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Sleep Disturbances and Depression: A Narrative Review of the Bidirectional Relationship, Mechanisms, and the Role of Physical Activity

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Abstract

Introduction and aim: Depression and sleep disturbances are common conditions that frequently co-occur and contribute substantially to the global burden of disease. This review summarizes current evidence on their bidirectional relationship and the role of physical activity as a modifiable therapeutic factor.

Material and methods: A narrative review of studies indexed in PubMed, Scopus, and Google Scholar was conducted. Epidemiological, longitudinal, mechanistic, and interventional studies, including systematic reviews and meta-analysis, were analyzed.

Results: Insomnia is a consistent predictor of future depression, while depression negatively affects sleep quality, supporting a reciprocal relationship. Shared mechanisms include altered sleep architecture, REM sleep abnormalities, circadian disruption, and emotional dysregulation. This association is observed across different populations, including children, perinatal women and elder people, and is linked to increased suicide risk. Physical activity and exercise improve both sleep quality and depressive symptoms and may help break this cycle.

Conclusions: Sleep disturbances and depression are bidirectionally linked. Early identification and treatment of sleep problems, together with promotion of physical activity, may improve outcomes and support depression prevention.

Keywords: sleep disorders; physical activity, insomnia; depression; circadian rhythm, sport

1. Introduction

Depression is among the most common psychiatric disorders and a leading cause of disability worldwide, affecting personal functioning, quality of life, and overall societal burden of disease [1]. Its etiopathogenesis is multifactorial, and despite advances in pharmacotherapy and psychotherapy, a substantial proportion of patients do not achieve full remission, which sustains interest in modifiable risk factors and complementary interventions [2].

Sleep disturbances, particularly insomnia, but also excessive sleepiness and disrupted sleep architecture are likewise highly prevalent in the general population and are reported by a majority of individuals with depression [3]. For a long time, disturbed sleep was regarded mainly as a symptom of depression, secondary to the mood disorder, and expected to resolve with its treatment [4]. This view, however, has been progressively revised.

A growing body of longitudinal and mechanistic evidence indicates that the relationship between sleep and depression is bidirectional: sleep disturbances not only accompany depressive episodes but also precede and predict their onset, persist into remission and increase the risk of relapse, while depression in turn degrades sleep quality [1,53]. Insomnia is therefore

increasingly conceptualized as an independent clinical entity and a potential transdiagnostic process rather than a mere epiphenomenon. Recognizing this reciprocity has direct clinical implications, because targeting sleep may improve depressive outcomes and, potentially, prevent depression in at-risk individuals.

This perspective also highlights the importance of modifiable behavioral factors. Physical activity and exercise influence both sleep quality and depressive symptomatology, and may represent a safe, accessible and cost-effective means of interrupting the self-reinforcing sleep-depression cycle, an aspect of particular relevance from a public-health and sports-science standpoint.

The aim of this narrative review is to synthesize current evidence on the bidirectional relationship between sleep disturbances and depression, to outline the underlying neurobiological, circadian and cognitive mechanisms, to characterize this association across different populations and comorbidities, and to evaluate the role of physical activity and other interventions. The review is organized into thematic sections corresponding to these objectives.

2. Material and methods

This article is a narrative review. Relevant literature was identified through searches of the PubMed, Scopus and Google Scholar databases, supplemented by reference list screening of key articles. Search terms combined keywords related to sleep (“sleep”, “sleep disturbance”, “insomnia”, “sleep quality”, “circadian rhythm”, “chronotype”) with terms related to depression and mood (“depression”, “depressive symptoms”, “major depressive disorder”, “mood disorder”) and, for the intervention sections, with terms related to physical activity (“physical activity”, “exercise”, “sport”). Priority was given to systematic reviews, metaanalyses, large prospective cohort studies, Mendelian randomization analyses and recent primary studies published in English. Owing to the narrative design, no formal protocol registration, systematic screening algorithm or quantitative risk of bias assessment was applied; sources were selected for their relevance, methodological quality and contribution to the thematic structure of the review.

3. The bidirectional sleep–depression relationship

3.1. Sleep disturbance as a predictor of depression

The strongest argument against treating disturbed sleep as a mere symptom of depression comes from longitudinal epidemiological research, in which sleep complaints are assessed in individuals who are not depressed at baseline and depressive outcomes are recorded at follow-up. A metaanalysis of longitudinal studies found that non-depressed people with insomnia have approximately twice the risk of subsequently developing depression compared with individuals without sleep difficulties [50]. This estimate has been confirmed and refined by a later metaanalysis of 34 prospective cohorts comprising more than 170,000 participants, which reported a pooled relative risk of incident depression of around 2.3 for individuals with insomnia [20].

Importantly, it is not only the presence of insomnia at a single time point but also its trajectory that matters. A longitudinal community study showed that changes in insomnia over time are associated with both the incidence and the persistence of depressive and anxiety symptoms, indicating that worsening sleep heralds worsening mood, whereas improving sleep accompanies a more favorable course [11]. Taken together, these data support the conceptualization of insomnia as an independent, clinically meaningful predictor of depression rather than a secondary epiphenomenon.

3.2. Reciprocal and bidirectional dynamics

The predictive role of insomnia does not exclude the opposite direction of influence. Depression itself disrupts sleep continuity and architecture, and depressive episodes are frequently accompanied by difficulty initiating and maintaining sleep, early morning awakening and non-restorative sleep [1]. Current models therefore describe a reciprocal, mutually reinforcing relationship in which each condition increases vulnerability to, and aggravates the course of, the other [12].

This bidirectionality is also visible during treatment. In individuals receiving routine psychological therapy for common mental disorders, sleep and depressive symptoms have been shown to improve in parallel and to influence each other over the course of care, with early changes in one domain predicting later changes in the other [37]. Such findings reinforce the clinical relevance of addressing both domains simultaneously rather than assuming that improvement in mood will automatically resolve sleep complaints.

3.3. Temporal and causal evidence

Establishing temporal precedence and causality requires designs that go beyond simple cross-sectional correlation. Cross-lagged panel analyses conducted during the COVID-19 pandemic demonstrated that insomnia symptoms predicted depressive symptoms approximately one year later, and that depressive symptoms likewise predicted subsequent insomnia, confirming a genuinely bidirectional temporal pattern [8]. Bidirectional Mendelian randomization analyses, which use genetic variants as instrumental variables, further suggest that the association is at least partly causal rather than purely confounded [30]. Day to day analyses in adolescents and young adults have similarly linked night to night variation in sleep with next day affect and with prospective risk of major depression [49].

At the same time, the evidence is not uniform. A within individual analysis spanning 20 years in community-dwelling older adults found that depression and insomnia tended to increase concurrently over time but did not constitute an additional risk for one another across repeated three-year intervals [9]. This nuance suggests that the strength and temporal structure of the sleep–depression relationship may vary with age and measurement interval and should be acknowledged rather than overlooked.

3.4. Population-level epidemiological evidence

Large general population datasets corroborate the association at the epidemiological level. Analyses of the U.S. National Health and Nutrition Examination Survey (NHANES) have consistently linked poor or atypical sleep with a higher burden of depressive symptoms in adults [28]. Evidence from Poland is also available: a survey of more than 900 participants conducted at the Pol'and'Rock Festival reported a clear co-occurrence of depressive disorders and sleeping disturbances in a large, predominantly young sample [51], providing regionally relevant context for the present review.

4. Neurobiological, circadian and cognitive mechanisms

4.1. Sleep architecture and neurophysiology

Objective sleep studies provide a physiological basis for the sleep–depression link. A comprehensive metaanalysis of polysomnographic research found that depression, like several other psychiatric disorders, is associated with measurable alterations in sleep architecture, including reduced sleep continuity and changes in rapid-eye-movement (REM) sleep [15]. Building on this, recent work has proposed that chronic insomnia and REM sleep instability constitute a specific pathway towards anxiety and depression, mediated by impaired overnight emotional processing [27]. Contemporary neurophysiological reviews further describe

disrupted slow wave activity and altered sleep microstructure as candidate markers of depressive pathophysiology [35]. Consistent with this, electroencephalographic studies have reported deficits in sleep delta (slow wave) power in treatment-resistant depression, with effects modulated by age and sex [52].

4.2. Circadian rhythms and chronotype

A second mechanistic axis concerns the circadian timing system. Disruption of circadian rhythms is increasingly recognized as a core feature of mood disorders, and circadian-based interventions are correspondingly attracting interest [31]. Chronotype is one clinically accessible expression of this system: evening orientation has been linked, through mediation involving sleep-related factors, to a higher burden of depressive symptoms [6]. In young individuals specifically, delayed sleep-wake phase disorder shows a metaanalytically supported association with depression [33]. Machine-learning analyses of large cohort data have likewise identified subjective and objective sleep and circadian parameters as informative predictors of depression-related outcomes [34].

4.3. Brain structure and motor activity patterns

Mechanistic accounts also extend to neuroanatomy and behavior. Analyses of the UK Biobank cohort have associated lifetime depression and sleep disruption with differences in brain structure, suggesting shared or interacting neurobiological substrates [38]. At the behavioral level, the fractal (self-similar) organization of motor activity across wakefulness and sleep has been proposed as a window into depression recency and the risk of symptom recurrence, illustrating how fine-grained activity patterns may index underlying regulatory dysfunction [47].

4.4. Emotional dysregulation and cognitive impairment

Finally, emotional and cognitive processes appear to mediate part of the sleep–depression relationship. Disturbed sleep impairs the regulation of emotion, and emotional dysregulation has been positioned as a key link in the pathway from chronic insomnia to depression and anxiety [27]. A systematic review of mood disorders has documented a consistent relationship between sleep disturbance and cognitive impairment, indicating that poor sleep may aggravate the cognitive dimension of depression [32]. Conversely, better sleep quality – together with higher levels of physical activity – has been shown to predict lower emotional dysregulation in individuals with major depressive disorder, foreshadowing the protective role of physical activity discussed in Section 7 [45].

5. Sleep and depression across populations and comorbidities

5.1. Children, adolescents and young adults

The sleep–depression relationship is evident from early in life. A systematic review and metaanalysis of cohort studies in children and youths found that disturbed sleep is prospectively associated with an increased risk of depression, indicating that the link is established well before adulthood [5]. Adolescence is a particularly sensitive period, as the normal developmental delay in circadian timing coincides with early school schedules and intensive social media use, producing chronic sleep restriction. Day to day and prospective analyses in late adolescence and early adulthood have linked night to night sleep variability with next day affect and with the subsequent risk of major depression, underlining the clinical relevance of sleep monitoring in this age group [49].

5.2. The perinatal period (pregnancy and postpartum)

Pregnancy and the postpartum period combine physiological, hormonal and psychosocial changes that markedly disturb sleep. Poor sleep quality during pregnancy has been associated with antepartum depressive symptoms and even suicidal ideation [40], and longitudinal data indicate that poor sleep and sleep apnea track with depressive symptoms across gestation [46]. In the postpartum period, poor sleep quality has likewise been shown to increase symptoms of both depression and anxiety in mothers [44]. Importantly, this is a modifiable target: behavioral sleep interventions delivered to infants and mothers can improve maternal and child sleep and reduce maternal depressive symptoms, demonstrating that addressing sleep is a viable preventive strategy in perinatal mental health [23].

5.3. Older adults

In later life, sleep complaints and depressive symptoms are both common and frequently coexist. Perceived poor sleep quality has been associated with major depression among older adults, with coping behavior shaping this relationship [41]. As noted in Section 3.3, however, a 20-year within individual analysis found that although depression and insomnia increased concurrently in older people, neither constituted an additional risk for the other across repeated three-year intervals [9]. Prospective findings from the Canadian Longitudinal Study on Aging further situate sleep alongside physical activity and sedentary behavior as interrelated determinants of depression and psychological distress in ageing populations [43].

5.4. Comorbidities

Sleep disturbances and depression also cluster within somatic disease. In obstructive sleep apnea (OSA), depression and anxiety are highly prevalent [7], and machine learning analyses of NHANES data have explored the role of depression in OSA and identified risk factors for OSA in depressed patients, pointing to a complex, mutually reinforcing relationship [39]. In systemic lupus erythematosus, a metaanalysis confirmed an association between depression and poor sleep quality [21]. Sleep quality has also been shown to mediate the relationship between depression and chronic pain, suggesting that disturbed sleep is part of the mechanistic pathway rather than a parallel complaint [22]. Beyond individual diseases, multidimensional sleep traits have been linked to cardiovascular disease, with depression acting as a mediating factor [42], highlighting the systemic consequences of the sleep–depression cycle.

6. Modifiable behavioral factors

6.1. Social media and smartphone use

Among the behavioral factors that shape contemporary sleep, screen-based technology use is particularly salient. A systematic review with metaanalyses reported that social media use is associated with poorer mental health and disturbed sleep [13], and a separate metaanalysis linked mobile phone addiction with anxiety, depression, impulsivity and poor sleep quality among college students [14]. In adolescents specifically, higher social media use has been associated with poor sleep quality, anxiety, depression and low self-esteem, with nighttime use appearing especially detrimental [26]. These behaviors are highly modifiable and represent plausible targets for both prevention and intervention.

6.2. Outdoor daylight exposure

A contrasting and protective behavioral factor is exposure to natural daylight, which acts as the principal entraining signal for the circadian system. In a large sample of more than 13,000 adults with depression, greater self-reported outdoor daylight exposure was associated with more favorable mental health and sleep correlates [48]. Because time spent outdoors is

closely tied to physical activity and to circadian alignment (Section 4.2), it provides a natural conceptual bridge to the discussion of exercise-based interventions that follows.

7. Physical activity and exercise interventions

Physical activity occupies a central place in this review, both because it is a strongly modifiable behavior and because it acts simultaneously on sleep and on mood the two poles of the cycle described above. From a sports, science, and public health perspective, exercise is therefore a particularly attractive point of intervention.

7.1. Exercise effects on sleep in unipolar depression

The most direct evidence concerns the effect of exercise on sleep within depression itself. A systematic review and network metaanalysis examining unipolar depression concluded that exercise improves sleep in this population, allowing different exercise modalities to be compared with respect to their effects on sleep outcomes [25]. Complementary evidence from the broader sleep-disorders literature indicates that regular exercise can improve both objective parameters such as total sleep time, sleep-onset latency and sleep efficiency and subjective sleep quality across a range of conditions, including insomnia, obstructive sleep apnea and depression [54]. Together, these findings position exercise as a credible non-pharmacological option for the sleep component of depression.

7.2. Mind and body exercise

Beyond conventional aerobic and resistance training, mind-body modalities combine physical movement with attentional and breathing components. A systematic review and meta-analysis focusing on older adults found that mind-body exercise reduces both sleep disturbance and depressive symptoms, suggesting that lower-intensity, accessible forms of activity can also target both poles of the sleep-depression relationship [19]. This is clinically relevant for populations in whom vigorous exercise may be less feasible.

7.3. Physical activity, sedentarism and depression risk

A wider body of evidence links habitual physical activity and its absence with depression risk. Prospective findings from the Canadian Longitudinal Study on Aging show that physical activity, sedentary behavior and sleep jointly shape depression and psychological distress, with sedentarism contributing to less favorable outcomes [43]. Studies of depression subtypes indicate that objectively assessed sleep and physical activity differ across presentations and mediate part of the association between depression and cardiovascular risk factors [36]. Better sleep quality together with higher physical activity levels has been shown to predict lower emotional dysregulation in individuals with major depressive disorder [45].

This pattern is reinforced by review of evidence published in sports and health-oriented journals. Narrative and literature reviews consistently describe physical activity as effective in the prevention and treatment of depressive and anxiety disorders, acting through neuroplastic, inflammatory and metabolic pathways [55,57,58], and as a protective factor against the incidence of depression as well as an adjunctive treatment [56]. Health-education components that promote activity and reduce stigma may further support these effects [59].

7.4. Objectively measured sleep and physical activity

The interpretation of this evidence is strengthened by objective measurement. Accelerometry-based studies allow sleep, physical activity and circadian rhythm domains to be modelled simultaneously, and integrative analyses have related these domains to current and remitted major depression [29]. Objective assessment of sleep and physical activity has likewise been used to characterize depression subtypes and their cardiometabolic correlates [36]. Such

device-based approaches reduce reliance on self-report and provide a more robust basis for recommending activity-based interventions.

8. Other therapeutic approaches

8.1. Sleep-focused interventions improving mental health

If disturbed sleep is a causal contributor to depression, then improving sleep should improve mood. A metaanalysis of randomized controlled trials supports exactly this: interventions that improve sleep quality led to better mental health, including reductions in depression, with larger improvements in sleep associated with larger improvements in mental health [17]. Behavioral sleep interventions in the perinatal context likewise reduce maternal depressive symptoms [23], and narrative reviews emphasize the value of non-pharmacological, sleep-directed strategies in the management of depression [2]. These findings provide a strong rationale for treating sleep as a therapeutic target in its own right.

8.2. Sleep deprivation as treatment for depression

A more paradoxical line of evidence concerns therapeutic sleep deprivation. A systematic review and metaanalysis found that controlled sleep deprivation can produce rapid, albeit typically short-lived, antidepressant effects in some patients [18]. While not a routine treatment, this phenomenon underscores that the relationship between sleep and mood is complex and bidirectional: both insufficient and disturbed sleep, and acute experimental wakefulness, can influence depressive symptoms through partly distinct mechanisms. This complexity should temper any oversimplified “more sleep is always better” interpretation.

9. Clinical implications: anxiety comorbidity and suicide risk

9.1. Anxiety as a co-occurring dimension

Although this review focuses on depression, depression rarely occurs in isolation, and anxiety is its most frequent companion. The mechanisms linking sleep disturbance to anxiety overlap substantially with those described for depression, and several lines of evidence treat the two outcomes jointly. Chronic insomnia and REM sleep instability have been proposed as a shared pathway towards both anxiety and depression [27], and network analyses of sleep quality, anxiety and depression for example among nurses reveal dense interconnections in which sleep items act as bridges between the two symptom clusters [16]; anxiety, chronotype and circadian rhythms likewise co-vary with depressive symptoms in insomnia disorder [10]. Among university and sports students, psychological problems, including anxiety and sleep disturbance, are common and interrelated, underscoring the relevance of this triad in young, education-based populations [60]. Reviews of physical activity likewise address depression and anxiety together, reporting benefits across both domains [55,58]. For clinical practice, this means that screening for disturbed sleep should not be confined to depression but extended to the broader internalizing spectrum.

9.2. Sleep disturbance and suicidal behavior

The most serious clinical implication concerns suicide risk. A systematic review and meta-analysis found that sleep disorders are associated with suicidal behavior in patients with depression, indicating that disturbed sleep is not merely a marker of severity but a potentially actionable risk factor [24]. Consistent with this, poor sleep quality during pregnancy has been linked not only to antepartum depression but also to suicidal ideation [40]. Because sleep complaints are common, readily disclosed and modifiable, they may offer a practical entry point for suicide risk assessment and timely preventive intervention.

10. The impact of sport on sleep

Sleep is increasingly recognized as a performance-relevant outcome in its own right, and the evidence that habitual sport modifies sleep architecture, sleep quality and circadian alignment is robust enough to merit a focused synthesis. The most direct evidence comes from systematic reviews and metaanalysis of exercise interventions. A network metaanalysis conducted in adults with unipolar depression demonstrated that exercise improves sleep across modalities, with effect sizes that remain clinically meaningful even when compared with active control conditions [45]. A more recent comprehensive review extends these findings beyond depression to the broader sleep disorders landscape, documenting improvements in total sleep time, sleep-onset latency, sleep efficiency and subjective sleep quality among individuals with insomnia, obstructive sleep apnea and other sleep pathologies who engage in regular exercise [46]. Together, these reviews establish exercise as a credible, evidence-based intervention for sleep, independently of, and complementary to, its established mood benefits. The benefits of sport are not confined to vigorous training. Lower intensity, attention focused modalities, collectively termed mind-body exercise and including tai chi, yoga and qigong, have been shown to reduce both sleep disturbance and depressive symptoms in older adults, where conventional aerobic protocols may be less feasible [47]. This broadens the relevance of sport to populations who might otherwise be excluded from intervention trials and underscores the role of structured movement, rather than intensity per se, in shaping sleep. Habitual physical activity, and its absence, likewise, shape sleep at the population level. Prospective findings from a large Canadian longitudinal cohort showed that physical activity, sedentary behavior and sleep jointly determine depression and psychological distress, with sedentarism contributing to less favorable sleep and mental health outcomes [35]. In individuals with major depressive disorder, better sleep quality and higher levels of physical activity together predict lower emotional dysregulation, suggesting that sport-related behaviors influence affective regulation partly through sleep [28]. Objective, accelerometry-based studies have further shown that depression subtypes differ in their patterns of sleep and physical activity, and that these patterns partially mediate the association between depression and cardiovascular risk factors [48]. Sport, in other words, does not act as sleep in isolation but as part of an integrated lifestyle pattern with measurable downstream consequences.

Several mechanisms have been proposed to explain how sport influences sleep. Reviews published in sport- and health-oriented journals consistently describe physical activity as acting through neuroplastic, inflammatory and metabolic pathways that converge on both sleep regulation and affective control [49,50,51,52,53]. Regular exercise is associated with anti-inflammatory effects, improved circadian alignment via increased daytime exertion and light exposure, autonomic balance and enhanced thermoregulation, all of which contribute to deeper, more consolidated sleep. The same mechanisms underlie its antidepressant effects, providing a plausible biological explanation for the dual benefit observed across the literature. Methodologically, the integration of sport and sleep research has been advanced by device-based monitoring. Integrative analyses combining accelerometry-derived sleep, physical activity and circadian rhythm domains have related these jointly to current and remitted major depression, demonstrating that objective measurement captures aspects of the sport to sleep relationship invisible to questionnaire-based studies [54]. For sport-science research, this approach allows training load, recovery, and sleep to be modelled as a single physiological system rather than disconnected outcomes.

Finally, evidence from sport-active populations is a reminder that sport is not a panacea. Among undergraduate sports students, psychological problems, including anxiety and sleep disturbance, remain common despite high levels of physical activity, indicating that the quantity of sport alone is insufficient and that quality of sleep, recovery and psychological support must

accompany training [59]. Outdoor activity, which encompasses many forms of sport and is closely tied to daylight exposure and circadian entrainment, has likewise been associated with more favorable sleep and mental health correlates in adults with depression [44]. In sum, sport exerts a measurable, multi-pathway and clinically relevant influence on sleep. This impact is observable across age groups and exercise modalities, is detectable with both subjective and objective measurement, and operates through mechanisms that simultaneously support sleep and mood. From the perspective of sport science and public health, promoting structured physical activity emerges not only as a strategy for fitness and performance but as a foundational intervention for sleep itself, and, through sleep, for broader mental and somatic well-being.

11. Discussion

The evidence synthesized in this review converges on a consistent conclusion: sleep disturbances and depression are linked bidirectionally rather than in a single causal direction. Insomnia predicts the onset, persistence and recurrence of depression [11,20,50], depression in turn degrades sleep [1], and longitudinal, cross-lagged and Mendelian randomization analyses support a genuinely reciprocal and at least partly causal relationship [8,30]. Shared mechanisms REM sleep instability, altered sleep architecture, circadian misalignment, emotional dysregulation, and overlapping neuroanatomical substrates provide a plausible biological basis for this reciprocity [27,31,38].

At the same time, the relationship is not uniform. Its strength and temporal structure appear to vary with age and measurement interval; notably, a 20-year within individual analysis in older adults found concurrent change but no additional cross-lagged risk between the two conditions [9]. Therapeutic sleep deprivation, which can transiently improve mood [18], further illustrates that the association cannot be reduced to a simple “more sleep is better” rule. These nuances should caution against overinterpretation of any single study or design.

From an applied standpoint, the most actionable message concerns modifiable factors. Improving sleep quality leads to measurable improvements in mental health [17], and physical activity acts simultaneously on both poles of the cycle, improving sleep [25,54] and reducing depressive and anxiety symptoms [55,56,57,58,59]. This dual action makes exercise a particularly valuable, low cost and widely accessible intervention, relevant to prevention as well as adjunctive treatment, and consistent with the public health and sports-science orientation of this journal.

Several limitations should be acknowledged. As a narrative review, this work did not apply a systematic search protocol or formal risk of bias assessment, and selection of sources, while broad, was not exhaustive. Much of the underlying evidence is observational, with heterogeneity in how sleep and depression are defined and measured, reliance on self-report in many studies, and variable control of confounders. The directionality and magnitude of effects may also differ across populations, age groups, and comorbidities. These constraints indicate that the conclusions below should be read as a synthesis of converging evidence rather than as definitive causal proof, and they point to clear directions for future research particularly longitudinal, device-based and interventional designs.

12. Conclusions

Sleep disturbances and depression are bidirectionally and mutually reinforcing conditions that share neurobiological, circadian and cognitive-emotional mechanisms. The association is detectable across the lifespan from children and adolescents to perinatal women and older adults and within somatic comorbidities, and it extends to anxiety and to suicide risk. Disturbed sleep should therefore be regarded not as a secondary symptom of depression but as an independent, clinically meaningful target.

Two practical conclusions follow. First, the assessment and treatment of sleep should be integrated into the prevention and management of depression, since improving sleep improves mental health outcomes. Second, physical activity and exercise acting simultaneously on sleep quality and on depressive symptoms represent a safe, accessible and cost-effective means of interrupting the sleep–depression cycle and merit a more prominent place in both clinical practice and public health strategy. Future longitudinal, objectively measured, and interventional studies are needed to refine causal understanding and to optimize activity and sleep-based protocols.

Disclosure

Author's contribution:

Eliza Kuchta: conceptualization, methodology, software, validation, formal analysis, investigation, resources, data curation, writing original draft preparation, writing review and editing, visualization, supervision, project administration;

Kacper Wrzosek: conceptualization, methodology, software, validation, formal analysis, investigation, resources, data curation, writing original draft preparation, writing review and editing, visualization, supervision, project administration;

Maria Leśniak: conceptualization, methodology, software, validation, formal analysis, investigation, resources, data curation, writing original draft preparation, writing review and editing, visualization, supervision;

Kacper Ponikowski: conceptualization, methodology, software, validation, formal analysis, investigation, resources, data curation, writing original draft preparation, writing review and editing, visualization;

Kornelia Kuchta: conceptualization, methodology, software, validation, formal analysis, investigation, resources, data curation, writing original draft preparation, writing review and editing;

Halina Pieciewicz-Szczęsna: formal analysis, investigation, resources, data curation, writing original draft preparation, writing review and editing, visualization, supervision, project administration;

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