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SLEEP QUALITY AS A MODULATOR OF IMMUNE RESPONSE AND INFECTIOUS RECOVERY: AN INTEGRATIVE REVIEW

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Abstract : Sleep quality and duration play a central role in regulating the immune response and individual well-being, influencing recovery from infections and maintaining physiological homeostasis. The objective of this study was to analyze, through an integrative review of the literature, the influence of sleep on innate and adaptive immunity, as well as its impacts on susceptibility to infections, vaccine efficacy, and psychophysiological balance. Studies published between 2002 and 2025 were selected from the PubMed/MEDLINE, Scopus, Web of Science, ScienceDirect, and Google Scholar databases. The results show that sleeping less than six hours per night is associated with up to a fourfold increase in the risk of respiratory infections and up to a 50% reduction in antibody titers after vaccination against influenza, hepatitis, and COVID-19. Sleep deprivation and fragmentation raise levels of pro-inflammatory cytokines, such as IL-6 and TNF-α, reduce natural killer cell activity, and impair T and B lymphocyte differentiation, compromising immune memory. In addition, inadequate sleep patterns are associated with worse clinical outcomes, greater severity of viral infections, and reduced quality of life, with a higher prevalence of anxiety and depressive symptoms. It can be concluded that sleep acts as a physiological modulator that determines immunocompetence and well-being, constituting a relevant non-pharmacological strategy for the prevention of infectious diseases and the optimization of the vaccine response.

Keywords: Sleep; Immune system; Inflammation; Vaccination; Infections.

INTRODUCTION

Sleep plays a fundamental role in physiological homeostasis and immune system regulation, acting as an essential modulator of inflammatory response and resistance to infections (Besedovsky; Lange; Born, 2012). Recent evidence shows that sleep deprivation or poor sleep quality compromises T lymphocyte differentiation and activity, alters cytokine production, and reduces the efficiency of the adaptive immune response, negatively impacting recovery from infectious processes and overall well-being (Feuth, 2024; Garbarino et al., 2021). Adequate periods of sleep, in turn, favor the consolidation of immunological memory and optimize vaccine efficacy, while sleep fragmentation is associated with lower antibody production and greater susceptibility to respiratory and viral infections (Izuhara et al., 2023; Spiegel et al., 2023).

The interaction between sleep and immunity is regulated by complex neuroendocrine mechanisms, in which the hypothalamic-pituitary-adrenal (HPA) axis and melatonin play central roles (Irwin, 2002; Poluektov, 2021). During sleep, reduced cortisol and increased release of growth hormone and prolactin favor the recruitment of immunocompetent cells, such as T lymphocytes and NK cells (Lee; Park, 2024). In contrast, sleep restriction triggers a pro-inflammatory state characterized by elevated cytokines such as IL-6 and TNF-α, contributing to the development of metabolic, cardiovascular, and infectious disorders (Kuna et al., 2022; Feuth, 2024).

In addition to immunological repercussions, sleep quality directly influences physical and mental well-being. Sleep disorders are associated with reduced quality of

life, increased fatigue, and increased anxiety and depressive symptoms (Rayatdoost et al., 2022; Li et al., 2025). During infectious processes, sleep fragmentation impairs the differentiation of follicular helper T cells, which are essential for B lymphocyte maturation and effective antibody production, contributing to the greater severity and duration of infections, including those related to COVID-19 (Fernandes et al., 2020; Shafiee et al., 2023; Walsh et al., 2023). Contemporary literature reinforces that the relationship between sleep and immunity is bidirectional: while chronic sleep deprivation induces immune dysfunctions similar to those observed in infectious processes, infection itself alters sleep patterns through the action of inflammatory mediators, such as interleukins and prostaglandins (Besedovsky; Lange; Born, 2012; Feuth, 2024).

OBJECTIVES

The main objective of this study is to analyze, in light of contemporary scientific evidence, the influence of sleep quality and duration on the immune response and well-being of individuals, emphasizing the role of sleep as an essential physiological modulator in the processes of recovery from infections and maintenance of systemic homeostasis.

In addition, we propose to investigate the neuroimmunological mechanisms that underpin the relationship between sleep and the regulation of innate and adaptive immunity, considering the modulation of pro- and anti-inflammatory cytokines, natural killer cell activity, and T and B lymphocyte dynamics. It also aims to assess the impacts of sleep deprivation and fragmentation on susceptibility to infections, vaccine response, and associated clinical outcomes.

Finally, it seeks to integrate evidence about the interactions between sleep quality, psychophysiological balance, and subjective well-being, discussing the potential for improving sleep hygiene as a non-pharmacological intervention aimed at optimizing immunocompetence and overall health.

METHODOLOGY

This study is an integrative literature review, whose objective is to critically analyze and synthesize the available knowledge on the effects of sleep on the immune response and well-being of individuals, with an emphasis on the role of sleep quality as a modulating factor in the recovery from infections. The choice of this type of review is justified by the possibility of bringing together results from theoretical and empirical research, allowing a comprehensive understanding of the topic, to integrate physiological, clinical, and epidemiological evidence that demonstrates the relevance of sleep for maintaining health and preventing diseases.

To construct the analytical body, 15 original scientific articles and reviews available in recognized academic databases, such as PubMed/MEDLINE, Scopus, Web of Science, ScienceDirect, and Google Scholar, were selected. The inclusion criteria defined were: studies published between 2002 and 2025, in Portuguese and English, that directly address the relationship between sleep quality or duration, immune response, infections, and indicators of physical and mental well-being. Studies ranging from physiological and immunological mechanisms (such as changes in T cells, cytokines, and antibodies) to clinical and epidemio-

logical research on infections, vaccination, inflammation, and sleep-related health outcomes were considered.

The search strategy used a combination of English and Portuguese descriptors: "sleep quality," "sleep deprivation," "immune response," "infection recovery," "well-being," "sleep and immunity," in addition to their Portuguese equivalents ("qualidade do sono," "privação do sono," "resposta imunológica," "recuperação de infecções," "bem-estar"), linked by the Boolean operators AND and OR, in order to broaden the scope and precision of the search.

The article selection process was conducted in two stages: initially, screening was performed based on titles and abstracts, excluding studies not directly related to the proposed theme; then, the selected texts were read in full to assess their relevance and contribution to the research objective. Duplicate studies were excluded, as were those with an approach limited to disorders not related to immunity or infections, as well as articles without original data or data relevant to the proposed discussion.

After selecting and analyzing the material, the data were organized systematically, allowing for the construction of a critical synthesis of the findings. The results were structured to elucidate the relationship between sleep quality, immune response, recovery from infections, and well-being, highlighting the pathophysiological mechanisms involved, the available clinical evidence, and the practical implications for health promotion and disease prevention.

RESULTS

Analysis of the included studies consistently shows that changes in sleep duration and architecture have a direct impact on central components of the immune system. In terms of innate immunity, both total and partial sleep deprivation trigger measurable inflammatory responses. Total sleep deprivation is associated with an immediate increase in the number of circulating leukocytes, especially neutrophils, characterizing an acute physiological stress response, with rapid reversal after a night of recovery (Ruiz et al., 2010; Garbarino et al., 2021). In contrast, partial sleep deprivation, even in the short term, promotes a sustained elevation of pro-inflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-α), in addition to an increase in C-reactive protein, configuring a state of low-grade systemic inflammation (Irwin, 2002; Feuth, 2024). Additionally, selective REM sleep deprivation is associated with a persistent reduction in serum immunoglobulin A, indicating compromised mucosal immunity, with relevant implications for the defense of the respiratory and gastrointestinal tracts (Ruiz et al., 2010; Kuna et al., 2022).

With regard to adaptive immunity, experimental and physiological evidence indicates that sleep deprivation compromises essential stages of activation, differentiation, and maintenance of the specific immune response. Studies show that total sleep deprivation results in a persistent increase in the proportion of CD4+ T lymphocytes, with slower recovery when compared to that observed in innate immunity parameters (Ruiz et al., 2010). In addition, the onset of NREM sleep, particularly slow-wave sleep, is accompanied by an increase in cytokines

such as IL-1 and IL-2, which are essential for clonal expansion and T cell differentiation (Besedovsky, Lange & Born, 2012). Sleep restriction in the second half of the night, a period characterized by a greater predominance of REM sleep, negatively interferes with this temporal sequence, impairing T and B lymphocyte differentiation and immune communication in germinal centers (Fernandes et al., 2020). These findings indicate that the physiological architecture of sleep is decisive for the formation and consolidation of immunological memory.

Circadian rhythms emerge as central elements in the coordination of immune function. Observational and experimental evidence shows that misalignment between the sleep-wake cycle and the endogenous circadian rhythm, as occurs in night workers, is associated with dysregulation of innate and adaptive immunity, increased risk of infections, and reduced vaccine response (Lee & Park, 2024). Molecules such as IL-1β, TNF-α, and nitric oxide exert dual functions as inflammatory mediators and sleep regulators, reinforcing the interdependence between the immune, neuroendocrine, and circadian systems (Poluektov, 2021; Feuth, 2024).

Chronic sleep loss is consistently associated with the development of a low-grade systemic inflammatory state. Longitudinal and experimental studies demonstrate persistent elevations of IL-6, TNF-α, and C-reactive protein in individuals with insufficient sleep, regardless of concomitant behavioral factors (Irwin, 2002; Garbarino et al., 2021). This inflammatory pattern is observed in both acute sleep deprivation and chronic sleep fragmentation, and is associated with immune dysfunction and the worsening of preexisting inflammatory conditions.

The relationship between sleep and vaccine response is one of the most robust findings in the literature. Meta-analyses have shown that individuals who sleep less than six hours per night have a significant reduction—up to 50%—in antibody production after vaccination against influenza, hepatitis A and B, and COVID-19 (Spiegel et al., 2023). Additional evidence indicates that both the duration and quality of sleep in the days following vaccination directly influence antibody titers after mRNA vaccines, regardless of age, sex, or intensity of adverse reactions (Izuhara et al., 2023). Systematic reviews reinforce the role of sleep as a natural physiological adjuvant of the adaptive immune response (Rayatdoost et al., 2022).

There is consistent consensus that insufficient sleep increases susceptibility to infections. Experimental trials show that individuals with less than six hours of sleep are up to four times more likely to develop respiratory infections after viral exposure, while low sleep efficiency increases this risk in a dose-dependent manner (Walsh et al., 2023). Population studies corroborate these findings by demonstrating that healthy sleep patterns are associated with a reduced risk of hospitalizations for infections in general and for liver infections in particular (Li et al., 2025).

In addition to increasing susceptibility, sleep disorders are associated with more severe infections. Recent meta-analyses confirm that poor sleep quality and chronic disorders significantly increase the risk of severe forms of COVID-19, including hospitalization (Shafiee et al., 2023). Proposed mechanisms include low-grade chronic in-

flammation, reduced natural killer cell activity, and impaired T-cell-mediated antiviral responses (Garbarino et al., 2021). In this context, melatonin has been investigated as a modulator of the inflammatory response in COVID-19, reinforcing the relevance of circadian rhythms in antiviral immunity.

Specific populations have increased immune vulnerability due to sleep disturbances. Individuals with depression often exhibit sleep disorders associated with reduced NK cell activity and lower lymphocyte responsiveness, increasing the risk of infections (Feuth, 2024). In individuals with alcoholism, fragmented sleep and reduced deep sleep persist even after prolonged periods of abstinence, accompanied by immunosuppression and increased susceptibility to infectious processes (Kuna et al., 2022).

Finally, recent studies suggest a bidirectional relationship between sleep architecture, gut microbiota, and immune function. Sleep disturbances can alter the composition and circadian rhythmicity of the gut microbiome, interfering with the production of immunomodulatory metabolites, such as short-chain fatty acids, which directly influence regulatory T cells and inflammatory mediators (Feuth, 2024). Conversely, changes in the microbiome can interfere with sleep quality by modulating neurotransmitters such as serotonin, highlighting the functional integration between sleep, the gastrointestinal tract, and systemic immunity. The main findings of the studies, as well as their designs and key findings, are summarized in Table 1.

When combining findings from different methodological designs, it is observed that the literature converges in demonstrating the central role of sleep as a modulator of innate and adaptive immunity, influencing everything from basic inflammatory processes to vaccine efficacy and susceptibility to acute infections. Although the studies are heterogeneous in terms of population, sleep assessment instruments, and immunological markers investigated, the results consistently indicate that changes in sleep quality, duration, and architecture directly affect immunological homeostasis. This integration of evidence provides a solid basis for understanding the physiological mechanisms involved and highlights gaps that warrant further investigation.

DISCUSSION

An integrated analysis of the literature consistently demonstrates that sleep plays a central role in modulating the immune response and psychophysiological balance, making it an essential determinant of health. The quality and duration of sleep influence both innate and adaptive immunity components, modulating the neuroendocrine environment that supports the body's defenses. Evidence from classical and contemporary studies reinforces that total or partial sleep deprivation triggers an inflammatory state characterized by elevated cytokines such as IL-6 and TNF-α, while adequate sleep favors the activation and recruitment of immunocompetent cells, such as T lymphocytes and NK cells (Lee; Park, 2024; Kuna et al., 2022). This body of evidence demonstrates that sleep not only reflects overall health, but also actively influences the mechanisms that preserve immune homeostasis.

Another key aspect concerns the influence of different sleep stages on the regulation of immune mechanisms. Studies by Besedovsky, Lange, and Born (2012)

Author/Year	Main Results	Conclusions
Besedovsky et al., 2012	Sleep, especially slow-wave NREM sleep, promotes an increase in pro-immune cytokines (IL-1, IL-2) and favors communication between the neuroendocrine and immune systems, enhancing the activation and differentiation of T lymphocytes.	Sleep plays an active and regulatory role in adaptive immunity and is essential for the formation of immunological memory.
Fernandes et al., 2020	Distúrbios do sono durante infecção prejudicam a diferenciação de células T foliculares auxiliares (Tfh), comprometendo a formação de centros germinativos e a resposta humoral.	Sleep disturbances during infection impair follicular helper T cell (Tfh) differentiation, compromising germinal center formation and humoral response.
Feuth, 2024	Sleep deprivation and fragmentation increase low-grade systemic inflammation, alter the function of NK cells, T and B lymphocytes, and increase the risk of infections and worsen clinical prognosis.	Adequate sleep modulates immunity, and its disruption contributes to chronic inflammation and increased susceptibility to infections.
Garbarino et al., 2021	Sleep deprivation is associated with increased inflammatory cytokines, reduced NK cell activity, and an increased risk of immune-mediated and infectious diseases, including COVID-19.	Insufficient sleep is a risk factor for immune dysfunction and worse clinical outcomes.
Irwin, 2002	Sleep loss elevates levels of IL-6, TNF-α, and C-reactive protein, in addition to compromising cellular and humoral immune responses.	Sleep acts as an essential regulator of inflammation and systemic immune function.
Izuhara et al., 2023	Shorter sleep duration after vaccination with mRNA against SARS-CoV-2 is associated with reduced anti-Spike antibody titers, regardless of age and sex.	Sleep duration directly influences the effectiveness of the vaccine response.
Kuna et al., 2022	Chronic sleep deprivation is associated with immunosuppression, reduced IgA, NK cell dysfunction, and increased susceptibility to infections, especially in clinical populations.	Sleep deprivation compromises multiple aspects of immunity, with significant clinical impact.
Lee & Park, 2024	Sleep disturbances and circadian misalignment impair innate immunity, affecting neutrophils, macrophages, and cytokine production.	Sleep and circadian rhythms are crucial for the effectiveness of innate immunity.
Li et al., 2025	Healthy sleep patterns are associated with a significant reduction in the risk of hospitalization for infections, including liver and respiratory infections.	Healthy sleep acts as a protective factor against infections in the population.
Poluektov, 2021	Inflammatory molecules such as IL-1β and TNF-α participate in both sleep regulation and immune response, demonstrating physiological interdependence.	Sleep and immunity form an integrated system mediated by common neuroimmune pathways.
Rayatdoost et al., 2022	Adequate sleep and timing of vaccination positively influence immune response and antibody production.	Sleep acts as a natural physiological adjuvant to vaccination.

Ruiz et al., 2010	Total sleep deprivation increases leukocytes and neutrophils, while selective REM sleep deprivation reduces serum IgA and compromises mucosal immunity.	Different stages of sleep have specific and complementary effects on immunity. REM sleep is essential for mucosal immunity.
Shafiee et al., 2023	Sleep disorders increase the risk of SARS-CoV-2 infection and progression to severe forms of COVID-19.	Inadequate sleep is a significant risk factor for the severity and acquisition of COVID-19.
Spiegel et al., 2023	Individuals who sleep less than 6 hours show up to a 50% reduction in post-vaccination antibody response.	Insufficient sleep substantially compromises vaccine efficacy.
Walsh et al., 2023	Good subjective sleep quality reduces the risk of respiratory infection even during periods of sleep restriction.	Sleep quality is an independent protective factor against respiratory infections.

Table 1 – Main Studies Included on Sleep and Immune Response

Source: produced by the authors

and Feuth (2024) highlight that deep sleep and REM sleep participate in lymphocyte differentiation and immune memory consolidation. During these periods, there is a reduction in cortisol and an increase in hormones such as prolactin and GH, creating an immunoprotective environment (Irwin, 2002; Poluektov, 2021). The disruption of this cycle—whether due to deprivation, fragmentation, or circadian misalignment—alters the dynamics between the neurological, endocrine, and immune axes, contributing to low-grade chronic inflammation and greater vulnerability to metabolic and infectious dysfunctions.

The findings also converge in demonstrating that sleep significantly influences vaccine efficacy. Studies by Spiegel et al. (2023) and Izuhara et al. (2023) show substantial reductions in antibody titers among individuals with insufficient sleep, especially after vaccines that rely heavily on the humoral response, such as influenza and COVID-19. Additional research shows impaired differentiation of follicular helper T cells in sleep disorder scenarios, a mechanism essential for B lymphocyte maturation and antibody production (Fernandes et al., 2020). Thus, it is clear that interventions aimed at optimizing sleep can represent a low-cost, high-impact complementary resource for increasing vaccine effectiveness in vulnerable populations.

In the clinical field, the studies reviewed indicate that healthy sleep patterns reduce susceptibility to respiratory, urinary, and systemic infections, in addition to decreasing the risk of progression to severe conditions, including COVID-19 (Shafiee et al., 2023; Walsh et al., 2023). These results highlight the potential of sleep as a prognostic marker and therapeutic target. However, the literature shows important disparities: while some observational studies indicate a strong association between sleep deprivation and infectious risk, studies using other methodologies suggest that behavioral and psychosocial factors may mitigate or amplify these effects. This variability highlights the need for caution when interpreting causal associations, especially in self-reported or uncontrolled sample-based research.

The psychophysiological impact of sleep disorders also occupies a relevant space in the discussion. Sleep disturbances are often associated with poorer mental health, with increased symptoms of depression and anxiety, which in turn modulate immunity through neuroendocrine pathways (Rayatdoost et al., 2022; Li et al., 2025). This interconnection reinforces that sleep-focused interventions can have multidimensional effects, benefiting not only the immune response but also emotional balance and overall well-being.

Despite this robust body of evidence, the literature has methodological limitations that must be considered. Among these are: wide heterogeneity in sleep assessment methods; predominance of observational studies; variation in the immune markers assessed; and a scarcity of longitudinal studies capable of elucidating cause-and-effect relationships. In addition, few studies consider confounding factors such as stress, dietary patterns, gut microbiota, and individual chronotype. These limitations reinforce the need for controlled clinical trials investigating the impact of sleep on specific immune parameters and long-term clinical outcomes.

From a clinical and public health perspective, the findings of this review point to the need to systematically incorporate sleep assessment into protocols for the prevention, monitoring, and treatment of infectious diseases. Measures such as sleep hygiene education, regularization of the sleep-wake cycle, reduction of nighttime exposure to artificial light, and stress management can be implemented as complementary strategies in medical practice. In specific populations—such as the elderly, night workers, immunosuppressed patients, and individuals with psychiatric disorders—such interventions take on even greater importance.

For future research, it is recommended to further investigate: (a) the impact of selective deprivation of specific sleep stages on distinct immune mechanisms; (b) the interaction between sleep, gut microbiota, and immunity; (c) the modulatory role of melatonin in respiratory and systemic infections; (d) personalized sleep strategies, considering chronotype and environmental factors; and (e) clinical trials testing behavioral or pharmacological interventions aimed at optimizing sleep as adjuvants in the vaccine response.

In summary, the results presented demonstrate that sleep plays a multifaceted role in regulating immunity, influencing everything from cellular mechanisms to clinical and psychosocial outcomes. The discussion of the findings highlights not only the biological relevance of this process, but also its clinical and translational potential. Thus, recognizing sleep as a central determinant of health represents a fundamental step toward the development of more integrated care practices and the advancement of scientific knowledge about the interface between sleep, immunity, and well-being.

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