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The Role of Sleep Disturbances in Depression and Anxiety: A Literature Review

Aleksandra Kowalczyk (corresponding author)
ORCID: https://orcid.org/0009-0002-9523-2367
E-mail: olakowalczyk2001@wp.pl
Medical University of Silesia in Katowice, Poland
Adrian Pączek
ORCID: https://orcid.org/0009-0009-5151-7556
E-mail: adrjanalan@gmail.com
Zagłębiowski Clinical Hospital in Czeladź, Poland
Paweł Dyczek
ORCID: https://orcid.org/0009-0008-6607-6231
E-mail: dyczek@onet.eu
Wojewódzki Hospital in Bielsko-Biała, Poland
Julia Hofman
ORCID: https://orcid.org/0009-0008-2609-8292
E-mail: juliahofman2001@gmail.com
Medical University of Silesia in Katowice, Poland
Wiktoria Staniszewska
ORCID: https://orcid.org/0009-0002-8964-404X
E-mail: wiktoria.staniszewska@gmail.com
Wojewódzki Hospital in Bielsko-Biała, Poland

Abstract

Background. Sleep disturbances are among the most frequent comorbid symptoms of mood and anxiety disorders. Contemporary evidence indicates they are robust predictors and likely contributory factors in the onset, maintenance, and relapse of these conditions.

Objective. To review and synthesize contemporary evidence on the associations between sleep disturbances and depression and anxiety, focusing on epidemiological data, mechanistic insights, and treatment implications.

Methods. A narrative review was conducted using PubMed and PMC databases, covering the years 2011–2025. Keywords included *sleep disturbance*, *insomnia*, *circadian rhythm*, *depression*, and *anxiety*. Preference was given to meta-analyses, longitudinal studies, and randomized controlled trials (RCTs).

Results. Sleep problems, especially insomnia and short sleep duration, predict the onset of depression and anxiety, with odds ratios of approximately 2–3. Disturbances are bidirectionally linked with mood disorders through mechanisms such as hyperarousal, dysregulated circadian rhythms, altered neurotransmission, and inflammation. Interventions improving sleep—especially cognitive-behavioural therapy for insomnia (CBT-I) and digital CBT-I—produce small to moderate reductions in depressive and anxiety symptoms.

Conclusions. Sleep disturbances are not merely symptoms of psychiatric illness but modifiable risk factors and therapeutic targets. Integrating sleep-focused assessment and intervention into mental health and primary care can improve clinical outcomes and possibly prevent new-onset depression or anxiety.

Keywords: anxiety; circadian rhythm; cognitive behavioural therapy for insomnia; depression; insomnia; sleep disturbance.

1. Introduction

Sleep plays a vital role in regulating emotional and cognitive processes. Chronic insomnia and other sleep problems are highly prevalent in patients with mood and anxiety disorders and predict later affective symptoms [1,2]. Its architecture and timing are controlled by two interdependent systems: the homeostatic drive, which builds pressure for sleep with wakefulness, and the circadian rhythm, which aligns physiological functions to the 24-hour light–dark cycle [3,6]. When these mechanisms become disrupted — through insomnia, irregular schedules, or circadian misalignment — emotional stability and stress resilience deteriorate markedly. Such disturbances are among the most prevalent biological correlates of mood and anxiety disorders [1–4].

Epidemiological studies show that chronic sleep problems, particularly insomnia, affect nearly 90% of individuals with major depressive disorder (MDD) and around half of those with generalized anxiety disorder [1,2]. Yet growing evidence indicates that these disturbances are not merely symptoms of psychiatric illness but significant predictors of its development.

A large meta-analysis by Hertenstein et al. (2019) demonstrated that people with insomnia had more than double the risk of developing depression and over three times the risk of developing anxiety disorders compared to good sleepers [1].

Similarly, Baglioni et al. (2011) reported consistent longitudinal evidence linking insomnia to later onset of depressive symptoms, even after controlling for baseline mood [7].

These findings highlight that sleep disturbances function as early, modifiable risk factors rather than passive correlates of psychological distress.

The association between sleep and mental health appears to emerge early in life. In a comprehensive meta-analysis of cohort studies, Marino et al. (2021) found that poor sleep quality and shorter sleep duration in children and adolescents were significantly associated with subsequent depression, independent of demographic and psychosocial factors [4]. Such results underscore the importance of preventive strategies focusing on sleep hygiene and circadian regularity in youth populations.

At the neurobiological level, circadian rhythm dysregulation has become a central explanatory framework for the link between sleep disturbance and mood disorders. Recent reviews emphasize that alterations in clock genes, melatonin secretion, and light exposure patterns may directly affect neural circuits regulating emotion and reward processing [3,6,20].

de Leeuw et al. (2023) showed that circadian misalignment modulates hypothalamic and limbic pathways involved in affect regulation, predisposing individuals to mood instability [3].

Neuroimaging and molecular studies further reveal that sleep deprivation affects regions such as the amygdala and prefrontal cortex, leading to exaggerated emotional reactivity and reduced top-down control [5,9].

Inflammatory mechanisms may also contribute: chronic insomnia has been linked to elevated interleukin-6 and C-reactive protein levels, which are known to influence neurotransmission and synaptic plasticity relevant to depression [9].

The relationship between sleep and affect is strongly bidirectional. On one hand, depressive or anxious states often lead to difficulties initiating and maintaining sleep, early-morning awakenings, and non-restorative rest. On the other, persistent sleep deprivation or circadian disruption aggravates existing affective symptoms and increases vulnerability to relapse [1,2,5]. Scott et al. (2021) demonstrated through a meta-analysis that interventions improving sleep quality produced moderate yet significant reductions in depressive and anxiety symptoms across diverse clinical populations [5]. This bidirectionality implies that addressing sleep is not only symptom management but also an opportunity for prevention and early intervention.

Clinically, the recognition of sleep disturbance as a transdiagnostic mechanism has reshaped approaches to mental-health care. Traditional psychiatric models often treated insomnia as secondary to mood disorders, but modern frameworks advocate for parallel and integrated treatment. Evidence-based therapies, such as cognitive behavioral therapy for insomnia (CBT-I) and its digital adaptations, have proven effective in alleviating both sleep and affective symptoms [10,11,16,17].

Such approaches reflect a broader paradigm shift: rather than viewing sleep as a passive outcome of emotional well-being, contemporary research positions it as a central determinant of mental health.

In summary, sleep disturbances represent a biologically and clinically meaningful bridge between physiological regulation and psychological resilience. Their high prevalence, predictive value, and treatability make them an essential target for prevention and therapy in depression and anxiety. Understanding the mechanisms underlying this relationship—ranging from circadian misalignment to inflammation—may lead to more effective and holistic approaches to mental-health care [1–3,5,6,9,10,20].

2. Methods

This paper is a narrative literature review summarizing current evidence on the relationship between sleep disturbances and depression and anxiety. The review covered studies published

between 2011 and 2025, with inclusion of key earlier meta-analyses on insomnia and mental health [1,2,7].

A structured search was performed in PubMed and PubMed Central (PMC) from January 2024 to October 2025.

Search terms included: “sleep disturbances AND depression”, “insomnia AND anxiety”, “circadian rhythm AND mood disorders”, and “CBT-I AND depression/anxiety”.

Only peer-reviewed English-language studies were considered. To ensure comprehensive coverage, reference lists of relevant reviews were also screened manually [3,5,6].

Included studies met at least one of the following criteria:

Systematic reviews, meta-analyses, or large cohorts assessing associations between sleep disturbances and depression or anxiety [1,4,5,12,13].

Randomized controlled trials or meta-analyses evaluating interventions focused on sleep, such as cognitive behavioral therapy for insomnia (CBT-I) and digital CBT-I [10,11,16–19].

Mechanistic or biological studies examining circadian rhythm disruption, inflammatory markers, or neural mechanisms linking sleep and mood [3,6,9,20].

Exclusion criteria comprised non-peer-reviewed works, conference abstracts, and studies unrelated to psychiatric outcomes.

Approximately 80 papers were screened, of which 20 high-quality publications were included for analysis. Extracted data covered study design, population, sleep and mood measures, and key results.

Meta-analyses such as those by Hertenstein et al. (2019), Baglioni et al. (2011), and Marino et al. (2021) served as the primary epidemiological sources [1,4,7].

Mechanistic insights were derived from de Leeuw et al. (2023) and Ballesio (2023) [3,9], while evidence on therapeutic outcomes was informed by recent CBT-I and digital CBT-I studies [10,11,16–19].

Due to the narrative design, no formal risk-of-bias scoring was performed. Instead, conclusions were guided by the hierarchy and consistency of evidence, emphasizing convergent findings across epidemiological, biological, and interventional research [5,6].

3. Results

1. Sleep disturbances as predictors of depression and anxiety

Epidemiological data consistently demonstrate that sleep disturbances, particularly insomnia and abnormal sleep duration, are strong predictors of subsequent depression and anxiety.

In a large meta-analysis including over 170 000 participants, Hertenstein et al. (2019) found that individuals with insomnia symptoms had a more than twofold increased risk of developing depression (odds ratio [OR] = 2.83) and a more than threefold increased risk of developing anxiety disorders (OR = 3.23) compared with individuals without insomnia [1].

Similarly, Baglioni et al. (2011) confirmed that insomnia predicted later onset of depressive episodes across multiple longitudinal cohorts, independent of baseline mood or anxiety levels [7]. The association was bidirectional, but the predictive pathway from insomnia to depression appeared stronger than the reverse.

Age-specific analyses reveal that the relationship emerges early in life. In a systematic review of 22 cohort studies, Marino et al. (2021) reported that insufficient or poor-quality sleep in children and adolescents significantly increased the risk of later depressive symptoms, even after controlling for baseline affective status and psychosocial factors [4].

In adults, sleep quantity also follows a U-shaped pattern in relation to mood outcomes. Li et al. (2023) demonstrated in a dose–response meta-analysis that both short sleep (< 6 hours per night) and long sleep (> 9 hours) were associated with higher risk of depression among middle-aged and older adults [12].

Recent work by Zhang et al. (2024) extended these findings, showing that short sleep duration predicted incident mental disorders, including depression and anxiety, across multiple populations [13].

Together, these results indicate that maintaining a regular and sufficient sleep pattern is essential for emotional stability, whereas chronic insomnia or irregularity may act as an early marker of affective vulnerability.

2. Mechanistic pathways

The connection between disturbed sleep and mood dysregulation is supported by converging biological evidence.

At the circadian level, disruptions in internal timing mechanisms contribute to altered emotional processing and stress reactivity. de Leeuw et al. (2023) and Dollish et al. (2024) highlighted that desynchronization of circadian rhythms leads to impaired regulation of the hypothalamic–pituitary–adrenal (HPA) axis, resulting in abnormal cortisol secretion and heightened emotional instability [3,6].

Misalignment between internal biological clocks and environmental light–dark cycles has also been shown to influence mood through changes in melatonin secretion and clock-gene expression, thereby affecting neurotransmission within limbic structures [3,20].

Physiological hyperarousal, a core feature of insomnia, mirrors the heightened sympathetic activity observed in anxiety disorders and may explain their frequent overlap [2]. Neurobiological studies indicate that both insomnia and depression involve imbalances in monoaminergic systems—particularly serotonin, dopamine, and noradrenaline—as well as reduced levels of brain-derived neurotrophic factor (BDNF), which are associated with diminished neural plasticity [5,9].

Sleep architecture alterations further support these links: patients with depression typically exhibit decreased slow-wave sleep and increased rapid eye movement (REM) density, both markers of dysregulated emotional processing [9].

Inflammatory mechanisms provide another pathway connecting sleep and mood. Ballesio (2023) proposed that chronic sleep deprivation and insomnia activate pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-α, which in turn contribute to depressive symptomatology via neuroimmune signaling [9].

Collectively, these findings suggest that sleep and mood share common neurobiological substrates, and that persistent disruption of circadian and homeostatic processes can directly influence emotional regulation and vulnerability to psychiatric disorders.

3. Sleep-focused interventions and mental-health outcomes

A growing body of evidence confirms that improving sleep quality exerts measurable benefits on depressive and anxiety symptoms. In a meta-analysis of 72 interventional studies, Scott et al. (2021) demonstrated that sleep improvement interventions produced medium-sized reductions in depressive symptoms (Hedges $g = 0.63$) and smaller but significant effects on anxiety (Hedges $g = 0.41$) [5].

Cognitive-behavioural therapy for insomnia (CBT-I) remains the most effective non-pharmacological treatment, showing sustained improvements even in patients with comorbid psychiatric conditions [16]. A network meta-analysis by Benz et al. (2020) confirmed CBT-I's superiority over pharmacological and relaxation-based approaches in improving both sleep parameters and mood outcomes [11].

Digital adaptations of CBT-I (dCBT-I) have expanded accessibility to evidence-based therapy. Meta-analyses by Lee et al. (2023) and Lin et al. (2023) reported that dCBT-I significantly reduced insomnia severity and simultaneously alleviated symptoms of depression and anxiety across diverse populations [10,17].

These results are reinforced by a large systematic review of fully automated dCBT-I programs conducted by Hwang et al. (2025), which demonstrated robust and sustained improvements in sleep quality and mental-health outcomes [19].

Beyond cognitive-behavioural approaches, circadian-based interventions—including morning bright-light therapy, regular wake-time scheduling, and melatonin supplementation—have shown additional benefits in stabilizing mood and sleep–wake cycles [6,20]. Such therapies are particularly effective in individuals with delayed sleep–wake phase disorder, while in non-circadian insomnia their mood effects appear smaller and more protocol-dependent [15].

Overall, the evidence indicates that targeting sleep through behavioural and circadian strategies not only improves sleep quality but also provides meaningful reductions in depressive and anxiety symptoms, supporting sleep health as a core component of mental-health care.

Table 1. Summary of key meta-analyses and recent reviews linking sleep disturbances with depression and anxiety

Study (Year)	Design / Population	Exposure / Intervention	Outcome(s)	Key Findings	Source / PMID
Hertenstein et al., 2019	Systematic review and meta-analysis (n = 170 000+)	Insomnia symptoms	Incidence of depression, anxiety	Insomnia doubled risk of depression (OR = 2.83) and tripled risk of anxiety (OR = 3.23).	*Sleep Med Rev.* 2019;43:96–105. PMID 30537570
Baglioni et al., 2011	Meta-analysis of longitudinal cohorts	Baseline insomnia	Onset of depressive episode	Insomnia predicted depression independent of baseline mood; stronger direction from	*J Affect Disord.* 2011;135:10–19. PMID 21300408

				insomnia → depression.	
Marino et al., 2021	Meta-analysis of 22 cohort studies (children/adolescents)	Poor sleep quality / short duration	Later depressive symptoms	Poor sleep associated with 58% higher risk of depression after adjustment for psychosocial factors.	*JAMA Netw Open* 2021;4(3):e212338. PMID 33749768
Li et al., 2023	Dose–response meta-analysis	Night-sleep duration	Depression incidence	Both short (<6 h) and long (>9 h) sleep linked to higher depression risk; U-shaped association.	*Front Physiol.* 2023;14:1085091. PMID 36935736
Zhang et al., 2024	Meta-analysis of prospective studies	Short sleep duration	Incident mental disorders	Short sleep increased risk of depression/anxiety across populations.	*J Affect Disord.* 2024;350:43–55. PMID 37642884
de Leeuw et al., 2023	Narrative/systematic review	Circadian misalignment	Mood dysregulation	Desynchronization of circadian rhythms impairs HPA-axis regulation; elevates cortisol,	*Front Neurosci.* 2023;17:1204382. PMID 37678570

				emotional instability.	
Ballesio, 2023	Review	Chronic insomnia / sleep deprivation	Inflammatory and neurobiological markers	Elevated IL-6 and CRP link sleep loss to depressive symptoms; supports neuroimmune hypothesis.	*Brain Behav Immun Health* 2023;31:100647. PMID 37408788
Scott et al., 2021	Meta-analysis of 72 RCTs	Sleep-improvement interventions (CBT-I, light therapy, etc.)	Depressive and anxiety symptoms	Medium effect on depression (g = 0.63), smaller but significant on anxiety (g = 0.41).	*Sleep Med Rev.* 2021;60:101557. PMID 34607184
Benz et al., 2020	Network meta-analysis	CBT-I vs. pharmacological / relaxation	Sleep and mood outcomes	CBT-I superior to other interventions; sustained benefits at follow-up.	*Sleep Med Rev.* 2020;52:101306. PMID 32777632
Lee et al., 2023	Systematic review and meta-analysis	dCBT-I	Depression, anxiety reduction	Digital CBT-I significantly reduced insomnia severity and	*NPJ Digit Med* 2023;6:64. PMID 36966184

	(digital CBT-I)			affective symptoms.	
Lin et al., 2023	Meta-analysis of RCTs	Digital CBT-I	Insomnia + comorbid depression	Improvement in both insomnia and depressive symptoms; effect comparable to face-to-face CBT-I.	*J Clin Sleep Med.* 2023;19(7):1325–1337. PMID 37927792
Hwang et al., 2025	Systematic review and meta-analysis	Fully automated dCBT-I	Sleep quality and mental-health outcomes	Automated dCBT-I significantly improved sleep and reduced depression/anxiety symptoms.	*NPJ Digit Med.* 2025;8(1):157. PMID 40075149

Abbreviations: OR – odds ratio; HPA – hypothalamic–pituitary–adrenal; CBT-I – cognitive-behavioural therapy for insomnia; dCBT-I – digital CBT-I; IL-6 – interleukin-6; CRP – C-reactive protein; g – Hedges' g (standardized mean difference).

4. Discussion

The evidence reviewed in this paper confirms that sleep disturbances play a bidirectional and clinically meaningful role in the onset and course of depression and anxiety, and that they are consistent predictors of later depressive and anxiety symptoms. However, most of the available data are observational (prospective cohorts, meta-analyses) and therefore cannot fully exclude residual confounding or shared vulnerability factors. Signals suggesting causality come mainly from intervention studies showing that improving sleep leads to reductions in depressive and anxiety symptoms, but these effects are small to moderate and not universal. Sleep disturbances should therefore be viewed as contributory and modifiable factors, rather than as single,

sufficient causes of depression or anxiety. Insomnia and short sleep duration consistently predict the development of depressive and anxiety disorders, while disturbances in circadian timing exacerbate existing symptoms and increase the risk of relapse [1,3,6,12]. This dual relationship supports the view of sleep as a transdiagnostic risk factor, operating across diagnostic boundaries and influencing multiple dimensions of emotional health.

1. Pathophysiological overlap

The connection between disordered sleep and affective dysregulation can be explained by several interrelated biological mechanisms.

Circadian dysregulation is one of the most robustly documented pathways. Misalignment between endogenous circadian rhythms and the external light–dark cycle alters the expression of core clock genes and disrupts melatonin secretion, leading to impaired synchronization of neural and endocrine systems responsible for mood regulation [3,5,6,20]. Individuals with delayed sleep–wake phase disorder, in particular, demonstrate higher depressive symptom severity and poorer stress adaptation [15].

A second mechanism involves hyperarousal and stress reactivity. Insomnia and anxiety share heightened sympathetic activity and excessive activation of the hypothalamic–pituitary–adrenal (HPA) axis, reflected in elevated evening cortisol and persistent physiological tension [2,6]. These factors impair restorative sleep and perpetuate anxiety and rumination, creating a self-reinforcing cycle of emotional and physiological hyperactivation.

Inflammatory signaling also contributes to the link between poor sleep and mood disturbance. Chronic sleep loss and fragmented sleep trigger immune activation, with increased levels of C-reactive protein and interleukin-6 correlating with depressive symptom severity [9]. Proinflammatory cytokines can influence monoamine metabolism and hippocampal neurogenesis, providing a plausible biological bridge between insomnia and depression. These biomarkers are also elevated in depression itself and in common comorbidities (e.g. obesity), so they should be interpreted as one of several interacting pathways, not as a standalone explanation.

Finally, neurotransmitter imbalance and reduced neuroplasticity play an important role. Sleep deprivation has been shown to decrease central serotonin availability and reduce brain-derived neurotrophic factor (BDNF) expression, both crucial for maintaining emotional regulation and cognitive flexibility [5]. Functional neuroimaging studies demonstrate reduced prefrontal inhibitory control and increased amygdala reactivity in sleep-deprived individuals—patterns closely resembling those observed in mood and anxiety disorders [3,9].

Together, these overlapping mechanisms explain why sleep disturbance not only accompanies but also precedes and sustains affective pathology. Addressing sleep therefore represents a biologically grounded approach to prevention and treatment.

2. Clinical implications

From a therapeutic standpoint, integrating sleep-focused interventions into psychiatric and primary care practice offers tangible benefits.

Cognitive-behavioural therapy for insomnia (CBT-I) is a first-line, evidence-based intervention that improves both sleep and emotional outcomes [11,16]. Importantly, its digital adaptations (dCBT-I) provide similar efficacy while reducing barriers to access, making them feasible for large-scale implementation, including in primary-care settings [10,17–19].

CBT-I and dCBT-I address maladaptive cognitions and behaviours that perpetuate insomnia, leading to sustained improvement in mood and anxiety symptoms. Meta-analytic data suggest that even partial improvements in sleep quality are associated with clinically significant reductions in depressive scores [5,10].

Beyond behavioural treatments, circadian-based strategies—such as morning bright-light exposure, consistent wake times, and melatonin administration—help restore alignment between biological rhythms and environmental cues [6,20]. These interventions can enhance the effectiveness of psychotherapy and pharmacotherapy by stabilizing daily physiological patterns that support emotional regulation.

For general practitioners and psychiatrists, early recognition of insomnia, hypersomnia, or irregular sleep timing should prompt proactive screening for depressive and anxiety symptoms. Incorporating structured sleep assessment tools (e.g., the Pittsburgh Sleep Quality Index) into routine visits may facilitate early intervention and reduce chronicity of affective disorders.

3. Limitations and future directions

Despite substantial progress, several gaps remain in the current literature. The definition of "sleep disturbance" varies considerably between studies, ranging from subjective insomnia symptoms to objective polysomnographic findings, which limits comparability of results [1,5,7]. Similarly, outcome measures for mood and anxiety are not always standardized, making effect size estimation inconsistent across trials.

Longitudinal studies with extended follow-up are still limited, and few have examined how specific dimensions of sleep (timing, architecture, variability) interact with particular symptom clusters. Future research should focus on integrating multimodal approaches—combining

actigraphy, hormonal profiles, inflammatory biomarkers, and neuroimaging—to clarify mechanistic pathways [3,6,9,20].

There is also a need for pragmatic trials assessing sleep-focused interventions in real-world primary-care settings, where insomnia and mild mood disturbances are most frequently encountered. Evaluating cost-effectiveness, adherence, and long-term relapse prevention would provide valuable insights for implementing these strategies into routine mental-health care [10,17–19].

Finally, advancing personalized sleep medicine—matching interventions to individual circadian profiles or biological markers—may enhance treatment outcomes and open new preventive avenues in psychiatry.

4. Summary

In summary, sleep disturbances are not secondary symptoms but central components of affective disorders, contributing to both their onset and persistence.

They interact with circadian, stress, inflammatory, and neuroplastic systems, forming a complex but modifiable network of vulnerability.

Addressing sleep through evidence-based behavioural and circadian interventions should be regarded as a core element of modern depression and anxiety management, both in psychiatric care and in primary practice.

5. Conclusions

Sleep disturbances represent a key and modifiable factor in the development and persistence of depression and anxiety. Consistent evidence from longitudinal and meta-analytic studies shows that insomnia and circadian disruption increase the risk of mood and anxiety disorders and worsen their clinical course [1,3,5,7,12].

Routine assessment of sleep quality should therefore form an essential part of psychiatric and primary-care evaluation.

Evidence-based treatments, particularly cognitive-behavioural therapy for insomnia (CBT-I) and digital CBT-I, have proven effective in improving both sleep and emotional outcomes, while circadian-based strategies such as light therapy and regular wake scheduling support long-term stability [10,11,16–20].

Integrating these interventions into standard care may enhance prognosis, improve quality of life, and reduce recurrence or new onset of affective disorders.

In summary, addressing sleep is not an adjunct to mental-health treatment but a fundamental component of effective prevention and therapy for depression and anxiety.

Disclosure and Declarations

Authors contribution:

Conceptualization: Aleksandra Kowalczyk, Adrian Pączek

Methodology: Julia Hofman, Aleksandra Kowalczyk

Formal analysis: Adrian Pączek, Wiktoria Staniszewska

Investigation: Aleksandra Kowalczyk, Paweł Dyczek

Writing- Rough Preparation: Adrian Pączek, Aleksandra Kowalczyk

Writing- Review and Editing: Wiktoria Staniszewska, Julia Hofman

Visualisation: Wiktoria Staniszewska, Paweł Dyczek

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