

Analysis of the impact of sleep duration and quality on tear-film in adults with complementary validation of a mobile healthy-lifestyle application

Aneta Buchtelova^{1,2}, Eliska Mikulaskova², Vojtech Malina¹, Karla Mothejllova³, Milada Luisa Sedivcova¹, Jan Kaspar¹, Karel Hana¹

Aims. This study aimed to investigate the relationship between sleep parameters and tear film hydration assessed by tear meniscus height (TMH), and to evaluate the usability of a mobile application for remote behavioral and physiological data collection.

Methods. Eighteen healthy adults (mean age 29.6 ± 6.4 years) completed five consecutive days of linked measurements combining objective sleep tracking (using Fitbit Charge 4) and slit-lamp imaging of TMH under standardized conditions. Behavioral data on stress, caffeine intake, and smartphone use were collected via a custom mobile application, whose usability was assessed by the System Usability Scale (SUS).

Results. Ninety valid daily records were analyzed. TMH correlated positively with total sleep time ($r=0.31$, $P=0.0033$), REM sleep duration ($r=0.28$, $P=0.0066$), and sleep quality ($r=0.32$, $P=0.0024$). Each additional 30 min of REM sleep increased TMH by approximately 0.014 mm. Short sleepers (<6 h) showed significantly lower TMH (0.121 mm) than normal sleepers (≥ 6 h; 0.153 mm; $P=0.0023$). Stress and evening smartphone use were associated with reduced sleep quality and lower TMH. The mobile application achieved excellent usability ($SUS = 87 \pm 6$).

Conclusions. Our data indicate that sleep duration and architecture play essential roles in maintaining tear film stability. Behavioral habits that impair sleep continuity may thus directly affect ocular surface physiology. The combined use of wearable sensors and mobile self-reporting represents a practical approach to studying the interaction between sleep and ocular physiology.

Association Between Sleep Architecture and Tear Film Hydration

Sleep plays an essential role in maintaining ocular surface homeostasis.

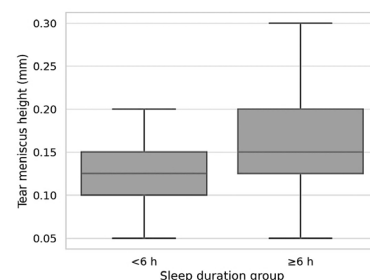
Behavioral and lifestyle factors – such as stress, evening screen exposure, and caffeine use – may disturb sleep quality and indirectly affect tear film stability.

This study combined wearable sleep tracking and mobile self-reporting to examine how total sleep duration and REM phase of sleep influence tear meniscus height (TMH), a clinical marker of tear film volume.



Participants sleeping less than 6 hours per night showed significantly lower TMH (0.121 ± 0.039 mm) compared with normal sleepers (0.153 ± 0.044 mm; $P=0.0023$).

Longer total sleep time and greater REM duration correlated positively with total sleep time ($r=0.31$, $P=0.0033$), REM sleep duration ($r=0.28$, $P=0.0066$), and sleep quality ($r=0.32$, $P=0.0024$).



Conclusion: Adequate sleep duration and sufficient REM phases support ocular surface hydration and tear film stability. Wearable and mobile tools provide a practical way to study sleep–eye interactions in real-world settings.

Buchtelova A. et al., doi: 10.5507/bp.2026.

Graphical Abstract

Biomedical Papers

<https://biomed.papers.upol.cz>

Key words: tear meniscus height, REM sleep, sleep quality, ocular surface, remote data collection

Received: November 13, 2025; Revised: January 27, 2026; Accepted: February 26, 2026; Available online: March 12, 2026

<https://doi.org/10.5507/bp.2026.006>

© 2026 The Authors; <https://creativecommons.org/licenses/by/4.0/>

¹Department of Information and Communication Technologies in Medicine, Faculty of Biomedical Engineering, Czech Technical University in Prague, Prague, Czech Republic

²Department of Natural Sciences, Faculty of Biomedical Engineering, Czech Technical University in Prague, Czech Republic

³Department of Biomedical Technology, Faculty of Biomedical Engineering, Czech Technical University in Prague, Kladno, Czech Republic

Corresponding author: Aneta Buchtelova, e-mail: aneta.buchtelova@fbmi.cvut.cz

INTRODUCTION

Sleep is a fundamental physiological process essential for systemic restoration, autonomic balance, and metabolic regulation. Beyond its established effects on neurological and cardiovascular health, growing evidence suggests that sleep also plays a key role in ocular surface homeostasis^{1,2}. The precocular tear film, composed of lipid, aqueous, and mucin layers, maintains lubrication, optical quality, and defense against environmental stressors³. Disruption of this balance leads to tear film instability and hyperosmolarity, key features of dry eye disease (DED) affecting millions worldwide.

Experimental and clinical studies have demonstrated that insufficient sleep negatively influences tear secretion and ocular surface integrity⁴. In an animal model, acute sleep deprivation increased tear osmolarity, shortened tear film break-up time (TBUT), and reduced tear secretion. Chronic sleep deprivation has been shown to impair lacrimal gland structure and function through oxidative stress and inflammatory pathways^{5,6}. Epidemiological research also links short sleep duration to higher prevalence of DED symptoms⁷. These findings support the hypothesis that sleep quality directly contributes to ocular surface stability.

Particular attention has recently turned to the role of rapid eye movement (REM) sleep, characterized by autonomic activation, fluctuating ocular blood flow, and potential lacrimal stimulation⁸. Parasympathetic activation during REM phases may enhance ocular perfusion and secretory activity, thereby supporting tear film maintenance and ocular surface hydration⁹. Sleep quality, encompassing continuity, efficiency, and restorative sleep architecture including adequate REM phases, plays a crucial role in maintaining systemic and ocular surface health. Poor sleep quality has been associated with autonomic imbalance, oxidative stress, and inflammatory changes that may impair lacrimal gland function and destabilize the tear film^{4,10}. Population-based studies have shown that individuals with low sleep quality or insomnia symptoms exhibit a higher prevalence of dry eye disease and reduced tear stability¹¹. The researchers suggest a bidirectional relationship: ocular discomfort and eyelid-closure defects may disrupt sleep, while sleep disturbances may impair parasympathetic regulation of lacrimal gland activity and tear film homeostasis. Although causality remains unproven, this body of work emphasizes that not only sleep duration, but sleep quality including continuity, latency and disturbance should be considered when exploring factors influencing tear film stability and ocular surface health^{7,11,12}. These findings suggest that both the quantity and restorative quality of sleep are key determinants of tear film homeostasis and ocular surface hydration.

Despite these insights, the quantitative relationship between sleep architecture, especially REM duration, and subclinical measures of tear volume such as tear meniscus height (TMH) remains poorly understood. Moreover, the integration of mobile technologies for behavioral and physiological tracking introduces new opportunities for ecological, real-world data collection in ophthalmic re-

search. Therefore, this study aimed to investigate the association between sleep quantity, sleep quality, and TMH as a marker of ocular surface hydration, and to evaluate the usability of a mobile application for remote behavioral monitoring in this context.

MATERIALS AND METHODS

This prospective observational study was carried out over a three-week period combining objective physiological sleep tracking, self-reported behavioral data, and ophthalmological examination. The research followed a within-subject repeated-measures design, which allowed the investigation of intra-individual variability between daily sleep parameters and ocular surface indicators. Each participant provided five complete daily records, yielding a total of ninety valid sleep-tear measurements. The study protocol was reviewed and approved by the Ethics Committee of the Faculty of Biomedical Engineering, Czech Technical University in Prague, prior to data collection, and all participants provided informed consent in accordance with the Declaration of Helsinki.

Inclusion and exclusion criteria and recruitment

Eighteen healthy adult volunteers, fourteen women and four men, with a mean age of 29.6 ± 6.4 years, were recruited from university staff and students. All participants had normal ocular health, were free from systemic or ocular medications, and did not engage in shift work. They were instructed to avoid contact lens use for at least twenty-four hours before each morning measurement. Written informed consent was obtained from all participants.

Procedures

Objective sleep assessment was performed using a validated wrist-worn sleep tracker (Fitbit Charge 4, Fitbit Inc., San Francisco, CA, USA), which has demonstrated high accuracy compared with polysomnography in estimating total sleep time and identifying sleep stages^{13,14}. The device recorded total sleep duration, REM sleep duration, light and deep sleep stages, number of awakenings, and sleep quality. Data were automatically synchronized each morning via Bluetooth to the Fitbit mobile application linked to each participant's private account, from which the study investigators subsequently retrieved the anonymized sleep records for analysis.

To complement the physiological data, participants also used a custom mobile application developed specifically for clinical data assessment. The app was built for both Android and iOS platforms and served as a digital diary and questionnaire tool. Each morning after waking, participants completed a short survey assessing perceived stress during the previous day, smartphone use in the thirty minutes before bedtime, caffeine intake after 5 PM, morning alertness, and subjective sleep satisfaction. Overall compliance was high, with 100 percent of days providing complete responses. The evaluation of the mobile application was conducted using the System

Usability Scale (SUS) and complemented by qualitative feedback obtained through structured interviews with participants after study completion.

Tear film measurements were performed each morning between 8:00 and 9:00 a.m. to minimize diurnal variation. TMH was measured for both eyes using slit-lamp biomicroscope Opton 30SL-M (Opton, Germany) via a standardized protocol. The device had undergone all required technical and safety inspections in accordance with applicable national medical device regulations. The slit beam was narrowed to a fine horizontal line and aligned along the upper border of the lower eyelid margin. TMH was measured at the central point beneath the pupil, where the tear meniscus is most distinct. For the measurement, the beam height was adjusted to 0.2 mm to serve as a visual reference against the meniscus height. Participants were instructed to fixate on a distant point and to blink normally before the measurement. Meniscus continuity along the eyelid margin was verified before each reading. Values markedly above or below the 0.2 mm reference were considered outside the physiological range, indicating potential abnormalities in tear film volume. Data from the wearable and the mobile application were merged by participant identification and date. Sleep and TMH measurements were linked at the daily level, such that each daily record comprised sleep parameters from the preceding night and TMH measured the following morning.

Statistical analysis

All statistical analyses were performed in Python version 3.12 using the pandas, SciPy, and statsmodels packages and R. Descriptive statistics were expressed as mean $\pm$ standard deviation (SD). Linear associations between TMH and continuous sleep variables, including total sleep time, REM sleep duration, and subjective sleep quality, were assessed using Pearson's correlation coefficient (r). Group differences in TMH between participants with short and normal sleep duration (<6 h vs ≥ 6 h) were evaluated with the non-parametric Mann-Whitney U test, because the distribution of TMH deviated from normality. The primary analysis focused on the association between sleep parameters (total sleep time and REM sleep duration) and tear meniscus height (TMH). Analyses involving behavioral factors (stress exposure, smartphone use, caffeine intake) and subjective sleep quality were considered secondary and exploratory. Given the exploratory nature of these analyses, no formal correction for multiple comparisons was applied.

To evaluate the association between REM sleep duration and tear meniscus height, the REM variable (in hours) was divided into three equal groups (terciles) according to its distribution across all measurements. The resulting terciles represented short (T1: 0.35–1.12 h), moderate (T2: 1.13–1.55 h), and long (T3: 1.56–2.78 h) REM sleep durations. Normality of the data distribution was assessed using the Shapiro-Wilk test. Because the data deviated from normality, non-parametric statistical methods were applied. Differences in TMH between REM terciles were analyzed using the Kruskal-Wallis test, and the direction and strength of the monotonic association were quantified using Spearman's rank correlation coefficient (ρ). All tests were two-tailed, and statistical significance was set at $P < 0.05$. To quantify the relationship between REM sleep duration and (TMH), a simple linear regression model was fitted with TMH (in millimeters) as the dependent variable and REM sleep duration (in hours) as the independent variable. The resulting slope coefficient was used to estimate the expected change in TMH per unit increase in REM duration (expressed also per 30-minute increment for interpretability). For each subgroup, Pearson's correlation coefficients and regression slopes were calculated.

Relationships between behavioral factors (stress exposure, smartphone use before bed, caffeine intake) and TMH or sleep quality were examined using the Mann-Whitney U test. All analyses were two-tailed, and statistical significance was set at $P < 0.05$.

RESULTS

Sample characteristics

A total of ninety valid daily records were collected from eighteen participants, each contributing five consecutive daily recordings. The mean age of the participants was 29.6 years (SD 6.4), and seventy-eight percent were female. The measurements were performed in stable environmental conditions.

Across all observations, the mean (TMH) was 0.144 ± 0.045 mm, ranging between 0.05 and 0.3 mm. Participants slept an average of 6.57 ± 1.26 hours per night, with an average REM sleep duration of 1.34 ± 0.46 hours, corresponding to approximately 20% of total sleep. Approximately 28% of all recorded nights were classified as short-sleep nights (<6 hours total sleep). Table 1 shows descriptive statistics of key sleep parameters and tear meniscus height.

Table 1. Descriptive statistics of key sleep parameters and tear meniscus height.

Parameter	Mean $\pm$ SD	Median	Min	Max
Tear meniscus height (mm)	0.144 ± 0.045	0.150	0.05	0.30
Total sleep duration (h)	6.57 ± 1.26	6.67	2.53	10.33
REM sleep (h)	1.34 ± 0.46	1.33	0.35	2.78
Light sleep (h)	3.86 ± 1.07	3.88	0.83	6.95
Deep sleep (h)	1.29 ± 0.32	1.29	0.40	2.12

Descriptive statistics of the main study variables, including tear meniscus height (TMH), total sleep time, REM, light and deep sleep duration, and subjective sleep quality. Data are presented as mean $\pm$ standard deviation (SD), median, minimum, and maximum values. Descriptive statistics are calculated at the level of daily records ($n=90$).

TMH, tear meniscus height; REM, rapid eye movement; SD, standard deviation.

Correlation between sleep and tear meniscus height

Significant positive correlations were observed between TMH and total sleep time ($r=0.31$, $P=0.0033$), REM sleep duration ($r=0.28$, $P=0.0066$), and sleep quality ($r=0.32$, $P=0.0024$). These results indicate that both the quantity and the restorative architecture of sleep are associated with increased tear volume and better ocular surface hydration. No significant correlations were found between TMH and the number of awakenings ($P>0.1$).

Influence of REM sleep on tear meniscus height

A particularly consistent relationship was found between the duration of REM sleep and TMH. Participants with longer REM phases exhibited higher TMH values, and this association was observed across different ranges of total sleep duration. To further explore the relationship between REM sleep and tear film volume, REM duration was divided into terciles based on its distribution in the collected data: T1: 0.35–1.12 h (short REM), T2: 1.13–1.55 h (moderate REM), and T3: 1.56–2.78 h (long REM). A significant positive association was found between the length of REM sleep and TMH (Spearman's $\rho=0.28$, $P=0.008$). Participants belonging to higher REM terciles exhibited progressively greater TMH values. The Kruskal–Wallis test confirmed significant overall group differences ($H=7.11$, $P=0.029$). These findings suggest a dose–response relationship, indicating that longer REM sleep duration is associated with improved ocular surface hydration.

In linear models, each additional 30 minutes of REM sleep was associated with an approximate 0.014 mm increase in TMH ($r=0.285$, $P=0.0066$). The association was not significantly different between nights with total sleep time <6 h and ≥ 6 h (interaction $P=0.60$), and within-stratum correlations were not statistically significant. These

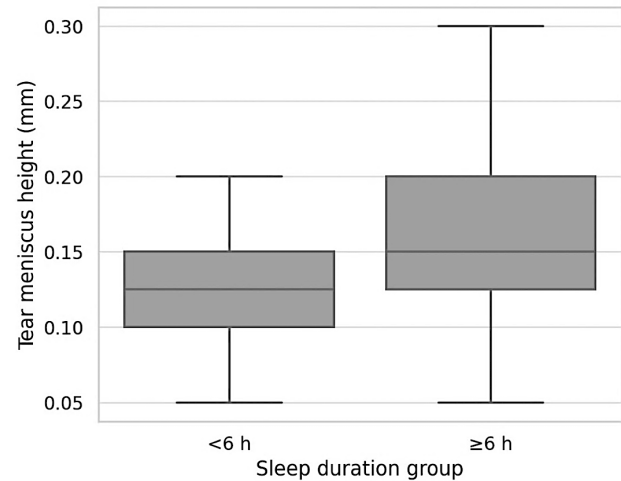


Fig. 1. Tear meniscus height by total sleep duration. Nights with total sleep duration <6 h exhibited significantly lower TMH compared with nights with total sleep duration ≥ 6 h ($P=0.0023$, Mann–Whitney U test).

results are consistent with a graded (dose–response) pattern across REM terciles.

From a physiological standpoint, longer REM phases likely facilitate parasympathetic activation, ocular blood flow enhancement, and lacrimal gland stimulation, all of which contribute to maintaining tear film stability. These mechanisms are compatible with prior evidence linking REM-related autonomic recovery to endocrine and immune regulation affecting ocular surface homeostasis. Sleep quality correlated positively with the REM proportion ($r=0.44$, $P<0.001$), suggesting that the restorative perception of sleep aligns with objective physiological processes beneficial to tear film function.

Comparison by total sleep duration

When grouped by total sleep duration, short-sleep nights (<6 h of total sleep time) exhibited significantly lower TMH (0.121 ± 0.039 mm) compared to nights with longer total sleep duration (≥ 6 h; 0.153 ± 0.044 mm; $P=0.0023$, Mann–Whitney U test) (see Fig. 1). This difference represents both a statistically and clinically meaningful reduction of approximately 21%. Group-level results are summarized in Table 2.

Behavioral factors and app-based observations

All participants completed the daily questionnaires, providing ninety valid records. Days when participants reported stress were associated with lower sleep quality ($P=0.03$) and slightly reduced TMH ($P=0.06$). Evening smartphone use before bedtime was common and linked to poorer sleep quality ($P=0.03$) and lower TMH ($P=0.05$). Caffeine intake after 5 PM was reported by only one participant, preventing meaningful statistical evaluation.

Overall, sleep quality correlated positively with TMH indicating that poorer sleep and less favorable behavioral habits were accompanied by mildly reduced tear film volume.

Table 2. Differences in tear meniscus height according to total sleep duration.

Group	n	Mean TMH (mm)	SD
< 6 h sleep	25	0.121	0.039
≥ 6 h sleep	65	0.153	0.044

Comparison of tear meniscus height (TMH) between nights classified as short sleep (<6 h) and normal sleep (≥ 6 h of total sleep time). Values are expressed as mean $\pm$ SD, and group differences were tested using the Mann–Whitney U test.

TMH, tear meniscus height; SD, standard deviation.

User satisfaction and acceptance of the mobile application

The usability and acceptance of the mobile application were evaluated through post-study feedback and a standardized System Usability Scale (SUS) questionnaire. The mean SUS score was 87 ± 6 out of 100, indicating excellent usability. Ninety-four percent of participants rated the app as “easy to use,” and 89% stated they would be willing to continue using it for longer studies or clinical monitoring.

Qualitative feedback indicated that participants appreciated the short and intuitive format of daily entries (mean completion time 54 s) and the integrated synchronization with their sleep tracker. In open comments, several participants mentioned that completing the questionnaire increased their awareness of sleep hygiene and encouraged behavioral changes, such as limiting phone use before bedtime. This secondary outcome suggests that the app may have a mild educational effect on sleep behavior, beyond data collection alone.

Overall, these results confirm the technical reliability and acceptability of the mobile application as a tool for remote behavioral data acquisition in ophthalmic and sleep research. The high adherence rate and consistency of subjective and objective measures demonstrate that mobile-based tracking can complement clinical and physiological studies effectively.

Summary of key findings

In summary, longer total and REM sleep durations were both associated with higher tear meniscus height, indicating better ocular surface hydration. REM sleep emerged as an independent and biologically plausible factor contributing to tear film stability, while behavioral and environmental variables such as stress and late-night smartphone use appeared to attenuate these beneficial effects. Furthermore, the high usability and participant satisfaction with the mobile application support the feasibility of integrating digital behavioral data collection into ophthalmic research, bridging laboratory physiology with real-world sleep hygiene monitoring.

DISCUSSION

The present study demonstrates that both sleep quantity and architecture are associated with tear film stability, as measured by TMH. In particular, REM sleep duration showed a consistent, moderate positive relationship with TMH. Behavioral and environmental factors such as stress and late-night smartphone use were linked to reduced TMH and poorer sleep quality. Together, these findings support the emerging concept of a “sleep-tear interface,” in which physiological and behavioral aspects of sleep jointly contribute to ocular surface homeostasis.

Our findings are consistent with a growing body of evidence linking poor sleep to ocular surface dysfunction and symptoms of dry eye disease (DED). Population-based studies have shown that individuals with low sleep quality or short sleep duration exhibit higher prevalence

of DED and lower basal tear secretion^{7,12,15}. Magno et al. reported that participants with poor sleep quality (PSQI > 5) had 20% higher odds of DED after adjusting for comorbidities¹². Similarly, Lee et al. and Ayaki et al. identified significant associations between reduced sleep quality, mood disorders, and increased subjective dryness^{7,15}. Experimental studies further indicate that sleep deprivation can directly impair lacrimal gland function through oxidative stress, inflammation, and altered lipid metabolism^{1,4,5,6,18}.

The present work extends this literature by showing, for the first time in a naturalistic human setting, a quantitative, dose-dependent association between REM sleep duration and TMH. While total sleep time has been repeatedly studied, the specific contribution of REM sleep to ocular physiology remains largely unexplored. To date, only few reports have addressed REM deprivation and its ocular consequences⁹. Human studies focusing specifically on REM phases and tear film parameters are virtually absent. Our findings may therefore represent one of the first empirical indications that REM sleep contributes measurably to ocular surface hydration.

The biological plausibility of this relationship is well supported by existing knowledge of REM physiology. During REM, parasympathetic activation predominates, promoting increased ocular perfusion and glandular secretion. This autonomic profile could enhance tear production, nutrient delivery, and epithelial repair¹⁶. Additionally, the characteristic rapid eye movements of this phase may provide mechanical stimulation aiding redistribution of the tear film. These mechanisms align with the observed positive association between REM duration and TMH, supporting the hypothesis that REM sleep plays a direct restorative role in maintaining tear film stability.

However, the current literature on this topic remains limited, and our findings should be viewed as an early contribution toward defining this relationship. Further studies incorporating polysomnography and advanced ocular imaging (e.g., OCT) are needed to confirm whether REM-related neural and vascular processes causally influence tear dynamics. The present study may thus serve as a preliminary step, a “first signal”, encouraging systematic investigation of sleep stage-specific effects on the ocular surface.

Methodologically, our approach combined objective wearable-derived sleep data with repeated slit-lamp biomicroscopy, yielding ninety valid linked measurements under controlled environmental conditions. While slit-lamp-based TMH assessment is less precise than OCT (ref.^{19,20}), the large number of observations per subject strengthens reliability and minimizes intraindividual variability. This pragmatic approach allowed continuous field monitoring that would not be feasible in a laboratory setting.

In contrast to previous studies relying on self-reported sleep questionnaires^{7,12,15}, our hybrid design improved ecological validity and sensitivity to subtle physiological effects. Still, the use of a consumer wearable (Fitbit Charge 4) instead of polysomnography introduces potential inaccuracies in sleep stage classification, especially for REM detection^{13,14}. Future validation studies comparing

wearable-derived REM metrics with gold-standard polysomnography in ophthalmic populations will be essential for confirming our observations.

Behavioral data collected via the mobile application provide additional insights into modifiable lifestyle factors affecting both sleep and ocular surface health. Stress was associated with poorer sleep quality and slightly lower TMH, consistent with studies linking psychological stress and elevated cortisol in tears to tear film instability and epithelial dysfunction²¹. Similarly, frequent smartphone use before bedtime correlated with lower REM duration and reduced TMH, supporting evidence that evening exposure to blue-enriched light suppresses melatonin secretion and disrupts REM sleep^{22,23}. These findings underline the need for behavioral interventions, such as digital hygiene and stress management, to support both sleep quality and ocular surface function.

Despite the robustness of repeated measurements and consistent results across sleep parameters, some limitations should be acknowledged. The study sample consisted of healthy young adults, limiting generalizability to older or clinical populations. The observational design precludes causal inference, and residual confounding by unmeasured factors (e.g., hormonal status, blinking frequency, daytime screen exposure) cannot be excluded. Because multiple nightly observations were obtained from each participant, the analyses did not explicitly account for within-subject clustering. Participant-specific factors, such as baseline tear meniscus height or habitual sleep patterns, may therefore have influenced the observed associations. Future studies with larger samples should apply mixed-effects or hierarchical models to better account for repeated measurements. Additionally, the use of slit-lamp-based meniscometry restricts precision compared to automated imaging, though consistency across repeated measurements partially mitigates this concern.

This study provides novel evidence linking sleep architecture, particularly REM sleep duration, to ocular surface hydration as reflected by TMH, suggesting that restorative sleep processes contribute to tear film stability. Given the current scarcity of research on REM sleep and the ocular surface, our results may represent an early indicator of a previously underrecognized link. Future studies employing polysomnography and advanced tear film imaging should further elucidate the mechanisms by which sleep architecture supports ocular surface homeostasis and potentially inform lifestyle-based strategies for preventing dry eye disease.

CONCLUSIONS

This study provides novel evidence linking sleep architecture, particularly REM sleep duration, to ocular surface hydration, as reflected by TMH. Both total sleep time and the proportion of REM sleep were positively associated with TMH, suggesting that restorative sleep processes play a role in maintaining tear film stability. Behavioral factors such as psychological stress and late-night smartphone use were found to attenuate these beneficial effects, un-

derscoring the interaction between lifestyle, sleep, and ocular physiology.

The findings highlight REM sleep as a potential physiological determinant of tear secretion and ocular surface homeostasis, an area previously underexplored in human research. Although the use of slit-lamp biomicroscopy and wearable-derived sleep data represents a methodological limitation, the high number of repeated measurements strengthens the reliability of the observed associations.

Future studies employing polysomnography and advanced ocular imaging (e.g., OCT meniscometry or interferometry) are warranted to elucidate the mechanisms through which sleep architecture influences tear film dynamics. Integrating digital behavioral monitoring into ophthalmic research may further enhance our understanding of sleep-eye interactions and support the development of lifestyle-based strategies for maintaining ocular surface health.

Taken together, the results highlight sleep as a modifiable determinant of ocular surface health. Promoting adequate, well-structured sleep and healthy pre-sleep behaviors may represent a novel, non-pharmacological avenue for preventing or mitigating tear film instability and dry eye symptoms.

Acknowledgements: The authors acknowledge financial support from the Faculty of Biomedical Engineering, Czech Technical University in Prague, through the Student Grant Competition project SGS23/203/OHK4/3T/17.

Author contributions: All authors contributed equally to preparing the manuscript.

Conflict of interest statement: The authors state that there are no conflicts of interest.

Ethical approval and consent to participate: The study protocol was reviewed and approved by the Ethics Committee of the Faculty of Biomedical Engineering, Czech Technical University in Prague and all participants provided informed consent.

REFERENCES

1. Lee YB, Koh JW, Hyon JY, Wee WR, Kim JJ, Shin YJ. Sleep deprivation reduces tear secretion and impairs the tear film. *Invest Ophthalmol Vis Sci* 2014;55(6):3525-31. doi: 10.1167/iops.14-13881
2. Li S, Tang L, Zhou J, Anchouche S, Li D, Yang Y, Liu Z, Wu J, Hu J, Zhou Y, Yin J, Liu Z, Li W. Sleep deprivation induces corneal epithelial progenitor cell over-expansion through disruption of redox homeostasis in the tear film. *Stem Cell Reports* 2022;17(5):1105-19. doi: 10.1016/j.stemcr.2022.03.017
3. Johnson ME, Murphy PJ. Changes in the tear film and ocular surface from dry eye syndrome. *Prog Retin Eye Res* 2004;23(4):449-74. doi: 10.1016/j.preteyeres.2004.04.003
4. Li S, Ning K, Zhou J, Guo Y, Zhang H, Zhu Y, Zhang L, Jia C, Chen Y, Sol Reinach P, Liu Z, Li W. Sleep deprivation disrupts the lacrimal system and induces dry eye disease. *Exp Mol Med* 2018;50(3):e451. doi: 10.1038/emmm.2017.285
5. Tang L, Wang X, Wu J, Li SM, Zhang Z, Wu S, Su T, Lin Z, Chen X, Liao X, Bai T, Qiu Y, Reinach PS, Li W, Chen Y, Liu Z. Sleep Deprivation Induces Dry Eye Through Inhibition of PPAR α Expression in Corneal Epithelium. *Invest Ophthalmol Vis Sci* 2018;59(13):5494-508. doi: 10.1167/iops.18-24504
6. Tang L, Zhang H, Liao Y, Zhou S, Yang Y, Zhang M, Guo Y, Xie T, Chen S, Ouyang W, Lin X, Wang S, Huang C, Zhang M, Zhuang J, Zhao

- J, Zhang R, Zhang C, Jin Z, Hu J, Liu Z. Chronic Sleep Deprivation Impairs Visual Functions via Oxidative Damage in Mice. *Am J Pathol* 2024;194(2):307-20. doi: 10.1016/j.ajpath.2023.11.004
7. Lee W, Lim SS, Won JU, Roh J, Lee JH, Seok H, Yoon JH. The association between sleep duration and dry eye syndrome among Korean adults. *Sleep Med* 2015;16(11):1327-31. doi: 10.1016/j.sleep.2015.06.021
8. Yamada RG, Ueda HR. Molecular Mechanisms of REM Sleep. *Front Neurosci* 2020;13:1402. doi: 10.3389/fnins.2019.01402
9. Murube J. REM sleep: tear secretion and dreams. *Ocul Surf* 2008;6(1):2-8. doi: 10.1016/s1542-0124(12)70101-5
10. Ayoubi M, Cabrera K, Mangwani S, Locatelli EVT, Galor A. Associations between dry eye disease and sleep quality: a cross-sectional analysis. *BMJ Open Ophthalmol* 2024;9(1):e001584. doi: 10.1136/bmjophth-2023-001584
11. Au NH, Mather R, To A, Malvankar-Mehta MS. Sleep outcomes associated with dry eye disease: a systematic review and meta-analysis. *Can J Ophthalmol* 2019;54(2):180-89. doi: 10.1016/j.jcjo.2018.03.013
12. Magno MS, Utheim TP, Snieder H, Hammond CJ, Vehof J. The relationship between dry eye and sleep quality. *Ocul Surf* 2021;20:13-19. doi: 10.1016/j.jtos.2020.12.009
13. Dong X, Yang S, Guo Y, Lv P, Wang M, Li Y. Validation of Fitbit Charge 4 for assessing sleep in Chinese patients with chronic insomnia: A comparison against polysomnography and actigraphy. *PLoS One* 2022;17(10):e0275287. doi: 10.1371/journal.pone.0275287
14. Schyvens AM, Van Oost NC, Aerts JM, Masci F, Peters B, Neven A, Dirix H, Wets G, Ross V, Verbraecken J. Accuracy of Fitbit Charge 4, Garmin Vivosmart 4, and WHOOP Versus Polysomnography: Systematic Review. *JMIR Mhealth Uhealth* 2024;12:e52192.
15. Ayaki M, Kawashima M, Negishi K, Tsubota K. High prevalence of sleep and mood disorders in dry eye patients: survey of 1,000 eye clinic visitors. *Neuropsychiatr Dis Treat* 2015;11:889-94. doi: 10.2147/NDT.S81515
16. Mukai Y, Yamanaka A. Functional roles of REM sleep. *Neurosci Res* 2023;189:44-53. doi:10.1016/j.neures.2022.12.009
18. Huang S, Si H, Liu J, Qi D, Pei X, Lu D, Zou S, Li Z. Sleep Loss Causes Dysfunction in Murine Extraorbital Lacrimal Glands. *Invest Ophthalmol Vis Sci* 2022;63(6):19. doi:10.1167/iops.63.6.19
19. Wang Y, Xu Z, Gong Q, Ren W, Chen L, Lu F, Hu L. The Role of Different Tear Volume Detection Methods in the Evaluation and Diagnosis of Mild Dry Eye Disease. *Transl Vis Sci Technol* 2022;11(3):15. doi: 10.1167/tvst.11.3.15
20. Niedernolte B, Trunk L, Wolffsohn JS, Pult H, Bandlitz S. Evaluation of tear meniscus height using different clinical methods. *Clin Exp Optom* 2021;104(5):583-8. doi: 10.1080/08164622.2021.1878854
21. Kaido M, Arita R, Mitsukura Y, Ishida R, Tsubota K. Variability of autonomic nerve activity in dry eye with decreased tear stability. *PLoS One* 2022;17(11):e0276945. doi: 10.1371/journal.pone.0276945
22. Heo JY, Kim K, Fava M, Mischoulon D, Papakostas GI, Kim MJ, Kim DJ, Chang KJ, Oh Y, Yu BH, Jeon HJ. Effects of smartphone use with and without blue light at night in healthy adults: A randomized, double-blind, cross-over, placebo-controlled comparison. *J Psychiatr Res* 2017;87:61-70. doi: 10.1016/j.jpsychires.2016.12.010
23. Silvani MI, Werder R, Perret C. The influence of blue light on sleep, performance and wellbeing in young adults: A systematic review. *Front Physiol* 2022;13:943108. doi: 10.3389/fphys.2022.943108