstrated feasibility in small pilot samples, but further conclusions must be deferred until a larger trial is reported.
- More comprehensive and dynamic pain assessment strategies may reveal effects for CBT-I and hybrid interventions not captured through single-occasion pain severity measures.

Table 1

Comparison of CBT-I and Hybrid Clinical Trials for Comorbid Insomnia and Chronic Pain

	Studies in which CBT-I was the active treatment									
	Treatment Types	**Study Sample**	**PSG Exclusion Criteria**	**Active Treatment Components**	**Treatment Duration**	**Treatment Format**	**Pain Measures**	**Sleep Measures**	**Significant CBT-Related Improvements in Pain?**	**Significant CBT-Related Improvements in Sleep?**
Currie etal. (2000)	CBT-I vs. Wait list control	Chronic Non-Malignant Pain + Insomnia N = 60	PSG not administered	GSE; SCT; SRT; SHE;RT;CT; Pain coping education	7 weekly sessions; 2 hrs/session	Group; 5-7 patients/group	MPI	Daily Diary; Actigraphy; PSQI	No	Yes **1** Diary SOL, SE, WASO **2** Sleep Quality **3** Nocturnal activity
Rybarczyk etal. (2005); Vitiello et al. (2009)	CBT-I vs. Stress Management Control	Osteoarthritis + Insomnia N = 51	AHI < 15 PLMI < 30	SCT; SRT; SHE; RT;CT	8 weekly sessions; 2hrs/session	Group; 5 patients/group	MPQ; SF-36 Bodily Pain	Daily Diary	Yes **1** SF-36 Bodily Pain	Yes **1** Diary SOL; SE; WASO
Edinger et al. (2005)	CBT-I vs. Sleep Hygiene Control vs. Usual Care	Fibromyalgia + Insomnia N = 47	AHI < 15 PLMI < 15	GSE; SCT; SRT	6 weekly sessions; 45-60 minutes in week 1, 15-30 minutes in weeks 2-6	Individual	MPQ; BPI	Daily Diary; Actigraphy; ISQ;	No	Yes **1** Diary SOL; SE; TWT **2** Actigraphy SOL and SOL variability; TST variability **3** Insomnia Severity
(Jungquist et al. 2010; 2012)	CBT-I vs. Contact Control	Chronic Non-Malignant Pain + Insomnia N = 28	AHI < 10 PLM not specified	SCT; SRT; SHE; CT	8 weekly session; 30-60 minutes/ session	Individual	Daily Diary; MPQ; MPI; PDI	Daily Diary; ISI; ESS	Yes **1** Pain Interference (MPI)	Yes **1** Diary SOL; SE; WASO; awakenings **2** Insomnia Severity
	Studies in which a hybrid CBT-I/P was the active treatment									
	Treatment Types	Study Population		Active Treatment Components	Treatment Duration	Treatment Format	Pain Measures	Sleep Measures	Significant Hybrid Treatment-Related Improvements in Pain?	Significant Hybrid Treatment - Related Improvements in Sleep?
Pigeon et al. (2012)	Hybrid CBT-I/P vs. CBT-I vs. CBT-Pvs. Wait list control	Chronic Non-Malignant Pain + Insomnia N = 21	AHI < 10 PLM I not specified	GSE; SCT; SRT; SHE; RT; sleep-related CT; sleep-related relapse prevention; pain education; pain-related CT;	10 weekly sessions; session time not reported	Individual	MPI; PDI	Daily Diary; ISI; ESS	No	Yes **1** Insomnia Severity[a]

	Studies in which CBT-I was the active treatment									
	Treatment Types	**Study Sample**	**PSG Exclusion Criteria**	**Active Treatment Components**	**Treatment Duration**	**Treatment Format**	**Pain Measures**	**Sleep Measures**	**Significant CBT-Related Improvements in Pain?**	**Significant CBT-Related Improvements in Sleep?**
				activity pacing; problem solving; communication skills training; pain-related relapse prevention activity pacing; problem solving; communication skills training; pain-related relapse prevention activity pacing; problem solving; communication skills training; pain-related relapse prevention activity pacing; problem solving; communication skills training; pain-related relapse prevention						
Tang et al. (2012)	Hybrid CBT-I/P vs. symptom monitoring control	Chronic Non-Malignant Pain + Insomnia N = 20	PSG not administered	GSE; SCT; SRT; CT; pain education; behavioral activation; pain-related CT	4 weekly sessions; 2 hours/session	Individual	Daily Diary; BPI; CISP; PSPS	Daily Diary; Actigraphy; ISI; DBAS; PSAS; APSQ	Yes **1** Pain Interference (BPI)	Yes **1** Insomnia Severity (ISI) **2** Diary SOL; SE; WASO; TST

Note. CBT-I = Cognitive-behavior therapy for insomnia; GSE = General sleep education; SCT = Stimulus Control Therapy; SRT = Sleep Restriction Therapy; SHE = Sleep Hygiene Education; RT = Relaxation Training; CT = Cognitive therapy; SOL = Sleep Onset Latency; WASO = Wake After Sleep Onset; SE = Sleep Efficiency; TWT = Total Wake Time; TST = Total Sleep Time; PSQI = Pittsburgh Sleep Quality Index; SII = Sleep Impairment Index; DBAS = Dysfunctional Beliefs About Sleep Scale; ISQ = Insomnia Symptom Questionnaire; ISI = Insomnia Severity Index; ESS = Epworth Sleepiness Scale; PSAS = Presleep Arousal Scale; APSQ = Anxiety and Preoccupation about Sleep Questionnaire; MPQ = McGill Pain Questionnaire; BPI = Brief Pain Inventory; MPI = Multidimensional Pain Inventory; PDI = Pain Disability Index; CISP = Catastrophizing in Pain Scale; PSPS = Pain Self-Perception Scale

[a]The hybrid group evidenced a larger effect size than CBT-I, but was not significantly different after adjusting for multiple comparisons

Table 2

Between-group effect size estimates for CBT-I and hybrid interventions on key outcomes at post-intervention

	Diary SOL	Diary SE	Diary WASO	Diary TST	Pain Severity	Functional Pain Measure
Studies in which CBT-I was the active treatment						
Currie et al.	0.76	1.02	0.94	0.40	0.51	--
Rybarczyk et al./Vitiello et al.	0.36	0.75	0.89	0.03	0.53	
Edinger et al.[a]	0.08	0.73	1.51	0.01	0.17	--
Jungquist et al.	2.28	1.95	1.69	1.12	0.81	0.67 (MPI) 1.06 (PDI)
Studies in which hybrid CBT-I/P was the active treatment						
Pigeon et al.[b]	--	1.48	--	0.66	0.37	0.39 (PDI)
Tang et al.	1.84	1.94	2.10	0.73	0.05	1.34 (BPI)

Note. Values are Cohen's d effect size estimates derived from between-group (treatment vs. control) comparisons at post-treatment time points.

[a] Study included multiple control groups; effect size derived from CBT-I/usual care comparison

[b] Study included multiple control groups; effect size derived from Hybrid/ wait-list control comparison

MPI = Multidimensional Pain Inventory, Pain-Interference Subscale; PDI = Pain Disability Index; BPI = Brief Pain Inventory, Pain-Interference Subscale