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Interactions between Sleep and the Immune, Nervous and Endocrine Systems: A Narrative Review

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Abstract

Background. Sleep is an elementary physiological process that is controlled by the circadian rhythm. It is crucial for maintaining homeostasis and proper functioning of many systems in the human body.

Aim. The aim of this review is to summarize current knowledge on the impact of sleep deprivation on the immune, neurological, and endocrine systems.

Material and methods. The narrative review of the literature was conducted using databases including PubMed. Studies were selected based on their clinical relevance. This article focuses on the latest observational studies, reviews and randomized trials.

Results. In the nervous system sleep deprivation disrupts several brain functions, including cognitive performance, memory consolidation and neurodevelopment. Insufficient sleep is also associated with increased risk of neurological disorders, such as stroke and neurodegenerative diseases. Endocrine disturbances caused by the lack of adequate sleep include changes in secretion of many hormones, namely growth hormone, cortisol, melatonin, leptin and insulin. As a result, those changes lead to metabolic dysregulation, increased risk of obesity and type 2 diabetes. Sleep deprivation also impairs the immune function by altering leukocyte activity, reducing natural killer cell function, and increasing pro-inflammatory cytokine levels. Those shifts may contribute to the development of autoimmune disease and increase the susceptibility to infections.

Conclusions. Sleep plays a crucial role in maintaining health and homeostasis. Its deprivation has many unfavorable effects on physiological pathways. Ensuring sufficient sleep duration and quality is essential for maintaining both, physical and mental health.

Key words: sleep deprivation, circadian rhythm, immune system, nervous system, endocrine system

Introduction

Sleep is a physiological activity, ensuring physical and mental health, which takes up around one third of our life.[1] This is a process that can be reversed by a stimulus of sufficient intensity. During sleep, a person ceases physical activity, loses conscious contact with the environment, and their responsiveness to external stimuli is limited. Nevertheless, the activity of these systems is preserved and undergoes cyclical changes.[2]

According to the American Sleep Academy criteria, sleep consists of wakefulness, NREM sleep (stages N1, N2, and N3), and REM sleep (stage R). REM sleep is characterized by rapid eye movements and high brain activity. This is accompanied by reduced muscle tone, irregular breathing, and an increased heart rate. It is during REM sleep that dreams occur. NREM sleep, on the other hand, is characterized by slow eye movements and a decrease in body temperature, heart rate and blood pressure.[3] During sleep, these two phases intertwine. Immediately after falling asleep, the NREM phase appears, which shortens and becomes shallower as sleep progresses, unlike the REM phase, which lengthens.[4]

The appropriate amount and quality of sleep determines the proper functioning of, among others, the immune, nervous and endocrine systems. The recommended amount of sleep changes with age, for example adults should sleep about 7-9 hours and children 9-11 hours. [5] These days, people are increasingly reporting sleep disorders. This process plays a significant role in regulating many processes and functions in the human body, affecting almost every organ and system.

The circadian rhythm (CR) plays a key role in maintaining the body's homeostatic mechanisms. The suprachiasmatic nucleus (SCN), located in the hypothalamus, regulates the brain's internal clock. By responding to changes in environmental light, the circadian rhythm is responsible for controlling sleep and wakefulness.[2], [6] The SCN receives signals from the retinal ganglion cells, which are transmitted to it via the optic nerve. This causes adjustments in neuronal activity to maintain the circadian rhythm. The SCN regulates the hypothalamic-pituitary-gland axis, influencing the control of melatonin secretion.[2], [6] This light-sensitive hormone has broad antioxidant properties and protects cells from oxidative stress. During darkness, melatonin production by the pineal gland increases. Under the influence of light, melatonin production by the pineal gland decreases.[2], [6], [7] A feedback loop of "clock genes" helps regulate circadian rhythms. These include brain and muscle ARNT-like protein-1-2 (BMAL1-2), circadian locomotor output cycles kaput (CLOCK), period circadian regulator 1-3 (PER1-3), and cryptochrome circadian regulator 1-2 (CRY1-2).[2], [8]

First of all, it affects the immune system, in many different ways. Wang and co-authors conducted a study to evaluate sleep quality and the prevalence of sleep disturbances among patients with various etiologies of liver cancer[9]1]. Their main focus was on examining the association between sleep quality and immune system among those patients. To analyze sleep quality they used the Pittsburgh Sleep Quality Index (PSQI). They found that increased CD3+ T cells and decreased NK cells are associated with sleep disturbances in patients with liver cancer of non-HBV cirrhosis. Additionally, Sun and co-authors investigated the association between gut microbiota dysbiosis and sleep deprivation (SD) in their review. [1]2] The analysis incorporated papers on many topics related to the immune system, including concentration of pro-inflammatory cytokines, immune cell counts and functions, antibodies and the intestinal barrier. They state that there is a connection between sleep deprivation and gut microbiota dysbiosis, which can affect many processes in the human body, also ones outside the gastrointestinal (GI) tract.

Sleep disturbances might be connected to an increased risk of obesity, which is one of the conditions included in the diagnosis of metabolic syndrome. Chaput and co-authors studied the effect of insufficient sleep and circadian misalignment on obesity risk in their review.[10] Energy intake and expenditure, changes in appetite hormones

and eating habits were considered in the paper. They revealed that both insufficient sleep and circadian misalignment may increase the risk of obesity by affecting several metabolism pathways and behavioural patterns.

Sleep is also closely connected with mental health. Cramm and co-authors performed a study where they examined the association between mental health and sleep among Canadian firefighters.[11] They analyzed data that was collected from a larger cross-sectional survey of PSP in Canada. Well-validated self-report screening tools were used to collect necessary information about indications of mental health disorders and their symptoms. Sleep disturbances were evaluated by using the Insomnia Severity Index (ISI). An association between positive screens for mental health disorders and clinical insomnia was found. In addition, the group with insomnia had a greater chance of screening positive for mental health disorders, including Post-Traumatic Stress Disorder (PTSD), General Anxiety Disorder (GAD), Panic Disorder (PD), Social Anxiety Disorder (SAD), and Major Depressive Disorder (MDD).

Furthermore, sleep may even play a role in reproductive health, including fertility. Wang and co-authors performed a cross-sectional study assessing a combined impact of insufficient sleep and overweight/obesity/abdominal obesity on the risk of female infertility.[12] The necessary data was drawn from the National Health and Nutritional Examination Survey (NHANES) database from 2017-2020. A total of 1,577 women participated in the study, among whom 191 were found to have infertility. They found that both, trouble sleeping and short sleep duration, are linked to an increased risk of female infertility. When combined with overweight/obesity/abdominal obesity, the potential risk is also higher.

Interestingly, sleep can even affect oral health. Jainy Shah and co-authors explored the impact of sleep duration and its quality on oral health in their scoping review, in which they included 14 studies[13]. The investigated papers regarded both children and adults. Authors found that poor sleep quality or short sleep duration are associated with an increased risk of periodontitis and dental decay.

Sleep also plays a profound role in many other different processes, including homeostasis, hormonal secretion and several brain functions. In this review we will mainly focus on the effects of sleep deprivation on immune, neurological and endocrine pathways.

Methodology

A systematic search was conducted in PubMed databases from December 2025 to April 2026. A combination of different keywords was used: “sleep impact” or “impact of sleep” or “sleep brain” and " nervous system” or “endocrine system” or “immune system”. Original studies in adults and animals regarding the influence of sleep on the immune, nervous and endocrine systems, published in English, were included in the analysis. Case reports and studies without access to the full text were excluded. Data on the authors, year of publication, type of study, population characteristics, and main results regarding the effect of sleep deprivation on function of the immune, nervous and endocrine systems were extracted from the eligible studies.

The impact of the sleep disorder on the nervous system

Sleep might affect the nervous system and brain functions in many different ways. First of all, there is an association between the act of sleep and cognitive performance. Gottesman and co-authors found a connection between sleep deprivation and worse cognitive performance, including impaired memory consolidation, by conducting a scientific statement, where they examined a link between disrupted sleep and brain health.[14] They also reached some other conclusions, which will be mentioned further. Additionally, Zimmerman and co-authors conducted a study, using a randomized, crossover design, to investigate the effect of sleep on cognitive performance.[15] A total of 65 participants, who regularly slept 7-9 h/night, were included in the sample. They completed two 6-week intervention conditions, adequate sleep and insufficient sleep. Cognitive performance was assessed with the NIH Toolbox Cognition Battery. They found that an adequate sleep, of at least 7 hours a night, can improve working memory in healthy adults. Furthermore, Brodt and co-authors conducted a review that focused on the process of long-term

memory consolidation during both wakefulness and sleep.[16] Many neural and synaptic processes and different stages of sleep were taken into consideration. They found evidence that sleep enhances memory formation through active systems consolidation in the hippocampus-dependent episodic memory system.

Sleep is also important in neurological development. Miletínová and co-authors performed a review about the potential functions of sleep, mainly focusing on brain functions.[17] They found that sleep, mostly REM sleep, might have an impact on neurodevelopment and neurogenesis. Synaptic plasticity may also be affected by sleeping, but circadian rhythms are a factor here as well. In addition, the review states that sleep is an important factor when it comes to memory consolidation but the effects of sleep on memory might be diverse, depending on features like the type of memory and age.

Interestingly, sleep can also have an impact on particular brain structures. Namsrai and co-authors studied this in their meta-analysis. [18] Several sleep disorders, such as insomnia, obstructive sleep apnoea (OSA), REM sleep behaviour disorder (RBD), restless legs syndrome (RLS) and narcolepsy, and sleep characteristics were included in the study. A conclusion that inadequate sleep is associated with lower volume in several parts of the brain was drawn. This shows that disrupted sleep may play a significant role in the process of neurodegeneration.

Sleep disruption may increase the risk of developing a stroke. Ungvari and co-authors conducted a systematic review and a meta-analysis to examine how sleep disturbances, including short and long sleep durations, impact stroke incidence and mortality.[19] First of all, a link between both, short and prolonged sleep duration, and an increased risk of stroke was found. Additionally, higher odds of stroke mortality were connected with short and long sleep duration. Titova and co-authors examined a relationship between sleep-disordered breathing (SDB) and the development of stroke, by performing a cohort study and a Mendelian randomization analysis.[20] The study included over 41,000 Swedish middle-aged and elderly people, who were followed up for incident stroke and death over 8 years. The authors used data from the National Research Infrastructure SIMPLER (Swedish Infrastructure for Medical Population-based Life-course Environmental Research). In the cohort study, having at least one SDB symptom was linked to an increased risk of total stroke and intracerebral hemorrhage. The MR analyses did not show such a connection. The previously mentioned scientific statement, performed by Gottesman and co-authors also found that sleep disturbances, such as obstructive sleep apnoea (OSA), are associated with a higher risk of stroke.

Neurodegenerative diseases are another group of disorders that are linked to sleep disturbances. Ungvari and co-authors performed a meta-analysis regarding sleep disorders and their contribution to cognitive decline, dementia, and neurodegenerative diseases.[21] Their main focus was on obstructive sleep apnea (OSA) and insomnia. First of all, they found that both apnoea and insomnia increase the odds of developing all-cause dementia. Additionally, insomnia was connected with a higher risk of vascular cognitive impairment. When it comes to Alzheimer's disease, they found that apnoea, as well as insomnia, are linked to an elevated risk of developing this particular disorder. Both sleep disorders were also associated with cognitive decline. In addition, Gottesman and co-authors found a link between sleep disturbances and an increased plausibility of dementia. Moreover, they also examined works about the glymphatic system and found that it is more efficient during sleep, which helps to remove metabolic waste from the brain, having an impact on some neurological disorders, including Alzheimer's disease (AD).

In summary, sleep has an impact on many neurological pathways and brain functions, including cognitive performance, memory consolidation, neurodevelopment and neurogenesis. Therefore, sleep deprivation may lead to an increased risk of stroke and higher odds of developing neurodegenerative diseases.

The impact of the sleep disorder on the endocrine system

Sleep is linked to the circadian rhythm, a clock that regulates cyclical body processes such as sleep, wakefulness, body temperature, and hormone production. Sleep disorders are associated with circadian rhythm disturbances, which in turn affect the abnormal secretion of hormones such as growth hormone, leptin, ghrelin, cortisol, melatonin and hormones related to carbohydrate and lipid metabolism.[22] The suprachiasmatic nucleus, located in the anterior part of the hypothalamus, is responsible for regulating circadian rhythms.

One hormone whose secretion is closely linked to circadian rhythms and sleep is growth hormone. Its secretion increases during sleep, peaking immediately after falling asleep.[23] Sleep deprivation significantly reduces growth hormone levels.[24] Sakia and co-authors studied people with post-traumatic stress disorder for sleep disturbances, growth hormone secretion, and memory consolidation. Sleep in the control and research groups was monitored via EEG. Growth hormone levels were assessed by monitoring blood levels every 20 minutes via catheter. Memory was assessed through a 15-word task that participants recalled step by step after waking. Researchers have shown that people with PTSD experience fragmented sleep, decreased GH levels, and impaired memory consolidation. Sleep disorders contribute to decreased growth hormone secretion.[25] Studies in animal models indicate that sleep deprivation not only reduces hormone concentration, but also increases tissue resistance to the effects of growth hormone.[26]

Sleep also affects the hypothalamic-pituitary-adrenal (HPA) axis and, consequently, cortisol secretion. During slow-wave sleep, in the early phase of sleep, the HPA axis and cortisol synthesis are inhibited. Later, as the duration of REM sleep increases, cortisol levels increase, reaching a peak just before the wakeful phase. Circadian rhythm disturbances and abnormal cortisol production have been observed in people working the night shift. This phenomenon was associated with a greater risk of developing metabolic, cardiovascular and mental disorders.[27] Even one night of complete sleep deprivation affects physiological stress, which manifests itself in large increases in cortisol, especially in the early morning and evening. It is most likely related to the lack of inhibitory effect of sleep on hormone secretion. [28]

Alongside cortisol, melatonin is considered the main hormone responsible for regulating the circadian rhythm. Cortisol is associated with mobilizing the body for upcoming activity. Melatonin, produced by the pineal gland, is responsible for regulating sleep, aiding in falling asleep, and has antioxidant properties. Melatonin reaches its highest concentrations at night. Its secretion is regulated by the suprachiasmatic nucleus in response to light and darkness. Melatonin plays a crucial role in immune system function. Long-term sleep deprivation has been associated with decreased melatonin levels, which in turn leads to increased proinflammatory cytokines, oxidative stress, and decreased immune cell activity. [29]

Glucose levels are controlled by the beta cells of the pancreas, which produce insulin. This hormone inhibits hepatic glucose production and increases glucose uptake by peripheral tissues. In cases of insulin resistance, higher insulin concentrations are required to regulate glycemia. There are also reports of a link between shortened sleep time and obesity and related metabolic disorders such as increased insulin resistance and diabetes mellitus. Three probable mechanisms for this process have been identified: changes in glucose metabolism, increased appetite, and reduced energy requirements.[30] In healthy people, glucose levels remain relatively constant during sleep, with minimal drops. Previous studies have focused on the effects of acute sleep deprivation on glucose levels. These changes were short-lived and easily corrected upon return to normal sleep. Glucose levels are lower than during normal sleep, but in the morning this trend reverses as regulatory mechanisms begin to operate. [30] There is a link between sleep deprivation and obesity, and obesity is a direct risk factor for developing diabetes.

Leptin is a hormone associated with feelings of satiety. It suppresses hunger, accelerates metabolism, and burns fat. Insufficient sleep time affects the increase in leptin secretion, which may promote the development of type 2 diabetes through several mechanisms. The first is increased appetite and food consumption, which contributes to weight gain. It's also important to note that leptin secretion disorders reduce insulin secretion and increase insulin resistance and inflammation. Both processes contribute to the development of diabetes.[31]

The impact of the sleep disorder on the immune system

Sleep is essential for the proper functioning of the immune system, which plays a key role in the body's defense against pathogens.[6, 8] Various studies have attempted to examine the relationship between sleep deprivation or sleep disturbances and changes in various white blood cell groups.[32] Zhou et al. described a subtle but significant effect of sleep disturbances on leukocytes, leading to changes in their number, distribution, and function. Increased secretion of glucocorticoids and catecholamines suppresses the number and cytotoxic activity of natural killer (NK) cells.[6, 8] During normal sleep-wake cycles, T lymphocytes tend to decrease at night and increase again in the morning. Sleep deprivation increases the levels of CD3+ T cells in the blood and spleen, CD4+ T cells in the blood and lymph nodes, and reduces the percentage of CD8+ T cells. In patients with chronic insomnia, a significant reduction in the number of total lymphocytes, CD3+ T cells, CD4+ T cells, and CD8+ T cells has been observed. [6] Ozturk et al. investigated the role of regulatory T cells (Treg) in response to sleep deprivation and the therapeutic effect of melatonin in modulating the response. They divided male mice into four groups: control, control with melatonin, sleep deprivation, and sleep deprivation with melatonin. The levels of regulatory T cells were assessed in peripheral blood and spleen samples. In the sleep deprived group, a decrease in CD3+ T cells was observed in blood samples and an increase in the transcription factor Helios in blood samples and spleen, which may indicate immunosuppression. Furthermore, melatonin administration restored the T cell population to normal levels. The results of this study group suggest that melatonin administration influences the regulation of immune balance during sleep deprivation.[32] Regulatory T cells play a role in maintaining immune tolerance and preventing excessive or misdirected immune responses. Their function and proliferation are regulated by the circadian rhythm, so sleep disorders may contribute to inflammation and autoimmune conditions. People with chronic insomnia have a 70% increased risk of developing autoimmune diseases.[2] Disease exacerbations may also occur. [2]Sleep deprivation may contribute to increased levels of antinuclear antibodies in patients with systemic lupus erythematosus. This may lead to an accelerated onset of the disease. [6] Furthermore, sleep deprivation may influence the growth of Th17 cells, thus inducing inflammation. [2] Chronic sleep deprivation reduces the suppressive function of T reg lymphocytes, resulting in an increase in proinflammatory cytokines—interleukin IL-1B and IL-6.[2, 6] Sleep disorders and the resulting immune dysfunction may increase susceptibility to infection by impairing the immune system's ability to properly regulate its response. [2, 32] Sleep deprivation exacerbates myocardial damage caused by ischemia, inflammation, and myocardial cell apoptosis. It has also been observed that circadian rhythm disruption can enhance the immune response and disrupt cytoskeletal gene expression, leading to acute lung injury.[6] Sleep disorders are an important factor in the pathophysiology of various chronic diseases by activating the immune response and leading to chronic inflammation. This contributes to the development of diseases such as atherosclerosis, where elevated inflammatory markers promote the development of atherosclerotic plaque in artery walls, and obesity, where sleep deprivation disrupts metabolic regulation, leading to insulin resistance and metabolic disorders.[6]

Conclusions

Sleep is a crucial process that has an impact on various human body systems and functions. Its proper duration and quality are significant for maintaining physical and mental health. The reviewed evidence indicates that sleep deprivation leads to impaired cognitive performance, memory consolidation and neurodevelopment. Regarding the nervous system it also increases the risk of neurological disorders, such as stroke and neurodegenerative diseases. Moreover, sleep plays a role in preserving brain structural integrity and enhancing the clearance of metabolic waste. Endocrine function is also highly dependent on proper sleep. Insufficient sleep causes alterations in concentration

of several hormones, including growth hormone, cortisol, melatonin, insulin and leptin, among others. Those shifts may lead to metabolic dysregulation and increased risk of obesity and type 2 diabetes. Additionally, sleep disturbances also contribute to immune dysregulation by increasing the levels of pro-inflammatory cytokine and causing shifts in the number and activity of certain immune cells. Those changes can lead to an increased risk of autoimmune and infectious diseases.

Disclosure

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reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication

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