

Original Research

Investigating the Influence of Sleep Quality and Duration on Immune System Function and Disease Susceptibility

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ABSTRACT

Background: The length and quality of sleep are important factors in controlling immune system activity. A higher risk of infections and chronic illnesses has been linked to inadequate sleep. To learn more about how sleep affects health outcomes, this research looks at the connection between immune response and sleep patterns.

Materials and methods: 150 adults between the ages of 20 and 50 who were chosen from a community health center participated in a cross-sectional research. For a week, actigraphy and the Pittsburgh Sleep Quality Index (PSQI) were used to measure the length and quality of sleep. White blood cell (WBC) count, C-reactive protein (CRP), and pro-inflammatory cytokine levels (IL-6 and TNF- α) were among the immunological indicators measured in blood samples. Multiple regression and Pearson correlation were used in the statistical analysis to look at the relationship between immune function and sleep characteristics.

Results: CRP levels were considerably higher (mean \pm SD: 5.8 \pm 1.2 mg/L vs. 3.2 \pm 0.9 mg/L, $p < 0.001$) and pro-inflammatory cytokine levels were higher (IL-6: 15.6 \pm 4.1 pg/mL vs. 9.3 \pm 2.7 pg/mL, $p < 0.01$) in participants with poor sleep quality (PSQI score > 5) and shorter sleep periods (< 6 hours). WBC count and sleep duration had a positive correlation ($r = 0.42$, $p < 0.05$), suggesting that those who get enough sleep had superior immunological responses. The results of regression analysis showed that shorter sleep duration and worse sleep quality independently predicted higher inflammatory markers ($p < 0.01$).

Conclusion: The importance of sleep in preserving immunological homeostasis is shown by this research. Inadequate sleep length and poor quality were linked to immunological dysregulation and elevated inflammation, underscoring the significance of good sleep hygiene in reducing the risk of illness. In order to investigate causal links and underlying processes, more longitudinal research are necessary.

Keywords: Sleep quality, sleep duration, immune system, inflammation, disease susceptibility, cytokines, CRP

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INTRODUCTION

A basic biological function, sleep is necessary to preserve general health and wellbeing. It is essential for controlling the immune system as well as other physiological processes. While inadequate sleep length and poor sleep quality are associated with heightened vulnerability to infections and chronic illnesses, including diabetes and cardiovascular problems, enough sleep has been shown to improve immune responses [1,2]. Neuroendocrine and inflammatory processes drive bidirectional signaling

pathways in the intricate relationship between immune function and sleep [3]. To defend the body against infections, the immune system uses both innate and adaptive processes. According to new research, sleep disruptions may change how important immune mediators like cytokines and acute-phase proteins are produced, which might result in systemic inflammation [4,5]. Sleep patterns have a major impact on pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF- α) and interleukin-6 (IL-6). Short sleep duration, for example, has been linked

to higher levels of IL-6, which may be a factor in chronic inflammation and a higher risk of illness [6]. Even with the increasing amount of studies, little is known about the precise pathways relating immune dysfunction and sleep quality. With little information on long-term effects and individual differences in sleep-immune interactions, the majority of research concentrate on short-term effects [7]. Additionally, objective sleep measurements like actigraphy are still neglected in assessing how sleep affects immunological markers. By measuring inflammatory biomarkers in a group of individuals, this research seeks to understand the connection between immune system activity, sleep length, and quality. Gaining knowledge of these connections might help us better understand how sleep contributes to both illness prevention and health promotion.

MATERIALS AND METHODS

Population and Study Design: This cross-sectional research, which included 150 adults between the ages of 20 and 50, was carried out at a community health centre. Convenience sampling was used to choose participants, and those without a history of mental health issues, sleep difficulties, or chronic diseases were accepted. Those using drugs that interfered with immune or sleep function were not included.

Evaluation of Sleep Duration and Quality: The Pittsburgh Sleep Quality Index (PSQI), a standardized questionnaire that evaluates many elements of sleep over a one-month period, was used to measure the quality of sleep. They were divided into two groups: those with poor sleep quality (PSQI score >5) and those with excellent sleep quality (PSQI score ≤ 5). Using actigraphy devices worn on the wrist for seven days in a row, which allowed for continuous monitoring of sleep-wake cycles, the length of sleep was objectively assessed.

Immune marker measurement: To reduce diurnal fluctuations in immunological markers, blood samples were taken in the morning after an overnight fast. Using enzyme-linked immunosorbent assay (ELISA) kits, serum levels of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α) as well as C-reactive protein (CRP) were determined in accordance with the manufacturer's instructions. Using an automated

hematology analyzer, the number of white blood cells (WBCs) was determined.

Gathering Information and Statistical Evaluation: Structured questionnaires were used to gather lifestyle and demographic data, including age, gender, body mass index (BMI), and smoking status. The associations between immunological markers and sleep measures were evaluated using Pearson correlation coefficients. Age, BMI, and smoking status were among the possible confounders that were taken into account using multiple linear regression models. SPSS software was used to conduct statistical analyses, with a significance level of $p < 0.05$.

RESULTS

The study included 150 participants, with a mean age of 35.4 ± 8.2 years, comprising 54% females and 46% males. Sleep quality and duration were analyzed in relation to immune markers, with the findings summarized below.

Sleep Quality and Immune Markers: Participants with poor sleep quality (PSQI >5) had significantly higher levels of inflammatory markers compared to those with good sleep quality (PSQI ≤ 5). The mean CRP levels were 5.6 ± 1.4 mg/L in the poor sleep group versus 3.1 ± 0.8 mg/L in the good sleep group ($p < 0.001$). Similarly, IL-6 levels were elevated in participants with poor sleep (16.2 ± 3.9 pg/mL) compared to those with good sleep (9.7 ± 2.3 pg/mL, $p < 0.01$) (Table 1).

Sleep Duration and Immune Response: Shorter sleep duration (<6 hours) was associated with increased levels of TNF- α and reduced WBC counts. Participants with short sleep duration had TNF- α levels of 12.4 ± 2.8 pg/mL, compared to 7.5 ± 1.9 pg/mL in those sleeping ≥ 6 hours ($p < 0.05$). WBC counts were lower in the short sleep group ($6.1 \pm 1.3 \times 10^3/\mu\text{L}$) versus the adequate sleep group ($7.4 \pm 1.2 \times 10^3/\mu\text{L}$, $p < 0.01$) (Table 2).

Correlation Analysis: Pearson correlation showed a significant negative correlation between PSQI scores and WBC count ($r = -0.45$, $p < 0.05$), and a positive correlation between poor sleep and CRP levels ($r = 0.51$, $p < 0.01$). Sleep duration was positively correlated with WBC count ($r = 0.42$, $p < 0.05$) (Table 3).

Table 1: Comparison of Inflammatory Markers Based on Sleep Quality

Parameter	Good Sleep Quality (n=80)	Poor Sleep Quality (n=70)	p-value
CRP (mg/L)	3.1 ± 0.8	5.6 ± 1.4	<0.001
IL-6 (pg/mL)	9.7 ± 2.3	16.2 ± 3.9	<0.01

Table 2: Comparison of Immune Markers Based on Sleep Duration

Parameter	Sleep ≥ 6 Hours (n=85)	Sleep <6 Hours (n=65)	p-value
TNF- α (pg/mL)	7.5 ± 1.9	12.4 ± 2.8	<0.05
WBC Count ($\times 10^3/\mu\text{L}$)	7.4 ± 1.2	6.1 ± 1.3	<0.01

Table 3: Correlation between Sleep Parameters and Immune Markers

Variable	CRP (r)	IL-6 (r)	TNF- α (r)	WBC Count (r)
PSQI Score	0.51**	0.48*	0.37	-0.45*

Sleep Duration (hours)	-0.46*	-0.39	-0.41	0.42*
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(*p< 0.05; **p< 0.01)

In summary, poor sleep quality and shorter duration were significantly associated with elevated inflammatory markers and reduced immune function (Tables 1–3). These findings emphasize the critical role of sleep in immune regulation.

DISCUSSION

This research emphasizes how important sleep length and quality are for immune system function, especially in relation to inflammatory indicators and immunological modulation in general. Our results add credence to the mounting evidence that sleep deprivation is associated with elevated inflammation and a higher risk of illness. According to earlier research, individuals with poor sleep quality had higher levels of C-reactive protein (CRP) and pro-inflammatory cytokines such interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α) (1,2). Increased production of pro-inflammatory mediators results from sleep disruptions that upset the autonomic nervous system and hypothalamic-pituitary-adrenal (HPA) axis balance [3,4]. The pathophysiology of chronic illnesses, such as diabetes, autoimmune disorders, and cardiovascular problems, may be influenced by these inflammatory alterations [5,6].

Reduced white blood cell (WBC) counts and increased inflammatory markers were also linked to short sleep duration, suggesting weakened innate immune responses. This result supports studies showing that sleep deprivation affects natural killer cell activity and neutrophil function, two essential elements of the body's defensive systems [7,8]. Additionally, the association between a short sleep duration and elevated TNF- α levels illustrates how this cytokine mediates inflammation and sleep-wake regulation [9,10].

Curiously, the study's findings highlight how crucial objective measurements—like actigraphy—are to precisely determining sleep characteristics. The reliability of traditional self-reported methods in establishing a correlation between immunological markers and sleep quality may be limited by their tendency to underreport sleep disruptions [11,12]. Future research on the relationship between sleep and the immune system may be more accurate if objective evaluations are included. The observed associations between inflammatory markers and sleep factors highlight how the sleep-immune interaction is reciprocal. Inflammation is exacerbated by inadequate sleep, but systemic inflammation may also interfere with sleep architecture, resulting in a vicious cycle [13]. Immune function may be enhanced and systemic inflammation may be decreased by treating sleep disorders with cognitive-behavioral therapy, relaxation methods, and pharmaceutical therapies [14,15]. The research has limitations in spite of these revelations. Its cross-sectional nature makes it

impossible to draw conclusions about causality, and depending just on a one-week actigraphy can miss long-term sleep habits. To investigate causation and ascertain if enhancing sleep quality might restore immunological dysregulation, further long-term research is required. Furthermore, generalizability would be improved by increasing the sample size and include a variety of groups.

CONCLUSION

To sum up, this research confirms how important sleep is for controlling the immune system. The significance of emphasizing sleep hygiene for general health and disease prevention is shown by the substantial correlation found between short sleep duration and poor sleep quality and elevated inflammatory markers. To investigate the processes behind these relationships and provide focused therapies, further study is necessary.

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