ŻEREK, Marta and SITAREK, Gracjan. Sleep Quality and Immune Function: Implications for Overall Health – A Literature Review. Journal of Education, Health and Sport. 2024;75:56048. eISSN 2391-8306.
https://dx.doi.org/10.12775/JEHS.2024.75.56048
https://apcz.umk.pl/JEHS/article/view/56048

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 07.11.2024. Revised: 20.11.2024. Accepted: 05.12.2024. Published: 05.12.2024.

Sleep Quality and Immune Function: Implications for Overall Health – A Literature Review

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Abstract:

Introduction

Sleep quality is increasingly acknowledged as critical for immune function and overall health. Poor sleep can disrupt immune response, elevating inflammation and susceptibility to infection and chronic diseases. Given the prevalence of sleep disturbances across populations, understanding their impact on immune health and overall wellness is essential.

Purpose of Work

This review aims to examine the relationship between sleep quality and immune function

by synthesizing findings on how poor sleep contributes to immune suppression, heightened inflammation, and mental health disturbances, particularly in vulnerable populations. This review also explores possible interventions to mitigate these effects.

Material and Methods

A literature search was conducted on databases including PubMed and Google Scholar, focusing on studies from 2012 to 2023. Articles assessing sleep quality, immune markers, and the mediating roles of stress and depression were reviewed. Criteria included studies with both subjective and objective assessments of sleep, immune function, and relevant mental health markers.

Summary

Findings indicate that poor sleep quality negatively impacts immune health, primarily through increased systemic inflammation and decreased immune cell functionality. Depression and stress further exacerbate these effects, particularly in populations with chronic illnesses or high psychological distress. Targeted interventions that improve sleep quality and manage stress may bolster immune resilience and overall health. Future research should continue to address these interactions across diverse populations to develop targeted interventions.

Keywords: Sleep quality, Immune function, Inflammation, Depression

Introduction:

Sleep plays a fundamental role in maintaining immune function, cognitive health, and emotional resilience, forming the basis of optimal health and well-being. Poor sleep quality, however, is prevalent across populations, affecting between 20% and 30% of adults globally, with higher rates among individuals with chronic conditions, high-stress occupations, and aging

populations. Increasingly, studies have shown that sleep disturbances can significantly impair immune responses, leading to heightened risks of infection, chronic inflammatory diseases, and even mental health disorders such as depression and anxiety (Cai et al., 2022; Passos et al., 2014).

Understanding the biological mechanisms through which poor sleep impacts immune function provides insight into the broader implications for overall health. Sleep disturbances lead to immune suppression and an inflammatory cascade, evidenced by elevated levels of markers such as cytokines and a reduction in critical immune cells like T-cells. In healthy sleep, a balance of pro- and anti-inflammatory responses supports immune regulation; in contrast, poor sleep disrupts this balance, contributing to chronic low-grade inflammation. This inflammatory state not only compromises the body's ability to fight infections but is also associated with increased risks for conditions such as cardiovascular disease and diabetes (Lueke & Assar, 2022).

The relationship between sleep and immune function is further complicated by the role of mental health. Poor sleep often contributes to stress and depressive symptoms, which can compound immune dysfunction. Depression, for example, is associated with elevated systemic inflammation and dysregulated immune markers, factors that may worsen immune outcomes in individuals already experiencing poor sleep (Passos et al., 2014). Furthermore, the interaction of sleep, mental health, and immunity can create a negative feedback loop. Chronic sleep issues exacerbate stress and depression, leading to further immune suppression and increasing vulnerability to physical and mental health complications.

As sleep and immune health become central topics in public health, particularly in the context of pandemic-related concerns and rising levels of chronic illness, it is crucial to investigate effective interventions. Aerobic exercise, cognitive-behavioral therapy, and relaxation techniques have shown promise in improving sleep quality, reducing depressive symptoms, and enhancing immune resilience. This review synthesizes current literature on the impact of sleep quality on immune function, examining the complex interactions of sleep, mental health, and immunity. The review also aims to highlight possible therapeutic approaches that can mitigate the adverse health impacts of poor sleep.

Methodology:

This literature review was conducted based on studies retrieved from PubMed, Google Scholar, and related academic databases. The search was limited to articles published between 2012 and

2023 to capture the most recent findings. Key search terms included "sleep quality," "immune function," "inflammation," "depression," and "stress." Studies were selected if they investigated the relationship between sleep and immune markers or mental health mediators such as stress and depression. Both observational and experimental studies were included to provide a comprehensive view of how sleep impacts immune function.

Discussion:

Sleep quality is a crucial determinant of immune function, with evidence indicating that insufficient or disturbed sleep weakens immune resilience. This relationship is especially complex because poor sleep not only affects immune markers directly but also interacts with psychological factors, such as stress and depression, which further exacerbate immune dysfunction.

1. Regulation of Sleep Homeostasis

Increased neural activity consumes energy, which may lead to the release of ATP into the extracellular space, resulting in a rise in extracellular adenosine concentration. Enhanced cytokine release can be directly triggered by neural activity or as a response to energy depletion. Cytokines are capable of activating multiple signaling pathways, including iNOS and NF-kB, which further induce an increase in adenosine levels, thereby reducing neural activity. TNFα may directly influence neural plasticity. The figure illustrates the key elements and their interactions in one configuration. While the main players remain the same, the order of events and the relative importance of each element are still under discussion (Porkka-Heiskanen, 2013).

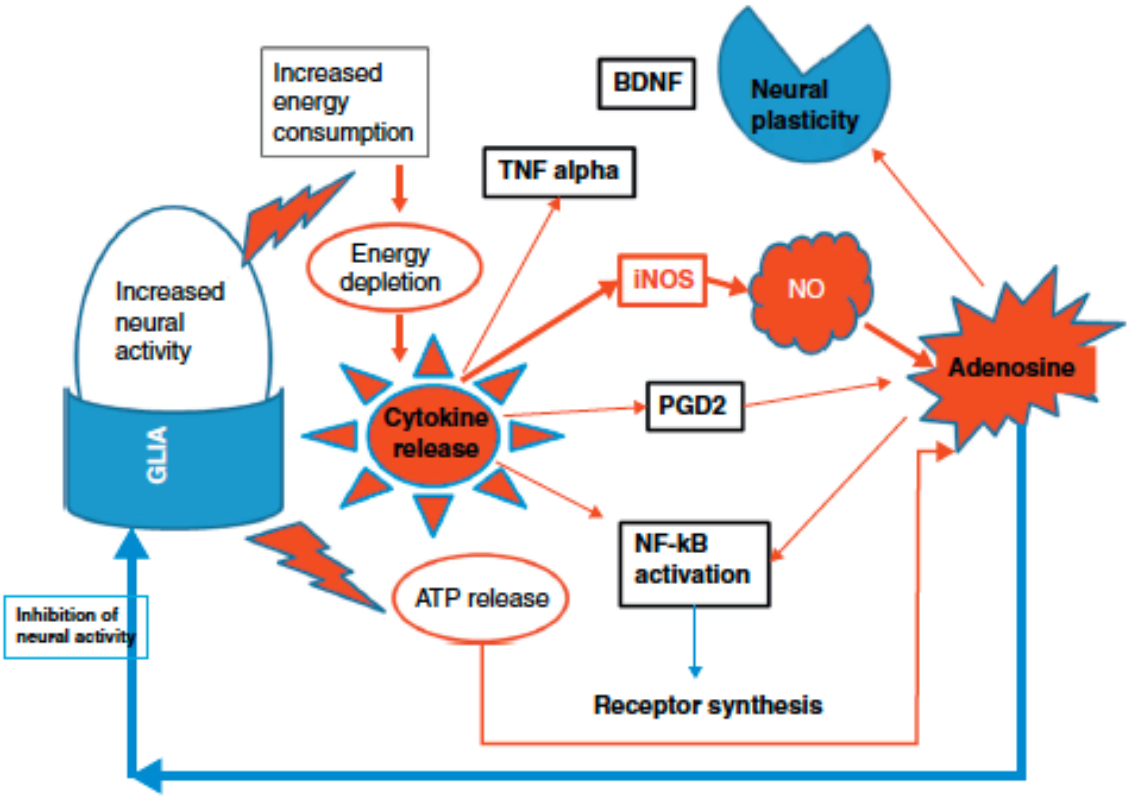

Figure 1. Key Players in the Regulation of Sleep Homeostasis (Porkka-Heiskanen, 2013).

The role of astrocytes in sleep and sleep homeostasis is a newly emerging area of study, with far more questions than certainties. Astrocytes influence neuronal activity through several mechanisms: gliotransmission, ion buffering, glutamate clearance, metabolic support, and non-vesicular exocytosis. The specific contributions of these processes to sleep homeostasis remain largely unknown, as does the precise brain region where astrocytes impact sleep. Since astrocytes are widely dispersed across subcortical and cortical areas, including regions associated with sleep and wake regulation, they may influence sleep and brain function either globally or through targeted modulation of established sleep and arousal centers. Moreover, it is unclear if similar astrocytic mechanisms regulate human sleep. Interestingly, recent studies highlight that human astrocytes are larger, more diverse, and more complex than those in other mammals, with a 2.6-fold larger diameter and numerous, long-reaching processes in the cortex compared to those in rodents. This suggests that astrocytic regulation of sleep in humans might be as extensive, if not greater, than observed in animal models. These intriguing questions highlight exciting possibilities for future research (Frank, 2013).

Lazarus et al. (2013) emphasize the basal ganglia's role in balancing wakefulness and sleep,

which is modulated by neurotransmitters like dopamine and adenosine. Since the basal ganglia help maintain wakefulness, any dysfunction in these circuits may lead to sleep fragmentation, impacting immune resilience by impairing restorative sleep phases. Such findings suggest potential therapeutic targets within the basal ganglia for improving sleep quality and, consequently, immune function.

In general, structural changes in synapses occur more slowly than molecular and electrophysiological changes, but they can still take place within a few hours—a timeframe compatible with the physiological sleep/wake cycle (Cirelli, 2013). Recent studies have documented the growth in the number and/or size of synapses over several hours of wakefulness in multiple neuronal circuits in flies. For example, the axonal tips of the γ neurons in the mushroom bodies, which play roles in olfactory learning and sleep regulation, increase in size after 7 hours of either spontaneous or enforced wakefulness compared to 7 hours of sleep. Similarly, the number of dendritic spines in the first giant tangential neuron of the lobula plate vertical system, part of the visual processing system, also increases after 7 hours of sleep deprivation (Cirelli, 2013). Notably, the number of spines in these neurons is similar in flies that are either sleeping or awake but housed in a small, empty tube. In contrast, when flies spend 12 hours of spontaneous wakefulness in an enriched environment, both dendritic branch length and spine number increase significantly compared to levels observed in sleeping flies. Furthermore, synaptic morphology returns to pre-enrichment levels only if flies are allowed to sleep; it does not revert if they are kept awake following the enrichment period. These findings suggest that in young adult flies, rapid synaptic growth can result from wakeful experiences, with sleep playing a restorative role that reverses these morphological changes (Cirelli, 2013).

2. Immune Implications of Poor Sleep

In healthy sleep, immune function follows a circadian rhythm, which optimally coordinates pro- and anti-inflammatory processes. During sleep, the body upregulates specific immune responses, preparing for potential threats upon waking. However, poor sleep disrupts this balance, reducing the body's defense capabilities. Sleep deprivation has been linked to increased levels of pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which contribute to a persistent state of low-grade inflammation. Chronic inflammation compromises immune health, rendering the body more susceptible to infections and chronic illnesses (Cai et al., 2022). For instance, Cai et al. (2022) demonstrated that patients with coronary artery disease who also had depression showed poorer sleep quality and elevated systemic inflammation compared to non-depressed patients with the same

condition. This indicates that depression amplifies the negative impact of poor sleep on immunity, likely through heightened inflammation. In addition to its impact on inflammation, poor sleep has been associated with decreased T-cell activity, including lower CD4+ and CD8+ T-cell ratios, which are essential for adaptive immunity. Mahlios et al. (2013) discuss how narcolepsy, a sleep disorder with autoimmune characteristics, involves specific immune pathways such as T-cell receptor involvement and associations with HLA gene polymorphisms. These genetic markers suggest that immune dysregulation may directly impact sleep, leading to potential implications for how immune-mediated pathways could disrupt sleep in other autoimmune contexts.

Recent studies have shown that clock genes, such as *Per2*, *Cry*, and *Npas2*, play a critical role in regulating sleep homeostasis, directly impacting circadian rhythms and immune functionality. These genes help synchronize sleep patterns with physiological processes, enabling the immune system to respond more effectively to threats. Disruptions in clock gene function, often caused by sleep deprivation or irregular sleep patterns, impair immune resilience and have been linked to chronic inflammatory states and metabolic disorders (Franken, 2013). This connection highlights the complex relationship between circadian rhythms, sleep quality, and immune health. Kurien et al. (2013) describe how genes like PER2, which regulates sleep timing, also play a role in immune responses, suggesting a genetic basis for the interplay between sleep and immune function. The paper notes that sleep phase genes might contribute to immune resilience, reinforcing how genetic variation in sleep-related genes may influence susceptibility to immune-related health conditions.

The interaction between sleep and immune function is also heavily influenced by glial cells, such as microglia and astrocytes, which play an active role in modulating immune responses to sleep deprivation. Glial cells respond to sleep loss by releasing pro-inflammatory cytokines like interleukin-1β (IL-1β) and tumor necrosis factor-alpha (TNF-α), which contribute to a low-grade inflammatory state. These responses suggest that microglia and astrocytes are essential mediators in the brain's immune response to disrupted sleep, linking chronic sleep disturbances with inflammation and neural dysfunction (Ingiosi et al., 2013). This glial modulation is especially relevant in aging populations, where sleep and immune functions tend to decline in tandem, increasing vulnerability to age-related diseases. Cytokines, especially interleukin-1 (IL-1) and tumor necrosis factor-alpha (TNF-α), are fundamental in regulating sleep and immune responses. Increased levels of these cytokines during prolonged wakefulness contribute to a low-grade inflammatory state, suggesting that disrupted sleep not only impacts immune resilience but can also drive inflammatory processes that lead to immune suppression

(Ganz, 2012).

As Hastings and Goedert (2013) discuss, disruptions in circadian rhythms influence protein aggregation, which has implications for neurodegenerative diseases. The effect of circadian misalignment on sleep indicates it may induce chronic inflammation within the immune system, raising susceptibility to infections and other chronic conditions. For example, the obstructive sleep apnea (OSA) is a highly complex disorder characterized by significant inter-individual variability in its pathophysiology. The reciprocal interactions between ventilation and behavioral state are believed to contribute to a vicious cycle of arousal, overbreathing, falling back asleep with underbreathing and airway closure, followed by arousal and overbreathing again. An especially low arousal threshold may predispose some individuals to OSA who might not otherwise experience it. OSA-related arousal is likely mediated by parabrachial (PB) neurons, which receive converging asphyxia-related signals and project to the arousal system. Moreover, a full understanding of hypercapnic arousal will require elucidating the role of serotonin, which may modulate responses at one or more synapses involved in detecting hypercapnia and relaying signals to arousal-inducing targets. Continued research into the pathogenesis of OSA-related arousal could pave the way for new therapeutic approaches (Chamberlin, 2013).

3. Role of Mental Health in Sleep-Immune Interactions

Mental health factors such as stress and depression serve as significant mediators in the sleep-immune function relationship. Stress and depressive symptoms, which often accompany poor sleep, further impair immune responses. Given the rise in mental health challenges, sleep quality in the United States has notably declined, with one in three American adults reporting sleep durations below the recommended minimum of 7 hours per night necessary for optimal health and general well-being. This increase in mental health concerns, alongside reduced sleep quantity and limitations in the American healthcare system, underscores the need for research into cost-effective, non-invasive strategies that may enhance mental well-being and sleep quality across the general population, including the often-overlooked younger adult demographic (Kisiolek et al., 2023). In a study on college students, found that poor sleep quality was linked to reduced immune function, with perceived stress and depression mediating this relationship. The sequential pathway they identified—where poor sleep increases perceived stress, which in turn exacerbates depressive symptoms and ultimately lowers immune resilience—illustrates a cascading effect of poor sleep on immune health. This cycle is supported by neurobiological evidence showing that sleep disturbances can dysregulate brain

regions associated with emotion regulation, such as the amygdala and prefrontal cortex. Poor sleep enhances amygdala reactivity to negative stimuli while weakening prefrontal control, leading to heightened stress perceptions and negative emotions. Over time, this neural pattern can contribute to chronic stress and depression, each of which further promotes inflammation and weakens immunity (Lueke & Assar, 2022).

Research highlights that non-REM (NREM) and REM sleep are regulated by distinct mechanisms, though both are subject to homeostatic control. This is particularly relevant as the restorative processes of sleep involve immune modulation, with prolonged wakefulness leading to increased sleep pressure, often marked by a rise in slow-wave activity (SWA) during subsequent sleep episodes (Porkka-Heiskanen, 2013).

A study involving partial sleep deprivation demonstrated that even short-term sleep loss could transiently impair immune function. Significant time-dependent changes were observed in immune markers like CD4 and CD8, as well as a decrease in HLA-DR expression, which could explain increased vulnerability to infections, particularly respiratory illnesses, after sleep disruption (Wilder-Smith et al., 2013).

Both acute and chronic sleep deprivation induce significant neurobehavioral changes that impact alertness, memory, and cognitive performance. Chronic sleep restriction appears to cause long-term neuromodulatory shifts that can take extended periods to recover fully, suggesting that consistent sleep loss impacts brain function at a fundamental level. Neurobehavioral responses to sleep loss show high individual variability, which may involve genetic factors, and evidence suggests that clock genes like *PER2* may serve as integrators of sleep and circadian signals, helping to regulate neurobehavioral stability (Basner et al., 2013).

In studies on sleep and memory, a frequent concern is the potential impact of circadian rhythms. Specifically, the question arises whether memory improvements seen after a night of sleep, as opposed to an equal period of wakefulness, reflect true sleep-dependent consolidation or merely circadian fluctuations in performance. For example, if individuals perform better in the morning than in the evening, it may create the appearance of memory improvement after sleep without actual changes in memory. One method for addressing this involves sleep deprivation studies, where participants are trained and tested at fixed times, with groups differing by whether sleep is allowed the night after training. Although sleep deprivation has its own potential confounds, studies that allow recovery sleep prior to testing consistently show poorer performance for sleep-deprived groups, suggesting it is the presence or absence of sleep, not circadian rhythms, that impacts performance (Stickgold, 2013).

Luppi et al. (2013) highlight the complex brainstem and hypothalamic interactions that underlie

the generation of REM sleep. Given that REM sleep is critical for immune and cognitive functions, understanding these mechanisms offers insight into how sleep architecture disturbances might exacerbate immune dysfunction, particularly in individuals with chronic stress or mental health disorders. Jagannath et al. (2013) emphasize the role of circadian rhythm disruption in mental health disorders, showing that circadian misalignment can exacerbate symptoms of schizophrenia and bipolar disorder. This supports the notion that immune system dysregulation due to poor sleep may also interact with the circadian misalignment observed in psychiatric disorders, thus creating a potential feedback loop between immune function and mental health.

4. Physical Interventions: The Role of Exercise

Physical activity, particularly aerobic exercise, has emerged as a promising non-pharmacologic intervention to improve sleep quality and, consequently, immune health. Passos et al. (2014) conducted a four-month aerobic exercise intervention in individuals with chronic insomnia and found improvements in sleep efficiency, reduced cortisol levels, and beneficial changes in immune markers, including increased T-cell activity. By enhancing sleep quality, exercise may reduce the body's inflammatory response and improve immune resilience, suggesting a beneficial feedback loop between physical activity and immune function. The benefits of exercise may also extend to mental health, as physical activity is known to lower stress and depressive symptoms. Exercise likely benefits immunity indirectly by improving sleep quality and reducing psychological stressors, thus mitigating the adverse effects of poor sleep on immune function.

5. Potential for Cognitive-Behavioral and Stress Management Interventions

Given the interplay of sleep, immune function, and mental health, cognitive-behavioral and stress management interventions may offer additional benefits. Cognitive-Behavioral Therapy for Insomnia (CBT-I), for example, has shown efficacy in treating sleep disturbances and has potential applications in populations vulnerable to immune suppression. By addressing sleep patterns and helping patients develop healthier coping mechanisms, CBT-I may reduce depressive symptoms and stress, ultimately benefiting immune health.

The significance of the relationship between sleep and immune function should not be underestimated. Effective treatment for insomnia may potentially reduce patients' susceptibility to immune-related diseases and enhance daytime functioning. Conversely,

improvements in sleep quality are likely to positively impact immune status. Research indicates that increasing sleep duration itself can reduce disease susceptibility (Donners et al., 2015).

Conclusion:

The literature reviewed highlights the complex relationship between sleep quality and immune function, showing that poor sleep can significantly impair immune resilience. This effect is compounded by stress and depression, which can create a feedback loop, further diminishing immunity. Aerobic exercise and behavioral therapies show promise as interventions to improve sleep quality and mitigate these effects. Addressing sleep quality as a core aspect of health interventions may offer meaningful improvements in immune health, particularly for populations at high risk for sleep disturbances and immune dysfunction.

Disclosure: Authors do not report any disclosures.
Author's contribution: All authors contribute to the article.
Conceptualization: Marta Żerek; **Methodology:** Marta Żerek; **Software:** Gracjan Sitarek; **Check:** Gracjan Sitarek; **Formal Analysis:** Marta Żerek; **Investigation:** Gracjan Sitarek; **Resources:** Gracjan Sitarek; **Data storage:** Marta Żerek; **Writing-Rough Preparation:** Gracjan Sitarek; **Writing - Review and Editing:** Gracjan Sitarek; **Visualization:** Marta Żerek; **Supervision:** Marta Żerek; **Project administration:** Marta Żerek;
All authors have read and agreed with the published version of the manuscript.
Funding statement: No external funding was received to perform this review
Statement of institutional review committee: not applicable
Statement of informed consent: not applicable
Statement of data availability: not applicable
Conflict of interest statement: The authors declare no conflict of interest.

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