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THE SIGNIFICANCE OF SLEEP DISTURBANCES IN THE CLINICAL AND PSYCHOSOCIAL PRESENTATION OF DEPRESSION - NARRATIVE LITERATURE REVIEW

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ABSTRACT

Background: Sleep disturbances are highly prevalent in depressive disorders and are increasingly recognized as both core symptoms and risk factors. Insomnia, hypersomnia, fragmented sleep, and circadian rhythm disruptions—including social jetlag—contribute to the onset, severity, and persistence of depressive symptoms, while impairing cognitive, emotional, and psychosocial functioning.
Methods: A narrative literature review was conducted using PubMed, Web of Science, and Scopus, focusing on peer-reviewed studies published between 2000 and 2025. Studies examining subjective and objective measures of sleep in adult populations with depressive disorders were included, with emphasis on clinical presentation, functional outcomes, and treatment response.
Results: Sleep disturbances are associated with greater depressive symptom severity, earlier onset of illness, impaired psychosocial functioning, and poorer response to treatment. Insomnia is the most common disturbance, while circadian misalignment, including social jetlag, is particularly relevant in adolescents, shift workers, and evening chronotypes. Mechanistic evidence links sleep disturbances to mood dysregulation through HPA axis disruption, inflammation, and altered melatonin secretion. Sleep-focused interventions, such as cognitive-behavioral therapy for insomnia, structured sleep schedules, and light therapy, improve both sleep quality and depressive outcomes, and enhance responsiveness to pharmacological treatments.
Conclusion: Sleep disturbances are integral to the clinical and psychosocial presentation of depression. Early identification and targeted management of insomnia, hypersomnia, and circadian misalignment should be incorporated into routine care to optimize symptom control, functional outcomes, and long-term prognosis.

KEYWORDS
Sleep Disturbances, Depression, Insomnia, Major Depressive Disorder

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Introduction

Depression is one of the most prevalent and disabling mental health disorders worldwide, affecting over 280 million individuals and ranking among the leading causes of disability and global disease burden (WHO, 2025). Clinically, depression is characterized by persistent low mood, anhedonia, cognitive deficits, impaired psychosocial functioning, and diminished quality of life. Sleep disturbances—including insomnia, hypersomnia, fragmented sleep, and circadian rhythm disruptions—are increasingly recognized as not merely secondary symptoms, but as core features and risk factors that influence the onset, severity, and maintenance of depressive disorders (Baglioni et al., 2016; Fang et al., 2019; Ballesio, 2023). These disturbances exert substantial impact on cognitive, emotional, and psychosocial functioning, highlighting the importance of assessing and addressing sleep in both research and clinical practice.

A growing body of evidence indicates that sleep disturbances can precede depressive episodes, suggesting a causal or contributory role in the pathophysiology of depression (Adam et al., 2022; Gebara et al., 2018). Insomnia, the most frequently reported sleep disturbance in depression, affects up to 70–90% of patients and is associated with greater symptom severity, earlier onset, and poorer treatment outcomes (Baglioni et al., 2016; Adam et al., 2022). Mechanistically, insomnia contributes to hyperarousal of the hypothalamic-pituitary-adrenal (HPA) axis, elevated inflammatory markers, altered neuroplasticity, and dysregulated sleep architecture, including reduced slow-wave sleep and shortened REM latency, all of which exacerbate depressive symptoms and prolong illness duration (Fang et al., 2019; Huang et al., 2025).

Circadian rhythm disruptions, including phase delays, irregular sleep-wake cycles, and social jetlag, are also closely linked to depression. Social jetlag, defined as the misalignment between biological and social clocks, is prevalent in adolescents, shift workers, and evening chronotypes, and is associated with increased depressive symptoms, impaired cognition, and daytime fatigue (Sun et al., 2025; Salfi et al., 2022). Circadian misalignment influences neuroendocrine regulation, including delayed melatonin secretion and evening hypercortisolemia, and interacts with insomnia and hypersomnia to form a complex, bidirectional network that amplifies mood dysregulation (Archer et al., 2021; Huang et al., 2025; Morin et al., 2006).

The bidirectional relationship between sleep disturbances and depression is a central concept in understanding their clinical significance. While poor sleep can increase vulnerability to depressive episodes, depression itself further disrupts sleep patterns, establishing a self-reinforcing cycle that complicates treatment and worsens functional outcomes (Baglioni et al., 2016; Fang et al., 2019; Nutt et al., 2008). Longitudinal studies demonstrate that chronic insomnia and circadian misalignment are predictive of both depression onset and relapse, highlighting the need for early identification and intervention (Adam et al., 2022; Troxel et al., 2012).

Targeted interventions addressing sleep disturbances have shown efficacy in improving both mood and functional outcomes. Cognitive-behavioral therapy for insomnia (CBT-I) is well-supported by meta-analytic evidence, demonstrating reductions in depressive symptoms and improved treatment response when combined with pharmacotherapy (Morin et al., 2006; Gebara et al., 2018). Structured sleep-wake schedules, light therapy, and interventions tailored to circadian misalignment have also been associated with improvements in mood, cognitive function, and psychosocial functioning (Salfi et al., 2022).

At the population level, the interplay between sleep disturbances and depression has substantial public health implications. Individuals with coexisting sleep problems exhibit longer illness duration, higher symptom burden, and increased healthcare utilization (WHO, 2025). Addressing sleep disturbances through educational programs, flexible work schedules, and circadian-friendly policies may reduce both the incidence and severity of depressive episodes, complementing individual-level interventions and enhancing overall psychosocial well-being (Sun et al., 2025; WHO, 2025).

Despite growing recognition of the centrality of sleep disturbances in depression, a comprehensive synthesis of recent evidence that integrates clinical, psychosocial, and mechanistic perspectives remains limited. This narrative review aims to address this gap by examining current literature on the significance of sleep disturbances in the clinical and psychosocial presentation of depression, emphasizing their impact on symptom severity, functional outcomes, and treatment response. By consolidating recent findings, this review seeks to inform both research and clinical practice, highlighting the importance of sleep-focused assessment and interventions in the management of depressive disorders.

Methodology

This study adopts a narrative literature review approach aimed at synthesizing clinically and psychosocially relevant evidence on the role of sleep disturbances in depression, rather than exhaustively cataloguing all available studies. This approach was selected to allow integration of findings across heterogeneous methodologies and to emphasize clinical applicability. As a narrative review, the study does not provide a quantitative synthesis or formal risk-of-bias assessment.

A literature search was conducted using PubMed, Web of Science, Scopus and Google Scholar to identify peer-reviewed publications from 2000 to 2025. The search focused on studies examining sleep disturbances—including insomnia, hypersomnia, sleep fragmentation, and circadian rhythm disruptions—in adult populations diagnosed with depressive disorders. Both subjective sleep assessments (e.g., questionnaires and clinical interviews) and objective measures (e.g., polysomnography and actigraphy) were considered.

The review included original clinical studies, systematic reviews, meta-analyses, and relevant international reports providing epidemiological or clinical context. Articles were selected based on their relevance to clinical presentation, symptom severity, illness course, treatment response, and psychosocial functioning in depression. Studies focusing exclusively on pediatric populations, non-English publications, and articles without accessible full texts were excluded.

Data extraction emphasized key study characteristics, including population features, sleep assessment methods, measures of depressive symptoms, and principal findings. Given the heterogeneity of study designs and outcome measures, results were synthesized narratively, with particular attention to patterns of association, proposed mechanisms, and clinical implications.

Results

Disruptions in sleep are consistently linked not only to the onset but also to the severity of depressive episodes. Insomnia, hypersomnia, and circadian rhythm disturbances have been shown to exacerbate depressive symptoms, impair cognitive and emotional functioning, and negatively influence psychosocial outcomes and response to treatment (Baglioni et al., 2016; Fang et al., 2019). These disturbances frequently co-occur with depression, suggesting a complex and mutually reinforcing relationship in which poor sleep contributes to mood dysregulation, while depressive symptoms further disturb sleep patterns.

Longitudinal evidence indicates that social jetlag—the misalignment between an individual's biological clock and socially imposed schedules—may be particularly detrimental, especially among adolescents and young adults. Individuals experiencing social jetlag demonstrate higher depressive symptom scores, increased daytime sleepiness, and reduced cognitive performance compared with peers maintaining regular sleep-wake schedules (Sun et al., 2025). Such findings highlight the clinical relevance of considering both the timing and quality of sleep, not merely sleep duration.

Mechanistically, chronic sleep disturbances are associated with dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, resulting in abnormal cortisol secretion, as well as elevated levels of pro-inflammatory cytokines and altered melatonin production. These physiological changes are implicated in the persistence of negative affect, impaired emotional regulation, and maintenance of depressive symptomatology (Fang et al., 2019; Huang et al., 2020). Alterations in sleep architecture, such as reduced slow-wave sleep and shortened REM latency, may further perpetuate mood disturbances by interfering with neuroplasticity and cognitive restoration.

Intervention studies demonstrate that targeted sleep therapies can meaningfully improve depressive outcomes. Cognitive-behavioral therapy for insomnia (CBT-I), either alone or in combination with pharmacotherapy, has been shown to reduce depressive symptom severity, enhance daytime functioning, and lower relapse rates over follow-ups extending up to twelve months (Scott et al., 2021). Meta-analytic evidence supports the notion that addressing sleep disturbances enhances the efficacy of standard antidepressant treatments, emphasizing the value of integrating sleep-focused interventions into routine clinical care (Gebara et al., 2018).

From a population perspective, depressive disorders remain highly prevalent and represent a substantial global burden. According to the World Health Organization, depression affects more than 280 million people worldwide, contributing significantly to disability, decreased productivity, and psychosocial impairment (WHO, 2025). These observations underscore the importance of public health strategies aimed at improving sleep hygiene, promoting flexible work and school schedules, and fostering circadian-friendly environments. Such interventions have the potential not only to reduce the incidence of depression but also to mitigate symptom severity and enhance functional outcomes among affected individuals.

Insomnia and depressive symptomatology

Insomnia is the most commonly reported sleep disturbance in individuals with depression, affecting a substantial proportion of patients (Baglioni et al., 2016; Fang et al., 2019). Clinically, insomnia manifests as difficulty initiating or maintaining sleep, frequent nocturnal awakenings, or early morning awakenings. These disruptions are often accompanied by daytime fatigue, irritability, and cognitive impairments, all of which exacerbate depressive symptoms and interfere with psychosocial functioning (Adam et al., 2022).

Longitudinal evidence indicates that insomnia is not merely a symptom of depression but also a predictor of its onset. Persistent difficulties in sleep initiation and maintenance increase vulnerability to major depressive episodes, highlighting insomnia as a potential early warning sign (Harvey, 2008; Gebara et al., 2018). Patients with coexisting insomnia exhibit higher depression severity scores, including greater mood disturbance, impaired cognition, and psychomotor slowing (Adam et al., 2022; Fang et al., 2019).

Mechanistically, insomnia contributes to hyperarousal of the hypothalamic-pituitary-adrenal (HPA) axis, increases inflammatory markers, and disrupts neuroplasticity, which collectively exacerbate mood dysregulation and prolong depressive episodes (Ballesio, 2023; Fang et al., 2019). Objective sleep studies also demonstrate reduced slow-wave sleep and altered REM patterns in patients with depression and insomnia, reinforcing the biological relevance of sleep disturbances in mood regulation (Baglioni et al., 2016; Goldstein et al., 2014).

Interventions targeting insomnia, particularly cognitive-behavioral therapy for insomnia (CBT-I), have shown robust benefits. Clinical trials indicate that combining CBT-I with pharmacological treatment improves sleep quality, reduces depressive symptom severity, and enhances functional outcomes (Morin et al., 2006; Scott et al., 2021; Gebara et al., 2018). These findings underscore that systematic assessment and management of insomnia should be an integral component of depression care, rather than an ancillary concern.

Circadian rhythm disruptions

Circadian rhythm disturbances are increasingly recognized as central contributors to depressive disorders, influencing both symptom severity and treatment outcomes. The circadian system regulates sleep-wake cycles, hormone secretion, body temperature, and mood, and its dysregulation can manifest as delayed sleep phase, irregular sleep-wake patterns, or misalignment between intrinsic biological clocks and social obligations (Archer et al., 2021; Baglioni et al., 2016).

A common manifestation of circadian misalignment is social jetlag, defined as the discrepancy between an individual's internal circadian timing and socially imposed schedules, such as school, work, or shift obligations. Longitudinal and cross-sectional studies show that adolescents and young adults experiencing social jetlag report higher depressive symptom scores, impaired daytime functioning, and increased fatigue (Sun et al., 2025; Salfi et al., 2022). Evening chronotypes, in particular, appear vulnerable, although flexible schedules such as remote work may reduce their risk of mood disturbances (Salfi et al., 2022).

Mechanistically, circadian disruptions alter hypothalamic-pituitary-adrenal (HPA) axis activity, leading to elevated evening cortisol levels, delayed melatonin secretion, and fragmented sleep architecture. These physiological changes contribute to emotional dysregulation, cognitive impairment, and rumination, all of which exacerbate depressive symptomatology (Archer et al., 2021; Ballesio, 2023). Polysomnographic and actigraphic studies in depressed populations confirm reduced slow-wave sleep, shortened REM latency, and increased nighttime awakenings, highlighting the biological relevance of circadian misalignment in mood disorders (Baglioni et al., 2016; Goldstein et al., 2014).

Interventions targeting circadian disturbances, including structured sleep-wake schedules and light therapy, demonstrate clinically meaningful improvements in both sleep and mood. Aligning sleep timing with endogenous circadian rhythms enhances treatment response to antidepressants and reduces relapse risk (Morin et al., 2006; Scott et al., 2021). Addressing social jetlag and irregular schedules at the population level, through education and flexible work policies, may further mitigate depressive burden, particularly in adolescents and shift workers (Sun et al., 2025; WHO, 2025).

Overall, circadian rhythm disruptions not only exacerbate depressive symptoms but also interact with insomnia and hypersomnia, forming a complex network of sleep-related vulnerabilities. Comprehensive assessment and intervention strategies must therefore address both the timing and quality of sleep to optimize clinical outcomes in depression.

Bidirectional relationship between sleep and depression

The relationship between sleep disturbances and depression is inherently bidirectional, forming a self-reinforcing cycle that amplifies symptom severity and complicates treatment. Insomnia, hypersomnia, and circadian misalignment increase vulnerability to depressive episodes, while depressive symptoms—including low mood, anhedonia, rumination, and heightened stress perception—further disrupt sleep patterns (Baglioni et al., 2016; Fang et al., 2019; Adam et al., 2022).

Longitudinal evidence supports this cyclical link. Individuals with persistent insomnia are at higher risk of developing depression, and once depressive symptoms emerge, sleep quality often deteriorates further, perpetuating cognitive and emotional dysfunction (Gebara et al., 2018; Troxel et al., 2012). Hypersomnia, frequently observed in atypical depression, may also disrupt circadian timing, further exacerbating mood disturbances and functional impairment (Baglioni et al., 2016).

Circadian rhythm disturbances, including social jetlag, further reinforce this feedback loop. Misalignment between internal biological clocks and social demands leads to delayed melatonin secretion, elevated evening cortisol, and fragmented sleep architecture, all of which contribute to heightened depressive symptoms and reduced daytime functioning (Sun et al., 2025; Salfi et al., 2022; Archer et al., 2021). Adolescents, young adults, shift workers, and urban residents are particularly vulnerable due to the frequent conflict between natural sleep-wake preferences and social schedules (Sun et al., 2025; WHO, 2025).

Evidence also suggests synergistic interactions between different types of sleep disturbances. Patients experiencing both insomnia and social jetlag report more severe depressive symptoms, higher fatigue, and reduced responsiveness to standard antidepressant therapies (Morin et al., 2006; Scott et al., 2021). These findings underscore the necessity of comprehensive assessment strategies that consider not only sleep quantity and quality but also circadian alignment and environmental factors.

Clinical interventions that target this bidirectional relationship demonstrate meaningful benefits. Cognitive-behavioral therapy for insomnia (CBT-I), structured sleep-wake schedules, and light therapy improve both sleep quality and depressive symptoms, enhance treatment response to pharmacotherapy, and reduce relapse risk (Gebara et al., 2018; Scott et al., 2021). Early identification and correction of circadian misalignment, particularly in adolescents and shift workers, may prevent progression to full depressive episodes and improve long-term outcomes (Sun et al., 2025; Salfi et al., 2022).

In conclusion, sleep disturbances are not merely secondary features of depression but active contributors to its onset, maintenance, and severity. Effective management requires integrated strategies addressing insomnia, hypersomnia, and circadian disruptions alongside standard depression treatments to optimize clinical and functional outcomes.

Clinical impact and population-level observations

Sleep disturbances in depression have profound consequences not only for individual patients but also for broader public health. According to the World Health Organization (WHO, 2025), depression affects over 280 million people worldwide, ranking among the leading causes of disability and contributing substantially to global morbidity and socioeconomic burden. Sleep problems—including insomnia, hypersomnia, and circadian rhythm disruptions—represent some of the most common and modifiable factors influencing the severity and functional impact of depression.

Epidemiological studies indicate that patients with coexisting sleep disturbances experience longer illness duration, greater symptom burden, and reduced treatment response. For instance, patients with persistent insomnia achieve lower remission rates after standard antidepressant therapy compared with patients without insomnia (Gebara et al., 2018; Troxel et al., 2012). Similarly, circadian misalignment, particularly social jetlag, substantially increases the risk of depressive relapse, emphasizing the prognostic relevance of assessing sleep patterns in depression management (Sun et al., 2025).

Certain populations are particularly vulnerable to sleep-related exacerbation of depressive symptoms. Adolescents, shift workers, and urban residents frequently exhibit irregular sleep-wake schedules. Studies indicate that over 40% of adolescents experience more than two hours of discrepancy between weekday and weekend sleep, correlating with higher depressive symptom scores (Sun et al., 2025). These misalignments disrupt circadian physiology and emotional regulation, increasing susceptibility to mood dysregulation, cognitive impairment, and functional deficits (Salfi et al., 2022; Morin et al., 2006; Riemann et al., 2017).

From a clinical perspective, targeted interventions addressing sleep disturbances are essential for comprehensive depression care. Cognitive-behavioral therapy for insomnia (CBT-I), structured sleep-wake schedules, and light therapy have been consistently shown to improve both sleep quality and depressive

outcomes, reduce relapse rates, and enhance responsiveness to pharmacological treatment (Morin et al., 2006; Scott et al., 2021). In addition, population-level strategies such as school-based sleep education, flexible work schedules, and promotion of circadian-friendly environments may mitigate the societal burden of depression by reducing symptom severity, absenteeism, and long-term disability (WHO, 2025).

Integrating sleep-focused interventions into routine mental health programs allows clinicians to break the self-reinforcing cycle between sleep disturbances and depression. Early identification and management of insomnia, hypersomnia, and circadian misalignment are not only beneficial for individual patients but also serve as preventive strategies at the community level. Addressing these modifiable factors has the potential to reduce both incidence and severity of depressive episodes, improving quality of life and functional outcomes across populations.

In summary, the clinical impact of sleep disturbances in depression is extensive, encompassing symptom severity, treatment efficacy, relapse risk, and public health burden. Effective management requires a multifaceted approach that integrates sleep assessment and targeted interventions alongside conventional pharmacological and psychotherapeutic treatments, with the goal of optimizing outcomes at both individual and societal levels.

Discussion

This narrative review highlights the multifaceted and clinically significant relationship between sleep disturbances and depression. Insomnia, hypersomnia, and circadian rhythm disruptions, including social jetlag, are not merely co-occurring symptoms but active contributors to the onset, severity, and persistence of depressive episodes (Baglioni et al., 2016; Fang et al., 2019; Sun et al., 2025). The bidirectional nature of this relationship underscores that poor sleep increases vulnerability to depression, while depressive symptoms exacerbate sleep disturbances, creating a self-perpetuating cycle that complicates clinical assessment and treatment (Adam et al., 2022; Harvey, 2008; Troxel et al., 2012).

Mechanistically, sleep disturbances impact key biological systems involved in mood regulation. Dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, elevated pro-inflammatory cytokines, altered melatonin secretion, and disrupted neuroplasticity provide robust pathways linking sleep deficits with depressive symptomatology (Ballesio, 2023; Fang et al., 2019). Chronic insomnia and circadian misalignment are also associated with changes in sleep architecture, such as reduced slow-wave sleep and shortened REM latency, further influencing mood regulation and cognitive functioning (Baglioni et al., 2016; Morin et al., 2006; Goldstein et al., 2014).

Clinically, the evidence strongly supports integrating sleep assessment and intervention into standard depression care. Cognitive-behavioral therapy for insomnia (CBT-I), structured sleep-wake schedules, and light therapy have demonstrated efficacy in improving both sleep quality and depressive outcomes, reducing relapse risk and enhancing responsiveness to pharmacological treatment (Morin et al., 2006; Scott et al., 2021; Gebara et al., 2018). Addressing circadian misalignment, particularly in adolescents, shift workers, and evening chronotypes, offers additional opportunities for non-pharmacological intervention and prevention of depressive episodes (Salfi et al., 2022; Sun et al., 2025).

At the population level, the prevalence of depressive disorders combined with widespread sleep disturbances represents a significant public health challenge. More than 280 million people are affected worldwide, with sleep disturbances contributing to functional impairment, reduced treatment response, and increased societal burden (WHO, 2025). Integrating sleep-focused strategies into mental health programs and public health policies—such as education on sleep hygiene, flexible work and school schedules, and circadian-friendly urban planning—may reduce both incidence and severity of depressive episodes (Riemann et al., 2017; WHO, 2025).

Finally, emerging evidence suggests the potential for personalized approaches. Genetic factors influencing circadian regulation may modulate individual susceptibility to depression in the context of sleep disruption (Archer et al., 2021). Understanding interactions between biological predispositions and environmental factors could guide tailored interventions, improving clinical outcomes and reducing relapse risk.

In summary, sleep disturbances are central to the pathophysiology, clinical expression, and psychosocial impact of depression. A comprehensive approach integrating assessment and intervention for insomnia, hypersomnia, and circadian misalignment is essential for optimizing patient outcomes and reducing population-level burden.

Conclusions

Sleep disturbances play a critical role in the development, clinical course, and prognosis of depression. Insomnia, hypersomnia, and circadian disruptions—including social jetlag—are consistently associated with increased symptom severity, poorer treatment response, and higher risk of relapse (Baglioni et al., 2016; Fang et al., 2019; Sun et al., 2025; Troxel et al., 2012). The bidirectional relationship between sleep and depression highlights the necessity of addressing sleep disturbances as part of comprehensive depression care.

Clinical interventions such as cognitive-behavioral therapy for insomnia (CBT-I), structured sleep-wake schedules, and light therapy have demonstrated efficacy in improving both sleep quality and depressive symptoms (Morin et al., 2006; Scott et al., 2021). At the population level, promoting sleep health through educational programs, flexible schedules, and circadian-friendly policies may reduce both individual and societal burden of depression (WHO, 2025; Riemann et al., 2017).

A holistic, sleep-inclusive approach should therefore be a standard component of depression management. Early identification and targeted intervention for sleep disturbances can prevent progression to full depressive episodes, improve treatment outcomes, and reduce relapse risk, ultimately enhancing quality of life and functional capacity at both individual and community levels.

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