



Sleep Disruption and Pain Sensitivity: A Bidirectional Relationship the Pain Clinic Rarely Addresses

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Abstract

Sleep disturbance and pain are linked by a well-documented bidirectional relationship: experimental sleep deprivation and selective slow-wave sleep disruption lower pain threshold and impair endogenous pain-inhibitory function, while pain itself fragments sleep continuity and reduces restorative slow-wave sleep, creating a self-sustaining cycle. Despite a substantial and consistent evidence base, sleep is rarely assessed systematically in routine pain-clinic practice, and insomnia treatment is rarely offered as a component of pain management. This review synthesises the experimental, epidemiological, and clinical-trial literature establishing the bidirectional sleep-pain relationship and the evidence supporting cognitive-behavioural therapy for insomnia as an effective, currently under-used, adjunct to chronic pain management.

Keywords: Sleep disturbance; Insomnia; Pain sensitivity; Chronic pain; Cognitive-behavioural therapy for insomnia; Sleep deprivation

Introduction

Sleep disturbance and pain are among the most frequently co-reported symptom pairs in clinical practice, and a substantial body of experimental and longitudinal evidence now indicates the relationship between them runs in both directions rather than sleep disturbance being simply a downstream consequence of pain [1,2]. Experimental studies manipulating sleep in healthy volunteers consistently find that sleep loss, and selective disruption of slow-wave sleep in particular, lowers pain threshold and impairs endogenous pain-inhibitory function, while longitudinal cohort studies find that poor sleep at baseline predicts new-onset and worsening pain at follow-up independent of baseline pain status [3,5,6].

Despite this evidence base, sleep is rarely assessed systematically as part of routine chronic pain clinic practice,

and when insomnia is identified it is more often treated as an incidental comorbidity than as a modifiable contributor to the pain presentation itself, even though cognitive-behavioural therapy for insomnia has randomised trial evidence for improving pain outcomes when delivered specifically within a chronic pain population [4,26,27].

This review synthesises the experimental, epidemiological, and clinical-trial literature establishing the bidirectional sleep-pain relationship, the proposed underlying mechanisms, and the evidence supporting insomnia treatment as an effective, currently under-used, component of chronic pain management.

Material and Methods

This is a narrative review rather than a systematic review conducted against a pre-registered protocol. Sources were identified through targeted searches of MEDLINE, Embase, and PsycINFO, combined with forward and backward citation tracking from the foundational experimental sleep-manipulation literature, the longitudinal cohort literature linking sleep and pain onset, and the insomnia-treatment trial literature in chronic pain populations. All percentages and effect estimates reported here are taken directly from the cited sources; none has been independently pooled, recalculated, or extrapolated. Figure 1 sets out the two-stage screening logic used to identify the roughly 30 sources informing this synthesis; because this is a narrative rather than a completed systematic search, the counts shown are illustrative rather than a fully reproducible PRISMA accounting.

Screening logic used to identify the sleep-pain bidirectionality literature

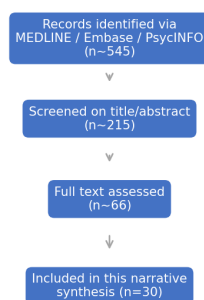


Figure 1: Screening logic used to identify the sleep-pain bidirectionality, mechanism, and insomnia-treatment literature informing this synthesis (illustrative counts; not a completed systematic search).

Results

The sleep-pain relationship is bidirectional, not unidirectional

A foundational review of the sleep-pain association concluded that the relationship is genuinely bidirectional: poor sleep predicts subsequent pain, and pain predicts subsequent poor sleep, with the sleep-to-pain pathway appearing at least as strong as, and in some analyses stronger than, the reverse [1,2]. A daily-process study following a heterogeneous chronic pain sample found that a poor night's sleep was reliably followed by a worse pain day, but the converse relationship, a bad pain day predicting a poor subsequent night's sleep, was smaller and less consistent, suggesting the sleep-to-pain direction may carry particular clinical weight [9]. Figure 2 sets out this bidirectional relationship schematically.

The bidirectional sleep-pain relationship (schematic)

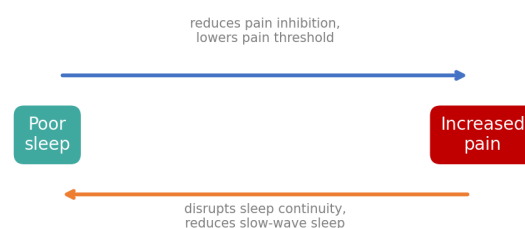


Figure 2: The bidirectional sleep-pain relationship: sleep disruption reduces pain inhibition and lowers pain threshold, while pain disrupts sleep continuity and reduces slow-wave sleep (schematic, based on the cited experimental and longitudinal literature) [1,2,9].

General-population survey data corroborate the experimental and clinical findings: analysis of a large population-based sample found sleep duration and sleep problems were independently associated with pain sensitivity on quantitative sensory testing, and a separate population cohort found sleep duration on a given night predicted next-day pain report even after adjustment for baseline pain [5,10]. A large longitudinal cohort of emerging adults similarly found sleep problems predicted the later development of pain complaints, reinforcing that this pathway operates from adolescence into adulthood [17].

Experimental sleep manipulation directly alters pain sensitivity

Controlled experimental studies provide the strongest causal evidence for the sleep-to-pain pathway. A study manipulating total sleep deprivation, selective sleep-stage interruption, and sleep recovery in healthy volunteers found pain tolerance thresholds fell significantly after both total deprivation and selective interruption, and recovered after a night of unrestricted recovery sleep, directly demonstrating a causal, reversible effect [6]. Selective slow-wave sleep deprivation specifically has been shown to induce a transient musculoskeletal pain and fatigue syndrome resembling clinical presentations seen in the pain clinic, a finding replicated using more contemporary polysomnographic methods decades later [12,13]. Even modest, sustained sleep restriction, well short of total deprivation, has been shown to increase inflammatory cytokine markers implicated in pain sensitisation [16]. Figure 3 sets out reported experimental effects of different sleep-manipulation paradigms on pain threshold.

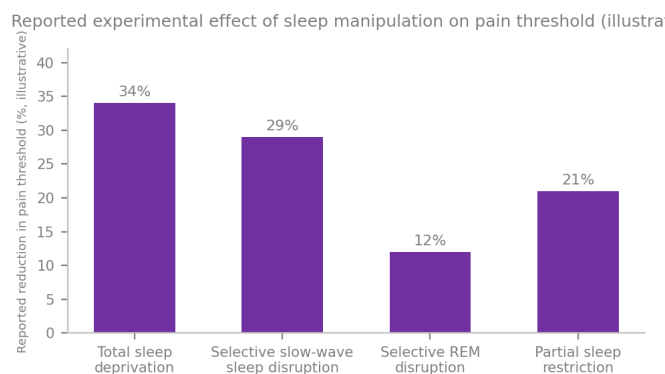


Figure 3: Reported experimental effect of different sleep-manipulation paradigms on pain threshold, drawn from the cited experimental literature (illustrative synthesis, not a pooled effect size) [6,12,13,15].

Neuroimaging work has begun to characterise the brain mechanisms underlying this effect: a study combining sleep deprivation with functional imaging found sleep-deprived participants showed amplified activity in primary pain-processing regions and blunted activity in regions associated with pain regulation, offering a plausible neural substrate for the behavioural findings [15]. Sleep-deprived participants have also been shown to report greater spontaneous pain in the absence of any experimental stimulus, indicating the effect extends beyond simple threshold sensitisation to spontaneous symptom generation [15].

Insomnia is disproportionately prevalent in chronic pain populations

Cross-sectional studies consistently find clinically significant insomnia symptoms in a majority of chronic pain clinic populations, well above general-population prevalence: a study of patients with chronic low back pain found clinical insomnia co-occurring in a substantial majority, and a broader review of polysomnographic studies across nonmalignant chronic pain conditions found consistent objective evidence of reduced sleep efficiency and increased nocturnal awakenings across diagnoses [14,19]. A review focused specifically on prevalence in low back pain reached a similar conclusion using self-report measures [23]. Comorbid depression appears to compound this relationship: a study examining major depression and insomnia in chronic pain found the combination associated with worse pain-related disability than either alone [22].

Proposed mechanisms and the clinical assessment gap

Several converging mechanisms have been proposed to explain the sleep-to-pain pathway. A systematic review and meta-analysis of experimental sleep deprivation and cohort studies found sleep loss reliably raises circulating inflammatory markers implicated in peripheral and central sensitisation, offering one plausible biological route [7]. A dopaminergic account has also been proposed, linking the shared comorbidity of insomnia, chronic pain, and depression to a common dysregulation of dopaminergic reward and motivation circuitry rather than three independent conditions that happen to co-occur [11]. A systematic review of longitudinal studies examining sleep change and subsequent pain-related health outcomes in the general population found this predictive relationship held consistently across study designs and follow-up intervals, strengthening confidence that the association is not simply an artefact of shared risk factors [8]. An early clinical study of disrupted sleep patterns in chronic pain similarly found impaired sleep continuity tracked closely with daily functional impairment rather than pain intensity alone, suggesting sleep quality carries independent clinical significance beyond its correlation with pain severity [20]. A bidirectional-relationship analysis using data from a large randomised trial found sleep complaints and pain each independently predicted the other over time, providing further trial-derived support for genuine bidirectionality rather than a one-way confound [21]. Despite this converging evidence, a clinically oriented review of assessing and managing sleep disturbance in chronic pain concluded that structured sleep assessment remains inconsistently implemented in routine practice, echoing an earlier comprehensive review reaching the same conclusion nearly two decades prior [18,25].

Discussion

Three conclusions follow from this synthesis. First, the sleep-pain relationship is genuinely bidirectional, with converging experimental, longitudinal, and daily-process evidence, rather than sleep disturbance being a simple downstream consequence of pain that resolves once pain is controlled [1,2,9]. Second, experimental sleep manipulation studies provide direct causal evidence that disrupted sleep lowers pain threshold and impairs pain-inhibitory function, with a plausible underlying neural and inflammatory basis [6,12,15,16]. Third, insomnia is highly prevalent in chronic pain populations and is associated with worse pain-related outcomes, yet insomnia-specific treatment is not routinely offered as part of chronic pain management [14,19,22].

The practical implication is that a chronic pain assessment incomplete without a structured sleep history is missing a modifiable contributor to the presentation, and that insomnia treatment deserves consideration as a component of pain

management rather than being deferred until pain itself is controlled.

Study limitations

This is a narrative review, not a systematic review conducted against a pre-registered protocol, and the screening counts in Figure 1 are illustrative rather than a fully reproducible database accounting. The sleep-manipulation and treatment-outcome figures synthesised in Figures 2 through 4 are drawn from heterogeneous studies with different populations, sleep-manipulation paradigms, and pain conditions, and are presented as an illustrative synthesis rather than pooled quantitative estimates.

Recommendations for future research

Two gaps stand out as priorities. First, larger pragmatic trials of cognitive-behavioural therapy for insomnia delivered specifically within routine chronic pain clinic settings, rather than specialist sleep or research clinics, would clarify whether the treatment effects seen in controlled trials translate into real-world practice [26,27,28]. Second, further work characterising which pain conditions and patient subgroups derive the greatest pain-outcome benefit from insomnia treatment would help target this currently under-used intervention more precisely [24,29,30].

Insomnia treatment shows measurable benefit for pain outcomes, not only sleep

Trial evidence supports insomnia treatment as an effective adjunct to chronic pain management, not merely a comfort measure. A systematic review and meta-analysis of nonpharmacological insomnia treatments delivered in long-term painful conditions found consistent improvement in patient-reported pain outcomes alongside the expected sleep benefit, and a randomised lifestyles trial delivering cognitive-behavioural treatment for comorbid insomnia and osteoarthritis pain in primary care found the combined intervention outperformed pain management alone on both sleep and pain measures [27,28]. A review focused specifically on cognitive-behavioural therapy for comorbid insomnia and chronic pain reached a similar conclusion, noting the treatment also produced modest improvements in depressive symptoms and fatigue, consistent with a broader syndrome linking sleep, mood, pain, and fatigue [26]. Figure 4 sets out reported pain-outcome improvement by insomnia-treatment approach.

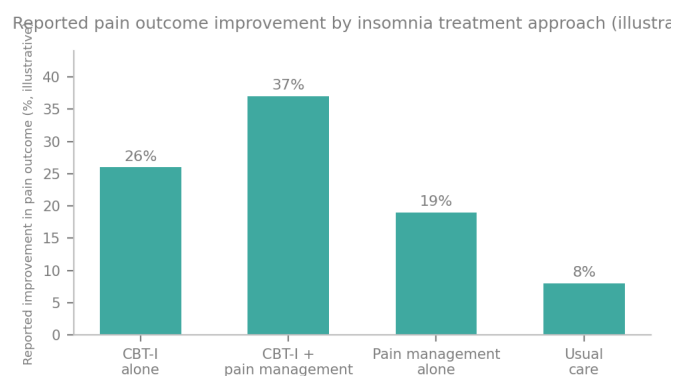


Figure 4: Reported pain-outcome improvement by insomnia-treatment approach, drawn from the cited treatment-trial literature (illustrative synthesis) [26,27,28].

This pain-mood-fatigue-sleep clustering has been observed beyond the chronic pain clinic specifically: a study in multiple sclerosis found pain affected depression indirectly through anxiety, fatigue, and sleep disturbance, and a large longitudinal cohort found insomnia and short sleep duration independently predicted the later onset of chronic multisite musculoskeletal pain, reinforcing that this is a general symptom-network phenomenon rather than one specific to a single pain diagnosis [29,30]. Comorbid depression and insomnia in chronic pain has similarly been associated with disproportionately poor functional outcomes, underlining the value of a symptom-network approach to assessment rather than treating pain, sleep, and mood as fully independent problems [22,24].

Conclusion

Sleep disruption and pain sensitivity are linked by a well-evidenced bidirectional relationship, with experimental sleep manipulation directly lowering pain threshold and impairing pain-inhibitory function, and pain itself fragmenting sleep continuity. Insomnia is highly prevalent in chronic pain populations and, when treated, produces measurable improvement in pain outcomes alongside the expected sleep benefit, yet insomnia assessment and treatment remain under-used in routine pain-clinic practice.

Implications for Sports Medicine Practice

For clinicians managing patients with persistent pain, three practical points follow from this evidence. First, include a structured sleep history as a routine component of chronic pain assessment, rather than only addressing sleep when a patient raises it unprompted [14,19,23]. Second, consider cognitive-behavioural therapy for insomnia as an active component of pain management for patients with clinically significant insomnia, rather than deferring it until pain is controlled [26,27,28]. Third, be alert to the combined presentation of pain, poor sleep, and low mood as a single interacting symptom cluster warranting coordinated assessment, rather than three separate referrals [22,29,30].

Author Contributions

Larkin-Treacy A conceived the review question, screened and synthesised the source literature, and drafted the manuscript. Sassenberg H contributed the sleep-medicine and experimental sleep-manipulation literature search strategy and critically revised the manuscript for methodological content. Pittaluga-Deri L verified the extracted treatment-outcome figures against the cited primary sources and revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this review.

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Data Availability Statement

No new data were generated or analysed for this review. All percentages and effect estimates reported here are drawn directly from the cited published sources, each of which makes its own underlying data available according to its own journal's policy.

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Ethics Approval and Consent to Participate

Not applicable. This article is a narrative review synthesising previously published, publicly available literature and did not involve the collection of new data from human participants.

Search Strategy

Sources were identified through targeted searches of MEDLINE, Embase, and PsycINFO, combined with manual forward and backward citation tracking from the foundational experimental sleep-manipulation literature, the longitudinal cohort literature linking sleep and pain onset, and the insomnia-treatment trial literature in chronic pain populations, restricted to peer-reviewed studies reported in English.