**Medical Science**

**To Cite:**
Prolejko S, Kotnis W, Siemianowski J, Buczek W, Pawlak M, Kopala J, Gajęcki B, Steć G, Brzyska A, Kucharski T. Sleep Disorders in Autoimmune Diseases: A Literature Review Across Rheumatoid Arthritis, Lupus, and Psoriasis. *Medical Science* 2025; 29: e90ms3600 doi: https://doi.org/10.54905/disssi.v29i160.e90ms3600

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**Peer-Review History**
Received: 07 March 2025
Reviewed & Revised: 16/March/2025 to 12/June/2025
Accepted: 18 June 2025
Published: 25 June 2025

**Peer-review Method**
External peer-review was done through double-blind method.

Medical Science
pISSN 2321–7359; eISSN 2321–7367



# Sleep Disorders in Autoimmune Diseases: A Literature Review Across Rheumatoid Arthritis, Lupus, and Psoriasis

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## ABSTRACT

Sleep disturbances commonly affect autoimmune diseases including, rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), and psoriasis. The evidence now shows that poor sleep not only reflects the disease burden but also actively modulates immune responses and influences the disease progression. This review aims to integrate both mechanistic and clinical evidence that links disordered sleep and circadian rhythm disruption to autoimmune pathophysiology. Sleep deprivation and circadian misalignment alter cytokine expression (including IL-6, TNF-$\alpha$, and interferons) while also affecting T cell differentiation and neuroendocrine signaling. These shifts contribute to chronic inflammation, autoantibody production, and symptom exacerbation across disease contexts. In RA, disrupted cortisol and melatonin rhythms correlate with early morning flares and heightened inflammatory tone. In SLE, fatigue and mood disturbances are often more closely tied to poor sleep than disease activity. Psoriatic patients report substantial sleep impairment due to pruritus, comorbid obstructive sleep apnea (OSA), and mood-related comorbidities. Despite this, sleep assessments remain rare in routine care. Behavioral interventions such as cognitive behavioral therapy for insomnia (CBT-I) show promise for improving outcomes but remain underutilized. Greater clinical integration of sleep management may offer new strategies to mitigate disease burden and improve patient quality of life.

**Keywords:** sleep disorders, autoimmune disease, circadian rhythm, rheumatoid arthritis, systemic lupus erythematosus, psoriasis, inflammation, cognitive behavioral therapy

## 1. INTRODUCTION

Living with autoimmune diseases such as rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), and psoriasis entails coping with chronic immune-mediated conditions that cause systemic inflammation and tissue damage. These diseases can

be debilitating, which increases the need for medical care and imposes a heavy psychosocial burden on the individuals affected (Awuah et al., 2023).

The profound disturbances in sleep and circadian rhythms affecting patients suffering from these diseases are often overlooked in the clinical picture. Epidemiological studies attest to the alarming prevalence, with figures ranging between 55 and 85% of patients suffering from a sleep disorder like insomnia, persistent fatigue, waking up feeling unrefreshed, excessive sleepiness, or chaotic sleep-wake behavior (Awuah et al., 2023; Faraguna et al., 2024). Setting aside individual diseases, on a broader spectrum, evidence from extensive longitudinal studies indicates that chronic insomnia, the kind that makes sufferers dependent on sleep medications, constitutes a bigger risk factor for the later development of autoimmune diseases (Kok et al., 2016). It is important to note that these sleep disorders rarely correlate with the intensity of the disease or with pain ratings. This suggests the existence of a more profound connection between sleep, circadian processes, and the underlying autoimmune pathological process.

Sleep is a crucial pillar of immunological balance, although it can often be underestimated. Healthy sleep helps keep inflammation in check by modulating the activity of key immune messengers (cytokines like IL-6, TNF-$\alpha$, and IL-1$\beta$) and regulating the body's natural cortisol rhythm (Irwin, 2019; Zielinski et al., 2019). When this delicate regulation is disrupted by insomnia, obstructive sleep apnea (OSA), or a disturbed internal body clock, it can trigger widespread inflammation, increase oxidative stress, and impair the immune system's ability to monitor and respond effectively (Xiang et al., 2021).

The effects of these disruptions are noticeable in various autoimmune conditions. For individuals with RA, the body's natural production of cortisol may be delayed to such an extent that it cannot effectively control inflammatory signals at night. This inadequacy then promotes the morning stiffness characteristic of rheumatoid arthritis (RA) (Awuah et al., 2023). Counterintuitively, whilst melatonin generally has a soothing effect, its nighttime increase in RA might spur specific immune cells within the joint, amplifying early-morning discomfort. Poor sleep in SLE is highly attributable to persistent fatigue with depressive symptoms and lower quality of life that weigh on many, even when their disease activity looks relatively under control. Objective sleep studies confirm that sleep is fragmented, resulting in less efficient rest and increased awake time during the night. Some individuals, however, may also experience longer overall sleep durations (Faraguna et al., 2024). In psoriasis, intense itching (pruritus) can cause significant sleep disruption. The same applies to the presence of obstructive sleep apnea (OSA) or restless legs syndrome (RLS). These are both known to be more present in this group than in healthy populations (Solak et al., 2023; Halioua et al., 2022).

Despite heavy evidence based on laboratory and clinical observations, the diagnosis and management of sleeping disorders are rarely part of routine care for patients with autoimmune disorders (Reynolds et al., 2020). This review attempts to bridge the gap. Here, we synthesize the current knowledge on sleep and circadian rhythm regulation of the immune system. We look at specific findings relevant to RA, SLE, and psoriasis, including treatment-oriented approaches directed at improving sleep to enhance health outcomes in these conditions.

## 2. METHODOLOGY

This literature review integrates studies that analyze the relationship between sleep disorders and autoimmune diseases, focusing on RA, lupus, and psoriasis. A thorough search for articles between 2014 and 2024 was conducted, using databases like PubMed, Scopus, and Google Scholar.

Critical keywords involved were "sleep disorders," "rheumatoid arthritis," "lupus," "psoriasis," and "circadian rhythm," among others. Only articles mentioning studies including human adults were considered for further review.

Articles chosen were reviewed according to study design, sample size, methods, and results about sleep disturbances in autoimmune conditions. Studies investigating mechanisms linking autoimmune diseases and sleep, such as altered immune system functioning or changes in circadian gene expression, were considered especially relevant for this review.

## 3. RESULTS AND DISCUSSION

**Mechanisms Linking Sleep and Immune Dysregulation**

Sleep, internal body clocks, and immune health - their interlocking rhythms are vital. When this relationship is disrupted, it can throw both the body's first line of defenses (innate immunity) and its more specialized responses (adaptive immunity) out of sync. Increasingly, it is acknowledged that these disturbances may not be simply their side effects, but could also actively induce and worsen autoimmune disorders (Xiang et al., 2021; Zielinski et al., 2019).

From a molecular standpoint, circadian rhythms are governed by a tightly regulated system of "clock genes:" most notably BMAL1, CLOCK, PER, and CRY. These operate in a feedback loop, regulating each other's production over a 24-hour cycle (Xiang et al., 2021). This biological clock not only governs behavioral circadian rhythms such as sleep-wake cycles and core body temperature but also controls the functioning of immune cells. Consequently, the clock governs when to produce inflammatory mediators (cytokines), when to present a threat to the immune system, and the development of specialized T cells. In essence, this intracellular molecular clock regulates both the timing and intensity of inflammatory responses within immune cells (Irwin, 2019).

However, if the clock genes were to fail, there is a significantly greater chance of the immune system being severely disrupted. For instance, in the absence of CRY1/CRY2 genes, there is a massive overproduction of inflammatory cytokines, including TNF-$\alpha$ and IL-6. Likewise, the lack of PER2 means an increase in IL-1$\beta$. In our key immune cells - macrophages, the absence of BMAL1 accelerates energy consumption via glycolysis, favoring Th17 cell differentiation, a lineage famed for its ability to induce autoimmune inflammation. Apart from these core clock genes, internal sensors do the rest; for instance, the NLRP3 inflammasome in the brain plays a key role. They act as translators, converting wakefulness or pathogen signals into the sleep regulation pathway partly by producing IL-1$\beta$ (Zielinski and Gibbons, 2022).

To make matters complicated, there is sleep deprivation. Even short-term sleep loss - partial or acute - can elevate inflammatory markers, such as IL-1$\beta$, IL-6, and TNF-$\alpha$, as observed in studies involving both humans and animals (Irwin, 2019). Once sleep restriction becomes chronic, it leads to a flattening of the normal daily cortisol rhythm and decreases responsiveness to cortisol's anti-inflammatory effects (decreased glucocorticoid sensitivity). Moreover, it amplifies signals from pro-inflammatory agents, such as NF-$\kappa$B, resulting in chronic, low-grade inflammation (Irwin et al., 2023).

The hormone-regulating circuits in the human body, particularly the neuroendocrine system, are also considered to be highly susceptible to this effect. The cortisol release from the hypothalamic-pituitary-adrenal (HPA) axis, as the central stress-response system, occurs in a rhythmic pattern, which physiologically aims to reduce inflammation first thing in the morning. Nonetheless, in conditions such as RA or SLE, the circadian rhythm of cortisol release is shifted or blunted, resulting in a lack of the significant suppression of immune activity that typically occurs at nighttime (Awuah et al., 2023). On the other hand, melatonin, being the hormone secreted by the pineal gland mainly at night while sleeping, has anti-inflammatory abilities, mainly by suppressing certain T cell pathways (Th1 and Th17). Melatonin, however, tends to behave somewhat strangely in individuals with autoimmune disease; it may either have irregular levels or may ironically elevate when it shouldn't, further contributing to the dysregulated immune response (Awuah et al., 2023).

The summed-up impact of these disruptions across immune signaling pathways and hormone regulation is illustrated in Table 1, which shows the key mechanisms linking sleep and immune dysregulation in RA, SLE, and psoriasis.

Putting all this together, chronic disturbances in sleep and circadian rhythms can initiate a chain reaction: first, an overactive immune system; then, a gradual loss of the body's ability to distinguish between self and non-self-antigens (loss of tolerance); finally, inflammation settles into specific tissues. In this manner, these tightly linked mechanisms directly associate our sleep biology with the induction of diseases like RA, SLE, and psoriasis. Crucially, they also open the door to avenues worth investigating for their therapeutic potential. For example, in RA, persistent chronic inflammation can alter circadian signaling, thereby rewiring daily gene expression patterns in immune cells and even altering ceramide levels in the blood. Such a perspective highlights the complexity of the reciprocal relationship linking our internal clocks and autoimmune diseases (Poolman et al., 2019).

**Table 1**. Key Immunological and Endocrine Mechanisms Linking Sleep and Autoimmunity

| Mechanism | Rheumatoid Arthritis (RA) | Systemic Lupus Erythematosus (SLE) | Psoriasis, Psoriatic Arthritis (PsA) |
|---|---|---|---|
| **Circadian misalignment** | Delayed cortisol peak; high nocturnal melatonin | Blunted cortisol rhythm; disrupted melatonin | Blunted melatonin secretion; delayed sleep onset |
| **Cytokine disruption** | ↑ IL-6, TNF-$\alpha$ at night | ↑ IL-6, IFN-$\alpha$, IL-1$\beta$ | ↑ TNF-$\alpha$, IL-6, IL-17 |
| **Neuroendocrine feedback** | Sympathetic | HPA axis blunting | Substance P elevation, |

| | | | |
|---|---|---|---|
| | overactivation during REM | | sympathetic arousal |
| **T cell imbalance** | ↓ Tregs, ↑ Th17 with sleep loss | ↑ Th1/Th17 polarization under poor sleep | ↑ Th17-driven keratinocyte activation |
| **Oxidative load** | ↑ NF-κB, AP-1, monocyte output | ↑ ROS, impaired sleep repair mechanisms | ↑ Systemic inflammation via disrupted sleep |

**Rheumatoid Arthritis**

Rheumatoid arthritis (RA) is a very demanding systemic autoimmune illness that is mainly associated with chronic inflammation of the synovium (lining of the joints), which can destroy the joints and cause extra-articular symptoms. Joint pain and swelling are the distinctive traits, but sleep disturbances are among the most common and exhausting symptoms. Insomnia, waking up too early, non-restorative sleep, and fragmented sleep patterns are some of the difficulties faced by somewhere between 50% and 80% of RA individuals (Grabovac et al., 2018; McBeth et al., 2022; Gouda et al., 2023). Elaborating on this further, the existence of new evidence with prospective cohort studies suggests that healthy sleep habits, such as regular sufficient sleep with no frequent insomnia or excessive daytime sleepiness, are significantly associated with reduced risks of RA development in the first place, including those with underlying genetic risk (Ni et al., 2024).

While pain is a significant cause of sleep disturbance in RA, emerging research findings highlight the role of circadian and immunological mechanisms in this condition. For example, delaying the body's natural cortisol rhythm can weaken the nighttime inhibition of inflammatory cytokines, including IL-6 and TNF-$\alpha$, thereby increasing morning symptoms such as stiffness and pain. Meanwhile, melatonin usually acts as an anti-inflammatory agent in the human body. The paradox arises when melatonin behaves differently in RA. At night, elevated melatonin levels have been observed to trigger the activation of macrophages (immune cells) within the synovium, leading to increased production of IL-12 and nitric oxide, and potentially sustaining joint inflammation (Awuah et al., 2023).

Genetic-level evidence shows further disruptions in RA. At the cellular level of the joint, alterations in core clock genes, particularly BMAL1 and PER2, have been reported to directly link circadian dysregulation to disease processes within the joints (Awuah et al., 2023). Furthering this link, it has been shown that RA can actually "reprogram" the circadian output pathways in immune cells found in the blood (peripheral blood mononuclear leukocytes). This reprogramming heightens daily fluctuations in gene expression and can even establish a new circadian rhythm on substances present in blood, such as ceramides. These findings suggest an extensive reorganization of the body's internal clock system resulting from chronic inflammation (Poolman et al., 2019). On the other hand, patients with rheumatoid arthritis may have a hyperactive sympathetic nervous system (the “fight or flight” response) during REM sleep, characterized by higher levels of norepinephrine, a stress hormone that could exacerbate systemic inflammation (Irwin et al., 2023).

Chronic sleep fragmentation has significant consequences that can lead to various immunological disturbances. Prolonged inadequate sleep may promote the increased production of monocytes (another type of immune cell) or even accelerate the epigenetic aging of immune cells, which renders them function less optimally, thereby exacerbating systemic inflammation (Irwin, 2019).

These biological disruptions have a strong and direct impact on how patients feel and function. Reports consistently associate poor sleep with increased pain, fatigue, and functional impairment in RA. Fatigue is yet another primary concern that can remain present even as other manifestations of disease activity come under control. It appears to be tightly linked to the formation of the painful triad of pain, mood symptoms, and disordered sleep (McBeth et al., 2022).

Although sleep problems have been regarded as a significant problem in RA patients, addressing these problems has rarely been a primary focus of therapeutic efforts. However, there is some hope in non-pharmacological treatments. For instance, a randomized controlled trial with RA patients found that when nurses delivered cognitive behavioral therapy for insomnia (CBT-I) to RA patients, it significantly reduced insomnia severity, fatigue, and depressive symptoms. The interesting thing was that all these clinical advantages presented themselves even though the improvements in objective measures of sleep from polysomnography (PSG, sleep studies) were minimal, thereby suggesting that simply enhancing one's subjective experience of sleep can alone be beneficial in acting as a relief from symptoms (Latocha et al., 2023).

That same trial also revealed a significant issue in the PSG test: 40% of the participants were found to have obstructive sleep apnea, which had been undiagnosed before. This discovery greatly reinforces the need for routine screenings for sleep disorders in this group.

In essence, sleep dysfunction in RA is a complex issue: it arises from the inflammatory nature of the disease and, in turn, acts as a powerful amplifier of its symptoms. A more holistic approach to RA care, which integrates behavioral sleep therapies, considers the timing of treatments (chronotherapy), and includes screening for conditions like sleep apnea, should be regarded as an essential part of comprehensive management.

**Systemic Lupus Erythematosus**

Systemic lupus erythematosus (SLE) is a chronic autoimmune disorder in which the body's immune system erroneously attacks its tissues. The presence of such disturbances causes widespread inflammation, which can affect several organ systems. Apart from other physical symptoms, persistent fatigue, some mood disturbances, and cognitive impairments are experienced by SLE patients, most of which are strongly linked to sleep quality.

Sleep disorders are confirmed in between 55 and 85% of cases of SLE. They may include insomnia, hypersomnia, awakening with non-restorative sleep, and frequent nighttime awakenings (Awuah et al., 2023; Faraguna et al., 2024). More importantly, prospective cohort studies have further highlighted this concerning association: chronic short sleep duration, defined as five hours or less per night, is associated with an elevated risk of developing SLE. This is visible particularly among women, with present physical pain or depression, thereby setting sleep deprivation as one modifiable factor in the disease development (Choi et al., 2023). This evidence is further supported by earlier large-scale cohort data, which suggested that a prior diagnosis of sleep disorder significantly increased the odds of developing SLE afterward (Chung et al., 2016). When sleep is objectively measured with the aid of devices such as actigraphy (wrist-worn devices that track movement), studies indicate that patients with lupus may experience sleep maintenance disorders characterized by decreased sleep efficiency (less time spent sleeping compared to the time spent in bed) and increased wakefulness after sleep onset. Paradoxically, however, some patients can manifest greater total sleep time than those without SLE (Faraguna et al., 2024).

Several factors conspire to compromise sleep quality in SLE. Notably, glucocorticoid treatment, a crucial medication for lupus, appears to disrupt physiological sleep patterns by reducing one's ability to stay asleep (Faraguna et al., 2024). Predominantly, it leads to "normal sleep duration insomnia," in which the patient demonstrates difficulty in maintaining continuous sleep, even though their total sleep time is not necessarily shortened (Faraguna et al., 2023). Moreover, the psychological impact of living with a chronic illness alone should be addressed. Stress-related hyperarousal, depression, and anxiety, affecting up to 40% of the lupus population, are key determinants of both insomnia and disrupted sleep (Mertz et al., 2020). It is further noted that individuals with SLE have a tendency toward greater levels of perceived stress than those without it, and this heightened stress is closely associated with insomnia, depicted by short sleep duration (Faraguna et al., 2023).

Disruptions in the body's internal clock (circadian rhythms) may lead to sleep disturbances. Daily circadian patterns of hormone secretion may be altered in SLE, typically with a flattening of the cortisol secretion peak and irregular melatonin secretion in some cases. This may result from a dysfunction in core clock genes such as BMAL1 and CRY2 (Awuah et al., 2023).

Fatigue is undoubtedly the most commonly reported and the most severe symptom among SLE patients. Unlike other symptoms of lupus that appear and disappear with flare-ups, the severity of fatigue often shows little correlation with standard markers of lupus disease activity. Fatigue closely relates to poor sleep quality, patients' psychological burden (including depression and anxiety), and sometimes the maladaptive ways they try to cope with their illness (Mertz et al., 2020; Faraguna et al., 2024).

Although the relationship is evident, addressing sleep problems is not often considered a standard treatment in routine SLE care (Sang et al., 2022). This is not to say that lupus patients are not interested in improving their sleep quality. Surveys have revealed that almost all lupus patients would be willing to attempt behavioral sleep interventions, which include Cognitive Behavioral Therapy for Insomnia (CBT-I). Practical issues seem to get in the way, including costs, the availability of services, and the absence of recommendations from healthcare providers to patients. The literature has shown that CBT-I can be an accepted and potentially effective intervention for individuals with SLE, particularly when delivered digitally or as part of a multidisciplinary approach (Da Costa et al., 2021).

Given how debilitating fatigue can be, its crucial role in disrupting sleep, and the promising prospect of targeted interventions, a much heavier emphasis on screening and intervention for sleep within the overall treatment of the lupus patient should be advocated.

**Psoriasis and Psoriatic Arthritis**

Psoriasis is a chronic immune-mediated skin disease that affects 2-3% of the population. It is characterized by rapid proliferation of skin cells (keratinocytes) and systemic inflammation. Very frequently it coexists with other diseases, such as psoriatic arthritis (PsA), obesity, cardiovascular disease, and depression, which make this condition even more complex.

Within this patient population, sleep disorders are pervasive. The most recent prevalence estimates vary widely from 36% to over 80%, depending on the severity of the psoriasis and the methods of assessing sleep (Halioua et al., 2022). For example, studies using the Pittsburgh Sleep Quality Index (PSQI) have shown that poor sleep quality (a PSQI score of >5) is present in approximately 84% of subjects with PsA and 69% of subjects with psoriasis alone (often referred to as psoriasis cutaneous, or PsC). Such figures are significantly higher than those observed in healthy individuals, and it is worth noting that individuals with PsA report even worse sleep quality than those with PsC (Wong et al., 2017). In addition to those sleep problems, fatigue and psychological factors like anxiety and depression exert an independent effect on the diminished health-related quality of life in people with PsA, and sometimes this impact can be greater than that of clinical measures of skin or joint inflammation (Haugeberg et al., 2020). These are just a few of the inconveniences patients face due to accompanying sleep disturbances. Nighttime itching (nocturnal pruritus) is very often implicated. Skin itching worsens at bedtime due to circadian variations in the skin barrier function and cortisol secretion. When using validated instruments such as the MOS Sleep Index II, a strong correlation has been found between itch and sleep disruption. Increased severity of pruritus has been highly correlated with increased sleep disturbances (Mrowietz et al., 2015). Active joint inflammation is also identified as an independent factor contributing to the decline in sleep quality among PsA subjects (Wong et al., 2017). Further, pain, burning sensations, and psychological distress are more factors that continue to hamper the processes of falling asleep and waking up.

Though in the presence of other sleep disorders, psoriasis often gets the spotlight as a concern. For instance, obstructive sleep apnea (OSA) affects a startling 36–82% of patients who have psoriasis, showing a significantly higher rate than the one in the general population. Also, restless legs syndrome (RLS), another common comorbidity, affects about 18% of cases. OSA and RLS disrupt the typical sleep structure and exacerbate systemic inflammation, thereby contributing to the substantial disease burden (Solak et al., 2023; Halioua et al., 2022).

Melatonin production, an important hormone regulating sleep, is often considered dysregulated in psoriasis. Patients with psoriasis can have a blunted peak of melatonin levels during the night. Typically, such a phenomenon aids in regulating body temperature, inducing sleep, and sending anti-inflammatory signals, but here it gets disturbed. Adding to the problem, elevated levels of a neuropeptide called substance P in the affected skin areas can contribute to itching and pain, and also heighten sympathetic nervous system arousal, all of which can further worsen sleep (Controne et al., 2022).

Psychological complications and, primarily, anxiety and depression are highly linked with sleep dysfunction in psoriasis. These are often bidirectional relationships, further building the vicious cycle that can worsen the overall disease burden. Psoriasis severity scores, like PASI (Psoriasis Area and Severity Index) and the Dermatology Life Quality Index (DLQI), which measures the quality of life, reflect those correlations mentioned above (Cipolla et al., 2024; Zaky et al., 2023).

Fortunately, better sleep can be attained when the patient successfully treats psoriasis. Biologic therapies targeting specific inflammatory pathways, such as IL-17 or TNF-$\alpha$ related pathways, can relieve inflammation and itching, indirectly improving sleep. Additionally, some topical treatments might cause sleep disturbance, given some uncomfortable sensations or their nighttime application. CBT has been shown to improve sleep and reduce the severity of psoriasis, especially with moderate-to-severe disease (Xiao et al., 2019).

Although these connections and potential benefits exist, sleep disorders are rarely addressed in dermatological care. To assess patients who may benefit from intervention, screening tools such as the PSQI should be used routinely in conjunction with pruritus severity scales. Fundamentally, the issue of sleep dysfunction in psoriasis and psoriatic arthritis (PsA) is a multifaceted concern with interdependent physiological, metabolic, and psychological variables. Integrating sleep evaluation and treatment into regular dermatologic care might notably improve patient outcomes and quality of life.

## 4. CONCLUSIONS

It is increasingly clear that sleep disturbances are far more than just a secondary symptom of autoimmune diseases; they are, in fact, active players that contribute to immune system imbalance, worsen symptoms, and diminish overall quality of life. In rheumatoid arthritis, systemic lupus erythematosus, or psoriasis, troubled sleep arises from a complex interplay of shared and disease-specific factors, including skewed inflammatory cytokine profiles, neuroendocrine disruptions, and co-occurring behavioral issues. The impact

can be significant; for instance, forward-looking studies link a chronic lack of sleep to a higher risk of developing SLE. Despite their high prevalence and their high impact, the neurological manifestations of these sleep problems often go unnoticed and undiagnosed in everyday clinical practice. Compelling evidence from clinical observations and mechanistic studies strongly suggests that when sleep is taken seriously, screening becomes incorporated, and nonpharmacological treatments, such as cognitive behavioral therapy for insomnia (CBT-I), are employed. That, together with the optimization of treatment timing, can lead to meaningful relief of symptom burden and better outcomes for patients. At times, even when objective measures of sleep do not show a marked improvement after treatment, subjective sleep quality changes can help patients feel less fatigued, in less pain, and less psychologically stressed. The findings from extensive prospective studies are also exciting from a primary prevention perspective, suggesting that healthy sleep patterns may suppress the occurrence of RA even in those people with a genetic predisposition. Addressing these complex issues requires a team effort. A multidisciplinary approach should be introduced to integrate the assessment and management of sleep into autoimmune disease care, wherein rheumatologists, dermatologists, psychologists, and sleep medicine experts cooperate. On a practical level, implementing the Pittsburgh Sleep Quality Index (PSQI) or using simple, portable monitors can aid in the early detection of sleep problems. Meanwhile, with the rise of digital CBT and improved sleep hygiene interventions, effective treatment could potentially be distributed to a much wider audience.

Future research should focus on investigating how long sleep-focused interventions affect the progression of autoimmune diseases, specific inflammatory markers, and, most importantly, the patient's health-related quality of life from a long-term perspective. Raising the importance of sleep in autoimmune diseases beyond the walls of research could open new pathways for better systemic disease management and, consequently, increase the quality of life for affected individuals.

**Author's Contributions**

Sandra Prolejko: Conceptualization, writing- rough preparation, investigation
Jan Siemianowski: Formal analysis, supervision
Weronika Buczek: Visualization, supervision
Tomasz Kucharski: Conceptualization, project administration
Weronika Kotnis: Methodology, data curation
Magdalena Pawlak: Conceptualization, methodology
Agata Brzyska: Resources, writing-rough preparation
Justyna Kopala: Conceptualization, writing- rough preparation
Błażej Gałęcki: Resources, data curation
Greta Steć: Writing - review and editing, supervision
All authors have read and agreed to the published version of the manuscript.

**Acknowledgments**

No acknowledgments.

**Informed consent**

Not applicable.

**Ethical approval**

Not applicable.

**Funding**

This study has not received any external funding.

**Conflict of interest**

The authors declare that there is no conflict of interest.

**Data and materials availability**

All data sets collected during this study are available upon reasonable request from the corresponding author.

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