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The Role of Sleep in Immune Function and Susceptibility to Infectious Diseases: A Narrative Review

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Abstract

Sleep and immunity are closely interconnected: sleep regulates immune function, while infection alters sleep architecture. This narrative review summarizes evidence linking insufficient sleep with impaired immunity and increased susceptibility to infectious diseases. Short sleep duration (≤ 6 hours) and poor sleep quality are associated with increased inflammation and a higher risk of viral infections, including rhinovirus and SARS-CoV-2. Insufficient sleep around vaccination may reduce antibody production and impair T-cell immune memory. In obstructive sleep apnea, severe disease is associated with impaired humoral immunity, while positive airway pressure therapy may improve immune function. Melatonin may further support immunomodulation by linking circadian rhythms with immune regulation. Overall, adequate, high-quality sleep contributes to infectious disease prevention and optimal vaccine-induced immunity.

1. Introduction

Sleep is a fundamental determinant of health and a critical factor influencing numerous physiological processes, including the regulation of immune responses (Asif et al., 2017; Chervin, 2024). Sleep and the immune system are closely interconnected through a bidirectional relationship (Besedovsky et al., 2019; Ibarra-Coronado et al., 2015). On the one hand, insufficient sleep duration and impaired sleep quality are recognized as factors that increase susceptibility to infections (Martínez-Albert et al., 2025; Poluektov, 2021). On the other hand, immune system activation during infection alters sleep patterns, often increasing sleep need, which is considered an adaptive response supporting host defense (Besedovsky et al., 2019; Feuth, 2024).

In modern societies, circadian rhythm disruptions are increasingly prevalent, largely due to irregular daily schedules, shift work, and exposure to artificial light (Schmitz et al., 2022). Chronic sleep deprivation is regarded as a risk factor for numerous disorders, including metabolic, cardiovascular, and neurodegenerative diseases (Kuna et al., 2023). With respect to immune function, sleep disturbances have been shown to impair immune competence and promote a chronic low-grade systemic inflammatory state (Irwin, 2023; Lee and Park, 2024). These alterations may compromise both innate and adaptive immune responses, thereby increasing vulnerability to infectious diseases (Asif et al., 2017; Irwin et al., 2006).

2. Methods

This study constitutes a narrative review of the literature aimed at synthesizing available evidence on the bidirectional interaction between sleep quality and susceptibility to infectious diseases (Besedovsky et al., 2019; Ibarra-Coronado et al., 2015). The objective was to collect

and interpret experimental, observational, and review studies describing both the impact of sleep deficiency on immune function and the mechanisms underlying increased infection risk (Irwin, 2023; Kuna et al., 2023).

Search strategy and inclusion criteria

Literature searches were conducted in major scientific databases, including PubMed/MEDLINE and Scopus. Given the dynamic development of this research field, both seminal studies dating back to the 1970s—when systematic investigations into sleep-immunity interactions began—and recent publications up to 2025 were included (Chen et al., 2025; Martínez-Albert et al., 2025).

Search terms encompassed combinations of keywords related to sleep and sleep disorders alongside immune function and infectious diseases, including:

1. Sleep duration and disorders: sleep deprivation, insomnia, obstructive sleep apnea (Nie et al., 2022; Quan et al., 2023).
2. Immune response and inflammation: immune response, inflammation, cytokines (IL-6, TNF- α), natural killer cells (Irwin et al., 2006; Lee and Park, 2024).
3. Infectious susceptibility: vaccination efficacy, infectious disease, viral infections (Athanasίου et al., 2022; Schmitz et al., 2022).

Eligible studies analyzed molecular mechanisms, clinical observations, and humoral and cellular immune responses. Data extraction focused on:

- the effects of sleep deprivation on inflammatory markers and cytokine profiles (Irwin et al., 2006; Kuna et al., 2023),
- associations between sleep disturbances and immune responses, particularly vaccine-induced immunity (Athanasίου et al., 2022; Schmitz et al., 2022),
- relationships between sleep deficiency and viral infection risk (Martínez-Albert et al., 2025; Quan et al., 2023).

Findings were synthesized according to major mechanistic pathways, including cytokine dysregulation and hypothalamic–pituitary–adrenal (HPA) axis activation (Asif et al., 2017; Besedovsky et al., 2019). Particular emphasis was placed on prospective study designs assessing sleep prior to infection, enabling stronger causal inference (Salfi et al., 2023). Methodological inconsistencies and knowledge gaps, such as those related to the "missing link"

role of melatonin, were also identified to inform future research directions (Szataniak and Packi, 2025).

3. Analysis and Synthesis of Literature

3.1 Short Sleep Duration, Insomnia, and Chronic Inflammation

Chronic sleep deprivation and persistent insomnia are recognized risk factors for inflammatory disorders such as type 2 diabetes and atherosclerosis (Kuna et al., 2023; Poluektov, 2021). Sustained sleep loss induces systemic inflammation, thereby increasing infection susceptibility through impaired immune function (Asif et al., 2017; Besedovsky et al., 2019). A key pathological mechanism involves increased secretion of pro-inflammatory cytokines. In individuals with chronic insomnia, the circadian peak of IL-6 and TNF- α secretion shifts from nighttime to daytime, in contrast to healthy sleepers, contributing to fatigue and immune dysregulation (Irwin, 2023; Irwin et al., 2006). Additionally, short sleep duration (<7 hours), particularly when accompanied by insomnia symptoms, is associated with elevated C-reactive protein levels (Irwin, 2023; Lee and Park, 2024).

Sleep deficiency adversely affects both innate and adaptive immunity. Natural killer (NK) cells, which constitute a first-line defense against viral infections, show reduced activity following partial sleep deprivation (Besedovsky et al., 2019; Ibarra-Coronado et al., 2015). Similarly, decreased numbers of CD3⁺, CD4⁺, and CD8⁺ T lymphocytes have been observed in individuals with insomnia, indicating impaired adaptive immune competence (Nie et al., 2022; Poluektov, 2021).

3.2 Sleep Quality and Vaccine Effectiveness

Sleep plays a fundamental role in the formation of immunological memory, directly influencing vaccine efficacy (Chervin, 2024; Schmitz et al., 2022). Multiple studies demonstrate that reduced sleep duration during the peri-vaccination period weakens immune responses. Among healthcare workers, insomnia severity measured using the Athens Insomnia Scale (AIS) independently predicted lower IgG antibody levels following the second dose of the BNT162b2 mRNA COVID-19 vaccine (Athanasίου et al., 2022). Poor sleep quality, assessed by the Pittsburgh Sleep Quality Index (PSQI >5), similarly correlated with reduced antibody titers (Athanasίου et al., 2022; Chen et al., 2025).

Experimental studies show that a single night of sleep deprivation following hepatitis A vaccination halves antibody production and impairs T-cell immunological memory, with effects persisting for up to one year (Besedovsky et al., 2019; Irwin, 2023). Objective measurements using actigraphy indicate that short sleep duration (≤ 6 hours) reduces antibody

responses to hepatitis B and influenza vaccines and decreases the likelihood of achieving clinical protection (Jaiswal et al., 2024; Schmitz et al., 2022).

Slow-wave sleep (SWS) is particularly important for immunological memory consolidation, facilitating interactions between antigen-presenting cells and T lymphocytes through a hormonal milieu characterized by increased growth hormone and prolactin and reduced cortisol levels (Asif et al., 2017; Besedovsky et al., 2019).

3.3 Increased Susceptibility to Viral Infections

Sleep disturbances increase vulnerability to viral infections. Individuals sleeping fewer than 7 hours per night exhibit nearly threefold higher risk of developing clinical rhinovirus infection compared with those sleeping at least 8 hours (Besedovsky et al., 2019; Poluektov, 2021). Low sleep efficiency ($\leq 92\%$) further amplifies this risk (Martínez-Albert et al., 2025). Short sleep duration is also associated with increased incidence of respiratory infections at the population level (Feuth, 2024; Martínez-Albert et al., 2025).

Epidemiological studies indicate that poor sleep quality is linked to higher risk of SARS-CoV-2 infection and hospitalization (Quan et al., 2023). A U-shaped association between sleep duration and COVID-19 risk has been observed, with both short (≤ 6 hours) and excessively long sleep durations conferring elevated risk (Quan et al., 2023; Salfi et al., 2023). Poor pre-infection sleep quality predicts prolonged recovery and increased likelihood of long COVID symptoms (Salfi et al., 2023).

3.4 Obstructive Sleep Apnea and Immune Dysfunction

Obstructive sleep apnea (OSA) is strongly associated with immune dysregulation resulting from intermittent hypoxia and sleep fragmentation (Chen et al., 2025; Kuna et al., 2023). Disease severity correlates inversely with vaccine-induced immune responses, particularly neutralizing antibody titers following SARS-CoV-2 vaccination (Chen et al., 2025). Severe untreated OSA is associated with reduced frequencies of circulating T follicular helper cells and antibody-secreting cells (Chen et al., 2025; Lee and Park, 2024). Positive airway pressure therapy effectively reverses these alterations by mitigating hypoxia and sleep fragmentation, thereby restoring immune competence (Chen et al., 2025).

4. Discussion

The analyzed literature confirms that sleep is not merely a passive resting state but an active homeostatic process through which the brain regulates immune function at cellular and genetic levels (Asif et al., 2017; Chervin, 2024). Chronic sleep deficiency induces low-grade inflammation via activation of NF- κ B signaling pathways, contributing to immune dysregulation and increased infection risk (Irwin, 2023; Irwin et al., 2006). The role of

melatonin as a circadian and immunomodulatory mediator further underscores the importance of sleep in maintaining immune homeostasis (Szataniak and Packi, 2025).

Clinical implications are significant, as improving sleep quality through behavioral or pharmacological interventions—including melatonin supplementation or treatment of OSA—may represent an effective strategy for enhancing immune defense and vaccine effectiveness (Chen et al., 2025; Szataniak and Packi, 2025). Despite robust evidence from experimental models, further longitudinal studies are needed to determine the clinical impact of sleep hygiene on infection prevention in diverse populations (Ibarra-Coronado et al., 2015; Schmitz et al., 2022).

5. Conclusions

1. Sleep is an essential modulator of innate and adaptive immunity, and its deficiency disrupts leukocyte trafficking and immunological memory formation (Asif et al., 2017; Besedovsky et al., 2019).
2. Sleep disorders, particularly severe OSA and insomnia, constitute significant risk factors for viral infections and adverse clinical outcomes (Chen et al., 2025; Quan et al., 2023).
3. Optimizing sleep duration and continuity during vaccination periods is crucial for maximizing immunological protection (Athanasίου et al., 2022; Schmitz et al., 2022).
4. Effective treatment of sleep disorders can reverse pro-inflammatory immune alterations and reduce infection-related risks (Chen et al., 2025; Irwin, 2023).
5. Sleep hygiene should be integrated into preventive healthcare strategies, especially for high-risk populations (Kuna et al., 2023; Martínez-Albert et al., 2025).

Disclosure:

Author's contribution:

Conceptualization: Anna Waldman, Maja Kowalczyk, Magdalena Wolff

Methodology: Kalina Kreft, Agata Sobczak, Lena Głuszkiewicz

Formal analysis: Anna Waldman, Lena Głuszkiewicz

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Artificial intelligence (AI) was used only for language enhancement purposes, such as grammar correction and stylistic refinement.

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