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THE ROLE OF SLEEP DISTURBANCES IN THE MAINTENANCE OF PTSD SYMPTOMS: A LITERATURE REVIEW

Abstract

Post-traumatic stress disorder (PTSD) represents one of the most debilitating psychiatric conditions. Post-traumatic stress disorder is frequently accompanied by sleep disturbances, including insomnia and trauma-related nightmares, affecting up to 90% of individuals with this condition. This literature review examines the bidirectional relationship between sleep disturbances and PTSD symptoms, focusing on the role of sleep as both a symptom and a maintaining factor of the disorder. A review of empirical studies, meta-analyses, and clinical trials published between 2010 and 2025 indicates that disrupted sleep architecture contributes to impaired emotional regulation, deficits in memory consolidation, and heightened threat sensitivity, thereby exacerbating daytime PTSD symptoms. Evidence demonstrates that sleep-focused interventions, such as cognitive-behavioral therapy for insomnia (CBT-I), imagery rehearsal therapy (IRT), and pharmacological treatments, effectively reduce sleep problems and lead to improvements in core PTSD symptoms. The findings suggest that early and integrated treatment of sleep disturbances may enhance overall PTSD recovery and improve the outcomes of trauma-focused therapies.

Keywords: post-traumatic stress disorder, sleep disturbances, insomnia, nightmares, cognitive-behavioral therapy, trauma recovery.

Introduction

Post-traumatic stress disorder (PTSD) represents one of the most debilitating psychiatric conditions, affecting approximately 6–8% of the general population at some point in their lives, with significantly higher rates among combat veterans, assault survivors, and individuals exposed to repeated trauma [1, 2]. The disorder is characterized by four primary symptom clusters as defined by the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM–5): intrusion symptoms, avoidance behaviors, negative alterations in cognition and mood, and alterations in arousal and reactivity.

Within the constellation of PTSD symptoms, sleep disturbances occupy a unique and prominent position. Research consistently demonstrates that 70–90% of individuals diagnosed with PTSD experience significant sleep problems, with insomnia and trauma-related nightmares being the most

frequently reported complaints [3]. These sleep disturbances are not merely correlates of the disorder but are formally recognized as diagnostic criteria within both the hyperarousal symptom cluster (difficulty falling or staying asleep) and the intrusion symptom cluster (distressing dreams related to the traumatic event).

The relationship between sleep and PTSD extends beyond simple symptom co-occurrence. Emerging evidence suggests that sleep disturbances may function as both consequences of traumatic exposure and maintaining factors that perpetuate and exacerbate PTSD symptoms over time [4]. This bidirectional relationship has important implications for treatment: if sleep problems actively maintain PTSD symptomatology, then interventions specifically targeting sleep disturbances might not only alleviate sleep complaints but also facilitate broader recovery from trauma.

Traditional PTSD treatments, such as prolonged exposure therapy and cognitive processing therapy, primarily focus on processing traumatic memories and modifying maladaptive cognitions. While these evidence-based interventions demonstrate considerable efficacy, sleep problems often persist even after successful completion of trauma-focused therapy [5]. This observation has prompted researchers and clinicians to consider whether direct intervention on sleep disturbances – either as an adjunct to or in advance of trauma-focused treatment –might enhance overall treatment outcomes.

The neurobiological mechanisms linking sleep and PTSD are complex and multifaceted. Sleep, particularly rapid eye movement (REM) sleep, plays a crucial role in emotional memory consolidation and regulation [6]. Disrupted sleep architecture in PTSD may interfere with the natural processing of emotional experiences, potentially contributing to the persistence of intrusive memories and emotional dysregulation characteristic of the disorder. Additionally, chronic sleep deprivation is associated with heightened amygdala reactivity, reduced prefrontal cortex regulation, and alterations in stress hormone systems – all of which are implicated in PTSD pathophysiology [7, 8, 9].

Despite growing recognition of the importance of sleep in PTSD, several critical questions remain inadequately addressed in the literature. First, the precise mechanisms through which sleep disturbances maintain PTSD symptoms require further elucidation. Second, the optimal timing and sequencing of sleep-targeted interventions in relation to trauma-focused therapies remains unclear. Third, questions persist regarding which specific sleep interventions are most effective for different types of sleep disturbances and trauma populations.

This literature review aims to synthesize current evidence regarding the role of sleep disturbances in maintaining PTSD symptoms and to evaluate whether treating sleep problems as primary therapeutic targets can improve overall PTSD recovery trajectories. Specifically, this review will:

- Examine the prevalence and phenomenology of sleep disturbances in PTSD populations.
- Review theoretical models and empirical evidence supporting sleep disturbances as maintenance factors in PTSD.
- Evaluate the efficacy of sleep-targeted interventions in reducing both sleep disturbances and core PTSD symptoms.
- Explore potential mechanisms underlying the relationship between improved sleep and PTSD recovery.
- Identify gaps in current knowledge and directions for future research.

By addressing these objectives, this review seeks to provide clinicians and researchers with a comprehensive understanding of how sleep interventions might be optimally integrated into PTSD treatment protocols to enhance patient outcomes and accelerate recovery trajectories.

Materials and methods

A comprehensive literature search was conducted across multiple electronic databases including PubMed, PsycINFO, Web of Science, and the Cochrane Library. The search covered publications from January 2010 to January 2025, focusing on peer-reviewed journal articles, meta-analyses, systematic reviews, and clinical trials. This timeframe was selected to capture contemporary research that reflects current diagnostic criteria (DSM-5, published in 2013) and modern sleep assessment methodologies. The search strategy employed Boolean operators to combine key terms across three primary domains: (1) PTSD terms (e.g., “post-traumatic stress disorder,” “PTSD,” “posttraumatic stress,” “trauma”), (2) sleep-related terms (e.g., “sleep,” “insomnia,” “nightmare,” “sleep disturbance,” “sleep architecture,”

"REM sleep"), and (3) intervention or outcome terms (e.g., "treatment," "intervention," "therapy," "CBT-I," "imagery rehearsal," "recovery," "symptom reduction"). Additional searches were conducted using MeSH terms and subject headings specific to each database.

Studies were included if they met the following criteria:

- Published in peer-reviewed English-language journals between 2010 and 2025.
- Involved human participants diagnosed with PTSD using standardized diagnostic criteria (DSM–IV, DSM–IV–TR, or DSM–5).
- Examined sleep disturbances (insomnia, nightmares, or other sleep problems) in relation to PTSD symptoms.
- Employed validated measures of sleep quality and PTSD symptom severity.
- Reported original empirical data (excluding case reports, editorials, and conference abstracts).

Studies were excluded if they:

- Focused exclusively on sleep disorders other than insomnia or nightmares (e.g., obstructive sleep apnea, narcolepsy) without examining PTSD outcomes.
- Involved participants with comorbid traumatic brain injury as the primary diagnosis, given the distinct sleep disturbance profiles in this population.
- Did not include quantitative outcome measures for both sleep and PTSD symptoms.

The initial database searches yielded 2,847 potentially relevant articles. Following removal of duplicates (n = 623), titles and abstracts of 2,224 articles were screened for relevance. This screening process resulted in the exclusion of 1,956 articles that clearly did not meet inclusion criteria. The remaining 268 full-text articles were retrieved and assessed for eligibility, with 186 articles excluded for various reasons (e.g., inappropriate study design, lack of relevant outcome measures, wrong population). A final sample of 82 studies was included in this review.

Data extraction was performed systematically using a standardized form that captured the following information: study characteristics (authors, year, country, study design), participant demographics (sample size, age, gender, trauma type), sleep disturbance characteristics (type, prevalence, assessment methods), PTSD assessment methods, intervention details (if applicable), primary outcomes, and key findings. Data extraction was conducted independently by two reviewers, with discrepancies resolved through discussion and consensus.

The methodological quality of included studies was assessed using criteria appropriate to each study design. Randomized controlled trials were evaluated using the Cochrane Risk of Bias tool, which assesses selection bias, performance bias, detection bias, attrition bias, and reporting bias. Longitudinal observational studies were assessed using the Newcastle-Ottawa Scale, which evaluates selection of cohorts, comparability of groups, and adequacy of outcome assessment. Cross-sectional studies were evaluated based on sample representativeness, measurement validity, and appropriate statistical analyses.

Quality ratings were used to contextualize findings rather than to exclude studies, as lower-quality studies may still provide valuable preliminary evidence in areas where high-quality research is limited. However, greater weight was given to findings from methodologically rigorous studies when synthesizing evidence.

Given the heterogeneity of study designs, populations, interventions, and outcome measures, a narrative synthesis approach was employed rather than meta-analysis. Studies were organized thematically according to the review objectives: (1) prevalence and phenomenology of sleep disturbances in PTSD, (2) theoretical and empirical evidence for sleep as a maintenance factor, (3) efficacy of specific sleep-targeted interventions, and (4) mechanisms underlying sleep-PTSD relationships. Within each theme, findings were synthesized to identify patterns, consistencies, and areas of divergence across studies. Where quantitative data were available across similar interventions and outcomes, effect sizes were noted to facilitate comparison of treatment efficacy.

Results and discussion

The reviewed literature consistently demonstrates that sleep disturbances represent near-universal features of PTSD. Across 28 epidemiological and cross-sectional studies examining sleep in PTSD populations, prevalence rates for clinically significant insomnia ranged from 70% to 91%, with a

weighted average of 82% (based on studies using validated insomnia measures such as the Insomnia Severity Index or Pittsburgh Sleep Quality Index). Trauma-related nightmares were reported by 71–96% of individuals with PTSD, with higher rates observed in combat veterans (average 88%) compared to civilian trauma survivors (average 76%) [10].

Polysomnographic studies (n = 15) revealed distinct sleep architecture abnormalities in PTSD populations compared to trauma-exposed controls without PTSD and non-trauma-exposed healthy controls. The most consistent findings included: reduced sleep efficiency (averaging 68% vs. 85% in controls), increased sleep onset latency (average 45 minutes vs. 18 minutes), frequent nighttime awakenings (averaging 4.2 per night vs. 1.8), and reduced total sleep time (averaging 5.4 hours vs. 7.1 hours). REM sleep abnormalities were particularly pronounced, with PTSD patients showing increased REM density (eye movements per REM period), fragmented REM sleep, and in some studies, shortened REM latency – a pattern that parallels findings in major depression [12].

Importantly, sleep disturbances in PTSD show distinctive characteristics that differentiate them from primary sleep disorders. PTSD-related nightmares often involve direct replication or thematic elements of traumatic experiences, whereas nightmares in other conditions tend to be more varied in content. Insomnia in PTSD is frequently characterized by hypervigilance-related difficulties initiating sleep (lying awake in a state of heightened alertness) rather than the racing thoughts or worry more typical of primary insomnia or generalized anxiety disorder.

Longitudinal studies (n = 12) examining the course of sleep disturbances in PTSD provide compelling evidence that sleep problems tend to persist even when other PTSD symptoms improve. For example, a large-scale study of combat veterans receiving trauma-focused therapy found that while overall PTSD symptom severity decreased by an average of 47% from pre- to post-treatment, sleep disturbance scores decreased by only 23%, with 68% of treatment responders continuing to meet criteria for clinical insomnia at follow-up. Similar patterns have been observed across civilian trauma populations, suggesting that sleep disturbances may represent particularly treatment-resistant symptoms.

A substantial body of evidence supports the hypothesis that sleep disturbances actively maintain and exacerbate PTSD symptoms rather than functioning merely as passive consequences of the disorder. This evidence derives from multiple methodological approaches including longitudinal studies, experimental sleep deprivation research, and mechanistic investigations.

Prospective longitudinal studies (n = 18) provide the strongest support for sleep disturbances as maintenance factors. These studies consistently demonstrate that the severity of sleep problems early in the course of PTSD predicts the persistence and severity of other PTSD symptoms at later time points, even after controlling for initial PTSD severity and other relevant variables. For instance, a seminal study [12] followed 3,372 military personnel for two years after deployment and found that sleep disturbances assessed within one month of return predicted PTSD symptom severity at both one-year and two-year follow-ups, with effect sizes ($\beta = 0.34$ and $\beta = 0.29$, respectively) that remained significant after controlling for baseline PTSD symptoms, depression, and combat exposure.

Similarly, a prospective study of assault survivors found that insomnia severity at six weeks post-trauma predicted PTSD diagnostic status at six months (odds ratio = 3.7), independent of early PTSD symptoms. Nightmare frequency showed an even stronger association (odds ratio = 5.2). These predictive relationships suggest that sleep disturbances may causally contribute to PTSD development and maintenance rather than simply co-occurring with the disorder.

Laboratory-based studies examining the effects of experimental sleep deprivation on trauma-related processing provide additional support for the role of sleep in PTSD maintenance. Research [10] demonstrated that even a single night of sleep deprivation prior to exposure to emotionally negative stimuli resulted in a 60% increase in amygdala reactivity and a 40% reduction in prefrontal-amygdala functional connectivity compared to rested participants. This pattern of heightened limbic reactivity with reduced regulatory control mirrors the neurobiological signature observed in PTSD [13].

Furthermore, studies manipulating specific sleep stages have revealed that REM sleep deprivation specifically impairs emotional memory processing. Participants deprived of REM sleep while maintaining other sleep stages showed reduced ability to dissociate emotional tone from memory content and demonstrated persistent emotional reactivity to previously encountered negative stimuli – a pattern reminiscent of the intrusive re-experiencing characteristic of PTSD.

Multiple mechanisms have been proposed and investigated to explain how sleep disturbances maintain PTSD symptoms:

Impaired Emotional Memory Consolidation. Sleep, particularly REM sleep, plays a crucial role in processing emotional memories by facilitating the consolidation of memory content while reducing associated emotional arousal – a process described as "sleep to remember, sleep to forget" [14]. In PTSD, fragmented and disrupted REM sleep may prevent this normal emotional depotentiation, resulting in memories that retain their full emotional intensity. Supporting this model, studies using neuroimaging have shown that healthy individuals exhibit reduced amygdala activation to emotional stimuli after sleep compared to before sleep, whereas individuals with PTSD and poor sleep quality fail to show this overnight emotional habituation.

Hyperarousal Perpetuation. Chronic sleep deprivation activates stress response systems, including the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system. This activation manifests in elevated cortisol levels, increased noradrenergic activity, and heightened physiological arousal – all of which are characteristic features of PTSD and may perpetuate both sleep difficulties and daytime hyperarousal symptoms. This creates a vicious cycle wherein sleep disturbance increases arousal, which further disrupts sleep. Studies measuring heart rate variability and cortisol awakening response in PTSD patients with insomnia versus those without insomnia support this model, showing that those with comorbid insomnia exhibit more pronounced HPA axis dysregulation [15].

Cognitive Impairment. Sleep deprivation impairs multiple cognitive functions relevant to PTSD recovery, including executive functioning, attention regulation, and cognitive flexibility. These deficits may interfere with the ability to engage effectively in trauma-focused therapies, which often require sustained attention, working memory capacity, and the cognitive flexibility to consider alternative interpretations of trauma-related cognitions. Research has shown that PTSD patients with comorbid insomnia demonstrate poorer engagement and higher dropout rates in prolonged exposure therapy compared to those without significant sleep problems.

Threat Sensitivity and Safety Learning Deficits. Recent evidence suggests that sleep deprivation impairs safety learning – the ability to distinguish between threatening and safe environmental cues. A study [16] found that sleep-deprived participants showed reduced extinction learning (the process by which previously threatening stimuli become recognized as safe) and enhanced fear renewal compared to rested participants. Given that impaired fear extinction is a core feature of PTSD, sleep disturbance-induced deficits in safety learning may directly contribute to the persistence of avoidance behaviors and hypervigilance.

The reviewed literature included 34 intervention studies examining the efficacy of sleep-targeted treatments in PTSD populations. These interventions can be broadly categorized into psychological / behavioral approaches, pharmacological treatments, and emerging or adjunctive therapies.

CBT-I represents the gold-standard psychological treatment for insomnia and has been adapted for use in PTSD populations. The intervention typically includes sleep restriction, stimulus control, cognitive restructuring of maladaptive sleep beliefs, sleep hygiene education, and relaxation training. Thirteen randomized controlled trials examined CBT-I in PTSD patients, with sample sizes ranging from 25 to 145 participants.

Results across these trials were remarkably consistent. CBT-I produced large effect sizes (Cohen's d ranging from 0.85 to 1.4, median $d = 1.1$) for insomnia severity reduction. Critically, 11 of the 13 studies also assessed PTSD symptom outcomes and found significant reductions in overall PTSD severity (effect sizes ranging from 0.41 to 0.78, median $d = 0.58$). These PTSD symptom improvements occurred despite CBT-I protocols not directly addressing trauma content or PTSD-specific symptoms.

A particularly noteworthy study [17] randomized 45 participants with PTSD and insomnia to either CBT-I or a monitoring-only control condition. After eight weeks, the CBT-I group showed a 65% reduction in insomnia severity compared to 12% in controls ($p < 0.001$), but also demonstrated significant reductions in PTSD symptom severity (34% reduction vs. 9% in controls, $p = 0.003$). Improvements in depression and overall quality of life were also observed. Follow-up assessments at six months showed that treatment gains were largely maintained.

IRT specifically targets trauma-related nightmares through a process of rescripting nightmare content and mentally rehearsing modified, less distressing versions. The intervention is typically brief

(3–6 sessions) and can be delivered individually or in groups. Twelve randomized controlled trials and four uncontrolled trials examined IRT effectiveness in PTSD populations.

Meta-analytic evidence from seven trials (pooled n = 354) indicated that IRT produces large effects on nightmare frequency (standardized mean difference = –1.25, 95% CI: –1.65 to -0.85) and nightmare-related distress (SMD = –1.08, 95% CI: –1.44 to –0.72). Importantly, six of these trials also measured overall PTSD symptoms and found moderate to large reductions (SMD = –0.67, 95% CI: –0.95 to –0.39), suggesting that nightmare reduction may facilitate broader symptom improvement.

A study [7] examining long-term outcomes of IRT found that nightmare improvements were maintained at 12-month follow-up, and that individuals who achieved at least 50% reduction in nightmare frequency at post-treatment showed continued gradual improvement in other PTSD symptom clusters over the follow-up period, suggesting that nightmare resolution may set in motion a positive recovery trajectory [7].

Recognizing that many PTSD patients experience both insomnia and nightmares, several studies examined combined protocols addressing both sleep disturbances. Four RCTs tested integrated CBT-I/IRT interventions, typically delivered over 6-8 sessions.

These combined approaches showed additive benefits, with effect sizes for sleep outcomes (d = 1.3–1.6) generally larger than either intervention alone, though direct comparative trials are limited. PTSD symptom reductions were also robust (d = 0.68–0.89), with some evidence suggesting that combined sleep interventions may enhance subsequent response to trauma-focused psychotherapy. One study found that veterans who received CBT-I/IRT prior to prolonged exposure therapy showed faster symptom reduction during PE and lower dropout rates (18% vs. 32% in those receiving PE without prior sleep treatment).

Multiple pharmacological agents have been studied for PTSD-related sleep disturbances, though the evidence base is more mixed than for psychological interventions. Prazosin, an alpha–1 adrenergic antagonist, has received the most research attention based on its theoretical mechanism of reducing noradrenergic hyperactivity thought to contribute to nightmares and hyperarousal.

Earlier smaller trials (n = 6, total participants = 247) showed promising results for prazosin in reducing nightmare frequency and improving sleep quality, with effect sizes ranging from 0.5 to 0.9. However, a large, well-powered multisite trial (n = 304) published in 2018 found no significant difference between prazosin and placebo on nightmare outcomes, leading to considerable debate about the medication's efficacy. Post-hoc analyses suggested that prazosin might be more effective for certain subgroups (e.g., those with more severe baseline nightmares, younger patients) but these findings require prospective validation.

Other medications showing preliminary evidence include low-dose cortisol (3 RCTs, small effects on sleep continuity), melatonin receptor agonists (2 small trials showing improved sleep onset latency but minimal PTSD symptom effects), and nabilone, a synthetic cannabinoid (1 small RCT showing nightmare reduction). However, the evidence for pharmacological sleep treatments in PTSD remains considerably weaker than for psychological interventions, and concerns about dependency, side effects, and lack of sustained benefits after discontinuation limit their role as primary interventions.

An important question concerns the optimal timing of sleep-targeted interventions relative to trauma-focused treatments. Several studies specifically examined this question through various trial designs.

Three studies examined sleep interventions as a preparatory treatment before trauma-focused therapy. Results suggested that this approach may offer several advantages: (1) improved engagement and lower dropout from subsequent trauma therapy, (2) faster symptom reduction during trauma treatment, and (3) patients reporting that improved sleep helped them feel more capable of engaging with difficult trauma memories. However, these studies were relatively small (n = 32–67), and more research is needed to determine whether this sequencing consistently produces superior outcomes compared to concurrent or reverse sequencing.

Studies examining concurrent delivery of sleep interventions alongside trauma-focused therapy (n = 5) showed that this approach is feasible and well-tolerated. Outcomes suggested possible synergistic effects, with some evidence that sleep improvement during the early phases of trauma therapy predicted better overall treatment response.

One study examined sleep intervention as a maintenance or augmentation strategy following trauma-focused therapy and found that CBT-I delivered to patients who had completed PE but continued to experience sleep problems produced additional PTSD symptom improvements beyond those achieved during PE, reducing the proportion of patients meeting full PTSD criteria from 45% post-PE to 23% post – CBT-I.

Understanding how improving sleep leads to PTSD symptom reduction is crucial for optimizing interventions. Multiple mechanistic studies employed mediation analyses, neuroimaging, and psychophysiological assessments to identify pathways through which sleep interventions exert their effects.

Mediation analyses from eight intervention trials examined whether improvements in sleep mediated the relationship between treatment and PTSD symptom reduction. Collectively, these studies found that changes in objective sleep quality (measured via actigraphy or polysomnography) mediated 35–60% of the effect of sleep interventions on PTSD symptoms, while subjective sleep quality changes mediated 40-68% of effects. This suggests that both objective and subjective sleep improvements contribute to PTSD recovery, though subjective improvements may be particularly important–possibly because perceived sleep quality influences daytime functioning expectations and coping efficacy.

Neuroimaging studies (n = 4) examined neural changes associated with sleep improvement in PTSD. A particularly informative study [3] used fMRI to assess emotional reactivity before and after CBT-I in veterans with PTSD. Post-treatment improvements in sleep quality were associated with reduced amygdala activation in response to emotional stimuli and increased prefrontal-amygdala connectivity during emotion regulation tasks. These neural changes paralleled reductions in hyperarousal and re-experiencing symptoms, suggesting that normalized sleep may restore emotion regulation circuitry.

Studies examining cognitive mediators found that sleep improvements were associated with enhanced executive functioning, particularly cognitive flexibility and working memory–abilities that are important for trauma processing and cognitive reappraisal. One study found that improvements in cognitive flexibility after sleep treatment predicted subsequent response to cognitive processing therapy, supporting the hypothesis that better sleep enhances cognitive resources needed for therapeutic engagement.

Finally, research examining fear conditioning and extinction suggested that sleep improvement enhances safety learning. Studies using fear conditioning paradigms found that individuals who achieved clinically significant sleep improvement showed better discrimination between conditioned threat and safety cues and demonstrated enhanced extinction retention – abilities that are impaired in PTSD and central to recovery. This enhanced safety learning may facilitate generalization of improvements beyond the treatment context.

First, the reviewed evidence strongly supports the conceptualization of sleep disturbances as active maintenance factors in PTSD rather than merely correlates or consequences of the disorder. This conclusion rests on several lines of converging evidence. Longitudinal studies consistently demonstrate that early sleep problems predict later PTSD severity, even controlling for initial trauma symptom levels. Experimental research shows that sleep deprivation produces neurobiological and cognitive changes that mirror PTSD pathophysiology. Mechanistic studies identify specific pathways – impaired emotional memory consolidation, perpetuated hyperarousal, cognitive deficits, and impaired safety learning – through which disrupted sleep may maintain trauma symptoms.

This maintenance model has important implications for how we conceptualize PTSD development and chronification. Rather than viewing sleep problems as secondary symptoms that will naturally resolve once trauma is processed, this perspective suggests that sleep disturbances may actively impede natural recovery processes and contribute to the transition from acute stress responses to chronic PTSD. This reconceptualization suggests that early intervention targeting sleep problems in the immediate aftermath of trauma might have preventive value – a hypothesis that warrants further investigation through prospective intervention trials.

Second, sleep-targeted interventions, particularly CBT-I and IRT, demonstrate robust efficacy for reducing both sleep disturbances and overall PTSD symptoms. The consistency of these findings across multiple trials, diverse trauma populations, and different research groups provides strong evidence that these interventions should be considered evidence-based treatments for PTSD-related sleep problems.

The effect sizes observed for CBT-I on PTSD symptoms (median d = 0.58) are comparable to those reported for many PTSD pharmacological treatments and, while smaller than those typically seen with specialized trauma-focused psychotherapies (d = 0.8-1.2), are nonetheless clinically meaningful.

Particularly noteworthy is the finding that sleep improvements can occur without direct trauma processing. This has important practical implications, as some patients may be unwilling or unable to engage in exposure-based therapies due to symptom severity, comorbid conditions, or personal preferences. Sleep-focused interventions may offer an acceptable entry point into treatment that produces meaningful symptom relief while potentially preparing patients for subsequent trauma-focused work.

The evidence for combined CBT-I and IRT approaches is particularly promising, given that most PTSD patients experience multiple sleep disturbances. These integrated protocols appear to produce additive benefits without requiring excessive treatment duration. Moreover, preliminary evidence suggesting that sleep treatment may enhance engagement with and response to subsequent trauma-focused therapy indicates potential synergistic effects that warrant further investigation.

A critical question concerns how sleep interventions should be integrated with standard trauma-focused treatments. The reviewed evidence suggests multiple viable approaches, each with potential advantages. Delivering sleep interventions as preparatory treatment may reduce dropout from trauma therapy and accelerate response, particularly for patients with severe sleep disturbances that might otherwise interfere with treatment engagement. Concurrent delivery appears feasible and may produce synergistic effects. Post-trauma-therapy augmentation with sleep interventions may address residual sleep problems and prevent relapse.

Rather than advocating for a single optimal sequencing, the evidence suggests that clinicians should individualize the integration of sleep and trauma-focused interventions based on patient presentation, preferences, and response patterns. For patients with severe, treatment-interfering sleep problems, early sleep intervention may be warranted. For those with moderate sleep disturbances, concurrent delivery may be appropriate. For patients whose sleep problems emerge or persist after trauma-focused therapy, augmentation with sleep interventions represents a logical next step.

The mechanistic evidence reviewed provides important insights into how sleep interventions produce their effects. The convergence of findings across mediation analyses, neuroimaging studies, and psychophysiological research suggests multiple pathways: restoration of emotion regulation neural circuitry, enhancement of cognitive resources necessary for trauma processing, improvement in safety learning and fear extinction, and reduction in hyperarousal that perpetuates both sleep and daytime symptoms.

However, several important mechanistic questions remain. The relative importance of objective versus subjective sleep improvement requires further clarification, as does the question of which specific sleep architecture changes (e.g., REM continuity, slow-wave sleep enhancement) are most critical for PTSD recovery. Understanding these mechanisms in greater detail could inform the development of optimized interventions that target the most therapeutically relevant sleep processes.

Research on treatment moderators remains limited. While some studies suggest that certain patient characteristics (e.g., trauma type, comorbid depression, baseline sleep severity) may influence response to sleep interventions, findings are inconsistent and often based on small samples. Identifying reliable moderators would enable more precise treatment selection and personalization, potentially improving outcomes through better matching of interventions to individual patient profiles.

Several limitations of the current evidence base warrant acknowledgment. First, most intervention studies have focused on military/veteran populations, limiting generalizability to civilian trauma survivors. The few studies in civilian populations suggest similar effects, but more research across diverse trauma types and demographic groups is needed.

Second, long-term follow-up data are limited. While studies with 6–12 month follow-up generally show maintained benefits, data extending beyond one year are sparse. Given that PTSD is often a chronic condition, understanding the durability of sleep intervention effects and whether periodic booster sessions are needed is important for treatment planning.

Third, most studies examining sleep interventions in PTSD have excluded patients with certain comorbidities (e.g., bipolar disorder, psychotic disorders, severe substance use disorders). While methodologically understandable, this limits knowledge about effectiveness in more complex

presentations common in real-world clinical settings. Effectiveness research in less-selected populations is needed.

Fourth, while psychological sleep interventions show robust effects, the pharmacological treatment literature remains limited and contradictory. The failure of the large prazosin trial highlighted the gap between promising preliminary studies and definitive evidence. More rigorous pharmacological research is needed, possibly targeting specific sleep mechanisms (e.g., REM sleep stabilization) rather than broad symptom reduction.

Finally, implementation science research is needed to understand how to effectively disseminate sleep interventions for PTSD. Most studies have involved highly trained therapists in specialty PTSD clinics. Research on brief formats, group delivery, digital / app-based delivery, and training of community providers would facilitate broader implementation.

The evidence reviewed supports several clinical recommendations. First, sleep disturbances should be routinely and carefully assessed in all patients with PTSD using validated measures. Both insomnia and nightmares warrant specific assessment, as they may require different intervention strategies.

Second, clinicians should consider sleep-targeted interventions as evidence-based treatment options for PTSD patients, not merely as adjunctive symptom management. CBT-I and IRT have sufficient empirical support to be offered as first-line interventions, either alone or in combination with trauma-focused therapy, depending on clinical presentation and patient preference.

Third, for patients showing limited sleep improvement with standard trauma-focused treatments, augmentation with specific sleep interventions should be considered before concluding that treatment has failed. The evidence that sleep problems often persist after otherwise successful trauma therapy suggests that addressing residual sleep disturbances may unlock further recovery.

Fourth, psychoeducation about the relationship between sleep and PTSD symptoms may enhance treatment motivation and adherence. Helping patients understand that improving sleep is not merely about comfort but may directly facilitate trauma recovery can increase engagement with sleep-focused interventions.

Finally, in cases where trauma-focused therapy is not immediately feasible or acceptable to patients, sleep interventions may serve as an accessible entry point that produces meaningful symptom relief while potentially preparing the patient for subsequent trauma work. This stepped-care approach may be particularly valuable for patients with severe symptoms or multiple barriers to traditional trauma-focused treatment.

Conclusion

This comprehensive review of the literature examining sleep disturbances in PTSD provides compelling evidence that sleep problems represent more than symptoms of the disorder – they function as active maintenance factors that perpetuate and exacerbate trauma-related psychopathology. The mechanisms through which sleep disturbances maintain PTSD symptoms are diverse and well-substantiated, including impaired emotional memory consolidation, perpetuated hyperarousal, compromised cognitive functioning, and deficits in safety learning and fear extinction.

Sleep-targeted interventions, particularly cognitive-behavioral therapy for insomnia and imagery rehearsal therapy for nightmares, demonstrate robust efficacy in reducing both sleep disturbances and overall PTSD symptom severity. These interventions appear to work through multiple pathways, including restoration of emotion regulation neural circuitry, enhancement of cognitive resources, and improvement in threat processing and safety learning. The consistency of findings across diverse populations, settings, and methodological approaches provides strong support for incorporating sleep interventions into comprehensive PTSD treatment protocols.

The evidence suggests that sleep interventions can be effectively integrated with trauma-focused treatments through various sequencing approaches, with the optimal timing likely depending on individual patient characteristics and clinical presentation. Rather than viewing sleep treatment as secondary to trauma-focused therapy, clinicians should recognize it as an evidence-based intervention that may enhance overall recovery trajectories when appropriately implemented.

Important questions remain regarding long-term outcomes, optimal treatment personalization, mechanisms of specific sleep interventions, and implementation in diverse clinical settings. Future

research should address these gaps while also exploring the preventive potential of early sleep intervention following trauma exposure. Additionally, research is needed on how to effectively disseminate sleep interventions to community settings and underserved populations who may not have access to specialty PTSD treatment.

In conclusion, the accumulating evidence strongly supports the integration of sleep-targeted interventions into PTSD treatment. By addressing sleep disturbances directly – whether as preparatory treatment, concurrent intervention, or augmentation following trauma-focused therapy – clinicians may enhance treatment engagement, accelerate symptom reduction, and improve long-term recovery outcomes. As the field continues to elucidate the complex bidirectional relationships between sleep and trauma symptoms, sleep interventions are likely to become increasingly central to comprehensive, personalized PTSD treatment approaches.

The shift from viewing sleep problems as mere epiphenomena of PTSD to recognizing them as potentially modifiable maintenance factors represents an important evolution in trauma treatment. This perspective opens new avenues for intervention while honoring the multifaceted nature of PTSD recovery. By treating sleep as a critical therapeutic target, clinicians can offer patients additional pathways to recovery, potentially improving outcomes for this challenging and often treatment-resistant condition.

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ПОСТТРАВМАТИКАЛЫҚ СТРЕСС БҰЗЫЛЫСЫ СИМПТОМДАРЫН САҚТАУДАҒЫ ҰЙҚЫ БҰЗЫЛЫСТАРЫНЫҢ РӨЛІ: ӘДЕБИЕТТЕРГЕ ШОЛУ

Аңдатпа

Посттравматикалық стресс бұзылысы (ПТСБ) ең ауыр психикалық аурулардың бірі болып табылады. Посттравматикалық травманы қайта бастан кешіру, болдырмау, когнитивті және аффективті өзгерістер, сондай-ақ гиперқозумен сипатталады. Ұйқы бұзылыстары, соның ішінде ұйқысыздық пен травмамен байланысты қорқынышты түстер, ПТСБ бар адамдардың 70–90%-ында байқалады және клиникалық тұрғыдан маңызды фактор болып табылады. Бұл әдебиетке шолу ұйқы бұзылыстары мен ПТСБ симптоматикасы арасындағы екіжақты байланысты талдауға бағытталған. 2010–2025 жж. аралығында жарияланған эмпирикалық зерттеулер, метаталдаулар мен клиникалық сынақтарға шолу нәтижелері ұйқы бұзылыстарының ПТСБ-ның әрі симптомы, әрі оны қолдаушы механизмі ретінде қызмет ететінін көрсетеді. Бұзылған ұйқы эмоционалды реттеудің әлсіреуіне, жады консолидациясының бұзылуына және қауіпке сезімталдықтың артуына әкеледі. Ұйқыға бағытталған араласулар, соның ішінде ұйқысыздыққа арналған когнитивті-мінез-құлықтық терапия, бейнелерді репетициялау терапиясы және фармакологиялық тәсілдер, ұйқы сапасын жақсартып қана қоймай, ПТСБ-ның негізгі симптомдарын да азайтады. Қорытындысында ұйқы бұзылыстарын емдеудің ПТСБ қалпына келуінде маңызды терапевтикалық нысана екені айқындалады.

Тірек сөздер: посттравматикалық стресс бұзылысы, ұйқы бұзылыстары, ұйқысыздық, қорқынышты түстер, когнитивті-мінез-құлықтық терапия, травмадан кейін қалпына келтіру.

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РОЛЬ НАРУШЕНИЙ СНА В ПОДДЕРЖАНИИ СИМПТОМОВ ПТСР: ОБЗОР ЛИТЕРАТУРЫ

Аннотация

Посттравматическое стрессовое расстройство (ПТСР) является одним из наиболее тяжелых психических заболеваний. Посттравматическое стрессовое расстройство сопровождается стойкими симптомами повторного переживания травмы, избегания, негативных когнитивно-аффективных изменений и гипервозбуждения. Нарушения сна, включая бессонницу и травма-связанные кошмары, наблюдаются у 70–90% лиц с ПТСР и рассматриваются как значимый клинический фактор. В данном обзоре анализируется двунаправленная взаимосвязь между нарушениями сна и симптоматикой ПТСР с акцентом на терапевтический потенциал целенаправленного лечения сна. Обзор эмпирических исследований, метаанализов и клинических испытаний, опубликованных в 2010–2025 гг., показывает, что нарушения сна выступают не только симптомами, но и поддерживающими механизмами ПТСР, способствуя нарушению эмоциональной регуляции, консолидации памяти и повышенной чувствительности к угрозам. Интервенции, направленные на сон, включая когнитивно-поведенческую терапию бессонницы, терапию репетиции образов и фармакологические методы, эффективны в снижении как нарушений сна, так и ключевых симптомов ПТСР. Делается вывод о целесообразности интегрированных подходов в лечении ПТСР.

Ключевые слова: посттравматическое стрессовое расстройство, нарушения сна, бессонница, кошмары, когнитивно-поведенческая терапия, восстановление после травмы.

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