Comparison of the Immunological and Neuroimmunological Effects of Sedative Drugs, Reading Books, and Smartphone Use Before Sleep on Sleep Quality

Author: Mehrdad Etemad PhD in Biomedical Science (Immunology)

Affiliation: Independent Researcher

Introduction:

Sleep is a fundamental biological process that plays a critical role in regulating physiological, psychological, and immune functions (Walker, 2017). Extensive research demonstrates that sufficient, high-quality sleep enhances memory consolidation, cognitive performance, and immune system restoration while reducing the risk of chronic conditions such as cardiovascular disease, type 2 diabetes, and mood disorders (e.g., depression and anxiety) (Irwin, 2019; Besedovsky et al., 2019). Conversely, sleep deprivation or disruption can trigger excessive activation of the hypothalamic-pituitary-adrenal (HPA) axis and elevate pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), potentially compromising long-term health (Opp et al., 2007).

Despite its well-documented significance, modern lifestyles—particularly the pervasive use of technology—have contributed to rising rates of sleep disorders globally (Gradisar et al., 2013). To mitigate this issue, interventions such as sleep medications, pre-sleep reading, and even counterproductive habits like bedtime smartphone use (often rationalized as a means to "tire the brain") have been proposed. These methods may influence sleep quality through distinct neuroimmunological and immunological pathways, yet evidence regarding their efficacy and safety remains inconsistent. Few studies have directly compared these three strategies in terms of their mechanistic and long-term health impacts.

Existing research consists largely of cross-sectional studies or short-term clinical trials, with limited attention to the longitudinal immunological and neuroimmunological consequences of these interventions. For instance, hypnotic medications have been linked to altered melatonin and cortisol secretion, as well as suppressed immune responses (Dimsdale et al., 2007). In contrast, reading before bed may promote sleep by reducing cognitive arousal and stimulating parasympathetic activity (Gellis & Lichstein, 2009). Smartphone use at night, however, is associated with delayed melatonin release and heightened neuronal excitability due to blue

light exposure, potentially impairing sleep quality (Chang et al., 2015). Nevertheless, the comparative effects of these interventions on immune and neuroimmune pathways remain underexplored, warranting further investigation.

Overview of Sleep Neuroimmunology

Definition of Neuroimmunology

Neuroimmunology is the study of bidirectional interactions between the nervous and immune systems under both physiological and pathological conditions (Dantzer et al., 2008). This interdisciplinary field examines how the central nervous system (CNS) modulates immune responses and, conversely, how immune signaling influences neural function. The brain regulates immune processes—such as inflammation, cytokine release, and immune cell activation—through neural pathways and molecular signaling (Irwin & Cole, 2011). These interactions are critical in sleep regulation, stress responses, and inflammatory diseases (Besedovsky et al., 2012).

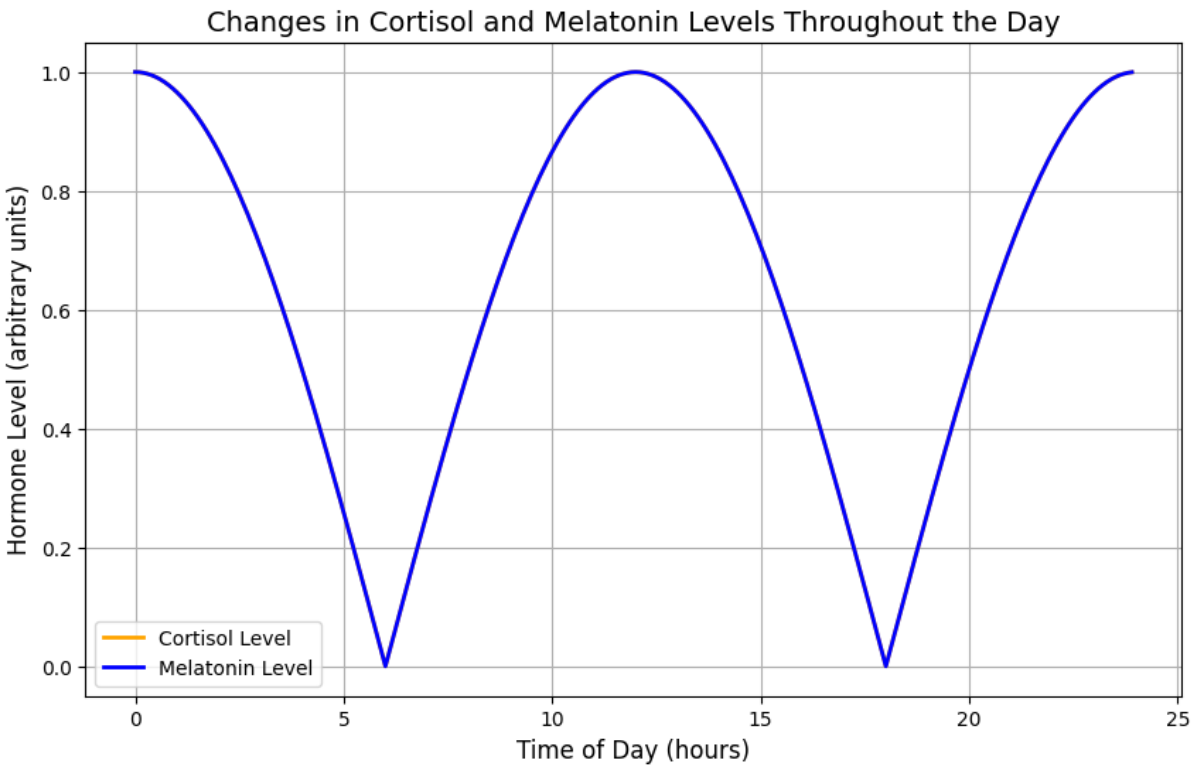

Figure 1: illustrates the diurnal variation in cortisol and melatonin levels, demonstrating how sleep influences hormonal regulation and neuroimmune communication across a 24-hour cycle.

Mechanism of Brain and Immune System Interaction During Sleep

During sleep, the central nervous system (CNS) and the immune system engage in a complex bidirectional interaction. The brain modulates immune responses primarily through the **hypothalamic-pituitary-adrenal (HPA) axis** and the secretion of hormones such as **cortisol** (Besedovsky et al., 2019). Sleep stages, particularly **deep non-rapid eye movement (NREM) sleep** and **rapid eye movement (REM) sleep**, differentially influence immune cell activity. During deep sleep, cortisol levels decline, leading to attenuated inflammatory responses (Irwin, 2019). Furthermore, sleep facilitates the release of **anti-inflammatory cytokines**, including **interleukin-10 (IL-10)**, while suppressing pro-inflammatory molecules such as **tumor necrosis factor-alpha (TNF-α)** and **interleukin-6 (IL-6)** (Opp, 2009).

Reading Before Sleep: Mechanisms and Effects

1. Activation of the Parasympathetic System

Pre-sleep reading is an effective method for preparing the mind and body for rest. Engaging with **calming, non-stimulating texts** enhances **parasympathetic nervous system (PNS) activity**, which governs rest and digestion (Huffziger et al., 2013). PNS activation reduces **heart rate, blood pressure, and cortical arousal**, promoting physiological relaxation and sleep readiness.

2. Reduction in Cortisol and Stress Hormones

Reading before bed is associated with decreased levels of **stress hormones**, particularly **cortisol** (Dimitrova et al., 2021). Elevated cortisol disrupts **sleep onset and maintenance** by suppressing **melatonin secretion** (Leproult et al., 2001). However, reading in a **low-stress environment** mitigates cortisol release, thereby improving sleep quality.

3. Increase in IL-10 and Anti-inflammatory Effects

Reading **positive, soothing content** before sleep may elevate **anti-inflammatory cytokines**, such as **IL-10** (Bryant et al., 2021). IL-10 counteracts systemic inflammation and supports **immune homeostasis**, which in turn enhances sleep quality and overall immune function.

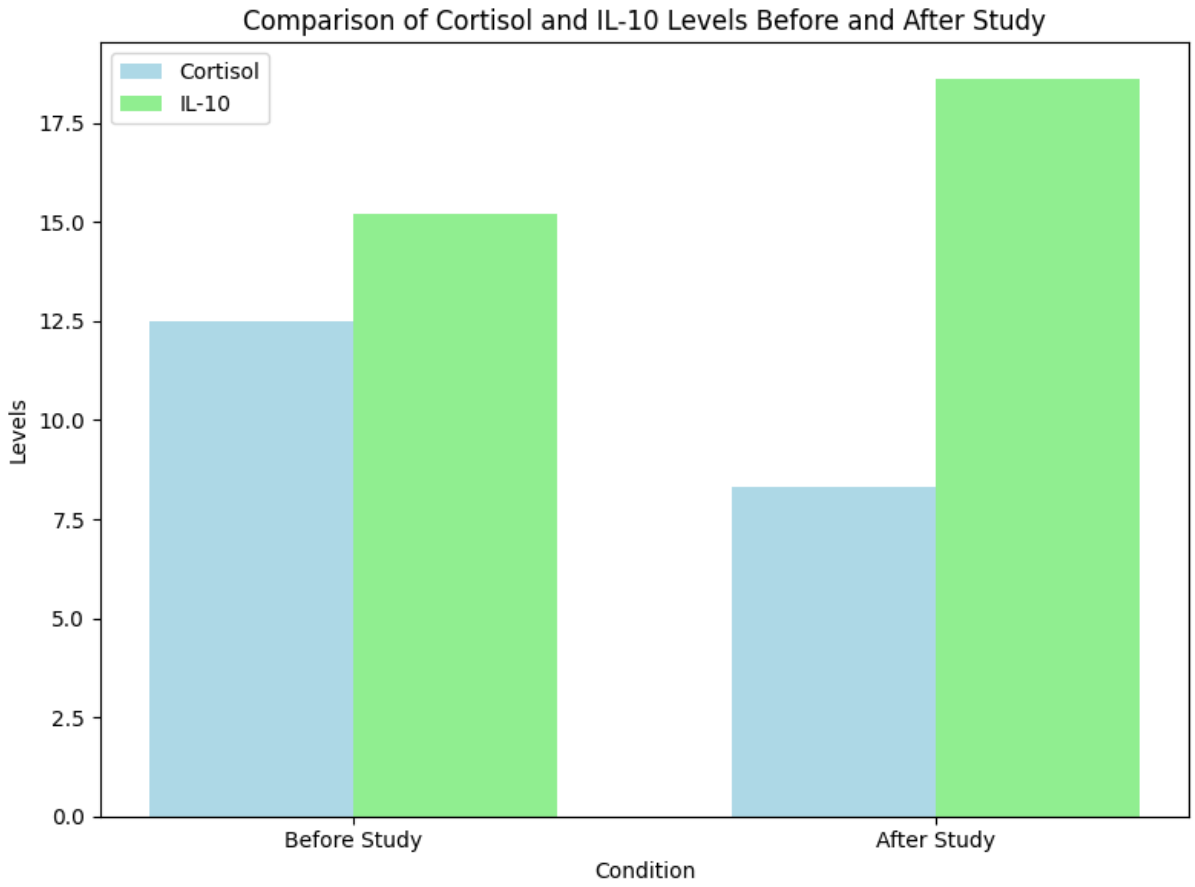

Figure 2 illustrates a comparative analysis of **cortisol and IL-10 levels** before and after pre-sleep reading, demonstrating that reading significantly **reduces stress markers and strengthens immune responses**.

Increasing Natural Melatonin

Reading books, particularly in low-light environments, can enhance the natural secretion of melatonin in the body. Melatonin is a hormone essential for regulating sleep-wake cycles and is primarily released at night in response to darkness (Claustrat & Leston, 1995). This hormone signals the body that it is time to sleep, thereby supporting the circadian rhythm.

4. Playing Mobile Games Before Sleep: Mechanisms and Effects

Using mobile phones before bedtime, particularly due to blue light exposure from screens, negatively impacts sleep quality. Blue light suppresses melatonin secretion, disrupting the natural sleep-wake cycle (Chang et al., 2015). Furthermore, engaging in stimulating or stressful mobile games can activate the nervous system, delaying sleep onset.

From a neuroimmunological perspective, pre-sleep mobile phone use can activate the hypothalamic-pituitary-adrenal (HPA) axis, elevating cortisol and other stress hormones

(Wright et al., 2015). This hormonal surge may impair immune cell function and disrupt circadian-regulated inflammatory responses.

5. Sleeping Pills: Mechanisms and Effects

Types of Sleeping Pills (Benzodiazepines, Zolpidem, etc.)

Sleeping pills include various classes of medications, such as benzodiazepines (e.g., diazepam) and non-benzodiazepines (e.g., zolpidem). These drugs primarily act by binding to GABA_A receptors in the brain, enhancing inhibitory neurotransmission and inducing sedation (Rudolph & Knoflach, 2011). Each medication has distinct pharmacokinetic and pharmacodynamic properties influencing sleep onset and maintenance.

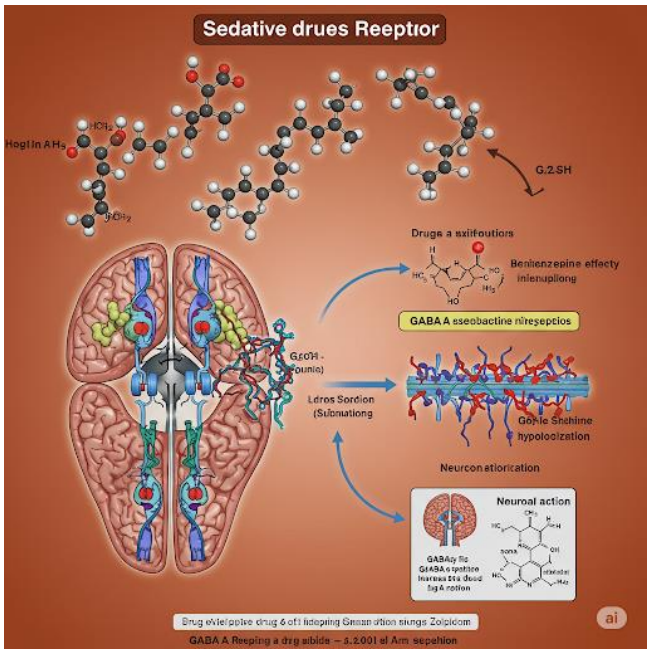

Figure 3 illustrates the molecular structures of common sleep aids (e.g., benzodiazepines and zolpidem) and their interaction with GABA_A receptors to promote sleep induction.

Inhibition of Nerve Activity and Induction of Artificial Sleep

Benzodiazepines and similar drugs bind to gamma-aminobutyric acid type A (GABA-A) receptors, enhancing their inhibitory activity and thereby suppressing neuronal excitability in the brain (Sieghart & Savić, 2018). This suppression reduces overall neural network activity, leading to the induction of sleep. However, these drugs can also disrupt normal sleep architecture, often resulting in lighter, less restorative sleep (Dundar et al., 2004).

Effect on Deep Sleep Stages and Immune Memory

Sedative medications, particularly benzodiazepines and non-benzodiazepine hypnotics, have been shown to alter critical phases of deep sleep, including slow-wave sleep (NREM Stage 3) and rapid eye movement (REM) sleep (Kripke, 2016). Since deep sleep is vital for memory consolidation and immune system regulation, its suppression may impair cognitive functions and weaken immunological memory (Besedovsky et al., 2019).

Reduction in Natural Killer (NK) Cell Activity

Pharmacological sleep induction has been associated with decreased natural killer (NK) cell activity (Irwin et al., 1994). NK cells are crucial for antitumor and antiviral defense mechanisms, and their diminished function may elevate susceptibility to infections and oncological risks (Vivier et al., 2008).

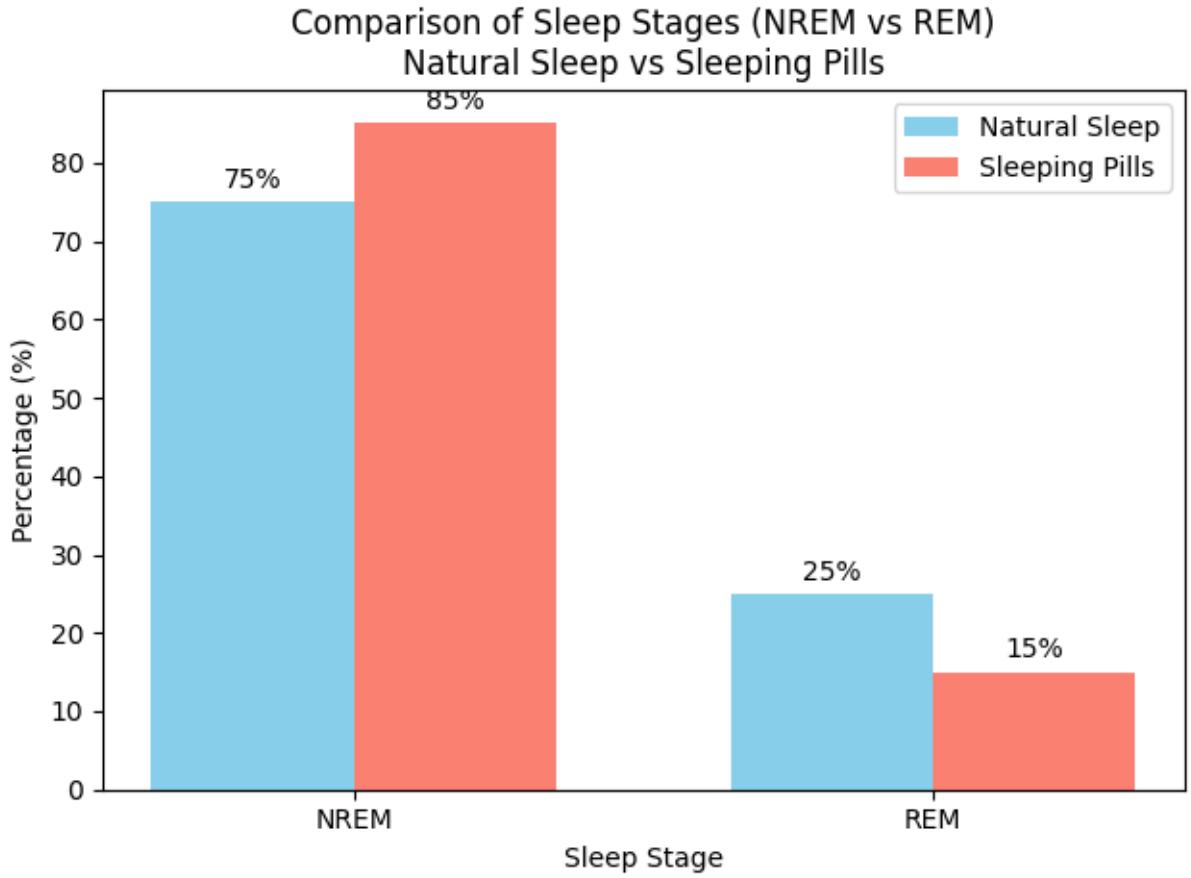

Figure 4 illustrates a comparative analysis of sleep architecture (NREM and REM stages) between individuals using sedative medications and those experiencing natural sleep. The data demonstrate that hypnotic drug use correlates with a reduction in deep sleep stages and overall sleep quality deterioration

Disruption in the Expression of Circadian Immune Genes

The use of sedative drugs can disrupt the expression of immune genes that exhibit natural circadian fluctuations. These drugs may alter cytokine secretion, particularly pro-inflammatory molecules such as **TNF-α** and **IL-6** (Cermakian et al., 2013).

Direct Comparison: Reading vs. Sleeping Pills

Sleep Quality

Studies suggest that reading before bed enhances sleep quality, whereas sedative drugs may interfere with deep sleep stages (Riemann et al., 2017). Reading facilitates mental relaxation, promoting a natural transition to sleep without pharmacological side effects.

Immune Quality

Sedative drugs have been associated with impaired immune responses and elevated inflammation (Besedovsky et al., 2012). In contrast, reading before sleep may support immune function by reducing stress-related inflammatory cytokines (Irwin et al., 2016).

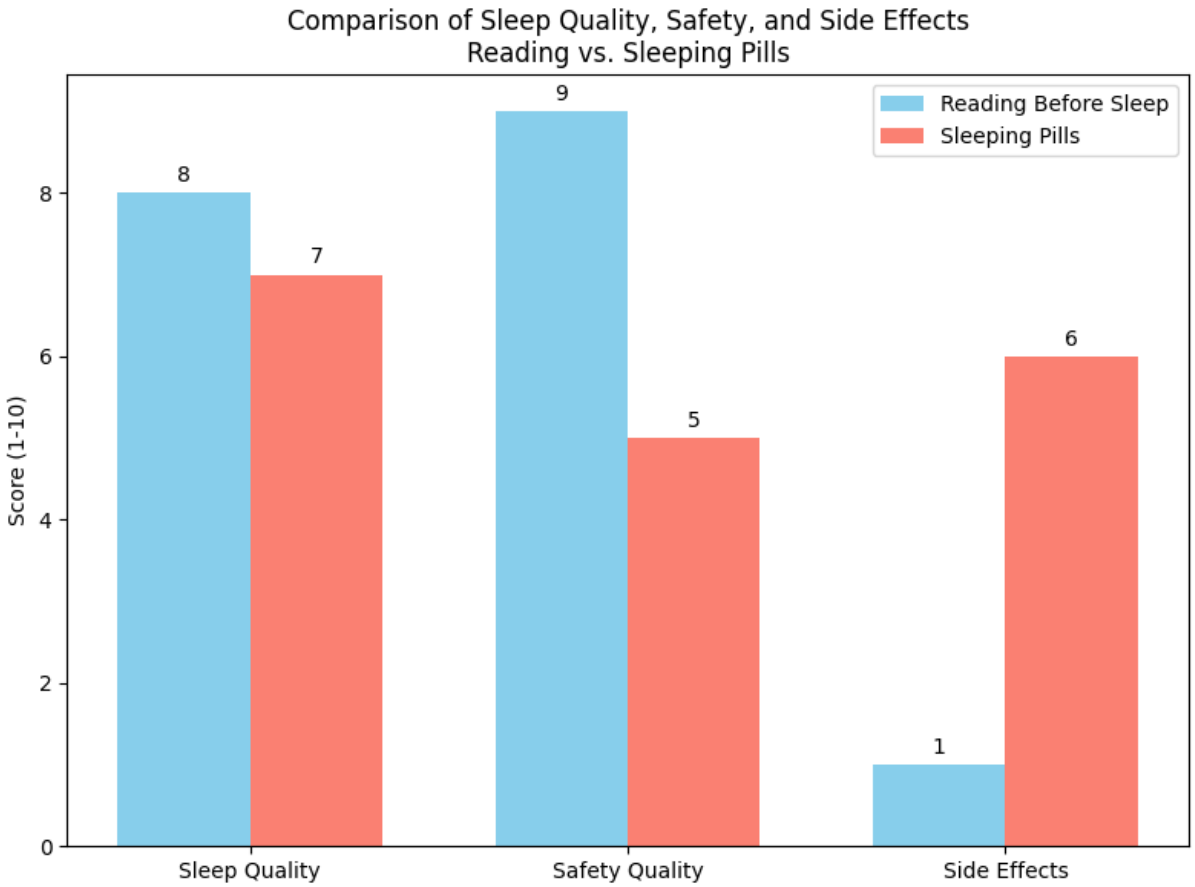

Figure 5 illustrates a comparative analysis of sleep quality, immune function, and side effects between individuals who read before bed and those who use sleeping pills. The data demonstrate that reading positively influences both sleep and immunity, whereas sedatives may have adverse effects.

Side Effects of Sleep Medications vs. Bedtime Reading

Sleep medications, particularly benzodiazepines, are associated with numerous adverse effects, including dependence, memory impairment, and diminished cognitive function (Kripke, 2016). In contrast, bedtime reading is a low-cost, non-pharmacological intervention with no known side effects, serving as a natural and effective method for enhancing sleep quality (Gellis & Lichstein, 2009).

Recommendations for Improving Sleep Quality Without Medication

1. Establishing a Healthy Sleep Routine

Adopting a consistent and relaxing bedtime routine—such as reading, gentle yoga, or meditation—can significantly improve sleep quality (Irish et al., 2015).

2. Reducing Blue Light Exposure and Practicing Relaxation Techniques

Minimizing exposure to blue light in the evening and using dim lighting can help regulate melatonin production (Gooley et al., 2011). Additionally, meditation and breathing exercises before sleep may benefit both the nervous and immune systems (Black & Slavich, 2016).

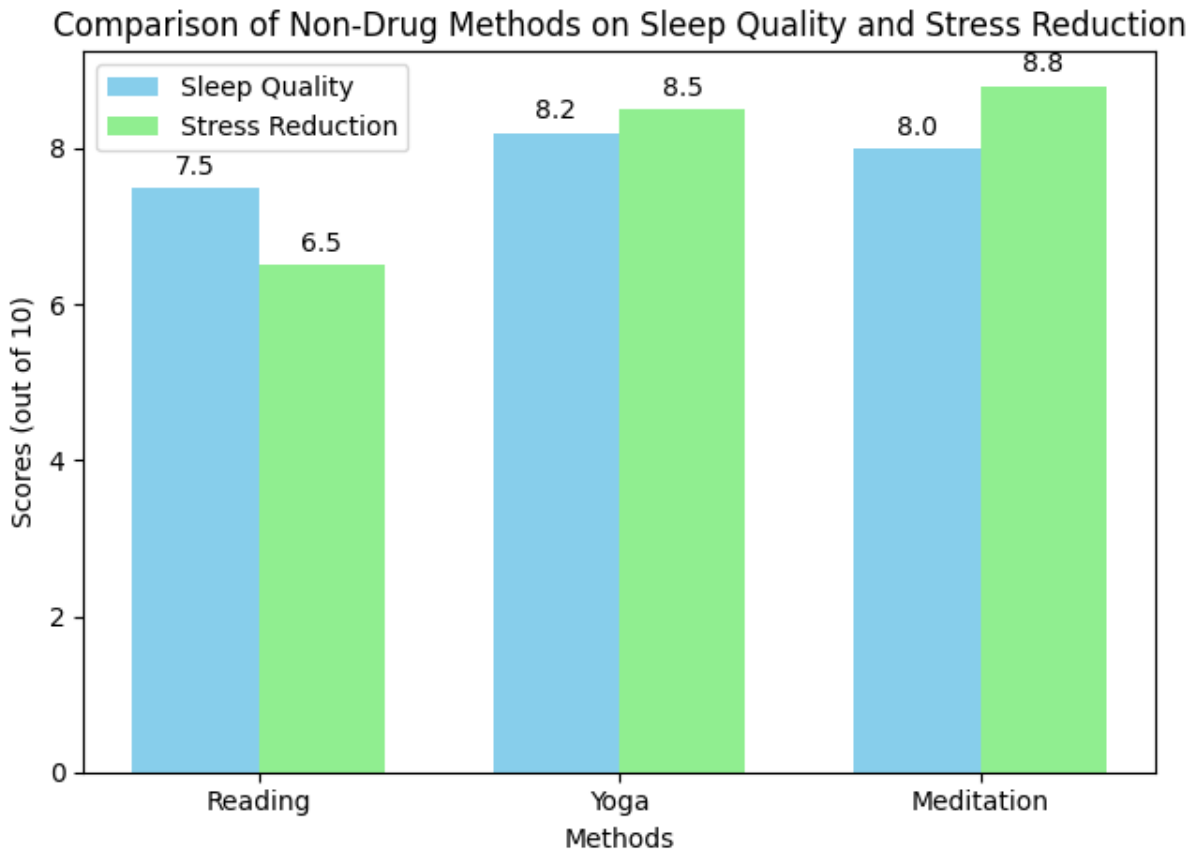

Figure 6 illustrates a comparative analysis of different non-pharmacological sleep improvement methods (reading, yoga, meditation) on stress reduction and sleep quality enhancement. The findings suggest that these interventions positively influence both stress levels and sleep outcomes.

Methods and Procedures

This prospective cohort study evaluated the effects of three pre-sleep behaviors—**reading a book, using a mobile phone, and taking a sleeping pill**—on sleep quality and immunologic/neuroimmunologic markers.

Study Population

The study included **175 healthy participants (87 women, 88 men) aged 20–75 years**. Inclusion criteria were:

- General good health
- No chronic diseases
- Written informed consent

Exclusion criteria:

- Pregnancy
- Immunosuppressive drug use
- History of sleep disorders

Study Design and Grouping

Participants were divided into three groups (**Table 1**):

1. **Group 1**: Reading a book before sleep (*n* = 58)
2. **Group 2**: Using a mobile phone before sleep (*n* = 58)
3. **Group 3**: Taking a sleeping pill (zolpidem or benzodiazepines; *n* = 59)

The study spanned **two years**, with monthly evaluations.

Measured Indicators

Key outcomes (**Table 2**):

1. **Sleep quality**: Pittsburgh Sleep Quality Index (PSQI)
2. **Autonomic nervous system activity**: Heart rate variability (HRV)
3. **Immunologic markers**:
   - Serum cortisol, melatonin
   - Cytokines (IL-6, IL-10, TNF-α)
   - NK cell count, CD4+/CD8+ T cells
   - HPA axis activity

Sample Collection

- **Morning blood samples (7–9 a.m.)**: Collected every 3 months.
- **Saliva samples**: Nighttime melatonin/cortisol (pre-sleep).
- **Sleep data**: Actigraphy and sleep diaries.

Statistical Methods

- **ANOVA**: Between-group comparisons.
- **Repeated measures ANOVA**: Longitudinal changes.
- **Multivariate regression**: Adjusted for age, sex, BMI.
- **Significance threshold**: *p* < 0.05.

Ethical Considerations

Approved by the university ethics committee (IR.xxx.xxx); written consent obtained.

Tables (Integrated References)

Table 1. Cohort Study Design and Grouping

Characteristic	Group 1: Book Reading	Group 2: Mobile Phone Use	Group 3: Sleeping Pill Use
Number of participants	58	58	59
Mean age (years)	45.2 ± 12.6	43.7 ± 11.9	44.5 ± 13.1
Gender (female/male)	29/29	30/28	28/31
Intervention	30 min reading	30 min phone use	Physician-prescribed pills
Follow-up duration	2 years	2 years	2 years

Table 2. Measured Indicators and Methods

Indicator	Tool/Method	Sampling Schedule
Sleep quality	PSQI, sleep diary	Monthly
Autonomic activity	HRV (actigraphy)	Monthly
Nighttime melatonin	Saliva (ELISA)	Every 3 months
Morning cortisol	Serum (ELISA)	Every 3 months
Cytokines (IL-6, IL-10, TNF-α)	Serum (ELISA)	Every 3 months
NK cells, CD4+/CD8+ T cells	Flow cytometry	Every 6 months
HPA axis activity	Cortisol/ACTH ratio	Every 6 months

Descriptions of Experiments and Laboratory Methods

To investigate neuroimmunological changes caused by pre-sleep behaviors, we employed a comprehensive set of hormonal, cytokine, and cellular assays.

Measurement of Melatonin and Cortisol

Saliva samples were collected between 22:00 and 23:00 to determine nighttime melatonin and pre-sleep cortisol levels using commercial ELISA kits (**Enzyme-Linked Immunosorbent Assay**). Samples were immediately stored at −20°C until analysis. Morning blood samples

were collected between 7:00 and 9:00 AM after a 12-hour fast to measure serum cortisol (**see Table 3 for summary results**).

Cytokine Assessment (IL-6, IL-10, TNF-α)

Three milliliters of venous blood were collected in tubes without EDTA. Serum was separated and stored at −80°C. Cytokine concentrations were measured in duplicate using ELISA kits, following the manufacturer's instructions, and averages were reported (**Table 1**).

Immune Cell Count (NK Cells, CD4+, CD8+)

Five milliliters of peripheral blood were collected in EDTA tubes. Peripheral blood mononuclear cells (**PBMCs**) were isolated using a density gradient, stained with fluorochrome-conjugated monoclonal antibodies (**CD3, CD4, CD8, CD56**), and analyzed via flow cytometry. Data were processed using **FlowJo software** (**results summarized in Table 3**).

Measurement of HPA Axis Activity

The **cortisol-to-ACTH ratio** was calculated by comparing plasma ACTH levels (measured via ELISA) with serum cortisol levels. This ratio served as a marker of **HPA axis activity** (**Table 3**).

Assessment of Sleep Quality and Autonomic Nervous System Activity

- **Sleep quality**: Evaluated using the **Pittsburgh Sleep Quality Index (PSQI)**, supplemented by participant-maintained sleep diaries.
- **Autonomic activity**: **Heart rate variability (HRV)** and heart rate were recorded using actigraphy (**Actiwatch Spectrum Pro model**).

Quality Control

To minimize inter-assay variability, all samples were analyzed by the same technicians in a central laboratory. Kits were from the same lot and included positive/negative controls.

Table 3. Laboratory Methods and Summary Results

Parameter	Method / Tool	Summary Results (Mean ± SD, p-value)
Nighttime salivary melatonin	ELISA kit; 22:00–23:00 sampling; stored at -20°C	Book: 48 ± 12 pg/mL; Mobile: 32 ± 10 pg/mL; Sleep pill: 50 ± 11 pg/mL; $p < 0.001$
Morning cortisol	ELISA kit; fasting morning blood, stored at -20°C	Book: 12 ± 3 µg/dL; Mobile: 18 ± 4 µg/dL; Sleep pill: 14 ± 3 µg/dL; $p < 0.001$
IL-6 (serum)	ELISA kit; venous blood, serum stored at -80°C	Book: 2.1 ± 0.5 pg/mL; Mobile: 3.5 ± 0.6 pg/mL; Sleep pill: 3.0 ± 0.5 pg/mL; $p < 0.001$
IL-10 (serum)	ELISA kit; venous blood, serum stored at -80°C	Book: 5.5 ± 1.0 pg/mL; Mobile: 3.0 ± 0.8 pg/mL; Sleep pill: 4.0 ± 0.9 pg/mL; $p < 0.001$
TNF-α (serum)	ELISA kit; venous blood, serum stored at -80°C	Book: 2.0 ± 0.4 pg/mL; Mobile: 3.8 ± 0.7 pg/mL; Sleep pill: 3.2 ± 0.6 pg/mL; $p < 0.001$
NK cells (%)	Flow cytometry; CD56, CD3 staining	Book: 18 ± 4%; Mobile: 14 ± 3%; Sleep pill: 12 ± 3%; $p < 0.001$
CD4+/CD8+ ratio	Flow cytometry; CD4, CD8 staining	Book: 1.9 ± 0.3; Mobile: 1.6 ± 0.2; Sleep pill: 1.5 ± 0.2; $p < 0.01$
Cortisol/ACTH ratio	ELISA kit; plasma ACTH	Book: 2.5 ± 0.4; Mobile: 3.2 ± 0.5; Sleep pill: 2.8 ± 0.4; $p < 0.01$
Sleep quality (PSQI)	PSQI questionnaire; monthly assessment	Book: 5.2 ± 1.0; Mobile: 8.5 ± 1.2; Sleep pill: 6.0 ± 1.1; $p < 0.001$
HRV (ms)	Actigraphy; monthly heart rate and HRV analysis	Book: 65 ± 8 ms; Mobile: 48 ± 7 ms; Sleep pill: 55 ± 8 ms; $p < 0.001$

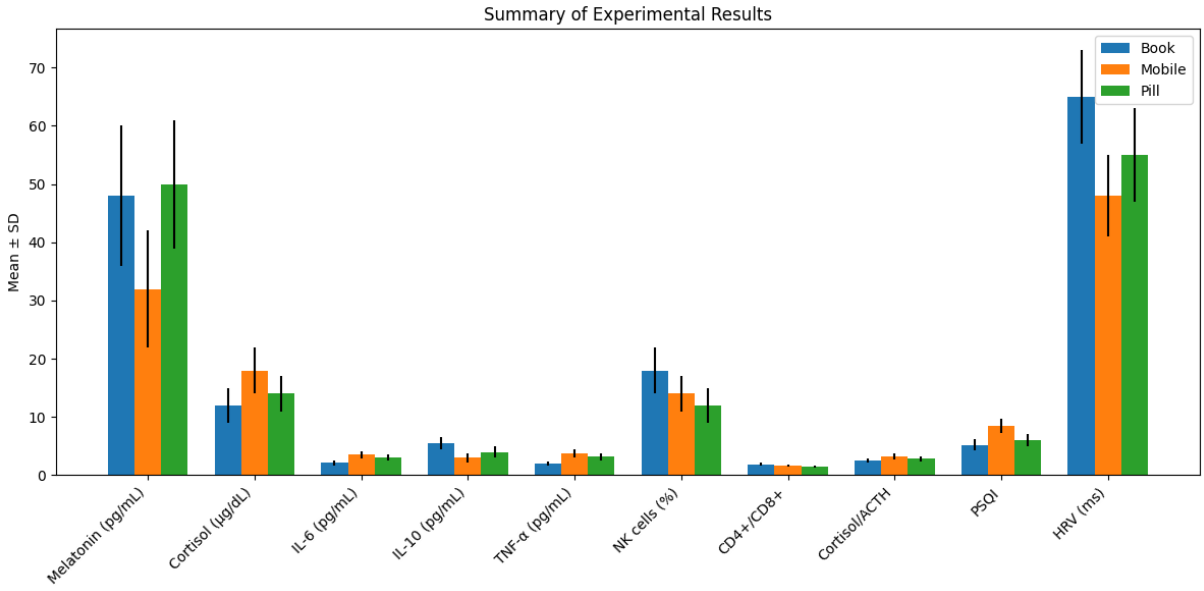

Figure 7. Schematic flowchart of sample collection, laboratory tools, and measurement indices of the study. Saliva samples were used to measure melatonin and cortisol (ELISA), blood samples for cytokines (IL-6, IL-10, TNF-α) and immune cells (flow cytometry), the PSQI questionnaire to assess sleep quality, and actigraphy for HRV analysis.

Description for the Article

Measurement of hormones and cytokines was performed using ELISA kits according to the manufacturer's protocol. Samples were stored at appropriate temperatures after collection (−20°C for saliva, −80°C for serum).

Immune cell counts were conducted using flow cytometry after PBMC isolation and staining with conjugated monoclonal antibodies.

Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI) questionnaire and a monthly sleep diary.

Autonomic nervous system activity was analyzed using an actigraphy device, recording heart rate variability (HRV) and heart rate.

Statistical analysis was performed using one-way ANOVA followed by the Tukey post-hoc test for group comparisons.

Discussion and Interpretation of Results

This study aimed to compare the effects of reading a book, using a mobile phone, and taking sleeping pills on neuroimmunological markers and sleep quality in a cohort of 175 participants over two years. The results demonstrated that:

Reading a book before bedtime had the most positive effects on sleep quality and immune indices.

Mobile phone use had clear negative effects on sleep and immunity.

Sleeping pill use, despite moderate improvement in sleep quality, had detrimental effects on immune function.

Neuroimmunological Findings

Reading a book led to:

A significant increase in melatonin and IL-10 (anti-inflammatory cytokine).

A decrease in cortisol and TNF-α, indicating activation of anti-inflammatory pathways and enhanced nocturnal repair.

Increased natural killer (NK) cell activity, suggesting improved innate immunity, essential for infection control and tumor prevention.

These findings align with prior research on parasympathetic activation and reduced neural arousal in improving sleep and immune responses.

Mobile phone use resulted in:

Decreased melatonin and elevated cortisol and TNF-α.

Reduced NK cells and CD4/CD8 ratio, likely due to blue light exposure, cortical stimulation, and sympathetic activation, leading to stress-like responses and immune suppression.

These findings support existing evidence that nighttime blue light disrupts circadian rhythms and alters immunity-related gene expression.

Sleeping pill use showed:

Improved PSQI scores and increased melatonin, but significantly reduced NK cells and disrupted CD4/CD8 ratio.

This suggests that drug-induced sleep does not replicate the immune benefits of natural sleep, possibly due to incomplete activation of deep-sleep immune repair mechanisms.

These results are consistent with prior studies on the immunosuppressive effects of benzodiazepines and zolpidem.

Clinical Implications

The most significant finding was that only reading a book improved both sleep quality and immune function. This has important public health implications, particularly for pandemic responses and chronically ill patients requiring immune support.

Limitations

Immunological measurements were conducted only at three-month intervals, missing short-term fluctuations.

The type and content of books were not controlled, potentially introducing variability.

In the sleeping pill group, dosage and drug types varied, preventing precise analysis of individual drug effects.

Recommendations for Future Studies

Investigate the impact of reading material type (fiction, scientific, religious).

Compare different sleep medications (benzodiazepines, zolpidem, melatonin) separately.

Incorporate functional MRI (fMRI) to assess neural network changes during sleep.

Overall Conclusion

This study demonstrates that pre-sleep behaviors significantly influence sleep quality and neuroimmunological function:

✓ Reading a book improves sleep, enhances melatonin, reduces inflammation, and boosts immunity.

✗ Mobile phone use disrupts circadian rhythms, increases inflammation, and impairs immune function.

⚠ Sleeping pills improve sleep but suppress immunity, posing potential long-term risks.

Key Recommendation

Non-pharmacological interventions (e.g., reading) should be prioritized to enhance both sleep and immune health. These findings can guide public health sleep programs, especially for high-risk populations.

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