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Original Article

ORIGINAL ARTICLE

Cognitive behavioral treatments for insomnia and pain in adults with comorbid chronic insomnia and fibromyalgia: clinical outcomes from the SPIN randomized controlled trial

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Abstract

Study Objectives: To examine the effects of cognitive behavioral treatments for insomnia (CBT-I) and pain (CBT-P) in patients with comorbid fibromyalgia and insomnia.

Methods: One hundred thirteen patients (M_{age} = 53, *SD* = 10.9) were randomized to eight sessions of CBT-I (*n* = 39), CBT-P (*n* = 37), or a waitlist control (WLC, *n* = 37). Primary (self-reported sleep onset latency [SOL], wake after sleep onset [WASO], sleep efficiency [SE], sleep quality [SQ], and pain ratings) and secondary outcomes (dysfunctional beliefs and attitudes about sleep [DBAS]; actigraphy and polysomnography SOL, WASO, and SE; McGill Pain Questionnaire; Pain Disability Index; depression; and anxiety) were examined at posttreatment and 6 months.

Results: Mixed effects analyses revealed that both treatments improved self-reported WASO, SE, and SQ relative to control at posttreatment and follow-up, with generally larger effect sizes for CBT-I. DBAS improved in CBT-I only. Pain and mood improvements did not differ by group. Clinical significance analyses revealed the proportion of participants no longer reporting difficulties initiating and maintaining sleep was higher for CBT-I posttreatment and for both treatments at 6 months relative to control. Few participants achieved >50% pain reductions. Proportion achieving pain reductions of >30% (~1/3) was higher for both treatments posttreatment and for CBT-I at 6 months relative to control.

Conclusions: CBT-I and CBT-P improved self-reported insomnia symptoms. CBT-I prompted improvements of larger magnitude that were maintained. Neither treatment improved pain or mood. However, both prompted clinically meaningful, immediate pain reductions in one third of patients. Improvements persisted for CBT-I, suggesting that CBT-I may provide better long-term pain reduction than CBT-P. Research identifying which patients benefit and mechanisms driving intervention effects is needed.

Clinical Trial: Sleep and Pain Interventions in Fibromyalgia (SPIN), clinicaltrials.gov, NCT02001077.

Statement of Significance

Insomnia disorder is highly prevalent in fibromyalgia (FM). The current trial examined the effects of cognitive behavioral treatments for insomnia (CBT-I) and pain (CBT-P) in patients with FM. Both treatments improved insomnia symptoms immediately and 6 months following treatment, relative to waitlist control. CBT-I generally resulted in larger improvements. However, at 6 months, about half of patients in both treatments no longer reported difficulties initiating and maintaining sleep. Neither treatment improved pain or mood relative to control. However, both prompted clinically meaningful, immediate pain reductions in about one third of patients that persisted at 6 months for CBT-I. Both treatments are efficacious for insomnia in patients with FM and may reduce pain in some patients. CBT-I holds promise for long-term pain reduction in some patients.

Key words: cognitive behavioral treatments; CBT; comorbid chronic insomnia; fibromyalgia; pain; sleep

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Introduction

Fibromyalgia (FM), a chronic pain condition characterized by widespread pain and mechanical hyperalgesia [1, 2], affects approximately 4 million Americans and is associated with US$50 billion in health care costs in the United States [3, 4]. Insomnia Disorder—defined as at least 3 months of difficulty initiating and/or maintaining restorative sleep (DSM-5)—is comorbid with chronic pain, affecting 50% of those with FM [1, 2]. Thus, research investigating the interplay and treatment of these conditions is needed.

Although the association between pain and insomnia is bidirectional, research over the past decade suggests that sleep impairment may have a stronger impact on chronic pain than vice versa [5]. Insomnia not only exacerbates chronic pain, but also leads to the development of painful conditions [5]. Given evidence regarding sleep and pain interactions, research has examined the impact of cognitive behavioral treatment for insomnia (CBT-I), an established and highly efficacious treatment [6], on chronic pain. In a meta-analysis of 11 randomized controlled trials of patients with chronic pain, Tang and colleagues found that treatments incorporating at least one CBT-I component improved sleep (standardized mean difference = .68), pain (.18), depression (.24), and fatigue (.38) in chronic pain populations [7]. A limited number of studies have examined the efficacy of CBT-I among patients with FM [8–12]. Specifically, CBT-I demonstrated efficacy over sleep hygiene in improving pain-related rumination/helplessness ("catastrophizing"), and use of CBT-I techniques was associated with greater posttreatment reductions in pain [8–10]. These findings are underscored by the fact that one of the recommended treatments for pain—cognitive behavioral therapy (CBT-P)—also has small effects on pain [13, 14].

The promise of CBT-I in improving insomnia and pain symptoms raises the question of whether CBT-P also reduces pain in patients with FM. To date, only one study has examined the efficacy of CBT-P among this population, but outcomes were not compared with CBT-I. Instead, Lami and colleagues compared CBT-P with a combination treatment for pain and insomnia (CBT-PI) and found that the combination treatment improved pain intensity at 3 month follow-up [12]. This effect was not observed following CBT-P, indicating that CBT-I may be necessary to effect long-term reductions in pain intensity. This is consistent with the findings of Pigeon and colleagues' pilot trial comparing CBT approaches for insomnia and pain in a chronic pain sample [15]. The authors found that neither intervention significantly improved pain severity over the waitlist control (WLC), and CBT-P had only modest advantage over CBT-I in improving pain-related disability [15]. Vitiello and colleagues also failed to find significant differences between CBT-PI, CBT-P, and an educational attention control in reducing pain severity in a sample of patients with osteoarthritis and insomnia at 9 month follow-up, although CBT-PI had the largest within-group effects [16]. Collectively, studies suggest that CBT-I may lead to comparable effects on pain. However, this has not been tested in larger samples or patients with FM.

The Sleep and Pain Interventions (SPIN) trial addresses this gap in the literature. Specifically, SPIN compares the efficacy (i.e. magnitude of improvement relative to baseline) of CBT-I, CBT-P, and a WLC condition on sleep- and pain-related outcomes among patients with FM. We aimed to improve on the limitations of previous CBT-I trials in patients with FM, which were conducted by only two research groups and provided CBT-I primarily in group settings, by implementing an 8-session CBT protocol that was delivered individually. Consistent with previous trials in FM and chronic pain [7, 17], participants in both interventions were expected to report greater improvements in sleep outcomes (self-reported sleep onset latency [SOL], wake after sleep onset [WASO], sleep efficiency [SE], sleep quality [SQ]) and clinical pain (pain intensity ratings) than the WLC. Furthermore, based on previous research, we hypothesized that CBT-I and CBT-P would show similar improvement in clinical pain, and the WLC would not show improvement in clinical pain. Furthermore, we hypothesized that CBT-I would have greater effects than CBT-P on sleep outcomes than CBT-P, and both interventions would show greater improvement than the WLC group. We also examined the effects of CBT-I and CBT-P on objectively measured (i.e. through actigraphy and polysomnography [PSG]) secondary sleep outcomes of SOL, WASO, SE, and total sleep time (TST). Because CBT-I is theorized to affect insomnia symptoms in part as a function of change in dysfunctional sleep-related beliefs [18], we examined change in sleep-related attitudes and beliefs as a secondary outcome. Finally, because individuals with chronic pain are susceptible to symptoms of anxiety and depression, and CBT-I has been associated with improvements in these symptoms [7, 19], we also examined secondary intervention effects on anxiety and depression.

Methods

Overview

The SPIN randomized controlled trial compared changes in sleep and pain in patients with comorbid chronic insomnia and FM immediately and 6 months following 8 weeks of CBT-I, CBT-P, or usual care WLC. The University of Florida Health Science Center Institutional Review Board (IRB-01) approved the trial protocol (#627–2007). All participants provided written informed consent. This trial is registered at www.clinicaltrials.gov (NCT02001077).

Participants

Participants (N = 113) were recruited from rheumatology and sleep clinics at the University of Florida and from the surrounding area through community advertisements. General inclusion criteria were aged 18 or older, willing to undergo randomization, and able to read and understand English. FM criteria were pain for at least 6 months and confirmation of FM by tender point testing, using guidelines established by the American College of Rheumatology (with application of 4 kg force, participants reported pain in at least 11 of 18 points, including points in all four body quadrants) [20]. Chronic insomnia criteria were insomnia complaints (sleep onset or awake time during night >30 min) at least three nights per week for more than 6 months; sleep diary confirmation of insomnia (sleep onset or awake time during night >30 min) at least six nights during the 2 week baseline period; daytime dysfunction due to insomnia (mood, cognitive, social, or occupational impairment); and no prescribed or over-the-counter sleep medications for at least 1 month or stabilized on sleep medication for at least 6 months.

Exclusion criteria were sleep disorders other than insomnia, specifically sleep apnea (apnea–hypopnea index greater than 15 per hr or between 10 and 15 per hr with oxygen saturation below

88%) or periodic limb movements (PLMS index greater than 15 per hr); bipolar or seizure disorders (due to contraindication for the sleep restriction component of CBT-I); significant medical (e.g. cancer) or neurological disorder (e.g. dementia); severe untreated psychiatric comorbidity (e.g. schizophrenia and substance abuse); cognitive impairment based on Mini-Mental State Examination (MMSE) score below 26 [21]; and concurrent participation in CBT or other nonpharmacological treatment outside of the study. Participants taking pain medications as well as those with common psychological comorbidities (e.g. depression and anxiety) were included to increase generalizability.

See Figure 1 for the CONSORT (Consolidated Standards of Reporting Trials) flow diagram. Participants reported withdrawing from the trial for the following reasons: CBT-I (3—not benefitting from treatment, 2—relocation, 2—familial obligations, 4—unwilling to undergo posttreatment or follow-up evaluation, 4—no reason given), CBT-P (3—not benefitting from treatment, 1—relocation, 2—familial obligations, 2—unwilling to undergo posttreatment or follow-up evaluation, 2—no reason given), and WLC (1—relocation, 4—familial obligations, 3—unwilling to undergo posttreatment or follow-up evaluation, 5—no reason given). Three participants in each active treatment condition withdrew due to reported lack of treatment benefit. Otherwise, there were no significant harms or unintended effects. Participants were compensated US$100 following each assessment period. They received the treatment and parking on the UF campus at no charge. WLC participants were offered treatment (a hybrid combining CBT-P and CBT-I) at no charge upon completion of their follow-up assessments.

Procedures

Screening

First, the project coordinator conducted a brief, structured telephone interview to address inclusion/exclusion criteria

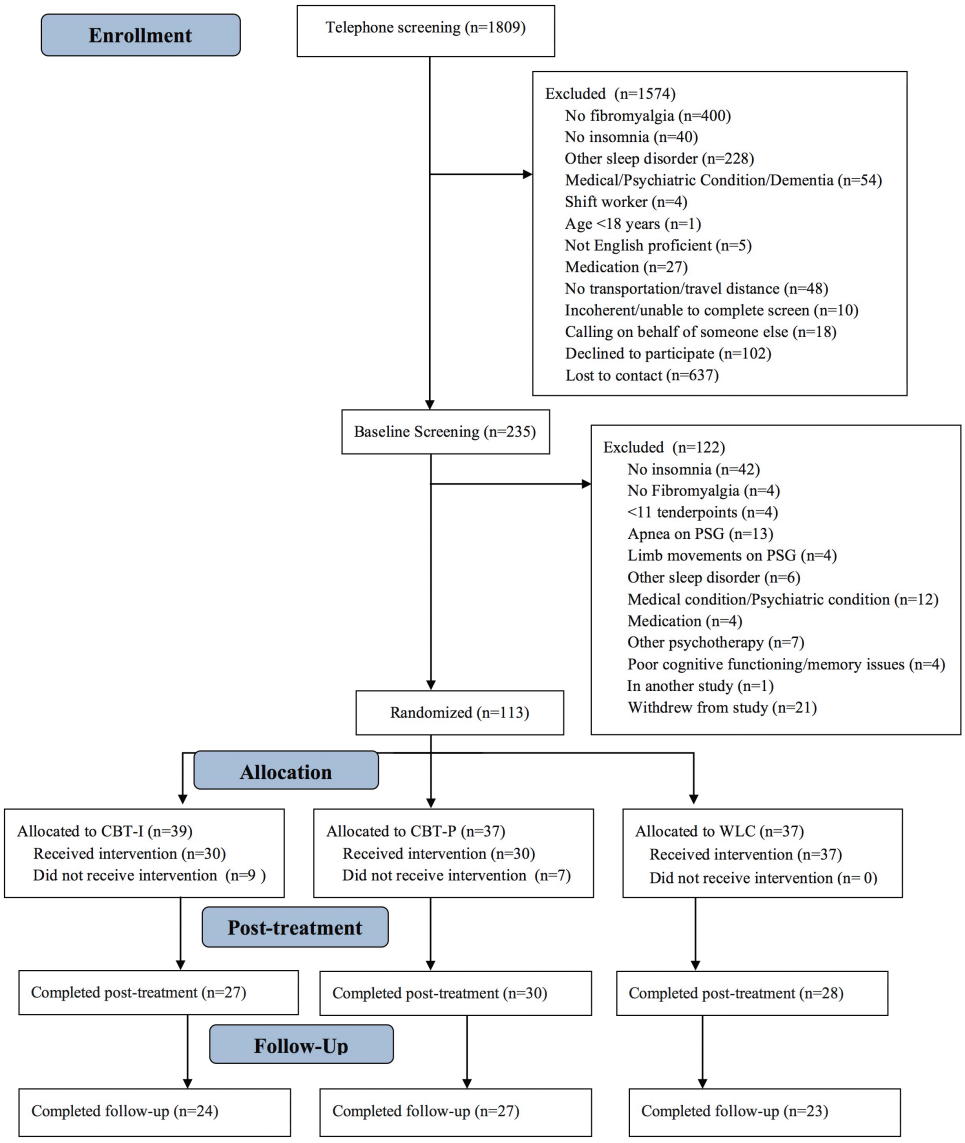

Figure 1. Participant recruitment CONSORT-style flow diagram. Allocated = randomized. Received intervention = completing all 8 sessions treatment sessions. *Details available from the first author upon request.

and establish probable FM and insomnia diagnoses. At the lab, study personnel conducted a semistructured clinical interview, performed tender point testing, and administered questionnaires to confirm FM diagnosis and assess mood. Then, participants underwent a single night of ambulatory PSG in their own homes to rule out sleep disorders other than insomnia. Finally, 2 weeks of sleep diaries were collected to confirm the insomnia diagnosis. Screening, with the exception of the telephone and clinical interviews and tender point testing, also served as the baseline assessment.

Randomization and masking

Participants were randomly assigned to condition by computer-generated block randomization (block size = 6). They provided informed consent and completed the baseline assessment period prior to being informed of their assignment by the project coordinator. Team members involved in recruitment data collection, and statisticians who conducted the analyses, were masked to assignment. Due to the nature of the treatment, interventionist and participant masking was not possible.

Interventions

Eight-session CBT-I and CBT-P protocols (Table 1) were manualized and individually delivered during 50 min sessions by predoctoral students in clinical psychology. There were three interventionists, and each delivered both interventions. Both treatments were developed by psychologists who had expertise in CBT-I (C.S.M.) and CBT-P (L.B.W.) and provided training, weekly supervision, and on-going monitoring of treatment delivery via audiotape. Lichstein, Riedel, and Grieve's model was used to guide training and treatment implementation [22]. Training involved mock therapy with corrective feedback and audiotaped practice sessions with volunteers. All treatment sessions were audiotaped. Half were randomly selected for scoring by another interventionist, and 25% of the scored tapes were double-scored for reliability by the lead supervising clinical psychologist (C.S.M.). Interventionist scoring of each other's tapes (not their own) was used because they were highly qualified to evaluate session content. Also, viewing others' tapes provided valuable booster training and enhanced consistency across interventionists. Session parts were weighted by importance and scored 0, 0.5, or 1 for no, part, or full delivery, respectively. Scores were summed to provide an index of the degree of treatment delivery. A separate index of treatment purity was calculated using a similar weighted scoring procedure. To ensure comprehension of treatment, participants were given a workbook detailing treatment instructions and rationale. They were also questioned during sessions about their home practice of techniques and procedural modifications were adopted as needed (e.g. pacing activities differently and adjusting bed/wake times). Interventionists encouraged adherence and emphasized the importance of regular home practice, which was monitored by daily practice logs. Participants also completed a 10-item quiz on treatment rationale and procedures at the beginning of session 3 and a treatment credibility questionnaire [23] at the end of session 3. Interventionists left the room prior to the completion of the credibility questionnaire, which was then completed by the participants, placed in a sealed envelope, and given to the project coordinator.

Measures

Sleep

Self-reported sleep. Participants were instructed to complete a daily diary assessing self-reported sleep and pain (see below) for 14 days at each assessment point. Diaries were completed via paper and pencil and were collected weekly. Participants provided subjective estimates of the following sleep variables: (1) SOL—time from initial lights out until sleep onset; (2) wake time after sleep onset (WASO)—time spent awake after initial sleep onset until last awakening; (3) TST—computed by subtracting total wake time (time spent awake from initial lights out until time out of bed in the morning) from the total time spent in bed; (4) sleep efficiency (SE)—ratio of total sleep time to total time spent in bed × 100%; and (5) sleep quality rating—rated from 1 (very poor) to 5 (excellent). Means were computed for these variables for each of the three 14 day assessment periods (baseline, posttreatment, and follow-up). Primary self-reported sleep outcomes are SOL, WASO, SE, and sleep quality.

Dysfunctional beliefs and attitudes about sleep. The dysfunctional beliefs and attitudes about sleep (DBAS) [18] consists of 30 questions intended to measure five dimensions: misconceptions about the causes of insomnia, misattributions or amplification of its consequences, unrealistic expectations, control and predictability of sleep, and faulty beliefs about sleep-promoting practices. Although the original scale used a 100 mm VAS, subsequent research with the DBAS [24] has used an 11-point Likert scale (0 = strongly disagree, 10 = strongly disagree). The latter response method was utilized in the present study. This measure had high internal consistency in our sample (α = .83).

Actigraphy. Participants wore an actigraph, the Actiwatch 2 (Phillips Respironics, Bend, OR), on their nondominant wrist 24 hr a day for 14 days at each assessment point (concurrent with daily diaries completion). The Actiwatch 2 monitors ambient light exposure and gross motor activity and contains an omnidirectional piezoelectric accelerometer with sensitivity of 0.01 λg-force or greater. The sensors of the Actiwatch 2 are sampled 32 times per second and record peak values for each second. Peak values are then summed into 30 s "activity" counts. Activity counts are downloaded onto a PC and analyzed using Actiware Sleep Analysis Software v.5.3.2, which uses a validated algorithm to identify each epoch as sleep or wake [25]. Bedtime and time out of bed in the morning were based on diary entries, as recommended in the software manual. Actiware Sleep determined sleep start automatically by searching for the first 10 min during which no more than one epoch was scored as wake. Likewise, sleep end was the last 10 min during which no more than one epoch was scored as wake. The software provides three default sensitivity settings (high, medium, and low). High sensitivity was used because it provides good correlation with PSG for SOL and TST (.70–.73) in individuals with insomnia [26]. Actigraphy provided behavioral estimates of the following secondary sleep outcomes: (1) SOL—interval between bedtime and sleep start; (2) WASO—sum of all the wake epochs within the time-in-bed period; (3) TST—sum of all sleep epochs within the time-in-bed period; and (4) SE—ratio of total sleep time to total time spent in bed × 100%. Variables were averaged for each 14 day assessment period.

Table 1. Treatment components

CBT for insomnia (CBT-I)
Session 1: Sleep Education
Education on sleep stages; sleep and fibromyalgia; and circadian rhythms and sleep was given to provide a heuristic background for the specific sleep techniques used.
Session 2: Sleep Hygiene and Stimulus Control
Sleep hygiene recommendations were:
(1) Avoid caffeine after noon
(2) Within 2 hr of bedtime, avoid exercise, nicotine, alcohol, and heavy meals
Stimulus control recommendations were:
(1) Do not use your bed or bedroom for anything (anytime) but sleep (or sex)
(2) If you do not fall asleep within 15–20 min, leave the bed and do something nonstimulating in another room. Return to bed only when sleepy. If you do not fall asleep within 20 min upon returning to bed, repeat this instruction as needed
(3) If you wake up and do not fall back asleep within 20 min, repeat #2
(4) Avoid napping
Session 3: Relaxation
A 10 min passive relaxation exercise was audiotaped and given to participants for daily practice at bedtime and once during the day
Session 4: Sleep Restriction
The amount of time spent in bed was tailored to the participants' reported total sleep time. A time-in-bed prescription was determined by adding 30 min to the participants' baseline average total sleep time. If this was <5 hr, the prescription was set at 5 hr. The interventionist and participant worked together to set regular/bed wake times to help the participant follow the prescription
Session 5: Cognitive Therapy—Monitoring Automatic Thoughts
Instruction was provided on recognizing thought patterns and emotional reactions that interfere with getting good sleep (i.e. I will never sleep well again)
Session 6: Cognitive Therapy—Challenging/Replacing Dysfunctional Thoughts
Instruction was provided on challenging the validity of sleep interfering thoughts and then replacing them with sleep conducive ones (i.e. There are things I can do to improve my sleep)
Session 7: Cognitive Therapy—Practical Recommendations
Established cognitive restructuring techniques (i.e. reappraisal, reattribution, and decatastrophizing) were taught
Session 8: Review of Skills and Long-Term Maintenance
Learned skills and the importance of maintaining a regular sleep schedule and good sleep habits were reviewed. Plans for gradually relaxing the sleep restriction schedule over time and continuing to use the techniques learned were discussed
CBT for pain (CBT-P)
Session 1: Pain Education and Diaphragmatic Breathing
Education on the gate control theory of pain (how pain signals are processed) was given to provide a heuristic background for the specific pain management techniques taught. Diaphragmatic (or belly) breathing was taught
Session 2: Progressive Muscle Relaxation
A progressive muscle relaxation exercise was audiotaped and given to participants for home practice
Session 3: Activity-Rest Cycle and Autogenic Relaxation
Relationship between pain and activity was discussed, and an individualized plan for adaptively pacing activities with appropriate rest was formed. An autogenic exercise was audiotaped and given to participants for daily practice
Session 4: Visual Imagery
A visual imagery exercise was audiotaped and given to participants for home practice
Sessions 5: Cognitive Therapy—Monitoring Automatic Thoughts
Instruction was provided on recognizing thought patterns and emotional reactions that contribute to pain (i.e. My pain is never going to get any better)
Session 6: Cognitive Therapy—Challenging/Replacing Dysfunctional Thoughts
Instruction was provided on challenging the validity of negative thoughts and then replacing them with more positive ones (i.e. I can't do everything the same as I used to but I can still do a lot of things). Importance of pleasant activities was discussed, and a plan for balancing work, responsibilities, and enjoyable activities was formed
Session 7: Cognitive Therapy—Balanced Thinking
Importance of creating balanced thoughts that incorporate all evidence to realistically appraise a situation was discussed. Practical recommendations for relaxing when there is insufficient time to do an entire session of one of the relaxation skills taught and when more than diaphragmatic breathing is needed
Session 8: Review of Skills and Long-term Maintenance
Learned skills and the importance of continued used of relaxation and the other techniques taught were reviewed. Plans were made for dealing with inevitable pain flare-ups

Ambulatory polysomnography. To rule out sleep disorders other than insomnia at the study outset and to obtain a physiological measure of sleep outcome, a single night of in-home ("ambulatory") PSG was conducted at the beginning of each assessment period using a 25-channel AURA Portable Recording System (Grass Technologies). Consistent with ambulatory PSG recommendations, monitoring consisted of 10 electroencephalography (EEG) measures (F2, C2, O2, ground, reference, M1, M2), 2 electro-oculography (EOG), and 3 chin electromyography (EMG) according to standard placements

[27–29]. Other standardized monitoring included respiratory inductance plethysmography (thoracic and abdominal effort), oximeter (pulse and oxygen saturation), electrocardiogram, right and left anterior tibialis EMG, oral-nasal airflow thermocouple, and nasal cannula pressure transducer. PSG records were scored by a registered polysomnographic technologist who was blinded to group assignment. Scoring procedures were based on those described by the Sleep Heart Health Study [29]. PSG provided the following secondary sleep outcomes: (1) SOL; (2) WASO; (3) TST; and (4) SE.

Pain

Clinical pain intensity. Pain intensity ratings were primary outcomes. On their daily diaries, participants were instructed to provide morning and bedtime ratings of current clinical pain intensity. Ratings were made using Visual Analogue Scales (VAS) with anchors of "no pain sensation" and "most intense pain imaginable." The VAS was instantiated on paper as a 10 cm horizontal line with the two anchors, and participants indicated their pain level by marking a spot on the line. To obtain a numerical score (0–10) for this rating, a ruler was used to measure the distance (in mm). Daily values were averaged to get a single rating of clinical pain per person. Morning and bedtime ratings of clinical pain intensity, a core pain outcome domain [30, 31], served as the primary self-reported pain outcomes and provided a pain corollary to the primary self-reported sleep outcomes, which were also collected daily. AM and PM pain ratings were collected to capture potentially important temporal relationships between pain and sleep [i.e. pain following a night of sleep (or lack thereof on some nights) versus pain following daytime activities] that might be differentially affected by the treatments.

Mcgill pain questionnaire. The McGill Pain Questionnaire (MPQ) [32] contains 78 items assessing participants' pain experiences and provides an overall total pain score (0 = no pain, 78 = severe pain) as well as evaluations of the sensory, affective, and evaluative dimensions of participants' pain experiences. Subscales on this measure had good internal consistency in our sample (α's = .72–.76). The MPQ was considered a secondary pain outcome.

Pain disability inventory. The Pain Disability Inventory (PDI) [33] includes 7 items rated on an 11-point scale (0 = no disability, 10 = total disability) indicating degree to which chronic pain interferes with participants' functioning in the following areas: family/home responsibilities, recreation, social activity, occupation, sexual behavior, self-care, and life-support activity. The seven ratings are summed to compute a total score (0–70). There was high internal consistency on this measure in our sample (α = .90). The PDI was considered a secondary pain outcome.

Mood

Two mood measures were evaluated as secondary outcomes.

Beck depression inventory-second edition. The Beck Depression Inventory-Second Edition (BDI-II) [34] contains 21 items that measure the severity of depressive symptomatology on a three-point scale (0 = absence of symptoms, 3 = most severe). Typically, respondents answer for the previous week, but the previous 2 weeks were used in this study to match the 2 week activity recording period for each assessment. Total scores range from 0 to 63. Ranges for clinical levels of depression are 0 to 13 (minimal), 14 to 19 (mild), 20 to 28 (moderate), and 29 to 63 (severe). There was high internal consistency on this measure in our sample (α = .86).

State-trait anxiety inventory-form y1. The State-Trait Anxiety Inventory-Form Y1 (STAI-YI) [35] asks respondents to rate how true 20 self-descriptive statements (e.g. "I feel calm") are on a 4-point scale (1 = not at all, 4 = very much so). Typically, respondents are asked to rate statements according to how they generally feel (trait–anxiety scale) and how they feel in the current moment (state–anxiety scale). However, for this study, participants based their ratings on how they generally felt over the preceding 2 weeks to match the 2 week activity recording period for each assessment. Total scores range from 20 to 80, with higher scores indicating greater maladjustment. There was high internal consistency within both the trait and state subscales in our sample (α's = .90–.93).

Statistical analyses

Power analysis

Sample size was determined by a power analysis in which moderate effect sizes (ESs of 0.5) were estimated for the group by time interactions based on prior studies of CBT-I (sleep ESs = 0.6–3, pain and mood ESs ~ 0.5) [8, 36, 37] and CBT-P (pain ES = 0.5 to 1.8, mood ESs = 0.4–1.9) [38, 39] in patients with chronic pain. Thus, for the group comparisons and the group by time comparisons with the WLC condition, power was expected to exceed .8, with alpha at .05, with 30 participants per group for the primary and secondary outcomes.

Baseline demographics and clinical characteristics

Group differences in baseline demographics and clinical characteristics were analyzed using analyses of variance (ANOVA) for continuous variables (age, number of health conditions, BMI, MMSE, duration of insomnia, and duration of FM) and chi-square analyses for categorical variables (gender, marital status, ethnicity, employment status, education, hypnotic use, and analgesic use).

Treatment outcomes

For each outcome variable, fixed effects of group (CBT-I, CBT-P, and WLC), time (baseline, posttreatment, and 6 month follow-up), and the group by time interaction were computed using Multilevel Modeling (MLM) analyses in SPSS software (version 25). Bonferroni-adjusted alphas were used to control for family-wise error, resulting in the following alpha criteria for group by time interactions for each outcome category: $p < .008$ (.05/6) for self-report sleep outcomes, $p < .0125$ (.05/4) for pain outcomes, $p < .0125$ (.05/4) for actigraphy and PSG outcomes, and $p < .025$ (.05/2) for mood outcomes. Significant main effects and interactions were followed by pairwise comparisons, with additional Bonferroni control ($p < .008$) applied to all outcomes to examine simple effects of group and/or time as appropriate. A priori planned comparisons revealed the magnitude of improvement over time for each group, as well as the difference between groups at each time point. Effect sizes were examined using hedges g (ES; .20 = small, .50 = medium, .80 = large) [40]. Hedges g was chosen as the effect size index as this has been

shown to impose less bias in the calculation of ES from smaller samples [41]. Across models, the most parsimonious random structure based on goodness of fit was used [22]. Factors were added to the random intercept of each model, and models were evaluated through restricted maximum likelihood estimation. Consistent with CONSORT guidelines on the reporting of RCTs [42], all available data from all randomized participants, including those who dropped out or were noncompliant, were analyzed using the "intention to treat" principle [43].

Clinical significance

Clinical significance was evaluated for insomnia and pain intensity. Because there are no established clinical significance guidelines for insomnia, participants were classified as no longer meeting trial criteria for difficulties initiating and maintaining sleep (i.e. self-reported SOL or WASO > 30 min on 3 or more days out of 14) at posttreatment and follow-up. In terms of pain, participants were classified as moderately and substantially improved (pain intensity decreases of 30%, and 50%, respectively) based on provisional benchmarks recommended for determining clinically important differences in pain intensity in clinical trials by the IMMPACT (Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials) Consensus Panel [41]. These improvement benchmarks were examined for both morning and evening pain intensity. Group differences were analyzed using chi-square.

Results

Participant characteristics

Table 2 provides the participant characteristics by group. The three groups did not differ on demographic or clinical characteristics with the exception of sex. CBT-P included three male participants, whereas the other two groups were exclusively female. Excluding male participants from the analyses did not affect the overall findings. Thus, the results reported below include all participants.

Baseline comparisons

There were no significant baseline group differences for any outcome (all *p*'s > .05).

Primary outcomes

Self-reported sleep. As shown in Table 3 and Figure 2, there were significant fixed effects for the group by time interaction for WASO, SE, and sleep quality rating. Within-group comparisons revealed significant posttreatment improvements in WASO and sleep quality for CBT-I and CBT-P (see Table 3 for within-group effect sizes). Posttreatment improvements in SE relative to baseline were observed for CBT-I, CBT-P, and WLC. These findings persisted at 6 month follow-up for SE and sleep quality. For WASO, the magnitude of improvement increased for CBT-P and decreased for CBT-I at 6 months. There were also significant main effects of time for SOL. Regardless of treatment condition, participants reported falling asleep faster at posttreatment (M = 29.23, SE = 3.17, p = .00) and follow-up (M = 34.66, SE = 3.42, p = .00) relative to baseline (M = 55.30, SE = 2.83).

Between group comparisons revealed that, at posttreatment, WASO only trended towards significantly lower values for CBT-I relative to both the CBT-P (p = .02) and WLC (p = .02) groups, which did not differ from each other. At posttreatment, SE was higher for CBT-I compared with the control (ES = 0.90) but only trended towards significantly higher values compared with CBT-P (p = .01, ES = 0.63), which did not differ from the control. In terms of sleep quality, posttreatment sleep quality was higher for both CBT-I (ES = 0.20) and CBT-P (ES = 0.13) relative to WLC, but the treatment groups did not differ from each other. At 6 months, WASO only trended toward significantly lower values for CBT-P (p = .02) and CBT-I (p = .06), relative to WLC, and the two treatments did not differ from each other. At 6 months, SE only trended towards higher values for CBT-I relative to WLC (p = .04), CBT-P did not differ from WLC, and the treatments did not differ from each other. Finally, at 6 months, sleep quality improvement was maintained for both treatments relative to the control, and again, the treatments did not significantly differ from each other.

Pain ratings. There were no significant group by time interactions for the morning and evening pain ratings. However, there was a main effect of time for morning pain. Regardless of treatment condition, participants reported less morning pain at posttreatment (M = 47.14, SE = 2.36) relative to baseline (M = 52.67, SE = 2.27, p = .004). This pattern persisted, with participants also reporting less morning pain at follow-up (M = 45.04, SE = 2.49, p = .006) relative to baseline.

Secondary outcomes

Sleep-related cognitions. As shown in Table 3 and Figure 2, there was a significant fixed effect of treatment group by time for dysfunctional attitudes and beliefs about sleep. At posttreatment, dysfunctional beliefs and attitudes about sleep decreased significantly for CBT-I compared with the control (ES = 0.90) and CBT-P (ES = 1.36) groups, which did not differ from each other. Similarly, at 6 months, dysfunctional beliefs and attitudes about sleep remained improved following CBT-I compared with CBT-P (ES = 0.78) and the control (ES = 1.27), which did not significantly differ from each other. Relative to baseline, there was significant posttreatment and follow-up improvement in dysfunctional attitudes and beliefs about sleep for CBT-I (ESs = 0.99) only. There were no significant group by time effects for total sleep time. However, there was a significant main effect of time for TST. Participants slept longer at posttreatment (M = 415.49, SE = 7.52, p=.00) and follow-up (M = 420.69, SE = 8.13, p = .00) compared with baseline (M = 385.06, SE = 6.75).

Actigraphy and polysomnography. There were no significant group by time interactions for either actigraphy or PSG (all *p*'s > .0125; see Table 4). However, there was a main effect of time and treatment group for polysomnographic WASO. Participants had less WASO at posttreatment (M = 58.01, SE = 4.97, p = .001) relative to baseline (M = 76.32, SE = 4.39). However, the comparison of follow-up (M = 66.83, SE = 5.36) to baseline did not survive Bonferroni control. When collapsed across time points, the CBT-I group (M = 54.37, SE = 6.17) had less WASO than WLC (M = 82.06, SE = 6.22; p = .002), but did not differ from CBT-P. CBT-P and WLC did not differ in WASO.

Pain questionnaires. There were no significant group by time interactions or main effects of group or time for any of the secondary pain outcomes (all *p*'s > .025; see Table 3).

Table 2 Participant characteristics by group

	CBT-I		CBT-P		WLC		
M	*n* = 39		*n* = 37		*n* = 37		P
Demographics							
Age, years; M (SD)	54.13	11.03	51.54	10.62	52.27	11.19	.56
Female (*n*; %)	39	100.00	34	91.89	37	100.00	.04
Marital Status (*n*; %)							.75
Single	9	23.08	6	16.22	14	37.84	
Married	18	46.15	18	48.65	13	35.14	
Cohabitating	0	0	1	2.70	0	0	
Widowed	2	5.13	2	5.41	2	5.41	
Divorced	9	23.08	9	24.32	7	18.92	
Separated	1	2.56	1	2.70	1	2.70	
Ethnicity (*n*; %)							.13
White	32	82.05	34	91.89	24	64.86	
Black	6	15.38	3	8.11	11	29.73	
Native Indian/Alaskan Native	1	2.56	0	0	1	2.70	
Biracia*	0	0	0	0	1	2.70	
Employed (*n*; %)	17	43.59	12	32.43	13	35.14	.57
Education (*n*; %)							.06
No High School Diploma	1	2.56	0	0	1	2.70	
High School Diploma	3	7.69	7	18.92	14	37.84	
Some College	7	17.95	7	18.92	3	8.11	
Associates Degree	11	28.21	7	18.92	6	16.22	
Bachelor's Degree	8	20.51	12	32.43	5	13.51	
Master's Degree	7	17.95	4	10.81	4	10.81	
Doctoral Degree	2	5.13	0	0	4	10.81	
Health Characteristics							
Conditions*	2.05	1.26	2.03	1.14	2.31	1.35	.58
BMI†; M (SD)	27.33	5.54	28.90	5.53	29.15	5.19	.27
MMSE; M (SD)	29.03	1.05	28.62	1.83	28.32	1.42	.12
Sleep characteristics							
Duration of insomnia; months; M (SD)	142.81	160.52	140.63	124.81	135.77	139.60	.98
Sleep Medication‡ (*n*; %)	13	33.33	17	45.95	17	29.73	.31
Benzodiazepines (*n*; %)	3	7.69	4	10.81	6	16.22	.50
Benzodiazepine-like Hypnotics (*n*; %)	2	5.12	4	10.81	0	0.00	.12
Antidepressants (*n*; %)	5	12.82	8	21.62	5	13.51	.51
Antihistamines (*n*; %)	5	12.82	5	13.51	3	8.11	.73
Pain Characteristics							
Duration of FM; months; M (SD)	114.52	91.10	94.64	76.16	109.46	88.62	.65
Pain medication§ (*n*; %)	12	30.77	18	48.65	10	27.03	.11
Opioids (*n*; %)	10	25.64	17	45.94	9	2.43	.08
NSAIDs (*n*; %)	5	12.82	1	2.70	2	5.41	.21

MMSE = Mini-Mental Status Exam; BDI-II = Beck Depression Inventory, Second Edition; STAI = State-Trait Anxiety Inventory; DBAS = Dysfunctional Beliefs and Attitudes about Sleep; MPQ = McGill Pain Questionnaire; PDI = Pain Disability Index; NSAIDs = Nonsteroidal anti-inflammatory agent.

*Total number of classes of health conditions from the following list: heart problems, cancer, hypertension, neurological disorder, breathing disorder, urinary problems, diabetes, pain, and gastrointestinal disorders.

†Body Mass Index = (weight/2.2046)/(height/39.37)^2.

‡Number of participants who reported currently using a prescribed sleep medication.

§Number of participants who reported currently using prescribed pain medication.

Mood. There were no significant group by time interactions for either mood outcome (both *p*'s > .025; see Table 3). However, there was a main effect of time for depression (i.e. scores on the Beck Depression Inventory–Second Edition). Regardless of treatment condition, participants endorsed fewer depressive symptoms at posttreatment (M = 13.68, SE = 1.03, p = .00) and follow-up (M = 12.54, SE = 1.09, p = .00) relative to baseline (M = 16.72, SE = .98).

Clinical significance

Insomnia-no longer reporting sleep initiation and maintenance difficulties. As shown in Figure 3, at posttreatment, CBT-I (χ^2 = 11.39, p = .00) had significantly more participants no longer reporting sleep initiation and maintenance difficulties than WLC (χ^2 = 9.70, p = .01), whereas CBT-P did not (χ^2 = 5.33, p = .07). At 6 months, both CBT-I (χ^2 = 5.88, p = .02) and CBT-P (χ^2 = 4.37, p = .04) had significantly more participants no longer reporting initiation and maintenance difficulties than WLC (χ^2 = 6.43, p = .04). CBT-I and CBT-P did not differ at either time point (χ^2 = 2.63, p = .11 and χ^2 = .18, p = .67, respectively).

Morning pain—moderately improved. As shown in Figure 4, CBT-I (χ^2 = 4.12, p = .04) and CBT-P (χ^2 = 6.77, p = .01) had significantly more participants who were moderately improved immediately following treatment than WLC, χ^2 = 6.78, p = .03. At

Table 3. Means and standard deviations for self-reported clinical outcomes

	Baseline		Posttreatment			6-mo Follow-up			Group		Time		Group x Time	
	M	*SD*	*M*	*SD*	ES	*M*	*SD*	ES	*F*	*P*	*F*	*P*	*F*	*P*
Morning pain intensity									.46	.64	5.91	.003	.97	.57
CBT-I	53.49	23.63	47.01	24.79	0.26	43.29	26.40	0.40						
CBT-P	54.04	23.67	46.72	23.81	0.30	47.78	24.45	0.25						
WLC	54.72	22.73	52.38	24.04	0.10	50.60	25.66	0.17						
Evening pain intensity									1.51	.225	3.04	.050	.49	.747
CBT-I	47.26	32.37	45.77	33.44	0.04	41.99	34.52	0.15						
CBT-P	54.26	32.04	49.39	32.61	0.15	49.77	33.35	0.13						
WLC	54.18	31.79	51.18	32.62	0.09	49.26	33.81	0.15						
McGill Pain Questionnaire									1.02	.364	2.05	.132	1.30	.273
CBT-I	25.85	13.15	26.26	15.01	0.03	23.62	16.22	0.15						
CBT-P	29.95	13.27	28.01	14.15	0.14	28.99	15.01	0.07						
WLC	28.53	13.40	29.84	14.53	0.09	23.30	16.02	0.35						
Pain Disability Index									2.90	.059	2.55	.081	1.46	.218
CBT-I	34.14	15.60	27.85	16.86	0.39	27.76	17.97	0.37						
CBT-P	37.27	15.25	38.03	15.95	0.05	36.37	17.20	0.05						
WLC	37.59	15.92	35.68	16.79	0.03	34.87	18.07	0.16						
Sleep onset latency (min)									.70	.50	33.36	.00	1.64	.17
CBT-I	58.30	30.03	22.25	34.77	1.09	33.97	37.50	0.70						
CBT-P	51.91	30.03	26.91	32.61	0.78	33.09	34.57	0.57						
WLC	55.68	30.03	38.52	33.53	0.53	36.91	37.07	0.55						
Wake after sleep onset (min)									1.39	.26	21.17	.00	4.72	.00
CBT-I	50.59	33.71	20.65*,†	14.67	0.86	33.34*	31.48	0.52						
CBT-P	50.39	30.72	37.90*	35.26	0.37	28.68*	22.67	0.80						
WLC	48.29	26.67	37.84†	26.05	0.32	46.85	35.04	0.02						
Total sleep time (min)									1.67	.19	11.88	.00	.92	.45
CBT-I	371.13	72.97	400.96	82.20	0.38	415.01	88.91	0.14						
CBT-P	397.56	72.97	426.52	77.96	0.38	442.50	82.56	0.56						
WLC	386.49	72.97	419.01	79.76	0.42	404.57	87.73	0.22						
Sleep efficiency (%)									2.84	.06	61.17	.00	5.82	.00
CBT-I	73.33	9.59	88.45*,†	9.60	1.55	84.59*,†	9.61	1.15						
CBT-P	75.51	9.38	82.44*	9.34	0.72	83.95*	9.35	0.88						
WLC	73.60	9.34	80.09*,†	9.35	0.68	79.23*,†	9.36	0.59						
Sleep quality rating									9.97	.00	34.91	.00	3.80	.006
CBT-I	2.62	3.43	3.32*,†	3.44	0.20	3.27*,†	3.45	0.19						
CBT-P	2.61	3.34	3.10*,‡	3.35	0.15	3.14*,‡	3.35	0.14						
WLC	2.47	3.34	2.66†,‡	3.35	0.06	2.65†,‡	3.36	0.05						
Dysfunctional attitudes and beliefs about sleep									13.03	.000	12.21	.000	8.29	.000
CBT-I	125.19	38.47	87.05*,†,§	37.21	0.99	86.41*,†,§	38.13	0.99						
CBT-P	136.74	37.48	137.93§	37.04	0.03	135.04§	37.68	0.04						
WLC	125.98	39.74	120.05†	38.14	0.15	116.93†	39.62	0.22						
Beck Depression Inventory—Second Edition									5.26	.007	9.56	.000	1.47	.215
CBT-I	14.08	10.37	8.52	11.12	0.51	8.22	11.93	0.51						
CBT-P	16.87	10.26	15.58	10.68	0.12	14.38	11.22	0.23						
WLC	19.12	10.53	16.94	10.94	0.20	15.01	11.68	0.36						
State-Trait Anxiety Inventory-Form Y1									4.00	.051	3.77	.025	1.00	.409
CBT-I	43.35	11.64	38.95	12.72	0.35	38.07	13.73	0.41						
CBT-P	45.55	11.76	45.22	12.12	0.03	43.86	12.78	0.13						
WLC	48.29	12.63	47.72	12.87	0.04	43.87	13.70	0.33						

Primary outcomes in bold text. CBT-I refers to the Cognitive Behavioral Treatment of Insomnia group (n = 39); CBT-P refers to the Cognitive Behavioral Treatment of Pain group (n = 37); WLC refers to Wait List Control group (n = 37). Sleep efficiency = Total Wake Time/Total Sleep Time X 100%. Sleep quality rated on a 0 (lowest) to 5 (highest) scale. Pain Intensity rated on a 0 (no pain sensation) to 100 (most intense pain imaginable) scale. Pain Unpleasantness rated on a 0 (not unpleasant) to 100 (most unpleasant pain imaginable) scale. ES = effect size (hedges g). Effect sizes represent magnitude of improvement relative to baseline. Guidelines for interpreting effect sizes (ES): .20 = small, .50 = medium, .80 = large[40].

*Indicates significant within-group difference compared with baseline (p < .008, Bonferroni adjusted).

†Indicates CBT-I and WLC differed significantly (p <.008, Bonferroni adjusted).

‡Indicates CBT-P and WLC differed significantly (p <.008, Bonferroni adjusted).

§Indicates CBT-I and CBT-P differed significantly (p <.008, Bonferroni adjusted).

6 months, CBT-I ($\chi^2 = 6.16$, $p = .01$) continued to have significantly more moderately improved participants than WLC ($\chi^2 = 6.23$, $p = .04$). However, the proportion of CBT-P participants with moderate improvement did not differ from that observed in WLC ($\chi^2 = 1.85$, $p = .17$). CBT-I and CBT-P did not differ from each other in terms of proportion of moderately improved participants at either time point ($\chi^2 = 1.42$, $p = .23$, and $\chi^2 = .43$, $p = .51$, respectively).

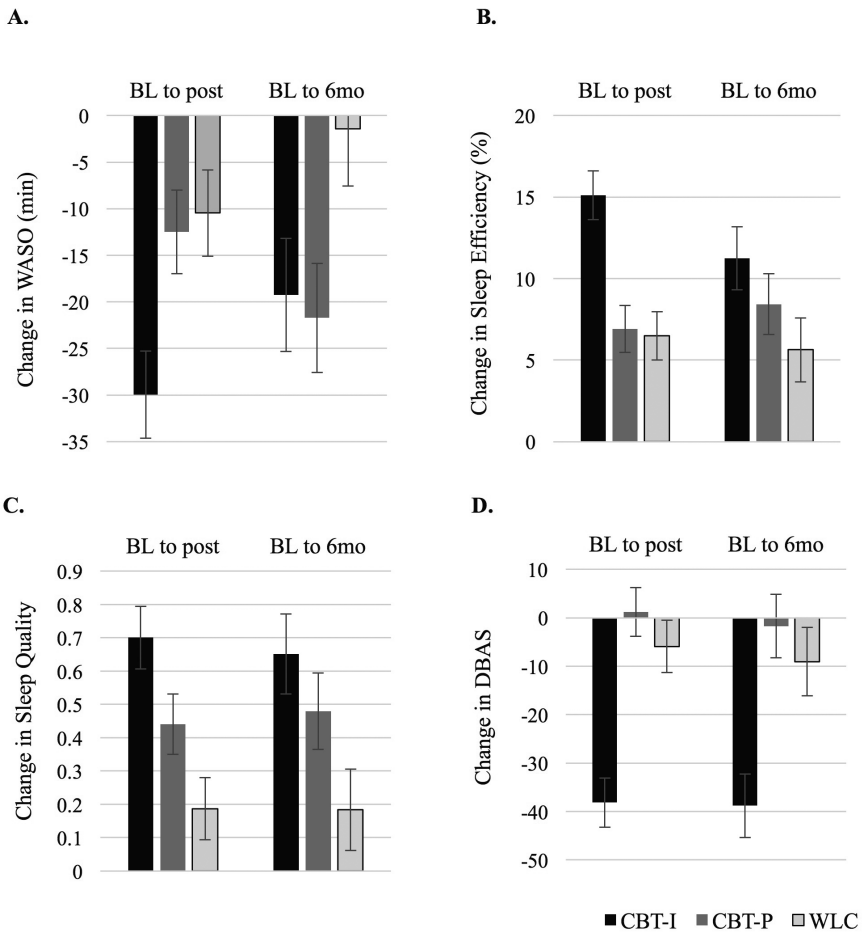

Figure 2. Within-group change in outcomes at posttreatment and 6 month assessments.

Morning pain—substantially improved. There were no significant group differences in proportion of participants substantially improved immediately following or 6 months following treatment. Each group's 50% response rate was low and remained the same from posttreatment to follow-up (Figure 4).

Evening pain—moderately improved. As shown in Figure 4, immediately following treatment, CBT-P had significantly higher proportion of participants with moderate improvement (χ^2 = 7.47, p = .01) than WLC (χ^2 = 6.66, p = .03). In contrast, immediately following treatment, CBT-I did not differ from CBT-P or WLC (p = .32). At 6 months, CBT-I had a significantly higher proportion of participants with moderate improvement (χ^2 = 6.45, p = .01) than WLC (χ^2 = 6.07, p = .048), whereas CBT-P did not differ from CBT-I (χ^2 = .82, p = .40) or WLC (χ^2 = 2.67, p = .10).

Evening pain—substantially improved. There were no significant group differences in proportion of participants substantially improved immediately or 6 months following treatment (Figure 4).

Treatment implementation

Sessions completed. Average number of sessions completed for CBT-I (M = 6.95, SD = 2.16) and CBT-P (M = 7.08, SD = 2.02) did not significantly differ.

Delivery. Average treatment delivery scores for CBT-I (M = 93.13, SD = 3.29) and CBT-P (M = 95.18, SD = 3.46) differed significantly, t = −2.52, p = .01. Although both group's averages were high (>90), scores for CBT-I were 2 points lower than CBT-P. The intraclass correlation coefficient between the 25% of sessions double-scored by lead supervising clinical psychologist (C.S.M.) and interventionist ratings was .93, indicating excellent reliability [44].

Receipt. Average score on the quiz assessing participants' comprehension of CBT-I (M = 9.3, SD = .83) and CBT-P (M = 9.4, SD = .92) was also high (highest possible score was 10).

Enactment. Average compliance rates for the sleep hygiene, stimulus control, and relaxation components of CBT-I were 91% during treatment, 86% at posttreatment, and 85% at follow-up. Likewise, the average compliance rates for the relaxation and activity pacing components of CBT-P were 90% during treatment, 90% at posttreatment, and 62% at follow-up.

Treatment credibility

Average total credibility scores for CBT-I (M = 8.93, SD = 1.21) and CBT-P (M = 8.38, SD = 1.37) did not significantly differ.

Drop-out analyses

Chi-square analyses to assess for systematic group differences in posttreatment [χ^2 (2, N = 113) = 1.44, p =.49] and follow-up [χ^2

Table 4. Means and standard deviations for actigraphy and polysomnography sleep outcomes for each treatment group as a function of time

	Baseline		Posttreatment			6 month follow-up			Group		Time		Group x Time	
	M	*SD*	*M*	*SD*	ES	*M*	*SD*	ES	*F*	*P*	*F*	*P*	*F*	*P*
Actigraphy														
Sleep onset latency (min)									2.63	.08	2.46	.09	1.31	.27
CBT-I	37.30	33.22	28.32	35.76	0.26	26.10	39.60	0.30						
CBT-P	40.55	33.03	42.69	34.79	0.06	44.42	37.29	0.11						
WLC	53.27	33.03	41.41	35.58	0.34	41.66	39.60	0.31						
Wake after sleep onset (min)									.38	.69	1.78	.17	.74	.57
CBT-I	52.09	24.68	45.33	26.47	0.26	47.92	28.28	0.15						
CBT-P	49.33	24.51	48.04	25.36	0.05	50.79	26.82	0.06						
WLC	53.71	24.51	52.72	25.79	0.04	52.15	28.10	0.06						
Total sleep time (min)									.88	.41	.93	.39	1.07	.37
CBT-I	392.20	75.06	375.82	83.81	0.21	402.18	90.37	0.12						
CBT-P	408.15	74.75	410.51	79.14	0.03	403.79	84.85	0.05						
WLC	394.63	74.73	386.26	80.99	0.11	388.96	90.39	0.07						
Sleep efficiency (%)									1.86	.16	2.17	.12	.89	.47
CBT-I	78.85	8.87	81.25	9.68	0.25	80.66	10.37	0.18						
CBT-P	78.49	8.82	78.98	9.18	0.05	77.35	9.79	0.12						
WLC	75.50	8.82	76.75	9.37	0.13	77.59	10.28	0.21						
Polysomnography														
Sleep onset latency (min)									.01	.99	.23	.80	1.64	.61
CBT-I	30.13	41.72	19.83	47.77	0.23	18.32	51.65	0.25						
CBT-P	22.27	41.73	22.96	45.01	0.02	23.92	47.68	0.04						
WLC	21.11	41.73	22.49	46.17	0.03	27.67	50.97	0.13						
Wake after sleep onset (min)									5.09	.008	5.92	.003	.38	.83
CBT-I	65.72	46.65	46.37	54.71	0.37	51.02	55.52	0.28						
CBT-P	77.08	46.65	52.32	51.03	0.50	64.83	40.04	0.69						
WLC	86.19	46.65	75.35	52.62	0.21	84.65	58.21	0.03						
Total sleep time (min)									.01	.99	.00	.99	1.02	.40
CBT-I	388.67	98.73	377.69	116.22	0.10	373.29	124.59	0.13						
CBT-P	367.27	98.72	393.12	108.27	0.24	373.02	114.66	0.05						
WLC	382.46	98.72	365.92	111.62	0.15	392.38	123.48	0.09						
Sleep efficiency (%)									3.44	.04	4.35	.01	.68	.61
CBT-I	81.08	11.74	86.33	13.80	0.40	85.83	14.80	0.35						
CBT-P	77.89	11.74	83.80	12.89	0.47	81.13	13.63	0.25						
WLC	78.43	11.74	79.64	13.26	0.09	78.41	14.65	0.001						

Primary outcomes in bold text. CBT-I refers to the Cognitive Behavioral Treatment of Insomnia group (n = 39); CBT-P refers to the Cognitive Behavioral Treatment of Pain group (n = 37); WLC refers to Wait List Control group (n = 37). Sleep efficiency = Total Wake Time/Total Sleep Time X 100%. Sleep quality rated on a 0 (lowest) to 5 (highest) scale. Pain Intensity rated on a 0 (no pain sensation) to 100 (most intense pain imaginable) scale. Pain Unpleasantness rated on a 0 (not unpleasant) to 100 (most unpleasant pain imaginable) scale. ES = effect size (hedges g). Effect sizes represent magnitude of improvement relative to baseline. Guidelines for interpreting effect sizes (ES) = .20 = small, .50 = medium, .80 = large[40].

(2, N = 113) = 1.37, p = .51] drop-out rates were not significant. Comparisons of drop-outs and completers revealed that participants who dropped out were more likely to be unemployed, have less education, and higher body mass indices (BMI). Specifically, individuals that completed the study (M = 15.13, SD = 2.44) reported 2 additional years of education, on average, compared with individuals that dropped out (M = 13.44, SD = 1.73), which was a significant difference, t = 4.25, p < .001. Moreover, although both groups were within the overweight classification, individuals that completed the study (M = 27.75, SD = 4.85) had a BMI that was, on average, 2 points lower than individuals that dropped out of the study (M = 29.82, SD = 5.60), p < .05.

Discussion

The primary objective of the SPIN randomized controlled trial was to compare the efficacy of CBT-I, CBT-P, and a WLC on insomnia and clinical pain symptoms immediately and 6 months following treatment in adults with comorbid FM and insomnia disorder.

Our hypotheses that both treatments would improve symptoms of insomnia (SOL, WASO, SE, and sleep quality), but that CBT-I would prompt greater improvements than CBT-P, were partially supported. CBT-I and CBT-P both prompted immediate improvements in 3 out of 4 insomnia symptoms—self-reported WASO, SE, and sleep quality, which were maintained at 6 months. The magnitude of improvement for WASO and SE for CBT-I can be considered large, whereas those for CBT-P generally ranged from small to moderate. For sleep quality, there was only a slightly larger effect size for CBT-I relative to CBT-P; however, both effects can be considered small in magnitude. Furthermore, CBT-P's impact on sleep efficiency at posttreatment and follow-up did not differ from controls. Additionally, unlike the findings for CBT-I, sleep efficiency at posttreatment did not

differ between CBT-P and controls. These findings are consistent with previous research, in which studies have typically found that CBT-I, either alone or combined with CBT for pain, has moderate to large effects on insomnia symptoms in comparison to usual care or educational control groups [8, 12, 15, 16]. In terms of clinical significance, the proportion of participants who no longer reported sleep initiation and maintenance difficulties was greater for CBT-I immediately following treatment and for both treatments at 6 months, compared with the WLC.

Both CBT-I and CBT-P were expected to improve insomnia symptoms based on prior research. However, CBT-I's larger impact on specific self-reported sleep parameters may be attributable to its use of techniques (e.g. stimulus control and sleep restriction) that specifically target insomnia symptoms. Another treatment-specific element of CBT-I, cognitive therapy focused on dysfunctional beliefs about sleep, may also partially account for the differential effects of the two treatments on insomnia symptoms. Dysfunctional sleep beliefs, another hypothesized mechanism of CBT-I, were the only sleep outcome that improved at both posttreatment and follow-up in CBT-I but not control or CBT-P groups. These findings also highlight the importance of understanding that CBT represents a class of interventions, and within that class, CBT-I and CBT-P are distinct interventions with distinct mechanistic impacts. For example, although CBT-P included cognitive therapy, it did not improve dysfunctional sleep beliefs. This is likely due to the focus in CBT-P on dysfunctional thinking related to pain. Such thinking is often referred to as pain catastrophizing or pain-related coping, and prior CBT-P trials have demonstrated improvements in these pain-related outcomes. Inclusion of pain catastrophizing and pain-related coping outcomes in future work in this area will help us to further clarify the differential effects of cognitive components of CBT-I and CBT-P on sleep and pain-related thought processes.

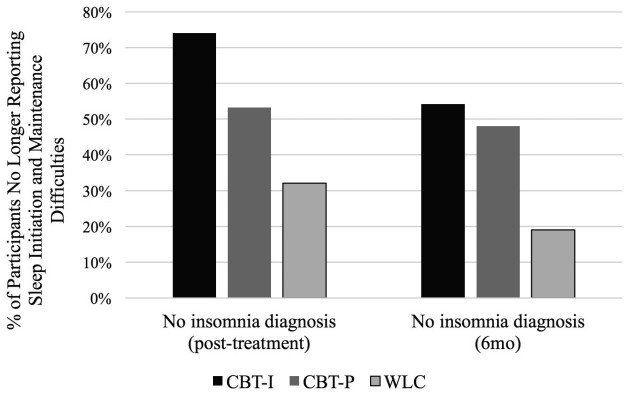

Figure 3. Percentage of participants no longer reporting difficulties initiating and maintaining sleep at posttreatment and 6 month assessments.

There were no significant group by time interactions for our objective sleep (actigraphy and polysomnograpy) outcomes. However, polysomnographically assessed WASO improved following treatment, regardless of condition. Furthermore, when collapsed across time points, CBT-I participants generally had less polysomnographically assessed WASO than CBT-P and control participants. Our lack of treatment-specific objective sleep improvements is consistent with other CBT-I trials [6, 45–47], and is not surprising, as insomnia (like chronic pain) is a condition diagnosed based upon patient complaints. For this reason, PSG is not recommended for the routine diagnosis of insomnia, but is indicated only if other sleep disorders are suspected (e.g. apnea and periodic limb movements disorder) [48]. Similarly, treatment response for both chronic pain and chronic insomnia is assessed based on patient self-report. Inclusion of PSG has increasingly become the standard in RCTs examining the impact of behavioral interventions on sleep. However, our lack of findings combined with those of prior trials raises the question of whether PSG should continue to be used as an outcome measure. Alternatively, given that PSG data were obtained through one night of assessment, this may not have captured the patterns of insomnia observed in daily self-report diaries. Potential night to night variation in polysomnographic values following CBT-I or CBT-P would be interesting to explore. However, other factors hinder this type of daily data collection of polysomnographic data, as well as its use in general as an outcome measure is sleep research studies. For instance, considerable resources are needed to conduct PSG (e.g. equipment and personnel), and patient burden is also quite high (e.g. hookup time, wearing the equipment overnight, and multiple assessments within in a relatively short period of time).

Our observed lack of improvement in actigraphy measures is, for the most part, consistent with prior insomnia intervention studies in FM. For instance, one study found that only one actigraphic sleep estimate (i.e. SOL) was associated with CBT-I [8]. That is, the authors reported overall better actigraphic SOL at

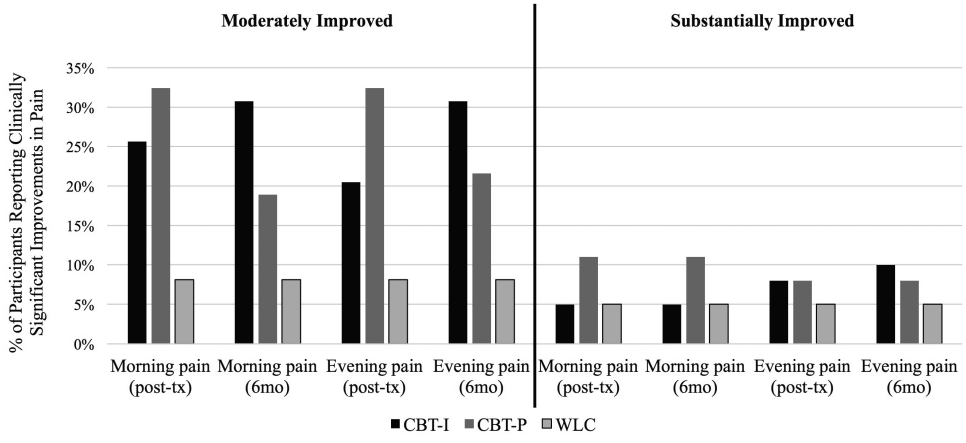

Figure 4. Percentage of participants reporting clinically significant improvements in pain at posttreatment and 6 month assessments.

posttreatment and 6 month follow-up for those who underwent CBT-I compared with usual insomnia care. Additionally, another non-CBT-I intervention pilot study (i.e. an 8 week functional respiratory intervention) found improvements in actigraphic SOL at posttreatment; however, the majority of improvement was observed for self-reported sleep (i.e. sleep quality, total sleep time, and sleep efficiency) [49, 50]. Taken together, research suggests that the improvement in actigraphy measures following insomnia treatments in FM miminal. Futhermore, given that there have been several reports of discrepancies between sleep diary and actigraphic measurements in FM [51], it is possible that actigraphy and diary measurements of sleep in FM are measuring different constructs. Overall, given the lack of objectively improved sleep in the present study, it may be important for future FM trials to consider other objective sleep measures (i.e. sleep microstructure/architecture obtained through PSG).

Although actigraphy outcomes did not improve, the collection of daily actigraphy has allowed us to address important questions about the daily relationship between sleep and pain [52]. Recent research suggests that actigraphy's usefulness may further extend to predicting treatment response. In a recent study, patients with primary sleep maintenance insomnia and objective short TST (<6 hr/night) based on 2 weeks of actigraphy prior to treatment did not respond as well to CBT-I as did patients with normal TST (≥6 hr) [53]. Specifically, patients with short TST on actigraphic evaluation reported significantly less improvement in terms of insomnia remission, SE, WASO, and total wake time compared with patients with normal sleep duration at 6 months after treatment. Given that study's focus on primary sleep maintenance insomnia, it is unclear whether those findings translate to the other populations, such as patients with comorbid insomnia and a broader range of insomnia symptoms (i.e. sleep onset difficulties alone or combined with sleep maintenance difficulties). Future research in this area appears warranted.

Contrary to expectations, improvements in pain were not treatment specific, as morning and evening clinical pain decreased significantly over time for all three groups. Additionally, there were no significant improvements in secondary pain outcomes of global pain experience or pain-related disability. Our examination of clinical significance revealed modest support for the clinical utility of both treatments for reducing clinical pain immediately following treatment, whereas long-term reductions were found for CBT-I only. Specifically, although very few participants achieved large reductions (>50%) in pain intensity, the number who achieved more modest pain reductions (at least 30%) was higher for both treatments for morning pain and for CBT-P for evening pain relative to the control at posttreatment. However, at 6 months, CBT-I (but not CBT-P) produced a greater proportion of participants who achieved reductions of at least 30% relative to the control. These findings provide limited support for our hypothesis that both treatments would affect pain. Consistent with Lami and colleagues' findings of long-term improvements in pain intensity following CBT-PI, but not CBT-P [12], these findings also suggest that CBT-I may be important for long-term pain reduction.

Findings regarding pain are not entirely surprising, given inconsistencies in the existing literature. Although prior research suggests that both CBT-I [8–10] and CBT-P [13, 14] hold promise for reducing clinical pain, findings have often shown greater improvement in pain-related symptoms (e.g. catastrophizing) rather than in clinical pain itself. Additionally, when pain improvements have been found, they have typically been small. Thus, one plausible explanation for our findings is that our trial was underpowered to detect small changes in pain. Tang and colleagues offered this same explanation for the contrast between their meta-analytic finding of significant improvement in pain following nonpharmacological treatments involving at least one component of CBT-I and the inconsistency in findings across individual trials [7]. Those researchers suggested the examination of individual response trajectories as one approach for increasing power to detect significant pain effects. Thus, future examination of patterns of within-person changes in pain over the course of treatment appears to be warranted.

The limited pain improvement in our trial may also be at least partially attributable to the presence of floor effects for pain. Pain severity was not considered when determining eligibility for the present trial and as a result, half of our participants reported average pain severity of less than 50 out of 100 possible at baseline. Use of insomnia severity criteria is an established methodological approach in the CBT-I literature, but has not been consistently used for clinical pain [8–10, 13, 14]. Additionally, previous research found group differences in pain only when limiting the sample to those with the most severe pain at baseline [16]; thus, we may have observed limited treatment-related pain improvement due to the lower average level of pain intensity reported by individuals in our study. Future examination of the potential impact of pain severity on pain outcomes is needed, and the adoption of pain severity criteria may be warranted.

Contrary to our prediction and previous research, there were no effects of treatment on self-reported mood, anxiety, and pain disability outcomes following either CBT-I or CBT-P. Given that we observed improvement in depressive symptoms (i.e. lower scores on the BDI-II) at posttreatment, regardless of treatment condition, it is also possible that mood changes are not specific to treatment. Instead, common elements of trial participation, such as the daily self-monitoring of behavior performed by all participants regardless of condition, may be associated with endorsing fewer depressive symptoms. This speculation is supported by the fact that participants did not continue reporting improvements at 6 months. Additionally, given that participants' average baseline scores on the BDI-II and STAI-Y1 would generally be considered representative of mild depression and anxiety [34, 35], it is possible that our results do not generalize to patients endorsing more severe symptoms. Therefore, future work should examine whether CBT-I and CBT-P improve anxiety and depression in patients with FM who have more severe symptoms at baseline. Furthermore, given that the pain disability index was assessed at one time point at baseline, posttreatment, and follow-up, it may not have captured the day to day changes regarding the impact of pain on daily functioning. Therefore, it may be important for future work to consider implementing daily assessments of this index in order to examine the impact of CBT-I and CBT-P on its daily variation.

Strengths of this trial include its RCT design, use of PSG to rule out sleep disorders other than insomnia, and assessment of both self-reported and objective sleep. Participants considered both treatments to be highly credible. Additionally, treatment integrity was strictly monitored using an established system [22] and was generally high. Treatment delivery was over 90% for both treatments. Participants understood their treatment based on scores over 90% on quizzes testing their knowledge

of their respective treatment. Likewise, logs indicated that participants were practicing the skills taught in treatment at home to a high degree (≥90%) immediately following treatment. Home practice was generally well-maintained at posttreatment for both treatments and at follow-up for CBT-I with a large drop (almost 30%) for CBT-P. Booster sessions may be useful in future research to help ensure high levels of continued skill enactment for CBT-P.

Potential limitations include lack of power (due to small sample size) to detect smaller effects and potential floor effects for pain and mood. Larger sample size and use of pain and/or mood severity criteria would increase the likelihood of detecting pain and mood effects in future trials. Our sample consisted primarily of middle-aged women, all of whom had comorbid FM and insomnia. Although this sex bias is representative of patients with FM, it is unclear whether our findings are more broadly generalizable to men with FM, patients with FM with subclinical insomnia or without insomnia, and individuals of both sexes with other chronic pain conditions. Use of paper and pencil daily diaries is a limitation, because there was no documentation of date of completion which could have implications for accuracy. Use of electronic daily diaries which provide a timestamp would increase the rigor of assessment of self-reported sleep and clinical pain outcomes in future trials. A global measure of insomnia severity was not included due to concerns about participant burden, given the trial's large number of outcomes, and represents a limitation. The use of a WLC is another potential limitation. Although our control participants were actively engaged in pharmacological treatment for insomnia and pain to a similar extent as our active treatment groups, an active control would provide a more rigorous test by controlling for nonspecific therapeutic factors.

Both CBT-I and CBT-P treatments led to improvements in self-reported sleep, with CBT-I prompting greater improvements that were maintained over time. Neither treatment prompted improvements in pain or mood relative to the control group. However, relative to the WLC, both CBT-I and CBT-P prompted clinically meaningful, immediate reductions in pain in about a third of patients that persisted at 6 months for CBT-I only. Future research that examines potential temporal effects of treatment on pain, identifies patients most likely to benefit from treatment (e.g. those with more severe levels of pain and mood), studies treatment effects on pain-related worry and coping, and investigates the mechanisms underlying treatment effects is encouraged.

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