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Sleep Disturbances in Psoriatic Arthritis: A Bidirectional Link Between Inflammation, Pain, and Disease Activity

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Abstract

Background. Psoriatic arthritis (PsA) is a chronic immune-mediated inflammatory disease associated with substantial musculoskeletal disability and multiple systemic comorbidities. Among these, sleep disturbances have emerged as an important yet frequently overlooked contributor to disease burden. Poor sleep in PsA is driven by a complex interplay of chronic inflammation, pain, fatigue, psychological comorbidities, and circadian rhythm dysregulation, while sleep impairment itself may further amplify inflammatory activity and worsen clinical outcomes.

Aim. To provide a comprehensive overview of current evidence on sleep disturbances in PsA, focusing on their epidemiology, pathophysiological mechanisms, clinical manifestations, assessment methods, impact on disease burden, and current management strategies.

Material and Methods. A narrative review of the literature was performed using PubMed and Scopus. PubMed was searched (2020–2026) using the terms "psoriatic arthritis" AND "sleep disorders", while Scopus was searched (2021–2026) using "sleep" AND "psoriatic arthritis". Following screening and eligibility assessment, 59 relevant publications, including original studies and review articles, were included. Additional studies identified through manual reference screening were incorporated when relevant.

Results. Sleep disturbances affect a large proportion of patients with PsA, with poor sleep quality reported in up to 84% of patients and overall sleep disorders affecting approximately 30–70%. Insomnia, obstructive sleep apnea, restless legs syndrome, excessive daytime sleepiness, and circadian rhythm disturbances were the most frequently reported conditions. Sleep impairment was consistently associated with increased pain, fatigue, anxiety, depression, reduced quality of life, impaired physical functioning, and higher disease activity. Current evidence supports a bidirectional relationship between sleep disturbances and systemic inflammation, mediated by pro-inflammatory cytokines, neuroendocrine dysregulation, and altered sleep architecture. Biologic therapies targeting inflammatory pathways appear to

improve sleep quality, although objective sleep assessment remains underutilized in clinical research.

Conclusions. Sleep disturbances represent a highly prevalent but underrecognized comorbidity in PsA and contribute substantially to disease burden and reduced quality of life. Their multifactorial pathogenesis highlights the need for a multidisciplinary approach that integrates routine sleep assessment into standard PsA management. Future studies employing standardized and objective sleep measures are needed to optimize screening strategies and develop targeted interventions aimed at improving both sleep and overall disease outcomes.

Keywords: psoriatic arthritis, sleep disorders

1. Introduction

Psoriatic arthritis (PsA) is a chronic, immune-mediated inflammatory disease that affects both peripheral joints and the axial skeleton, often occurring in association with psoriasis. Beyond musculoskeletal symptoms, PsA is increasingly recognized as a systemic condition with broad impacts on patients' overall health and quality of life. Among the most frequently reported comorbidities are sleep disturbances, which affect a substantial proportion of individuals with PsA [1, 2, 3].

Sleep disturbances are highly prevalent in PsA, affecting approximately 30-70% of patients, depending on disease activity, assessment method, and comorbid conditions [4, 5]. Sleep disorders in this population are multifactorial and may result from a complex interplay of chronic pain, joint stiffness, fatigue, psychological comorbidities such as anxiety and depression, and systemic inflammation. In addition, circadian rhythm disruption and pro-inflammatory cytokines have been suggested to contribute to impaired sleep regulation. Poor sleep quality, in turn, may exacerbate disease activity, increase pain perception, and negatively affect functional status, creating a bidirectional relationship between sleep disturbances and PsA severity [6, 7].

Despite growing awareness of this association, sleep disorders in PsA remain underdiagnosed and insufficiently addressed in routine clinical practice. There is a need for a better understanding of their prevalence, underlying mechanisms, and clinical consequences,

as well as for effective screening and management strategies. Therefore, this paper aims to review current knowledge on sleep disorders in PsA, with particular emphasis on their pathophysiology, clinical presentation, and impact on disease outcomes.

2. Methodology

This narrative review was conducted to summarize evidence on sleep disturbances in PsA. A structured search was performed in PubMed and Scopus. PubMed (2020-2026) using ("psoriatic arthritis" AND "sleep disorders") yielded 31 records, and Scopus (2021-2026) using ("sleep" AND "psoriatic arthritis") identified 183 records. After screening for relevance, 59 studies were included in the final analysis (14 from PubMed and 45 from Scopus). Additional relevant publications identified through manual search were also included, covering sleep quality, obstructive sleep apnea, and related conditions in PsA. Studies were selected based on relevance to sleep outcomes in PsA and psoriasis, with emphasis on recent original research and reviews (2020-2026). The review also explored emerging evidence on the bidirectional relationship between sleep disturbances and systemic inflammation in PsA.

3. Epidemiology

Sleep disturbances are a frequent comorbidity in patients with PsA, although reported prevalence rates vary considerably across studies, largely due to differences in definitions, diagnostic criteria, and assessment methods. Broadly defined sleep disturbances, encompassing impaired nocturnal rest, insomnia symptoms, and reduced subjective sleep quality, have been reported in 22-46.6% of patients with PsA [8, 9, 10]. However, when sleep quality is assessed using the Pittsburgh Sleep Quality Index (PSQI), the most commonly applied instrument in this population, poor sleep quality (defined as a PSQI score $\geq$6) is identified in as many as 61.5-84% of patients [4, 5, 11, 12, 13]. Importantly, poor sleep quality appears to be more common in PsA than in psoriasis without joint involvement, suggesting that the musculoskeletal manifestations of the disease may contribute substantially to sleep impairment [5, 14].

Several specific sleep disorders have also been investigated in PsA. Sleep-related breathing disorders have been reported in approximately 29.4% of patients, whereas the prevalence of obstructive sleep apnea (OSA) ranges widely from 0% to 50% across studies [10, 15, 16]. Excessive daytime sleepiness (EDS) was observed in 17.6% of patients in the study by Petzinna et al. Notably, this study conducted in newly diagnosed PsA patients found no significant differences in the prevalence of sleep-related breathing disorders or daytime sleepiness compared with healthy controls and patients with newly diagnosed rheumatoid

arthritis (RA) [15]. Nevertheless, population-based data suggest a potential long-term association between PsA and sleep apnea. In the study by Egeberg et al., patients with PsA had a significantly increased incidence of sleep apnea compared with the general population (incidence rate ratio 1.75; 95% CI 1.35-2.26) [17].

Restless legs syndrome (RLS) has also been described in patients with PsA, although prevalence estimates are highly heterogeneous, ranging from nearly 0% to 64%. Despite this variability, studies reporting RLS generally indicate a higher frequency among patients with PsA than among those with psoriasis alone. Furthermore, Sandikci et al. demonstrated that moderate and severe forms of RLS were significantly more common in PsA, suggesting a greater burden of clinically relevant symptoms in this population [18].

Taken together, the available evidence indicates that sleep disturbances are highly prevalent in PsA and may occur more frequently than in patients with cutaneous psoriasis alone. While the marked heterogeneity of prevalence estimates limits direct comparisons between studies, the consistently high rates of poor sleep quality and the observed associations with conditions such as sleep apnea and RLS support the notion that sleep impairment represents an important yet often underrecognized aspect of PsA. These findings further suggest that routine assessment of sleep-related symptoms may provide valuable information for the comprehensive management of patients with PsA.

4. Pathophysiology

4.1 The Role of Inflammatory Processes and Pro-Inflammatory Cytokines in Sleep Disturbances

Under physiological conditions, particularly during the non-rapid eye movement (NREM) phase of sleep, modulation of the immune response and controlled secretion of inflammatory mediators occur. In PsA, chronic activation of the immune system leads to persistently elevated levels of pro-inflammatory cytokines, which affect both peripheral tissues and structures of the central nervous system responsible for regulating the sleep-wake cycle [5].

Among the mediators of the inflammatory response, tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6) play a particularly important role in the pathogenesis of sleep disturbances. These cytokines are key components of the inflammatory cascade responsible for the development and persistence of inflammatory changes in PsA, while also participating in the regulation of sleep processes [5, 19]. TNF-α is involved in the physiological regulation of

NREM sleep; however, its chronically elevated levels lead to alterations in sleep architecture, an increased number of nocturnal awakenings, and greater daytime sleepiness [19, 20]. The significance of this cytokine is supported by clinical observations showing that treatment with TNF-α inhibitors not only reduces disease activity but also improves sleep quality and decreases fatigue [5, 20].

Similarly, IL-6 is closely associated with the regulation of the sleep-wake rhythm. In patients with chronic inflammatory diseases, elevated IL-6 levels correlate with poorer sleep quality, more frequent nocturnal awakenings, and increased fatigue [21, 22]. Consequently, chronic inflammation may result in prolonged sleep latency, a higher number of awakenings, and reduced sleep efficiency.

However, these disturbances are not merely a consequence of disease activity. It has been demonstrated that even short-term sleep deprivation leads to increased production of pro-inflammatory cytokines, including TNF-α, IL-1β, and IL-6, which may further exacerbate the inflammatory process and PsA activity [21, 23]. This creates a vicious cycle in which chronic inflammation contributes to poor sleep quality, while sleep disturbances enhance the production of inflammatory mediators and sustain disease activity.

4.2. Chronic Pain and Its Impact on Sleep Architecture

Pain is one of the predominant symptoms of PsA and is likely the most important direct factor responsible for impaired sleep quality in these patients. Pain symptoms result from inflammation of peripheral joints, involvement of tendon and ligament insertions (enthesitis), as well as inflammatory changes affecting the spine and sacroiliac joints [24]. Pain in PsA exhibits a complex nature, combining features of nociceptive, inflammatory, and neuropathic pain. Its circadian pattern is characterized by increased intensity during the night and early morning hours, which directly disrupts normal sleep architecture [5].

The impact of chronic pain on sleep architecture is primarily manifested by a reduction in deep NREM sleep (stage N3), an increased number of microarousals, shortened total sleep time, and a decrease in rapid eye movement (REM) sleep [1, 23, 25]. Of particular importance is the reduction of slow-wave sleep, which is essential for tissue regeneration and restorative physiological processes. Sleep fragmentation is often triggered by sudden increases in pain stimuli during nocturnal changes in body position, resulting in numerous awakenings, many of which may remain unrecognized by the patient.

A characteristic phenomenon observed in polysomnography of patients with chronic pain is the alpha-delta sleep pattern, which involves the intrusion of fast alpha waves (8-12 Hz)

into slow-wave delta sleep (0.5-4 Hz). First described by Moldofsky in patients with fibromyalgia, this phenomenon has also been reported in individuals with PsA and is considered a marker of impaired restorative function of deep sleep. The consequences include a subjective feeling of non-restorative sleep, increased morning fatigue, and increased pain sensitivity, all of which further perpetuate the vicious cycle of pain and insomnia [26].

4.3. Hypothalamic-Pituitary-Adrenal Axis Dysregulation

The hypothalamic-pituitary-adrenal (HPA) axis is a key neuroendocrine system regulating the stress response, immune function, and circadian rhythms. Under physiological conditions, cortisol secretion follows a distinct circadian pattern, reaching its lowest levels during the night and increasing in the morning, which enables the transition from sleep to wakefulness. In addition, cortisol exerts important anti-inflammatory effects by suppressing the production of pro-inflammatory cytokines [27].

In patients with PsA, chronic inflammation and persistent pain may disrupt HPA axis function. Pro-inflammatory cytokines, particularly IL-6 and IL-1β, stimulate the release of corticotropin-releasing hormone (CRH) and activate the HPA axis, leading to alterations in cortisol secretion and a flattening of its normal circadian rhythm [28]. As a result, physiological feedback mechanisms may become impaired, contributing to both persistent inflammation and sleep disturbances.

Chronic HPA axis activation is associated with difficulties initiating and maintaining sleep. Elevated evening levels of cortisol and CRH promote a state of hyperarousal, which increases nocturnal awakenings and reduces overall sleep quality [27]. Consequently, HPA axis dysregulation represents an important mechanism linking chronic inflammation with sleep disturbances in patients with PsA [23].

5. Sleep disorders

5.1. Insomnia

Insomnia is the most commonly diagnosed sleep disorder among patients with PsA. In a recent meta-analysis by Grant et al., which included 36 studies, the prevalence of self-reported sleep problems among patients with PsA ranged from 30% to 85%, while approximately 73% of patients demonstrated significantly impaired sleep quality [29]. In the study by Toledano et al., as many as 63% of patients with PsA exhibited poor sleep quality as assessed by the PSQI, with the most prominent disturbances involving reduced sleep duration, frequent awakenings, and prolonged sleep latency [30].

Subclinical nociceptive stimuli generated by peripheral inflammation lead to a pathological increase in the density of fast alpha and beta on electroencephalography (EEG) activity during NREM sleep. Consequently, patients subjectively perceive their sleep as shallow, fragmented, and entirely lacking restorative properties [31, 32]. The severity of insomnia shows a strong and independent association with levels of anxiety and depression, while chronic sleep deprivation alters pain perception through mechanisms of central sensitization, markedly lowering the threshold for sensory stimuli during the daytime [33].

As a result, patients with PsA suffering from insomnia tend to exhibit higher clinical disease activity scores and a poorer response to treatment with disease-modifying antirheumatic drugs [1, 34].

5.2. Restless Legs Syndrome

RLS is a neurological disorder characterized by unpleasant sensations in the lower limbs and an irresistible urge to move them, particularly during periods of rest and in the evening hours. These symptoms lead to difficulties falling asleep, frequent awakenings, and reduced sleep quality.

In the study by Sandikci and colleagues, the prevalence of RLS was significantly higher in patients with PsA than in patients with cutaneous psoriasis without joint involvement and in healthy controls. RLS was diagnosed in 64.0% of patients with PsA, compared with 20.0% of patients with psoriasis and 14.0% of individuals in the control group ($p < 0.001$). Furthermore, moderate and severe forms of RLS were considerably more common among patients with PsA (68.7%) than among patients with psoriasis (30.0%) and were not observed in the control group [18].

Regression analysis demonstrated an independent association between greater RLS symptom severity and poorer sleep quality, as assessed by the PSQI ($\beta = 0.269$; $p = 0.002$), higher levels of fatigue measured using the Fatigue Severity Scale ($\beta = 0.243$; $p = 0.003$), worse physical functioning evaluated with the SF-36 Health Survey ($\beta = 0.242$; $p = 0.004$), and more severe depressive symptoms assessed using the Beck Depression Inventory ($\beta = 0.177$; $p = 0.036$) [18].

5.3. Obstructive sleep apnea

Sleep-disordered breathing (SDB) encompasses a spectrum of conditions including primary snoring, OSA, central sleep apnea, Cheyne-Stokes respiration, and sleep-related

hypoventilation disorders. In the context of PsA, the most frequently reported form of SDB is OSA [35].

Sleep-related breathing disorders were reported in 29.4% of patients in the study conducted by Petzinna et al. The authors used the Epworth Sleepiness Scale (ESS) and respiratory polygraphy (RPG). However, the study included a small sample size and was limited to newly diagnosed, untreated patients with PsA. The authors emphasized the usefulness of the routine use of the ESS and RPG as simple and cost-effective screening tools in patients with newly diagnosed inflammatory arthritis [15].

According to a recent meta analysis, OSA affects approximately half of the global population with high body mass index, with increasing age and male sex identified as major risk factors [36]. An older population-based study in Switzerland reported a prevalence of OSA, affecting 23% of women and 50% of men [37]. In a population based study by Egeberg et al., patients with PsA had an incidence rate ratio for sleep apnea of 1.75 (95% CI 1.35-2.26) compared with the reference population [17].

The prevalence of OSA varies substantially across studies, ranging from 0 to 50%, with the lowest estimate reported in a cohort of newly diagnosed patients [10, 15, 16].

5.4. Circadian Rhythm Disorders

The circadian rhythm is an endogenous mechanism that regulates cyclical physiological processes, including sleep-wake patterns, hormone secretion, body temperature, metabolism, and immune system activity. Its proper functioning depends on synchronization between the internal biological clock and environmental cues, particularly the light-dark cycle [20].

In recent years, growing attention has been directed toward the relationship between chronic inflammation and circadian rhythm disruption. It has been demonstrated that pro-inflammatory cytokines central to the pathogenesis of PsA, including TNF-α, IL-17, IL-23, and IL-6, may influence the expression and function of genes responsible for circadian rhythm regulation [38, 39].

A key factor influencing the clinical manifestation of circadian rhythm sleep-wake disorders in PsA is the nocturnal and early morning intensification of pain and joint stiffness. These symptoms are closely correlated with the physiological fluctuations of pro-inflammatory cytokines such as TNF-α, IL-17, and IL-23. These cytokines exhibit peak plasma concentrations during the early morning hours. In patients with active PsA, however, this nocturnal amplitude becomes pathologically amplified, resulting in a marked reduction in the pain threshold during the night and destabilization of normal sleep architecture [40].

Despite the growing number of scientific reports, data specifically addressing circadian rhythm disorders in PsA remain limited. Most available studies have been conducted in populations of patients with cutaneous psoriasis. Nevertheless, due to the shared immunological and metabolic mechanisms underlying both conditions, it is assumed that similar relationships are also present in patients with PsA [38, 39]. Therefore, assessment of sleep quality and potential circadian rhythm disturbances may represent an important component of comprehensive patient care in PsA.

5.5. Excessive Daytime Sleepiness

EDS is a common consequence of sleep disturbances occurring in patients with inflammatory diseases, including PsA.

In the study by Petzinny et al., EDS was assessed using the ESS, with a score >10 points considered abnormal. The authors demonstrated a high prevalence of EDS among patients with newly diagnosed PsA and RA. An elevated ESS score proved to be a useful predictor of coexisting SDB and OSA syndrome [15].

6. Sleep assessment methods in clinical trials

6.1. Subjective tools (questionnaires: PSQI, ESS, ISI)

Sleep quality in PsA remains underexplored, as most studies evaluate sleep disturbances in patients with psoriasis without stratifying for the presence of PsA [13].

The Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) is used to assess disease activity in patients with PsA. One of its components is fatigue, which may result from sleep disturbances, and highlights that fatigue is a significant problem for patients with PsA, although it remains highly subjective and dependent on the patient's perception [41].

Polak and colleagues suggest that the Multidimensional Health Assessment Questionnaire (MDHAQ) could be used as a screening tool to identify patients with poor sleep quality. The questionnaire includes items on physical function, pain, fatigue, and overall well-being, which may indirectly reflect sleep disturbances. However, its results should be followed by further evaluation using more detailed sleep assessment tools [13].

In another study, Polak and colleagues noted that research on sleep in patients with PsA has mostly relied on questionnaires. They reported that the PSQI is the most commonly used tool for subjective assessment of sleep [42].

Yet, the PSQI has not been validated in patients with PsA. Furthermore, even in other populations, its dimensionality (i.e., factor structure) warrants further investigation.

The Numerical Rating Scale (NRS) has also been applied, but it only captures the perceived severity of sleep problems. Using the PSQI, patients with PsA were found to have impaired subjective sleep quality, longer sleep latency, shorter sleep duration, reduced sleep efficiency, more frequent sleep disturbances, and greater daytime dysfunction. No significant differences were observed in the use of sleep medications between PsA patients and healthy controls [42].

The ESS is a widely used questionnaire to assess daytime sleepiness in patients with PsA. It evaluates the likelihood of falling asleep in eight common daily situations, providing insight into fatigue and sleep-related problems that may affect disease outcomes [15].

In the study by Sandikci et al. (2019), the Insomnia Severity Index (ISI) was used to assess the severity of insomnia in patients with PsA, alongside other measures such as the PSQI, ESS, and Fatigue Severity Scale [18].

6.2. Objective methods (polysomnography, actigraphy)

There is a limited number of studies investigating sleep disturbances in patients with PsA using objective methods such as polysomnography or actigraphy. Most research still relies on subjective questionnaires.

In one study, Mustafa et al. used PSG to assess the point-prevalence of OSA in PsA patients. Among 42 participants, OSA was diagnosed in 50%, with 36% exhibiting moderate to severe disease. The presence of OSA was not associated with disease activity, but older age and higher BMI were identified as risk factors [16].

In another study, the authors noted that PsA patients commonly report insomnia, pain-related awakenings, and fatigue - all potentially measurable via actigraphy. These findings suggest that objective sleep assessment could provide valuable insights beyond subjective questionnaires [43].

6.3. Advantages and limitations of the methods

Although existing sleep assessment scales, such as the PSQI and MDHAQ, are commonly used in patients with PsA, they do not capture the underlying causes of sleep disturbances in this population, including pruritus, skin pain, joint pain, and stiffness. Given these limitations, the content validity and other measurement properties of current sleep patient-reported outcome measures (PROMs) in PsA require further evaluation. To address this gap and improve the comprehensiveness of sleep assessment, a set of PsA-specific sleep instruments - the “PsO-Sleepy-Q” and the “PsA-Sleepy-Q”- are currently under validation [1].

7. Risk factors for sleep disorders in PsA

Disease activity and pain severity are among the strongest predictors of poor sleep. Patients with active PsA report significantly worse sleep quality than those with low disease activity, with studies showing that up to 60-80% of individuals with high disease activity experience clinically relevant sleep impairment [31]. Nocturnal pain, morning stiffness, and elevated inflammatory cytokines (e.g., TNF-α, IL-6) disrupt sleep continuity and reduce sleep efficiency. A bidirectional relationship has also been described, where poor sleep further increases pain sensitivity and fatigue [24, 44].

Psychological comorbidities, particularly depression and anxiety, are also strongly associated with sleep disturbances. Depression is present in approximately 20-30% of PsA patients, while anxiety affects up to 30-40%, and both conditions significantly increase the risk of insomnia and non-restorative sleep. These disorders contribute to emotional hyperarousal and impaired sleep regulation, further worsening disease burden [45].

Metabolic comorbidities, especially obesity, represent another important risk factor. Obesity affects approximately 35-45% of PsA patients and is strongly associated with OSA, which itself occurs in up to 25-40% of patients with PsA. Chronic low-grade inflammation linked to adiposity further exacerbates both disease activity and sleep disruption [46].

Finally, pharmacological treatment may influence sleep patterns. Glucocorticoids can induce insomnia in up to 30-50% of patients, particularly with higher doses or evening administration. Conversely, biologic therapies targeting TNF-α or IL-17 pathways have been associated with improvements in sleep quality in a substantial proportion of patients, likely secondary to reduced inflammation and pain [47].

Across studies involving patients with PsA, with or without concomitant psoriasis, pain emerged as the factor most consistently reported to be independently associated with sleep disturbances, followed by the presence of actively inflamed or tender joints. In studies that included patients with both psoriasis and PsA, the presence of PsA was identified as an independent predictor of sleep disturbances [5, 8, 48].

8. Effects of sleep disorders in patients with PsA

Sleep disturbance was ranked in the top 4 health-related quality of life domains affected by PsA [1].

Health-related quality of life (QoL) is one of the determinants of health status. Studies have shown that the quality of sleep has a substantial impact on the overall health-related QoL, emphasising that sleep disturbance is linked to lower QoL [49].

Sleep disturbance has been defined as one of the most prevalent issues affecting QoL in patients with PsA. Some of the factors strongly linked to the diminished quality of sleep were pain and actively inflamed or tender joints [1]. Although the issue of decreased QoL due to sleep disturbance and poor quality of sleep encompasses both patients with psoriasis (PsO) and PsA, those affected with PsA report a higher severity of consequences [8].

Both lower quality of sleep, as well as sleep disorders affect the QoL in patients with PsA. Patients with PsA experience a higher occurrence of sleep disorders, such as OSA, restless leg syndrome, insomnia, circadian rhythm disturbances in comparison to the general public. Moreover, patients with PsA record lower quality of sleep, meaning less than seven hours of restful sleep a night [15, 50]. The issue is especially prevalent in female patients, who according to studies, report a significantly lower quality of sleep, emphasising the sleep latency, unrestful sleep and subdued QoL, as a result [9].

Low quality of sleep is directly linked with reported worse mental health among patients with PsA. Studies have shown that patients disclosing poor sleep quality are often more liable to symptoms of depression and anxiety, which in result lessens the overall QoL and health [9]. Depression and anxiety are important issues concerning patients with PsO and PsA. Literature suggests that more than 10% of patients with psoriasis deal with clinical depression, while twice as many show symptoms of depression and anxiety. Furthermore, patients with PsA are more likely to develop these symptoms during the course of the disease [24].

Patients with PsA who exhibit sleep dysfunctions may be at a higher risk of other comorbidities such as cardiovascular diseases or obesity. Studies have shown that the risk of developing ischemic heart disease or stroke is higher in PsA patients who report troubles with sleep in comparison to those without sleep dysfunction. There are also interesting findings on the influence of gut microbiome in patients with PsA, and the effect the underlying dysbiose, exaggerated by sleep disturbance, may pose on the development of the disease [50].

Sleep disturbance and disruption of nighttime rythm may lead to lower energy-levels during the day, increased fatigue and lower daytime activity in patients with PsA. Patients may

report higher irritability, sleepiness, as well as disrupted memory and concentration and lower motivation and energy for everyday tasks [9].

One of the important aspects of QoL reported by patients with PsA is the influence of disease-related sleep dysfunction on the social and work environment. Patients face multiple challenges concerning their employment and the quality and quantity of work they are able to provide on a daily basis. Productivity loss, absenteeism, presenteeism are all impairments that patients with PsA-related sleep issues face, which in turn negatively impacts their overall QoL and well-being [51].

The link between PsA and sleep disturbance is bilateral and comprises multiple factors. Sleep disturbance in patients with PsA may be related to the disease activity, mainly inflammation and pain of the joints. However, sleep dysfunction such as fragmented sleep, sleep disturbances and diminished amount of sleep per night, may lower the pain threshold and in turn intensify pain. Furthermore, sleep disturbance may trigger flares of PsO and PsA, which has been shown in studies on night-shift workers with a disrupted circadian rhythm [9,15,50].

Sleep disturbance is one of the most important, alas insufficiently addressed issues regarding the QoL of patients with PsA. Holistic approach, engrossing both pharmacological and non-pharmacological treatment is crucial. Strategies such as patient education, physical therapy and psychotherapeutic interventions, such as cognitive-behavioural therapy, are key in managing the needs of patients with PsA-related sleep dysfunction.

9. Treatment and management of sleep disorders

Modern treatment of PsA includes both pharmacological, as well as non-pharmacological interventions, providing a multidisciplinary approach to treatment. The dialogue between the patient and medical professional is crucial in developing and maintaining an effective treatment plan, including not only the disease activity, but also the comorbidities such as mental health issues, pain, fatigue and sleep disturbance. Biologic therapy of PsA has been shown to provide a great impact on depression symptoms in patients, in comparison to other treatments such as phototherapy or conventional systemic therapy. Other studies have shown that patients on biologic therapy such as ixekizumab, adalimumab and ustekinumab therapies of PsA presented better work productivity enhancement in contrast to other pharmacological treatments. Some of the studies suggest that adalimumab therapy of PsA significantly enhanced sleep quality and overall QoL, as well as pain. Literature shows that health-related QoL has been shown to improve in patients on guselkumab treatment of PsA.

However, more research is needed to determine the best treatment options for management of PsA comorbidities [24,52].

Interdisciplinary approach towards treatment of PsA includes diet, sleep and physical activity, as well as cognitive-behavioural therapy and balneotherapy. It has been shown that adequate, safe physical activity such as moderate aerobic activities, muscle strengthening and stretching all benefit disease activity and lower pain levels and overall fatigue. Another intervention which may improve disease activity levels is weight loss in overweight patients [26].

Although many techniques and treatments have already been studied, further research is essential to determine the best course of action in an interdisciplinary approach towards sleep disturbance in patients with PsA. The patient-physician dialogue is crucial in building a sustainable treatment plan that is tailored to the patient. Education and increasing patient awareness is vital in management of sleep disturbances in PsA.

10. Discussion and conclusions

This narrative review highlights that sleep disturbances represent one of the most prevalent and clinically relevant comorbidities in patients with PsA. The available evidence indicates that sleep impairment affects a substantial proportion of patients, with poor sleep quality reported in up to 84% of individuals when assessed using the PSQI. Importantly, sleep disturbances appear to be more common and more severe in PsA than in psoriasis without musculoskeletal involvement, suggesting that the inflammatory, painful, and disabling nature of joint disease contributes significantly to sleep dysfunction.

One of the most important findings emerging from the reviewed literature is the multifactorial nature of sleep disturbances in PsA. Sleep impairment cannot be attributed solely to pain or disease activity. Instead, it results from a complex interaction between chronic inflammation, pain, neuroendocrine dysregulation, psychological comorbidities, metabolic factors, and sleep-specific disorders such as insomnia, RLS and OSA. This complexity may explain the considerable heterogeneity in prevalence estimates reported across studies.

The relationship between inflammation and sleep appears to be bidirectional. Elevated levels of pro-inflammatory cytokines, particularly TNF-α and IL-6, contribute to alterations in sleep architecture, increased sleep fragmentation, and reduced sleep efficiency. At the same time, sleep deprivation itself promotes the production of inflammatory mediators, potentially exacerbating disease activity. This vicious cycle may partially explain why patients with poor sleep frequently report higher disease activity scores, greater pain intensity, and increased

fatigue. Similar mechanisms have been described in other inflammatory rheumatic diseases, including RA and axial spondyloarthritis, suggesting that sleep disturbances may represent an integral component of systemic inflammatory burden.

Pain emerged as the factor most consistently associated with impaired sleep across studies. Nocturnal pain, morning stiffness, and enthesitis directly interfere with sleep initiation and maintenance, while chronic pain contributes to long-term alterations in sleep architecture, including reductions in restorative slow-wave sleep and REM sleep. These findings support previous observations that sleep quality should be considered an important patient-reported outcome in PsA management. Notably, sleep impairment may also lower pain thresholds through central sensitization mechanisms, further reinforcing the reciprocal relationship between pain and sleep disturbances.

Among specific sleep disorders, insomnia appears to be the most prevalent condition in PsA. The reviewed studies consistently demonstrated associations between insomnia severity, fatigue, depression, anxiety, and reduced physical functioning. These findings suggest that insomnia should not be viewed merely as a symptom of active disease but rather as a distinct clinical problem requiring targeted assessment and intervention. Similarly, RLS was reported more frequently in patients with PsA than in individuals with psoriasis alone or healthy controls, and its severity was associated with poorer sleep quality, fatigue, depressive symptoms, and impaired QoL. Although the mechanisms underlying this association remain incompletely understood, chronic inflammation and altered dopaminergic pathways may play contributory roles.

The available evidence also suggests an increased risk of OSA in patients with PsA. Population-based studies indicate a significantly elevated incidence of sleep apnea compared with the general population, although prevalence estimates vary substantially between studies. This variability likely reflects differences in patient characteristics, diagnostic methods, and disease duration. Obesity, which is highly prevalent in PsA, may act as an important mediator of this association. Given the well-established links between OSA, cardiovascular disease, and systemic inflammation, routine screening for SDB may be particularly important in patients with additional cardiometabolic risk factors.

An important observation from this review is the relative scarcity of objective sleep assessments in PsA research. Most studies rely on subjective questionnaires, particularly the PSQI, ESS, and ISI. While these tools are practical and easy to implement, they provide limited insight into the physiological mechanisms underlying sleep disturbances. Objective methods such as polysomnography and actigraphy remain underutilized despite their potential to

identify specific sleep abnormalities and quantify sleep architecture disturbances. Furthermore, currently available sleep assessment tools have not been specifically developed for PsA and may fail to capture disease-specific contributors to sleep impairment, such as joint pain, stiffness, enthesitis, and skin symptoms. The ongoing validation of PsA-specific instruments, including the PsA-Sleepy-Q, represents an important step toward more accurate evaluation of sleep outcomes in this population.

The clinical consequences of sleep disturbances extend far beyond nocturnal symptoms. Poor sleep quality is consistently associated with reduced health-related QoL, impaired physical functioning, fatigue, mood disorders, cognitive dysfunction, and decreased work productivity. The evidence reviewed suggests that sleep disturbances contribute significantly to the overall disease burden experienced by patients with PsA. Moreover, sleep dysfunction may potentially increase the risk of cardiovascular and metabolic comorbidities, which are already overrepresented in this population. These findings support the concept that sleep health should be regarded as an essential component of comprehensive PsA management.

The review also highlights the potential benefits of effective disease control on sleep outcomes. Biologic therapies targeting inflammatory pathways, particularly TNF-α and IL-17 inhibitors, have been associated with improvements in sleep quality, fatigue, pain, and overall QoL. However, it remains unclear whether these benefits result primarily from reduced inflammation and symptom burden or from direct effects on sleep-regulating mechanisms. Future studies should explore this question further and investigate whether sleep-specific interventions can provide additional benefits beyond disease-targeted therapies.

Several limitations of the currently available literature should be acknowledged. First, substantial heterogeneity exists regarding study design, patient populations, diagnostic criteria, and sleep assessment methods, limiting direct comparisons between studies. Second, many investigations include mixed populations of patients with psoriasis and PsA, making it difficult to distinguish the specific impact of joint disease on sleep outcomes. Third, most studies are cross-sectional, preventing conclusions regarding causality. Finally, objective sleep assessments remain relatively rare, and longitudinal studies evaluating the impact of treatment on sleep outcomes are limited.

Future research should focus on prospective studies incorporating both subjective and objective sleep measures, standardized diagnostic criteria, and comprehensive assessment of disease activity, pain, psychological health, and comorbidities. Particular attention should be given to the evaluation of circadian rhythm disturbances, which remain poorly studied in PsA despite increasing evidence linking circadian dysregulation with inflammatory pathways.

Furthermore, randomized controlled trials assessing multidisciplinary interventions that combine pharmacological treatment with behavioral and sleep-focused therapies are needed.

In conclusion, sleep disturbances are highly prevalent in PsA and represent a major contributor to disease burden, impaired QoL, and potentially worse clinical outcomes. Their pathogenesis is multifactorial and involves complex interactions between inflammation, pain, neuroendocrine dysfunction, psychological factors, and comorbid sleep disorders. Given the bidirectional relationship between sleep and disease activity, routine screening and management of sleep disturbances should become an integral part of comprehensive PsA care. Improved recognition and treatment of sleep disorders may not only enhance sleep quality but also contribute to better overall disease control and patient well-being.

Declaration of AI Use

In preparing this manuscript, the authors used ChatGPT (OpenAI) for the purpose of improving the quality of the English language, including grammar, spelling, clarity, and stylistic refinement. After using this tool, the authors carefully reviewed and edited the manuscript as needed and accept full responsibility for the accuracy, integrity, and substantive content of the publication.

Disclosure

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