Cognitive Behavioral Therapy for Insomnia Improves Sleep and Decreases Pain in Older Adults with Co-Morbid Insomnia and Osteoarthritis

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Study Objectives: Osteoarthritis pain affects more than half of all older adults, many of whom experience co-morbid sleep disturbance. Pain initiates and exacerbates sleep disturbance, whereas disturbed sleep maintains and exacerbates pain, which implies that improving the sleep of patients with osteoarthritis may also reduce their pain. We examined this possibility in a secondary analysis of a previously published randomized controlled trial of cognitive behavioral therapy for insomnia (CBT-I) in patients with osteoarthritis and co-morbid insomnia.

Methods: Twenty-three patients (mean age 69.2 years) were randomly assigned to CBT-I and 28 patients (mean age 66.5 years) to an attention control. Neither directly addressed pain management. Twelve subjects crossed over to CBT-I after control treatment. Sleep and pain were assessed by self-report at baseline, after treatment, and (for CBT-I only) at 1-year follow-up.

Results: CBT-I subjects reported significantly improved sleep and significantly reduced pain after treatment. Control subjects reported no significant improvements. One-year follow-up found maintenance of improved sleep and reduced pain for both the CBT-I group alone and among subjects who crossed over from control to CBT-I.

Conclusions: CBT-I but not an attention control, without directly addressing pain control, improved both immediate and long-term self-reported sleep and pain in older patients with osteoarthritis and co-morbid insomnia. These results are unique in suggesting the long-term durability of CBT-I effects for co-morbid insomnia. They also indicate that improving sleep, per se, in patients with osteoarthritis may result in decreased pain. Techniques to improve sleep may be useful additions to pain management programs in osteoarthritis, and possibly other chronic pain conditions as well.

Keywords: CBT-I, sleep, pain, osteoarthritis, insomnia, co-morbid

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Osteoarthritis is a common cause of pain and disability among older adults, affecting 20 million Americans. The prevalence of osteoarthritis is rapidly increasing with the accelerating growth of the older portion of the US population.1 Osteoarthritis is characterized by joint degeneration, pain, and dysfunction, with 80% of patients with osteoarthritis experiencing limitations of movement.1 Osteoarthritis demonstrates a broad spectrum of symptom severity, ranging from intermittent aching and joint stiffness to loss of motion and severe chronic pain.2 Severity and disability tend to increase with age, although severity can fluctuate markedly over short periods of time.

Sleep quality is a major concern among persons with osteoarthritis, with 60% of people with osteoarthritis reporting pain during the night.3 In fact, pain secondary to arthritis is the most common factor predicting sleep disturbance in the population at large.4 It is well established that pain interferes with sleep5 and, more recently, that disturbed sleep lowers the pain threshold.6,8 Whether sleep disturbance precedes or follows pain onset is unclear, but reciprocal effects are likely.3 Patients with osteoarthritis who report having pain and stiffness in the morning have more sleep-related muscle spasms and objectively assessed sleep disturbance.9 Even after treatment with anti-inflammatory medications, patients with osteoarthritis show significantly greater objective sleep disturbance, as compared with age-matched control subjects.10 Chronic sleep disturbance, so common among older patients with osteoarthritis, is itself associated with impaired daytime function, daytime sleepiness and fatigue, reduced quality of life, and increased health care utilization.11,12

Given the likely reciprocal effects between pain and sleep disturbance, teasing apart unique causal pathways is difficult. Chronic pain initiates and exacerbates sleep disturbance; disturbed sleep in turn maintains and exacerbates chronic pain and related dysfunction.5,13-14 Sleep disruption, fragmentation, or restriction produces hyperalgesia6,8 and can interfere with analgesic treatments involving opioidergic and serotonergic mechanisms of action.13 The basis for this reciprocal relationship may be the modulation of pain during sleep and waking by reciprocally ac-

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Questionnaire (MPQ) across the
group showed no reductions in pain report on the McGill Pain
of measures were included in their analyses. The CBT-I–treated
investigated the hypothesis that improvements in sleep would
not specifically address pain management. However, the study
investigated the hypothesis that improvements in sleep would
result in improvements in daytime functioning, so a broad array
of measures were included in their analyses. The CBT-I–treated
group showed no reductions in pain report on the McGill Pain
Questionnaire (MPQ) across the 3 co-morbid medical illnesses,
or for the osteoarthritis group alone, relative to an attention-
control group.15 However, Rybarczyk and colleagues analyzed
neither a second available pain measure (ie, SF-36 pain sub-
scale) nor the within-group effects. Given the possibility that
the attention-control group might have received some pain
benefits, examining within-group effects is an important ana-
lytic consideration. To better explore the potential impact of
improved sleep on osteoarthritis pain, we reanalyzed the Ryba-
czyk et al. data, using within-group analyses to examine both
available measures of pain, as well as previously unavailable
1-year follow-up sleep and pain data, for osteoarthritis partici-
pants only. We also examined effects among participants who
crossed over from the control group to the CBT-I treatment.

METHODS

A full description of the parent study—detailing patient re-
cruitment and screening, etc.—may be found in the report by
Rybarczyk et al.15 Here we report only the methodological
information relevant to our above-stated purpose. The study
protocol was approved by the Rush University Medical Center
Institutional Review Board, and all participants gave written
informed consent at the time of enrollment.

PARTICIPANTS

Twenty-three patients with osteoarthritis (mean age 69.2, 18
women and 5 men) were randomly assigned to CBT-I and 28 pa-
tients with osteoarthritis (mean age 66.5, 27 women and 1 man)
to an attention-control stress management and wellness (SMW)
intervention. Demographic and other characteristics of study
subjects by treatment group are reported in Table 1. Subjective
ratings of sleep quality and pain were assessed by patient self-
report before treatment, after treatment, and at 1-year follow-up.

Procedures

Participants were recruited by placement of brochures, mem-
os, and flyers in places where medical patients who qualified
for the study might see them, ie, community presentations (e.g.,
senior centers) and letters to individuals from mailing lists ob-
tained from non-patient sources (e.g., American Arthritis Foun
dation membership mailing list) and physicians supporting the
study. Participants were paid up to $200, based on how much
of the protocol they completed. Enrollment occurred between

Potential volunteers who contacted the investigators about
the study were initially screened by telephone. The inclusion
criteria used at this stage were (1) age 55 or older; (2) at least 3
episodes of insomnia per week for at least 6 months (problems
with sleep onset, sleep maintenance, or a combination of both
were allowed, defined as taking at least 30 minutes to fall asleep,
being awake for at least 60 minutes after falling asleep, or ac-
cumulating less than 6.5 hours of sleep per night); and (3) day-
time consequences of insomnia, such as fatigue, irritability, or
difficulty concentrating. After passing the telephone screening,
participants were required to undergo a night of home polysom-
ographic assessment to exclude individuals with sleep apnea
or sleep disorders other than insomnia. Twenty-two individuals
with suspected sleep apnea were eliminated from the study, and
2 additional individuals were eliminated based on t-scores of 70
or higher on subscales other than anxiety and somatization on
the Brief Symptom Inventory.18

To meet criteria for osteoarthritis, an individual needed to
report physician-diagnosed osteoarthritis (confirmed by a radi-
ograph or magnetic resonance imaging study) in 1 or more joints
(including the knees, hips, lower back, neck, fingers, thumb, or

### Table 1—Demographic and Other Baseline Characteristics by Treatment Group

<table>
<thead>
<tr>
<th></th>
<th>CBT-I (n = 23)</th>
<th>SMW (n = 28)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, no.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Women</td>
<td>18</td>
<td>27</td>
</tr>
<tr>
<td>Age, y&lt;sup&gt;a&lt;/sup&gt;</td>
<td>69.2 (8.9)</td>
<td>66.5 (7.7)</td>
</tr>
<tr>
<td>Education, y&lt;sup&gt;a&lt;/sup&gt;</td>
<td>14.7 (3.8)</td>
<td>13.8 (2.6)</td>
</tr>
<tr>
<td>Race, no.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>16</td>
<td>17</td>
</tr>
<tr>
<td>African American</td>
<td>5</td>
<td>11</td>
</tr>
<tr>
<td>Hispanic</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Chronic illnesses, no&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.57 (.66)</td>
<td>.59 (.46)</td>
</tr>
<tr>
<td>Insomnia duration, y&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.5 (0.6-7.5)</td>
<td>4.8 (0.6-49.0)</td>
</tr>
<tr>
<td>Medications&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep</td>
<td>30% (7)</td>
<td>29% (8)</td>
</tr>
<tr>
<td>Pain</td>
<td>35% (8)</td>
<td>50% (14)</td>
</tr>
</tbody>
</table>

Abbreviations: CBT-I refers to cognitive behavioral therapy for insomnia; SMW, stress management and wellness.
<sup>a</sup>Data are presented as mean (SD).
<sup>b</sup>Data are presented as median (range).
<sup>c</sup>Percent of subjects (n) taking over-the-counter or prescription medications for sleep at least once in a 2-week period, or daily medications for pain.
big toe), ongoing treatment from a physician, and at least “moderate” pain ratings on the SF-36 pain item addressing the degree of “bodily pain” or the pain-rating item from the Arthritis Impact Measurement Scales 2.19 One exception was made for an osteoarthritis participant who reported mild pain but had the highest possible Arthritis Impact Measurement Scales 2 scores for the number of joints that are painful and the degree of joint stiffness.

Per the design of the study, participants in the SMW treatment who continued to demonstrate sleep difficulties at the post-treatment assessment were offered the opportunity to cross over to CBT-I treatment after the post-treatment assessment. This design was based on previous experience by the authors suggesting that study retention over a 1-year follow-up period in this population was likely to be substantially compromised if or when sleep was not improved after treatment. Twenty-three of the 28 SMW subjects continued to have sleep problems that met study criteria. Of these, 13 began CBT-I treatment, and 12 completed both the treatment and the 1-year follow-up assessment. The 2 primary reasons for subjects choosing not to cross over were schedule conflicts and not wanting to participate in another class. Data from these additional 12 subjects were added to the CBT-I group for analyses presented in Table 3.

Interventions

The CBT-I and SMW (attention-control condition) interventions consisted of 8 weekly 2-hour classes matched in as many characteristics as possible. Class sizes ranged from 4 to 8 participants, averaging 5 members per class. All classes were conducted at an academic medical center in downtown Chicago and were spread out over the calendar year. Participants who missed a class were given make-up classes for up to 2 missed sessions.

CBT-I Protocol

The CBT-I intervention protocol closely followed Morin’s insomnia treatment protocol,20 with the exception of an added relaxation-training component. Sessions were led by 2 experienced clinical psychologists. Each session included a didactic presentation, a question-and-answer period, a review of each individual’s sleep log, and group discussion to solve problems encountered during implementation of the techniques. The 2 main behavioral components, stimulus control21 and sleep restriction22 were introduced and emphasized during the first 3 sessions. A strict schedule of bedtimes and arising times was prescribed to consolidate sleep and decrease time spent awake during the night. Patients initially reduced their time in bed to the amount of time they were actually sleeping, according to their pretreatment sleep logs, but not less than 4.5 hours. Sleep logs were completed continuously during treatment, and the bedtime was moved earlier by a maximum of one-half hour each week if there was sufficient improvement in sleep efficiency, usually defined as achieving 85% sleep efficiency. Participants were also instructed to lie awake in bed no longer than 15 minutes, at which time they were to go to another room, engage in a non-stimulating task in a dimly lit room, and return to the bed only when they felt sleepy again. No activities were permitted in bed other than sleep and sex. The third component to be introduced was cognitive restructuring, which emphasized changing unrealistic beliefs and irrational fears regarding sleep or loss of sleep. The fourth component was relaxation training, designed to decrease anxiety and reduce cognitive and physiologic arousal at bedtime. Each participant was given a relaxation audiotape that included the following 4 commonly used modalities: deep breathing, progressive muscle relaxation, autogenic training, and imagery. A final component of the intervention was sleep-hygiene education, including the use of increased daytime bright-light exposure to address any circadian causes of insomnia. Topics included increasing natural-light exposure, daytime activity, and exercise; reducing caffeine and alcohol intake; keeping an appropriate bedroom temperature; reducing ambient noise in the bedroom; using warm baths in the evening; and using appropriate food choices and eating patterns. The CBT-I treatment condition made no mention of pain management.

SMW Treatment

The SMW treatment, adapted from Rybarczyk et al.,23 consisted of didactic presentations and corresponding skill training covered the following 6 topics: (1) the mind-body relationship, (2) modifying self-talk for the reduction of stress and anxiety, (3) effective communication and assertiveness, (4) problem solving and goal setting, (5) nutrition, and, (6) exercise for individuals with chronic conditions. Topics 1 and 2, covered over 4 separate class sessions, were presented by a physician with extensive training and speaking experience regarding mind-body health. Topics 3 and 4 by were presented by psychologists, and Topics 5 and 6 were presented by an expert nutritionist and exercise physiologist. At the beginning of the SMW class, participants were given a presentation of a treatment rationale based on the common public perception of an interrelationship between sleep problems and stress, nutrition, and exercise. This model suggests that improvements in daytime coping and wellness lead to improvements in sleep. As a substitute for an active treatment of relaxation training, participants were given brief instruction in breath awareness. Providing such a quasi-relaxation training procedure was deemed essential to increasing the credibility of this program as a treatment for insomnia.

The SMW intervention was designed as an attention control for CBT-I and did not include an active treatment for relaxation or specifically mention pain control, but it did have several components that have been included in effective multi-component interventions for management of chronic pain. Although the SMW intervention targeted coping with chronic medical conditions in general (not chronic pain, per se), it included problem-solving, goal-setting, cognitive approaches to reducing stress and anxiety, interpersonal skills training, and education about exercise enhancement. These components have been used in various forms in previous studies of CBT interventions for coping with chronic pain.24 We assumed that analgesic effects of these nonspecific interventions were likely to be modest, but SMW conceptually might have had some therapeutic benefit for pain outcomes.

Study Measures

Although the parent study employed an extensive battery of self-report measures, here we report only those measures directly relevant to our specific stated purpose.
The SF-36 is a 36-item scale designed to assess the following 8 health concepts: physical functioning, role limitations due to physical health problems, social functioning, general mental health, role limitations due to emotional problems, general health perceptions, vitality, and bodily pain. The SF-36 has demonstrated adequate reliability and validity. For the present study, we report only the Bodily Pain Subscale (SF-PAIN), which is comprised of a question addressing “How much bodily pain have you had during the past 4 weeks?” (ranging from 1 “none” to 6 “very severe”) and a question asking “During the past 4 weeks, how much did pain interfere with your normal work (including both work outside the home and housework)?” (ranging from 1 “not at all” to 5 “extremely”).

**Geriatric Depression Scale**

The Geriatric Depression Scale (GDS) was developed specifically for use with older adults and uses a simple “yes/no” answer format. The GDS scale comprises 30 items, none of which reflect the somatic and vegetative aspects of depression, thus reducing possible confounding of depressive and age-related medical-illness symptoms. We report GDS data to address the possibility that any observed reduction in pain might be the result of improvements in depression status.

**Statistical Analysis**

In light of the small sample of patients with osteoarthritis who were available, we assessed within-subject pre-treatment to post-treatment change in each of the 2 groups separately using paired t-tests to maximize the ability of the analysis to detect clinically important change. The α level was set at ≤ 0.05. The parent study only tested for treatment effects on pain by using a repeated measures analysis of variance examining group X time effects. Within-group effect sizes (Cohen d) for CBT-I and SMW treatments were also calculated.

**RESULTS**

As shown in Table 2, CBT-I subjects reported significantly decreased SLAT and WASO and increased SE after treatment, compared with before treatment. They also reported significant changes in any measure of sleep quality or pain from before to after treatment. Average within-group pretreatment to post-treatment effect sizes of the 5 sleep measures and of the pain measures are presented as mean (SD), significance level (p), and effect sizes (ES) for pretreatment and post-treatment sleep measures. SMW refers to stress management and wellness; CBT-I, cognitive behavioral therapy for insomnia; TST, total sleep time; SE, sleep efficiency; SLAT, sleep latency; WASO, wake after sleep onset; GDS, Geriatric Depression Scale. Data extracted from Rybarczyk et al., 2005.

One subject had missing data for the McGill Pain Questionnaire (MPQ), and another had missing data for the Medical Outcomes Study Short Form-36 Pain (SF-PAIN).

Effect size (ES) refers to within-subjects Cohen d.

Higher MPQ scores indicate more pain. Higher SF-PAIN scores indicate less pain.

| Parameter | SMW<sup>a</sup> (n = 28) | | | | CBT-I<sup>b</sup> (n = 23) | | |
|-----------|------------------|-------------|-------------|------------------|-------------|-------------|
| | Before | After | p Value<sup>c</sup> | ES<sup>c</sup> | Before | After | p Value<sup>c</sup> | ES<sup>c</sup> |
| TST, min | 342 (84) | 370 (71) | 0.059 | 0.255 | 351 (60) | 372 (59) | 0.069 | 0.250 |
| SE, % | 70.2 (14.1) | 75.2 (14.0) | 0.069 | 0.252 | 71.0 (12.3) | 84.0 (8.1) | 0.000 | 0.883 |
| SLAT, min | 36.9 (27.1) | 33.4 (31.0) | 0.360 | 0.085 | 40.4 (21.4) | 23.5 (22.0) | 0.014 | 0.551 |
| WASO, min | 67 (45) | 55 (41) | 0.134 | 0.197 | 62 (47) | 25 (21) | 0.000 | 0.719 |
| Naps, min/wk | 15.5 (20.7) | 11.4 (15.7) | 0.084 | 0.158 | 9.9 (31.1) | 5.4 (6.8) | 0.067 | 0.141 |
| MPQ score<sup>e</sup> | 11.1 (9.6) | 11.6 (10.8) | 0.704 | 0.035 | 10.1 (9.6) | 8.0 (7.1) | 0.221 | 0.176 |
| SF-PAIN score<sup>e</sup> | 50.3 (21.4) | 53.1 (25.0) | 0.371 | 0.085 | 56.4 (19.7) | 66.1 (24.3) | 0.010 | 0.310 |
| GDS score | 5.3 (4.5) | 4.6 (4.5) | 0.327 | 0.110 | 5.6 (3.8) | 5.1 (4.7) | 0.608 | 0.083 |

Data are presented as mean (SD), significance level (p), and effect sizes (ES) for pre-treatment and post-treatment sleep measures. SMW refers to stress management and wellness; CBT-I, cognitive behavioral therapy for insomnia; TST, total sleep time; SE, sleep efficiency; SLAT, sleep latency; WASO, wake after sleep onset; GDS, Geriatric Depression Scale. Data extracted from Rybarczyk et al., 2005. *Significance levels for pre-treatment to post-treatment paired t-tests (2-tailed). Effect size (ES) refers to within-subjects Cohen d. One subject had missing data for the McGill Pain Questionnaire (MPQ), and another had missing data for the Medical Outcomes Study Short Form-36 Pain (SF-PAIN). One subject had missing data for both post-treatment pain measures. Higher MPQ scores indicate more pain. Higher SF-PAIN scores indicate less pain.

**Table 2** — Comparison of Sleep Parameters and Pain Scores in Older Patients with Osteoarthritis and Co-Morbid Insomnia Treated with Stress Management and Wellness or Cognitive Behavioral Therapy for Insomnia

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**Sleep Log**

Sleep logs were paper-and-pencil records that participants completed each morning for 2 weeks before treatment (during the month prior to treatment), after treatment (during the month after treatment ended), and at the 1-year follow-up. They were also completed on a weekly basis during the CBT-I class. The logs included sleep latency (SLAT), nighttime awakenings (quantity and duration), time in bed, naps, and any medication used for sleep. For the present study, average SLAT, total sleep time (TST), wake after sleep onset (WASO), and sleep efficiency (SE) were calculated for each of the 3 two-week assessment periods for each study subject.

**Short-Form MPQ**

The Short-Form MPQ was designed to be a brief version of the long-form MPQ, which has been widely used in the measurement and study of pain. The short-form MPQ has been shown to correlate highly with the standard MPQ and to be sensitive to pain-management interventions. For the present study, the total score was used.

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CBT-I improves both immediate and long-term reported pain in these patients—also in subjects who did not experience significant pain improvement at post-treatment assessment. Despite this, the CBT-I group demonstrated a significant reduction in pain at 1-year follow-up, which was not observed in the SMW group. This finding is consistent with previous studies demonstrating the efficacy of CBT-I for reducing pain in patients with osteoarthritis and co-morbid insomnia. The majority of CBT-I subjects (19 of 23) were further assessed at 1-year follow-up. As shown in the left panel of Table 3, at 1-year follow-up, they reported significantly decreased SLAT and WASO and increased SE and TST relative to pre-treatment. They also reported non-significant trends for reduced perceived pain on both the SF-PAIN and MPQ. Average within-group pre-treatment to 1-year follow-up effect sizes of the 5 sleep measures and of the 2 pain measures were 0.473 and 0.167 (Table 3). SMW subjects were not assessed at 1-year follow-up.

Data from the pretreatment versus 1-year follow-up analyses that included both the 19 original CBT-I subjects, as well as the 12 SMW subjects who chose to cross over to CBT-I and finished treatment and 1-year follow-up, are reported in the right panel of Table 3. This group of 29 CBT-I treated subjects reported significantly decreased SLAT and WASO and significantly increased SE and TST relative to pretreatment baselines. They also reported significantly less pain on the MPQ and a non-significant trend for reduced pain on the SF-PAIN. Average within-group pre-treatment to 1-year follow-up effect sizes of the 5 sleep measures and of the 2 pain measures were 0.410 and 0.184 (Table 3).

GDS scores for both CBT-I and CBT-I plus crossover subjects were low at pre-treatment and essentially unchanged at 1-year follow-up.

DISCUSSION

CBT-I improved both immediate and long-term self-reported sleep quality in this sample of older patients with osteoarthritis and co-morbid insomnia. These results are unique in demonstrating the long-term durability of CBT-I effects for co-morbid insomnia. The other major finding of this study—that CBT-I, without specifically addressing pain management, appeared to reduce both immediate and long-term reported pain in these patients—is also unique. This last finding is in contrast with the failure of the SMW treatment to significantly reduce reported pain at post-treatment assessment, despite the fact that the SMW protocol contained several treatment components that have been included in effective multi-component interventions for management of chronic pain. There were non-significant trends for improved sleep in the SMW group, as might be expected for an intervention targeting stress, but SMW effect sizes were substantially smaller than those of the CBT-I group.

These findings extend those of the parent study by suggesting the durability of CBT-I treatment effects on both sleep and pain across a 1-year follow-up. Several previous studies of CBT-I for insomnia co-morbid with chronic medical illness have demonstrated durability of treatment effects, but the longest follow-up to date has been 4 months in a sample of older adults with mixed chronic medical conditions. This is a potentially important finding given the common view that insomnia in medical populations is considered to be largely secondary to medical factors, such as pain or discomfort, and any worsening of those symptoms over time is believed by some to result in relapse of the insomnia.

The recent finding of Manber and colleagues, who reported that CBT-I enhanced depression outcome in patients with co-morbid insomnia and major depression, provides additional support that improving sleep can result in an improvement in a co-morbid disorder, be it medical (osteoarthritis) or psychiatric (major depression). Another recent study by Steptanski and colleagues found that increased difficulty sleeping predicted increased pain ratings in people with cancer. If pain management for people with cancer can be enhanced through successful treatment of sleep disturbance, this would have important clinical implications for improving quality of life in this patient population. These results support the need for large-scale randomized controlled trials in diverse patient populations in which chronic pain and insomnia commonly co-occur (e.g., osteoarthritis, chronic back pain, cancer).
How might improving sleep decrease perceived pain among older patients with osteoarthritis? Figure 1 schematically depicts possible mechanisms through which osteoarthritis pain and associated sleep disturbance are believed to be dysregulated daily activities and sleep schedule. As illustrated on the left side of Figure 1, when people with osteoarthritis must cope with arthritis pain and sleep disturbance, their daily activities and sleep schedules are altered. Sleep disturbance lowers the pain threshold and amplifies the transmission of pain signals, resulting in increased attention to pain, compromised function, and more negative pain-focused emotions and cognitions. Thus, via sleep disturbance-induced hyperalgesia, patients with osteoarthritis experience increased pain, reduced activity levels, and further disrupted sleep in a positive feedback-loop pattern.

Correspondingly, as shown on the right side of Figure 1, CBT-I enhances sleep, which raises the pain threshold, and amplification of pain-signal transmission is reduced. Thus, in turn, results in less perceived pain, less compromised function, and less pain-focused and more positive emotions and cognitions. This decreased pain and increased activity is then likely to further improve sleep, and the reciprocal interaction between sleep and pain helps to maintain both improved sleep quality and decreased perceived pain, again forming a positive feedback-loop.

Although the findings of this study support the need for further research, the current study has limitations. First, as noted previously, the parent study’s SMW control group, which contained several components typically employed in CBT intervention protocols for pain control, was not an optimal control condition. Because the SMW control group received interventions that may be beneficial for pain, it was employed as a comparison group with separate within-subject analyses rather than as a control group employing a more rigorous between-groups analysis to control for type I error. Also, the SMW comparison group was not followed for 1 year. Even though pain is thought to progressively worsen in osteoarthritis, this study design was unable to control for spontaneous improvements in pain levels unrelated to improved sleep that may have occurred during the follow-up period. Second, the criteria for osteoarthritis in the present study allowed participants with only moderate pain levels in a single joint to participate in the study. The primary purpose of the parent study was to address co-morbid insomnia, so emphasis was placed on verifying that subjects had the requisite level of insomnia and a verifiable diagnosis of osteoarthritis (rather than a significant level of osteoarthritis pain). Third, although insomnia is more common among older women, as compared with men, this study sample had a preponderance of women, which might diminish the generalizability of the study findings to men with osteoarthritis and co-morbid insomnia.

Finally, greater confidence in the reliability of the results would require a larger sample size, which would also provide an opportunity to test related hypotheses, such as what factors predict successful sleep and pain outcomes. Given these limitations, we regard the results of this reanalysis of Rybarczyk et al.’s data to yield only preliminary evidence in favor of the hypothesis that a successful sleep intervention has analgesic benefits among patients with osteoarthritis and co-morbid insomnia. Further randomized trials, with larger subject samples and intent-to-treat analyses, are needed to definitively evaluate this important clinical effect.

The evidence regarding the efficacy of cognitive-behavioral interventions specifically for arthritis pain and functioning is not strong. Astin et al. reported a meta-analysis of 25 trials of CBT-based pain interventions for rheumatoid arthritis and estimated an effect size of .22 for post-treatment benefits in pain, with somewhat larger effect sizes for improvements in functional disability, coping, and self-efficacy. The evidence regarding efficacy of cognitive behavioral interventions for osteoarthritis is also weak. Dixon and colleagues, in their meta-analysis of CBT-based pain interventions for osteoarthritis and rheumatoid arthritis, estimated a small pooled effect size of 0.18 for pain intensity and effect sizes of similar magnitude for functional outcomes. However, the effectiveness of a CBT-based sleep intervention for osteoarthritis pain has not been previously evaluated until the current study. Interestingly, the average effect size for the current study’s CBT-I–related post-treatment reduction of pain was 0.24.

Although some CBT-based pain interventions for osteoarthritis discuss sleep disturbances, sleep is typically not addressed.
in a systematic fashion, and interventions rarely go beyond basic sleep-hygiene recommendations that, by themselves, have little impact on sleep outcomes.\textsuperscript{37-38} Incorporating CBT-I, which specifically targets sleep, into behavioral interventions for osteoarthritis is a potential approach to increase the overall effectiveness for pain and functional outcomes that merits further research.

The ability of CBT-I to improve both short- and long-term sleep quality has been well demonstrated. The current study indicates that such long-term improvements may also be obtained in patients with insomnia and co-morbid osteoarthritis. The current study further suggests that, in addition to improving sleep, and even without directly addressing pain management, CBT-I appears to decrease pain in older patients with osteoarthritis and co-morbid insomnia both after treatment and at 1-year follow-up. These results, while preliminary, support the hypothesis that improving sleep, per se, in patients with osteoarthritis may be analgesic, such that perceived pain is reduced without being specifically targeted. These results further suggest that techniques to improve sleep, such as CBT-I, should be considered as additions to the various existing behavioral treatment programs for pain management in osteoarthritis, and possibly in other chronic pain conditions as well.

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\textbf{DISCLOSURE STATEMENT}

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\textbf{REFERENCES}