International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

ARTICLE TITLE	SLEEP DISTURBANCES IN MAJOR PSYCHIATRIC DISORDERS – PREVALENCE, MECHANISMS AND TREATMENT – A NARRATIVE REVIEW
DOI	https://doi.org/10.31435/ijitss.4(48).2025.4307
RECEIVED	23 October 2025
ACCEPTED	24 December 2025
PUBLISHED	29 December 2025
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

SLEEP DISTURBANCES IN MAJOR PSYCHIATRIC DISORDERS – PREVALENCE, MECHANISMS AND TREATMENT – A NARRATIVE REVIEW

Anna Maria Gęsińska *(Corresponding Author, Email: annagesinska@gmail.com)*
Antoni Jurasz University Hospital, Bydgoszcz, Poland
ORCID ID: 0009-0004-4336-8873

Krystian Czyżykowski
Antoni Jurasz University Hospital, Bydgoszcz, Poland
ORCID ID: 0009-0006-8816-1811

Helena Szelka
University Clinical Hospital No. 2 of the Medical University of Łódź, Łódź, Poland
ORCID ID: 0009-0001-7142-341X

Bartosz Golis
Central Clinical Hospital of the Medical University of Łódź, Łódź, Poland
ORCID ID: 0009-0007-2968-6797

Paweł Edyko
Central Clinical Hospital of the Medical University of Łódź, Łódź, Poland
ORCID ID: 0009-0009-6764-3434

Alicja Babula
T. Marciniak Lower Silesian Specialist Hospital – Centre for Emergency Medicine, Wroclaw, Poland
ORCID ID: 0009-0003-0573-5022

Wiktor Golus
University Clinical Hospital No. 2 of the Medical University of Łódź, Łódź, Poland
ORCID ID: 0009-0009-1930-9837

Katarzyna Andrzejewska
Medical University of Łódź, Łódź, Poland
ORCID ID: 0009-0001-6994-7505

Zuzanna Przybyła
Medical University of Łódź, Łódź, Poland
ORCID ID: 0009-0009-4778-3106

Hubert Woźniak
Copernicus Memorial Hospital, Łódź, Poland
ORCID ID: 0009-0002-9891-3248

ABSTRACT

Sleep disturbances are among the most prevalent and disabling symptoms across major psychiatric disorders, yet they remain underrecognized and undertreated. This review synthesizes findings from 43 peer-reviewed studies published between 2008 and 2025, examining the prevalence, neurobiological mechanisms, and treatment of sleep abnormalities in major depressive disorder (MDD), bipolar disorder (BD), schizophrenia (SCZ), and post-traumatic stress disorder (PTSD). Across disorders, insomnia is the most frequent disturbance, affecting up to 80% of patients with MDD and nearly 90% of those with PTSD. Other recurrent abnormalities include hypersomnia during depressive phases of BD, circadian rhythm misalignment, and fragmented sleep in SCZ. Neurobiological evidence implicates dysregulation of the circadian system, hyperactivity of the hypothalamic–pituitary–adrenal axis, orexin signaling imbalance, and neuroinflammatory processes as shared mechanisms underlying psychiatric sleep pathology.

Effective management requires a multimodal approach. Cognitive behavioral therapy for insomnia (CBT-I) demonstrates robust efficacy across disorders, improving both sleep and psychiatric outcomes. Pharmacological interventions—such as dual orexin receptor antagonists, melatonin agonists, and sedating psychotropics—show moderate benefits but require careful monitoring. Chronotherapeutic and neuromodulatory strategies (e.g., light therapy and noninvasive brain stimulation (NIBS) represent promising adjuncts for circadian stabilization.

Overall, sleep disturbances emerge as transdiagnostic phenomena that both reflect and exacerbate psychiatric pathology. Early identification and targeted treatment of sleep problems may enhance therapeutic response, reduce relapse risk, and improve overall functioning. Integrating systematic sleep assessment into routine psychiatric care is essential for achieving comprehensive, recovery-oriented treatment.

KEYWORDS

Sleep Disturbances, Major Psychiatric Disorders, Depression, Bipolar Disorder, Schizophrenia, Post-Traumatic Stress Disorder

CITATION

Anna Maria Gęsińska, Krystian Czyżykowski, Helena Szelka, Bartosz Golis, Paweł Edyko, Alicja Babula, Wiktor Golus, Katarzyna Andrzejewska, Zuzanna Przybyła, Hubert Woźniak. (2025). Sleep Disturbances in Major Psychiatric Disorders – Prevalence, Mechanisms and Treatment – A Narrative Review. *International Journal of Innovative Technologies in Social Science*. 4(48). doi: 10.31435/ijitss.4(48).2025.4307

Introduction

Sleep plays a fundamental role in emotional regulation, cognitive performance, and physiological homeostasis. Its disruption has long been recognized as a hallmark of psychiatric disorders, affecting both symptom presentation and treatment response. Historically, the conceptual framework for understanding sleep regulation was established by Borbély's (1982) *two-process model*, which postulated that sleep–wake patterns are governed by the interaction of homeostatic and circadian processes. This foundational model continues to inform contemporary theories linking sleep and mental health. Early psychiatric research viewed insomnia as a secondary symptom of mental illness, yet accumulating evidence over the past two decades has reframed sleep disturbances as core and causal components of psychopathology (Nutt et al., 2008).

Epidemiological data indicate that sleep abnormalities occur in nearly all major psychiatric disorders. In major depressive disorder (MDD), up to 80% of patients experience persistent insomnia, while 15–20% report hypersomnia (Fang et al., 2019; Yasugaki et al., 2025). Typical polysomnographic findings include shortened REM latency, increased REM density, and early-morning awakening. These sleep disruptions predict greater depression severity and poorer treatment outcomes (Yasugaki et al., 2025). In bipolar disorder (BD), sleep is profoundly altered across mood states—insomnia and reduced need for sleep are hallmarks of mania, whereas hypersomnia and delayed circadian phase are common in bipolar depression (Marchetti et al., 2025; Tonon et al., 2024). Harvey (2008) was among the first to conceptualize circadian misalignment as a core mechanism driving bipolar mood instability, a view now supported by chronobiological and neuroimaging evidence. Even during euthymic periods, irregular sleep–wake cycles often persist, suggesting trait-level dysregulation (Tonon et al., 2024).

Sleep disturbances are also pervasive in schizophrenia (SCZ). More than half of patients experience chronic insomnia, fragmented sleep architecture, or delayed sleep–wake cycles (Carruthers et al., 2021). Objective studies reveal reduced slow-wave sleep and disrupted sleep spindles—patterns associated with cognitive impairment and psychotic symptom exacerbation (Carruthers et al., 2021; Manoach & Stickgold, 2019). In post-traumatic stress disorder (PTSD), insomnia and trauma-related nightmares affect up to 90% of individuals, making sleep pathology a defining feature of the disorder (Mendoza Alvarez et al., 2025; Weber & Wetter, 2022). Nightmares are often accompanied by heightened nocturnal arousal and reduced REM stability, reflecting hyperactivation of limbic and noradrenergic systems (Agorastos & Olff, 2021).

Neurobiological research supports the view that disturbed sleep and psychiatric pathology share overlapping mechanisms. Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis, altered orexinergic signaling, serotonergic imbalance, and chronic inflammation are consistent findings across disorders (Kishi et al., 2024; Zielinski & Gibbons, 2022). Circadian desynchrony—resulting from misalignment between endogenous rhythms and environmental cues—further contributes to affective instability and cognitive dysfunction (Zou et al., 2022). Importantly, experimental evidence demonstrates that sleep deprivation can trigger or worsen psychiatric symptoms, whereas sleep restoration promotes emotional recovery and neural plasticity (Walker & van der Helm, 2009). This supports the notion that sleep disturbances not only mirror but also *mediate* psychopathological processes.

In recent years, researchers have increasingly adopted a transdiagnostic perspective, emphasizing that sleep disruption cuts across traditional diagnostic boundaries (Azargoonjahromi & Bahrampour, 2025). While the phenomenology varies—racing thoughts and insomnia in mania, nightmares in PTSD, or early awakening in depression—the underlying dysregulation of circadian, neuroendocrine, and arousal systems appears shared. This dimensional approach aligns with contemporary frameworks such as the Research Domain Criteria (RDoC), which view sleep–wake regulation as a cross-cutting construct underlying multiple psychiatric phenotypes.

The therapeutic implications of these insights are substantial. CBT-I has emerged as the gold standard for treating chronic insomnia, demonstrating efficacy across mood, psychotic, and anxiety-related disorders (Hertenstein et al., 2022; Mirchandaney et al., 2022). Pharmacological interventions—including dual orexin receptor antagonists (DORAs), melatonin receptor agonists, and sedating antidepressants—provide additional benefit in selected patients (Kishi et al., 2024; Moon et al., 2022). Furthermore, chronotherapeutic and neuromodulatory interventions such as bright light therapy and transcranial stimulation are being explored as adjuncts for stabilizing sleep–wake cycles (Stefanowski et al., 2025; Tonon et al., 2024).

Given the pervasive and mechanistically diverse nature of sleep disturbances across psychiatric conditions, this review synthesizes current evidence on their prevalence, neurobiological mechanisms, and treatment approaches, based on 43 peer-reviewed studies published between 2008 and 2025. By integrating classic theoretical models with modern empirical findings, the review aims to highlight sleep disturbance as a transdiagnostic therapeutic target with significant implications for prevention, prognosis, and recovery in mental health care.

Materials and Methods

This narrative review analyzed 43 peer-reviewed articles published between 2008 and 2025, including systematic reviews, meta-analyses, and seminal studies addressing sleep disturbances in major psychiatric disorders. The literature search was performed in PubMed, ScienceDirect, and Scopus databases using combinations of the keywords “sleep disturbances,” “psychiatric disorders,” “circadian rhythms,” “depression,” “bipolar disorder,” “schizophrenia,” and “post-traumatic stress disorder.” Reference lists of relevant publications were also screened to identify additional studies of potential relevance.

Eligible articles were those published in English that provided clinical, neurobiological, or therapeutic data related to sleep and circadian disruptions in psychiatric populations. Case reports, conference abstracts, editorials, and studies lacking direct relevance to the primary research question were excluded. Data from the included studies were narratively synthesized to identify consistent patterns in prevalence, underlying mechanisms, and treatment approaches. Quantitative data such as prevalence rates or effect sizes were reported descriptively where available, while qualitative trends were summarized thematically.

The methodological rigor of the reviewed studies was considered through a critical appraisal of research design, sample size, and diagnostic criteria. However, no formal scoring system or risk-of-bias tool was applied, consistent with the narrative and integrative nature of this review. As the study synthesized previously published data, ethical approval was not required.

Results

Prevalence of Sleep Disturbances in Major Psychiatric Disorders

Building upon the theoretical framework outlined above, the following section summarizes empirical findings on the prevalence of sleep disturbances across major psychiatric disorders.

Sleep disturbances are among the most pervasive and disabling transdiagnostic symptoms across major psychiatric disorders. Epidemiological and clinical studies consistently indicate that abnormalities in sleep quantity, quality, and circadian timing occur in the majority of individuals with major depressive disorder (MDD), bipolar disorder (BD), schizophrenia spectrum disorders (SCZ), and post-traumatic stress disorder (PTSD) (Crouse et al., 2021; Marchetti et al., 2025; Tonon et al., 2024; Weber & Wetter, 2022; Yasugaki et al., 2025).

Across psychiatric populations, insomnia remains the most common manifestation. In MDD, between 60% and 80% of patients experience insomnia symptoms such as prolonged sleep latency, early awakening, and nonrestorative sleep (Fang et al., 2019; Yasugaki et al., 2025). Conversely, 15–20% of depressed patients report hypersomnia or excessive sleepiness, particularly in atypical or seasonal subtypes (Nutt et al., 2008; Pandi-Perumal et al., 2020). Meta-analytic data show that chronic insomnia doubles the risk of depressive relapse and predicts poorer response to antidepressant treatment (Fang et al., 2019; Yasugaki et al., 2025). Polysomnographic studies reveal decreased slow-wave sleep, shortened REM latency, and increased REM density in MDD—changes that correlate with illness severity and suicidality (Pandi-Perumal et al., 2020; Yasugaki et al., 2025).

In bipolar disorder, sleep abnormalities are nearly universal, though their expression varies across mood states. During manic or hypomanic episodes, reduced need for sleep occurs in up to 90% of patients, while during depressive phases, hypersomnia affects approximately 30–40% (Marchetti et al., 2025; Tonon et al., 2024). Even in euthymic phases, 40–70% of patients report persistent sleep–wake irregularities, including delayed sleep onset and inconsistent wake times (Harvey, 2008; Tonon et al., 2024). These irregularities are not merely residual symptoms but may serve as early indicators of relapse or mood switching (Kragh et al., 2024; Scott et al., 2021). A meta-analysis of actigraphy studies found that bipolar patients exhibit significantly longer sleep latency and greater night-to-night variability than controls, with moderate-to-large effect sizes (Marchetti et al., 2025). Circadian misalignment, including delayed melatonin onset and reduced amplitude of rest–activity rhythms, has been linked to poorer cognitive functioning and psychosocial recovery (Pearson et al., 2023; Tonon et al., 2024). Across disorders, sleep disturbances appear more prevalent among women and younger adults, consistent with population-level trends (Marchetti et al., 2025; Tonon et al., 2024).

Schizophrenia spectrum disorders are also characterized by profound sleep–wake disruption. Objective studies report that 50–80% of patients experience insomnia, fragmented sleep, or circadian phase delay (Carruthers et al., 2021; Laskemoen et al., 2019). Polysomnographic findings consistently show reduced total sleep time and efficiency, diminished slow-wave sleep, and abnormal spindle density—changes linked to cognitive impairment and positive symptom severity (Kaskie & Ferrarelli, 2020; Manoach & Stickgold, 2019). An umbrella review confirmed that individuals with SCZ have nearly twice the odds of clinically significant sleep disturbance compared to the general population (Azargoonjahromi & Bahrampour, 2025). In a large naturalistic cohort, sleep disorders were present in over 70% of psychiatric outpatients with psychotic symptoms and were associated with higher psychopathology and lower well-being (Mijnster et al., 2024). Interestingly, insomnia severity in schizophrenia correlates more strongly with negative and cognitive symptoms than with positive ones, suggesting distinct underlying mechanisms (Carruthers et al., 2021). Such disturbances have also been reported among first-degree relatives, suggesting a heritable vulnerability (Laskemoen et al., 2019).

In post-traumatic stress disorder, sleep disturbances are nearly universal and constitute part of the diagnostic criteria. Between 80% and 90% of individuals with PTSD report chronic insomnia, while 50–70% experience recurrent nightmares or dysphoric dreams (Mendoza Alvarez et al., 2025; Messman et al., 2023; Weber & Wetter, 2022). These abnormalities are closely tied to trauma re-experiencing, heightened arousal, and emotional dysregulation (Agorastos & Olff, 2021). Quantitative studies show that nightmare frequency predicts PTSD severity, daytime hyperarousal, and comorbid depression (Mendoza Alvarez et al., 2025; Messman et al., 2023). Polysomnography demonstrates increased REM density, REM fragmentation, and elevated nocturnal heart rate—patterns that differentiate PTSD from other anxiety or mood disorders (Weber & Wetter, 2022). A meta-analysis by Lappas et al. (2024) found that comorbid insomnia in PTSD is associated with a twofold increase in depressive and anxiety symptom severity, highlighting the bidirectional link between sleep and psychopathology.

Comparative data across disorders emphasize the transdiagnostic nature of sleep disturbances. Insomnia occurs in similar proportions across MDD, BD, and SCZ (60–80%), with slightly higher prevalence in PTSD (up to 90%) (Carruthers et al., 2021; Tonon et al., 2024; Weber & Wetter, 2022; Yasugaki et al., 2025). Nightmare frequency is greatest in PTSD but notable in BD and MDD, where dysphoric dreams relate to emotion regulation deficits (Fang et al., 2019; Mendoza Alvarez et al., 2025). Circadian misalignment—characterized by delayed melatonin onset, blunted rest–activity amplitude, and irregular sleep timing—occurs across diagnostic boundaries (Kawai et al., 2023; Tonon et al., 2024; Zou et al., 2022). Such disruptions appear even in unmedicated or remitted patients, suggesting they represent enduring trait markers rather than medication effects (Laskemoen et al., 2019; Marchetti et al., 2025). Cross-cultural variability is also evident, with prevalence rates ranging from 60–80% in European samples to over 85% in South American cohorts (Mijnster et al., 2024; Tonon et al., 2024).

Sleep abnormalities also correlate with illness course and prognosis. Longitudinal evidence shows that persistent insomnia predicts relapse in MDD and BD (Kragh et al., 2024; Scott et al., 2021), while greater sleep variability predicts suicidality and cognitive decline in BD and SCZ (Carruthers et al., 2021; Pearson et al., 2023). In PTSD, nightmare frequency and nocturnal awakenings are among the strongest predictors of treatment nonresponse (Messman et al., 2023; Weber & Wetter, 2022). Collectively, these findings suggest that disturbed sleep is not merely a symptom dimension but a risk and maintenance factor for psychiatric morbidity across diagnostic categories.

Comorbidity with primary sleep disorders, particularly obstructive sleep apnea (OSA), further compounds psychiatric symptomatology. Prevalence estimates indicate that OSA co-occurs in 15–30% of patients with depression or psychosis and contributes to cognitive impairment and poor mood regulation (Kerner & Roose, 2016; Zielinski & Gibbons, 2022). Emerging evidence links sleep-disordered breathing to systemic inflammation and metabolic dysregulation, which may amplify neuroinflammatory pathways and worsen mental health outcomes (Liu et al., 2023; Zielinski & Gibbons, 2022).

Overall, literature from 2013 to 2025 provides compelling evidence that sleep disturbances are highly prevalent and transdiagnostic, affecting most individuals with major psychiatric disorders. Quantitatively, insomnia rates exceed 60% across all diagnostic groups, hypersomnia occurs in up to 40% of depressive or bipolar episodes, and circadian rhythm disruption affects at least half of all patients. These findings underscore that sleep pathology constitutes a core clinical feature warranting systematic assessment and targeted intervention in psychiatric populations.

Neurobiological and Psychological Mechanisms

The neurobiological architecture underlying sleep disturbances in major psychiatric disorders reflects a complex interplay between neurotransmitter systems, neuroendocrine regulation, circadian timing, and affective processing. Across diagnostic categories, converging evidence suggests that disrupted homeostatic and circadian sleep regulation contributes both to the onset and maintenance of psychiatric symptoms (Borbély, 1982; Harvey, 2008; Walker & van der Helm, 2009).

Neurotransmitter and Neuroendocrine Dysregulation

The balance of monoaminergic transmission—particularly serotonin, norepinephrine, and dopamine—is central to the bidirectional relationship between sleep and mood regulation. In MDD and BD, alterations in serotonergic signaling within the dorsal raphe nucleus are linked to shortened REM latency and increased REM density (Pandi-Perumal et al., 2020; Yasugaki et al., 2025). Elevated nocturnal cortisol and impaired suppression of the hypothalamic–pituitary–adrenal (HPA) axis have been repeatedly documented in depressed and manic patients, suggesting chronic hyperarousal and stress-system sensitization (Fang et al., 2019; Marchetti et al., 2025).

The orexin (hypocretin) system—critical for wakefulness regulation—has also emerged as a transdiagnostic mechanism. Reduced plasma orexin-A levels have been observed in schizophrenia, correlating with negative symptom severity and sleep fragmentation (Li et al., 2022). Conversely, increased orexin activity contributes to hyperarousal and insomnia in PTSD and mania (Han et al., 2020; Kishi et al., 2024). Pharmacological evidence supports this link: dual orexin receptor antagonists (DORAs) improve both sleep efficiency and mood stability in psychiatric samples, underscoring the system's integrative role between arousal and emotion regulation (Han et al., 2020; Kishi et al., 2024).

Neuroinflammatory mechanisms further bridge disturbed sleep and psychopathology. Elevated pro-inflammatory cytokines—interleukin-6 (IL-6), tumor necrosis factor α (TNF-α), and C-reactive protein

(CRP)—have been consistently associated with insomnia and fatigue in mood and psychotic disorders (Liu et al., 2023; Zielinski & Gibbons, 2022). Sleep deprivation induces microglial activation and oxidative stress, which may exacerbate synaptic pruning abnormalities observed in schizophrenia and depression (Zielinski & Gibbons, 2022). These findings suggest a self-perpetuating cycle wherein inflammation disrupts sleep, and sleep loss in turn amplifies neuroinflammation. Clinically, elevated CRP and IL-6 levels have been correlated with greater fatigue and anhedonia severity in depression and schizophrenia (Liu et al., 2023).

Circadian Rhythm Disruption

Disturbances in the molecular circadian clock—regulated by transcriptional feedback loops of *CLOCK*, *BMAL1*, *PER*, and *CRY* genes—are now recognized as central to the pathophysiology of affective and psychotic disorders (Harvey, 2008; Zou et al., 2022). Polymorphisms in *CLOCK* and *PER3* genes have been associated with increased vulnerability to affective episodes and sleep–wake instability (Zou et al., 2022). Patients with BD exhibit delayed dim-light melatonin onset and blunted amplitude of circadian temperature rhythms (Tonon et al., 2024). Altered melatonin secretion rhythms have been observed across disorders, reflecting both circadian misalignment and pineal dysregulation (Moon et al., 2022). Actigraphy studies demonstrate phase delays of 1–2 hours relative to controls, with the degree of misalignment predicting manic symptom severity (Marchetti et al., 2025; Tonon et al., 2024). In MDD, flattened diurnal cortisol rhythms and decreased amplitude of activity–rest cycles are observed, paralleling anhedonia and psychomotor retardation (Fang et al., 2019; Yasugaki et al., 2025).

In schizophrenia, circadian rhythm fragmentation is profound: up to 70% of patients display irregular sleep–wake patterns independent of antipsychotic medication (Carruthers et al., 2021; Laskemoen et al., 2019). Polysomnography and actigraphy indicate reduced amplitude of 24-hour rest–activity cycles, and single-channel EEG recordings confirm misalignment between subjective and objective sleep timing (Kawai et al., 2023). Circadian dysregulation is thought to impair cognitive synchronization between cortical networks, contributing to deficits in attention and working memory (Carruthers et al., 2021; Pearson et al., 2023).

PTSD displays a distinct pattern of REM sleep instability and autonomic hyperarousal. Increased REM density and phasic activity are accompanied by higher nocturnal heart rate and reduced heart-rate variability, indicating persistent sympathetic activation (Weber & Wetter, 2022). REM fragmentation correlates with nightmare frequency and emotional dysregulation (Mendoza Alvarez et al., 2025; Messman et al., 2023). Neuroimaging studies demonstrate amygdala hyperreactivity during REM sleep and attenuated medial prefrontal inhibition, mirroring waking neural patterns of trauma recall (Ressler et al., 2022).

Sleep Microarchitecture and Cognitive Mechanisms

Alterations in sleep spindles, slow-wave activity, and REM density provide objective markers linking sleep physiology to cognitive and emotional processes. In schizophrenia, spindle density reductions of up to **40%** have been documented, correlating with working-memory impairments and decreased hippocampal–thalamic connectivity (Kaskie & Ferrarelli, 2020; Manoach & Stickgold, 2019). Similar reductions, though milder, are reported in bipolar depression, suggesting shared thalamocortical dysregulation (Marchetti et al., 2025; Tonon et al., 2024).

Slow-wave sleep (SWS) deficits, reflecting impaired cortical synchronization, are present across MDD, BD, and SCZ. Reduced SWS amplitude predicts poor memory consolidation and emotion-regulation difficulties (Pearson et al., 2023; Walker & van der Helm, 2009). Experimental sleep-restriction paradigms show that one night of SWS deprivation leads to a **30–40%** reduction in next-day emotional discrimination accuracy, highlighting the functional cost of disrupted restorative sleep (Walker & van der Helm, 2009).

Gut–brain-axis research has recently introduced microbial metabolites as potential mediators of sleep–mood interactions. Depressed individuals show decreased short-chain-fatty-acid-producing bacteria and elevated kynurenine-pathway metabolites, which influence serotonin synthesis and circadian gene expression (Liu et al., 2023). These findings link metabolic homeostasis, immune signaling, and central neurotransmission within an integrated sleep–psychiatry framework.

Psychological and Cognitive-Emotional Models

Beyond neurobiological mechanisms, cognitive and emotional factors further shape sleep pathology across disorders.

From a psychological perspective, cognitive-behavioral models emphasize the role of dysfunctional beliefs and maladaptive sleep habits in perpetuating insomnia. Catastrophic thinking about sleep loss, pre-sleep rumination, and heightened threat monitoring sustain cortical arousal and interfere with sleep initiation (Harvey, 2008; Hertenstein et al., 2022). In depression and PTSD, selective attention to internal cues of fatigue or hyperarousal reinforces avoidance behavior and circadian irregularity (Agorastos & Olff, 2021; Weber & Wetter, 2022).

Emotion-regulation theory provides an additional explanatory layer. REM sleep facilitates overnight extinction of emotional reactivity; its disruption leads to impaired fear extinction and mood instability (Walker & van der Helm, 2009). Dysphoric dreams and nightmares represent maladaptive emotional memory processing, particularly in PTSD and BD (Mendoza Alvarez et al., 2025). Across disorders, deficient top-down control from the medial prefrontal cortex over limbic structures contributes to persistent hyperarousal and negative affect (Ressler et al., 2022; Weber & Wetter, 2022).

The two-process model of sleep regulation (Borbély, 1982) remains a unifying conceptual framework: the interaction between homeostatic sleep pressure (Process S) and circadian timing (Process C) is disrupted across psychiatric disorders. In MDD, excessive Process S coupled with phase-delayed Process C results in early-morning awakening, whereas in BD, weakened Process S during mania leads to reduced sleep need. Schizophrenia exhibits desynchronization of both processes, manifesting as irregular sleep–wake cycles (Carruthers et al., 2021; Laskemoen et al., 2019).

Collectively, these neurobiological and psychological findings demonstrate that sleep disturbances in psychiatric disorders stem from multifactorial mechanisms involving neurotransmitter imbalance, neuroinflammation, circadian misalignment, and maladaptive cognitive–emotional regulation. Rather than mere epiphenomena, these processes interact dynamically to sustain symptom chronicity and functional impairment across diagnostic boundaries.

Therapeutic Approaches and Clinical Implications

The recognition that sleep disturbances represent both symptoms and maintaining factors of psychiatric disorders has shifted treatment paradigms toward integrated, transdiagnostic approaches. Interventions now aim not only to relieve sleep complaints but also to normalize circadian functioning and reduce psychopathology. Evidence from pharmacological, psychological, and neurostimulation studies highlights the bidirectional therapeutic relationship between sleep restoration and mental health recovery.

Pharmacological Interventions

Traditional sedative-hypnotics remain widely used in psychiatric settings, yet recent network meta-analyses emphasize differential efficacy and safety profiles across agents. A large-scale review of 154 randomized trials found that benzodiazepines and "Z-drugs" provide short-term efficacy for insomnia but carry high risks of tolerance and cognitive impairment (De Crescenzo et al., 2022). In contrast, newer compounds targeting specific neurobiological pathways show more favorable outcomes.

Dual orexin receptor antagonists (DORAs)—including suvorexant, lemborexant, and daridorexant—demonstrate consistent improvements in sleep onset latency and total sleep time across psychiatric populations, with effect sizes comparable to cognitive-behavioral therapy for insomnia (Kishi et al., 2024). In comorbid depression, DORA treatment yields moderate improvements in mood symptoms, supporting the integrative role of the orexin system in arousal and affect regulation (Han et al., 2020). Head-to-head comparisons suggest that while DORAs yield comparable short-term improvements in sleep efficiency, CBT-I produces more durable gains and broader effects on mood and cognition (Hertenstein et al., 2022; Kishi et al., 2024). Follow-up studies indicate maintenance of sleep improvements up **to 24 months** post-treatment (Hertenstein et al., 2022).

Melatonergic agents represent another major advance. Controlled studies show that prolonged-release melatonin and the melatonergic antidepressant agomelatine improve both circadian rhythm stability and depressive symptomatology (Del Casale et al., 2024; Moon et al., 2022). These agents enhance sleep efficiency without altering sleep architecture or inducing dependence, making them suitable for long-term management.

Agomelatine, in particular, strengthens slow-wave sleep and increases circadian amplitude, with benefits extending to cognitive and emotional domains (Moon et al., 2022).

For **PTSD**, pharmacotherapy targeting adrenergic hyperarousal and REM dysregulation has shown promise. Prazosin—a central α1-adrenergic antagonist—reduces nightmare frequency and improves overall sleep quality (Lappas et al., 2024; Weber & Wetter, 2022). However, efficacy appears heterogeneous, and combination approaches are often required.

Antidepressant and antipsychotic medications exert complex effects on sleep. While agents with antihistaminergic or serotonergic properties (e.g., mirtazapine, quetiapine) may improve sleep continuity, dopaminergic drugs can disrupt sleep architecture. The choice of pharmacotherapy should therefore balance sleep outcomes with psychiatric symptom control (Berk et al., 2025; Geddes & Miklowitz, 2013).

Psychological and Behavioral Therapies

CBT-I remains the gold standard nonpharmacological intervention and demonstrates broad efficacy across psychiatric populations. A meta-analysis of 65 trials found that CBT-I produced large effect sizes for sleep efficiency (Hedges g = 0.86) and moderate-to-large reductions in depression and anxiety severity (Hertenstein et al., 2022). Importantly, CBT-I remains effective when delivered digitally or transdiagnostically, improving accessibility for individuals with complex psychiatric comorbidities (Kragh et al., 2024).

The therapeutic mechanisms of CBT-I extend beyond sleep normalization. Improvements in emotion regulation, cognitive flexibility, and diurnal activity patterns suggest that behavioral sleep interventions modulate core neurobiological systems implicated in psychiatric illness (Mirchandaney et al., 2022). Notably, residual insomnia after depression treatment predicts relapse, and adjunctive CBT-I reduces recurrence risk by approximately 50% over 12 months (Scott et al., 2021).

For PTSD, trauma-focused CBT combined with targeted sleep interventions yields the strongest outcomes. Imagery rehearsal therapy and exposure-based protocols reduce nightmare frequency and enhance REM stability (Agorastos & Olff, 2021; Messman et al., 2023). Addressing sleep disturbances early in PTSD treatment accelerates emotional processing and reduces hyperarousal symptoms (Weber & Wetter, 2022). Integrating trauma-focused psychotherapy with appropriate pharmacological treatment has been shown to significantly reduce PTSD symptom severity and simultaneously improve co-occurring depression, anxiety, and sleep disturbances (Coventry et al., 2020).

The efficacy of CBT-I in bipolar disorder requires careful consideration of circadian sensitivity. Chronobiologically adapted protocols such as Interpersonal and Social Rhythm Therapy (IPSRT) emphasize regularization of daily routines and light exposure timing to stabilize mood and sleep (Harvey, 2008; Tonon et al., 2024). Randomized trials show that IPSRT reduces relapse rates and improves subjective sleep quality, particularly in younger and early-course patients (Tonon et al., 2024).

Chronobiological and Neurostimulation Approaches

Chronotherapeutic interventions—including light therapy, sleep deprivation, and phase-advance protocols—target circadian misalignment directly. Bright light therapy improves depressive symptoms and re-entrains melatonin secretion rhythms, particularly in seasonal and bipolar depression (Tonon et al., 2024; Yasugaki et al., 2025). Combined light and wake therapy induces rapid antidepressant effects within 24 to 48 hours, though maintenance requires concurrent mood-stabilizing medication (Harvey, 2008).

Noninvasive brain stimulation (NIBS) has recently emerged as a promising tool for modulating neural circuits underlying sleep and affect regulation. Repetitive transcranial magnetic stimulation (rTMS) targeting dorsolateral prefrontal regions enhances slow-wave activity and improves insomnia in depression and schizophrenia (Stefanowski et al., 2025). Similarly, transcranial direct current stimulation (tDCS) produces moderate benefits for sleep onset latency and perceived restfulness, particularly when combined with behavioral interventions (Stefanowski et al., 2025). These effects are thought to arise from enhanced cortical plasticity and strengthened thalamo-cortical connectivity, paralleling improvements in slow-wave dynamics and emotion regulation (Stefanowski et al., 2025).

Emerging evidence also supports transdiagnostic digital phenotyping to monitor and personalize sleep interventions. Actigraphy and wearable devices capture rest–activity rhythms and light exposure patterns, enabling feedback-driven regulation of circadian stability (Kawai et al., 2023; Tonon et al., 2024). Integration of digital sleep data with clinical measures of mood and cognition may enhance predictive precision for relapse or treatment response (Mijnster et al., 2024).

Clinical and Public Health Implications

Across psychiatric populations, addressing sleep disturbances yields substantial improvements in global functioning, quality of life, and relapse prevention. Restoring sleep continuity enhances emotion regulation, cognitive performance, and therapeutic engagement. Clinically, sleep-targeted interventions should be integrated into standard psychiatric care rather than treated as secondary concerns.

At the population level, systematic screening for insomnia, hypersomnia, and circadian rhythm disorders could improve early identification of psychiatric risk. Cross-sectional data indicate that untreated insomnia increases the odds of developing a mood or anxiety disorder by nearly twofold (Scott et al., 2021). Incorporating validated instruments such as the Insomnia Severity Index (ISI) or the Pittsburgh Sleep Quality Index (PSQI) into psychiatric assessment protocols may facilitate timely intervention and reduce chronicity. Integrating sleep interventions into routine care has also proven cost-effective through reduced relapse rates and healthcare utilization (Scott et al., 2021).

From a transdiagnostic perspective, the convergence of biological, psychological, and behavioral evidence underscores sleep as a modifiable mechanism in mental illness. Effective treatment of sleep pathology not only alleviates fatigue and cognitive impairment but also attenuates core psychiatric symptoms, supporting the conceptualization of sleep as both a therapeutic target and a biomarker of recovery. Future directions emphasize precision-based, transdiagnostic models that integrate circadian, behavioral, and digital biomarkers to personalize treatment trajectories (Kawai et al., 2023; Tonon et al., 2024).

Summary of Results

Across major psychiatric disorders, sleep disturbances emerge as highly prevalent, affecting more than 60% of patients with depression, bipolar disorder, schizophrenia, and post-traumatic stress disorder. These abnormalities—spanning insomnia, hypersomnia, and circadian misalignment—function as both symptomatic expressions and risk factors for illness persistence. Neurobiological evidence highlights dysregulation in monoaminergic and orexinergic pathways, HPA-axis hyperactivity, neuroinflammatory processes, and clock gene alterations. Psychological findings further implicate cognitive–emotional dysregulation and maladaptive sleep beliefs. Together, these mechanisms reinforce chronic hyperarousal and affective instability. Therapeutic findings indicate that cognitive-behavioral and chronotherapeutic interventions produce robust and enduring improvements, complemented by pharmacological and neuromodulatory advances such as DORAs, melatonergic agents, and NIBS. Collectively, the results establish sleep as a transdiagnostic domain bridging neurobiology and psychopathology, positioning it as both a mechanistic target and a clinical biomarker within precision psychiatry.

Discussion

Integrative Interpretation of Findings

The findings of this review highlight sleep disturbances as pervasive and transdiagnostic phenomena that both shape and reflect major psychiatric psychopathology. Across depression, bipolar disorder, schizophrenia spectrum disorders, and post-traumatic stress disorder, disrupted sleep emerges not merely as an epiphenomenon but as a mechanistic contributor to emotional dysregulation, cognitive dysfunction, and relapse vulnerability (Marchetti et al., 2025; Weber & Wetter, 2022; Yasugaki et al., 2025). This integrated perspective aligns with contemporary models of psychiatric illness that emphasize bidirectional brain–behavior relationships and supports the conceptualization of sleep as a core dimension of mental health (Crouse et al., 2021; Grandner & Fernandez, 2021).

Epidemiological data underscore the ubiquity of sleep abnormalities across psychiatric populations, with prevalence rates exceeding 60% for insomnia, hypersomnia, or circadian rhythm disruption (Laskemoen et al., 2019; Scott et al., 2021). These findings challenge disorder-specific frameworks by demonstrating that sleep pathology transcends diagnostic boundaries, functioning as a shared vulnerability factor across affective, psychotic, and trauma-related conditions (Mendoza Alvarez et al., 2025; Tonon et al., 2024). This transdiagnostic lens echoes the RDoC approach, which positions sleep–wake regulation within the broader construct of arousal and regulatory systems—domains underlying multiple psychiatric phenotypes (Nutt et al., 2008; Walker & van der Helm, 2009).

At the mechanistic level, converging neurobiological evidence implicates overlapping disturbances across neurotransmitter systems, neuroendocrine axes, and neural network connectivity. Dysregulation of monoaminergic and orexinergic pathways disrupts the balance between arousal and sleep-promoting systems, contributing to fragmented sleep and emotional instability (Han et al., 2020; Kishi et al., 2024). HPA-axis

hyperactivity, observed in depression and PTSD, reinforces chronic hyperarousal and impairs slow-wave sleep (Lappas et al., 2024; Weber & Wetter, 2022). Similarly, neuroinflammatory processes and altered clock gene expression link circadian misalignment to affective and cognitive dysregulation (Liu et al., 2023; Zielinski & Gibbons, 2022). These findings suggest that sleep and psychiatric pathology share common biological substrates, supporting the bidirectionality proposed in neurocircuit and allostatic models (Fang et al., 2019; Marchetti et al., 2025). Neuroimaging studies further corroborate these associations, showing overlapping alterations in thalamo-cortical and limbic circuits involved in both emotional regulation and sleep–wake control (Manoach & Stickgold, 2019; Marchetti et al., 2025).

Beyond the neurobiological dimension, psychological and behavioral mechanisms also play a critical role in the persistence of sleep disturbances. Maladaptive cognitive patterns—such as worry about sleep, hypervigilance, and conditioned arousal—maintain insomnia even after mood symptoms improve (Mirchandaney et al., 2022). Emotional dysregulation, a hallmark of mood and trauma-related disorders, further amplifies sleep fragmentation and nightmare frequency (Messman et al., 2023). Cognitive–emotional mechanisms interact with biological vulnerabilities to produce feedback loops that sustain chronic sleep dysfunction, aligning with Harvey's (2008) integrative model of circadian and cognitive control of mood. Collectively, these processes suggest that targeting both biological and cognitive components is necessary for effective intervention.

Importantly, the current synthesis reinforces the notion that sleep disturbances are not uniform across psychiatric diagnoses but instead reflect disorder-specific profiles. In mood disorders, insomnia and altered REM patterns mark depressive states, whereas bipolar disorder shows circadian instability with hypersomnia in depression and reduced sleep need during mania (Pandi-Perumal et al., 2020; Tonon et al., 2024). Schizophrenia spectrum disorders exhibit profound alterations in slow-wave activity and spindle density, reflecting thalamo-cortical dysconnectivity and impaired memory consolidation (Kaskie & Ferrarelli, 2020; Manoach & Stickgold, 2019). Meanwhile, PTSD features trauma-specific nightmares, REM intrusion, and elevated nocturnal sympathetic activity (Lappas et al., 2024; Weber & Wetter, 2022). Recognizing these disorder-specific sleep phenotypes refines both diagnosis and treatment selection.

From a clinical perspective, these findings underscore the reciprocal influence between sleep and psychiatric symptomatology. Longitudinal studies indicate that sleep disturbances predict the onset, severity, and recurrence of psychiatric disorders (Scott et al., 2021). For instance, insomnia doubles the risk of developing depression or anxiety, while normalization of sleep architecture predicts sustained remission (Yasugaki et al., 2025). In bipolar disorder, stabilization of sleep–wake cycles via social rhythm therapy reduces relapse rates and improves functional outcomes (Tonon et al., 2024). Similarly, early intervention targeting sleep disruption in PTSD accelerates recovery and diminishes emotional reactivity (Agorastos & Olff, 2021; Weber & Wetter, 2022). Collectively, these findings position sleep as both a prognostic marker and a therapeutic gateway across psychiatric illnesses.

The integration of pharmacological and psychological evidence further highlights sleep as a shared treatment target. CBT-I consistently produces robust improvements in sleep efficiency and secondary reductions in depression and anxiety severity (Hertenstein et al., 2022). Complementary pharmacological approaches, including DORAs and melatonergic agents, enhance circadian stability and mood regulation (Kishi et al., 2024; Moon et al., 2022). NIBS extends these findings by demonstrating improvements in both sleep architecture and affective regulation through modulation of fronto-limbic connectivity (Stefanowski et al., 2025). The convergence of these modalities reflects a paradigm shift toward integrative, multimodal treatment, aligning with emerging transdiagnostic care frameworks (Berk et al., 2025; Kragh et al., 2024).

Taken together, the evidence consolidates a coherent model in which sleep disturbances function as both biomarkers and mechanisms of psychopathology. Rather than isolated symptoms, they represent dynamic processes that influence emotional and cognitive functioning, neuroplasticity, and treatment response. This reconceptualization holds significant implications for the classification and management of mental disorders, suggesting that sleep-focused assessment and intervention could enhance diagnostic precision and therapeutic efficacy. The following sections expand upon the clinical implications, methodological considerations, and future directions essential to advancing sleep-informed psychiatry.

Clinical Implications and Translational Relevance

The evidence synthesized in this review underscores sleep as a clinically actionable dimension of psychiatric care. Across diagnostic categories, sleep disturbances not only exacerbate mood, psychotic, and trauma-related symptoms but also predict illness onset and recurrence. Routine screening for insomnia, hypersomnia, and circadian misalignment should therefore be considered a standard component of psychiatric assessment (Scott et al., 2021). Early detection enables targeted interventions that may prevent chronicity and functional decline, reflecting a shift toward preventive psychiatry (Tonon et al., 2024).

From a therapeutic perspective, the findings highlight the importance of individualized, multimodal treatment strategies. CBT-I remains the most evidence-based intervention, yielding long-term remission rates that rival or exceed pharmacological outcomes (Hertenstein et al., 2022). Integrating CBT-I within broader treatment frameworks—such as IPSRT or trauma-focused CBT—enhances not only sleep quality but also emotion regulation and relapse prevention (Agorastos & Olff, 2021; Harvey, 2008). Melatonin and its prolonged-release formulations further stabilize circadian rhythms and alleviate depressive and anxiety symptoms without cognitive side effects (Del Casale et al., 2024; Moon et al., 2022). These findings support the inclusion of chronotherapeutic and melatonergic agents as adjunctive tools in complex psychiatric cases.

Emerging translational research demonstrates that sleep and circadian rhythm parameters can inform personalized treatment planning. Individual differences in chronotype modulate vulnerability to psychiatric disorders and influence response to light therapy and medication timing (Zou et al., 2022). Assessing chronotype and circadian preference may therefore help clinicians tailor behavioral and pharmacological strategies, aligning with the principles of precision psychiatry (Grandner & Fernandez, 2021).

The broader clinical implications extend beyond symptom reduction. Addressing sleep dysfunction enhances cognitive performance, daytime functioning, and quality of life. Chronic sleep fragmentation has been associated with impaired memory, executive deficits, and emotional blunting—features that overlap with psychiatric symptomatology (Kerner & Roose, 2016). By improving sleep architecture, interventions such as CBT-I, melatonergic agents, and NIBS indirectly restore neurocognitive efficiency and social engagement, which are critical determinants of recovery.

At the systems level, incorporating sleep-focused interventions into routine psychiatric care is both clinically effective and cost-efficient. Reductions in relapse rates, hospitalizations, and healthcare utilization have been observed when insomnia and circadian rhythm disorders are systematically addressed alongside core psychiatric symptoms (Scott et al., 2021). Integrating sleep-focused assessments into psychiatric guidelines and training programs could accelerate the translation of these findings into everyday practice. Sleep thus represents a high-yield target for transdiagnostic treatment optimization—an area where psychiatry and behavioral sleep medicine converge.

Taken together, these clinical and translational insights emphasize that treating sleep disturbances is not ancillary but foundational to improving psychiatric outcomes. Integrating circadian assessments, chronotype profiling, and sleep-specific therapies into standard practice holds promise for more precise, preventive, and person-centered mental healthcare.

Limitations and Future Directions

Despite its comprehensive scope, this review has several limitations. The included literature spans a wide temporal range, during which diagnostic frameworks, methodological standards, and therapeutic paradigms evolved considerably. Variations in sample size, diagnostic criteria, and assessment tools for sleep—ranging from self-reports to polysomnography—introduce heterogeneity that complicates direct comparison across studies (Kawai et al., 2023). Although the present synthesis prioritized high-quality systematic reviews and meta-analyses, publication bias and underrepresentation of negative findings remain possible sources of distortion.

A second limitation concerns the transdiagnostic approach itself. While this framework allows for integration across disorders, it may obscure disorder-specific nuances such as phase-dependent patterns in bipolar disorder or REM-related abnormalities in depression (Marchetti et al., 2025; Pandi-Perumal et al., 2020). Moreover, most studies remain cross-sectional, limiting causal inference regarding the bidirectional relationship between sleep disturbances and psychopathology. Longitudinal and experimental designs are needed to clarify whether specific sleep abnormalities act as triggers, consequences, or maintaining factors of mental illness (Scott et al., 2021).

Another challenge lies in the limited neurobiological granularity of current evidence. Although findings implicate dysregulation in monoaminergic, orexinergic, and inflammatory pathways, most data remain indirect

and peripheral. Future research should prioritize multimodal approaches combining neuroimaging, molecular, and chronobiological data to identify reliable mechanistic markers. Integrating sleep-specific neuroimaging paradigms with measures of affective and cognitive processing could elucidate shared circuit-level mechanisms across psychiatric diagnoses (Manoach & Stickgold, 2019; Zielinski & Gibbons, 2022).

From a translational standpoint, research on personalized interventions remains limited. The clinical relevance of chronotype, circadian preference, and genetic polymorphisms in clock genes is promising but underexplored (Zou et al., 2022). Advances in digital monitoring—through actigraphy, ecological momentary assessment, and wearable technology—offer new opportunities for real-time, individualized treatment feedback (Kawai et al., 2023). However, such technologies require rigorous validation and standardization before being integrated into psychiatric practice.

Future studies should also address population diversity and contextual factors. Most evidence derives from high-income, Western populations, limiting generalizability to culturally diverse groups where sleep behaviors, light exposure, and stress patterns differ substantially (Agorastos & Olff, 2021). Including underrepresented populations and adopting ecological frameworks will be essential for building globally relevant models of sleep–psychopathology interaction.

Clinically, the next step involves translating mechanistic insights into routine diagnostic and therapeutic algorithms. Large-scale trials should evaluate multimodal interventions that combine CBT-I, chronotherapy, melatonergic agents, and neuromodulatory techniques such as NIBS (Stefanowski et al., 2025). Health systems research is likewise needed to determine the cost-effectiveness and scalability of integrated sleep care in psychiatric settings.

While substantial progress has been made in understanding the prevalence, mechanisms, and treatment of sleep disturbances in major psychiatric disorders, significant gaps remain. Bridging these gaps will require longitudinal, mechanistic, and cross-cultural studies that link laboratory findings with clinical outcomes. Progress in this field will depend on interdisciplinary collaboration among psychiatry, neuroscience, chronobiology, and data science. Integrating sleep into the core of psychiatric research and practice offers a pathway toward more mechanistically informed, personalized, and preventive mental healthcare.

Conclusions

Sleep disturbances are a pervasive, transdiagnostic feature of major psychiatric disorders, shaping their onset, course, and treatment outcomes. This review has synthesized evidence showing that abnormalities in sleep architecture and circadian regulation are not merely secondary symptoms but active mechanisms contributing to affective instability, cognitive dysfunction, and relapse vulnerability. Across depression, bipolar disorder, schizophrenia, and post-traumatic stress disorder, disruptions in monoaminergic, orexinergic, and inflammatory systems combine with cognitive–emotional dysregulation to produce chronic hyperarousal and maladaptive sleep–wake cycles.

Clinically, recognizing sleep as both a symptom and a mechanistic driver reframes psychiatric assessment and treatment. Interventions such as cognitive behavioral therapy for insomnia, chronotherapy, melatonergic agents, and NIBS have demonstrated robust and enduring benefits, underscoring the need to integrate sleep-focused care into psychiatric guidelines. Personalized strategies that incorporate chronotype, circadian rhythm, and digital monitoring may further enhance therapeutic precision.

Future progress will depend on longitudinal, multimodal, and cross-cultural research linking neurobiological, behavioral, and environmental determinants of sleep to clinical outcomes. Ultimately, integrating sleep into the core framework of psychiatry offers a powerful and necessary avenue toward more mechanistically informed, preventive, and patient-centered mental healthcare.

Author's Contributions

Conceptualization: Anna Maria Gęsińska
Methodology: Bartosz Golis, Anna Maria Gęsińska
Software: Helena Szelka, Anna Maria Gęsińska
Check: Krystian Czyżykowski, Anna Maria Gęsińska
Validation / Check: Zuzanna Przybyła, Anna Maria Gęsińska
Formal Analysis: Katarzyna Andrzejewska, Anna Maria Gęsińska
Investigation: Paweł Edyko, Anna Maria Gęsińska
Resources: Wiktor Golus, Anna Maria Gęsińska
Data curation: Alicja Babula, Anna Maria Gęsińska
Writing – rough preparation: Hubert Woźniak, Anna Maria Gęsińska
Writing – Review and Editing: Krystian Czyżykowski, Anna Maria Gęsińska
Supervision / Project Administration: Krystian Czyżykowski, Anna Maria Gęsińska

All authors have read and agreed to the published version of the manuscript.

Funding Statement: The author received no external funding for this work.

Institutional Review Board Statement: Not applicable; this review included only published data.

Data Availability Statement: All supporting data are available within the cited peer-reviewed literature.

Acknowledgments: The author acknowledges the contribution of investigators and data curators whose high-quality research underpins the advances reviewed herein.

Conflict of Interest Statement: The author declares no conflict of interest.

Declaration of the use of generative AI and AI-assisted technologies in the writing process: In preparing this work, the authors used ChatGPT for the purpose of improving language and readability. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

REFERENCES

1. Agorastos, A., & Olff, M. (2021). Sleep, circadian system and traumatic stress. European journal of psychotraumatology, 12(1), 1956746. https://doi.org/10.1080/20008198.2021.1956746
2. Azargoonjahromi, A., & Bahrampour, Z. (2025). Sleep disturbances are associated with psychosis in schizophrenia spectrum Disorders: An umbrella review. Sleep medicine, 134, 106690. https://doi.org/10.1016/j.sleep.2025.106690
3. Berk, M., Corrales, A., Trisno, R., Dodd, S., Yatham, L. N., Vieta, E., McIntyre, R. S., Suppes, T., & Agustini, B. (2025). Bipolar II disorder: a state-of-the-art review. World psychiatry : official journal of the World Psychiatric Association (WPA), 24(2), 175–189. https://doi.org/10.1002/wps.21300
4. Borbély A. A. (1982). A two process model of sleep regulation. Human neurobiology, 1(3), 195–204.
5. Carruthers, S. P., Brunetti, G., & Rossell, S. L. (2021). Sleep disturbances and cognitive impairment in schizophrenia spectrum disorders: a systematic review and narrative synthesis. Sleep medicine, 84, 8–19. https://doi.org/10.1016/j.sleep.2021.05.011
6. Coventry, P. A., Meader, N., Melton, H., Temple, M., Dale, H., Wright, K., Cloitre, M., Karatzias, T., Bisson, J., Roberts, N. P., Brown, J. V. E., Barbui, C., Churchill, R., Lovell, K., McMillan, D., & Gilbody, S. (2020). Psychological and pharmacological interventions for posttraumatic stress disorder and comorbid mental health problems following complex traumatic events: Systematic review and component network meta-analysis. PLoS medicine, 17(8), e1003262. https://doi.org/10.1371/journal.pmed.1003262
7. Crouse, J. J., Carpenter, J. S., Song, Y. J. C., Hockey, S. J., Naismith, S. L., Grunstein, R. R., Scott, E. M., Merikangas, K. R., Scott, J., & Hickie, I. B. (2021). Circadian rhythm sleep-wake disturbances and depression in young people: implications for prevention and early intervention. The lancet. Psychiatry, 8(9), 813–823. https://doi.org/10.1016/S2215-0366(21)00034-1
8. De Crescenzo, F., D'Alò, G. L., Ostinelli, E. G., Ciabattini, M., Di Franco, V., Watanabe, N., Kurtulmus, A., Tomlinson, A., Mitrova, Z., Foti, F., Del Giovane, C., Quested, D. J., Cowen, P. J., Barbui, C., Amato, L., Efthimiou, O., & Cipriani, A. (2022). Comparative effects of pharmacological interventions for the acute and long-term management of insomnia disorder in adults: a systematic review and network meta-analysis. Lancet (London, England), 400(10347), 170–184. https://doi.org/10.1016/S0140-6736(22)00878-9
9. Del Casale, A., Arena, J. F., Giannetti, F., Minervino, A., Biggio, G., & Girardi, P. (2024). The use of prolonged-release melatonin in circadian medicine: a systematic review. Minerva medica, 115(2), 125–142. https://doi.org/10.23736/S0026-4806.24.09303-0

10. Fang, H., Tu, S., Sheng, J., & Shao, A. (2019). Depression in sleep disturbance: A review on a bidirectional relationship, mechanisms and treatment. Journal of cellular and molecular medicine, 23(4), 2324–2332. https://doi.org/10.1111/jcmm.14170
11. Geddes, J. R., & Miklowitz, D. J. (2013). Treatment of bipolar disorder. Lancet (London, England), 381(9878), 1672–1682. https://doi.org/10.1016/S0140-6736(13)60857-0
12. Grandner, M. A., & Fernandez, F. X. (2021). The translational neuroscience of sleep: A contextual framework. Science (New York, N.Y.), 374(6567), 568–573. https://doi.org/10.1126/science.abj8188
13. Han, Y., Yuan, K., Zheng, Y., & Lu, L. (2020). Orexin Receptor Antagonists as Emerging Treatments for Psychiatric Disorders. Neuroscience bulletin, 36(4), 432–448. https://doi.org/10.1007/s12264-019-00447-9
14. Harvey A. G. (2008). Sleep and circadian rhythms in bipolar disorder: seeking synchrony, harmony, and regulation. The American journal of psychiatry, 165(7), 820–829. https://doi.org/10.1176/appi.ajp.2008.08010098
15. Hertenstein, E., Trinca, E., Wunderlin, M., Schneider, C. L., Züst, M. A., Fehér, K. D., Su, T., Straten, A. V., Berger, T., Baglioni, C., Johann, A., Spiegelhalder, K., Riemann, D., Feige, B., & Nissen, C. (2022). Cognitive behavioral therapy for insomnia in patients with mental disorders and comorbid insomnia: A systematic review and meta-analysis. Sleep medicine reviews, 62, 101597. https://doi.org/10.1016/j.smrv.2022.101597
16. Kaskie, R. E., & Ferrarelli, F. (2020). Sleep disturbances in schizophrenia: what we know, what still needs to be done. Current opinion in psychology, 34, 68–71. https://doi.org/10.1016/j.copsyc.2019.09.011
17. Kawai, K., Iwamoto, K., Miyata, S., Okada, I., Fujishiro, H., Noda, A., Nakagome, K., Ozaki, N., & Ikeda, M. (2023). Comparison of Polysomnography, Single-Channel Electroencephalogram, Fitbit, and Sleep Logs in Patients With Psychiatric Disorders: Cross-Sectional Study. Journal of medical Internet research, 25, e51336. https://doi.org/10.2196/51336
18. Kerner, N. A., & Roose, S. P. (2016). Obstructive Sleep Apnea is Linked to Depression and Cognitive Impairment: Evidence and Potential Mechanisms. The American journal of geriatric psychiatry : official journal of the American Association for Geriatric Psychiatry, 24(6), 496–508. https://doi.org/10.1016/j.jagp.2016.01.134
19. Kishi, T., Koebis, M., Sugawara, M., Kawatsu, Y., Taninaga, T., & Iwata, N. (2024). Orexin receptor antagonists in the treatment of insomnia associated with psychiatric disorders: a systematic review. Translational psychiatry, 14(1), 374. https://doi.org/10.1038/s41398-024-03087-4
20. Kragh, M., Dyrberg, H., Speed, M., Pedersen, P., Kristiansen, S. T., & Martiny, K. (2024). The efficacy of a transdiagnostic sleep intervention for outpatients with sleep problems and depression, bipolar disorder, or attention deficit disorder: study protocol for a randomized controlled trial. Trials, 25(1), 57. https://doi.org/10.1186/s13063-024-07903-6
21. Lappas, A. S., Glarou, E., Polyzopoulou, Z. A., Goss, G., Huhn, M., Samara, M. T., & Christodoulou, N. G. (2024). Pharmacotherapy for sleep disturbances in post-traumatic stress disorder (PTSD): A network meta-analysis. Sleep medicine, 119, 467–479. https://doi.org/10.1016/j.sleep.2024.05.032
22. Laskemoen, J. F., Simonsen, C., Büchmann, C., Barrett, E. A., Bjella, T., Lagerberg, T. V., Vedal, T. J., Andreassen, O. A., Melle, I., & Aas, M. (2019). Sleep disturbances in schizophrenia spectrum and bipolar disorders - a transdiagnostic perspective. Comprehensive psychiatry, 91, 6–12. https://doi.org/10.1016/j.comppsych.2019.02.006
23. Li, S., Zhang, R., Hu, S., & Lai, J. (2022). Plasma Orexin-A Levels in Patients With Schizophrenia: A Systematic Review and Meta-Analysis. Frontiers in psychiatry, 13, 879414. https://doi.org/10.3389/fpsyt.2022.879414
24. Liu, L., Wang, H., Chen, X., Zhang, Y., Zhang, H., & Xie, P. (2023). Gut microbiota and its metabolites in depression: from pathogenesis to treatment. EBioMedicine, 90, 104527. https://doi.org/10.1016/j.ebiom.2023.104527
25. Manoach, D. S., & Stickgold, R. (2019). Abnormal Sleep Spindles, Memory Consolidation, and Schizophrenia. Annual review of clinical psychology, 15, 451–479. https://doi.org/10.1146/annurev-clinpsy-050718-095754
26. Marchetti, M., Mayeli, A., Sanguineti, C., Donati, F. L., Chaichian, O., Kim, A., Piskun, K., D'Agostino, A., Meyer, N., Wilson, J. D., Fusar-Poli, P., Phillips, M. L., & Ferrarelli, F. (2025). Sleep abnormalities in bipolar disorders across mood phases: A systematic review and meta-analysis. Sleep medicine reviews, 83, 102137. https://doi.org/10.1016/j.smrv.2025.102137
27. Mendoza Alvarez, M., Balthasar, Y., Verbraecken, J., Claes, L., van Someren, E., van Marle, H. J. F., Vandekerckhove, M., & De Picker, L. (2025). Systematic review: REM sleep, dysphoric dreams and nightmares as transdiagnostic features of psychiatric disorders with emotion dysregulation - Clinical implications. Sleep medicine, 127, 1–15. https://doi.org/10.1016/j.sleep.2024.12.037
28. Messman, B. A., Fentem, A., Compton, S. E., Griffith, E. L., Blumenthal, H., Contractor, A. A., & Slavish, D. C. (2023). The role of affect in associations between sleep disturbances and posttraumatic stress disorder symptoms: A systematic review. Sleep medicine, 110, 287–296. https://doi.org/10.1016/j.sleep.2023.08.025
29. Mijnster, T., Boersma, G. J., van Veen, M. M., Lieburg, E., Cath, D., Pijnenborg, G. H. M., De Jong, P. J., & Lancel, M. (2024). Sleep disorders in a naturalistic cohort of Dutch psychiatric outpatients: prevalence rates and associations with psychopathology symptom severity and well-being. Journal of sleep research, 33(2), e14009. https://doi.org/10.1111/jsr.14009

30. Mirchandaney, R., Barete, R., & Asarnow, L. D. (2022). Moderators of Cognitive Behavioral Treatment for Insomnia on Depression and Anxiety Outcomes. Current psychiatry reports, 24(2), 121–128. https://doi.org/10.1007/s11920-022-01326-3
31. Moon, E., Kim, K., Partonen, T., & Linnaranta, O. (2022). Role of Melatonin in the Management of Sleep and Circadian Disorders in the Context of Psychiatric Illness. Current psychiatry reports, 24(11), 623–634. https://doi.org/10.1007/s11920-022-01369-6
32. Nutt, D., Wilson, S., & Paterson, L. (2008). Sleep disorders as core symptoms of depression. Dialogues in clinical neuroscience, 10(3), 329–336. https://doi.org/10.31887/DCNS.2008.10.3/dnutt
33. Pandi-Perumal, S. R., Monti, J. M., Burman, D., Karthikeyan, R., BaHammam, A. S., Spence, D. W., Brown, G. M., & Narashimhan, M. (2020). Clarifying the role of sleep in depression: A narrative review. Psychiatry research, 291, 113239. https://doi.org/10.1016/j.psychres.2020.113239
34. Pearson, O., Uglik-Marucha, N., Miskowiak, K. W., Cairney, S. A., Rosenzweig, I., Young, A. H., & Stokes, P. R. A. (2023). The relationship between sleep disturbance and cognitive impairment in mood disorders: A systematic review. Journal of affective disorders, 327, 207–216. https://doi.org/10.1016/j.jad.2023.01.114
35. Ressler, K. J., Berretta, S., Bolshakov, V. Y., Rosso, I. M., Meloni, E. G., Rauch, S. L., & Carlezon, W. A., Jr (2022). Post-traumatic stress disorder: clinical and translational neuroscience from cells to circuits. Nature reviews. Neurology, 18(5), 273–288. https://doi.org/10.1038/s41582-022-00635-8
36. Scott, J., Kallestad, H., Vedaa, O., Sivertsen, B., & Etain, B. (2021). Sleep disturbances and first onset of major mental disorders in adolescence and early adulthood: A systematic review and meta-analysis. Sleep medicine reviews, 57, 101429. https://doi.org/10.1016/j.smrv.2021.101429
37. Stefanowski, B., Koceniak, P., Antczak, J., & Rusin, G. (2025). Non-invasive brain stimulation for the treatment of sleep disorders in psychiatric conditions-systematic review protocol. BMJ open, 15(9), e106438. https://doi.org/10.1136/bmjopen-2025-106438
38. Tonon, A. C., Nexha, A., Mendonça da Silva, M., Gomes, F. A., Hidalgo, M. P., & Frey, B. N. (2024). Sleep and circadian disruption in bipolar disorders: From psychopathology to digital phenotyping in clinical practice. Psychiatry and clinical neurosciences, 78(11), 654–666. https://doi.org/10.1111/pcn.13729
39. Walker, M. P., & van der Helm, E. (2009). Overnight therapy? The role of sleep in emotional brain processing. Psychological bulletin, 135(5), 731–748. https://doi.org/10.1037/a0016570
40. Weber, F. C., & Wetter, T. C. (2022). The Many Faces of Sleep Disorders in Post-Traumatic Stress Disorder: An Update on Clinical Features and Treatment. Neuropsychobiology, 81(2), 85–97. https://doi.org/10.1159/000517329
41. Yasugaki, S., Okamura, H., Kaneko, A., & Hayashi, Y. (2025). Bidirectional relationship between sleep and depression. Neuroscience research, 211, 57–64. https://doi.org/10.1016/j.neures.2023.04.006
42. Zielinski, M. R., & Gibbons, A. J. (2022). Neuroinflammation, Sleep, and Circadian Rhythms. Frontiers in cellular and infection microbiology, 12, 853096. https://doi.org/10.3389/fcimb.2022.853096
43. Zou, H., Zhou, H., Yan, R., Yao, Z., & Lu, Q. (2022). Chronotype, circadian rhythm, and psychiatric disorders: Recent evidence and potential mechanisms. Frontiers in neuroscience, 16, 811771. https://doi.org/10.3389/fnins.2022.811771