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The Hidden Epidemic-How Modern Society's Clock is Making Us Sick: A Review of Social Jetlag and Its Health Consequences

Gizli Salgın: Modern Toplumda Sosyal Jetlag ve Sağlık Üzerindeki Etkilerinin Derlemesi

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Abstract

Social jetlag (SJL), a pervasive public health issue in modern society, is the chronic misalignment between an individual's internal biological clock and the social demands of their daily schedule. Unlike temporary jetlag from travel, SJL represents a continuous weekly cycle of circadian disruption. It is primarily driven by fixed work and school schedules, evening exposure to artificial light, and increased use of digital devices. This review focuses on the causes and health consequences of this chronic condition, which is increasingly recognized as a significant public health concern and has been estimated to affect around 70% of individuals in industrialized nations. The consequences of SJL extend far beyond sleepiness, contributing to an increased risk of obesity, metabolic syndrome, cardiovascular disease, and a weakened immune system. It also has significant negative impacts on mental health, correlating with higher rates of depression and anxiety, as well as cognitive deficits and poor academic performance, particularly among adolescents. SJL also imposes a substantial economic burden on society through healthcare costs and lost productivity. Addressing SJL requires a multifaceted approach, including individual behavioral changes, such as consistent sleep schedules and reduced evening light exposure, alongside broader societal and policy interventions, such as flexible work hours and later school start times. Raising public awareness of SJL is crucial to transforming it from a silent epidemic into a recognized public health priority.

Keywords: Social jetlag, circadian rhythm, public health, chronotype, sleep-wake cycle

Öz

Sosyal jetlag (SJL), modern toplumda yaygın görülen bir halk sağlığı sorunu olup, bireyin iç biyolojik saati ile günlük yaşamın sosyal gereklilikleri arasındaki kronik uyumsuzluğu ifade eder. Seyahat kaynaklı geçici jetlagden farklı olarak SJL, sabit iş ve okul saatleri, akşamları yapay ışığa maruz kalma ve dijital cihaz kullanımının etkisiyle ortaya çıkan, haftalık döngüler halinde devam eden bir sirkadiyen bozulmadır. Bu derleme, sanayileşmiş ülkelerde bireylerin %70'ine kadarını etkileyen bu kronik durumun nedenlerini ve yaygın sağlık sonuçlarını incelemektedir. SJL'nin sonuçları yalnızca uykululukla sınırlı kalmamakta; obezite, metabolik sendrom, kardiyovasküler hastalıklar ve zayıflamış bağışıklık sistemi riskinde artışa da yol açmaktadır. Ayrıca SJL'nin ruh sağlığı üzerinde de ciddi olumsuz etkileri bulunmakta; depresyon ve anksiyete oranlarının yükselmesi, bilişsel yetersizlikler ve özellikle ergenlerde akademik başarı düşüklüğü ile ilişkilendirilmektedir. Bunun yanı sıra SJL, sağlık harcamaları ve üretkenlik kaybı nedeniyle toplum üzerinde önemli bir ekonomik yük oluşturmaktadır. SJL ile mücadele bireysel davranış değişikliklerini (düzenli uyku saatleri, akşam ışığa maruziyetin azaltılması) kapsayan, bununla birlikte esnek çalışma saatleri ve okulların geç başlaması gibi daha geniş kapsamlı toplumsal ve politik düzenlemeleri de içeren çok boyutlu bir yaklaşım gerektirmektedir. SJL'nin sessiz bir salgın olmaktan çıkarılıp tanınan bir halk sağlığı önceliğine dönüştürülmesi için toplumsal farkındalığın artırılması büyük önem taşımaktadır.

Anahtar Kelimeler: Sosyal jetlag, sirkadiyen ritim, halk sağlığı, kronotip, uyku-uyanıklık döngüsü

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Introduction

The human body operates according to a complex, endogenous 24-hour internal clock known as the circadian rhythm. This rhythm primarily governs wakefulness and sleep cycles by responding to changes in environmental light (1). Located in the suprachiasmatic nucleus (SCN) of the hypothalamus, this intricate biological system coordinates physiological processes and behaviors, including sleep-wake cycles, hormone secretion, metabolism, and immune function, to optimize energy expenditure and the body's internal physiology (1). The fundamental functioning of this circadian system is vital for human health and well-being, as disruptions in circadian activity are associated with various adverse physiological, psychological, and clinical outcomes (2).

As a modern phenomenon, social jetlag (SJL) describes the chronic misalignment between an individual's biological clock and the social clock imposed by societal constraints, such as work and school schedules (3). Unlike travel-induced jetlag, SJL arises from persistent discrepancies in sleep timing between work or school days and free days, driven by rigid institutional schedules conflicting with natural light-dark cycles and individual chronotypes. This discrepancy often leads individuals to deviate from their natural sleep-wake cycles during weekdays, attempting to compensate on weekends. This creates a "jetlag-like" experience without actual travel (3). This chronic incongruity stems from a continuous conflict between the body's natural rhythms and social expectations, disrupting them and leading to wide-ranging health consequences. It is typically quantified as the difference in mid-sleep timing between workdays and free days (3,4). First described by Wittmann et al. (3) in 2006, SJL arises when people use alarms to wake before their natural wake time on workdays and then "oversleep" on weekends, leading to a weekly cycle of circadian disturbance (4,5).

The concept of SJL has gained prominence in recent years due to its high prevalence and potential health impacts. Epidemiological studies indicate that up to 70% of individuals in industrialized nations experience at least one hour of SJL weekly, with approximately 20–30% exhibiting misalignment exceeding two hours—a threshold consistently linked to adverse health outcomes (5-7). This widespread prevalence underscores its significance as a growing public health concern in modern 24/7 societies, where artificial lighting and around-the-clock activities contribute to circadian disruption (8).

Given the high prevalence of SJL and its initial symptoms often being perceived as milder than those of travel-induced jetlag (9), many individuals may live with chronic circadian disruption without fully realizing it or understanding its severity. Although the term "jetlag" typically refers to a temporary condition, SJL is chronic (10). Chronic circadian disruption (such as that from night shift work or jet lag) can increase risks for metabolic, cardiovascular, immune and psychiatric diseases. This chronic, often unnoticed condition, namely SJL, is becoming a kind of "invisible epidemic" silently eroding public health, as the internal-external time conflict affects sleep, behavior, and well-being on a large scale.

This review article has delved into the underlying mechanisms, causes, health effects, measurement methods, and potential prevention strategies of SJL, highlighting its critical importance as a public health issue in contemporary society.

Defining SJL: The Mismatch of Clocks

The concept of SJL emerged from observations using the Munich ChronoType Questionnaire (MCTQ), which enabled the quantitative assessment of chronic inconsistencies in sleep timing (10). The MCTQ provides a measure of chronotype and calculates SJL as the absolute difference in mid-sleep between workdays and free days (4,10).

Historically, before the introduction of time zones in the late 19th century, the "social clock" was largely synchronized with the "sun clock" (noon being when the sun reached its zenith). Modern life, with its fixed schedules and widespread use of artificial light, has significantly weakened the influence of natural light-dark cycles (zeitgebers), leading to a greater divergence between biological and social time (10). This phenomenon was first described in 2006 when researchers noticed that individuals in sleep diaries consistently appeared to be "flying west on Friday evenings and returning on Monday mornings" without actually traveling (10).

SJL represents a chronic discrepancy between internal and external timing (10). While SJL often involves accumulated sleep debt during weekdays due to early wake-up times and late bedtimes, it fundamentally concerns the misalignment of circadian rhythms, not merely insufficient sleep duration (10). Individuals can experience SJL even without a weekday sleep deficit (8). This distinction is crucial because the health effects of SJL are attributed to this chronic circadian disruption, not solely to sleep deprivation (11). SJL is defined by differences in sleep midpoints, suggesting that individuals try to "catch up on sleep" on weekends (3). However, oversleeping on weekends, while compensating for sleep debt, increases SJL by delaying the free-day sleep midpoint (7). This indicates that the act of "catching up" perpetuates the misalignment, creating a continuous cyclical problem. As the body constantly tries to adapt, societal pressures pull it back.

Circadian Mechanisms and Chronotypes - Mechanisms of Circadian Misalignment

Biological clocks coordinate almost every aspect of human physiology with the 24-h day. The human circadian system is regulated by a central "master clock" located in the SCN of the hypothalamus (2). The SCN synchronizes the 24-hour rhythms of various physiological functions and behaviors throughout the body (2). The SCN is reset daily by "zeitgebers" or time cues from the external environment, which allows the body's internal rhythms to synchronize with the outside world (12). Circadian rhythms are synchronized to the external environment primarily by "zeitgebers" (German for "time givers"), with light being the most dominant and powerful cue (8). Natural light-dark cycles entrain the biological clock to solar time, ensuring that

the internal biological night begins near sunset and ends near sunrise (13). Reduced exposure to daylight and increased exposure to artificial light at night can delay circadian and sleep timing (13).

Humans exhibit significant individual differences in their preferred sleep and activity timing, known as chronotypes, which are largely regulated by genetic variations in clock genes and environmental influences (3). These chronotypes range from "early types" (larks) who prefer earlier sleep and wake times, to "late types" (owls) who prefer later timings, with most individuals falling into "intermediate types" between these extremes (3). Most individuals are intermediate, but chronotype follows a near-normal distribution across populations (7,14). Chronotype depends partly on genetics and age: children tend to be early types, while puberty shifts clocks later; adolescents peak in "night owl" preference around ages 16–18 (7,15). Morning types preferentially wake and sleep early, with peak alertness in the morning, whereas evening types naturally go to bed late and wake late (7,14). Evening chronotypes report feeling and performing best in the late afternoon or evening (16).

The standard organization of modern society often favors early chronotypes, forcing late chronotypes to conform to routines that conflict with their internal biological time (8). This is particularly evident in fixed school and work schedules that require early wake-up times (3). This interference creates a chronic discrepancy between an individual's preferred sleep timing and their actual sleep timing on workdays, leading to SJL (3).

The human circadian system has evolved over billions of years to synchronize with natural light-dark cycles (1). However, modern society, especially since the widespread availability of electricity, has significantly weakened natural zeitgebers and introduced artificial light at night (10). This means the "social clock" has largely decoupled from the "sun clock," forcing human biology to work against its evolutionary programming. This suggests that addressing SJL is not merely about individual discipline but requires a fundamental re-evaluation of societal structures and environmental design. Public health strategies should acknowledge this "forced evolution" and advocate for changes in the built environment and societal schedules to better align with natural human biology.

Furthermore, light is the most powerful natural zeitgeber (8). However, modern lifestyles involve widespread exposure to artificial light, particularly blue light from digital devices, which directly suppresses melatonin secretion and delays circadian timing (17). This indicates that digital devices may not only contribute to SJL but may also act as a new, artificial zeitgeber that actively misaligns our biological clocks, often overriding natural light cues.

Causes and Contributing Factors of SJL

SJL arises whenever societal schedules force a mismatch with biological timing (7,10). Because adolescents naturally shift toward a later chronotype during development, they are particularly vulnerable to SJL when required to adhere to conventional early school start times (3,5,18). The increasing demand for productivity and competitiveness in modern society contributes to routines that diverge from an individual's internal time (8). Shift work, particularly night shifts, is a major contributor, forcing individuals into unnatural light-dark patterns, leading to severe circadian disruption and high levels of SJL. Permanent night shift workers experience the highest SJL (e.g., 5:08 hours) (8). Early school start times are also a significant contributing factor. Conventional 8–9 AM school start times are often too early for adolescents and many young adults. To meet those obligations, they must wake before their internal clock signals full alertness, using an alarm to truncate sleep (3,5).

The widespread availability of artificial light, especially in the evenings, has profoundly impacted circadian rhythms (10). Living predominantly indoors shields individuals from full daylight exposure, while evening artificial light delays biological clocks (10). Excessive exposure to blue light from electronic devices (smartphones, laptops, tablets) is a critical modern factor (19). Blue light, particularly at wavelengths of 400-500 nm, suppresses melatonin secretion, which is vital for sleep regulation, and can significantly delay sleep onset, thereby disrupting circadian rhythms (17). Recent adolescent studies show that bedtime screen use correlates with a later chronotype and increased SJL (20). Studies show that even 2 hours of LED tablet exposure can reduce melatonin levels by 55% and delay its onset by 1.5 hours (17). Thus, digital lighting and 24/7 media consumption exacerbate the misalignment. In summary, chronotype–social clock conflict is at the core: evening types are forced to conform to early schedules, while society seldom adjusts to their biology (3). Any pressure to advance sleep (social clock) or delay it beyond natural timing can produce SJL.

Health Effects of SJL: A Broad Spectrum of Adverse Outcomes

SJL, as a consequence of disrupted circadian rhythms, is a complex public health issue leading to a wide range of physical, mental, and behavioral health problems. This condition is not merely limited to sleep patterns but represents a chronic state of misalignment affecting multiple bodily systems. SJL is strongly associated with poor sleep quality and increased daytime sleepiness (3). Studies show that individuals with ≥2 hours of SJL experience significantly higher daytime sleepiness (21). Studies have found that high-SJL individuals exhibit reduced heart rate variability during sleep (a sign of poorer sleep quality) and report more fatigue (7). In effect, SJL often doubles as an ongoing form of insomnia/deprivation on weekdays.

SJL is significantly associated with various cardiometabolic risk factors. High-quality studies indicate that SJL is associated with a higher body mass index (BMI), increased waist circumference, higher systolic blood pressure, and elevated glycated hemoglobin (22). Studies found that people with longer SJL had higher fasting cortisol levels and a dyslipidemic profile (higher triglycerides, total cholesterol, and glucose) than those with minimal SJL (4,23-26). At the population level, SJL has been associated with higher BMI and waist circumference.

Several epidemiological studies report that greater SJL predicts a higher prevalence of obesity and metabolic syndrome (7,15). Thus, SJL appears to contribute to the modern epidemics of overweight, metabolic syndrome and type 2 diabetes.

SJL is also positively correlated with mental health issues, particularly depressive and anxiety symptoms in young people and workers, with individuals experiencing ≥2 hours of SJL showing higher odds of clinically significant depression and anxiety (5,27-29).

Behavioral risk factors linked to SJL include higher rates of smoking (30,31), increased alcohol consumption (32), and poorer diet quality with higher intake of sugary beverages, total fat, and saturated fat (33).

In one survey, teens reporting night-time screen use had significantly more SJL, which mediated effects on mood and anxiety (20). SJL has been associated with multiple adverse health outcomes. It is linked to a weakened immune system and circadian rhythm disruptions.

Furthermore, SJL is associated with cognitive deficits, including reduced crystallized intelligence and lower academic performance from early adolescence through university (30,31), as well as a lower overall quality of life (27). In children with autism spectrum disorders, SJL correlates positively with core symptoms, particularly in 2–3-year-olds, indicating their sensitivity to routine changes (34).

While direct associations with occupational accidents are less clear, shift work—a major cause of SJL—is linked to increased accident risk, reduced psychomotor vigilance, and impaired workplace alertness and performance (7,35,36). Collectively, these findings underscore that SJL is a significant public health concern that affects multiple domains of well-being. Assessment and management of SJL should be integrated into clinical practice, especially for patients predisposed to metabolic, sleep, or mental health disorders, and public health screenings could incorporate SJL evaluation to identify at-risk populations where disease progression might be prevented.

Economic and Societal Burden of SJL

The economic burden of SJL is substantial and quantifiable. A spatial regression discontinuity study conducted along US time zone borders takes advantage of a unique natural experiment. Counties on the western edge of a time zone experience later sunsets but follow the same clock times and social schedules as counties on the eastern edge. Utilizing data from the American time use survey and the behavioral risk factor surveillance system, researchers found that an additional hour of evening natural light reduces average sleep duration by 19 minutes and significantly increases the probability of insufficient sleep (defined as <6 hours) (37). The misalignment leads to a 0.3 standard deviation decline in a composite health index, reflecting higher population-level rates of obesity, diabetes, cardiovascular disease, and breast cancer on the late sunset side of the border (37). Notably, no significant residential sorting—measured through home values, rents, or commuting patterns—was observed, suggesting that individuals do not relocate to mitigate circadian disruption, likely due to structural constraints or behavioral trade-offs between evening leisure and sleep (37). This natural experiment provides robust causal evidence on the health effects of socially imposed timing. It shows that circadian misalignment—independent of individual behavior—contributes to population-level health disparities. The economic burden of SJL is estimated to result in at least $2 billion in annual healthcare expenditures and the loss of 4.4 million workdays due to reduced productivity and increased illness (37). At the individual level, SJL exceeding two hours is associated with an 18% reduction in work ability index scores, a validated metric of work capacity and functional ability (36).

Measurement of SJL

Accurate assessment of SJL is a fundamental step for research, clinical awareness, individual interventions, and public health strategies. The MCTQ is the most widely used self-report tool for assessing chronotype and quantitatively determining SJL (38). It calculates SJL as the absolute difference between the midpoint of sleep on free days and the midpoint of sleep on work/school days. A "sleep-corrected" version (SJLsc) has been proposed to account for accumulated sleep debt, which might otherwise overestimate SJL (39). The MCTQ is a quick, cost-effective, and non-invasive tool that has been validated across diverse populations, making it well-suited for large-scale epidemiological studies of chronotype and SJL. In research, SJL is often categorized (e.g., <1 h, 1–2 h, ≥2 h) to examine dose–response relationships with health outcomes. The Morningness-Eveningness Questionnaire is another tool used to assess diurnal preferences and categorize individuals into morningness-eveningness types, showing good correspondence with MCTQ chronotype (40). Other approaches include actigraphy and circadian phase markers. Wrist actigraphy (wrist motion tracking) is used to objectively measure sleep-wake intervals and derive sleep parameters, including sleep midpoint, which can then be used to calculate SJL. It provides a more objective assessment compared to self-reports. Objective physiological markers of circadian phase include Dim light melatonin onset (DLMO) and core body temperature acrophase (7,41). DLMO is considered the gold standard for assessing the phase of entrainment (41).

SJL research often suffers from varying methodologies and definitions, making comparisons across studies challenging. Self-reported sleep data, while practical, can be prone to bias, especially in individuals with emotional problems. Objective measures like DLMO are expensive and invasive, limiting their use in large-scale epidemiological studies. The initial SJL calculation can be confounded by sleep debt, leading to overestimation; sleep-corrected versions aim to address this (39). This creates a "measurement dilemma" for public health in accurately assessing SJL prevalence and impact across large populations without prohibitive costs or methodological compromises. Surveys like the MCTQ remain the standard for epidemiological assessment of SJL.

Prevention and Mitigation Strategies: A Multifaceted Approach

Given the high prevalence and wide-ranging health effects of SJL a multifaceted approach is necessary to address this public

health issue. These strategies extend from individual behavioral changes to technological solutions and broad societal policies.

Individual Strategies

The cornerstone is promoting regular sleep–wake schedules even on free days. Maintaining consistent sleep-wake patterns, even on weekends, is critical to minimize SJL. Reducing evening blue light exposure can advance melatonin secretion and sleep onset, helping late chronotypes better cope with early social schedules (42). Conversely, increasing morning light exposure can also help advance circadian timing (42). These behavioral changes are key for individuals to take control of their circadian health. Educational programs, such as teacher-led sleep education and awareness interventions, show promise in improving total sleep time and reducing SJL in adolescents (43). Such programs can advance sleep onset and significantly decrease SJL, especially in those with severe SJL at baseline (43).

Given the impact of evening blue light from digital devices on melatonin suppression and circadian disruption (17), technological interventions, such as blue light-blocking glasses or screen filters (e.g., orange or amber-tinted lenses) can reduce short-wavelength light exposure before bedtime (42). While findings are mixed, some studies suggest these filters can improve sleep efficiency and total sleep time, particularly in individuals prone to sleep disturbances (44). Overall screen time management, especially at night, is also critical (19). Many sleep-tracking apps help individuals monitor their sleep timing and consistency, potentially flagging excessive weekend oversleep. These digital tools could be adapted to target SJL by promoting consistent sleep schedules and adjusting light exposure.

Societal and Policy Interventions

Aligning societal schedules with human biology is crucial for reducing SJL. Flexible work hours and later school start times for adolescents can help minimize the chronic discrepancy between work or school demands and biological time, particularly for late chronotypes (3,10). The abolition of daylight saving time has also been proposed to reduce the circadian misalignment imposed by it (11). Urban planning that maximizes daytime light exposure while minimizing night-time artificial light can support circadian rhythms and further reduce SJL (12).

Public health initiatives play a key role in raising awareness about the significance of SJL (8,19). While individual strategies—such as managing evening light exposure and maintaining consistent sleep routines—can provide meaningful benefits, they are insufficient on their own. Effectively addressing SJL requires systemic changes in work, school, and societal scheduling practices, particularly for vulnerable groups such as adolescents and shift workers. A comprehensive, "whole-of-society" approach is therefore needed, in which individual behaviors are supported by enabling environments and reinforced through evidence-based policy measures.

Conclusion

SJL, characterized by the chronic misalignment between our biological clocks and societal demands, is a prevalent and often underestimated public health issue in modern industrialized societies. Its high prevalence in various age groups, particularly among adolescents and young adults, underscores its profound societal impact.

The roots of SJL are deeply embedded in modern lifestyles, including fixed work/school schedules, shift work, and the pervasive presence of artificial light, especially blue light from digital devices, which disrupts natural circadian entrainment. The consequences extend beyond mere sleep disturbance, encompassing a broad spectrum of adverse health outcomes. Despite increasing awareness, the precise causal pathways and long-term consequences of SJL remain poorly understood. Addressing these gaps will require large-scale, population-based longitudinal studies. Interdisciplinary research, integrating chronobiology, public health, medicine, and the social sciences, is essential to fully unravel the complex interplay of factors contributing to SJL and its health effects. Given these findings, an equity-focused perspective is essential. Data suggest that the prevalence and health consequences of SJL may differ across socioeconomic, ethnic, and gender groups, potentially reinforcing existing health disparities. Public health research should explicitly investigate the differential prevalence and impact of SJL in diverse demographic groups. Interventions should be tailored to address specific vulnerabilities and ensure equitable access to circadian health resources, making SJL a component of broader health equity initiatives. Public health organizations must lead awareness campaigns to educate communities about the profound importance of circadian health. Prioritizing circadian alignment is not just about improving sleep; it is about building a healthier, more resilient society.

Footnotes

Authorship Contributions

Concept: S.U.U., Design: S.P., S.U.U., Data Collection or Processing: S.P., Analysis or Interpretation: S.P., S.U.U., Literature Search: S.P., S.U.U., Writing: S.P., S.U.U.

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Review / *Derleme*

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Sleep, the Immune System, and Inflammation: A Comprehensive Review and Update

Uyku, İmmün Sistem ve Enflamasyon: Kapsamlı Derleme ve Güncelleme

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Abstract

Neurotransmitters in the central nervous system, such as serotonin, noradrenaline, and dopamine, play important roles not only in sleep-wake cycle regulation and neurobehavioral modulation, but also in the regulation of immune system functions. Sleep and the immune system exhibit a bidirectional relationship. Beyond immune regulation, cytokines are involved in various other physiological processes, including memory, appetite, cognition, pain, fatigue, and sleep. Pro-inflammatory cytokines released from Th1 cells predominantly increase the duration of the non-rapid eye movement sleep stage and exert a pro-somnogenic effect. Other molecules such as nitric oxide and adenosine are also known mediators of sleep-immune interactions. During sleep, the production and release patterns of neurotransmitters and neurohormones undergo substantial changes, which are accompanied by alterations in immune cell functions and cytokine levels. In addition to circadian effects, sleep itself, and especially slow-wave sleep, modulates the expressions and levels of cytokines and hormones. Consequently, sleep disturbances, fragmentation, and disorders such as sleep apnea are associated with a significantly increased risk of developing cardiovascular disorders and cancer, driven by elevated inflammation.

Keywords: Sleep, immune system, cytokines, inflammation

Öz

Santral sinir sisteminde serotonin, noradrenalin veya dopamin nörotransmiterler, sadece uyku-uyanıklık döngüsü ya da davranışsal düzenlemede rol almakla kalmaz, aynı zamanda immün sistem ile ilişkili pek çok fonksiyonun düzenlenmesinde de önemli görev üstlenirler. Uyku ve immün sistem arasında iki yönlü bir ilişki mevcuttur. Sitokinlerin sadece immün sistem içerisinde değil, hafıza, iştah, bilişsel fonksiyonlar, ağrı, yorgunluk ve uyku gibi pek çok fonksiyonlarda görev aldıkları tanımlanmıştır. Özellikle Th1 hücrelerinden salınan pro-enflamatuvar sitokinler, çoğunlukla hızlı olmayan göz hareketli uyku, uyku evresinde artışa yol açarak, pro-somnojenik etki de gösterirler. Sitokinlerin yanı sıra, nitrik oksit ve adenozin gibi moleküllerin de immün sistem ve uyku aracılı etkileşimde rol oynadığı bilinmektedir. Uyku esnasında ise, hem nörotransmiterlerin ve nörohormonların salınım paterni değişir, hem de immün sistem ile ilişkili hücrelerin fonksiyonlarında ve sitokin seviyelerinde değişiklikler meydana gelir. Sirkadiyen etkilerin yanı sıra, uyku ve özellikle yavaş dalga uykusu hormon ve sitokin seviyeleri üzerinde etkilidir. Uykunun bozulması, fragmante olması ya da eşlik eden uyku apnesi gibi bozuklukların varlığında ise, enflamasyon artışı ile ilişkili kardiyovasküler hastalıklar ve kanser gelişim riskinde belirgin bir artış izlenir.

Anahtar Kelimeler: Uyku, bağışıklık sistemi, sitokinler, enflamasyon

Introduction

Every organ and physiological system within the human body relies on homeostatic mechanisms that are inextricably linked to immune and inflammatory functions. The cells mediating these functions interact continuously, utilizing a shared repertoire of peptides, neurotransmitters, and soluble molecules (1). Neurotransmitters such as serotonin, noradrenaline, and dopamine in the central nervous system (CNS) not only play a role in the sleep-wake cycle and behavioral regulation but also perform critical regulatory functions within the immune system (2-4). Immune system responses during inflammation are regulated by the "stress axis," which is formed by interactions among the hypothalamus, pituitary gland, and adrenal glands (5). Alongside this axis, various organs modulate the immune system (Figure 1). For example, skeletal muscle exerts an anti-inflammatory effect via myokines secreted into the circulation following sustained contraction during exercise (6). Lipopolysaccharides entering the bloodstream due to impaired gastrointestinal epithelial function can readily trigger

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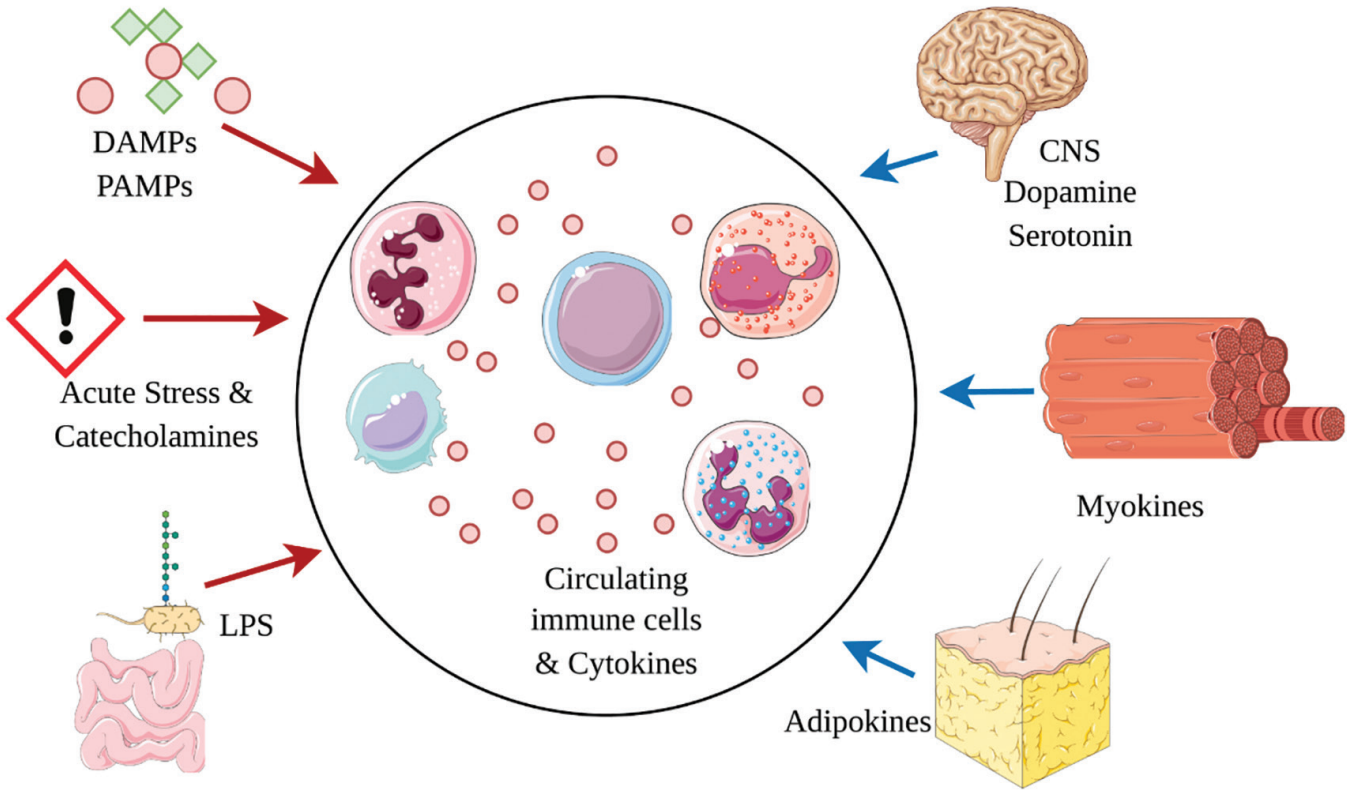

Figure 1. Regulators of circulating immune cells and cytokines. Peripheral inflammatory cues, including damage-associated molecular patterns, pathogen-associated molecular patterns, and bacterial lipopolysaccharide, as well as acute stress-related catecholamines promote immune activation and cytokine release (red arrows). In parallel, central and peripheral modulatory signals including central nervous system neurotransmitters (dopamine and serotonin), skeletal muscle-derived myokines, and adipose tissue-derived adipokines regulate and constrain immune cell function and cytokine profiles in a context-dependent manner (blue arrows). Some icons used in this figure were sourced from bioicons.com and are used under CC BY 3.0 and CC BY 4.0 licenses.
DAMPs: Damage-associated molecular patterns, PAMPs: Pathogen-associated molecular patterns, LPS: Lipopolysaccharide.

an immune response (7). Adipose tissue also suppresses immune function through the secretion of adipokines (8). Finally, immune-mediated molecules act across multiple areas of the CNS, including sleep-wake centers, exerting autoregulatory effects.

The relationship between the CNS and the immune system is highly integrated and bidirectional (9). Communication between these systems via cytokines and neurohormones was first suggested by Besedovsky et al. (10) in the 1970s (11). Subsequent studies have established that cytokines, once thought to operate exclusively within immunological boundaries as messengers between leukocytes, are actually pleiotropic regulators of memory, appetite, cognition, pain, fatigue, and sleep architecture (12). They can exert both pro- and anti-inflammatory, as well as somnogenic and anti-somnogenic effects. Moreover, they can also modulate circadian gene expressions (13).

In this review, we aimed to synthesize recent, clinically relevant literature on the interactions among sleep, the immune system, and inflammation, integrating experimental and human data to provide an updated framework for clinicians and researchers.

The Immune System as a Regulator of Sleep Architecture

The inflammatory response to invading pathogens or tissue injury fundamentally alters healthy sleep architecture, a phenomenon recognized clinically and biologically as "sickness behavior" (14). From an evolutionary perspective, sickness behavior—which includes lethargy, social withdrawal, loss of appetite, and increased sleepiness—is a highly conserved adaptive response designed to conserve energy and redirect metabolic resources toward mounting a robust immune defense. In general, pro-inflammatory cytokines exhibit pro-somnogenic effects, predominantly increasing the duration and intensity of non-rapid eye movement (NREM) sleep, while anti-inflammatory cytokines largely exert anti-somnogenic or wake-promoting effects (Table 1).

The interleukin (IL) 1 family represents a broad and complex network of cytokines comprising 11 different members that

exhibit both pro-inflammatory (e.g., IL-1α, IL-1β, IL-18, IL-33) and antagonistic or anti-inflammatory (e.g., IL-36Ra, IL-37, IL-38) properties (15). The profound relationship between IL-1 and sleep regulation was first identified in 1984, and its somnogenic impact is highly dependent on both the dosage and the circadian timing of administration. Under normal, healthy conditions, IL-1 levels fluctuate with the sleep-wake cycle, peaking at sleep onset. At low physiological doses, IL-1α and IL-1β can trigger sleep without inducing a febrile response, specifically enhancing the duration and electroencephalographic delta-wave intensity of NREM sleep. However, at higher, pathological doses mimicking a severe infection, IL-1 induces fever while simultaneously suppressing rapid eye movement (REM) sleep to prioritize deep, restorative NREM stages (12). The endogenous or exogenous inhibition of IL-1 signaling and the action of anti-inflammatory cytokines such as IL-4, IL-10, and IL-13 inhibit NREM sleep (16,17).

IL-6 is another highly potent pro-inflammatory and pro-somnogenic cytokine. Elevated levels of circulating IL-6 are known to increase NREM and decrease REM sleep duration. IL-6 is linked to the circadian system, modulating the molecular transcription of the *Per1* gene, thereby bridging the immune response with the body's internal clock (18,19). A pivotal study investigated the administration of tocilizumab, a targeted IL-6 receptor inhibitor, to patients suffering from rheumatoid arthritis. The intervention resulted in significant, rapid improvements in sleep quality and a marked reduction in daytime sleepiness (20). The authors stated that these beneficial effects occurred independently of the decrease in disease activity. However, another comprehensive study failed to replicate such effects, instead reporting that tocilizumab was associated with poorer sleep quality (21).

Tumor necrosis factor alpha (TNF-α) shares similar somnogenic properties with the IL-1 family. Recognized for its sleep-promoting effects in 1987, TNF-α is essential not only to inflammation but also to apoptosis, neuroplasticity, and appetite regulation. The expression of TNF-α in the brain increases proportionally with the duration of wakefulness. Like IL-1 and IL-6, TNF-α enhances NREM sleep duration and intensity while causing a compensatory reduction in REM sleep duration, playing a central role in the homeostatic sleep drive (12,22).

Beyond the classical cytokine networks, nitric oxide (NO) and adenosine are also known to mediate interactions between the immune system and sleep. NO, which has important functions in various immunological and physiological processes, demonstrates a circadian pattern in brain tissue that parallels the homeostatic need for sleep; increased NO levels correlate with a heightened urge to sleep. Studies have shown that both NREM and REM sleep stage duration increased following the administration of NO or L-arginine (an NO precursor) (12,23,24). Adenosine is a nucleoside associated with increased homeostatic drive after a period of prolonged wakefulness. It is released from astrocytes during inflammation, especially as part of the cerebral response to lipopolysaccharides originating from the gastrointestinal tract. It exerts a sleep-enhancing effect by stimulating adenosine-1 receptors (25).

Table 1. Classification of cytokines according to their pro-/anti-inflammatory effects, pro-/anti-somnogenic effects, and their associations with the innate versus adaptive immune system (the effects of immune cell populations and certain interleukins (*) may be dose-dependent or, when administered exogenously may vary according to circadian rhythm).

Cytokines	Pro-somnogenic	Anti-somnogenic	Immune system
Pro-inflammatory	IL-1β* IL-1α* TNF-α TNF-β IL-6* IL-15 IL-18 NGF Neurotrophin-3 Neurotrophin-4 Interferon-α* Interferon-γ*	IL-1β* IL-1α* IL-6* Interferon-α* Interferon-γ*	Innate
	IL-2		Adaptive
Anti-inflammatory	Epidermal growth factor	IL-10 IL-1Ra Insulin-like growth factor* Soluble TNF receptor Soluble IL-1 receptor	Innate
		IL-4 IL-5 IL-13 Transforming growth factor-β	Adaptive

IL: Interleukin, TNF-α: Tumor necrosis factor-alpha, TNF-β: Tumor necrosis factor-beta, NGF: Nerve growth factor

The immune system and inflammation have sleep-regulating effects. Inflammation in response to a pathogen alters healthy sleep structure. The inflammatory process is highly complex, exerting its effects on sleep by interacting with both neurons and glial cells. Through these interactions, neuronal cellular pathways are activated, and localized inflammation arising in specific brain regions accelerates sleep onset. In other words, local inflammation in the brain naturally increases during sleep (26).

Neuroinflammation

Pro-inflammatory cytokines can cross to the CNS, interact with the brain's cytokine network, and contribute to the regulation of multiple behavioral functions (27). Cytokines released from activated monocytes and macrophages reach the CNS through three main routes: humoral, neural, and cellular. In the humoral pathway, entry occurs through regions with higher blood-brain barrier permeability, such as the choroid plexus and circumventricular organs. Within the brain parenchyma, activated endothelial cells promote the release of mediators such as NO, thereby driving the activation of specific brain regions. In the neural pathway, peripheral afferent fibers of the vagus nerve, upon stimulation by pro-inflammatory cytokines, relay signals to the nucleus of the solitary tract and the area postrema. Finally, in the cellular pathway, circulating pro-inflammatory cytokines—particularly TNF-α—can directly activate brain-resident monocytes and related myeloid cells, thereby contributing to the development of neuroinflammation.

Cytokines influence a wide range of physiological domains within the CNS. For example, their effects on the basal ganglia can modulate motivation and motor activity, whereas their impact on the anterior cingulate cortex can shape circuits related to mood and anxiety. Cytokines also regulate neuronal growth, neurogenesis, and synaptic plasticity through interactions with neurotrophic factors. In addition, they affect the metabolism of key neurotransmitters, including serotonin, dopamine, and glutamate, thereby contributing to mood regulation and sleep control. Neuronal metabolic activity leads to the release of "sleep-regulatory molecules" such as adenosine, NO, IL-1β, and TNF-α, which shift local networks toward sleep. These same mediators also act at larger circuit levels. For instance, microinjections of IL-1β or TNF-α into the anterior hypothalamus markedly increase total sleep time, while unilateral cortical cytokine administration increases ipsilateral slow-wave activity. Thus, the complex relationship between local networks and whole-organism sleep is coupled through shared cytokines and metabolites (28).

A broad range of cytokines participate in sleep regulation (27,29). Pro-inflammatory cytokines, particularly those associated with Th1 responses, often exert pro-somnogenic effects by promoting NREM sleep. Key pro-somnogenic mediators, including IL-1, TNF-α, and neurotrophic factors, activate intracellular inflammatory mediators such as nuclear factor κB (NF-κB) and increase the production of prostaglandin D2, adenosine, NO, and growth hormone-releasing hormone. This strengthens the homeostatic sleep drive and facilitates NREM sleep initiation. Pro-inflammatory cytokines also bias the balance between inhibitory and excitatory neuronal and glial signaling—mediated by gamma-aminobutyric acid and glutamate—toward sleep. Beyond their effects on homeostatic regulation, pro-inflammatory cytokines also influence circadian processes. They inhibit the effect of the *BMAL1–CLOCK* complex on the *CRY–PER* complex, further contributing to sleep initiation (29). Moreover, cytokines and neurohormones exhibit circadian rhythmicity. During wakefulness, the increase in cortisol (associated with increased CXCR4 expression) promotes the trafficking of naïve T helper cells toward the bone marrow (30). Similarly, the high levels of adrenaline during wakefulness facilitate the mobilization of cytotoxic natural killer cells from marginal pools into the circulation by reducing CX3CR1/CD11a signaling. During sleep, both neurotransmitter and neurohormone release patterns shift, accompanied by changes in immune cell function and cytokine levels.

CX3CR1 signaling is also critical for microglial activity and the maintenance of neuroimmune homeostasis. CX3CR1, a receptor predominantly expressed on microglia in the CNS, follows a circadian rhythm that dictates microglial morphology and their surveillance of neuronal synapses. Healthy sleep-wake cycles ensure the proper regulation of this signaling, facilitating synaptic pruning and preventing excessive neuroinflammation. However, sleep disruption can impair CX3CR1-mediated pathways, leading to aberrant microglial activation and a shift toward a pro-inflammatory state. Consequently, nocturnal sleep serves as a fundamental regulator of the brain's internal immune environment through these specific molecular checkpoints (31).

Beyond the CNS, nighttime sleep also reshapes peripheral immune trafficking between the circulation and lymphoid tissues. Lower cortisol levels during sleep compared to wakefulness facilitate the egress of T helper cells from the bone marrow into the circulation and their subsequent homing to lymph nodes (30). Thus, circulating T helper cell levels are higher during nocturnal sleep. In contrast, lower adrenaline levels promote redistribution of cytotoxic natural killer cells from the circulation to marginal pools, resulting in lower circulating natural killer cell counts during sleep.

These changes in neurohormonal tone and immune cell distribution translate into substantial alterations in cytokine profiles. Decreases in cortisol and adrenaline/noradrenaline reduce anti-inflammatory cytokine production by monocytes, particularly IL-10. Conversely, increases in growth hormone and prolactin are accompanied by marked rises in pro-inflammatory cytokines released from monocytes and dendritic cells, including IL-6, TNF-α, and IL-12. Collectively, these shifts contribute to a more pro-inflammatory milieu in the brain during sleep, consistent with enhanced nocturnal neuroinflammatory activity.

Effects of Sleep on the Immune System

Circadian rhythms shape the secretion profiles of many hormones and cytokines. Sleep—especially slow-wave sleep—exerts an additional, state-dependent influence on their circulating levels (32). During this sleep phase, a significant increase in growth hormone and prolactin secretion is observed,

as well as a decrease in cortisol levels. Comparisons with nocturnal wakefulness demonstrate that this is a true "sleep" effect rather than merely a "night" effect (30). Catecholamine levels also decrease during sleep compared to periods of nocturnal wakefulness, particularly during the REM sleep phase. Consequently, sleep increases TNF-α and IL-12 production while suppressing IL-10 production via its modulation of neurohormonal levels. Conversely, IL-6 production is controlled solely by circadian effects. Indeed, comparisons with nocturnal wakefulness reveal no additional effect of sleep.

As discussed above, a decrease in cortisol and epinephrine/norepinephrine levels and an increase in growth hormone, prolactin, and melatonin levels occur during healthy sleep via the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system (33). When sleep is disrupted, increased cortisol levels suppress pro-inflammatory gene transcription by stimulating glucocorticoid receptors, directly inhibiting pro-inflammatory mediators such as NF-κB through protein-protein interactions and increasing anti-inflammatory gene transcription. As a result, Th1 cell counts and interferon (IFN) levels decrease, while Th2 cell count and IL-10 levels increase. The grand sum of these effects is the anti-inflammatory effect associated with the adaptive immune system.

In the presence of sleep disruption, fragmentation, or comorbid sleep disorders such as sleep apnea, sympathetic nervous system activation occurs. This causes an increase in epinephrine/norepinephrine levels, which normally decrease during healthy sleep. As a result, β-adrenergic receptors (such as ADRB2) in leukocytes are stimulated, increasing pro-inflammatory *IL-1β, IL-6* and *TNF* gene transcription and suppressing anti-inflammatory *IFN* gene transcription. This leads to susceptibility to infection and poor vaccine response. Sleep disruption also negatively impacts the innate immune system. Similarly, CNS activation and the stimulation of leukocyte β-adrenergic receptors increase NF-κB secretion, resulting in the production of IL-6 and TNF-α. The interaction between the brain and peripheral tissues enables the brain's modulating effect on the immune system, whereas inflammatory activity reciprocally affects neural processes and sleep patterns. Disruption of this dynamic is associated with increased inflammation and heightened risk of cardiovascular diseases and cancer.

Sleep exerts a distinct effect on leukocyte trafficking beyond circadian timing. Although circulating leukocytes show robust day–night rhythms, controlled human studies demonstrate that nocturnal sleep itself acutely lowers blood counts of monocytes, natural killer cells, and major lymphocyte subsets compared with sustained nocturnal wakefulness, consistent with redistribution from the blood to tissue/lymphoid compartments (33,34). This sleep-related reduction extends across multiple T-cell populations. In detailed phenotyping, sleep uniformly decreased the numbers of diverse CD4+ and CD8+ T-cell subsets during the night relative to nocturnal wakefulness (35). Sleep selectively enhances the migratory potential of several T-cell subsets toward CCL19, a key chemokine for lymph node homing. This effect can be reproduced using plasma from sleeping participants and is dependent on growth hormone and prolactin signaling (36). Complementing these physiological observations, evidence from recent human data suggests that sleep disruption or deprivation can shift innate immune composition, with reversible increases in non-classical ($CD14^{low}CD16^{+}$) monocytes, supporting the view that insufficient sleep biases leukocyte distribution toward a more inflammatory profile (37).

Sleep, especially slow-wave sleep, works in cooperation with the circadian system to ensure the formation of an adaptive immune response. Changes in anti- and pro-inflammatory mediators and hormone levels first initiate an adaptive immune response in the lymph nodes. Following the entry of antigens into the body, they are retained by antigen-presenting cells (APCs) and presented to Th cells. IL-12 produced by APCs stimulates Th1 cells and activates antigen-specific Tc cells, which initiates antibody production by B-cells (30). In the first half of the night especially, the pro-inflammatory state created by the circadian system and slow-wave sleep—driven by increased growth hormone and prolactin secretion and the suppression of cortisol—lays the groundwork for this pathway, which establishes long-term immune memory against the antigen. Growth hormone, prolactin, and IL-12 increase the Th1 immune response at the cellular level. The neurobehavioral changes of the adaptive immune system take place in the lymph nodes through coding, consolidation, and recall phases, similar to what occurs in the brain. Thus, sleep provides memory against infections in both the brain and the peripheral immune system (30). In addition, cytokines released from Th1 cells are also known to induce slow-wave sleep in a positive feedback loop (as discussed above).

Natural killer cell activity in the innate immune system begins to increase in the first hours of sleep and reaches a peak in the second half of the night. Neutrophil and monocyte cell levels also increase with sleep duration (38). IL-5 and IL-6 levels also peak during sleep, especially during the REM sleep phase. In the event of a mismatch between daylight hours and the endogenous circadian clock, or in any situation that disrupts the healthy sleep architecture, an initial increase in cortisol levels occurs via HPA axis activation. Consequently, contrary to what occurs during healthy sleep, increased production of both pro- and anti-inflammatory cytokines is observed in the peripheral blood and the CNS, thereby initiating an inflammatory immune response.

Sleep Diseases and Inflammation

Disruption of sleep structure and shortening of total sleep time cause inflammation and inflammation-related disorders such as cardiovascular diseases, arthritis, diabetes mellitus, and some cancers (33,39,40). Sleep deprivation causes spontaneous activation of the cellular innate immune system and the release of inflammatory cytokines (e.g., IL-6 and TNF-α) from monocytes as a result of the aforementioned effects on the HPA axis. It also activates proteins involved in signaling and transcription, creating an inflammatory microenvironment that poses an increased risk for disease development. These effects may be reversible following correction of sleep deprivation (33,34).

Altered cytokine gene expressions were also reported in experimental models featuring short-term sleep fragmentation (41). A significant increase in *IL-1β* gene expression levels in adipose tissue, myocardium, and specific brain regions (the hypothalamus and hippocampus) has been observed, as well as an increase in *TGF-β1* gene expression levels in the same brain regions. Studies in humans have further demonstrated that the regulation of immune-associated signaling pathways is impaired after partial sleep restriction. Changes in sleep structure due to altered cytokine levels, especially the decrease in NREM sleep duration, may also explain subsequent daytime fatigue and excessive sleepiness (42,43). A significant proportion of fatigue complaints, which are highly prevalent in multiple sclerosis (an immune-mediated neurodegenerative disorder), are associated with these negative inflammatory effects on the brain and sleep cycles (44,45). The persistent nature of some of these effects seems to be important in triggering pathological processes such as inflammation-related cardiovascular disease. Gastrointestinal conditions such as metabolic syndrome or inflammatory bowel diseases can also manifest or exacerbate due to the inflammatory processes associated with sleep disorders (46,47). In addition to increased inflammation, sleep restriction has also been shown to play an important role in the development of cancer (48). Ultimately, considering the regulatory role of sleep on the immune system, it is evident that sleep deprivation poses a significant risk for the development of numerous diseases by triggering inflammatory cascades. Acute sleep deprivation leads to an increase in pro-inflammatory cytokines and enlargement of the perivascular spaces, which also indicates glymphatic dysfunction (49). Prolongation of this process and chronic glymphatic system involvement is known to trigger the long-term development of neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease (50).

The coronavirus disease 2019 (COVID-19) pandemic significantly highlighted the intersection of sleep medicine and infectious disease. While the pandemic itself triggered a surge in insomnia—largely driven by heightened levels of anxiety and depression—evidence consistently demonstrated that pre-existing sleep disturbances were associated with higher susceptibility to infection and more severe clinical courses. Specifically, patients with short sleep duration and insomnia exhibited higher rates of hospitalization and poorer prognoses (51,52). Furthermore, sleep deprivation was linked to diminished antibody responses and a higher incidence of fatal acute respiratory failure in COVID-19 cases (53). In this context, it has been reaffirmed that a healthy sleep is one of the most basic requirements for having a properly functioning immune system. Recognizing the highly integrated, bidirectional relationship between sleep and the immune system sheds light on the pathophysiology of various diseases. Furthermore, it highlights the clinical necessity of routinely assessing sleep health and implementing corrective interventions in both the prevention and treatment of disease.

Inflammatory Diseases and Sleep

In addition to the higher risk of developing immune and chronic inflammatory diseases in those with sleep disorders, the reverse is also true: sleep-related disorders are more common among patients with immune-mediated inflammatory diseases (54). A recent review comparing disease groups such as inflammatory bowel disease, systemic lupus erythematosus, rheumatoid arthritis, psoriasis, and fibromyalgia with control groups revealed higher rates of disturbed nocturnal sleep, insomnia, restless legs syndrome, and sleep apnea in patients with inflammatory diseases. Given the complex etiopathogenesis of sleep disorders, it can be said that although the magnitude of risk varies across specific disease groups, the overall prevalence of sleep disorders remains significantly elevated in these populations.

Beyond the primary inflammatory drivers, specific clinical manifestations of immune-mediated diseases further complicate the sleep architecture through multifactorial pathways. Insomnia is frequently exacerbated not only by the systemic inflammatory load but also by comorbid chronic pain and depressive symptoms prevalent in these patient populations. Furthermore, anatomical and structural changes directly influence sleep-disordered breathing. For instance, rheumatoid arthritis may predispose patients to obstructive sleep apnea through temporomandibular and cricoarytenoid joint involvement (55). Similarly, the systemic nature of inflammatory bowel diseases, often accompanied by concomitant obesity or iron deficiency, can independently trigger sleep apnea or restless legs syndrome, respectively (56). Consequently, these disease-specific findings create a complex clinical picture where inflammatory activity and physical symptoms synergistically degrade sleep quality.

Conclusion

In conclusion, the relationship between immune-mediated inflammatory diseases and sleep disorders is characterized by a sophisticated, bidirectional interplay where dysregulated sleep architecture and chronic inflammation reinforce one another. Beyond the higher prevalence of sleep disturbances in these populations, the evidence suggests that poor sleep quality acts as a potent driver of disease progression, potentially worsening clinical outcomes and diminishing treatment efficacy. Recognizing sleep not merely as a quality-of-life issue but as a core physiological component of immune homeostasis is essential for modern clinical practice. Consequently, integrating sleep health assessments into the management protocols of inflammatory diseases—and targeting these shared neurobiological pathways—represents a critical frontier for improving both patient prognosis and the overall burden of chronic immune-mediated conditions.

Footnotes

Authorship Contributions

Concept: U.T., G.B.Ş., Design: U.T., G.B.Ş., Data Collection or Processing: U.T., G.B.Ş., Analysis or Interpretation: U.T., G.B.Ş., Literature Search: U.T., G.B.Ş., Writing: U.T., G.B.Ş.

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Prevalence and Patterns of Sleep Disturbances Among Doctors in a Tertiary-Care Institution: A Cross-sectional Survey

Üçüncü Basamak Bir Sağlık Kurumunda Hekimler Arasındaki Uyku Bozukluklarının Prevalansı ve Örüntüleri: Kesitsel Bir Araştırma

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Abstract

Objective: Sleep disturbances are common among healthcare professionals and have profound implications for their health and performance. However, data capturing a wide range of sleep disorders among medical personnel in India are limited. The lack of routine screening and institutional emphasis on sleep hygiene adds to the risk.

Materials and Methods: This cross-sectional study was conducted between October 2023 and January 2024 at PSG Institute of Medical Sciences and Research, Coimbatore. Doctors of all designations were surveyed using a 26-item self-report questionnaire based on the International Classification of Sleep Disorders, 3rd edition. Collected responses were statistically analyzed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY). Face-to-face clinical evaluation or polysomnography was not performed.

Results: A total of 252 doctors participated (mean age 30.4 ± 6.1 years; 56% female). The most prevalent sleep complaints were insufficient sleep (93.7%), insomnia (88.6%), and circadian rhythm disturbances (88.6%). Females had significantly more symptoms of parasomnias and insomnia, while males more frequently reported symptoms suggestive of sleep-disordered breathing (snoring, witnessed apnea, dry mouth on waking). Younger professionals (<30 years) had a greater burden of symptoms. Comorbidities included hypothyroidism (13%), diabetes (10%), and hypertension (7%).

Conclusion: Although the lack of polysomnographic confirmation limits diagnostic certainty, sleep disturbances appear to be highly prevalent among doctors, especially early-career professionals. Institutional-level reforms in shift scheduling, screening, and sleep-health education are urgently warranted to preserve workforce health and patient safety.

Keywords: Sleep disorders, insomnia, medical workforce, occupational health, India

Öz

Amaç: Uyku bozuklukları sağlık çalışanları arasında yaygın olup sağlıkları ve mesleki performansları üzerinde önemli etkilere sahiptir. Bununla birlikte, Hindistan'daki sağlık personelinde geniş bir uyku bozukluğu yelpazesini değerlendiren veriler sınırlıdır. Rutin taramaların yapılmaması ve kurumlarda uyku hijyenine yeterince önem verilmemesi riski daha da artırmaktadır.

Gereç ve Yöntem: Bu kesitsel çalışma, Ekim 2023 ile Ocak 2024 tarihleri arasında Coimbatore'daki PSG Tıp Bilimleri ve Araştırma Enstitüsü'nde gerçekleştirilmiştir. Tüm unvanlardaki hekimlere, Uyku Bozukluklarının Uluslararası Sınıflandırması, 3. baskıya dayalı 26 maddelik bir öz bildirim anketi uygulanmıştır. Elde edilen yanıtlar, IBM SPSS Statistics for Windows, sürüm 26.0 (IBM Corp., Armonk, NY) kullanılarak istatistiksel olarak analiz edilmiştir. Yüz yüze klinik değerlendirme veya polisomnografi uygulanmamıştır.

Bulgular: Çalışmaya toplam 252 hekim katılmıştır (ortalama yaş: 30,4 ± 6,1 yıl; %56'sı kadın). En yaygın uyku yakınmaları yetersiz uyku (%93,7), insomnia (%88,6) ve sirkadiyen ritim bozuklukları (%88,6) olarak belirlenmiştir. Kadınlarda parasomni ve insomnia belirtileri anlamlı düzeyde daha fazla görülürken, erkeklerde uykuda solunum bozukluğunu düşündüren horlama, tanıklı apne ve sabah ağız kuruluğu gibi belirtiler daha sık bildirilmiştir. Genç hekimlerde (<30 yaş) belirti yükünün daha yüksek olduğu saptanmıştır. Eşlik eden hastalıklar arasında hipotiroidi (%13), diyabet (%10) ve hipertansiyon (%7) yer almıştır.

Sonuç: Polisomnografik doğrulamanın bulunmaması tanısal kesinliği sınırlandırmakla birlikte, uyku bozukluklarının özellikle meslek yaşamının ilk dönemlerindeki hekimler arasında oldukça yaygın olduğu görülmektedir. Sağlık çalışanlarının sağlığını ve hasta güvenliğini korumak amacıyla vardiya düzenlemeleri, tarama programları ve uyku sağlığı eğitimleri konusunda kurumsal düzeyde reformların acilen hayata geçirilmesi gerekmektedir.

Anahtar Kelimeler: Uyku bozuklukları, insomnia, sağlık çalışanları, iş sağlığı, Hindistan

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Introduction

Sleep is a fundamental biological process essential for human health and well-being (1). It is particularly crucial for cognitive performance, emotional regulation, and physiological restoration. In high-stakes professions such as medicine, adequate sleep becomes even more critical, as sleep deprivation can impair clinical judgment, compromise patient safety, and affect the overall quality of care (2). Healthcare professionals often operate in high-pressure environments with irregular schedules, night shifts, and demanding workloads, which collectively disrupt circadian rhythms and negatively impact sleep quality (3). Studies globally have shown that doctors, nurses, and allied healthcare workers are disproportionately affected by sleep disturbances, with long-term consequences to both their physical and mental health (4). Despite these concerns, sleep health among Indian healthcare professionals has not received adequate attention in research (5). Most Indian studies have focused on medical interns or emergency staff, leaving a considerable gap in understanding how sleep disorders affect different tiers of the medical hierarchy across departments.

The current study addresses this gap by providing a comprehensive, symptom-based analysis of sleep disturbances among doctors in a major tertiary-care teaching hospital in India, using the International Classification of Sleep Disorders, 3rd edition (ICSD-3) (6). By examining multiple sleep domains and correlating them with demographic variables, we sought to identify high-risk groups and propose targeted interventions. A secondary aim was to highlight occupational and systemic contributors to poor sleep, which are currently under-recognized.

Materials and Methods

This cross-sectional observational study was conducted between October 10, 2023 and January 31, 2024 at PSG Institute of Medical Sciences and Research, a 1400-bed tertiary-care teaching hospital in Coimbatore, Tamil Nadu, India. The study population comprised all categories of medical professionals at the institution, including compulsory rotating residential interns (CRRIs), junior residents, and faculty members (senior residents, assistant professors, associate professors, professors, and consultants) across both clinical and preclinical departments. Data were collected through an anonymous online survey administered via Google Forms. Participation was voluntary, and informed consent was obtained digitally prior to commencement of the survey.

The questionnaire consisted of 26 items adapted from the ICSD-3, encompassing 7 major domains of sleep dysfunction: insufficient sleep, insomnia, circadian rhythm disorders, parasomnias/rapid eye movement (REM)-related disturbances, sleep-disordered breathing (SDB), movement disorders, and excessive daytime sleepiness (EDS). Each item was scored using a five-point Likert scale, ranging from "Never" to" ≥3 times per week."

Demographic variables included age, sex, professional designation, departmental affiliation, and presence of self-reported chronic comorbidities. Variables such as work hours, shift schedule, screen use before bed, family stress, smoking/alcohol use, or physical activity were not included and represent an important limitation in interpreting the results. Similarly, parameters such as usual bedtime, wake time, and total sleep duration were not assessed.

The study received ethical clearance from the PSG Institute of Medical Sciences and Research Institutional Ethics Committee (IEC no: 2023/457, date: 06.10.2023) and adhered to guidelines set forth by the Indian Council of Medical Research.

Statistical Analysis

Survey responses were exported to IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY) for analysis. Descriptive statistics were used to summarize the prevalence of sleep-related symptoms. Associations between demographic variables and symptom categories were evaluated using chi-square (χ^2) tests. A p-value of <0.05 was considered indicative of statistical significance.

Results

A total of 252 completed responses were included in the final analysis, representing a 90% response rate (Figure 1). The mean age was 30.4±6.1 years, with a median age of 29. Females constituted 56% of the sample. CRRIs were the largest group (43.7%), followed by junior residents (15.1%), faculty members (20.6%), and consultants (18.3%).

Overall, 93.7% of respondents reported symptoms of insufficient sleep, and 88.6% reported symptoms consistent with insomnia. Circadian rhythm disturbances were reported by 88.6% of doctors, followed by parasomnias (70.9%), symptoms suggestive of SDB (62.2%), movement disorders (34.5%), and EDS (27.8%). A striking observation was that nearly 77% of respondents reported disturbances across at least three domains.

Statistically significant sex-based differences were observed in the prevalence of insufficient sleep (females 98.6% vs. males 89.3%, p = 0.003) and parasomnias (females 79.3% vs. males 61.6%, p = 0.003). Females also reported more insomnia, although the difference was not significant (91.4% vs. 85.5%, p = 0.16). Conversely, male doctors had a higher prevalence

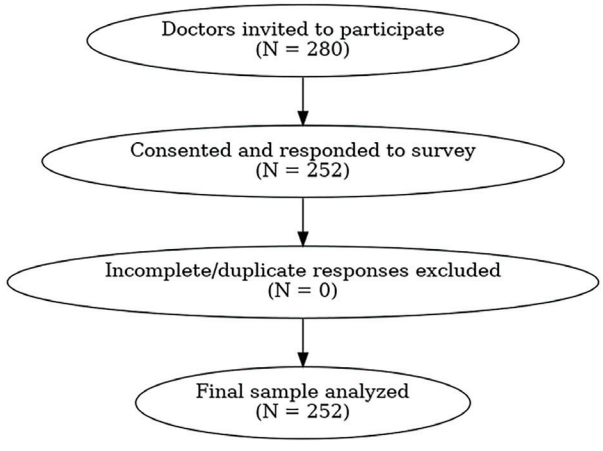

Figure 1. Participant flowchart.

of SDB symptoms (75.0% vs. 52.9%, p = 0.001), including snoring and dry mouth on waking.

Age-based trends revealed that younger doctors, particularly those below 30 years of age, were most affected, with a symptom prevalence as high as 96.5% for insufficient sleep. Those aged ≥50 years had significantly lower prevalence across most domains. Additionally, interns and junior residents were disproportionately affected, reporting the highest burden of symptoms.

Comorbid conditions such as diabetes, hypothyroidism, and hypertension were present in 26.5% of participants and showed significant association with insomnia and SDB symptoms.

Tables 1 and 2 present the demographic breakdown and domain-based prevalence of reported symptoms, respectively. Figures 2 through 5 visually summarize symptom frequencies, sex-wise differences, comorbidities, and designation-based trends.

Table 1. Demographic characteristics of participants (n = 252).

Variable	Value
Age (years), mean ± SD (median)	30.4 ± 6.1 (29)
Age range (years), n (%)	
<30	134 (53.2)
30–39	66 (26.2)
40–49	34 (13.5)
≥50	18 (7.1)
Sex, n (%)	
Male	111 (44.0)
Female	141 (56.0)

SD: Standard deviation.

Table 2. Prevalence of sleep symptom domains.

Sleep domain	n (%)
Insufficient sleep	236 (93.7)
Insomnia	223 (88.6)
Circadian rhythm disturbances	223 (88.6)
Parasomnias/REM-related symptoms	178 (70.9)
Sleep-disordered breathing	157 (62.2)
Movement disorders	87 (34.5)
Excessive daytime sleepiness	70 (27.8)

REM: Rapid eye movement.

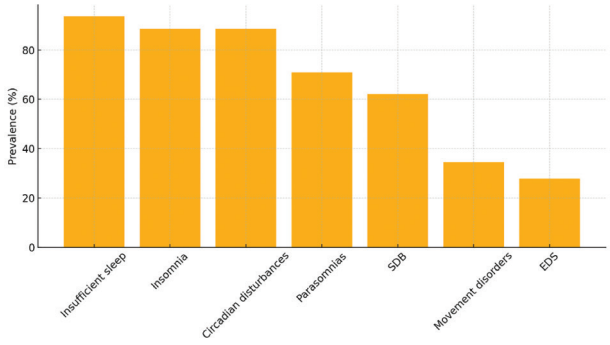

Figure 2. Overall prevalence of sleep disturbance domains.
SDB: Sleep disordered breathing, EDS: Excessive daytime sleepiness.

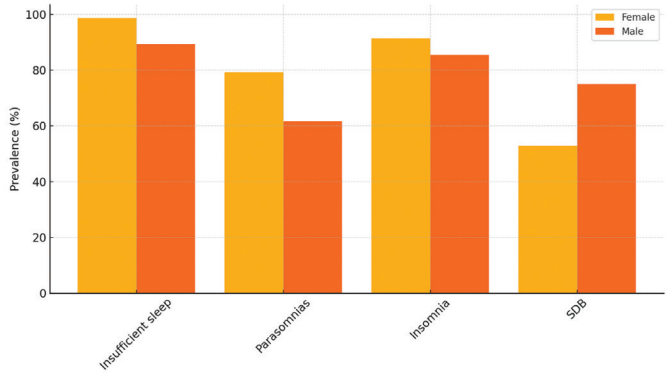

Figure 3. Sex-wise differences in sleep symptom prevalence.
SDB: Sleep-disordered breathing.

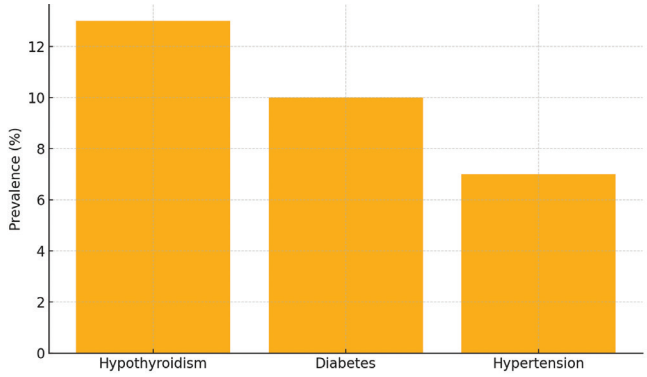

Figure 4. Frequency of reported comorbidities.

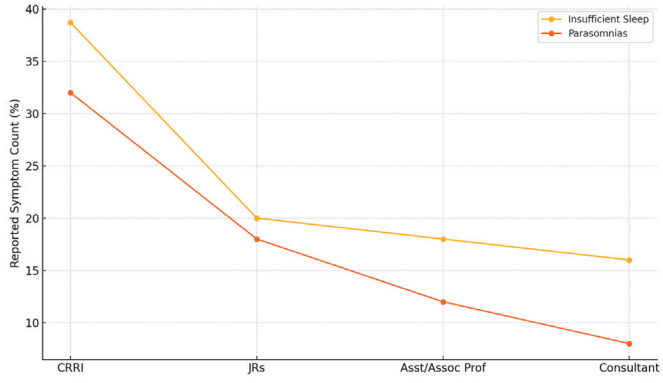

Figure 5. Symptom distribution by professional designation.
CRRI: Compulsory rotating residential interns, JRs: Junior residents.

Discussion

This study reveals a strikingly high prevalence of sleep disturbances among doctors in a large tertiary-care academic hospital in India. The most frequently reported symptoms were insufficient sleep and insomnia, each affecting over 88% of respondents. These findings are consistent with global literature indicating that healthcare workers are disproportionately

affected by sleep-related complaints due to extended shifts, erratic working hours, and high occupational stress (3-5).

Our data show that younger doctors, particularly those under the age of 30, report the highest burden of symptoms. This is likely due to inflexible and demanding work schedules, which often include prolonged overnight duties and academic obligations. Similar age-related findings have been reported in other studies, underscoring the need for policy interventions early in medical careers (4).

Sex-wise differences were notable. Female doctors had a higher prevalence of insufficient sleep, insomnia, and parasomnias. This finding may reflect both biological (e.g., hormonal) and psychosocial (e.g., dual responsibilities of work and home) contributors to sleep fragmentation and poor sleep quality (7,8).

In contrast, male doctors exhibited greater prevalence of symptoms suggestive of SDB, consistent with known anatomical and behavioral risk factors (9). However, it should be noted that these symptoms were not confirmed with polysomnography.

Another important observation was the high burden of symptoms across multiple domains. More than three-quarters of respondents reported disturbances in at least three symptom categories. This clustering increases the likelihood of cumulative negative effects on cognitive functioning, psychomotor performance, and emotional regulation—all of which are vital for medical decision-making and patient safety (4,10).

Comorbid conditions such as hypothyroidism, diabetes mellitus, and hypertension were common among the study population. These conditions are both influenced by and contributors to sleep dysfunction, suggesting a bidirectional relationship that merits integrated management strategies (4,11).

Compared to Western data, the prevalence of parasomnias and circadian misalignment was relatively high. In a Turkish sample during the coronavirus disease-2019 pandemic, poor sleep quality was reported in 55% of physicians, comparable to our findings, and was significantly associated with female sex, frontline worker status, low social support, and heightened stress levels (12). This could reflect cultural and systemic differences in healthcare delivery, including more frequent night duties, higher patient loads, and limited institutional emphasis on sleep hygiene in the Indian context (3,9).

Study Limitations

This study has several limitations. It was conducted in a single tertiary-care center, which may limit the generalizability of the findings. Data were self-reported and may be affected by recall bias. As a cross-sectional study, it cannot establish causality. The questionnaire, though based on ICSD-3 criteria (6), is not a validated diagnostic tool. Polysomnography and clinical examination were not performed. Other sleep-related factors such as bedtime, sleep latency, nocturnal awakenings, and screen time before bed were also not assessed, and the participants' work schedules and shift timings were not recorded. Moreover, although lifestyle-related factors (e.g., smoking, alcohol, caffeine use, mobile device usage) can strongly influence sleep, these were not assessed. Future studies should consider including these important determinants.

Conclusions

This study underscores the widespread and multidimensional burden of sleep disturbances among doctors, with younger age, female sex, and early-career professional status emerging as significant risk factors. The most prevalent disturbances identified were insufficient sleep, insomnia, and parasomnias/REM-related symptoms. These conditions frequently co-occurred and were often associated with underlying metabolic and endocrine comorbidities, highlighting the complex interplay between occupational stressors and sleep health.

Given the high prevalence and clinical relevance of these disturbances, there is an urgent need for institutional reforms to address this silent epidemic. Key recommendations include:

- Implementing duty-hour regulations to prevent chronic sleep deprivation,
- Integrating sleep health education into routine medical training and wellness programs,
- Periodic screening for sleep-related disorders among healthcare personnel, and
- Establishing dedicated sleep clinics to facilitate early diagnosis and intervention.

Recognizing sleep health as a fundamental component of clinician well-being is essential for sustaining a safe, efficient, and compassionate healthcare system. By prioritizing the sleep health of medical professionals, institutions can not only enhance clinician resilience and reduce burnout but also improve the quality of patient care through better attention, empathy, and clinical decision-making.

Ethics

Ethics Committee Approval: The study received ethical clearance from the PSG Institute of Medical Sciences and Research Institutional Ethics Committee (IEC no: 2023/457, date: 06.10.2023) and adhered to guidelines set forth by the Indian Council of Medical Research.

Informed Consent: Participation was voluntary, and informed consent was obtained digitally prior to commencement of the survey.

Footnotes

Authorship Contributions

Surgical and Medical Practice: J.R., T.G., R.K., G.S., Concept: J.R., R.K., Design: J.R., R.K., Data Collection or Processing: J.R., T.G., G.S., Analysis or Interpretation: J.R., T.G., R.K., G.S., Literature Search: J.R., T.G., G.S., Writing: J.R., T.G., R.K., G.S.

Conflict of Interest: No conflict of interest was declared by the authors.

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Effect of Cluster versus Traditional Set Resistance Exercises on Sleep Architecture and Oxygen Saturation

Küme Set ve Geleneksel Set Direnç Egzersizlerinin Uyku Mimarisi ve Oksijen Satürasyonu Üzerine Etkisi

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Abstract

Objective: The aim of this study was to compare the effect of cluster set (CLS) and traditional set (TS) resistance exercises on sleep architecture and nocturnal oxygen saturation (SpO_2) in healthy recreationally active males.

Materials and Methods: Fourteen males (22.41 ± 2.38 years; 177.79 ± 6.55 cm; 77.15 ± 12.65 kg) completed CLS or TS resistance training in a randomized crossover design (72 ± 24-hour washout). Exercises were performed at 85% intensity. TS was applied with 4 x 6 repetitions and 3-minute rest between sets and exercises; CLS was performed as 4 x (3 x 2) repetitions with 30–second intraset and 3-minute rest interset and exercise. Blood lactate was assessed pre- and post-exercise, anxiety was evaluated pre-sleep, and polysomnography was conducted post-exercise (11:00 p.m.–7:00 a.m.).

Results: Repeated-measures ANOVA and paired samples t-tests indicated that sleep architecture parameters and SpO_2 after TS or CLS resistance exercise did not differ significantly from the control sleep session ($p > 0.05$).

Conclusion: In this study, neither set configuration significantly affected sleep architecture parameters or nocturnal SpO_2 compared to control sleep.

Keywords: Polysomnography, REM sleep, NREM sleep, strength training, set structure

Öz

Amaç: Bu çalışmanın amacı, sağlıklı rekreasyonel aktif erkeklerde küme set (CLS) ve geleneksel set (TS) direnç egzersizlerinin; uyku mimarisi ve uyku sırasındaki oksijen satürasyonu (SpO_2) üzerine olan etkilerini karşılaştırmaktır.

Gereç ve Yöntem: On dört erkek (22,41 ± 2,38 yaş; 177,79 ± 6,55 cm; 77,15 ± 12,65 kg) rastgeleleştirilmiş çapraz geçişli bir tasarımda (72 ± 24 saat aralıkla) CLS veya TS direnç antrenmanlarını tamamladı. Egzersizler %85 şiddette uygulandı. TS 4 x 6 tekrar, set arası ve hareketler arası 3 dakika dinlenme ile; CLS 4 x (3 x 2) tekrar, set içi 30 saniye, setler ve hareketler arası 3 dakika dinlenme ile gerçekleştirildi. Kan laktadı egzersiz öncesi ve sonrasında değerlendirildi. Uyku öncesi anksiyete değerlendirildi; polisomnografi egzersiz sonrasında (23:00–07:00) uygulandı.

Bulgular: Tekrarlı ölçümlerde ANOVA ve eşleştirilmiş örneklemler t-testleri, TS veya CLS direnç egzersizleri sonrasında uyku mimarisi parametreleri ve SpO_2'nin kontrol uykusuna kıyasla anlamlı farklılık göstermediğini ortaya koydu ($p > 0,05$).

Sonuç: Çalışmanın sonuçlarına göre, hiçbir set konfigürasyonu uyku mimarisi parametrelerini veya SpO_2'yi kontrol uykusu değerlendirmesinden farklı etkilememiştir.

Anahtar Kelimeler: Polisomnografi, REM uykusu, NREM uykusu, kuvvet antrenmanı, set yapısı

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Introduction

Despite evidence showing the effects of training and competition on athletes' sleep (1), the magnitude and underlying mechanisms of these effects have not yet been fully elucidated (2). Although the neural mechanisms involved in sleep (3) and central or peripheral nervous system fatigue (4) may appear different, exercise protocols are known to impact sleep (5). Aerobic exercise is recognized as a non-pharmacological method to increase both the amount and quality of sleep (6). In addition to aerobic exercise, reports indicate that progressive resistance exercises can also improve sleep quality (7). However, resistance exercises have numerous training variables (exercise type, set configuration, load lifted, training volume, rest between sets, and repetition speed), with manipulation of these variables producing different results. Therefore, it is challenging yet important to determine the effects of resistance exercises on human health.

Kovacevic et al. (7) reported that chronic resistance exercise improves various aspects of sleep, particularly sleep quality. Previous studies examining the effects of acute and chronic resistance exercise on sleep architecture and quality have typically utilized traditional set (TS) protocols (8,9). The TS method causes a temporary decrease in muscle power and movement velocity due to phosphocreatine depletion and increased blood lactate [$(La^-)_b$] concentrations (10). High ammonia concentrations and decreased neural activation can also lead to muscle fatigue (11,12). Therefore, it has been suggested that TS configurations may not be the most appropriate choice in clinical applications (13).

In contrast to these disadvantages of the TS method of resistance training, cluster set (CLS) configurations have been associated with lower [$(La^-)_b$] concentrations and increased repetition velocity compared to TS in both acute and long-term training (14,15). Given that sleep and exercise interact through complex physiological and psychological pathways (5), the effects of fatigue induced by TS and CLS exercise on sleep architecture and oxygen saturation (SpO_2) during sleep are still not fully understood. Therefore, this study aimed to examine the acute effects of TS and CLS resistance training on sleep architecture and nocturnal SpO_2. We hypothesized that CLS would lead to improved sleep architecture and nocturnal SpO_2 compared to TS.

Materials and Methods

Experimental Design

This study employed a randomized, controlled, crossover experimental design with repeated-measures. Participants underwent three conditions-control (no exercise), TS exercise, and CLS exercise-each separated by a 72 ± 24-hour washout period. Condition order was randomized using a computer program to mitigate the "first-night effect" associated with polysomnography (PSG) and potential carry-over effects between interventions.

During the initial screening session, participants' anthropometric data, sleep quality, anxiety level, and chronotype (natural circadian rhythm) were assessed. In the second session, a 10-repetition maximum (10-RM) test was conducted for the six resistance exercises. In subsequent sessions, participants followed one of three assigned conditions. In the control condition, resting [$(La^-)_b$] concentration was measured, followed by overnight PSG evaluation. In the TS and CLS conditions, [$(La^-)_b$] concentration was measured pre- and post-exercise, and the resistance training protocol was followed by overnight PSG evaluation.

Participants

A G*Power F-test analysis was performed to determine the number of volunteers to enroll in the study. With effect size (f) = 0.36, type I error rate (α) = 0.05, and power (1-β) = 0.80 for a single-group, repeated-measures design with 3 repetitions, the required sample size was 14. Accordingly, 14 recreationally active men (age; 22.41 ± 2.38 years; height; 177.79 ± 6.55 cm; weight; 77.15 ± 12.65 kg) were recruited to participate in this study. The participants were involved in sports such as swimming, football, basketball, tennis, athletics, and fitness.

Inclusion criteria were as follows: (a) male sex, aged 18 to 30 years; (b) recreational sports participation at least 3 days per week for at least 3 months; (c) no physical or psychological illness; (d) no tobacco, alcohol, medication, or caffeine use; (e) no diagnosed sleep disorder; and (f) intermediate chronotype.

Ethics committee approval for the study was obtained from the Medical Research Ethics Committee of the Ege University Faculty of Medicine (decision number: 21-3.1T/60, date: 18.03.2021). All participants were informed about the purpose of the study and signed a voluntary consent form prior to participation, in accordance with the Declaration of Helsinki.

Data Collection Tools

Anthropometric Measurements

Height was measured barefoot using a Seca stadiometer. Body weight, body mass index, and body fat percentage were assessed using a Tanita BC-418 body composition analyzer, at the same time of day to ensure consistency.

Chronotype Assessment

Chronotype was assessed using a culturally adapted and validated version of the Horne-Ostberg questionnaire (16). Only participants with intermediate types were included in the study.

Sleep Quality Assessment

A culturally adapted and validated version of the Pittsburgh Sleep Quality Index was used to evaluate the participants' sleep quality prior to the training sessions (17).

Anxiety Assessment

A validated, culturally adapted version of the State-Trait Anxiety Inventory (Form X) was administered before each PSG evaluation to assess anxiety levels (18).

Calorie Determination

Daily caloric requirements were estimated using the Harris-Benedict equation (basal metabolic rate x 1.55 activity factor) (19). The timing and content of pre-sleep meals were standardized across all conditions.

Ten-Repetition Maximal Strength

Individual training loads were determined using a 10-RM protocol for each prescribed exercise (20).

Blood Lactate Measurement

Resting $[(La^-)_b]$ was measured after 10 minutes of passive rest upon arriving at the laboratory using a YSI 1500 Lactate Analyzer (Yellow Springs Instruments, Ohio, USA). For the TS and CLS conditions, a second measurement was performed 5 minutes post-exercise.

Polysomnographic Sleep Analysis

PSG recordings were carried out in the Sleep Disorders Monitoring Center in the Neurology Department of İzmir Bozyaka Training and Research Hospital. The sleep room was a controlled environment with a noise level of 27 dB (decibels), temperature of 21 °C, and 0 lx (lux) illumination. Recordings commenced at 11:00 p.m. and concluded at 7:00 a.m.

Sleep data were acquired and analyzed using an EMBLA N7000 PSG system and RemLogic software (version 3.4.1). Neuro-ocular recordings were obtained using three-channel (F4-M1, C4-M1, O2-M1) electroencephalography in the frontal, central, and occipital regions of the head and two-channel electrooculography near the right and left eyes. Myographic data were obtained by surface electromyography of the chin and bilateral tibialis anterior muscles. Respiratory and position data were obtained via cannula and thermistor for oronasal airflow to the nose; respiratory belts in the relevant areas for thoracic and abdominal respiratory movements; a body position control belt; finger pulse oximeter for SpO_2; and respiratory sound and synchronous video recording.

Sleep datasets were staged in 30-second epochs and scored according to the 2016 American Academy of Sleep Medicine (AASM) Manual for the Scoring of Sleep and Associated Events (21).

Strength Training Program

The resistance training protocol (85% intensity) included six exercises: Smith machine front squat, bench press, lat pull-down, cable face pull, barbell preacher curl, and triceps pushdown. The TS condition comprised 4 x 6 reps with 3-min rest interset and exercise. The CLS configuration involved 4 sets of 3 x 2 reps with 30-s intraset and 3-min rest interset and exercise.

Participant Schedule

Participants continued their routine daily activities (without exercise) and maintained a regular sleep schedule (11:00 p.m. to 7:00 a.m.) on non-testing days and during washout periods (Figure 1A). On sleep measurement days, participants rose at 7:00 a.m. and reported to the laboratory in a rested state. After baseline $[(La^-)_b]$ measurements, participants trained from 4:00 p.m. to 6:00 p.m., and the final meal was consumed between 6:00 p.m. and 7:00 pm. Between 7:00 p.m. and 11:00 p.m., the participants rested, traveled to the clinic, and underwent preparations for PSG evaluation, which was conducted between 11:00 p.m. and 7:00 a.m. (Figure 1B).

Statistical Analysis

Results were evaluated using SPSS 25.0 (IBM Corp., Armonk, NY). According to the Shapiro-Wilk test, 15 parameters were found to be normally distributed (TRT, TST, N1, N2, N3, R, RL, SE, mean SpO_2 during non-rapid eye movement (NREM), rapid-eye movement (REM), wakefulness, state anxiety, pre-exercise $[(La^-)_b]$, post-exercise $[(La^-)_b]$ and $\Delta[(La^-)_b]$) and 9 parameters were non-normally distributed (SOL, N1L, N2L, N3L, W, WASO, AHI, PLMS, and trait anxiety).

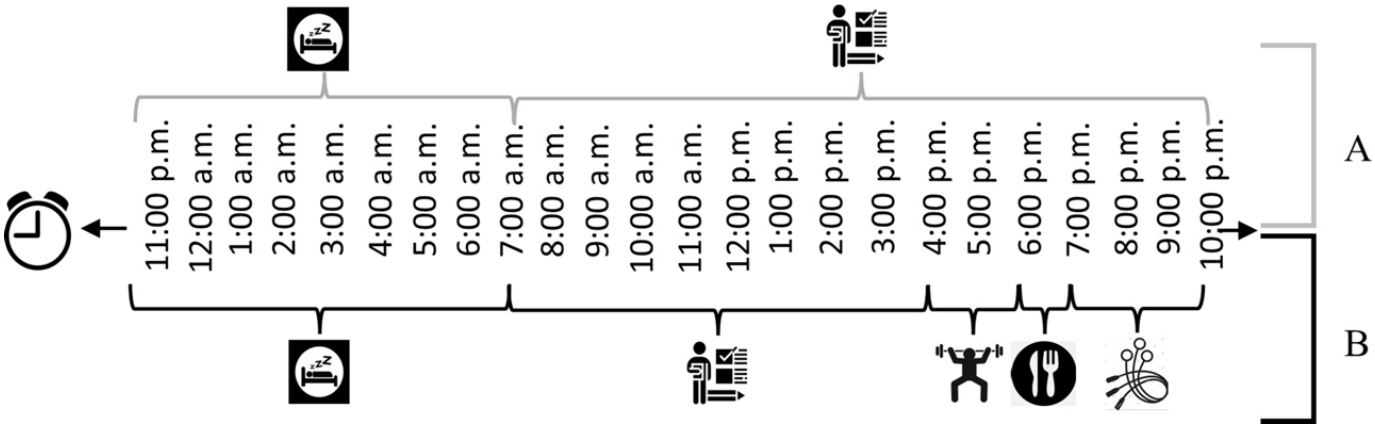

Figure 1. A) Participants' daily schedule before and between sleep measurement. B) Daily schedule on the day of sleep measurement.

Parametric data were analyzed using repeated-measures ANOVA. Partial eta-squared (η_p^2) was calculated as the effect size and was categorized as small (0.01–0.06), medium (0.06–0.14), and large (>0.14) (22). Pairwise comparisons were performed with Bonferroni correction, and Cohen's d was determined as the effect size, categorized as small (0.2–0.5), medium (0.5–0.8), and large (>0.8) (22). Paired samples t-test and Cohen's d effect size were used for pairwise comparisons of post-exercise $[(La^-)_b]$ and $\Delta[(La^-)_b]$.

Repeated measures analysis of non-parametric data was performed using the Friedman test with Kendall's W effect sizes, categorized as small (0.1–0.3), medium (0.3–0.6), large (0.6–1.0), and very large (>1.0). For pairwise comparisons of non-parametric data, the Conover test was used and effect sizes were presented with Cohen's d. Statistical significance was set at $p < 0.05$.

Results

Descriptive statistics of the participants are given in Table 1.

Based on the repeated-measures ANOVA test and Friedman test, a significant difference was observed only in REM latency (RL) ($F_{2,26}=3.812$, $p=0.035$, $\eta_p^2=0.227$). No statistically significant difference was found in repeated measures for any other parameter ($p>0.05$) (Table 2). Paired samples t-test revealed a significant difference in post-exercise $[(La^-)_b]$ ($t_{13}=5.766$, $p\leq0.001$) and $\Delta[(La^-)_b]$ ($t_{13}=5.375$, $p\leq0.001$) (Table 2). No other significant differences were identified in pairwise comparisons between interventions ($p > 0.05$) (Table 2).

Discussion

This study aimed to examine the effects of strength training with CLS and TS configurations on sleep architecture and changes in SpO_2 during sleep. Contrary to our hypothesis that the CLS resistance training protocol would improve sleep architecture and nocturnal SpO_2 values compared to the TS protocol, our findings did not support this prediction. The main results indicated that the CLS and TS interventions yielded no statistically significant differences in sleep architecture parameters or SpO_2 during sleep ($p>0.05$).

Table 1. Descriptive statistics of the participants (n = 14).

	$\bar{X}\pm$ SD
Age (years)	22.41 ± 2.38
Height (cm)	177.79 ± 6.55
Weight (kg)	77.15 ± 12.65
Body mass index (kg/m²)	24.36 ± 3.21
Body fat ratio (%)	12.27 ± 4.85
Pittsburgh Sleep Quality Inventory score	3.07 ± 1.38
Horne–Östberg chronotype score	51.29 ± 6.01
Daily calorie requirement (kcal.)	2886.54 ± 310.11

$\bar{X}$ ± SD: Mean ± standard deviation

The absence of a statistical difference in any of the study parameters may be explained by the ceiling and floor effect hypothesis. This theory suggests that better baseline sleep equates to less potential for improvement in sleep parameters (23). As our participants were recreationally active and reported no sleep problems, there may not have been a measurable change or improvement in their sleep architecture (24,25). Acute physical exercise can increase REM latency, reduce REM duration, and slightly increase slow-wave sleep (25). It has also been reported that the positive effects of acute exercise on sleep are observable after exercise sessions lasting at least one hour (24). Nevertheless, acute physical exercise is reported to have a small to moderate impact on sleep (26). This suggests that although the exercise protocol in our study lasted longer than one hour, the time spent under load in the single sessions was not sufficient to alter sleep parameters in these participants.

Although numerous studies have examined the relationship between exercise and sleep, comparing their results is challenging because of differences in sample groups (e.g., age, clinical diagnoses), sleep evaluation methods, specific parameters assessed, and interventions (8,27). In the literature, resistance training studies employing TS configurations with comparable research designs and study populations have consistently reported no statistically significant differences in any parameters related to sleep architecture ($p > 0.05$) (8,28,29).

Ramos-Campo et al. (8) evaluated 15 resistance-trained men (age; 23.4 ± 2.4) performing 4 sets of 10 repetitions or 5 sets of 8 repetitions at the same intensity (75% of 1-RM). Using actigraphy to assess sleep quality, they reported no statistically significant difference in sleep onset latency (SOL), sleep efficiency (SE), or time in bed between the intervention groups ($p > 0.05$). Although their study is the most similar to ours in terms of participant characteristics and research design, the use of actigraphy rather than PSG is a major methodological distinction (8). Nevertheless, some of their findings align with ours, particularly regarding the lack of significant impact on SOL and SE following TS exercise.

In a study by de Faria et al. (28) 27 sedentary men with no sleep problems or diagnosed disease performed TS resistance exercise in 3 sets of 15 repetitions at 50% of 1-RM at one of three times of day: morning, afternoon, or evening. The training lasted 40–45 minutes with 90-second intraset rest, and sleep was assessed by PSG on the same night after a single exercise session. The authors reported no significant difference in any sleep parameter (SOL, RL, TST, SE, N1, N2, N3, N4, SWS, R, and W) compared to the baseline measurements ($p > 0.05$). Although this study is also similar to ours in that the sample group comprised healthy young men and sleep was evaluated by PSG, the lower intensity exercise and inclusion of sedentary participants are major differences. However, the results reported by de Faria et al. (28) are similar to the findings we obtained after TS.

Table 2. Comparisons of sleep architecture and oxygen saturation parameters.

	Control $\bar{X} \pm SD$	TS $\bar{X} \pm SD$	CLS $\bar{X} \pm SD$	ANOVA p	ηp^2	Friedman p	Kendall's W	Control vs. TS			Control vs. CLS			TS vs. CLS		
								Δ	p	ES	Δ	p	ES	Δ	p	ES
TRT (min)	434.47 ± 37.93	451.87 ± 36.72	425.40 ± 49.92	0.069	0.186			17.4	0.45	0.46	9.07	0.826	0.20	26.47	0.195	0.60
TST (min)	368.91 ± 63.87	394.84 ± 58.16	378.21 ± 58.93	0.211	0.113			25.92	0.44	0.42	9.30	0.988	0.15	16.62	0.965	0.28
N1 (min)	31.77 ± 14.32	28.71 ± 13.74	27.57 ± 18.99	0.670	0.030			3.057	1.0	0.21	4.20	1.0	0.24	1.14	1.0	0.24
N2 (min)	249.94 ± 56.26	268.80 ± 50.33	257.19 ± 58.89	0.352	0.077			18.86	0.527	0.35	7.25	1.0	0.12	11.61	1.0	0.21
N3 (min)	38.78 ± 25.45	39.0 ± 23.51	38.56 ± 28.48	0.997	< 0.001			0.214	1.0	0.008	0.221	1.0	0.008	0.221	1.0	0.016
R (min)	48.41 ± 21.99	58.32 ± 24.23	52.97 ± 18.49	0.375	0.151			9.907	0.684	0.42	4.564	0.727	0.22	5.343	1.0	0.24
RL (min)	172.92 ± 90.86	126.10 ± 64.42	109.35 ± 46.43	0.035*	0.227			46.82	0.229	0.62	63.57	0.079	0.88	16.75	1.0	0.88
SE (%)	84.82 ± 12.09	87.27 ± 9.48	88.39 ± 8.09	0.325	0.083			2.457	1.0	0.22	3.571	0.194	0.34	1.114	1.0	0.12
SpO_2 during wakefulness (%)	95.62 ± 1.00	95.55 ± 1.18	95.55 ± 0.87	0.905	0.008			0.071	1.0	0.05	0.071	1.0	0.07	<0.001	1.0	0.0
SpO_2 during NREM (%)	94.63 ± 1.20	94.72 ± 1.35	94.75 ± 1.12	0.839	0.013			0.086	1.0	0.07	0.121	1.0	0.10	0.036	1.0	0.02
SpO_2 during REM (%)	95.10 ± 1.11	95.27 ± 1.30	95.42 ± 1.15	0.334	0.081			0.164	1.0	0.14	0.314	0.334	0.28	0.150	1.0	0.12
State anxiety	46.57 ± 3.18	45.50 ± 4.25	46.36 ± 3.67	0.627	0.035			1.071	1.0	0.018	0.214	1.0	0.06	0.857	1.0	0.21
Pre-exercise $[(La^-)_b]$ $(mmol \cdot L^{-1})$	1.47 ± 0.38	1.51 ± 0.32	1.46 ± 0.46	0.887	0.009			0.048	1.0	0.11	0.006	1.0	0.02	0.054	1.0	0.12
Post-exercise $[(La^-)_b]$ $(mmol \cdot L^{-1})$	—	7.14 ± 1.77	4.57 ± 1.00	—	—			—	—	—	—	—	—	2.56	<0.001*	1.78
$\Delta[(La^-)_b]$ $(mmol \cdot L^{-1})$	—	5.62 ± 1.72	3.11 ± 0.90	—	—			—	—	—	—	—	—	2.51	<0.001*	1.82
SOL (min)	10.56 ± 8.03	10.10 ± 11.80	11.63 ± 12.30			0.751	0.020	0.464	1.0	0.043	1.071	1.0	0.09	1.536	1.0	0.14
N1L (min)	10.56 ± 8.03	10.10 ± 11.80	11.63 ± 12.30			0.751	0.020	0.464	1.0	0.043	1.071	1.0	0.09	1.536	1.0	0.14
N2L (min)	16.49 ± 12.14	12.52 ± 11.85	14.58 ± 12.62			0.607	0.036	3.964	1.0	0.325	1.907	1.0	0.15	2.057	1.0	0.16
N3L (min)	45.10 ± 34.38	54.17 ± 47.43	58.33 ± 51.73			0.849	0.012	9.071	1.0	0.201	13.23	1.0	0.29	4.164	1.0	0.09
W (min)	65.55 ± 52.32	57.01 ± 42.83	49.07 ± 36.38			0.109	0.158	8.543	1.0	0.193	16.47	0.1	0.37	10.89	0.5	0.17
WASO (min)	54.97 ± 49.47	46.92 ± 33.38	37.49 ± 37.42			0.257	0.097	8.057	1.0	0.198	17.48	0.428	0.43	9.429	0.5	0.23
AHI	1.95 ± 2.35	2.30 ± 2.77	2.38 ± 2.29			0.982	0.001	0.350	1.0	0.143	0.436	1.0	0.178	0.086	1.0	0.03
PLMS/h	0.357 ± 1.19	0.036 ± 0.13	0.0001 ± 0.0001			0.368	0.071	0.321	1.0	0.461	0.357	0.237	0.512	0.036	1.0	0.05
Trait anxiety	42.50 ± 4.70	42.21 ± 4.13	41.21 ± 3.16			0.819	0.014	0.286	1.0	0.071	1.286	1.0	0.371	1.00	1.0	0.24

*p < 0.05: $\bar{X} \pm SD$: Mean ± SD, η_p^2: Partial eta-squared, Kendall's W: Friedman test effect size, Δ: Difference of means, ES: Cohen's d effect size, TS: Traditional set, CLS: Cluster set, TRT: Total record time, SOL: Sleep onset latency, SE: Sleep efficiency, N1: NREM stage 1, N2: NREM stage 2, N3: NREM stage 3, R: REM stage, W: Wakefulness during sleep period, N1L: NREM stage 1 latency, N2L: NREM stage 2 latency, N3L: NREM stage 3 latency, RL: REM stage latency, TST: Total sleep time, SpO2: Oxygen saturation, WASO: Wake after sleep onset, AHI: Apnea-hypopnea index, PLMS: Periodic limb movements in sleep, $[(La\text{-})_b]$: Blood lactate.

Another important parameter for sleep architecture and quality is the Apnea-Hypopnea Index (AHI), which is the average number of apneas and hypopneas occurring per hour of sleep (30). According to the AASM, obstructive sleep apnea (OSA) is classified according to AHI as mild (5–15), moderate (15–30), and severe (>30). The prevalence of OSA in the general population varies between 9% and 38%, with rates higher in men and increasing with age (31). The participants in our study had a normal AHI (<5) in the control sleep session. Although an increase was observed after the TS and CLS interventions, it was not statistically significant. Rossi et al. (29) reported that acute resistance training with 3 sets of 15 repetitions at 50% of 1-RM with 90-s interset rest did not result in significant changes in AHI or SpO_2 parameters. Similarly, we observed no statistically significant difference in mean SpO_2 during NREM or REM sleep. This is likely related to the low AHI values in our participants.

Study Limitations

This study has several limitations that should be considered. First, the sample consisted of recreationally active men without sleep problems. Therefore, the findings of this study cannot be generalized to clinical populations with sleep disorders, sedentary individuals, elite athletes, or female athletes. Second, as this study examined only acute effects, the findings may not reflect the chronic effects of different resistance training set configurations. Finally, the time under tension (TUT) employed in our protocol may not have provided a sufficient stimulus to elicit measurable changes. Future research should include participants with clinically diagnosed sleep disorders, recruit across different performance levels and both sexes, and employ protocols with longer TUT to determine whether different outcomes emerge. In addition, to ensure sample homogeneity, future studies may consider incorporating biochemical and neuronal homogeneity criteria related to sleep.

Conclusion

A single session of resistance training using either a TS or CLS configuration did not significantly alter sleep architecture or nocturnal SpO_2 in healthy, recreationally active young men without sleep disorders.

Ethics

Ethics Committee Approval: Ethics committee approval for the study was obtained from the Medical Research Ethics Committee of the Ege University Faculty of Medicine (decision number: 21-3.1T/60, date: 18.03.2021).

Informed Consent: All participants were informed about the purpose of the study and signed a voluntary consent form prior to participation, in accordance with the Declaration of Helsinki.

Footnotes

Authorship Contributions

Concept: O.D., C.K., Design: O.D., P.O., B.Ö., Data Collection or Processing: O.D., P.O., Analysis or Interpretation: O.D., C.K., P.O., B.Ö., Literature Search: O.D., Writing: O.D.

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Association of Problematic Smartphone Use with Sleep Quality and Hygiene: A Study in University Students

Problemli Akıllı Telefon Kullanımının Uyku Kalitesi ve Uyku Hijyeni ile İlişkisi: Üniversite Öğrencileri Üzerine Bir Çalışma

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Abstract

Objective: The university period is a time characterized by significant lifestyle and psychological changes, during which smartphone use is widespread and often excessive. The aim of this study was to examine the relationship between smartphone addiction, sleep quality, and sleep hygiene.

Materials and Methods: This descriptive cross-sectional study was conducted with 408 students. Data were collected through face-to-face surveys, including a sociodemographic form, the Smartphone Addiction Scale (SAS)-Short Form, the Pittsburgh Sleep Quality Index, and the Sleep Hygiene Index (SHI).

Results: The mean age was 20.7 ± 1.9 years; 48.5% were male. According to SAS, 34.3% had smartphone addiction. Poor sleep quality was observed in 81.9% overall and 91.4% among those with addiction. SHI scores were significantly higher in addicted students.

Conclusion: Smartphone addiction is prevalent and negatively affects sleep quality and hygiene. Poor sleep hygiene is associated with low sleep quality. Promoting healthy screen use and sleep habits is essential to support student well-being.

Keywords: Students, smartphone, internet addiction disorder, sleep, sleep hygiene

Öz

Amaç: Üniversite dönemi, yaşam tarzı ve psikolojik değişimlerin yoğun yaşandığı bir süreç olup akıllı telefon kullanımının yaygın ve çoğu zaman aşırı olduğu bir dönemdir. Bu çalışmanın amacı, akıllı telefon bağımlılığı ile uyku kalitesi ve uyku hijyeni arasındaki ilişkiyi incelemektir.

Gereç ve Yöntem: Bu tanımlayıcı kesitsel çalışma, 408 üniversite öğrencisiyle yürütülmüştür. Veriler, yüzyüze uygulanan anketler aracılığıyla toplanmıştır. Anket formları; sosyodemografik özellikler, Akıllı Telefon Bağımlılığı Ölçeği (ATBÖ)-Kısa Versiyonu, Pittsburgh Uyku Kalitesi İndeksi ve Uyku Hijyeni İndeksi (UHI)'ni içermektedir.

Bulgular: Katılımcıların yaş ortalaması 20,7 ± 1,9 yıldır ve %48,5'i erkektir. ATBÖ sonuçlarına göre öğrencilerin %34,3'ünde akıllı telefon bağımlılığı saptanmıştır. Genel olarak katılımcıların %81,9'unda, akıllı telefon bağımlılığı olan grupta ise %91,4'ünde kötü uyku kalitesi görülmüştür. UHI puanları akıllı telefon bağımlılığı olan öğrencilerde anlamlı düzeyde daha yüksektir.

Sonuç: Akıllı telefon bağımlılığı yaygın olup, uyku kalitesi ve hijyenini olumsuz etkilemektedir. Kötü uyku hijyeni, düşük uyku kalitesiyle ilişkilidir. Öğrencilerin sağlığını desteklemek için sağlıklı ekran kullanımı ve uyku alışkanlıklarının teşvik edilmesi önemlidir.

Anahtar Kelimeler: Öğrenciler, akıllı telefon, internet bağımlılığı bozukluğu, uyku, uyku hijyeni

Introduction

In recent years, smartphones have become one of the most prominent technological tools in the daily lives of university students (1). Functioning similarly to computers, these devices support a wide range of activities such as social interaction, entertainment, and access to information by providing constant internet access (2). The rapid development of technology and the widespread availability of smartphones have led to a global increase in their use (3). The multifunctional nature of smartphones contributes to their widespread adoption among university students (4). However, excessive participation in

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social media and gaming applications has been shown to be associated with an increased risk of addiction (5).

Smartphone addiction (SA), although not officially recognized as a clinical disorder, has attracted significant attention in research. Problematic smartphone usage is classified as a behavioral addiction, and validated scales have been developed to identify the presence of SA using specific score thresholds (2,6-8). Evidence suggests that SA can negatively affect not only academic performance but also physical and mental health indicators such as neck-shoulder pain, depression/anxiety, and sleep (9,10). Given the growing concern about this issue, it is crucial to examine the underlying causes and mechanisms of SA among university students (10).

Sleep is a fundamental requirement for overall health and quality of life (11), and maintaining good sleep quality is important for individuals of all ages (12). Insufficient or poor-quality sleep can negatively affect both mental and physical well-being (11). Although various factors can impair sleep quality, smartphone use is increasingly recognized as a significant contributor that adversely affects sleep primarily by prolonging nighttime wakefulness and disrupting natural sleep patterns (12).

In adults, high-quality sleep is generally defined by the ability to fall asleep within a reasonable period, maintain uninterrupted deep sleep during the night, and resume sleep quickly following nocturnal awakenings (12). Sleep hygiene is a fundamental determinant of sleep health, encompassing daily habits, behavioral routines, and environmental conditions that promote restorative sleep and enhance overall sleep quality (13,14). Ensuring proper sleep quality and adherence to good sleep hygiene practices is crucial for general health and is particularly important for university students. Sleep can be negatively impacted by various environmental factors, including noise, lighting, and room temperature, as well as lifestyle behaviors such as caffeine and alcohol consumption, tobacco use, irregular sleep schedules, and low physical activity levels. The use of electronic devices before bedtime is particularly disruptive, as it interferes with circadian rhythms and alters normal sleep-wake cycles (14). Previous studies demonstrate that increased smartphone use is associated with poorer sleep outcomes, and that disturbed sleep is one of the primary consequences of problematic or addictive smartphone use (9,12,15,16).

Based on the context outlined above, the present study investigated how SA is related to sleep quality and sleep hygiene among university students. The study sought to answer the following questions: What is the prevalence of SA among university students? What factors are associated with SA? How is SA related to sleep quality and sleep hygiene?

Materials and Methods

Study Design

This research was conducted as a descriptive cross-sectional study.

All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1975 Helsinki Declaration, as revised in 2000. Ethical approval for the study was obtained from the Sivas Cumhuriyet University Scientific Research and Publication Ethics Committee for Social and Human Sciences (approval number: 47, date: 06.01.2022).

Population and Sample

The study population comprised 27,000 students enrolled in faculties located on the main campus of Sivas Cumhuriyet University. Using a 95% confidence level and a 5% margin of error, the minimum required sample size was calculated as 379 participants, assuming an unknown prevalence. To reach students, a survey stand was established in the university cafeteria, where student density is highest, and the questionnaires were administered in person by the research team.

Data Collection Tool

Before beginning data collection, participants were fully informed about the study, and only those who provided written consent were included. The questionnaire consisted of a total of 60 items. The first 26 items gathered demographic information (e.g., age, gender, place of residence) and sleep-related habits. The remaining 34 items included the Smartphone Addiction Scale-Short Form (SAS-SF), the Pittsburgh Sleep Quality Index (PSQI), and the Sleep Hygiene Index (SHI).

Smartphone Addiction Scale-Short Form

The SAS-SF, developed by Kwon et al. (17) to evaluate the risk of SA among adolescents, consists of 10 items rated on a six-point Likert scale ranging from 1 to 6. Total scores can range from 10 to 60, with higher scores indicating a greater risk of SA. The scale is unidimensional and does not include any subscales. In the original Korean sample, cut-off values were set at 31 for males and 33 for females. The scale demonstrated strong internal consistency and concurrent validity, with a Cronbach's alpha of 0.91. The Turkish version of the SAS-SF was validated and adapted by Noyan et al. (18).

Pittsburgh Sleep Quality Index

The PSQI, developed by Buysse et al. (19), is designed to assess sleep quality over the preceding month. The Turkish version was validated by Ağargün et al. (20). The instrument includes 24 items, 19 of which are self-reported, while 5 are answered by a spouse or roommate and are not included in the scoring. The PSQI evaluates seven components: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. Each component is scored from 0 to 3, resulting in a global score ranging from 0 to 21. Scores of ≤5 indicate good sleep quality, whereas scores >5 denote poor sleep quality. The scale demonstrated good internal consistency, with a reported Cronbach's alpha of 0.80.

Sleep Hygiene Index

The SHI, which was adapted into Turkish by Özdemir et al. (21), comprises 13 items rated on a five-point Likert scale (1 = never, 2 = rarely, 3 = sometimes, 4 = often, 5 = always). Higher

scores indicate poorer sleep hygiene practices. The items were developed based on the insufficient sleep hygiene criteria outlined in the International Classification of Sleep Disorders. The SHI demonstrated acceptable reliability, with a Cronbach's alpha of 0.70.

Statistical Analysis

Data were analyzed using SPSS for Windows, version 25. The normality of continuous variables was assessed with the Shapiro-Wilk test. Descriptive analyses were conducted first, with categorical variables reported as frequencies and percentages, and continuous variables summarized as mean ± standard deviation. A p-value of <0.05 was considered statistically significant, with a 95% confidence interval. Inferential statistical tests included chi-square tests, Spearman correlation, independent-samples t-tests, and one-way ANOVA followed by Tukey test for post-hoc pairwise comparisons.

Results

Participant Demographics

A total of 408 students participated voluntarily in the study. The average age of the participants was 20.7 ± 1.9 years (range: 16–25), with 48.5% (n = 198) identifying as male and 51.5% (n = 210) as female. Of the students participating in the study, 55.7% (n = 227) were studying health sciences, 25.6% (n = 104) were studying social sciences, 14.5% (n = 59) were studying natural sciences and engineering, and the remainder were studying in other departments. A chronic health condition was reported by 12.7% of the participants (n = 52). Most students (72.8%, n = 297) were living away from their families. Regarding lifestyle factors, 33.3% (n = 136) were smokers and 29.4% (n = 120) reported alcohol consumption. The descriptive characteristics of the participants are presented in Table 1.

Smartphone Usage Characteristics and SAS-SF Scores

Regarding smartphone use, 49% (n = 200) primarily used their devices for social media, 36% (n = 147) for communication, 12.5% (n = 51) for general internet access, and 2.5% (n = 10) for gaming. Daily smartphone use was reported as less than 1 hour by 1.7% (n = 7), 1–4 hours by 45.6% (n = 186), 4–8 hours by 44.9% (n = 183), and more than 8 hours by 7.8% (n = 32). The mean SAS-SF score was 28.6 ± 11.8 (range: 10–60), and based on the SAS-SF cut-off points, 34.3% of students (n = 140) were classified as having SA. Those without chronic illnesses had significantly higher average SAS-SF scores than those with chronic illnesses ($p < 0.001$). The mean SAS-SF score was significantly higher for smokers than for non-smokers ($p = 0.037$). There was no significant difference in mean SAS-SF score according to participants' gender, place of residence, or alcohol use ($p > 0.05$). The majority of students (58.8%) went to bed after 1:00 a.m. Those who went to bed between 10:00 p.m. and 12:00 a.m. had a significantly higher mean SAS-SF score than those who went to bed after 1:00 a.m. ($p < 0.05$). Demographic characteristics and average SAS-SF score comparisons are shown in Table 1.

Table 1. Sociodemographic characteristics of the participants and their comparison with the SAS-SF average score.

	n/%	Mean ± SD	p
Gender			
Female	210 (51.5)	28.6 ± 12.5	0.538
Male	198 (48.5)	29.0 ± 11.3	
Place of residence			
With family	111 (27.2)	28.9 ± 11.4	0.719
Away from family	297 (72.8)	28.4 ± 12.1	
Chronic disease			
Yes	52 (12.7)	19.5 ± 8.3	**<0.001**
No	356 (87.3)	29.9 ± 11.8	
Smoking			
Yes	136 (33.3)	30.3 ± 13.1	**0.037**
No	272 (66.7)	27.7 ± 11.2	
Alcohol consumption			
Yes	120 (29.4)	28.1 ± 14.4	0.608
No	288 (70.6)	28.8 ± 10.8	
Bedtime			
10:00 p.m.–12:00 a.m.	63 (15.4)	32.6 ± 11.5	**0.014[a]**
12:00–1:00 a.m.	105 (25.7)	27.7 ± 10.5	
After 1:00 a.m.	240 (58.8)	27.9 ± 12.4	
Primary purpose of smartphone use			
Communication	147 (36)	24.7 ± 14.4	**<0.001[b]**
Social media	200 (49)	31.9 ± 12.8	
Gaming	10 (2.5)	31.2 ± 8.8	
Internet access	51 (12.5)	26.3 ± 9.1	

n: Number of participants, SD: Standard deviation, SAS-SF: Smartphone Addiction Scale–Short Form, Post-hoc analyses were significant ($p < 0.05$) for: [a]: 10:00 pm.–12:00 am .vs. 12:00–1:00 am., [b]: Communication vs. social media.

Sleep Quality and Sleep Hygiene Scores

The mean PSQI score was 7.6 ± 3.9 (range: 2–21). PSQI scores indicate sleep quality, with values ≤5 representing "good sleep" and scores >5 indicating "poor sleep." In this study, 81.9% of participants (n = 334) were classified as having poor sleep quality.

The distribution of sleep quality according to demographic and behavioral factors is presented in Table 2. The prevalence of poor sleep quality was greater among female students than males and higher among smokers compared to non-smokers. Additionally, poor sleep quality differed according to wake-up time ($p = 0.001$), with post-hoc analyses revealing a significantly higher prevalence among students waking after 12:00 pm (97.1%) compared to those waking before 10:00 am (81.9%) and those waking between 10:00 am and 12:00 pm (70.3%) ($p < 0.05$ for both). In contrast, no statistically significant association was found between the use of electronic devices before bedtime and sleep quality ($p = 0.072$), with a high prevalence of poor sleep quality observed in both groups. Comparisons of gender, lifestyle factors, and SAS-SF and SHI scores according to sleep quality are shown in Table 2.

Table 2. Factors associated with sleep quality in university students.

	Good sleep quality (PSQI < 5)	Poor sleep quality (PSQI ≥ 5)	p
Gender	**n (%)**	**n (%)**	
Female	29 (13.8%)	181 (86.2%)	**0.021**
Male	45 (22.7%)	153 (77.3%)	
Age	20.6 ± 1.8	20.4 ± 1.7	0.704
Smoking			
Yes	17 (12.5%)	119 (87.5%)	**0.041**
No	57 (21.0%)	215 (79.0%)	
Alcohol consumption			
Yes	15 (12.5%)	51 (91.1%)	0.050
No	59 (20.5%)	229 (79.5%)	
Bedroom occupancy			
One person	43 (22.6%)	147 (77.4%)	0.081
Two people	11 (12.8%)	75 (87.2%)	
Three or more people	20 (15.2%)	112 (84.8%)	
Eating before bedtime			
Yes	54 (20.8%)	205 (79.2%)	0.056
No	20 (13.4%)	129 (86.6%)	
Consumption of caffeinated beverages after 6:00 pm			
Yes	16 (13.6%)	102 (86.4%)	0.657
Sometimes	14 (18.2%)	63 (81.8%)	
No	5 (13.5%)	32 (86.5%)	
Using electronic devices before bedtime			
Yes	74 (18.5%)	326 (81.5%)	0.072
No	0 (0%)	8 (100%)	
Wake-up time			
Before 10:00 a.m.	45 (18.1%)	203 (81.9%)	**0.001**[a,b]
10:00 a.m.-12:00 p.m.	27 (29.7%)	64 (70.3%)	
After 12:00 p.m.	2 (2.9%)	67 (97.1%)	
SAS-SF score	26.3 ± 8.7	29.1 ± 12.6	**0.026**
SHI score	33.3 ± 7.0	37.2 ± 10.4	**0.003**

PSQI: Pittsburgh Sleep Quality Index, SAS-SF: Smartphone Addiction Scale–Short Form, SHI: Sleep Hygiene Index. Post-hoc analyses were significant (p < 0.05) for: [a]: Before 10:00 AM vs. after 12:00 pm, [b]: 10:00 am–12:00 pm vs. after 12:00 pm.

The mean SHI score among the participants was 36.5 ± 10.5 (range: 15–65). Participants who lived away from their families, smoked, and consumed alcohol had significantly higher average SHI scores compared to others (p < 0.001). Additionally, students who did not wake up feeling rested in the morning and those who slept during the day had significantly higher average SHI scores (p < 0.001). As expected, those who ate and used electronic devices before bed also had significantly higher average SHI scores (p < 0.001), and those who often drank tea or coffee in the evening had significantly higher average SHI scores than those who never or only occasionally drank tea or coffee in the evening (p < 0.05). Table 3 shows a comparison of the participants' mean SHI scores according to demographic data and sleep habits.

Table 3. Demographic data of participants and comparison of their sleep-related habits with their mean SHI scores.

	Mean ± SD	p
Gender		
Female	35.9 ± 9.9	0.221
Male	37.1 ± 10.1	
Place of residence		
With family	28.9 ± 6.2	**<0.001**
Away from family	39.4 ± 9.7	
Smoking		
Yes	43.7 ± 10.9	**<0.001**
No	32.9 ± 7.3	
Alcohol consumption		
Yes	45.0 ± 10.5	**<0.001**
No	33.0 ± 7.3	
Sleeping during the day		
Yes	40.6 ± 9.2	**<0.001**
No	35.0 ± 9.9	
Waking up feeling rested in the morning		
Yes	32.7 ± 7.4	**<0.001**
No	38.8 ± 10.6	
Eating before bedtime		
Yes	38.4 ± 10.9	**<0.001**
No	33.3 ± 7.3	
Using electronic devices before bedtime		
Yes	39.0 ± 5.1	**<0.001**
No	34.5 ± 10.1	
Drinking tea or coffee in the evening		
Yes	37.8 ± 10.8	**<0.005**[a,b,c]
Sometimes	36.2 ± 9.4	
No	32.7 ± 7.4	

SHI: Sleep Hygiene Index, SD: Standard deviation, Post-hoc analyses were significant (p < 0.05) for: [a]: Yes vs. sometimes, [b]: Yes vs. no, [c]: Sometimes vs. no.

Association of SA with Sleep Quality and Sleep Hygiene Scores

Among students with SA, 91.4% experienced poor sleep quality, a prevalence significantly higher than that of non-addicted students (Table 4).

Students identified as having SA exhibited significantly higher SHI scores (37.1 ± 8.7) compared to their non-addicted peers (34.3 ± 7.6; p = 0.012).

Discussion

Smartphones serve as multifunctional tools that meet university students' needs for communication, access to information, social interaction, gaming, and entertainment. Studies show that smartphone use is widespread among university students

Table 4. Comparison of smartphone addiction prevalence according to categorical PSQI scores.

	Good Sleep Quality (PSQI < 5)	Poor Sleep Quality (PSQI ≥ 5)	p
SA	**n (%)**	**n (%)**	
Yes	12 (8.6)	128 (91.4)	**<0.001**
No	61 (23.1)	206 (76.9)	

n: Number of participants, PSQI: Pittsburgh Sleep Quality Index, SA: Smartphone addiction according to the Smartphone Addiction Scale–Short Form.

and significantly influences their daily routines (12). Problematic smartphone use can negatively affect both physical and psychological health (3). Reported prevalence rates of SA in different countries range from 38.9% to 67% (3,5,6). The results of our study are consistent with the literature.

Findings regarding gender differences in SA are conflicting. Some studies suggest that female students have higher SA levels, often attributed to more intensive use of social networking platforms (3,10,16). In contrast, other studies report higher SA scores among males (22,23), while several investigations have found no gender-based differences (5,15,24). Similarly, in our study, no significant difference was observed between male and female participants in terms of SAS-SF scores.

There is also evidence in the literature indicating significant associations between the purposes of smartphone use and addiction levels. In one Turkish study, more than half of students reported using smartphones primarily to access social media, and those who did so had higher SAS-SF scores than students who mainly used their phones for studying or communication (24). Likewise, a study conducted in Saudi Arabia found that most participants used smartphones predominantly for social networking platforms (5). Consistent with these findings, the majority of students in our study reported using their smartphones mainly for social media, and this group had significantly higher SAS-SF scores. These results support the view that social media use is one of the major contributors to SA, particularly among young adults.

The literature includes numerous studies examining the relationship between SA and sleep quality, and these studies generally report that individuals with SA experience significantly poorer sleep quality compared to their non-addicted peers (12,15,16,22). However, some studies have found no significant association between these variables (23). In our study, nearly all students diagnosed with SA had poor sleep quality, and this proportion was markedly higher than that observed among students without SA. Our findings are consistent with the literature indicating that SA negatively affects sleep quality. Possible explanations include intensive social media use, prolonged nighttime use of digital devices, and a persistent need to remain online.

Sleep hygiene refers to avoiding behaviors that disrupt sleep, particularly the use of cigarettes, alcohol, or caffeine in the evening and the use of electronic devices before bedtime. Poor sleep hygiene negatively affects daytime alertness and overall daily functioning (11). In our study, the mean SHI score was 36.5 ± 10.5. Our findings are consistent with previous studies conducted in Türkiye, although the scores are higher than those reported in international studies (11,25-27). These results suggest that sleep hygiene among students in Türkiye may be poorer compared to international standards. Possible contributing factors include cultural differences, social lifestyle patterns, academic pressure, and living arrangements.

In this study, students with SA had significantly higher SHI scores than their non-addicted peers. Likewise, students who used electronic devices before bedtime also had significantly higher SHI scores. Problematic smartphone use in the evening increases physiological and cognitive arousal, making it more difficult to fall asleep and delaying sleep onset. In addition, blue light emitted from smartphones suppresses melatonin secretion, creating a biological mechanism that disrupts sleep initiation and restorative sleep (22). These findings indicate that SA affects sleep quality not only through behavioral pathways but also via biological mechanisms. Therefore, regulating sleep hygiene practices and screen time is critical for protecting sleep health in young adults.

Numerous studies have demonstrated a strong association between SHI scores and overall sleep quality (25-28). Poor sleep hygiene practices are linked to shorter sleep duration and reduced sleep quality (28). Consistent with these findings, students classified as having poor sleep quality in our study had markedly higher SHI scores than those reporting good sleep quality. When factors associated with sleep hygiene were examined, no significant gender difference was found. Some studies support this finding (11,14,25,29), whereas others report poorer sleep hygiene among males (27,30). Students living with their families had significantly better sleep hygiene. Because sleep hygiene is influenced by environmental factors such as light exposure, noise, external stimuli, and room temperature, having a private and more controlled sleeping environment at home may reduce exposure to these disruptors and contribute to better sleep hygiene (30). Although some studies support this finding (27,30), others report no significant association between place of residence and sleep hygiene (14,29).

Study Limitations

This study has several limitations. First, its cross-sectional design and reliance on self-report questionnaires limit the ability to draw causal inferences. Sleep quality was assessed solely based on participants' subjective reports; incorporating objective measurements could have enhanced the reliability of the findings. In addition, data were collected at a single time point, which may not reflect fluctuations in sleep patterns throughout the academic year, such as during examination periods. Longitudinal and follow-up studies are needed to more accurately capture temporal variations in sleep quality.

Furthermore, certain factors known to affect sleep quality, such as dietary habits, physical activity, and genetic predispositions, were not evaluated. Finally, since the sample was drawn from a single university, the generalizability of the findings to other student populations may be limited.

Conclusion

In conclusion, this study demonstrates that SA is associated with poorer sleep hygiene and sleep quality among university students. The findings indicate that promoting conscious and balanced technology use, along with increasing awareness of sleep hygiene, is essential for maintaining healthy sleep patterns in young adults. Raising awareness among university students about how excessive smartphone use may lead to addiction and reduced sleep duration, organizing educational programs on this topic, and encouraging participation in alternative social and physical activities instead of spending all leisure time on smartphones are important steps. Behavioral strategies such as putting the phone on silent mode and using the "do not disturb" or airplane mode before going to bed may help reduce nighttime screen exposure, shorten sleep onset latency, and contribute to better sleep quality. In this context, individual sleep hygiene habits and self-regulation strategies related to technology use can be considered preventive interventions.

Our study contributes to the literature by highlighting the relationship between the commonly observed problems of SA and sleep disturbances among university students and emphasizes the importance of preventive approaches. Future research should evaluate the effectiveness of structured intervention programs targeting SA and examine in greater detail the determining roles of individual characteristics, environmental conditions, and behavioral factors on sleep.

Ethics

Ethics Committee Approval: Ethical approval for the study was obtained from the Sivas Cumhuriyet University Scientific Research and Publication Ethics Committee for Social and Human Sciences (approval number: 47, date: 06.01.2022).

Informed Consent: Informed consent was obtained from all individual participants included in the study.

Footnotes

Authorship Contributions

Concept: S.K., E.A., Design: S.K., E.A., Data Collection or Processing: S.K., E.A., Analysis or Interpretation: S.K., Y.A., Literature Search: Y.A., Writing: S.K., E.A., Y.A.

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Adaptation of the Light Exposure Behavior Assessment into Turkish: A Validity and Reliability Study

Işık Maruziyeti Davranışı Anketi'nin Türkçeye Uyarlanması: Geçerlik ve Güvenirlik Çalışması

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Abstract

Objective: This study aimed to translate and culturally adapt the light exposure behavior assessment (LEBA) questionnaire into Turkish and evaluate its psychometric properties in terms of reliability and validity.

Materials and Methods: The LEBA was translated using the translation, review, adjudication, pretesting, and documentation method. Data were collected online from 445 Turkish-speaking adults residing in Türkiye and abroad. Statistical analyses included content validity, confirmatory and exploratory factor analyses, reliability testing (Cronbach's α, McDonald's ω), and non-parametric comparisons across demographic and cultural groups.

Results: The Turkish version of the LEBA demonstrated high content validity and acceptable construct validity. The exclusion of items 05 and 06 further improved the factorial fit of the model. Internal consistency was adequate overall (α = 0.70; ω total = 0.83), although the fifth subscale showed weak reliability (ordinal α = 0.54, ω = 0.53). Weak but significant associations were observed between LEBA factors and sleep quality, particularly for the subscale "Using a phone or smartwatch in bed", which was negatively associated with sleep quality. Cross-cultural comparisons indicated convergence in light exposure behaviors between Turkish residents and expatriates. Exploratory factor analysis revealed culturally specific factors, including behaviors related to controlling light in the sleep environment.

Conclusion: The Turkish LEBA is a valid and reliable tool for assessing light-related behaviors in Turkish-speaking populations. These findings highlight the importance of cultural adaptation in evaluating light exposure behaviors and their associations with sleep and circadian health.

Keywords: Light exposure, circadian rhythm, sleep, LEBA, psychometric, sociocultural, reliability, validity, Turkish

Öz

Amaç: Bu çalışma, ışık maruziyeti davranış değerlendirme (LEBA) anketinin Türkçeye çevrilmesi, kültürel uyarlanması ve psikometrik özelliklerinin (güvenirlik ve geçerlik) değerlendirilmesini amaçlamıştır.

Gereç ve Yöntem: LEBA, gözden geçirme, uzlaşma, ön test ve belgelendirme yöntemi kullanılarak Türkçeye çevrilmiştir. Veriler, Türkiye'de ve yurt dışında yaşayan toplam 445 Türkçe konuşan yetişkinden çevrim içi olarak toplanmıştır. İstatistiksel analizlerde kapsam geçerliği, doğrulayıcı ve açımlayıcı faktör analizleri, güvenirlik analizleri (Cronbach's α, McDonald's ω) ve demografik ile kültürel gruplar arasında parametrik olmayan karşılaştırmalar yapılmıştır.

Bulgular: Türkçe LEBA yüksek kapsam geçerliği ve kabul edilebilir yapı geçerliği göstermiştir. Madde 5 ve 6'nın çıkarılması faktör modelinin uyumunu artırmıştır. Ölçeğin genel iç tutarlılığı yeterli bulunmuştur (α = 0,70; ω toplam = 0,83), ancak beşinci alt boyut düşük güvenirlik göstermiştir (ordinal α = 0,54, ω = 0,53). LEBA faktörleri ile uyku kalitesi arasında zayıf fakat anlamlı ilişkiler gözlenmiş; özellikle "yatakta telefon veya akıllı saat kullanımı" alt boyutu düşük uyku kalitesi ile ilişkilendirilmiştir. Kültürlerarası karşılaştırmalarda, Türkiye'de yaşayanlar ile yurt dışındaki Türk katılımcılar arasında ışık maruziyeti davranışlarında benzerlik saptanmıştır. Açımlayıcı faktör analizi, uyku ortamında ışığın kontrolü ile ilişkili davranışlar gibi kültüre özgü faktörleri ortaya koymuştur.

Sonuç: Türkçe LEBA, Türkçe konuşan popülasyonlarda ışıkla ilişkili davranışları değerlendirmek için geçerli ve güvenilir bir araçtır. Bulgular, ışık maruziyeti davranışlarının değerlendirilmesinde kültürel uyarlamanın ve bu davranışların uyku ile sirkadiyen sağlıkla ilişkilerinin önemini vurgulamaktadır.

Anahtar Kelimeler: Işık maruziyeti, sirkadiyen ritim, uyku, LEBA, psikometrik, sosyokültürel, güvenirlik, geçerlik, Türkçe

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Introduction

Light is an essential driver of the synchronization of circadian rhythms and sleep with the external world (1). Light exposure can shift circadian rhythms, suppress melatonin secretion at night, and directly regulate cognitive performance and sleep through neural connections from the retina (2,3). Intrinsically photosensitive retinal ganglion cells are responsible for the above-mentioned non-visual effects of light and have photopigment melanopsin, whose peak sensitivity to light is approximately 480 nm (4,5). In general, exposure to bright light in the morning can support wakefulness and cognitive performance and advance the biological clock, whereas evening-time light exposure can delay the biological clock and sleep and reduce sleep quality (6,7). Owing to the widespread use of electrical lighting and digital screens, individual retinal light exposure extends beyond natural light-dark cycles. Therefore, optimizing both natural and artificial light exposure is crucial for maintaining and improving quality of life, since inappropriate light exposure is associated with several psychiatric and physiological disorders (8,9).

Understanding human light exposure in real-world settings is inherently complex. Light today functions as more than a mere visual stimulus; it represents a dynamic medium through which individuals engage to meet their visual, aesthetic, and psychosocial needs for overall well-being (10). The increasing use of adjustable lighting systems and the incorporation of light-modulating elements, such as blinds, illustrate how people actively regulate and personalize their light environments (11). Similarly, an individual's preferred activity related to natural light exposure, such as spending time outdoors or observing the sunset, reflects their preferred light exposure (12). Cultural, religious, and traditional practices, including early morning prayers, further shape an individual's pattern of light exposure. Rather than experiencing light as a passive environmental factor, individuals consciously construct and manipulate their preferred luminous surroundings as part of their daily lives.

The initial step in understanding human light exposure involves continuous measurement over extended periods using instruments such as sensors, photometers, or spectroradiometers. However, these approaches to measurement are limited by the high cost of equipment and the logistical challenges associated with long-term monitoring (13). Moreover, light exposure is influenced by numerous internal and external factors, including lifestyle habits, geographical location, weather conditions, and work schedules, which are often not captured by wearable light monitors (10). Consequently, while such devices provide objective data, they fail to reflect the contextual and behavioral dimensions of light exposure. An alternative and complementary approach is the use of light-use preference surveys, which can be easily, quickly, and seamlessly integrated into daily life. Such surveys enable scalable assessments of personal light exposure patterns and facilitate epidemiological research on the human light exposome.

The light exposure behavior assessment (LEBA) is an inventory developed in English to assess an individual's behaviors related to light exposure (14). This psychometrically validated tool assesses five main behaviors: the use of blue light filters, spending time outdoors, electronic device use in bed, light use preferences before sleep, and light exposure throughout the day. The LEBA can be used to understand the impact of varying light exposure on physiology and psychology and inform interventions targeting these behaviors. One study revealed associations between healthy light exposure measured by the LEBA and positive emotions, improved memory and concentration, and sleep quality (15). Another study involving participants from diverse geographical and cultural backgrounds indicated that certain subfactors of light exposure behavior may vary across cultures (16). The LEBA, which was used to examine these cultural differences, has not yet been translated into other languages. Therefore, the aim of this study is to provide a Turkish translation of the LEBA and assess its validity and reliability.

Materials and Methods

The Light Exposure Behavior Assessment

LEBA is a self-report survey designed to investigate behaviors that may influence an individual's light exposure. The initial item pool consisted of 48 questions identified by an expert panel as relevant to light exposure behaviors. Following systematic item reduction and psychometric analyses, the final version of the instrument comprised 23 items. Each statement uses a five-point Likert scale with a range of 1 (Never) to 5 (Always). Respondents are instructed to complete the survey with reference to their behaviors during the past four weeks, providing a consistent and recent observation window. The LEBA assesses five behavioral factors: (F1) wearing blue light filters, (F2) spending time outdoors, (F3) using a phone or a smartwatch in bed, (F4) controlling environmental light before bedtime, and (F5) using light in the morning or during daytime. Subscale scores were calculated as the mean of the relevant items, with F1 derived from items 01–03, F2 from items 04 (reverse-coded) and 05-09, F3 from items 10-14, F4 from items 15-18, and F5 from items 19-23 (14).

Translation Protocol

The original LEBA was developed in English and subsequently translated into Turkish using the method translation, review, adjudication, pretesting, and documentation (17). This systematic approach was designed to maximize both the linguistic accuracy and cultural validity of the instrument. Three independent bilingual translators first produced forward translations of all items. These translations were then reviewed and harmonized by an additional independent expert to resolve discrepancies and ensure conceptual equivalence. An adjudication panel, consisting of another independent specialist and the reviewer, finalized the wording. The pre-final version was then subjected to a cognitive pretest with a small sample from the target population (n = 5) to evaluate clarity, comprehensibility, and cultural appropriateness. Following this step, the adjudication panel conducted a final review and confirmed the definitive Turkish version of the instrument.

Sample and Recruitment

Data were collected between April 2024 and September 2025, spanning multiple seasons to minimize potential seasonal bias in light-related behaviors. Participants were recruited digitally through an anonymous Qualtrics survey link (Qualtrics, Provo, UT). Eligibility was restricted to adults aged 18-65 years. In Türkiye, recruitment was facilitated through three university hubs located in İzmir, İstanbul, and Nevşehir. Additionally, Turkish expatriates were recruited via advertisements posted on the Prolific platform (www.prolific.com). The inclusion criteria for expatriate participants included fluency in Turkish and reporting Turkish as their primary and native language while currently residing outside of Türkiye. Quality control procedures led to the exclusion of six survey responses because of completion durations shorter than 120 seconds, two survey responses because of completion by an individual outside the eligible age range, and one survey response because of extremely low variance (≥80% identical responses across LEBA items). No duplicate responses were identified. After these criteria were applied, the final sample for analysis included 445 valid survey responses, including 370 participants residing in Türkiye and 75 Turkish expatriates living abroad. The İzmir Institute of Technology Research Ethics Committee reviewed and approved the protocol (approval no: 26.01.2024-01/06, date: 26.01.2024). Each participant provided written informed consent before participating in the study.

Study Design

This study was designed as a translation and cultural adaptation study in which a random population completed an online survey to evaluate the reliability and validity of the LEBA in Turkish. The survey instrument comprised three main sections. The first section included demographic and health-related questions concerning age, sex, employment status, shift work, diagnosed sleep disorders, diagnosed psychological disorders, chronic diseases affecting sleep, visual impairments, and the use of visual aids with filter features (e.g., UV, orange, or blue light blocking), as well as any medically diagnosed light sensitivity or chronic illnesses causing light sensitivity. The second section consisted of the final translated version of the 23-item LEBA. In addition, participants were presented with the Turkish translations of the remaining items from the original 48-item pool that were not included in the final LEBA. The third section consisted of the Pittsburgh Sleep Quality Index (PSQI), which is designed to collect information concerning sleep, as sleep represents one of the primary physiological effects of light exposure (18).

Statistical Analysis

All statistical analyses were performed using R software (version 4.4.1), and visualizations were created with GraphPad Prism 10 (GraphPad Software, San Diego, CA). Descriptive statistics were calculated as means and standard deviations for continuous variables and as counts and percentages for categorical and ordinal variables. The item response distributions were examined using histograms and density plots.

Content Validity

Items were evaluated by a panel of ten experts who rated the relevance on a four-point scale (1 = not relevant, 2 = somewhat relevant, 3 = quite relevant, 4 = highly relevant). The ratings predominantly fell between 3 and 4, indicating high relevance. The content validity index (CVI) was calculated for each item and for the overall instrument to quantify the degree of expert agreement.

Construct Validity

Prior to confirmatory factor analysis (CFA), the appropriateness of the sample and the suitability of the factor model were assessed. Multivariate normality was examined using skewness and kurtosis statistics, including Mardia's test. Post hoc sampling adequacy was evaluated using the Kaiser–Meyer–Olkin (KMO) measure and Bartlett's test of sphericity to confirm that correlations among items were sufficient for factor analysis. Given the ordinal nature of the survey items, a polychoric correlation matrix was employed to more accurately estimate inter-item associations. The CFA model was specified according to the factor structure proposed in the original study, with each factor treated as independent and without shared variance, and equality constraints were applied where appropriate. Model estimation was performed using the weighted least squares mean and variance adjusted (WLSMV) estimator to account for the ordinal nature of the data. Model fit was evaluated using multiple indices: scaled Comparative Fit index (CFI), scaled Tucker-Lewis index (TLI), Adjusted Goodness-of-Fit index (AGFI), Goodness-of-Fit index (GFI), Relative Fit index (RFI) and Normed Fit index (NFI) ≥ 0.90 and scaled root mean square error of approximation (RMSEA) and standardized root mean square residual (SRMR) ≤0.08, which were considered indicators of acceptable model fit. To further improve the structural equation model, modification indices and negative factor loadings were examined.

Reliability

Internal consistency and reliability analyses were conducted for the LEBA factors using a polychoric correlation matrix to account for the ordinal nature of the items. Cronbach's alpha (α), ordinal alpha, and McDonald's omega (ω) coefficients were calculated for each factor. At the global scale level, Cronbach's α, ordinal α, McDonald's ω total, and ω hierarchical were computed to assess the overall reliability of the instrument.

Criterion Validity

Convergent validity with sleep outcomes was assessed using Spearman's rank correlations between the LEBA factor scores and the PSQI total score (range 0-21; higher scores indicate poorer sleep quality), its seven subcomponents, self-reported sleep duration (hours), and sleep midpoint (clock time). Results were visualized using heatmaps. In addition, associations between LEBA subscale scores and employment status (as a major determinant of real-world light exposure) were examined using the Kruskal–Wallis test followed by Dunn's multiple comparisons test for post hoc comparisons. Further validation analyses were performed to test the associations between the LEBA subscales

and self-reported clinically diagnosed conditions, including sleep disorders, psychiatric disorders, chronic conditions with sleep impairment, ocular problems, light sensitivity, and the use of filtered visual aids, using Wilcoxon rank-sum tests. The false discovery rate correction method was applied to control for multiple comparisons.

Cross-Cultural Validity

To examine whether the LEBA subscale scores differed across cultural and environmental contexts, three subgroups were compared: Turkish participants residing in Türkiye, Turkish expatriates, and an external dataset from Malaysia (15). Group differences in each LEBA subscale were tested using the Kruskal-Wallis test, with post hoc Dunn's multiple comparisons test. As a further test of cultural influences on light-exposure behavior, the content validity of the original LEBA item pool was revisited. Specifically, the 48 items developed during the original LEBA construction were reanalyzed in the Turkish sample. A polychoric correlation matrix was analyzed using a scree plot and parallel analysis to identify the appropriate number of latent factors. An exploratory factor analysis (EFA) was subsequently performed with principal axis factoring and varimax rotation. Items with factor loadings <0.30 or cross-loadings >0.30 were iteratively excluded. The final retained factor structure, including the number of factors, item composition, and conceptual meaning, was compared with the five subscales of the LEBA to evaluate overlap and potential cultural differences.

Results

Sample Description

The final sample for analysis consisted of 445 participants (M age = 29.18 years, standard deviation (SD) = 10.58). Of these, 62.22% were female, and 37.78% were male. In terms of employment, 8.09% were not employed, 50.56% were students, 6.07% worked part-time, and 35.28% worked full-time. Among the total sample, 19.57% reported night-shift work. When stratified by residency, 370 participants were Turkish residents (M age = 28.16, SD = 10.96; 64.31% female), and 75 were Turkish expatriates living abroad (M age = 34.19, SD = 6.46; 52.00% female).

In terms of health characteristics, 1.80% reported a physician-diagnosed sleep disorder, with insomnia (n = 4) and sleep apnea (n = 4) reported. A total of 11.24% reported a psychiatric disorder, most commonly anxiety disorders (n = 33) and depression (n = 26), followed by attention-deficit/hyperactivity disorder (n = 13), among others. Additionally, 4.04% reported a chronic disorder affecting sleep, 7.87% reported light sensitivity, and 2.70% reported a chronic condition affecting light sensitivity. Eye disorders, including glaucoma (n = 2), macular degeneration (n = 1), cataracts (n = 1), and color blindness (n = 2), were reported by 65.84% of the participants, along with refractive errors. All these participants reported using glasses or contact lenses, and 47.71% reported wearing filtered glasses.

Content Validity of the Turkish LEBA

The Turkish translation of the LEBA items can be found in Table 1. Content validity was evaluated using ratings from 10 expert reviewers for the 23-item LEBA long version. The item-level content validity indices ranged from 0.90 to 1.00, with a mean score per item between 3.6 and 4.0. The scale-level CVI, calculated by averaging across items, was 0.99, and the proportion of items on which all the experts rated the item as relevant was 0.91.

During the translation and adaptation process, one item ("I use an alarm with a dawn simulation light") was identified as potentially problematic, as dawn simulation devices are not widely known or used in Türkiye, and participants reported difficulty understanding the concept. The feedback collected at the end of the survey revealed several additional points: some participants noted that the questionnaire increased their awareness of light sensitivity and sleep habits, and many described it as useful and reflective. However, several limitations also emerged, including concerns about whether the instrument adequately captured the unique experiences of pregnant and breastfeeding mothers, who reported frequent nighttime awakenings due to childcare.

The distribution of item response patterns is presented in Table 2. Preliminary analyses indicated significant multivariate non-normality (Mardia's skewness = 5933.91, $p < 0.001$; kurtosis = 21.35, $p < 0.001$). The KMO measure of sampling adequacy (MSA) was acceptable (overall MSA = 0.64), with individual item values ranging from 0.54 to 0.80. Bartlett's test of sphericity was significant, χ^2 (253) = 2713.17, $p < .001$, indicating that the correlation matrix was suitable for factor analysis. Polychoric correlations were calculated to account for the ordinal nature of the items, ranging from –0.65 to 0.88, with 7.51% of correlations exceeding |0.30|, suggesting adequate inter-item relationships for factor extraction. The strongest associations were observed between LEBA01–LEBA02 ($\rho = 0.88$), LEBA13–LEBA14 ($\rho = 0.80$), LEBA15–LEBA18 ($\rho = 0.77$), and LEBA01–LEBA03 ($\rho = 0.76$). A few moderate negative associations also emerged (e.g., LEBA04–LEBA05, $\rho = –0.65$). Overall, these results support subsequent CFA, although the violation of multivariate normality indicates that robust estimation methods, such as the WLSMV adjusted estimator, CFA was conducted to test the five-factor structure proposed in the original study (14). Consistent with the initial specification, equality constraints were imposed on the loadings of LEBA01–LEBA02 and LEBA15–LEBA18, and a residual covariance was freely estimated between LEBA13 and LEBA14 to account for shared variance. LEBA04 was reverse coded to align its scoring direction with the latent construct. The initial model demonstrated a reasonable but suboptimal fit, $\chi^2(231) = 1228.24$, CFI = 0.896, TLI = 0.886, GFI = 0.949, AGFI = 0.923, NFI = 0.880, RFI = 0.869, SRMR = 0.110, RMSEA = 0.074. On the basis of negative factor loadings and high modification indices, LEBA05 and LEBA06 were removed, and the error terms of LEBA07 and LEBA08 were allowed to correlate. The respecified model showed improved fit, $\chi^2(189) = 801.72$, CFI = 0.928, TLI = 0.920, GFI = 0.963, AGFI = 0.942, NFI = 0.912, RFI = 0.902, SRMR = 0.111, RMSEA = 0.066, indicating

Table 1. Original LEBA items and their Turkish translations.

Item no.	English original	Turkish translation
1	I wear blue-filtering, orange-tinted, and/or red-tinted glasses indoors during the day.	Gün içinde kapalı mekanlarda mavi filtreli, turuncu veya kırmızı camlı gözlük takarım.
2	I wear blue-filtering, orange-tinted, and/or red-tinted glasses outdoors during the day.	Gün içinde açık havada mavi filtreli, turuncu veya kırmızı camlı gözlük takarım.
3	I wear blue-filtering, orange-tinted, and/or red-tinted glasses within 1 hour before attempting to fall asleep.	Uykuya dalmaya çalışmadan önceki 1 saat içinde mavi filtreli, turuncu veya kırmızı camlı gözlük takarım.
4	I spend 30 minutes or less per day (in total) outside.	Dışarıda günlük toplam 30 dakika veya daha az vakit geçiririm.
5	I spend between 30 minutes and 1 hour per day (in total) outside.	Dışarıda günlük toplam 30 dakika ila 1 saat arasında vakit geçiririm.
6	I spend between 1 and 3 hours per day (in total) outside.	Dışarıda günlük toplam 1 ila 3 saat arasında vakit geçiririm.
7	I spend more than 3 hours per day (in total) outside.	Dışarıda günlük toplam 3 saatten fazla vakit geçiririm.
8	I spend as much time outside as possible.	Dışarıda olabildiğince çok vakit geçiririm.
9	I go for a walk or exercise outside within 2 hours after waking up.	Uyandıktan sonraki 2 saat içinde yürüyüş ya da egzersiz için dışarı çıkarım.
10	I use my mobile phone within 1 hour before attempting to fall asleep.	Uykuya dalmaya çalışmadan önceki 1 saat içinde cep telefonumu kullanırım.
11	I look at my mobile phone screen immediately after waking up.	Uyanır uyanmaz telefonumun ekranına bakarım.
12	I check my phone when I wake up at night.	Gece uyandığımda telefonumu kontrol ederim.
13	I look at my smartwatch within 1 hour before attempting to fall asleep.	Uykuya dalmaya çalışmadan önceki 1 saat içinde akıllı saatime göz atarım.
14	I look at my smartwatch when I wake up at night.	Gece uyandığımda akıllı saatime göz atarım.
15	I dim my mobile phone screen within 1 hour before attempting to fall asleep.	Uykuya dalmaya çalışmadan önceki 1 saat içinde cep telefonu ekranımın parlaklığını kısarım.
16	I use a blue-filter app on my computer screen within 1 hour before attempting to fall asleep.	Uykuya dalmaya çalışmadan önceki 1 saat içinde bilgisayarımın ekranında mavi filtre uygulaması kullanırım.
17	I use as little light as possible when I get up during the night.	Gece yataktan kalktığımda mümkün olduğunca az ışık kullanırım.
18	I dim my computer screen within 1 hour before attempting to fall asleep.	Uykuya dalmaya çalışmadan önceki 1 saat içinde bilgisayar ekranımın parlaklığını kısarım.
19	I use tunable lights to create a healthy light environment.	Sağlıklı bir aydınlatma sağlamak için rengi veya parlaklığı ayarlanabilen ışıklar kullanırım.
20	I use LEDs to create a healthy light environment.	Sağlıklı bir aydınlatma sağlamak için LED kullanırım.
21	I use a desk lamp when I do focused work.	Odaklanma gerektiren bir iş yaparken masa lambası kullanırım.
22	I use an alarm with a dawn simulation light.	Gün doğumunu taklit eden ışıklı bir alarm kullanırım.
23	I turn on the lights immediately after waking up.	Uyanır uyanmaz ışıkları açarım.

Light exposure behavior assessment, ("ışık maruziyeti davranışı anketi"). Instructions: Please indicate how often you performed the following behaviors in the past 4 weeks ("Lütfen son 4 hafta içinde aşağıdaki davranışları ne sıklıkla gerçekleştirdiğinizi belirtiniz"). Response categories: Never ("Asla"), Rarely ("Nadiren"), Sometimes ("Bazen"), Often ("Sıklıkla"), and Always ("Her zaman"). Factors: F1 (items 01– 03) – wearing blue light filters; F2 (items 04–09) – spending time outdoors; F3 (items 10–14) – using phone or smart watch in bed; F4 (items 15–18) – controlling environmental light before bedtime; F5 (items 19–23) – using light in the morning or during daytime.

LEBA: Light exposure behavior assessment.

Table 2. Summary of LEBA item-level results (n = 445).

	Summary statistics			Response pattern				
Item	Mean	Median	SD	Never	Rarely	Sometimes	Often	Always
F1: Wearing blue light filters								
LEBA01	1.6	1.6	1.2	70.79% (315)	11.69% (52)	5.62% (25)	5.84% (26)	6.07% (27)
LEBA02	1.8	1.8	1.2	64.49% (287)	13.26% (59)	9.21% (41)	6.97% (31)	6.07% (27)
LEBA03	1.5	1.5	1.1	80.00% (356)	6.97% (31)	4.49% (20)	4.27% (19)	4.27% (19)

Table 2. Continued.

	Summary statistics			Response pattern				
Item	**Mean**	**Median**	**SD**	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
F2: Spending time outdoors								
LEBA04	3.9	3.9	1.1	4.72% (21)	9.21% (41)	17.08% (76)	32.58% (145)	36.40% (162)
LEBA05	2.6	2.5	1.2	19.10% (85)	31.01% (138)	28.99% (129)	15.51% (69)	5.39% (24)
LEBA06	3.0	3.0	1.1	12.36% (55)	20.67% (92)	33.48% (149)	26.52% (118)	6.97% (31)
LEBA07	3.4	3.4	1.1	4.72% (19)	22.47% (100)	19.78% (88)	35.96% (160)	17.53% (78)
LEBA08	3.3	3.3	1.2	6.52% (29)	21.80% (97)	26.52% (118)	28.76% (128)	16.40% (73)
LEBA09	2.2	2.2	1.1	30.79% (137)	34.83% (155)	21.80% (97)	9.21% (41)	3.37% (15)
F3: Using phone and smartwatch in bed								
LEBA10	4.1	4.1	1.0	2.70% (12)	6.97% (31)	11.01% (49)	40.22% (179)	39.10% (174)
LEBA11	3.9	3.9	1.0	2.92% (13)	8.31% (37)	13.03% (58)	44.04% (196)	31.69% (141)
LEBA12	3.1	3.1	1.2	12.81% (57)	19.78% (88)	23.15% (103)	31.01% (138)	13.26% (59)
LEBA13	1.6	1.6	1.1	69.66% (310)	11.69% (52)	7.42% (33)	7.64% (34)	3.60% (16)
LEBA14	1.5	1.5	0.9	76.63% (341)	8.76% (39)	7.64% (34)	5.62% (25)	1.35% (6)
F4: Controlling environmental light before bedtime								
LEBA15	3.5	3.5	1.3	11.91% (53)	11.69% (52)	17.98% (80)	29.21% (130)	29.21% (130)
LEBA16	2.1	2.1	1.5	58.43% (260)	10.11% (45)	8.99% (40)	9.66% (43)	12.81% (57)
LEBA17	3.9	3.9	1.1	4.49% (20)	9.66% (43)	11.69% (52)	42.25% (188)	31.91% (142)
LEBA18	3.0	3.0	1.5	22.92% (102)	15.96% (71)	15.51% (69)	24.94% (111)	20.67% (92)
F5: Using light in the morning and during daytime								
LEBA19	2.2	2.2	1.3	42.47% (189)	20.22% (90)	16.85% (75)	11.46% (51)	8.99% (40)
LEBA20	2.6	2.6	1.4	32.81% (146)	16.18% (72)	20.90% (93)	20.00% (89)	10.11% (45)
LEBA21	2.5	2.5	1.3	30.79% (137)	24.04% (107)	19.33% (86)	19.55% (87)	6.29% (28)
LEBA22	1.3	1.3	0.7	85.39% (380)	6.97% (31)	4.94% (22)	1.35% (6)	1.35% (6)
LEBA23	2.4	2.4	1.2	25.84% (115)	34.38% (153)	21.57% (96)	13.03% (58)	5.17% (23)

LEBA: Light exposure behavior assessment, SD: Standard deviation.

good overall model adequacy despite a slightly elevated SRMR, which is considered acceptable for ordinal indicators analyzed with WLSMV estimation. The standardized factor loadings ranged from 0.13 to 0.94, with most exceeding 0.60, suggesting adequate item representation of the latent constructs. The full CFA results are presented in Table 3.

Reliability of the LEBA

The LEBA subscales were calculated after items 05 and 06 were excluded because of inadequate factorial validity. Internal consistency and reliability analyses were conducted for the five factors. Factor 1 demonstrated excellent reliability (α = 0.84, ordinal α = 0.91, ω = 0.87). Factor 2 demonstrated marginal reliability (α = 0.63, ordinal α = 0.67, ω = 0.55). Factor 3 demonstrated comparable moderate reliability (α = 0.65, ordinal α = 0.71, ω = 0.55). Factor 4 reached acceptable levels (α = 0.66, ordinal α = 0.72, ω = 0.74). Factor 5, however, showed poor internal consistency (α = 0.47, ordinal α = 0.54, ω = 0.53). At the global scale level, Cronbach's α was 0.70, and McDonald's ω total was 0.83, indicating adequate overall internal consistency. However, ω hierarchical was 0.29, suggesting that most reliable variance was attributable to the group (factor-specific) components rather than a general factor. Overall, these findings indicate that certain subscales (particularly F5) exhibit weak internal consistency and should be interpreted with caution.

Associations between LEBA Subscales and Sleep, Health, and Employment Status

Composite LEBA factor scores were calculated after items 05 and 06 were excluded (Figure 1A). Descriptive statistics indicated that mean scores ranged from 1.62 (SD = 1.01) for Factor 1 to 3.18 (SD = 0.78) for Factor 2. Median values were generally close to the means, with interquartile ranges suggesting moderate variability across factors. Shapiro–Wilk tests indicated significant deviations from normality for all factors ($p < 0.001$), supporting the use of non-parametric methods in subsequent analyses.

Sleep quality was assessed using the PSQI. The mean PSQI global score was 6.30 (SD = 2.88; range = 0-17), with higher scores indicating poorer sleep quality. Subscale means ranged from 0.19 (sleep medication use) to 1.51 (daytime dysfunction). The mean reported sleep duration was 7.34 hours (SD = 1.57), and the mean midsleep time was 4:10 a.m. (M = 4.18, SD = 1.31). Spearman correlations revealed generally weak associations between LEBA factors and PSQI indicators

(Figure 1B). Notably, Factor 1 (Wearing blue light filters) showed a small but significant positive correlation with sleep disturbances on the PSQI ($\rho = 0.11$, $p = 0.027$). Factor 3 (using a phone or a smartwatch in bed) was weakly but significantly associated with the global PSQI score ($\rho = 0.21$, $p < 0.001$) and midsleep timing ($\rho = 0.20$, $p < 0.001$), suggesting that later light exposure coincided with poorer sleep quality and a delayed sleep midpoint. Factor 4 (controlling environmental light before bedtime) showed a weak positive correlation with daytime dysfunction ($\rho = 0.17$, $p < 0.001$).

Factor 1 (Wearing blue light filters) was highly significantly different across health-related variables (Figure 1C). Participants who reported using glasses with filters scored significantly higher on Factor 1 ($W = 36519$, $p < 0.001$). Similarly, individuals reporting ocular problems also differed significantly in terms of Factor 1 scores ($W = 16303$, $p < 0.001$). These results indicate that Factor 1 differentiates individuals on the basis of the use of visual aids with filtering features.

A Kruskal-Wallis test was conducted to examine whether employment status groups differed in terms of LEBA factor

Table 3. The original (Model 1) and improved (Model 2) CFA models of the LEBA dataset.

	Model 1					Model 2				
Item	**Estimate**	**Std.err**	**z-value**	**P(>|z|)**	**Std.est**	**Estimate**	**Std.err**	**z-value**	**P(>|z|)**	**Std.est**
F1: Wearing blue light filters										
LEBA01	1.000				0.936	1.000				0.936
LEBA03	0.837	0.034	24.451	0.000	0.783	0.837	0.034	24.451	0.000	0.783
LEBA02	1.000				0.936	1.000				0.936
F2: Spending time outdoors										
LEBA07	1.000				0.657	1.000				0.562
LEBA08	1.046	0.058	17.895	0.000	0.687	1.096	0.080	13.699	0.000	0.616
LEBA04	1.196	0.054	22.162	0.000	0.785	1.288	0.408	3.160	0.002	0.723
LEBA09	0.212	0.075	2.812	0.005	0.139	0.465	0.125	3.719	0.000	0.261
LEBA05	-1.060	0.063	-16.709	0.000	-0.697					
LEBA06	-0.485	0.061	-8.010	0.000	-0.319					
F3: Using phone and smartwatch in bed										
LEBA10	1.000				0.748	1.000				0.748
LEBA11	1.217	0.085	14.258	0.000	0.910	1.217	0.085	14.258	0.000	0.910
LEBA12	0.829	0.052	15.903	0.000	0.620	0.829	0.052	15.903	0.000	0.620
LEBA13	0.168	0.076	2.208	0.027	0.126	0.168	0.076	2.208	0.027	0.126
LEBA14	0.286	0.082	3.467	0.001	0.214	0.286	0.082	3.467	0.001	0.214
F4: Controlling environmental light before bedtime										
LEBA15	1.000				0.875	1.000				0.875
LEBA16	0.705	0.048	14.586	0.000	0.617	0.705	0.048	14.586	0.000	0.617
LEBA18	1.000				0.875	1.000				0.875
LEBA17	0.256	0.057	4.507	0.000	0.224	0.256	0.057	4.507	0.000	0.224
F5: Using light in the morning and during daytime										
LEBA19	1.000				0.808	1.000				0.808
LEBA20	0.754	0.150	5.017	0.000	0.609	0.754	0.150	5.017	0.000	0.609
LEBA21	0.374	0.087	4.285	0.000	0.302	0.374	0.087	4.285	0.000	0.302
LEBA22	0.347	0.132	2.622	0.009	0.280	0.347	0.132	2.622	0.009	0.280
LEBA23	0.210	0.083	2.524	0.012	0.170	0.210	0.083	2.524	0.012	0.170
Model statistics:	$\chi^2(231) = 1228.240$, CFI (scaled) = 0.896, TLI (scaled) = 0.886, GFI = 0.949, AGFI = 0.923, NFI = 0.880, RFI = 0.869, SRMR = 0.110, RMSEA (scaled) = 0.074					$\chi^2(189) = 801.720$, CFI (scaled) = 0.928, TLI (scaled) = 0.920, GFI = 0.963, AGFI = 0.942, NFI = 0.912, RFI = 0.902, SRMR = 0.111, RMSEA (scaled) = 0.066				

Confirmatory factor analysis using the weighted least squares mean and variance adjusted estimator. Model 1 specified equality constraints on the loadings of LEBA01–LEBA02 and LEBA15–LEBA18; included a residual covariance between LEBA13 and LEBA14; reverse-coded LEBA04; included latent factors modelled as independent. Model 2 further refined the structure by removing LEBA05 and LEBA06 and allowing the error terms of LEBA07 and LEBA08 to correlate.

LEBA: Light exposure behavior assessment, CFA: Confirmatory factor analysis, CFI: Comparative Fit index, TLI: Tucker–Lewis index, GFI: Goodness-of-Fit index, AGFI: Adjusted Goodness-of-Fit index, NFI: Normed Fit index, RFI: Relative Fit index, SRMR: Standardized root mean square residual, RMSEA: Root mean square error of approximation.

scores, with pairwise comparisons presented in Figure 1D. For Factor 1, there was no significant difference between groups ($\chi^2(3) = 3.40$, $p = 0.334$), indicating similar scores across employment groups. Significant differences were found for Factor 2 ($\chi^2(3) = 26.46$, $p < .001$), Factor 3 ($\chi^2(3) = 28.20$, $p < 0.001$), Factor 4 ($\chi^2(3) = 38.67$, $p < 0.001$), and Factor 5 ($\chi^2(3) = 12.59$, $p = 0.006$). In general, unemployed participants scored lower than other employment groups did on Factors 2, 3, and 4. These results indicate that employment status is associated with differences in LEBA factor scores, except for Factor 1.

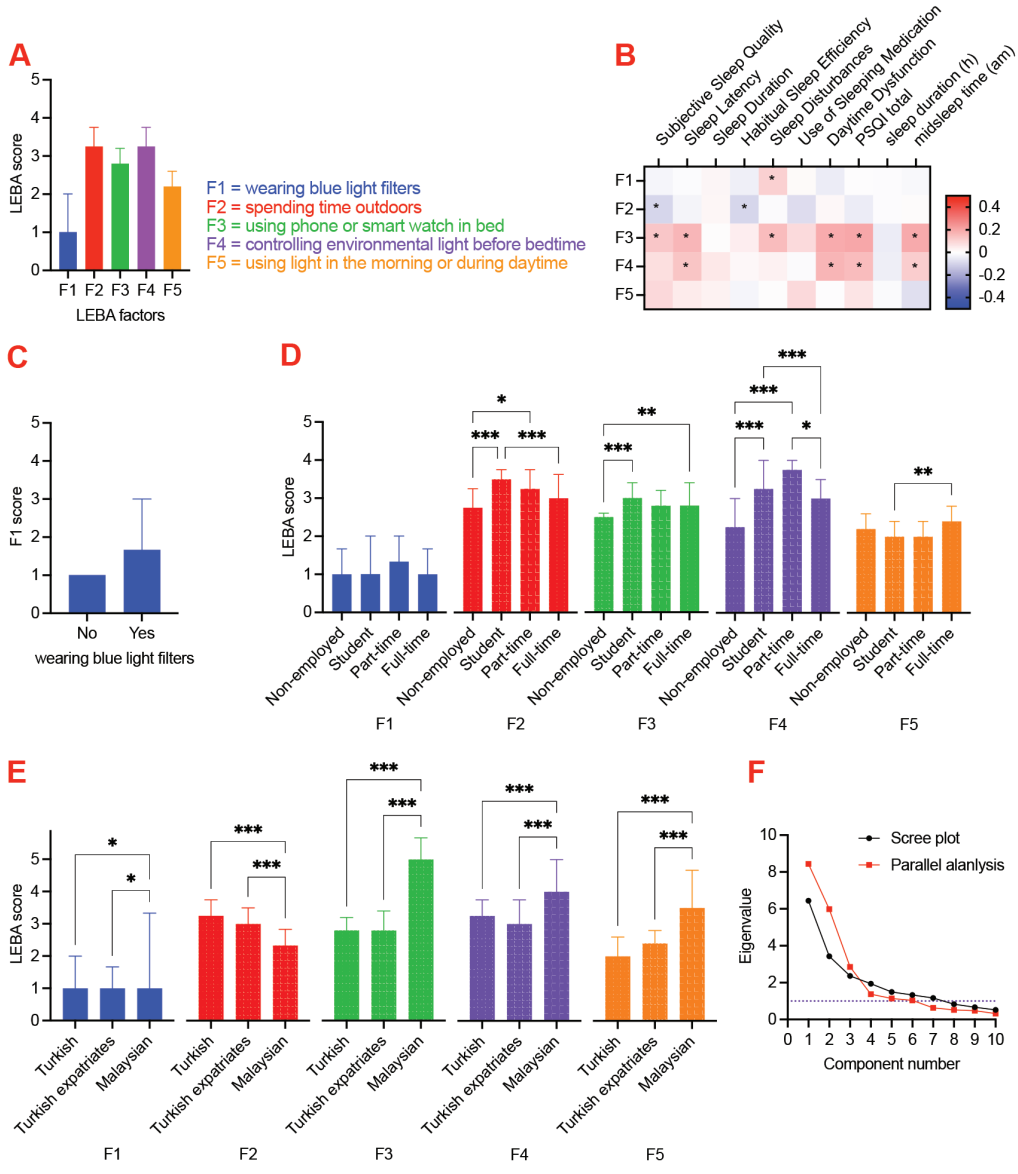

Figure 1. (A) Median and interquartile ranges of the LEBA factor subscale scores. (B) Spearman correlation coefficients between LEBA factors, PSQI scores, sleep duration, and sleep midpoint time are displayed as heatmaps (*p < 0.05). (C) Median and interquartile ranges of LEBA Factor 1 scores among participants with and without filtered glasses. (D) Comparison of LEBA factor scores by employment status (median and interquartile ranges). Kruskal-Wallis test with Dunn's correction for multiple pairwise comparisons; *p < 0.05, **p < 0.01, ***p < 0.001, p < 0.001. (E) Comparison of LEBA factor scores by country of residency (median and interquartile ranges). Kruskal–Wallis test with Dunn's correction for multiple pairwise comparisons; *p < 0.05, **p < 0.01, ***p < 0 0.001. (F) Scree plot showing the eigenvalues for the first ten components, including the results of parallel analysis.
LEBA: Light Exposure Behavior Assessment, PSQI: Pittsburgh Sleep Quality index.

Effects of Country of Residency and Language on LEBA Scores

A Kruskal-Wallis test revealed significant differences in LEBA factor scores across residency groups (Figure 1E): Factor 1 ($\chi^2(2) = 11.30$, $p = 0.004$), Factor 2 ($\chi^2(2) = 194.71$, $p < 0.001$), Factor 3 ($\chi^2(2) = 433.10$, $p < 0.001$), Factor 4 ($\chi^2(2) = 85.71$, $p < 0.001$), and Factor 5 ($\chi^2(2) = 265.58$, $p < 0.001$). Post hoc pairwise comparisons revealed notable differences: Malaysian participants scored higher on Factors 1, 3, 4, and 5 than Turkish residents and Turkish expatriates did, whereas Turkish residents and Turkish expatriates scored higher on Factor 2 than Malaysians did.

EFA of Turkish Light Exposure Behaviors

Using the 48 original LEBA items, Horn's parallel analysis indicated six components (Figure 1F). Iterative removal of items with low or cross-loadings resulted in a final six-factor model (Table 4). Factor loadings ranged from 0.33 to 0.96, with the six new factors together accounting for 50% of the total variance (0.11, 0.10, 0.08, 0.08, 0.08, 0.06). Factor 1 (wearing blue light filters) and Factor 2 (spending time outdoors) largely replicated the original LEBA domains, although Factor 2 excluded items 06 and 09. Factor 3 (using a phone or a smartwatch in bed) included items reflecting nighttime awakenings and screen use, suggesting a broader "screen-in-bed" construct. In this version, items 12-14 related to smartwatch use and phone usage during night awakenings were replaced with an added item ("I use my computer/laptop/tablet within 1 hour before attempting to fall asleep"). Factor 4 (controlling environmental light before bedtime) also aligned with the original structure but incorporated the item "I use a blue-filter app on my mobile phone screen within 1 hour before attempting to fall asleep" instead of item 17, which referred to light use during nighttime awakenings. Factor 5 (using light in the morning or daytime) emerged with modifications, excluding items 22 and 23 on dawn simulation lamps and light exposure immediately after waking, but adding two items on sunglass and visor use while outdoors in bright daylight. Finally, a culturally specific sixth factor emerged, representing behaviors not captured in the original LEBA, indicating a potential context-dependent dimension that warrants further investigation. This new factor included items such as "I purposely leave a light on in my sleep environment while sleeping", "I use as little light as possible when I get up during the night", "I turn on the lights when I get up during the night", "I close curtains or blinds to prevent light from entering the bedroom if I want to sleep", "I turn on my ceiling room light when it is light outside" and "I use an alarm with a dawn simulation light." These items clustered around a theme of managing artificial and natural light within the sleep environment, reflecting culturally specific sleep–light regulation behaviors.

Table 4. Factor loadings for the EFA model (6-Factor model).

Question no.	Question	Item no.	PA1	PA2	PA3	PA4	PA5	PA6
32	I dim my mobile phone screen within 1 hour before attempting to fall asleep.	LEBA15	0.68					
33	I dim my computer screen within 1 hour before attempting to fall asleep.	LEBA18	0.73					
34	I use a blue-filter app on my mobile phone screen within 1 hour before attempting to fall asleep.		0.85					
35	I use a blue-filter app on my computer screen within 1 hour before attempting to fall asleep.	LEBA16	0.88					
16	I wear blue-filtering, orange-tinted, and/or red-tinted glasses indoors during the day.	LEBA01		0.94				
17	I wear blue-filtering, orange-tinted, and/or red-tinted glasses outdoors during the day.	LEBA02		0.83				
36	I wear blue-filtering, orange-tinted, and/or red-tinted glasses within 1 hour before attempting to fall asleep.	LEBA03		0.79				
08	I spend 30 minutes or less per day (in total) outside.	LEBA04			-0.73			
09	I spend between 30 minutes and 1 hour per day (in total) outside.	LEBA05			-0.55			
11	I spend more than 3 hours per day (in total) outside.	LEBA07			0.71			
12	I spend as much time outside as possible.	LEBA08			0.69			
37	I purposely leave a light on in my sleep environment while sleeping.					0.69		
38	I use as little light as possible when I get up during the night.	LEBA17				-0.67		
39	I turn on the lights when I get up during the night.					0.55		
03	I look at my mobile phone screen immediately after waking up.	LEBA11					0.70	

Table 4. Continued.

Question no.	Question	Item no.	PA1	PA2	PA3	PA4	PA5	PA6
27	I use my mobile phone within 1 hour before attempting to fall asleep.	LEBA10					0.96	
28	I use my computer/laptop/tablet within 1 hour before attempting to fall asleep.						0.55	
46	I use tunable lights to create a healthy light environment.	LEBA19						0.62
04	I use an alarm with a dawn simulation light.	LEBA22				0.33		
13	I use sunglasses when I go outside in bright daylight.							0.46
14	I wear a visor or cap when I go outside in bright daylight.							0.50
25	I use a desk lamp when I do focused work.	LEBA21						0.39
26	I turn on my ceiling room light when it is light outside.					0.42		
42	I close curtains or blinds to prevent light from entering the bedroom if I want to sleep.					-0.50		
45	I use LEDs to create a healthy light environment.	LEBA20						0.48

Exploratory factor analysis was conducted using principal axis factoring with varimax rotation on a polychoric correlation matrix. PA1 = F4: Controlling environmental light before bedtime. PA2 = F1: Wearing blue light filters. PA3 = F2: Spending time outdoors. PA4 = New factor: Controlling light within the sleep environment. PA5 = F3: Using phone and smartwatch in bed. PA6 = F5: Using light in the morning and during daytime.

EFA: Exploratory factor analysis, PA: Principal axis

Discussion

The present study evaluated the validity, reliability, and cultural adaptation of the LEBA for Turkish-speaking populations. To our knowledge, this is the first study to examine the psychometric properties of the LEBA in a Turkish context and explore potential cross-cultural differences in light exposure behaviors. Overall, the findings provide partial support for the Turkish version of the LEBA as a useful tool while highlighting areas that may require refinement.

The expert review results provided strong evidence for content validity. Item-level and scale-level content validity indices indicated that the translated items were relevant and conceptually sound. However, one item related to the use of dawn simulation alarms was identified during the cognitive pretest as culturally problematic, reflecting limited familiarity with such devices in Türkiye. This suggests that while the LEBA captures a broad range of light exposure behaviors, certain items may need sociocultural adaptation to ensure conceptual clarity and applicability across diverse populations.

The CFA provided moderate support for the five-factor model proposed in the original LEBA validation. The respecified model, excluding items 05 and 06, demonstrated acceptable overall fit. These items exhibited negative factor loadings, indicating an inverse relationship with the intended latent construct. Although reverse coding could statistically address this issue when subscales are computed via summation, the persistent negative loadings suggested potential conceptual ambiguity rather than a simple scoring-direction artifact. Items 05 and 06 assess spending 0.5 to 3 hours outdoors and were designed to capture greater daylight exposure. However, in the present sample, the responses appeared to reflect perceptions of insufficient time spent outdoors, implying that the items may have been interpreted relative to subjective adequacy rather than absolute duration. This pattern may stem from cultural or contextual influences, including lifestyle differences, work–study schedules, and varying normative expectations regarding outdoor activity. Additionally, environmental factors such as urban living conditions and seasonal daylight variability may further affect item interpretation.

The internal consistency of Factor 5 (using light in the morning or during daytime) was notably low. From a measurement perspective, reduced reliability indicates increased measurement error and diminished precision of subscale scores; therefore, findings associated with this factor should be interpreted with caution. Several factors may account for these results. First, daytime light exposure behaviors are inherently context-dependent and strongly influenced by external constraints, including occupational schedules, commuting routines, and seasonal variability, which may introduce substantial situational noise. Second, the relatively young and student-dominated sample may have exhibited restricted variability in behaviors such as the use of desk lamps during focused work, attenuating inter-item correlations. Third, certain items within this factor, particularly those assessing the use of dawn simulation alarms and tunable lighting systems, may reflect technologies that are not yet widely adopted or culturally familiar in Türkiye, thereby reducing response consistency. Additionally, behaviors related to turning on lights immediately after waking may be influenced by seasonal daylight availability, which may be perceived as less critical at the latitudes represented in the present sample. Cultural differences in the perception, regulation, and prioritization of daytime light exposure, for example, norms regarding sunlight exposure, indoor lighting preferences, and the use of shading strategies, may have further contributed to heterogeneity in item interpretation. Finally, variability in participants' awareness of circadian health and attitudes toward light-related behavioral regulation may also have influenced response patterns.

Factor 1 (wearing blue light filters) distinguished individuals with ocular problems and those who reported using glasses with filtering features, providing initial evidence for the discriminant validity of the LEBA. Employment status also emerged as a significant correlate, with students and full-time employees reporting greater engagement in several light-related behaviors than unemployed participants did. These patterns support that occupational demands and daily routines influence light exposure behaviors and that the Turkish LEBA is a valid tool for measuring them (19,20).

Consistent with prior literature linking light exposure to sleep timing and quality, weak but significant associations emerged between the LEBA subscales and PSQI outcomes (21,22). Notably, phone and smartwatch use in bed was associated with poor sleep quality and delayed midsleep timing, supporting evidence that evening screen exposure contributes to circadian misalignment (23). Similarly, controlling environmental light before bedtime was associated with daytime dysfunction, suggesting that light-related behaviors may extend their influence on daytime functioning (24). However, the effect sizes were generally small, indicating that while the LEBA captures meaningful behaviors, sleep quality is influenced by multiple additional factors.

According to the original LEBA development study, the factorial characteristics of light exposure behavior are the same for both native and non-native English speakers (14). However, significant differences in LEBA scores were observed across language groups in this study. Malaysians scored higher on most factors, except for Factor 2 (outdoor light exposure), in which both Turkish groups scored higher and showed convergence. These differences may reflect sociocultural norms that shape outdoor activity preferences, potential linguistic or response biases in the self-report instrument, or the possibility that the construct of light exposure behavior is not entirely equivalent across cultures. Moreover, in one study, correlations between LEBA items and objective light exposure metrics emerged in Switzerland but were absent in Malaysia, suggesting that subjective reports of light exposure are influenced by cultural context and may not directly correspond to objectively measured light exposure (16). Previous literature also indicates that ethnicity may modulate light sensitivity; for instance, greater light-induced melatonin suppression has been observed in Caucasians than in Asian participants, and lower nocturnal melatonin excretion has been reported in African Americans than in European Americans (25,26). Collectively, these results highlight the importance of cross-cultural evaluation in understanding light exposure behaviors.

EFA of the broader 48-item pool revealed a six-factor structure that differed from that of the original five-factor LEBA model. The emergence of an additional factor in the EFA does not necessarily contradict the CFA findings but rather suggests that light-exposure-related behaviors may be organized somewhat differently within this cultural context. Importantly, the remaining five factors were thematically consistent with the original LEBA framework, and the majority of the items were retained. The most notable thematic deviations involved the absence of smartwatch-related items and the inclusion of behaviors related to wearing hats or sunglasses during bright outdoor daytime conditions. These differences further support the potential influence of contextual factors, such as socioeconomic accessibility to wearable technologies and seasonal daylight variability. From a practical standpoint, we do not recommend replacing the theoretically grounded five-factor LEBA structure with the exploratory six-factor solution without replication in independent Turkish cohorts. Instead, the EFA findings should be considered hypothesis-generating, highlighting potential culture-sensitive behavioral dimensions that warrant further investigation.

Notably, the new factors identified in the EFA clustered around behaviors related to managing light within the sleep environment, including leaving lights on during sleep, controlling light exposure during nighttime awakenings, and regulating the entry of external light via curtains or blinds. This pattern may reflect sociocultural influences specific to the Turkish context. Possible interpretations include culturally shaped norms regarding privacy within the home, security-related behaviors, comfort-seeking strategies, fear or avoidance of darkness, and household practices concerning energy use. Additionally, variability in awareness of circadian health and attitudes toward light-related behavioral regulation may contribute to these response patterns. The identification of this factor suggests that sleep-environment light management may represent a meaningful, context-dependent behavioral construct not fully captured by the original LEBA framework.

Satellite-based high-resolution analyses have revealed an increase in the total luminous energy emitted from Türkiye into space in recent years (27-29). Given that Türkiye is among the countries at heightened risk for nocturnal light pollution and a high incidence of sleep disorders, it is crucial to identify culturally specific dimensions and adapt standardized assessment tools accordingly (30). In clinical and public health contexts, the Turkish version of the LEBA may serve as a valuable instrument for detecting maladaptive light exposure patterns, informing interventions to improve sleep and circadian health, and supporting research on light-related risks for public health (9).

Study Limitations

Several limitations should be acknowledged. First, the sample included relatively young individuals, most of which were students; thus, the generalizability of the findings may be limited, particularly to older adults. Age-related differences in lifestyle, occupational demands, sleep patterns, and light exposure behaviors may influence responses to the LEBA, and future studies should include more age-diverse samples. Second, reliance on self-report measures introduces the possibility of recall and social desirability bias. Test–retest reliability was not examined. Integrating objective light exposure measures, such as actigraphy with light sensors, would strengthen the evidence of validity. Although the data were collected from three different regions in Türkiye, they may not fully reflect the cultural diversity of light-related practices across the country. Replication in independent samples is necessary to confirm the

stability of the factor structure. Additionally, external data from Malaysia were collected via an 18-item LEBA, which differed from the version applied in this study.

Conclusions

In summary, the Turkish version of the LEBA demonstrated strong content validity, acceptable factorial validity, and adequate overall reliability. The subscale "Using light in the morning or during daytime" showed low reliability and should therefore be interpreted cautiously. The second factor, "Spending time outdoors," should be calculated excluding specified items. The emergence of a culturally specific factor structure underscores the importance of cultural adaptation in LEBA. We do not recommend adopting an exploratory six-factor structure without replication in independent samples; however, the collection of an additional question on lighting preferences during sleep could be beneficial in Türkiye. Associations with sleep and health outcomes provide preliminary evidence for construct validity and highlight the importance of light exposure in sleep and circadian health. The Turkish LEBA represents a promising tool for advancing research and practice in circadian public health.

Ethics

Ethics Committee Approval: The İzmir Institute of Technology Research Ethics Committee reviewed and approved the protocol (approval no: 26.01.2024-01/06, date: 26.01.2024).
Informed Consent: Each participant provided written informed consent before participating in the study.

Footnotes

Authorship Contributions

Concept: A.D., S.K.K., P.C., Design: A.D., Z.K., İ.A., B.Y., S.K.K., M.N.Ö.Ü., P.C., Data Collection or Processing: A.D., Z.K., İ.A., B.G., S.K.K., P.C., Analysis or Interpretation: A.D., B.G., Literature Search: A.D., Z.K., İ.A., Writing: A.D., Z.K., İ.A., B.G., S.K.K., B.Y., M.N.Ö.Ü., P.C.
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