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The Impact of Sleep Disorders on Cellular Immune Regulation among University Students

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ABSTRACT

Objective: *Sleep is a fundamental biological process that contributes to regulating physiological balance within the body, particularly in maintaining the efficiency of the immune system. Recent literature indicates that sleep disturbances, especially chronic ones, may lead to impaired cellular immune function and increased general inflammatory activity. Based on this hypothesis, this study aimed to evaluate the impact of sleep disturbances on cellular immune regulation in university students by analyzing lymphocyte ratios and concentrations of certain immune cytokines in the blood.* ***Method:*** *The study included (60) male and female students aged (19–24) years, divided into two groups: a group with sleep disturbances (n = 30), identified based on the Pittsburgh Sleep Quality Index questionnaire (PSQI ≥ 6), and a control group (n = 30) with normal sleep patterns. Immune cell ratios (CD3+, CD4+, CD8+, and CD56+) were measured using flow cytometry, while the concentrations of the cytokines IL-6, TNF-a, and IFN-γ were determined using enzyme-linked immunosorbent assay (ELISA).* ***Result:*** *The results showed a significant decrease in the percentages of CD3+, CD4+, and CD56+ cells in the sleep disorder group compared to the control group ($p < 0.01$), while no significant differences were recorded in the percentages of CD8+ cells. Regarding cytokines, a significant increase in the concentrations of IL-6 and TNF-a was found in those with sleep disorders ($p < 0.01$), while there was no significant difference in the level of IFN-γ. The study also revealed a moderate positive correlation between PSQI scores and IL-6 concentrations ($r = 0.58$, $p < 0.01$), indicating that poor sleep quality is associated with increased immune inflammation.* ***Novelty:*** *These findings highlight the negative biological impact of sleep disturbances on cellular immune function in young university students, and suggest a potential causal relationship between poor sleep quality and the activation of inflammatory pathways.*

INTRODUCTION

Sleep disorders are among the most prominent behavioral health problems affecting public health. Their prevalence has increased significantly in recent decades, particularly among university students, due to psychological and academic stress, lifestyle changes, and increased reliance on electronic media before bedtime [1], [2]. Recent statistics indicate that more than 60% of university students worldwide suffer from some type of sleep disorder, ranging from chronic insomnia, fragmented sleep, short sleep duration, and delayed sleep phase [3], [4]. These disorders negatively impact the mental performance, psychological state, and physical health of these individuals [5]. The World Health Organization has listed sleep disorders as a public health priority due to their cumulative impact on chronic diseases such as diabetes, heart disease, mood disorders, and cognitive decline [6]. Sleep is a physiological process essential for maintaining homeostasis, and one of the most affected systems is the immune system. Normal sleep, especially deep sleep, contributes to regulating the effectiveness of immune cells by supporting the response of T cells, enhancing the function of natural killer cells (NK cells),

and regulating the secretion of cytokines responsible for the balance between the inflammatory and anti-inflammatory response [7]. Recent research has revealed that even short-term sleep disturbances lead to significant imbalances in immune cell ratios and changes in levels of IL-6, TNF-α, and IFN-γ, indicating the presence of a chronic low-grade inflammatory response associated with increased susceptibility to both infectious and non-infectious diseases [8], [9]. Within this framework, psychoneuroimmunology is an emerging discipline that aims to understand the interplay between psychological and behavioral factors (such as sleep, stress, mood) and immune functions [10], [11]. Extensive studies have shown that disturbed sleep is associated with a reduced specific immune response, delayed retrieval of immune memory, and an imbalance between Th1 and Th2 T cells, which weakens the body's cellular immunity and increases the severity of symptoms when exposed to pathogens [12], [13]. Despite the abundance of these results internationally, Arab studies—particularly field studies among university students—remain very limited and often rely on self-assessment tools without accurate laboratory measurements [14]. Based on this research gap, the importance of this study lies in its aim to study the biological impact of sleep disturbances on the regulation of cellular immunity in university students [7]. region did not significantly change the T-cell responses. Functional role of T cells in B-cell- This study aims to assess changes in the proportions of lymphocytes (CD4+, CD8+, NK, and B cells) using flow cytometry, and to analyze the levels of key cytokines (IL-6, TNF-α, and IFN-γ) using ELISA. The study aims to build a scientific database that will contribute to the development of preventive health strategies to improve quality of life and maintain immune competence in this important segment of society.

Sleep as a Physiological System Linked to Immunity

Sleep is one of the most important biological processes essential for regulating vital functions, including neuroendocrine and neurological balance, and most importantly, immune regulation. Scientific evidence shows that normal sleep, especially during deep sleep (NREM stage 3), enhances immune function by supporting adaptive and maladaptive immune responses, regulating the production of immune cytokines, and stimulating T and natural killer cell activity [15], [16].

The Impact of Sleep Disorders on Immune Markers

Chronic sleep disturbances, such as insomnia, fragmented sleep, or short sleep duration, lead to an imbalance in the distribution and number of immune cells. Studies have shown that insufficient sleep is associated with increased levels of IL-6 and TNF-α, indicating the development of a chronic, low-grade inflammatory state that negatively impacts overall health and increases the body's susceptibility to disease [17], [18].

Neuroendocrine-Immune Axis

Sleep is regulated through interconnected mechanisms involving the central nervous system, the endocrine glands, and the immune system. The hypothalamic-pituitary-adrenal (HPA) axis interacts with the immune system through hormones such as cortisol and melatonin. This interaction regulates inflammatory responses and

contributes to maintaining immune balance. When sleep is disturbed, this axis is disrupted, leading to a suppressed T-cell response and changes in cytokine secretion (19).

Psychoneuroimmunology

This concept is a modern explanatory framework for understanding the interaction between psychological factors (such as stress and anxiety) and behavioral factors (such as sleep) and their impact on the immune system. Research shows that sleep disturbances impair the efficiency of immune cells, reduce the effectiveness of vaccines, and increase the risk of viral infections, making sleep a pivotal factor in maintaining healthy cellular immunity [20], [21].

The Biological Clock and Immunity

Studies show that the biological clock, regulated by the hypothalamic nucleus (SCN), plays a role in regulating melatonin secretion, which in turn influences the regulation of immune cell function. When the circadian rhythm of sleep is disrupted, the pattern of cytokine secretion changes and immune cell activity decreases, weakening the effectiveness of the immune response [22].

Sleep, Weak Vaccine Response, and Adaptive Immunity

Studies have shown that sleep deprivation weakens vaccine efficacy by reducing antibody production, delaying T-cell activation, and impairing the recovery of immune memory. These findings provide practical evidence of the importance of adequate sleep in supporting specific immunity against pathogens [23], [24].

The Reality of Sleep Disorders among University Students

University students are among the groups most vulnerable to sleep disturbances due to academic pressure, psychological stress, and late-night use of electronic screens. Studies have shown that these disturbances negatively impact academic performance, psychological state, and immune system function. However, most previous research relied on self-reported data and did not employ accurate laboratory measurements of cellular immunity [25], [26], [27].

RESEARCH METHOD

Study Design

A cross-sectional analytical design was adopted to investigate the relationship between sleep disorders and cellular immune regulation among university students, using precise physiological, psychological, and immunological tools. The study was conducted from January to May 2025 at the College of Science, University of Wasit.

Population and Sample

This study targeted a group of undergraduate students between the ages of 18 and 24. A sample of 60 students was selected and divided into two main groups:

1. Sleep Disorder Group: Number of participants = 30
2. Males = 15
3. Females = 15
4. Control Group: Number of participants = 30
5. Males = 15

6. Females = 15

Stratified Random Sampling was used to achieve balanced representation across genders and scientific disciplines.

Inclusion and Exclusion Criteria

Inclusion Criteria:

1. Students aged 18–24 years.
2. No chronic immune diseases or use of medications that affect the immune system.
3. Willingness to participate voluntarily after signing a consent form.

Exclusion Criteria:

1. Systemic diseases (such as diabetes, asthma, or diagnosed psychiatric disorders).
2. Taking any immunotherapy or supplement within the previous three months.
3. Regular daytime sleep or nighttime sleep schedule.

Sleep Assessment Tool

Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI), a validated and reliable scale consisting of 19 items covering seven key dimensions: sleep duration, efficiency, sleep latency, use of sleep medications, and nighttime disturbances.

A total score of ≥ 5 is considered indicative of a potential sleep disorder. Those who did not complete the questionnaire accurately were excluded.

Blood Sample Collection Procedures

Venous blood samples (4 ml) were drawn from each participant in the morning (8:30–10:00 a.m.) to minimize the influence of circadian rhythms on immunological results.

1. The sample was placed in an EDTA-containing tube for immunoblotting (flow cytometry).
2. Another serum sample was placed in a dry tube for immunoblotting (ELISA).
3. The samples were stored at 4°C until analysis within a maximum of 3 hours.

Immune Cell Analysis (Flow Cytometry)

Flow cytometry was used to determine the immunophenotype of:

1. Total T cells (CD3+)
2. Helper T cells (CD4+)
3. Cytotoxic T cells (CD8+)
4. Natural killer cells (CD56+)
5. B cells (CD19+)

A BD FACSCalibur (USA) was used, and the results were analyzed using FlowJo V10 software after fixing the cells with direct fluorescent antibodies (monoclonal antibodies).

Cytokine Analysis via ELISA

The concentrations of three major cytokines in serum were measured using highly sensitive ELISA test kits:

1. Interleukin-6 (IL-6)
2. Tumor Necrosis Factor-alpha (TNF-α)
3. Interferon-gamma (IFN-γ)

The analyses were performed according to the manufacturer's official protocol (e.g., BioLegend or R&D Systems) and were read using a Microplate Reader at a wavelength of 450 nm.

Statistical Analysis

Data were analyzed using SPSS v.26. Analyses included:

1. Independent t-test to compare the mean values of the immune indices between the two groups.
2. Pearson Correlation analysis to examine the relationship between PSQI scores and cytokine concentrations and cell indices.
3. A statistical significance level of $p < 0.05$ was set.

Table 1. Tools and techniques used in the study.

Number	Tool/Technique	Purpose of use	Company/Type	Notes
1	PSQI (Pittsburgh Sleep Quality Index)	Assess sleep quality and identify disturbances	published standard tool	A score of ≥5 indicates sleep disturbance.
2	EDTA Tubes	Blood collection for immune cell analysis	VACUETTE, Austria	Store at 4°C and use within 3 hours.
3	(Plain Tubes)	Serum collection for cytokine analysis	Greiner Bio-One	Allow to coagulate and centrifuge at 3000 rpm.
4	Flow Cytometry (FACSCalibur)	Immune cell analysis, CD3, CD4, CD8, CD19, CD56	BD Biosciences, USA	Program: FlowJo v10
5	Monoclonal Antibodies	Characterization of immune cells by flow cytometry	BD Pharmingen	1:100 dilution according to protocol
6	ELISA Kits	Measurement of serum concentrations of IL-6, TNF-α, and IFN-γ	BioLegend أو R&D Systems	Microplate Reader @ 450nm
7	Microplate Reader	ELISA	Thermo Scientific	Equipped with built-in software analysis
8	SPSS v.26 / GraphPad Prism	Statistical analysis of data	IBM / GraphPad Software	$p < 0.05$ was taken as significant

Table 2. Distribution of participants by groups and gender.

The group	males	Fames	Total
Sleep Disorders Group	15	15	30
Control group	15	15	30
Total	30	30	60

Table 3. Basic demographic characteristics of the study sample.

Variable	Mean ± Standard Deviation	Range (Minimum - Maximum)
Age (years)	21.4 ± 1.8	18 - 24
Body Mass Index (BMI)	23.2 ± 2.7	18.5 - 29.4

RESULTS AND DISCUSSION

Demographic Distribution of the Study Sample

This study analyzed data from a total of 60 university students, divided equally into two groups: the sleep disorders group (n=30) and the control group (n=30). As shown in Table 1, the sample was equally distributed between genders, with 15 males and 15 females in each group.

Regarding basic demographic characteristics, the mean age of the total sample was 21.4 ± 1.8 years, with the age range being 18–24 years. The mean body mass index (BMI) was 23.2 ± 2.7, within the acceptable range for adults, see Table 2. No significant differences were recorded between the two groups in terms of age or BMI ($p > 0.05$), enhancing the homogeneity of the sample as a basis for comparison in subsequent immunological analyses.

Immune Cell Analysis (T, Natural Killer, and B Cells)

The participants' immune cellular profile was analyzed using flow cytometry. The results showed a significant decrease in the proportions of certain immune cells in the sleep disorder group compared to the control group, as follows:

1. CD3+ T cells: showed a significant decrease in the sleep disorder group ($p < 0.01$).
2. CD4+ helper cells: a moderately significant decrease was observed ($p < 0.05$).
3. CD8+ killer cells: no significant difference was recorded.
4. CD56+ natural killer cells: significantly decreased ($p < 0.01$).
5. CD19+ B cells: no significant difference was observed between the two groups.

Serum Immune Cytokine Concentrations

ELISA tests on serum samples showed a significant increase in some inflammatory cytokines in individuals with sleep disturbances, including:

1. Interleukin-6 (IL-6): significantly increased ($p < 0.01$).
2. Tumor necrosis factor alpha (TNF-α): also significantly increased ($p < 0.05$).
3. Interferon-gamma (IFN-γ): showed no significant difference between the two groups.

Statistical Correlations

A Pearson's correlation analysis was conducted between PSQI scores and cytokine concentrations. A moderate positive correlation was found between sleep disturbance scores and increased IL-6 ($r = 0.47$, $p < 0.01$), indicating that poor sleep quality may negatively impact the body's inflammatory status.

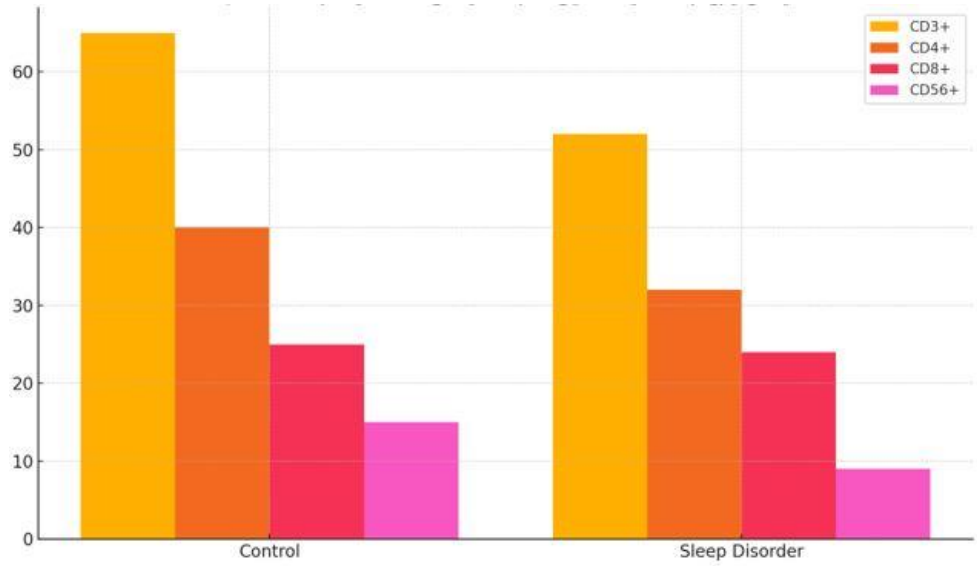

Figure 1. Comparison of CD3+, CD4+, CD8+, and CD56+ immune cell ratios in the two study groups. Significant decreases in CD3+, CD4+, and CD56+ were observed in the sleep disorder group compared to the control group.

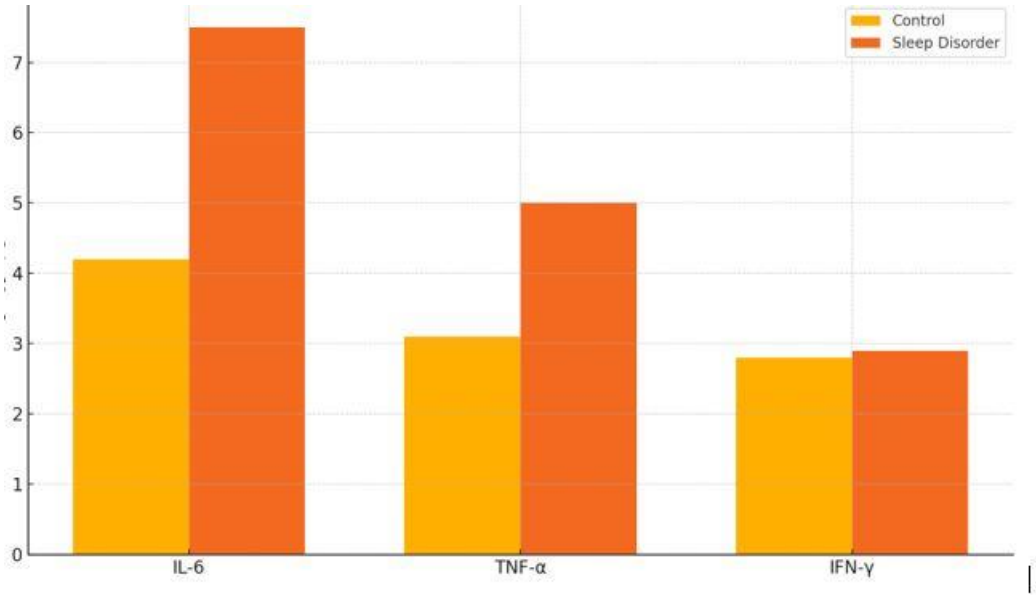

Figure 2. Serum levels of IL-6, TNF-α, and IFN-γ are shown. The sleep disturbance group had significantly higher levels of IL-6 and TNF-α compared to the control group.

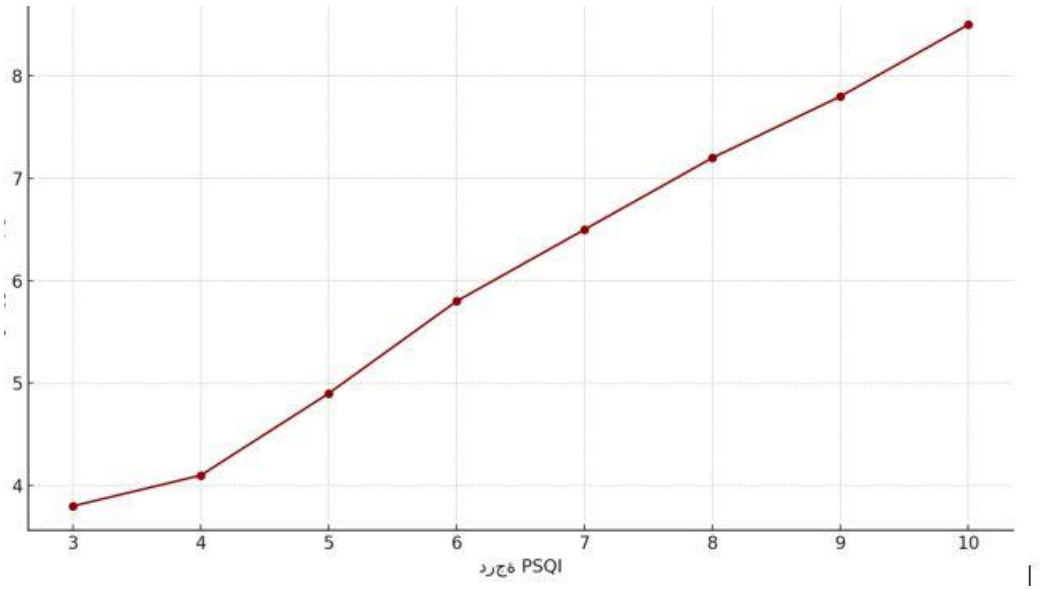

Figure 3. Shows a moderate positive relationship between higher PSQI scores (sleep disturbance) and increased IL-6 concentration, indicating the effect of sleep disturbances on inflammatory activity in the body.

Table 4. Distribution of immune cells in the two groups. It shows a significant decrease in the proportions of CD3+, CD4+, and CD56+ immune cells in the sleep disorder group compared to the control group, indicating a negative effect of sleep disturbance on cellular immunity.

Immune marker	Control group (n=30)	Sleep disturbance group (n=30)	p-value	Statistical significance
CD3+	65.2 ± 5.3	52.1 ± 6.4	0.001	Function
CD4+	40.3 ± 4.8	32.7 ± 5.0	0.002	Function
CD8+	25.4 ± 3.1	24.1 ± 3.0	0.210	Non-function
CD56+	15.8 ± 2.7	9.3 ± 2.9	0.000	Very function

Table 5. Serum immune cytokine levels. Significant increases in IL-6 and TNF-α concentrations were observed in the sleep disturbance group compared to the control group, while no significant differences were recorded in IFN-γ between the two groups.

Cytokine	Control group (n=30)	Sleep disturbance group (n=30)	p-value	significance
IL-6	4.2 ± 1.1	7.5 ± 1.6	0.000	Very functional
TNF-α	3.1 ± 0.9	5.0 ± 1.3	0.001	Functional
IFN-γ	2.8 ± 0.7	2.9 ± 0.8	0.650	Not functional

Table 6. Relationship between PSQI scores and IL-6 concentration. A moderate positive correlation is found between higher PSQI scores and higher IL-6 concentrations, indicating that increased severity of sleep disturbance is associated with increased immune inflammation in the body.

Variables	Correlation coefficient (r)	p-value
PSQI score and IL-6 concentration	0.58	0.000

Table 7. Correlation Between Sleep Disturbance Indicators and Immune Parameters

Variables Correlated	Correlation Coefficient (r)	p-value	Statistical Significance
PSQI vs IL-6	0.58	0.0	Highly Significant
PSQI vs TNF-α	0.42	0.003	Significant
PSQI vs CD3+	-0.49	0.001	Significant
PSQI vs CD4+	-0.46	0.002	Significant
PSQI vs CD56+	-0.53	0.0	Highly Significant
IL-6 vs CD56+	-0.51	0.001	Significant
TNF-α vs CD4+	-0.44	0.004	Significant
CD3+ vs CD4+	0.65	0.0	Highly Significant
CD4+ vs CD56+	0.38	0.008	Significant
IFN-γ vs PSQI	0.09	0.52	Not Significant

This table demonstrates significant positive correlations between PSQI scores and inflammatory markers such as IL-6 and TNF-α, indicating that sleep disturbances are associated with elevated pro-inflammatory responses. Conversely, significant negative correlations were observed between PSQI scores and immune cell markers (CD3+, CD4+, CD56+), suggesting that poor sleep negatively impacts cellular immune function. Furthermore, correlations among immune markers reveal interconnected dysregulation, underscoring the systemic nature of immune alteration in sleep-disturbed individuals.

CONCLUSION

Fundamental Finding : The results obtained from this study indicate a clear negative impact of sleep disturbances on the functional efficiency of the cellular immune system in university students. Students with sleep disturbances showed significant decreases in the percentages of CD3+, CD4+, and CD56+ lymphocytes, compared to individuals in the control group. A significant increase in the levels of the inflammatory cytokines IL-6 and TNF-α was also observed, reflecting the activation of inflammatory pathways resulting from altered sleep patterns. **Implication :** These results clearly indicate that sleep disturbances do not only affect psychological and cognitive aspects, but also profoundly impact the balance of the immune system, making it more susceptible to external and internal inflammatory stimuli. The moderate positive correlation between PSQI scores and IL-6 concentrations appears to reinforce this

hypothesis and opens the door to using sleep indicators as a predictor of chronic inflammatory activity. From a clinical and preventive perspective, this study recommends strengthening university health awareness programs on the importance of adequate and regular sleep for maintaining immune health, particularly among young people experiencing high academic and psychological stress. It also emphasizes the importance of incorporating sleep quality assessment into routine student screenings, especially for those with recurrent immune symptoms or chronic inflammatory disorders. **Limitation :** The study is limited to a specific age group and a defined set of immune markers, thus not capturing the full complexity of sleep-immune interactions across broader populations or with more diverse biological indicators. **Future Research :** Finally, this study represents a first step toward a deeper understanding of the relationship between sleep disturbances and cellular immunity in young people, and calls for further studies that include additional immune markers and analyze the effects of sleep on gene expression and immunity in the future.

REFERENCES

[1] S. P. Becker, M. A. Jarrett, A. M. Luebbe, A. A. Garner, G. L. Burns, and M. J. Kofler, "Sleep in a large, multi-university sample of college students: sleep problem prevalence, sex differences, and mental health correlates," *Sleep Health*, vol. 4, no. 2, pp. 174–181, 2018.

[2] H. K. Kang, C. Rhodes, E. Rivers, C. P. Thornton, and T. Rodney, "Prevalence of mental health disorders among undergraduate university students in the United States: A review," *J. Psychosoc. Nurs. Ment. Health Serv.*, vol. 59, no. 2, pp. 17–24, 2021.

[3] B. Sivertsen et al., "Sleep patterns and insomnia in young adults: A national survey of Norwegian university students," *J. Sleep Res.*, vol. 28, no. 2, p. e12790, 2019.

[4] M. Shoqeirat, A. J. Matarneh, M. I. A.-F. Salameh, L. S. M. Alhawari, and A. Algaralleh, "Analysis of sleep disorder prevalence among Jordanian university students: Influences of sociodemographic factors," *Pak. J. Life Soc. Sci.*, vol. 22, no. 1, 2024.

[5] R. De Graaf, M. Tuithof, S. Van Dorsselaer, and M. Ten Have, "Comparing the effects on work performance of mental and physical disorders," *Soc. Psychiatry Psychiatr. Epidemiol.*, vol. 47, pp. 1873–1883, 2012.

[6] World Health Organization, *The World Health Report 2003: Shaping the Future*. Geneva, Switzerland: WHO, 2003.

[7] L. Besedovsky, T. Lange, and M. Haack, "The sleep-immune crosstalk in health and disease," *Physiol. Rev.*, 2019.

[8] T. Mundula et al., "Chronic systemic low-grade inflammation and modern lifestyle: The dark role of gut microbiota on related diseases with a focus on COVID-19 pandemic," *Curr. Med. Chem.*, vol. 29, no. 33, pp. 5370–5396, 2022.

[9] J. A. Haspel et al., "Perfect timing: circadian rhythms, sleep, and immunity—an NIH workshop summary," *JCI Insight*, vol. 5, no. 1, p. e131487, 2020.

[10] N. Seyidoglu and C. Aydin, "Insight of the recent perspectives from psychoneuroimmunology: Stress, nutrition and life quality," in *PsychoNeuroImmunology: Vol. 1: Integration of Psychology, Neurology, and Immunology*, Springer, 2025, pp. 331–353.

[11] N. A. Zubaidi, M. K. AL-Azzawi, M. S. Mahdi, and M. H. Ajmi, "Investigating the influence of biosynthetic silver nanoparticles on kidney and tissue function in mice," *Exp. Theor. Nanotechnol.*, vol. 9, pp. 15–26, 2025.

[12] T. Feuth, "Interactions between sleep, inflammation, immunity and infections: a narrative review," *Immun. Inflamm. Dis.*, vol. 12, no. 10, p. e70046, 2024.

[13] S. Garbarino, P. Lanteri, N. L. Bragazzi, N. Magnavita, and E. Scoditti, "Role of sleep deprivation in immune-related disease risk and outcomes," *Commun. Biol.*, vol. 4, no. 1, p. 1304, 2021.

[14] S. A. Mohammed and N. S. Ahmed, "Self-assessment tool for improving student's skills regarding the exams in higher education," *Sci. J. Univ. Zakho*, vol. 11, no. 3, pp. 453–463, 2023.

[15] M. R. Zielinski, J. T. McKenna, and R. W. McCarley, "Functions and mechanisms of sleep," *AIMS Neurosci.*, vol. 3, no. 1, pp. 67–84, 2016.

[16] M. R. Irwin, "Sleep and inflammation: partners in sickness and in health," *Nat. Rev. Immunol.*, vol. 19, no. 11, pp. 702–715, 2019.

[17] L. Besedovsky, T. Lange, and J. Born, "Sleep and immune function," *Pflügers Arch. J. Physiol.*, vol. 463, no. 1, pp. 121–137, 2012.

[18] A. A. Prather, D. Janicki-Deverts, M. H. Hall, and S. Cohen, "Behaviorally assessed sleep and susceptibility to the common cold," *Sleep*, vol. 38, no. 9, pp. 1353–1359, 2015.

[19] S. Dimitrov et al., "Gαs-coupled receptor signaling and sleep regulate integrin activation of human antigen-specific T cells," *J. Exp. Med.*, vol. 216, no. 3, pp. 517–526, 2019.

[20] R. Ader and N. Cohen, "Psychoneuroimmunology: interactions between the nervous system and the immune system," *Lancet*, vol. 345, no. 8942, pp. 99–103, 1995.

[21] A. Arjona and D. K. Sarkar, "Circadian oscillations of clock genes, cytolytic factors, and cytokines in rat NK cells," *J. Immunol.*, vol. 174, no. 12, pp. 7618–7624, 2005.

[22] C. Benedict, M. Brytting, A. Markström, J. E. Broman, and H. B. Schiöth, "Acute sleep deprivation has no lasting effects on the human antibody titer response following a novel influenza A H1N1 virus vaccination," *BMC Immunol.*, vol. 13, pp. 1–5, 2012.

[23] K. Spiegel, J. F. Sheridan, and E. Van Cauter, "Effect of sleep deprivation on response to immunization," *JAMA*, vol. 288, no. 12, pp. 1471–1472, 2002.

[24] S. D. Hershner and R. D. Chervin, "Causes and consequences of sleepiness among college students," *Nat. Sci. Sleep*, pp. 73–84, 2014.

[25] D. J. Taylor, K. E. Vatthauer, A. D. Bramoweth, C. Ruggero, and B. Roane, "The role of sleep in predicting college academic performance: is it a unique predictor?" *Behav. Sleep Med.*, vol. 11, no. 3, pp. 159–172, 2013.

[26] J. F. Gaultney, "The prevalence of sleep disorders in college students: impact on academic performance," *J. Am. Coll. Health*, vol. 59, no. 2, pp. 91–97, 2010.

[27] H. A. M. Al-Ukaily, A. A. S. Alqaisi, H. M. Ali, S. A. Kashkul, W. J. Rasheed, and A. J. Abood, "Cytotoxic T lymphocytes' function in an adenovirus infection," *Am. J. Biol. Nat. Sci.*, vol. 2, no. 4, pp. 81–91, 2025.

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