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Journal

Journal of Sleep Research, 33(5)

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Publication Date

2024-10-01

DOI

10.1111/jsr.14147

Peer reviewed

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HHS Public Access

Author manuscript

J Sleep Res. Author manuscript; available in PMC 2024 October 18.

Published in final edited form as:

J Sleep Res. 2024 October ; 33(5): e14147. doi:10.1111/jsr.14147.

Pain in your day? Get sleep treatment anyway! The role of pain in insomnia treatment efficacy in women veterans

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Summary

Insomnia and pain disorders are among the most common conditions affecting United States adults and veterans, and their comorbidity can cause detrimental effects to quality of life among other factors. Cognitive behavioural therapy for insomnia and related behavioural therapies are

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Alexander J. Erickson: Conceptualization; writing – original draft; writing – review and editing; formal analysis. **Scott G. Ravyts**: Conceptualization; writing – review and editing. **Joseph Dzierzewski**: Conceptualization; writing – review and editing. **Gwendolyn Carlson**: Writing – review and editing. **Monica Kelly**: Writing – review and editing. **Yeonsu Song**: Writing – review and editing. **Sarah Kate McGowan**: Writing – review and editing. **Michael Mitchell**: Writing – review and editing; formal analysis. **Donna L. Washington**: Writing – review and editing. **Elizabeth M. Yano**: Writing – review and editing. **Cathy A. Alessi**: Writing – review and editing. **Jennifer Martin**: Conceptualization; writing – review and editing; formal analysis; supervision.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

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recommended treatments for insomnia, but chronic pain may hinder treatment benefit. Prior research has not addressed how pain impacts the effects of behavioural insomnia treatment in United States women veterans. Using data from a comparative effectiveness clinical trial of two insomnia behavioural treatments (both including sleep restriction, stimulus control, and sleep hygiene education), we examined the impact of pain severity and pain interference on sleep improvements from baseline to post-treatment and 3-month follow-up. We found no significant moderation effects of pain severity or interference in the relationship between treatment phase and sleep outcomes. Findings highlight opportunities for using behavioural sleep interventions in patients, particularly women veterans, with comorbid pain and insomnia, and highlight areas for future research.

Keywords

insomnia; insomnia treatment; pain; sleep; United States veterans; women

1 | INTRODUCTION

Pain affects a substantial 50.2 million United States adults and plays a role in many areas of daily living, with its relationship to sleep being of particular importance (Yong et al., 2022). Problems with sleep are similarly pervasive, with an estimated 14.5%–17.8% of adults having difficulties falling asleep or staying asleep most nights and 19.2% of the United States population meeting criteria for insomnia disorder (Adjaye-Gbewonyo et al., 2022; Ford et al., 2015). Prior research has shown that pain and poor sleep frequently co-occur, with insomnia and chronic pain comorbidity rates being as high as 50% in some study samples (Taylor et al., 2007). Both of these conditions also disproportionally affect women who appear to have higher rates of insomnia (Kessler et al., 2011), several pain-related conditions, and pain-associated disability (Pavlović & Derby, 2022). These heightened rates are also reflected in the United States veteran population, with higher rates of pain-related conditions overall as well as higher rates of severe pain from these conditions compared to non-veterans (Nahin, 2017). Women veterans represent a group who are at heightened risk of both insomnia disorder and pain (Colvonen et al., 2020). Based on a 2017 report from the National Center for Veterans, migraines, back pain, and degenerative arthritis of the spine represented three of the 10 most common service-connected conditions in women veterans (National Center for Veterans Analysis and Statistics, 2017). Despite the high comorbidity of insomnia disorder and pain in this population, insomnia treatment-focused studies for women veterans remain generally sparse.

The cross-sectional association between pain, particularly chronic pain, and poor sleep has been a longstanding focus of scientific inquiry. Several studies have illustrated pervasive issues with sleep in individuals with chronic pain, including difficulties with sleep onset latency (SOL), sleep efficiency (percentage of time in bed spent asleep), and total time asleep (Mathias et al., 2018). In addition, difficulties with SOL, sleep efficiency, and insomnia symptoms have been associated with increased sensitivity to pain (Sivertsen et al., 2015). Although pain and poor sleep appear to share a bidirectional association, some research further contends that poor sleep has much more of an impact on the experience

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of pain than the reverse, which has motivated multiple studies evaluating how improving sleep serves to manage pain symptoms (Whibley et al., 2019). Multiple studies have also sought to examine potential mediating factors that underly the sleep-pain relationship, though current literature is limited by methodological constraints such as measurement and historical effects (Whibley et al., 2019).

The treatment of sleep-related difficulties could be a valuable approach to supporting the treatment of chronic pain. In a meta-analysis that illustrated the significant problem of disordered sleep in chronic pain, Mathias et al. (2018) highlighted the centrality of assessing and treating sleep disturbance in conjunction with chronic pain conditions, amplifying the call for evidence-based insomnia treatment in populations with pain. Cognitive behavioural therapy for insomnia (CBT-I) is the recommended first-line treatment for insomnia disorder, and has been shown to provide reductions in insomnia symptoms that are long-lasting compared to pharmacological interventions for sleep (Mitchell et al., 2012; Qaseem et al., 2016; Rios et al., 2019). CBT-I typically includes three treatment components: stimulus control, sleep restriction, and cognitive strategies. Stimulus control includes interventions that help foster associations between a person's sleep environment and sleep, such as having individuals leave their bed if they cannot sleep and return once they feel sleepy again. Sleep restriction involves temporarily reducing time in bed to increase sleep drive, with gradual increases for time in bed being added as sleep quality improves. Cognitive strategies in CBT-I are designed to address sleep-discouraging thought processes, including thoughts or beliefs that lead to anxiety or frustration when sleeping. CBT-I is recognised to not only improve sleep, but also improve other outcomes often comorbid with sleep difficulties including depression (Cunningham & Shapiro, 2018) and poor quality of life (Alimoradi et al., 2022).

Martin et al. (2023) conducted a comparative effectiveness trial comparing CBT-I against a novel acceptance and commitment therapy (ACT)-based psychotherapy for insomnia (e.g., acceptance and behavioural changes for insomnia [ABC-I]) to establish non-inferiority as well as examine treatment completion and adherence rates. Across sleep-related outcomes, CBT-I and ABC-I were found to be comparable, indicating statistical equivalence of effects (Martin et al., 2023). Structured pain assessment was included among measures used in that trial, but the study did not directly focus on pain. Despite this study not targeting women veterans with pain specifically, over half of the sample endorsed arthritis (51%) and chronic back pain (52%), warranting further study on how pain may have had a role in treatment outcomes. Pain has been shown in multiple contexts to increase treatment dropout and limit symptom improvement in pain as well as conditions like depression (Ogrodniczuk et al., 2008; Oosterhaven et al., 2019). Based on literature examining clinician-related barriers to CBT-I utilisation, many clinicians seek to treat pain instead of insomnia despite their comorbidity, due to common beliefs among clinicians that insomnia is more of a symptom of pain rather than an independent condition (Koffel et al., 2018). There are also contrasting view-points that individuals experiencing chronic pain may experience the most benefit from evidence-based behavioural insomnia treatment compared to those without chronic pain, and that individuals with chronic pain may not see their pain itself as a major barrier to CBT-I treatment (Koffel et al., 2020; McCurry et al., 2014). The underlying question regarding

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pain's relationship with behavioural insomnia treatment outcomes, particularly in women veterans, stands as the focus of this study.

The purpose of the present study was to examine the potential influences of pain, particularly patient self-rated pain severity and interference in day-to-day life, on the effectiveness of insomnia treatment for sleep outcomes (e.g., insomnia symptoms, perceived sleep quality, etc.). The data used for secondary data analysis comes from the randomised comparative effectiveness trial mentioned above examining CBT-I versus ABC-I in a sample of women veterans (Martin et al., 2023). Based on the original study, both interventions resulted in similar reductions in insomnia symptoms and other improvements to sleep outcomes, and we further hypothesised that both pain severity and interference would significantly moderate insomnia symptom improvement, with higher pain being associated with less improvement in insomnia after treatment and at 3-months follow-up.

2 | METHODS

2.1 | Participants

Data used in this study were from a sample of 149 women veterans who engaged in a double-blind randomised comparative effectiveness trial (NCT02076165) conducted by Martin et al. (2023). After removing data from participants who dropped out of the original study prior to completion, secondary data analysis was conducted on data from 135 women veterans who had access to and recently engaged with one large urban Veterans Affairs (VA) healthcare system. Relevant demographic characteristics are summarised in Table 1. Participants identified primarily as White (51 [34.23%]), African American (40 [26.85%]), or Hispanic/Latinx (28 [18.79%]). Average age of the sample was ~48 years and the average years of education for the sample was ~16 years. All participants completed baseline, post-treatment, and 3-month follow-up assessments in the original study conducted by Martin et al. (2023). For details regarding participant recruitment and attrition within the original study, please see Figure 1.

In the original study, inclusion criteria included having a diagnosis of insomnia disorder as determined through diagnostic criteria from the Diagnostic and Statistical Manual for Mental Disorders (American Psychiatric Association, 2013). Exclusion criteria for the original study included unmanaged sleep apnea, unmanaged serious mental illness (e.g., bipolar disorder), and factors that would impact ability to participate in a research trial (e.g., lack of transportation to medical centre, lack of stable housing, etc.). The present study did not impose any additional inclusion or exclusion criteria.

2.2 | Measures

2.2.1 | Brief Pain Inventory (BPI)—The short form of the BPI is a nine-item self-administered questionnaire that was used in this study to operationalise level of pain severity as well as pain-related interference in functioning. Response scales within the BPI vary by item, and include rating scales (e.g., 'which number best describes your pain on the average?') with a response scale from zero (no pain) to 10 (pain as bad as you can imagine). The BPI is also composed of two subscales: the Interference and Severity subscales. The

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Interference subscale score is formed from averaging responses across sub-items contained within item nine of the BPI (e.g., 'Mark the box beside the number that describes how, during the past 24 h, pain has interfered with your:… General Activity'). The Severity subscale score is formed from averaging responses across items three through six of the BPI. Global BPI severity and interference composite scores similarly range from zero to 10. High construct validity and convergent validity ($0.61 \leq r \leq 0.74$) with other pain measures has been established for the BPI in the context of non-cancer pain (Keller et al., 2004).

2.2.2 | Insomnia Severity Index (ISI)—The ISI is a seven-item instrument that was used to assess insomnia symptom severity. All items utilise a similar 4-point Likert-style scale, with initial items rating perceived symptoms (e.g., 'Please rate the CURRENT [i.e., LAST 2 WEEKS] SEVERITY of your insomnia problem(s): 1. Difficulty falling asleep'), and later items rating other areas of experience regarding insomnia (e.g., 'How SATISFIED/DISSATISFIED are you with your CURRENT sleep pattern'). Total scores within the ISI range from zero to 28. Literature on the ISI has illustrated evidence of internal consistency ($0.71 < \alpha < 0.91$) as well as convergent validity with measures of sleep quality ($r_{PSQI} = 0.8$), fatigue ($0.41 \leq r_{MFI} \leq 0.55$), sleep diaries ($=0.32 \leq r \leq 0.91$), and other similar instruments (Bastien et al., 2001; Morin et al., 2011).

2.2.3 | Pittsburgh Sleep Quality Index (PSQI)—Utilised as a measure of global sleep quality, the PSQI is an 18-item questionnaire that includes items with multiple response scales, including open-field response entries (e.g., 'During the past month, what time have you usually gone to bed at night?') and fixed choice frequency items (e.g., 'During the past month, how often have you had trouble sleeping because you wake up in the middle of the night or early morning,' with response choices ranging from 'not during the past month' to 'three or more times a week'). The total score of the PSQI ranges from zero to 21. Evidence of test–retest reliability ($\alpha = 0.87$) and correlation with commonly used sleep diaries has been established for patients with primary diagnosis of insomnia (Backhaus et al., 2002; Buysse et al., 1989).

2.2.4 | Glasgow Sleep Effort Scale (GSES)—The GSES is a seven-item measure of perceived effort to fall asleep (Broomfield & Espie, 2005). Individuals are asked to rate their agreement with statements related to sleep effort, including 'I put too much effort into sleeping when it should come naturally' and 'I worry about not sleeping if I cannot sleep.' Responses to these statements range from zero (not at all) to two (very much), with total scores ranging from zero to 14. Initial psychometric development and evaluation illustrated generally adequate internal consistency ($\alpha = 0.77$), as well as evidence of concurrent and discriminant validity (Broomfield & Espie, 2005).

2.2.5 | Epworth Sleepiness Scale (ESS)—The ESS was employed to measure perceived daytime sleepiness. The ESS is an eight-item measure that has participants rate their likelihood of nodding off or falling asleep unintentionally in provided situations (e.g., 'Sitting and reading') from zero ('would never nod off') to 3 ('high chance of nodding off'). An ESS total summed score can range from zero to 24. Studies have illustrated evidence of test–retest reliability within sleep-focused clinical trials (Rosenberg et al., 2022), as well as

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evidence of validity in multiple contexts and populations though the ESS warrants further investigation into its validity (Schokman et al., 2022).

2.3 | Procedure

All participants within the original study (for details, see Martin et al., 2023) gave written informed consent to study participation, and study procedures were approved by the Institutional Review Board of the VA Greater Los Angeles Healthcare System. The original study is registered as a Clinical Trial (NCT02076165). Study activities started in October 2014 (recruitment) and concluded in February 2018. Both participants and research staff conducting assessments were blinded to participant randomisation and group assignment. Participants were initially recruited through basic postal surveys containing items related to insomnia disorder diagnostic criteria and related measures. For participants who returned these postal surveys and endorsed insomnia symptoms, telephone screeners were completed.

Following telephone screening and enrolment in the study, a pre-treatment face-to-face baseline assessment, including demographic, health status (e.g., self-reported medical history), mental health symptoms, sleep measures, and other measures was completed in interview format with all participants. Following completion of baseline assessment, participants were randomised 1:1 (using random allocation concealment) to a behavioural insomnia treatment condition (CBT-I or ABC-I) via a randomisation sequence developed by the study statistician using Stata (version 13.1). To be randomised in the study, women veterans had to have a diagnosis of insomnia disorder, which was confirmed through baseline assessments as well as final determination by a study clinical psychologist and board-certified sleep medicine physician based on Diagnostic and Statistical Manual of Mental Disorders fifth edition criteria (American Psychiatric Association, 2013). After randomisation, participants completed five sessions of behavioural insomnia treatment. Additional assessments, containing the same measures used in the baseline assessment, were conducted directly post-treatment and in a 3-month follow-up. Martin et al. (2023) can be referenced for additional details regarding original study procedures.

2.4 | Treatment conditions

In the original study conducted by Martin et al. (2023), the main research goal was to establish the efficacy of an ACT-based insomnia treatment, ABC-I, in comparison to CBT-I. Both treatment conditions included five weekly individual sessions. Treatments were provided by clinical psychologists with expertise in CBT-I delivery as well as knowledge and training in ACT and the ABC-I protocol. Full information regarding these treatments can be found in the original article by Martin et al. (2023), with brief summaries of the treatment conditions provided in Table 2.

2.5 | Data analysis

Analyses were conducted using Stata (version 17.1). Mixed-effects models were conducted utilising Stata's *mixed* command, with further exploration of effects being evaluated through the *contrast* and *margins* commands. Observations with missing values were omitted from analyses, which is the default missing data method within the Stata *mixed* command. Initial scatterplots were created to evaluate data spread, and, based on examining these

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plots, investigators found it valuable to evaluate pain factors both as linear predictors of sleep outcomes (termed 'linear models' in the remaining content of the manuscript) as well as quadratic predictors of sleep outcomes (termed 'quadratic models' in the remaining content of the manuscript). Linear mixed-effects analyses modelled outcomes of interest as a function of treatment phase as a factor variable with three levels (baseline, post-treatment, 3-month follow-up), pain as a continuous variable (baseline pain severity, baseline pain interference), and the interaction between treatment phase and pain factors. A significant regression coefficient regarding treatment phase represents a post-treatment/3-month follow-up versus baseline difference in an outcome of interest when a pain value is at its zero point. A significant regression coefficient regarding pain represents baseline differences in an outcome of interest at different degrees of baseline pain severity or interference. Quadratic mixed-effects analyses modelled outcomes of interest as a function of the aforementioned predictors, with the addition of a quadratic term for pain factors (e.g., pain severity squared, pain interference squared) as well as a term for the interaction between treatment phase and the quadratic pain term. A significant quadratic regression coefficient regarding pain represents baseline differences in an outcome of interest at different degrees of baseline pain severity or interference, with the effect of pain severity or interference compounding as the pain score increases. Both pain interference and pain severity were treated as time invariant predictors across analyses, with these variables representing pain inference and severity scores at baseline. Linear and quadratic models were compared using a likelihood ratio (LR) test to determine which model was most appropriate to interpret. Additional sensitivity analyses were conducted using linear mixed-effects models, testing if presence of self-reported chronic pain conditions (arthritis, chronic back pain) at baseline moderated treatment effects on sleep outcomes.

All models also contained random intercepts at the subject level. In testing additional random effects at the subject level within models, all other random intercepts and slopes led to a lack of convergence of calculated models, resulting in a lack of inclusion of those random effects to support model convergence and subsequent interpretation. In the original Martin et al. (2023) article, analyses illustrated non-inferiority between CBT-I and ABC-I outcomes, establishing equivalence between conditions. Given the findings of Martin et al. (2023), treatment type was not included as a predictor of interest.

Post hoc power analyses were conducted using the Stata *power* command to determine the detectable effect given the study sample size. Detectable effect size was interpreted based on f^2 thresholds previously established (Cohen, 1992). Based on these analyses, with $\alpha = 0.05$ and a sample size of 135, analyses had the potential to detect a small-to-medium effect through either the linear models ($f^2 = 0.059$) or the quadratic model ($f^2 = 0.073$). Of the outcomes, insomnia symptom (ISI scores) were the primary outcome of interest given that insomnia is the primary target of both treatment conditions. Supplementary graphs within this study were produced with Stata's *marginplot* command and include 95% confidence intervals (CIs).

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3 | RESULTS

Univariate statistics of variables included within the models below can be found in Table 1. Initial linear and quadratic models were compared utilising a LR test, of which findings and model selection details can be found in Table 3. Findings interpreted below are from statistically significant models, which can be reviewed in Table 4. The LR results regarding model comparison between models without or with random intercept can be viewed in the Supplementary Materials (Table S1). Sensitivity analysis results are summarised below, with an associated table (Table S2) and figure (Figure S1) available in the Supplementary Materials. Marginal means, with shading for 95% CIs, of pain factors (*x*-axis), sleep outcomes (*y*-axis), and treatment phase comparison (plotted lines) are plotted in Figure 2.

3.1 | Insomnia

In the pain interference model, treatment phase was a significant predictor of insomnia symptoms, with significant reductions in insomnia symptoms noted between baseline and post-treatment ($b = -8.31$, standard error [SE] = 0.652, $p < 0.001$) and between baseline and the 3-month follow-up ($b = -8.17$, SE = 0.761, $p < 0.001$). While baseline pain interference as a linear predictor was not significant, baseline pain interference as a quadratic predictor was significant ($b = 0.136$, SE = 0.048, $p = 0.005$). No significant interactions were found between treatment phase and pain interference regarding their relationship with insomnia symptoms.

In the pain severity model, treatment phase was a significant predictor of insomnia symptoms, with significant reductions in insomnia symptoms noted between baseline and post-treatment ($b = -8.05$, SE = 0.69, $p < 0.001$) and between baseline and the 3-month follow-up ($b = -8.05$, SE = 0.85, $p < 0.001$). While baseline pain severity as a linear predictor was not significant, baseline pain severity as a quadratic predictor was significant in both models ($b = 0.154$, SE = 0.049, $p = 0.002$). No significant interactions were found between treatment phase and pain severity regarding their relationship with insomnia symptoms.

3.2 | Sleep quality

Treatment phase was a significant predictor of perceived sleep quality within the pain interference model, with significant improvements in perceived sleep quality noted between baseline and post-treatment ($b = -5.321$, SE = 0.484, $p < 0.001$) and between baseline and the 3-month follow-up ($b = -4.849$, SE = 0.546, $p < 0.001$). Baseline pain interference significantly predicted sleep quality scores as a quadratic predictor ($b = 0.09$, SE = 0.037, $p = 0.015$) but not as a linear predictor. No significant interactions were found between treatment phase and pain interference in regard to their relationship with sleep quality.

Treatment phase was a significant predictor of perceived sleep quality within both pain interference and pain severity models, with significant improvements in perceived sleep quality noted between baseline and post-treatment ($b = -4.965$, SE = 0.479, $p < 0.001$) and between baseline and the 3-month follow-up ($b \leq -4.403$, SE = 0.566, $p < 0.001$). Baseline

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pain severity significantly predicted sleep quality scores as a linear predictor ($b = 0.334$, SE = 0.099, $p = 0.001$). No significant interactions were found between treatment phase and pain severity in regard to their relationship with sleep quality.

3.3 | Sleep effort

Treatment phase was a significant predictor of sleep effort within the pain interference model, with significant reductions in sleep effort noted between baseline and post-treatment ($b = -2.37$, SE = 0.46, $p < 0.001$) and between baseline and the 3-month follow-up ($b = -2.78$, SE = 0.518, $p < 0.001$). Baseline pain interference was not a significant linear or quadratic predictor of baseline sleep effort. No significant interactions were found between treatment phase and pain interference regarding their relationship with sleep effort.

Treatment phase was a significant predictor of sleep effort within the pain severity model, with significant reductions in sleep effort noted between baseline and post-treatment ($b = -2.222$, SE = 0.456, $p < 0.001$) and between baseline and the 3-month follow-up ($b = -2.671$, SE = 0.521, $p < 0.001$). Baseline pain severity significantly predicted baseline sleep effort scores as a linear predictor ($b = 0.308$, SE = 0.1, $p = 0.002$). No significant interaction were found between treatment phase and pain severity regarding their relationship with sleep effort.

3.4 | Daytime sleepiness

Treatment phase was a significant predictor of daytime sleepiness within the pain interference model, with significant reductions in daytime sleepiness noted between baseline and post-treatment ($b = -3.089$, SE = 0.55, $p < 0.001$) and between baseline and the 3-month follow-up ($b = -3.02$, SE = 0.593, $p < 0.001$). Baseline pain interference ($b = 0.377$, SE = 0.11, $p = 0.001$) significantly predicted baseline daytime sleepiness scores, with higher baseline levels of pain interference associated with higher baseline levels of daytime sleepiness. No significant interactions were found between treatment phase and pain interference in regard to their relationship with daytime sleepiness.

Treatment phase was a significant predictor of daytime sleepiness within the pain severity model, with significant reductions in daytime sleepiness noted between baseline and post-treatment ($b = -3.231$, SE = 0.592, $p < 0.001$) and between baseline and the 3-month follow-up ($b \leq -3.058$, SE = 0.647, $p < 0.001$). Baseline pain severity ($b = 0.307$, SE = 0.13, $p = 0.018$) significantly predicted daytime sleepiness scores, with higher baseline levels of pain severity associated with higher levels of baseline daytime sleepiness. No significant interactions were found between treatment phase and pain severity in regard to their relationship with daytime sleepiness.

3.5 | Sensitivity analysis

The results of sensitivity analysis were generally consistent with primary analysis outcomes. Treatment phase was a significant predictor across all models, with sleep outcomes improving at post-treatment and the 3-month follow-up following behavioural insomnia treatment. In regard to chronic pain conditions, chronic low back pain was a significant predictor of insomnia symptoms and sleep effort, while arthritis was not a significant

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predictor of any sleep outcomes. A significant interaction was found between treatment phase and chronic back pain in regard to sleep effort, with individuals who reported having chronic low back pain at baseline exhibiting slightly greater improvement in sleep effort at post-treatment ($b = -1.43$, SE = 0.606, $p = 0.019$) and the 3-month follow-up ($b = -1.38$, SE = 0.692, $p = 0.046$) compared to individuals who denied having chronic back pain. No other significant interactions were present, consistent with primary statistical analyses. For model-specific statistics and figures, see the Supplementary Materials.

4 | DISCUSSION

Sleep-related disturbances and disorders like insomnia are often comorbid with pain, and improvement in sleep has been shown to influence pain-related outcomes. Given evidence from previous studies of pain's potential negative influence on engagement and outcomes of some treatments and psychotherapies (e.g., Ogrodniczuk et al., 2008, etc.), this study sought to examine if pain severity and pain-related interference impacted insomnia treatment outcomes in women veterans. We found that higher pain severity and interference at baseline were associated with heightened insomnia symptoms and daytime sleepiness, as well as poorer sleep quality, at baseline. In relation to insomnia symptoms and sleep quality, the effect of baseline pain interference appeared to be more pronounced at higher levels of baseline pain interference. Baseline pain severity also demonstrated a similar quadratic trend, with its influence on insomnia symptoms specifically getting stronger with greater levels of baseline pain severity. Baseline pain severity also shared a generally minor linear relationship with baseline sleep effort, with more intense baseline pain severity being associated with higher baseline sleep effort. These results are consistent with previous studies demonstrating the cross-sectional bidirectional relationship between pain and sleep (Finan et al., 2013; Whibley et al., 2019).

The central question in the present analysis was: does pain influence the effectiveness of treatment for insomnia? Across primary models, no significant interactions were found between pain factors and treatment phase (before treatment, after treatment, and the 3-month follow-up), which suggests that pain does not influence the degree of improvement in sleep-related outcomes following behavioural insomnia treatments. These results were generally replicated in sensitivity results that used self-reported pain conditions, with some evidence of slightly greater improvements in sleep effort for individuals with self-reported chronic back pain. Given the results of post hoc power analyses, with each model representing an opportunity to detect a small-to-medium sized effect if one was present, it appears that this lack of association is unlikely to be the result of a type II error. It is also important to highlight that despite the appearance of some visual trends from primary analyses (particularly differences of trajectories in insomnia symptoms and sleep quality based on pain interference in Figure 2), these trends are not statistically different and treatment improvements were statistically meaningful for all outcomes of interest, with only one exception regarding sleep effort where treatment effects may be minimal with high levels of pain interference (e.g., CI contains zero).

These findings can yield multiple possible implications to both the research and clinical investigation of comorbid pain and insomnia. Based on this study, mild-to-moderate pain

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does not appear to meaningfully limit benefits to sleep from behavioural insomnia treatment in women veterans; a population who are at heightened risk of comorbid insomnia disorder and pain. This is in line with other quantitative studies that have demonstrated meaningful improvement in sleep outcomes following CBT-I for individuals with pain conditions (Selvanathan et al., 2021; Vitiello et al., 2009) as well as qualitative evidence of pain not being a central barrier to CBT-I treatment for patients with chronic pain (Koffel et al., 2020).

There could be many potential explanations for why these insomnia treatment approaches are not impacted by pain-related symptoms. First, some behavioural interventions contained within both the CBT-I and ABC-I treatments can be useful for addressing pain symptoms as well. For example, increasing moderate daytime activity can be an intervention that reduces pain impacts on daily living. If someone lays in bed for extended periods during the day because of pain, stimulus control activities such as identifying other comfortable areas to rest during the day could benefit both sleep and pain-related symptoms. These skills also could address factors comorbid with or associated with pain, such as depression and anxiety, which also may serve to mitigate the impact of pain on treatment efficacy. CBT-I and ABC-I also instil skills (e.g., cognitive/acceptance techniques, behavioural stimulus control practises) that patients can use to help themselves through daily challenges independently (Beck, 2020; Hayes et al., 2012), even if pain is present.

Although pain does not appear to significantly interfere with insomnia treatment benefits, pain may contribute to insomnia relapse over the long-term. Sleep improvements may dwindle (e.g., beyond a 3-month follow-up) if pain is not adequately addressed. Individuals with mild-to-moderate pain may more easily maintain stimulus control, sleep hygiene, and other practises given that their pain levels may be easier to tolerate; however, a patient with high levels of pain severity or interference may experience challenges in sustaining more difficult practises and may be prone to insomnia relapse. Our study could not directly address this question. Additionally, most sleep and pain focused literature has conveyed that although sleep and pain are associated, improving sleep alone may not sufficiently improve pain and additional intervention may be required (Tang et al., 2015). Insomnia treatment may be important to optimise improvements in pain but is unlikely to be sufficient without corresponding pain-focused interventions.

Insomnia treatment could be a complementary approach to incorporate with pain-focused care and models of pain management or related care, such as the stepped care model of pain management (Von Korff, 1999) and whole health model (Gaudet & Kligler, 2019) that has been adopted by the US Veteran's Health Administration (VHA). These models infer baseline health and mental health factors, that could include sufficient sleep, as foundational to pain-focused care, although the discussion of sleep's role in pain or methods of improving sleep in populations with pain is generally lacking. Importantly, ensuring sufficient, high quality sleep may enhance engagement with pain-focused care. Some research has also the explored a combined cognitive behavioural approach for comorbid pain and insomnia, such as CBT for Pain and Insomnia (CBT-PI; Vitiello et al., 2013). A recent systematic review highlighted CBT-I or CBT-PI as potentially being the most effective behavioural treatment for comorbid insomnia and pain compared to CBT for chronic pain alone (Enomoto et

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al., 2022). Further research in this area is warranted, including system-focused studies examining implementation of CBT-I and CBT-PI within interdisciplinary pain settings.

4.1 | Limitations/future directions

Given that many individuals with chronic pain have comorbid insomnia, the incorporation of CBT-I and ACT-informed approaches could be a valuable addition to best practice treatment for comorbid pain and insomnia. Both CBT and ACT are also evidenced-based approaches for treating chronic pain and have been highlighted as best-practice approaches in pain management within the United States (U.S. Department of Health and Human Services, 2019). Given that this study design is focused on sleep outcomes following insomnia treatment, findings do not directly illustrate how these treatments could be incorporated in pain treatment. Future studies, as well as programme development and evaluation at the clinical system level, should explore the implementation and effectiveness of incorporating insomnia-focused behavioural treatments into pain-focused care settings.

Although there is limited literature assessing how long it takes for sleep-interfering behaviours or cognitions to develop, it is possible that pain-informed behaviour and cognitions play a role in the development of insomnia symptoms; particularly if pain remains unresolved. In this, pain also could be associated with the development of other symptoms or factors, such as depression or anxiety, which serve to worsen insomnia symptoms. As mentioned previously, although this study was able to evaluate sleep outcomes up to a 3-month follow-up, the potential influence of pain or associated factors on long-term sleep disturbance and insomnia relapse requires exploration. This not only presents an opportunity for longitudinal research, but also for clinical research to use designs examining how pain can give rise to sleep-interfering behaviours and cognitions over time. With larger samples, studies could account for potential indirect mediating factors underlying the association between pain, sleep, and sleep treatment efficacy.

The influence of sleep-focused interventions on pain outcomes more generally is a compelling area of research and warrants further study. Opportunities also exist for this study to be replicated within the context of a pain-focused clinical settings. Pain also can present with more facets than severity and interference with day-to-day life, and future studies could examine other facets such as biomarkers, pain duration, or chronic pain diagnosis to provide additional depth of understanding regarding pain's impact on behavioural insomnia treatment efficacy. In regard to subjective measurement of pain, future studies may also seek to establish empirically supported severity thresholds for BPI scores, strengthening practical interpretability of findings beyond what this study provides. Additionally, given that the recruitment focus of this study's data source was not specifically for individuals with pain or pain conditions, findings of this study are generally limited to individuals with mild-to-moderate levels of pain severity and pain-related activity interference. This study also did not utilise data from treatment non-completers given the analytical approach, which may impact findings. This study also does not account for the impact of pain or sleep-focused medication, which could have an impact on study outcomes and pain ratings. Studies should also seek to include more individuals with high levels of pain severity and interference and account for the role of medication use, which would serve

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to strengthen findings and their generalisability. Lastly, to further support generalisability, future studies should include other genders and non-veterans.

5 | CONCLUSION

Comorbid insomnia and pain affects many people in the United States and worldwide, with substantial literature illustrating how these comorbid conditions impact day-to-day living. Although some studies have highlighted evidence of pain interfering with psychotherapeutic treatments and their associated outcomes, this study illustrated that the benefits of insomnia treatment on sleep outcomes appear robust to mild-to-moderate pain or pain-related activity interference. These findings support the value of insomnia treatment in the context of comorbid sleep problems and pain, and further open paths to future research into the use of insomnia treatment in pain management settings, the role of pain in sleep-interfering behaviour/cognitions, and other areas. Ultimately, our study shows that mild-to-moderate pain, or associated interference to daily activity from that pain, does not appear to be a barrier to behavioural insomnia treatment efficacy, particularly in women veterans.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGEMENTS

Data used were from an original project funded by VA/Health Services Research and Development Service (HSR&D) IIR-HX002300 (Principal Investigator: Jennifer L. Martin; NCT02076165). Dr Jennifer L. Martin is also supported by National Institutes of Health (NIH)/National Heart, Lung, and Blood Institute (NHLBI) K24HL143055 and VA HSR&D Research Career Scientist Award RCS 20-191. Dr Alexander J. Erickson was supported by the VA Office of Academic Affiliations through the Advanced Fellowship Program in Geriatrics. Dr Gwendolyn C. Carlson is also supported by VA HSR&D CDA 20-227. Dr Monica R. Kelly is also supported by NIH/NHLBI K23 HL157754. Dr Elizabeth M. Yano is funded by a VA HSR&D Senior Research Career Scientist Award RCS 05-195. Support was also provided by the VA Greater Los Angeles Geriatric Research, Education and Clinical Center.

Funding information

National Institutes of Health/National Heart, Lung, & Blood Institute, Grant/Award Numbers: K23 HL157754, K24HL143055; VA Health Services Research & Development, Grant/Award Number: RCS 20-191; VA Office of Academic Affiliations; VA Greater Los Angeles Geriatric Research, Education and Clinical Center; VA Human Services Research & Development, Grant/Award Numbers: CDA 20-227, IIR-HX002300, RCS 05-195, NCT02076165

DATA AVAILABILITY STATEMENT

Data may be available through execution of a data use agreement between the requestor's institution and the VA Greater Los Angeles Healthcare System. Study materials can be requested from the corresponding author.

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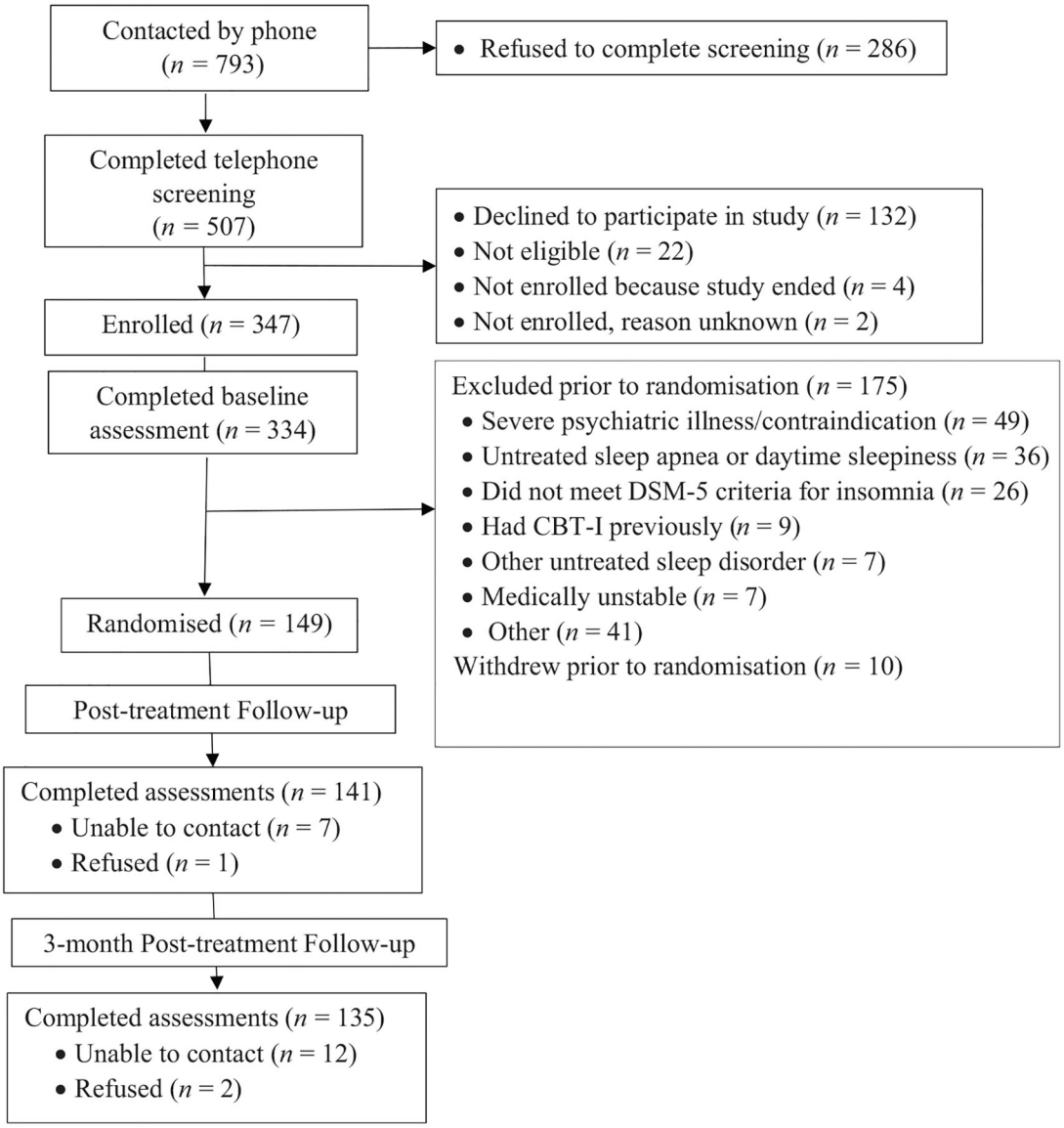

FIGURE 1.

Summarised recruitment and sample from original Martin et al. (2023) study. CBT-I, Cognitive Behavioural Therapy for Insomnia; DSM-5, *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition.

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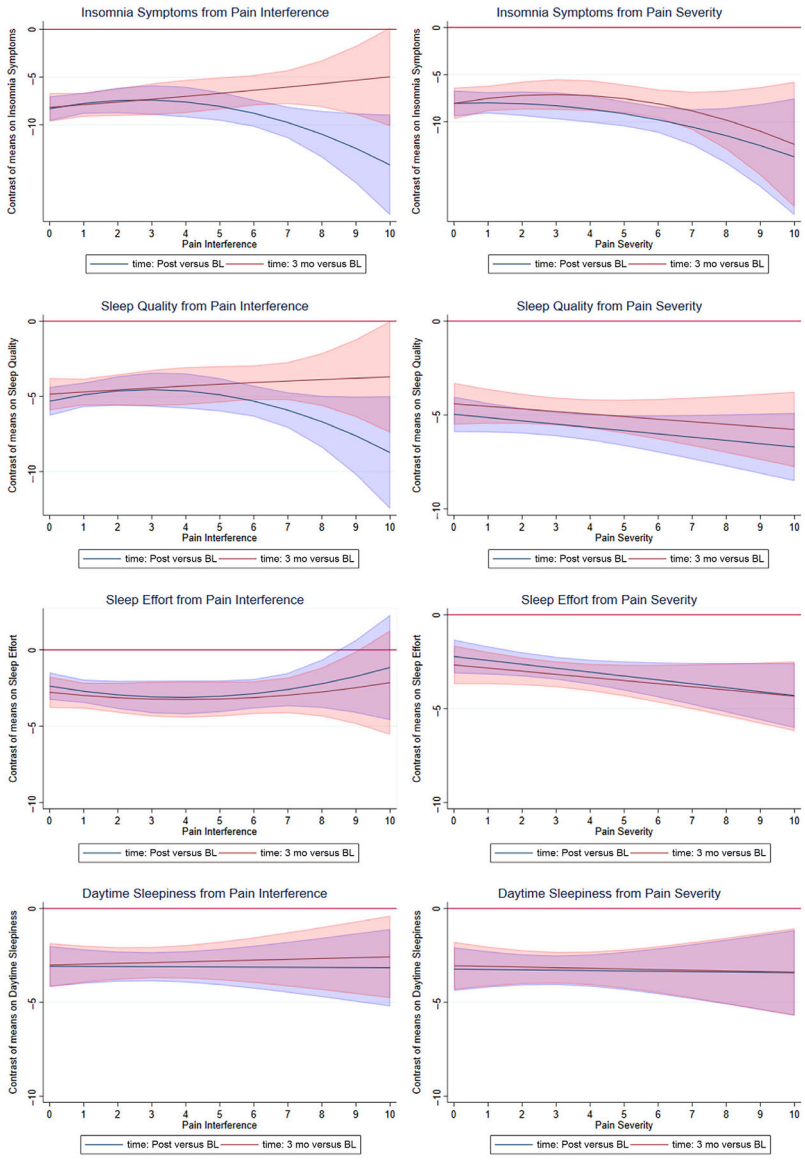

FIGURE 2.

Change in sleep outcomes across treatment phase by pain factor levels. BL, baseline; 3 mo, 3-month follow-up; Post, post-treatment.

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TABLE 1

Sample demographics and descriptive statistics (n = 149).

	Baseline (n = 149)		Post-treatment (n = 141)	3-month follow-up (n = 135)
Variable	***N* (%)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**
Race				
African American	40 (26.9)	-	-	-
Hispanic/Latinx	28 (18.8)	-	-	-
Asian American	5 (3.4)	-	-	-
White	51 (34.2)	-	-	-
Multi-racial	19 (12.8)	-	-	-
Other	6 (4.0)	-	-	-
Treatment condition				
CBT-I	75 (50.3)	-	-	-
ABC-I	74 (49.7)	-	-	-
Pain conditions				
Arthritis/rheumatism	73 (49.0)	-	-	-
Chronic back pain	72 (48.3)	-	-	-
Chronic headaches	84 (56.3)	-	-	-
Age	-	47.8 (13.1)	-	-
Education	-	16.3 (2.68)	-	-
BPI – Severity	-	3.1 (2.8)	2.6 (2.7)	3.1 (2.8)
BPI – Interference	-	3.2 (3.2)	2.4 (2.8)	2.6 (2.8)
ISI	-	14.4 (5.0)	5.4 (4.9)	6.5 (5.7)
PSQI	-	10.7 (3.7)	5.1 (3.4)	5.9 (4.2)
GSES	-	6.6 (3.7)	3.7 (2.9)	3.4 (3.2)
ESS	-	8.3 (4.9)	4.9 (4.2)	5.2 (4.67)

Note: ABC-I, acceptance and behavioural changes for insomnia; BPI, Brief Pain Inventory; CBT-I, cognitive behavioural therapy for insomnia; ESS, Epworth Sleepiness Scale; GSES, Glasgow Sleep Effort Scale; ISI, Insomnia Severity Index; PSQI, Pittsburgh Sleep Quality Index; SD, standard deviation.

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TABLE 2

Cognitive behavioural therapy for insomnia/acceptance and behavioural changes for insomnia treatment session descriptions.

Session	CBT-I	ABC-I
Session 1	Psychoeducation (3P model, stimulus control); sleep hygiene	Psychoeducation (3P model, stimulus control); sleep hygiene
Session 2	Sleep diary review; psychoeducation (sleep restriction) and development of sleep restriction schedule	Sleep diary review; psychoeducation (Sleep restriction) and development of sleep restriction schedule; ACT metaphors around long-term benefit versus short-term discomfort
Session 3	Sleep diary review; adjustment of sleep schedule; psychoeducation (cognitive skills); coping cards	Sleep diary review; adjustment of sleep schedule; psychoeducation (ACT skills); cognitive defusion
Session 4	Sleep diary review; adjustment of sleep schedule; cognitive skills; addressing barriers and issues to treatment adherence	Sleep diary review; adjustment of sleep schedule; ACT-based discussion of overcoming barriers to treatment adherence
Session 5	Review of improvements; relapse prevention strategies	Review of improvements; relapse prevention strategies

Abbreviations: ABC-I, acceptance and behavioural changes for insomnia; ACT, acceptance and commitment therapy; CBT-I, cognitive behavioural therapy for insomnia.

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TABLE 3

Likelihood ratio tests comparing linear and quadratic mixed-effects models.

Models	LR χ^2	*p*	Model interpreted
ISI and pain interference	13.96	0.003	Quadratic
PSQI and pain interference	9.75	0.021	Quadratic
GSES and pain interference	11.19	0.011	Quadratic
ESS and pain interference	7.61	0.055	Linear
ISI and pain severity	10.28	0.02	Quadratic
PSQI and pain severity	4.94	0.18	Linear
GSES and pain severity	1.45	0.6948	Linear
ESS and pain severity	3.78	0.2858	Linear

Abbreviations: ESS, Epworth Sleepiness Scale; GSES, Glasgow Sleep Effort Scale; ISI, Insomnia Severity Index; LR, likelihood ratio; PI, pain interference; PSQI, Pittsburgh Sleep Quality Index.

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TABLE 4

Pain factors and sleep outcomes models.

Models	Pain interference *b* (SE)	Pain severity *b* (SE)
ISI	Wald $\chi^2 = 519.55^{***}$	Wald $\chi^2 = 471.84^{***}$
Time (baseline to post-treatment)	-8.31^{***} (0.652)	-8.05^{***} (0.69)
Time (baseline to 3-month follow-up)	-8.17^{***} (0.761)	-8.05^{***} (0.846)
Pain	−0.47 (0.379)	−0.431 (0.359)
Pain*Time (baseline to post-treatment)	0.683 (0.53)	0.127 (0.522)
Pain*Time (baseline to 3-month follow-up)	0.265 (0.561)	0.635 (0.599)
Pain^2	0.136^{**} (0.048)	0.154^{**} (0.049)
Pain^2*Time (baseline to post-treatment)	−0.127 (0.072)	−0.069 (0.076)
Pain^2*Time (baseline to 3-month follow-up)	0.005 (0.073)	−1.07 (0.084)
PSQI	Wald $\chi^2 = 355.4^{***}$	Wald $\chi^2 = 319.71^{***}$
Time (baseline to post-treatment)	-5.321^{***} (0.484)	-4.965^{***} (0.479)
Time (baseline to 3-month follow-up)	-4.849^{***} (0.546)	-4.403^{***} (0.566)
Pain	−0.357 (0.292)	0.334^{**} (0.099)
Pain*Time (baseline to post-treatment)	0.514 (0.387)	−0.174 (0.122)
Pain*Time (baseline to 3-month follow-up)	0.149 (0.408)	−0.137 (0.137)
Pain^2	0.09^{**} (0.037)	-
Pain^2*Time (baseline to post-treatment)	−0.086 (0.052)	-
Pain^2*Time (baseline to 3-month follow-up)	−0.003 (0.053)	-
GSES	Wald $\chi^2 = 145.86^{***}$	Wald $\chi^2 = 114.22^{***}$
Time (baseline to post-treatment)	-2.37^{***} (0.46)	-2.222^{***} (0.456)
Time (baseline to 3-month follow-up)	-2.78^{***} (0.518)	-2.671^{***} (0.521)
Pain	−0.01 (0.296)	0.308^{**} (0.1)
Pain*Time (baseline to post-treatment)	−0.391 (0.364)	−0.208 (0.116)
Pain*Time (baseline to 3-month follow-up)	−0.241 (0.383)	−0.167 (0.127)
Pain^2	0.043 (0.038)	-
Pain^2*Time (baseline to post-treatment)	0.051 (0.049)	-

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Models	Pain interference *b* (SE)	Pain severity *b* (SE)
Pain^2*Time (baseline to 3-month follow-up)	0.031 (0.05)	-
ESS	Wald $\chi^2 = 110.03^{***}$	Wald $\chi^2 = 92.13^{***}$
Time (baseline to post-treatment)	-3.089^{***} (0.55)	-3.231^{***} (0.592)
Time (baseline to 3-month follow-up)	-3.02^{***} (0.593)	-3.058^{***} (0.647)
Pain	0.377^{**} (0.11)	0.307^{***} (0.13)
Pain*Time (baseline to post-treatment)	−0.007 (0.136)	−0.02 (0.153)
Pain*Time (baseline to 3-month follow-up)	0.043 (0.145)	−0.034 (0.16)
Pain^2	-	-
Pain^2*Time (baseline to post-treatment)	-	-
Pain^2*Time (baseline to 3-month follow-up)	-	-

Note:

** $p < 0.01$,

*** $p < 0.001$; Single * between variable names represent interaction terms.

Abbreviations: ISI, Insomnia Severity Index; PSQI, Pittsburgh Sleep Quality Index; GSES, Glasgow Sleep Effort Scale; ESS, Epworth Sleepiness Scale; SE, standard error.