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SLEEP AND MENTAL HEALTH: THE RELATIONSHIP BETWEEN SLEEP DISORDERS AND DEPRESSION

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ABSTRACT

Sleep disturbances are a core feature of depressive disorders, occurring in most patients during both acute episodes and remission. Insomnia is the most prevalent form and significantly affects treatment response and relapse risk. The relationship between sleep and depression is bidirectional—sleep problems may precede, co-occur with, or result from depression. Non-pharmacological approaches such as cognitive behavioral therapy for insomnia (CBT-I) have proven effective in improving both sleep quality and depressive symptoms, especially when combined with antidepressants. Somatic interventions like chronotherapy, light therapy and sleep cycle manipulation also show promising outcomes. Recognizing and treating sleep disturbances as a primary therapeutic target can enhance the overall effectiveness of depression management and support long-term remission.

KEYWORDS

Sleep Disturbances, Depression, Insomnia, Cognitive Behavioral Therapy for Insomnia (CBT-I), Sleep Architecture, Somatic Therapy, Chronotherapy, Light Therapy, Sleep Deprivation

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Introduction

Depression is one of the most common mental disorders of our time, currently affecting over 280 million individuals globally, according to the World Health Organization (WHO). Early symptoms are frequently subtle and may be overlooked or underestimated by patients. However, as the disorder progresses, it significantly impairs personal and professional functioning, leading to a considerable decline in quality of life. Despite the availability of various treatment methods, treatment resistance and relapses are frequently observed among individuals.

Sleep plays a crucial role in both physical and mental health. Sleep disturbances are an integral component of the clinical presentation of depressive disorders. Nevertheless, despite their significant influence on the course and therapeutic process of depression, they are often overlooked. Insomnia is the most prevalent manifestation, although other forms such as pavor nocturnus, nightmares and hypersomnia may also occur [1].

An increasing number of studies are evaluating the impact of improved sleep quality on the dynamics and efficacy of depression treatment. There is a growing use of approaches based on somatic strategies, encompassing both pharmacotherapy and non-pharmacological therapeutic interventions aimed at modulating environmental stimuli [1]. Emerging evidence also suggests that the use of cognitive behavioral therapy for insomnia (CBT-I) in combination with antidepressant medication in patients diagnosed with both insomnia and depression may result in a greater remission of depressive symptoms compared to pharmacotherapy alone [2].

The aim of this paper is to analyze the current scientific literature on the relationship between sleep disorders and depression, with particular emphasis on their mutual interaction and the bidirectional nature of this relationship. Special attention will also be given to the treatment of sleep disturbances as an integral component of comprehensive depression therapy.

Changes in sleep architecture in individuals with depression

Physiological sleep is divided into two main states: rapid eye movement sleep (REM) and non-rapid eye movement sleep (NREM). Sleep begins with the NREM stage, which accounts for approximately 80% of the entire sleep process, followed by a transition into the REM stage. This cycle repeats several times throughout the night [3].

Sleep plays a crucial role in secretion of growth hormone, the clearance of harmful metabolites from the brain and memory consolidation [4][5][6][7]. Additionally, acute sleep deprivation leads to elevated plasma cortisol levels [8], indicating that sleep may also contribute to stress resilience. On the other hand, accumulated stress negatively impacts mental health and contributes to the development of psychiatric and sleep disorders [3]. This underscores the strong association between sleep and depressive disorders.

Patients diagnosed with depression frequently exhibit abnormalities in sleep architecture (see summary in Fig.1) [3]. Starting from sleep onset, notable alterations include prolonged sleep latency and an increased number of nocturnal awakenings [9]. Subsequent dysfunctions are observed within the NREM stage, including reduced slow-wave activity (SWA), particularly within the 0.25–2.50 Hz frequency range [10] and decreased delta wave power which is more pronounced in patients experiencing a depressive relapse [11]. This suggests that reduced delta power may serve as a risk factor for depression recurrence [3]. Moving to the next stage of sleep, above all, we can observe an increased amount of REM sleep [12]. Furthermore, there is a shortened interval from the moment of falling asleep to the onset of the first episode of this phase, referred to as sleep latency, increased density meaning a higher frequency of eye movements and a prolonged average duration of rapid eye movement sleep episodes [13][14][15][16]. Additionally, long-term reduction in REM latency has

been associated with a heightened risk of depression relapse [17]. Based on these findings, alterations in sleep architecture may serve as a biomarker of depressive disorders [18][19].

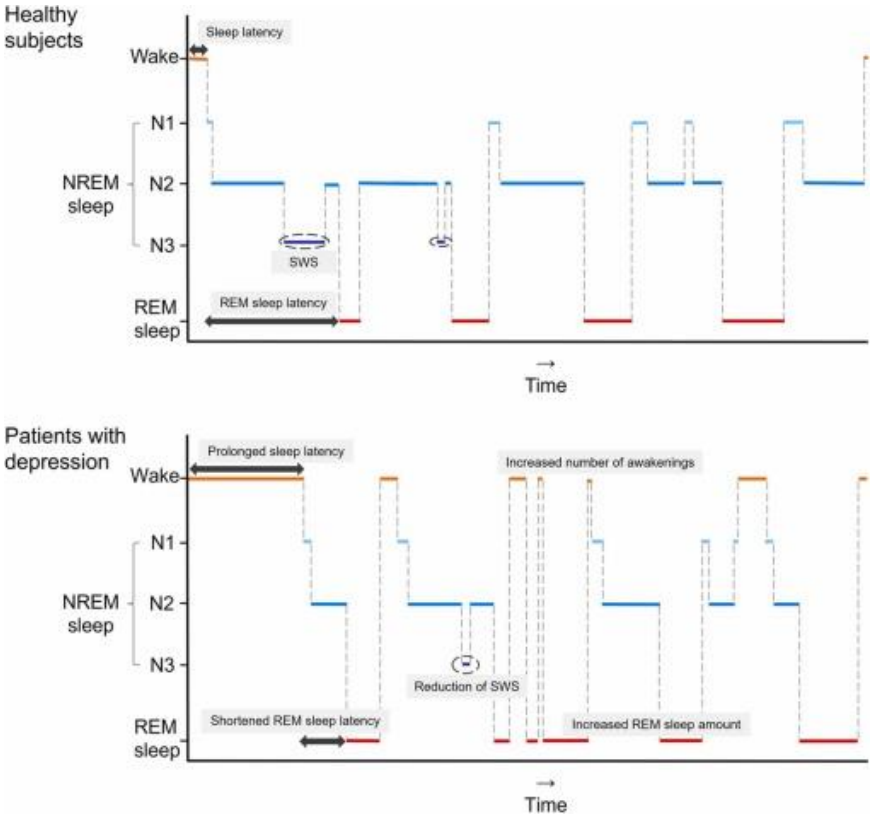

Fig.1. *Schematics of sleep architecture in healthy subjects and patients with depression Sleep abnormalities are observed in patients with depression. [3]*

The relationship between sleep and depression

Scientific literature clearly indicates the bidirectional relationship between sleep and depression. Sleep dysfunctions, in addition to being a diagnostic criterion for depression [3] also represent a risk factor for its development [20][21]. A one-year longitudinal study demonstrated that patients diagnosed with insomnia were at significantly higher risk of developing major depression [22]. Moreover, research findings suggest that sleep rhythm disturbances occurring in early childhood may serve as potential risk factors for the development of depressive disorders later in life [23]. On the other hand, persistent sleep disturbances following remission of depressive symptoms may reflect previously existing sleep difficulties [24]. Furthermore, insomnia has been identified as a predictor of depression relapse risk [25][26][27][28][29].

Sleep disturbances whether subjectively reported or objectively recorded via electroencephalography are significantly associated with reduced effectiveness and delayed achievement of remission during depressive episodes [30][31][32]. These findings suggest that sleep dysfunctions may serve as important prognostic indicators in evaluating the efficacy of depression treatment and underscore the necessity of incorporating sleep quality assessments into therapeutic planning. Among patients diagnosed with depression who exhibit abnormal sleep patterns, significantly less favorable clinical outcomes have been observed. including higher symptom severity, increased rates of premature treatment discontinuation, lower likelihood of achieving remission and reduced stability of therapeutic response, compared to individuals with relatively normal sleep profiles. [31][33] In patients with major depressive disorder, the presence of disrupted sleep continuity and early morning awakenings is associated with a higher likelihood of experiencing suicidal ideation compared to individuals without these symptoms [34]. This suggests that insomnia symptoms may significantly limit the effectiveness of antidepressant therapy [2]. Conversely, depressive symptoms are strongly correlated with an increased risk of developing insomnia, constituting one of its key risk factors. Depression is commonly identified as a significant comorbid condition in patients with chronic insomnia, regardless of its etiopathogenesis. The clinical relevance of this relationship is further amplified by the fact

that certain classes of antidepressant medications may exacerbate insomnia symptoms, thereby hindering the achievement of full clinical remission [35].

As previously mentioned, patients with depression commonly exhibit alterations in the REM stage of sleep [13][16][36]. Pharmacological agents classified as antidepressants, including tricyclic antidepressants (TCAs), tetracyclic antidepressants, monoamine oxidase inhibitors (MAOIs), selective norepinephrine reuptake inhibitors (NARIs), selective serotonin reuptake inhibitors (SSRIs), and serotonin-norepinephrine reuptake inhibitors (SNRIs) affect this sleep phase in various ways (see Fig. 2) [15]. Suppression of REM sleep has been observed in patients treated with tricyclic antidepressants [37], selective serotonin reuptake inhibitors, and serotonin-norepinephrine reuptake inhibitors [38]. This effect is attributed to the influence of serotonergic neurons in the dorsal raphe nucleus on cholinergic regions of the pons [39].

Antidepressants	Changes in sleep
TCAs, MAOIs, NARIs, SSRIs, SNRIs	REM sleep latency ↑ REM sleep amount ↓ REM sleep frequency ↓ REM density ↓ Wang et al., 2015
Agomelatine	Sleep latency ↓ San and Arranz et al., 2008 Sleep efficiency ↑ SWS amount ↑ Quera Salva et al., 2007
Ketamine	REM sleep amount ↑ NREM sleep amount ↑ SWA ↑ Duncan et al., 2013; Duncan et al., 2017

Fig.2. *Antidepressants have diverse effects on sleep in humans. [3]*

However, there are antidepressant medications that are effective in treating depression without reducing REM sleep. One such drug is agomelatine, a melatonergic receptor agonist and a serotonin receptor subtype antagonist. The mechanism of action of agomelatine may be independent of the REM stage, as suggested by findings that long-term treatment with this antidepressant results in reduced sleep latency [40], increased overall sleep efficiency, and enhanced slow-wave sleep (SWS) [41]. The efficacy of agomelatine has been demonstrated in clinical trials [42]. Another agent that has shown positive effects in patients diagnosed with both insomnia and depression is ketamine [43]. In addition to being a muscarinic receptor antagonist and interacting with opioid receptors, ketamine is a non-competitive antagonist of the N-methyl-D-aspartate (NMDA) receptor. Its effects include increased amounts of both REM and NREM sleep, as well as enhanced slow-wave activity (SWA) [44][45]. In patients with depressive disorders, a reduction in suicidal ideation can be observed as early as 40 minutes after administration, indicating ketamine's rapid antidepressant action [46][47][48].

Types of sleep disorders in depression

In the course of depression, sleep disturbances are so prevalent that some authors claim a diagnosis of depressive disorder should be made with caution in their absence [25]. The accompanying sleep dysfunctions can present in various forms.

The most common form of sleep disturbance is insomnia. By definition, it involves difficulty falling asleep, maintaining sleep, and/or experiencing non-restorative sleep, accompanied by impaired daytime functioning for a period of at least four weeks [49]. Insomnia is further classified into initial insomnia (difficulty falling asleep), middle insomnia (difficulty maintaining sleep) and terminal insomnia (early morning awakenings). Initial and terminal insomnia each occur in approximately 23% of cases, while middle insomnia is the most prevalent, affecting 24.4% of individuals [50]. Overall, insomnia may affect 60% to 80% of patients with major depressive disorder [51].

The second type of sleep disturbance is hypersomnia, characterized by excessive daytime sleepiness and a need for napping that fails to produce a sense of refreshment. Importantly, hypersomnia does not imply insufficient nocturnal sleep [1]. Although it occurs more frequently in bipolar depression [52], it may affect up to 30% of patients with unipolar depression [53], including during inter-episode periods [54]. Hypersomnia is also associated with chronic and treatment-resistant forms of depression [55]. It is more commonly observed in atypical depression and tends to affect younger individuals and women across all age groups [1]. Notably, some patients may experience both insomnia and hypersomnia during the same depressive episode [56].

In depression, nightmares are the most frequently occurring parasomnia, defined as"extremely frightening dreams" from which individuals awaken abruptly with rapid reorientation and detailed recall of the dream content—typically involving threats to life, safety, or self-esteem—often followed by difficulty returning to sleep [57]. In the general population, nightmares occur with a prevalence ranging from 1% [58] to 8% [58][59]. They are more commonly reported by female patients and are associated with an increased number of nocturnal awakenings, difficulties in falling asleep, daytime memory impairment and anxiety resulting from inadequate sleep quality [60].

The second parasomnia are night terrors, characterized by sudden arousal from deep sleep, during which the individual may have limited awareness of their surroundings [61]. Symptoms include a scream followed by autonomic nervous system activation, accompanied by stereotypical and persistent motor activity [62]. Such episodes of night terrors appear to occur more frequently in adults with a history of psychiatric disorders [63].

Somatic therapy

Somatic therapy aimed at treating depression includes pharmacotherapy as well as medical interventions designed to modify the patient's perception of external stimuli, such as chronotherapy, light therapy, cycles of sleep and manipulation of the sleep-wake rhythm. The advantages of such medical strategies include their accessibility and an effectiveness that appears to be comparable to that of pharmacotherapy [64].

Light therapy was originally developed as the treatment of choice for seasonal affective disorder [65], depression during pregnancy [66] and eating disorders [67]. The physiological effects of light on the human body depend on the timing of its administration [1]. For example, considering that circadian rhythms in older adults tend to shift toward earlier hours, morning light therapy may help advance sleep onset in cases of difficulty falling or staying asleep [68]. During the first week of light therapy, especially when it is administered in the morning, it typically produces a moderate antidepressant effect with efficacy significantly increasing in patients who are responsive to sleep deprivation [69].

Sleep deprivation is frequently used in individuals with depressive disorders [70][71]. In cases where there is no response to pharmacological or psychotherapeutic treatment within the first 15 days of light therapy, the introduction of sleep cycle manipulation can lead to clinically significant improvement within a few hours in approximately 60% of patients diagnosed with major depression [72]. In these individuals, a single night of sleep deprivation has been associated with a reduction in self-reported depressive symptom severity compared to assessments made the day before the intervention [73]. However, this effect is typically short-lived, with depressive symptoms often returning once normal sleep cycles are restored [73]. To enhance the effectiveness of treatment and prevent relapse, some authors recommend combining sleep cycle manipulation with lithium salts or selective serotonin reuptake inhibitors (SSRIs) [74]. The antidepressant efficacy of sleep deprivation depends on several factors. A meta-analysis has shown that the prediction of changes in depressive symptoms following total sleep deprivation is significantly influenced by baseline diurnal mood variability [75]. The therapeutic effect of this method may also be potentiated when combined with morning light therapy [69].

There is also selective REM sleep deprivation. It is a more complex method involving waking the patient upon entering the rapid eye movement stage, as monitored by electroencephalographic recording (EEG). Given that patients with depressive disorders typically exhibit increased REM sleep, this stage is thought to have a potentially detrimental impact on the course of illness [3]. Studies have shown that after several weeks of selective REM deprivation, patients tend to demonstrate a deterioration in scores on the Hamilton Depression Rating Scale, particularly within the first 24 hours following the completion of the treatment cycle [76][77]. This method commonly induces a significant REM rebound phenomenon during the next period of uninterrupted sleep, suggesting that this rebound may be crucial to the observed antidepressant effect [3]. In patients with insomnia, REM sleep is characterized by an increased frequency of awakenings and more intense eye movements compared to the physiological state [78], thereby reducing REM sleep quality, which may negatively affect the course of depression [3]. Even in healthy individuals, reduced sleep quality has been found to correlate with a slower reduction of emotional stress, as measured by self-report questionnaires [79].

Consequently, the therapeutic mechanism of REM sleep deprivation in patients with depression may be related to limiting exposure to low-quality REM sleep, which could potentially contribute to the persistence of depressive symptoms.

Cognitive Behavioral Therapy for Insomnia (CBT-I)

Cognitive behavioral therapy for insomnia (CBT-I) is currently one of the primary treatment modalities for insomnia. It is a non-pharmacological intervention based on techniques such as sleep restriction, stimulus control aimed at breaking the association between the bedroom environment and wakefulness, cognitive restructuring targeting dysfunctional beliefs and attitudes about sleep, sleep hygiene education and relaxation training [80]. Numerous studies, summarized in several meta-analyses [81][82][83][84], have demonstrated that CBT-I achieves efficacy in the treatment of primary insomnia comparable to that of acute pharmacological therapy [85][86]. Moreover, the scientific literature supports the effectiveness of CBT-I in treating insomnia co-occurring with depression [87][88][89][30][90]. Importantly, CBT-I has been shown to be effective in treating both insomnia symptoms [91][92] and comorbid depression, functioning both as a standalone therapeutic approach and as an adjunct to pharmacological antidepressant treatment [93]. In light of these findings, CBT-I may serve as an effective alternative therapy for individuals suffering from both insomnia and depression, particularly in cases of antidepressant resistance [3]. A notable advantage is the long-term durability of sleep improvements achieved through CBT-I, which can persist for up to two years following the intervention [85]. This is especially relevant in depressive disorders, as maintaining healthy sleep increases the likelihood of sustained remission, in contrast to cases where insomnia relapse occurs [28][94].

A randomized controlled pilot study was conducted to evaluate the feasibility, acceptability and preliminary efficacy of combining escitalopram treatment with CBT-I in patients diagnosed with both major depression and insomnia. The results demonstrated a significantly higher depression remission rate in the group receiving combined escitalopram and CBT-I treatment (62%) compared to those treated with escitalopram and a control intervention alone (33%) [2]. This finding holds considerable clinical significance, as achieving remission in depression is considered the primary therapeutic goal [95]. Furthermore, the combination therapy also resulted in a significantly higher remission rate of insomnia, as defined by the Insomnia Severity Index (ISI), reaching 50% versus only 8% in the escitalopram-only group with the control intervention. This suggests that CBT-I contributes meaningfully to sleep quality improvements beyond those attained with antidepressant monotherapy, emphasizing its additional clinical value in treating comorbid insomnia and depression [2].

Conclusions

This literature review indicates that sleep disturbances are an integral component of the clinical presentation of depression. Although their role in the pathogenesis, course, and treatment of depressive disorders is increasingly well understood, it remains insufficiently addressed in clinical practice.

On one hand, sleep disturbances are a symptom of depression and may serve as a predisposing factor that precedes the onset of the disorder and increases vulnerability to its development. On the other hand, depression itself can lead to sleep disturbances, confirming the bidirectional relationship between sleep and depression. Sleep dysfunctions are present in the majority of patients during both - acute and remission phases. Most commonly, they manifest as insomnia, which not only co-occurs with depression but also significantly influences its clinical course, treatment efficacy, and risk of relapse. Therefore, sleep disturbances should not be regarded merely as secondary symptoms of depression, but rather as comorbid conditions that directly affect therapeutic outcomes.

There is evidence supporting the effectiveness of cognitive behavioral therapy for insomnia (CBT-I) as a valuable adjunct in the treatment of depression. Increasing attention is also being given to the role of somatic therapies, which, in addition to pharmacological treatment, include interventions such as chronotherapy, light therapy, sleep scheduling and manipulation of the sleep–wake cycle. Incorporating strategies aimed at improving sleep quality may significantly accelerate the reduction of depressive symptoms, enhance patients' quality of life, and reduce the risk of relapse.

In conclusion, sleep disturbances should be recognized as an equally important diagnostic and therapeutic target in the treatment of depression. Regardless of their severity, interventions aimed at improving sleep should constitute a consistent component of clinical practice. The integration of pharmacological, psychotherapeutic and somatic approaches in the treatment of sleep disorders may significantly enhance the overall effectiveness of depression therapy.

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