

UDC 612.821:159.944

DOI: 10.31651/2076-5835-2018-1-2026-1-4-16

Bohdan Olehovych Chernolevskiy

Oles Honchar Dnipro National University

bogdanchernolevskij@gmail.com

ORCID ID: <https://orcid.org/0009-0009-9089-1380>

Olena Viktorivna Severynovska

Oles Honchar Dnipro National University

eseverinovskaya@gmail.com

ORCID ID: <https://orcid.org/0000-0002-0002-1237>

DYNAMICS OF SLEEP ARCHITECTURE RESTORATION AND AUTONOMIC RESPONSES AFTER NIGHTLY WAR-RELATED STRESS

Objective. To investigate neurophysiological patterns of sleep recovery after acute stress and to evaluate the capabilities of consumer-grade wearable devices for recording microstructural changes in sleep, using the Xiaomi Smart Band 9 as an example.

Methods. Data from 39 individual sleep cycles recorded over five consecutive nights were analyzed (Baseline – control night, Night 0 – night of missile attack, Night 1-3 – recovery period). Sleep architecture and heart rate indicators were assessed using wearable data. Additionally, an exploratory single-case observation with parallel EEG (Hypnodyne ZMax) recording was performed. Statistical analysis included RM-ANOVA, Bonferroni post hoc test, Pearson correlation, and event-based metrics (PPV, F1-score, Cohen's κ).

Originality. The study provides the quantitative evidence of dissociation between REM sleep macro- and microstructure under real war-related stress conditions. It offers a novel approach for remote sleep assessment using heart rate dynamics as a proxy for detecting microarousals.

Results. The sleep macrostructure remained relatively stable despite acute stress, whereas REM sleep showed pronounced fragmentation. Detection of microarousals based on heart rate demonstrated consistency with EEG and greater informational value of autonomic responses under stress.

Conclusions. Sleep parameter recovery after acute stress follows a delayed, gradual pattern. Although consumer-grade wearable devices have limitations in estimating absolute sleep parameters, they can be used to monitor stress-related dynamics and indirectly assess REM-fragmentation.

Keywords: sleep neurophysiology; REM sleep; microarousals; sleep microstructure; autonomic regulation; wearable devices; post-stress recovery.

Problem statement. Sleep is one of the main integrative mechanisms for maintaining the physiological homeostasis. It is involved in regulating cellular metabolism, hormonal balance, and energy balance [1, 2]. Sleep architecture is formed by the complex interaction of homeostatic and circadian processes, which ensure the alternation of slow and fast sleep stages [3]. Each stage has a specific functional significance. Slow-wave sleep is associated with somatic recovery, metabolic regulation, and maintenance of neural plasticity [4], while the REM phase is involved in the processing of emotional experience and memory consolidation [5]. That is why the stability of the sleep architecture is an important condition for the normal functioning of the nervous system. Numerous studies show that sleep is highly sensitive to stressors. Emotional imbalance is accompanied by changes in the neurophysiological mechanisms of sleep regulation, manifested by increased short awakenings, fragmented sleep stages, and decreased overall sleep efficiency [5]. It is believed that the key role in these processes is played by the activation of limbic structures, primarily the amygdala, which maintains increased sympathetic activity even after the stressful event ends. Such prolonged autonomic activation can change the stability of neural networks responsible for maintaining sleep [6, 7]. REM sleep is considered particularly sensitive to emotional stress. This is because REM sleep plays an important role in adaptive responses to stressful experiences and in the processing of affective information [8, 9]. Accordingly, clinical studies have

shown that trauma-related conditions, including posttraumatic stress disorder, are associated with increased REM sleep fragmentation and instability of REM episodes, suggesting disruption of REM-related regulatory processes [10]. In response to a deficit of REM sleep, the phenomenon of the so-called REM rebound is sometimes observed - a compensatory increase in the intensity or frequency of REM episodes in subsequent nights [11].

Under conditions of stress, particularly those associated with war-related threats, sleep disorders often become persistent with alterations in their structure and quality. Notably, nocturnal stressors can affect recovery processes during sleep.

In this context, it is important to study how the architecture of sleep disruptions following such influences and how these alterations relate to the regulation of physiological processes. Of particular interest are the associations between sleep's structural characteristics and autonomic activity parameters.

Therefore, the problem lies in clarifying the features of changes in sleep architecture under acute nocturnal stress and their dynamics during subsequent recovery.

Analysis of recent studies and publications. Sleep disorders are especially often recorded in populations located in areas of prolonged social or armed conflicts. Under such conditions, the frequency of insomnia, nightmares, and sleep fragmentation increases significantly, which is often combined with symptoms of posttraumatic stress disorder [12, 13, 14, 15]. Studies conducted among the civilian population in war zones show that one of the most characteristic manifestations is REM sleep disturbance, which is considered an important neurophysiological marker of emotional maladaptation [9].

After the start of the war in Ukraine, the problem of sleep disorders has become particularly urgent. According to epidemiological studies, a significant proportion of the population reports deterioration in sleep quality, increased frequency of night awakenings, and reduced duration of recovery phases [12, 15]. Similar changes are observed among students living under conditions of regular air raid alerts and missile attacks [13]. Chronic stress in such conditions can lead to a gradual destabilization of sleep regulatory mechanisms and affect cognitive performance [3, 6].

At the same time, most studies assess mainly subjective indicators of sleep quality or symptoms of insomnia, while objective changes in sleep architecture during real stressful events remain insufficiently studied. The architecture of sleep reflects the complex organization of the NREM and REM cycles, their duration, sequence, and stability throughout the night. These parameters may serve as sensitive indicators of impaired neurophysiological regulation under strong emotional stress [3, 6]. Therefore, the study of changes in sleep architecture after nightly stressful events can provide a deeper understanding of the nervous system adaptation mechanisms to extreme conditions.

In recent years, wearable devices have been increasingly used in sleep research. Modern wearable activity trackers allow recording motor activity and heart rate parameters for extended periods without significant interference with a person's usual routine [16, 17]. This makes them a convenient tool for field observations, when full polysomnography is technically difficult or not feasible [18].

Highlighting previously unresolved parts of the general problem. Despite the significant research on the associations between stress and sleep, the dynamics of sleep architecture restoration after acute stressful events have not been sufficiently studied. Most of the work is based either on laboratory experiments or on subjective assessments of sleep quality. Data on short-term neurophysiological changes in the first days after real extreme events, especially in conditions of threats related to armed conflict, remain limited. Of particular interest is the potential to use cardiovascular autonomic signals as indicators of microstructural sleep disorders. Short-term increases in heart rate may accompany microarousals and reflect sympathetic nervous system activation [21, 22, 23]. However, the possibility of using such reactions as proxy markers of microarousal during REM sleep has not yet been sufficiently investigated, especially in field conditions using wearable devices [24].

In this regard, the study of changes in sleep architecture and related cardiovascular responses under natural conditions of exposure to intense stressors is of particular importance.

The aim of the study was to analyze the dynamics of sleep architecture on the night of a war-related stressful event and on subsequent recovery nights, and to assess the potential of cardiovascular-autonomic signals as proxy markers of sleep microstructural disturbances.

Scientific novelty. The dynamics of sleep recovery following acute nocturnal stress were assessed using monitoring with wearable activity trackers. The dissociation between relatively stable REM sleep macroarchitecture and disrupted microstructure was observed under real war-induced stress conditions. Autonomic responses, particularly heart rate spikes, showed potential as proxy markers of REM microarousals, with their informative value depending on stress exposure conditions.

Participants and study design. The study involved 20 civilians aged 21-35 years who resided in Ukraine and were within the potential range of military strikes. The inclusion criteria were the absence of diagnosed sleep disorders and severe psychiatric illnesses in the medical history, as well as no regular use of medications that may affect sleep or psychological state (e.g., hypnotics, sedatives, antidepressants, or anxiolytics).

The study was conducted as a prospective naturalistic observation. A total of 420 sleep recordings were collected. Since the occurrence of night-time stressful events, including missile strikes, drone attacks, and air defense system activity, was unpredictable, only a subset of the collected data met the criteria for night-time stressful exposure, which led to the selection of 39 individual five-day observation cycles for further analysis. Each participant contributed multiple observation cycles.

Each cycle included five consecutive nights: a control night (Baseline), the night of the stressful event (Night 0), and three subsequent recovery nights (Night 1-3).

The stress exposure night was defined based on multiple sources, including participants' self-reports, official air-raid alerts issued by regional authorities, and verified reports of missile or drone attacks. In most cases, the event was accompanied by the audible explosions or the operation of air defense systems in the participants' city of residence.

Only complete five-day observation cycles containing a single nocturnal attack were included in the analysis to ensure interpretability of recovery dynamics. Cycles with multiple attacks were excluded.

Participants followed their usual sleep schedule and were not subjected to any experimental interventions.

Hardware and monitoring. During sleep, heart rate (photoplethysmography) and motor activity (accelerometry) were recorded using the Xiaomi Smart Band 9.

The device's photoplethysmographic sensor provided continuous heart rate monitoring, while the accelerometer assessed motor activity during sleep.

Sleep stage estimation and autonomic responses was performed based on synchronized heart rate and motor activity data.

Hypothesis-generating naturalistic observation. During the devices' pilot validation, naturalistic observation was recorded. One participant simultaneously used two sleep monitoring devices for 5 consecutive nights: the Xiaomi Smart Band 9, a photoplethysmographic wearable device, and the Hypnodyne ZMax portable EEG system (Hypnodyne Corp., USA).

The second night of the study coincided with an acute stressful event - a massive missile and drone attack. Despite the discomfort of the EEG headband and the emotional strain, the participant decided to continue recording his sleep. The next three nights were used to observe the recovery period.

This situation was not experimentally induced; it occurred naturally, so the data obtained are considered a descriptive single-case observation rather than a controlled experiment or clinical validation.

The ZMax system provided frontal EEG, triaxial accelerometry, and photoplethysmography recording. Sleep stages were defined according to the American Academy of Sleep Medicine (AASM) criteria.

The naturalistic observations obtained during the devices' validation procedure allowed evaluation of their performance under conditions of real acute stress. In this case, the heart rate spikes recorded by the wearable device's photoplethysmographic sensor showed temporal coherence with EEG-recorded REM sleep microactivations. Although these results are descriptive and cannot be generalized, they provide a basis for a hypothesis-generating observation regarding the use of photoplethysmographic signals as indirect markers of microarousals.

Microarousals were identified in EEG recordings according to AASM criteria as abrupt frequency shifts lasting at least 3 seconds.

Based on photoplethysmographic data from the wearable activity tracker, heart rate spikes exceeding 15% of the individual's nocturnal median heart rate were identified. Such events were considered as potential proxy indicators of sleep fragmentation.

Statistical analysis. Statistical analyses were conducted using Statistica 12.0 software. (StatSoft Inc., USA). Normality was assessed using the Shapiro-Wilk test. Data with a normal distribution are presented as mean \pm standard deviation ($M \pm SD$).

The dynamics of indicators during the observation period (Baseline-Night 3) were assessed using repeated-measures analysis of variance (RM-ANOVA with time as a within-subject factor) with Bonferroni post hoc correction.

The associations between sleep architecture parameters and autonomic regulation indicators were analyzed using Pearson's correlation matrices.

Agreement between the two devices under acute stress exposure was assessed using Cohen's kappa coefficient. The level of statistical significance was set at $p < 0,05$.

Ethical aspects. The study was approved by the Ethics Committee of Oles Honchar Dnipro National University (Dnipro, Ukraine) (protocol No. 4 dated 05/19/2025). All participants provided written informed consent. Personal data were anonymized in accordance with the Law of Ukraine "On the Protection of Personal Data".

The study was non-invasive, did not involve interference with participants' behavior during air raid alerts, and was conducted in accordance with the Declaration of Helsinki (2013). Participants were free to withdraw from the study at any time without providing a reason.

Artificial intelligence tools were used exclusively to improve the grammar, clarity, and coherence of the text. Data analysis, result interpretation, and conclusion formulation were performed by the authors.

Exploratory single-case observation data analysis. Assessing the consistency of integral sleep parameters obtained over five consecutive days during naturalistic observation using the Xiaomi Smart Band 9 and Hypnodyne ZMax in parallel revealed varying levels of coherence between devices, depending on the metric studied. Table 1 shows the mean values with standard deviation ($M \pm SD$) and mean absolute error (MAE) values.

Table 1

Comparison of sleep architecture parameters

Parameter	ZMax ($M \pm SD$)	Xiaomi Smart Band 9 ($M \pm SD$)	MAE \pm SD
Total sleep time (TST), min	356,6 \pm 40,9	367,2 \pm 36,8	14,2 \pm 10,9
Duration of deep sleep (N3), min	113,8 \pm 20,5	113,2 \pm 6,1	13,6 \pm 12,4
Duration of REM sleep, min	102,0 \pm 27,2	94,2 \pm 18,6	17,8 \pm 9,8
Duration of light sleep (N1-N2), min	126,0 \pm 34,1	160,2 \pm 22,2	40,2 \pm 20,3
Wake after sleep onset (WASO), min	13,4 \pm 13,1	0,6 \pm 0,55	12,8 \pm 13,7

The smallest absolute error was observed for the wake after sleep onset (WASO) parameter; however, this value was accompanied by a systematic underestimation of wakefulness duration by the wearable device. According to ZMax, the average WASO was over 13 min, while the Xiaomi Smart Band 9 almost did not register such episodes, suggesting a limited ability to detect short-term awakenings.

For the deep sleep duration (N3) parameter, the mean absolute error was $13,6 \pm 12,4$ min, one of the smallest among the analyzed sleep stages. Moderate discrepancies were recorded for the parameters of total sleep duration (TST) and REM sleep. The largest differences were observed in the total duration of light sleep stages (N1-N2), with an MAE exceeding 40 min.

Event analysis of REM fragmentation. To test the hypothesis that heart rate spikes could serve as a proxy marker of REM sleep fragmentation, an event analysis was performed. A heart rate spike was defined as a short-term increase in heart rate of more than 15% above the individual's average during a REM epoch. Microarousals were identified in EEG recordings collected by the ZMax system according to AASM criteria.

In total, during the observation period, an average of 28 microarousals were recorded during REM sleep according to EEG data (ZMax) and 25 cardiovascular reactions (heart rate spikes) according to Xiaomi Smart Band 9 data.

Event classification matrix. To evaluate the correspondence between heart rate spikes and EEG-verified microarousals, events within REM sleep epochs were compared. For each REM sleep epoch, it was determined whether a microarousal was observed from EEG data and whether a heart rate spike was recorded from Xiaomi Smart Band 9 data. An event classification matrix was constructed.

Four types of results were distinguished: True Positive (TP) – simultaneous registration of microarousal and heart rate spike (20 events); False Negative (FN) – microarousal without heart rate response (8 events); False Positive (FP) - heart rate spike without EEG event (5 events); True Negative (TN) – absence of both events in the REM epoch (15 events).

Based on this classification matrix, the main accuracy indicators of matching heart rate spikes as a proxy marker of microarousals were calculated.

The positive predictive value (PPV) of the method was 80,0%, while the sensitivity and specificity were moderate (Sensitivity = 71,4%, Specificity = 75,0%). The overall classification accuracy was 72,9%, and the harmonic mean of precision and recall (F1-score) was 0,755.

To assess the overall agreement between the methods, Cohen's kappa coefficient (Cohen's κ) was calculated based on the event classification matrix in REM epochs. The obtained value of $\kappa \approx 0,66$ indicates a moderately high level of agreement between the event detection methods.

Dependence of detection on the stress exposure condition. Since cardiovascular reactions during sleep can vary with the emotional and physiological state of the person, it was further examined whether the accuracy of heart rate spike detection depends on the physiological context of the night. For this purpose, microarousals detected from EEG data (ZMax) were distributed across study periods: baseline night, night of the acute stressful event, and three subsequent nights. For each period, the match rate (proportion of microarousals accompanied by heart rate spikes) was calculated.

The analysis showed that heart rate spike detection accuracy varied depending on stress exposure conditions (Table 2).

Table 2

Match rate by study period

Period	Match Rate (TP/Events ZMax)	Detection Characteristics
Baseline	20% (1/5)	Low detection of "quiet" microarousals
Night 0 (Attack)	78% (7/9)	High sensitivity to acute events
Night 1-3 (Aftereffect)	86% (12/14)	Maximum identification accuracy

During the baseline night, only 1 out of 5 microarousals coincided with a heart rate spike. During the stress exposure night, such coincidences were observed more frequently, with 7 of 9 microarousals accompanied by autonomic responses. The association between heart rate spikes and microarousals was highest during the three subsequent nights after the attack, with 12 out of 14 paired events (86%).

These results suggest that heart rate responses preferentially reflect microarousals associated with emotionally salient events rather than spontaneous microarousals during stable sleep.

Results of the main phase of the study using the Xiaomi Smart Band 9. Table 3 presents the mean values and standard deviations ($M \pm SD$) of sleep architecture and autonomic parameters derived from the wearable device data over 39 five-day observation cycles, along with the results of a repeated-measures analysis of variance (RM-ANOVA) for the time factor. For indicators with a statistically significant time effect, post hoc comparisons were performed using the Bonferroni correction.

Table 3

Mean values and variability of sleep architecture and autonomic sleep parameters derived from wearable device data ($n = 39$ sleep cycles)

Cycle day	Baseline ($M \pm SD$)	Night 0 ($M \pm SD$)	Night 1 ($M \pm SD$)	Night 2 ($M \pm SD$)	Night 3 ($M \pm SD$)	F (4,152)	p
Total sleep duration, min	466,87 $\pm 77,24^{a,b}$	419,00 $\pm 69,86^b$	482,77 $\pm 92,25^a$	466,51 $\pm 71,31^{a,b}$	458,38 $\pm 67,32^{a,b}$	4,7	0,001
Duration of deep sleep (N3), min	155,56 $\pm 25,42^{a,b}$	139,54 $\pm 30,13^b$	156,62 $\pm 32,61^a$	157,82 $\pm 28,27^{a,b}$	148,36 $\pm 31,62^{a,b}$	3,41	0,011
Duration of light sleep (N1-N2), min	197,69 $\pm 45,38^a$	171,69 $\pm 40,44^{b*}$	203,28 $\pm 50,90^a$	197,69 $\pm 38,14^a$	194,18 $\pm 36,90^{a,b}$	4,54	0,002
Duration of REM sleep, min	115,08 $\pm 24,36$	107,05 $\pm 25,78$	117,74 $\pm 9,34$	113,92 $\pm 28,45$	116,41 $\pm 29,18$	1,45	0,221
Wake after sleep onset (WASO), min	8,82 $\pm 14,24$	10,13 $\pm 15,92$	16,69 $\pm 28,12$	7,36 $\pm 12,25$	6,85 $\pm 11,95$	2,42	0,051
REM episode count, (n)	9,51 $\pm 1,93$	9,31 $\pm 3,03$	10,77 $\pm 3,44$	10,79 $\pm 3,30$	9,03 $\pm 2,98$	2,77	0,029
Mean REM episode duration, min	12,14 $\pm 3,42$	12,54 $\pm 4,27$	11,85 $\pm 4,50$	11,57 $\pm 3,81$	13,23 $\pm 4,32$	1,57	0,186
Heart rate spikes, (n)	3,15 $\pm 1,60^a$	6,03 $\pm 2,45^{b, c*}$	6,36 $\pm 2,36^{c*}$	7,08 $\pm 2,31^{b*}$	4,10 $\pm 1,93^a$	47,31	<0,01
Minimum heart rate, bpm	57,56 $\pm 5,52$	57,46 $\pm 6,07$	57,72 $\pm 5,34$	57,95 $\pm 5,49$	57,92 $\pm 6,00$	0,16	0,959
Mean heart rate, bpm	68,21 $\pm 6,53$	68,79 $\pm 7,28$	68,41 $\pm 6,15$	69,31 $\pm 7,43$	69,08 $\pm 7,18$	0,40	0,805
Maximum heart rate, bpm	79,49 $\pm 8,25^a$	88,92 $\pm 8,82^{b*}$	83,46 $\pm 10,16^{a*}$	82,28 $\pm 9,14^a$	82,31 $\pm 9,53^a$	19,48	<0,01

Notes: * – statistically significant differences ($p < 0,05$) compared to Baseline.

Letter indices (^a, ^b, ^c) reflect the results of multiple comparisons between observation nights (Post-hoc Bonferroni test). Values with different letters within the same row differ significantly ($p < 0,05$).

Repeated analysis of variance revealed that some sleep architecture parameters varied across nights. The most pronounced changes concerned the total sleep duration, deep sleep duration, and light sleep duration.

During the night of the attack, total sleep duration decreased, followed by an increase on the first night after the event. Similar dynamics were observed for deep sleep duration, which decreased during the attack night and gradually returned to baseline levels on subsequent nights. Light sleep showed a slightly different pattern: its duration also decreased during the attack night, but recovered already on the first post-event night. No significant time effect was found for total REM sleep duration. The values of this parameter remained relatively stable throughout the observation period. Similarly, the mean duration of a single REM episode did not differ significantly between nights.

Wake after sleep onset showed only a trend toward change. The highest value was observed on the first night after the event; however, due to substantial inter-individual variability, the effect did not reach statistical significance.

The REM episode count varied across observation days, but post-hoc comparisons did not reveal statistically significant differences between individual nights, suggesting that the overall organization of REM sleep, as estimated by wearable device, remained relatively stable.

A different pattern of cardiovascular reactivity was observed. The frequency of heart rate spikes during REM sleep showed a significant time effect. It increased markedly on the attack night compared to baseline and remained elevated over the following two nights, before returning to baseline levels on the third night after the event. The minimum and mean heart rates during sleep remained stable throughout the observation period. In contrast, the maximum heart rate showed significant changes: it increased during the attack night, remained elevated on subsequent nights, and gradually declined during the recovery period.

In general, sleep architecture parameters demonstrated moderate, relatively short-term changes following a stressful event, whereas autonomic responses, particularly heart rate spikes and maximum heart rate, displayed more pronounced, longer-lasting dynamics.

These findings suggest that, under acute stress conditions, autonomic regulation may serve as a more sensitive indicator of physiological response than sleep architecture metrics derived from consumer-grade wearable devices.

In addition, these results are consistent with the event-based analysis, which showed that heart rate responses during REM sleep are more often associated with microarousals linked to stressful or emotionally salient events.

Correlations between sleep parameters and cardiovascular regulation. To assess the dynamics of the associations between sleep architecture and cardiovascular regulation parameters, a correlation analysis was performed for each study stage. Table 4 shows the Pearson's correlation coefficients between sleep architecture parameters and heart rate indicators.

Throughout the observation period, a stable pattern of associations among the main components of sleep architecture was maintained. Total sleep duration showed strong positive correlations with both deep and light sleep durations, as well as moderate positive correlations with REM sleep duration. During REM sleep, characteristic internal associations were maintained: REM sleep duration was positively correlated with the REM episode count, while the mean duration of REM episodes showed a stable negative correlation with episode count. Indicators of cardiovascular regulation also demonstrated high internal consistency across all observation periods: the minimum, mean, and maximum heart rates showed strong positive correlations with each other.

The most stable correlation structure was observed in the baseline period. In addition to the expected internal associations between sleep phases, positive correlations were found between the WASO duration and heart rate indicators. During the attack night (Night 0), the overall pattern of correlations between sleep stages was preserved, whereas associations between sleep parameters and heart rate measures weakened. In particular, correlations between WASO and heart rate parameters were no longer significant. During the first two post-event nights (Night 1, Night 2), the correlation pattern became more pronounced again, with the main changes observed during REM sleep.

Table 4

Correlation coefficients between sleep parameters
and heart rate variables (n = 39 sleep cycles)

Pair of variables	Baseline	Night 0	Night 1	Night 2	Night 3
Duration of deep sleep, min - Minimum heart rate, bpm	0,04	-0,13	-0,21	-0,18	-0,13
Duration of deep sleep, min - Mean heart rate, bpm	0,11	-0,09	-0,11	-0,32*	-0,23
Duration of REM sleep, min - Minimum heart rate, bpm	-0,23	-0,17	-0,43**	-0,51***	-0,35*
Duration of REM sleep, min - Mean heart rate, bpm	-0,14	-0,07	-0,30	-0,51***	-0,30
Duration of REM sleep, min - Maximum heart rate, bpm	-0,30	-0,17	-0,14	-0,35*	-0,24
REM episode count, (n) - Minimum heart rate, bpm	0,13	0,05	0,21	0,01	-0,20
Mean duration of REM episode, min - Minimum heart rate, bpm	-0,37*	-0,13	-0,52***	-0,18	-0,11
Duration of awakenings, min - Minimum heart rate, bpm	0,63***	0,26	0,17	0,57***	0,05
Duration of awakenings, min - Mean heart rate, bpm	0,47**	0,21	0,15	0,51***	-0,01
Duration of awakenings, min - Maximum heart rate, bpm	0,27	0,22	0,07	0,46**	0,13

Note: correlations significance levels: * – $p < 0,05$; ** – $p < 0,01$; *** – $p < 0,001$.

Marked negative correlations were observed between REM sleep duration and heart rate parameters. Similar correlations were observed for the mean duration of REM episodes. On the second night after the event (Night 2), positive correlations between the duration of awakenings and heart rate indicators were also restored, approaching the baseline pattern. However, this pattern was associated with a stronger negative correlation between REM sleep and heart rate. On the third night after the event (Night 3), the correlation structure almost fully recovered to baseline levels.

Overall, the results showed that an acute stressful event was associated with a temporary weakening of connections between the sleep-regulating systems and cardiovascular activity. Over the following nights, this interaction gradually returned, with the most pronounced changes in the correlation structure occurring during REM sleep. This may indicate a special role for REM sleep in restoring autonomic regulation after stress.

Discussion. During naturalistic observation, the performance of a consumer-grade wearable device and an EEG system was analyzed in assessing microarousals during REM sleep. Heart rate spikes recorded with the Xiaomi Smart Band 9 photoplethysmographic sensor showed temporal coherence with microactivation episodes recorded with EEG data. Although the findings cannot be generalized due to the lack of a representative sample, they suggest that consumer-grade wearable devices may be useful for detecting physiologically meaningful responses during REM sleep under acute stress.

Comparison of sleep architecture parameters obtained from the two monitoring systems under real war-induced stress conditions revealed variable agreement across sleep stages. In particular, the consumer device showed sufficient accuracy in detecting deep sleep but differed markedly from the reference system in estimating WASO and light sleep stages (N1-N2). The observed tendency to classify nocturnal awakening periods as light sleep suggests that wearable activity device algorithms underestimate the fragmentation indices of sleep's macrostructure.

The sleep architecture dynamics assessed during the main study generally corresponded to the principles of homeostatic regulation and compensatory recovery. On the night of the extreme event, total sleep duration decreased, consistent with the known effects of acute stress on the regulation of the sleep-wake cycle. On subsequent nights, this indicator gradually increased, which may reflect compensatory prolongation of sleep after disruption of its structure.

Similar dynamics were observed for slow-wave sleep. Deep sleep duration decreased during the attack night and gradually recovered over subsequent nights. This pattern is consistent with the concept that slow-wave sleep contributes to the restoration of neural resources and the maintenance of brain homeostasis.

In contrast, the architecture of REM sleep appeared relatively stable on initial analysis. The total REM sleep duration did not change significantly during the studied period. The results obtained may indicate a high adaptive potential of the nervous system and the development of mechanisms to maintain sleep homeostasis under prolonged war-related stress. At the same time, significant alterations in REM episode count, alongside a relatively stable total REM duration, suggest an internal reorganization of this sleep stage. Accordingly, while the macroarchitecture of REM sleep appears externally stable, processes of destabilization and redistribution of phase cycles may occur at the microstructural level. A similar dissociation between macro- and microstructure of sleep has been described in studies analyzing the effects of stress on REM sleep, as well as in works focused on sleep state misperception in paradoxical insomnia [25, 26]. Accordingly, sleep disorders in the context of war-induced stress occur mainly at the microstructural level, underscoring the importance of using highly sensitive methods for quantitative analysis of sleep microarchitecture rather than relying on standard assessment of temporal parameters alone.

A key component of stress-induced changes observed in this study was autonomic regulation. Elevations in maximum heart rate and in the frequency of heart rate spikes during REM sleep may reflect enhanced autonomic reactivity. Such cardiovascular responses are often associated with brief cortical arousals and transient disruptions in sleep stability [27].

Considering heart rate spikes as proxy markers of microarousals suggests that increased autonomic reactivity may contribute to microstructural fragmentation of REM sleep even after the end of direct stress exposure. At the same time, the stability of the minimum heart rate throughout the entire observation period indicates the preservation of stable autonomic homeostasis. Therefore, the results obtained indicate a certain dissociation between the basic autonomic tone and the peak reactivity of the cardiovascular system.

This dissociation was also manifested in the structure of correlations between sleep parameters and heart rate. In the baseline period, a relatively stable system of correlations was observed. WASO demonstrated positive correlations with the minimum and mean heart rate parameters, reflecting coordinated autonomic responses and sleep maintenance.

During acute stress (Night 0), these correlations were significantly weakened. Such desynchronization may indicate an increased variability of autonomic responses and a temporary disruption of the usual regulatory connections between the autonomic system and the architecture of sleep.

During the first two post-event nights (Night 1 and Night 2), the correlation structure was partially rebuilt, with the main changes concentrated in the REM sleep. In particular, negative correlations were recorded between the duration of REM episodes and heart rate parameters. This may indicate that increased autonomic activation limits the duration of individual REM cycles and contributes to their premature interruption. Concurrently, on the second night after the event, positive correlations between WASO and heart rate re-emerged, suggesting a gradual reintegration of sleep regulatory processes and autonomic activity. The negative correlation between REM sleep and WASO during this period reflects the competitive nature of the interaction between sleep maintenance mechanisms and destabilizing factors. On the third night after the event (Night 3), the correlation structure almost completely returned to the baseline pattern. This may indicate the completion of the acute adaptation phase and the gradual restoration of the coordinated work of the sleep regulation systems and the autonomic nervous system.

The results obtained are also consistent with modern ideas about the functional role of REM sleep in the processing of emotional experience and the regulation of stress reactions. A number of studies have shown that REM sleep is involved in emotional adaptation and recovery from stress. From this point of view, the detected changes in the REM structure may reflect the active phase of the neurophysiological adaptation to the experienced stressful impact [3, 6].

However, the study's results should be interpreted in light of certain methodological limitations. The assessment of sleep architecture was carried out using a consumer wearable device, whose algorithms may have limited accuracy in determining individual sleep stages and short episodes of awakening. That is why the interpretation of changes in sleep architecture should be considered alongside additional indicators, particularly the dynamics of cardiovascular responses.

Overall, the results indicate that, even with the relative stability of the macroarchitecture of REM sleep after acute stress exposure, significant changes in its microstructure may occur, reflected in increased autonomic reactivity and in the restructuring of associations between sleep parameters and heart rate.

Conclusions and future research directions. The restoration of sleep architecture after acute stress is gradual. The most pronounced instability of parameters is observed during the first two nights after the event, whereas the correlation structure between sleep indicators and heart rate returns to baseline pattern only by the third night.

Despite the relative stability of sleep macrostructure, stress is associated with microstructural alterations, such as REM sleep fragmentation and autonomic desynchronization, indicating a dissociation between macro- and microarchitectural levels of sleep organization.

The Xiaomi Smart Band 9 wearable activity tracker can be used for longitudinal monitoring of general sleep trends, but has limitations in detecting WASO, potentially leading to an underestimation of sleep fragmentation.

Heart rate spikes during REM sleep can serve as proxy markers of microarousals. The proposed approach demonstrated sufficient agreement with EEG recordings ($F1 = 0,755$; Cohen's $\kappa = 0,66$), supporting the feasibility of using cardiovascular-autonomic parameters for indirect evaluation of REM sleep fragmentation.

Future research directions include expanding the sample across demographic groups, conducting detailed analyses of REM sleep microstructure under chronic stress, and developing machine-learning-based methods for automated sleep fragmentation detection. Further work should focus on integrating wearable device data with polysomnography to improve the accuracy of ambulatory sleep disorder assessment and on establishing a quantitative index of REM sleep fragmentation based on cardiovascular-autonomic markers derived from wearable devices.

REFERENCES (in language original)

1. Liu P. Y., Reddy R. T. Sleep, testosterone and cortisol balance, and ageing men. *Reviews in endocrine & metabolic disorders*. 2022. Vol. 23, No. 6. P. 1323–1339. DOI: <https://doi.org/10.1007/s11154-022-09755-4>.
2. Feeney S. P., McCarthy J. M., Petruconis C. R., Tudor J. C. Sleep loss is a metabolic disorder. *Science signaling*. 2025. Vol. 18, No. 881. Art. eadp9358. DOI: <https://doi.org/10.1126/scisignal.adp9358>.
3. Franken P., Dijk D. J. Sleep and circadian rhythmicity as entangled processes serving homeostasis. *Nat. Rev. Neurosci*. 2024. Vol. 25. P. 43–59. DOI: <https://doi.org/10.1038/s41583-023-00764-z>.
4. Xu Y., Schneider A., Wessel R. et al. Sleep restores an optimal computational regime in cortical networks. *Nat Neurosci*. 2024. Vol. 27. P. 328–338. DOI: <https://doi.org/10.1038/s41593-023-01536-9>.
5. Tempesta D., Soggi V., De Gennaro L., Ferrara M. Sleep and emotional processing. *Sleep medicine reviews*. 2018. Vol. 40. P. 183–195. DOI: <https://doi.org/10.1016/j.smrv.2017.12.005>.
6. Van Someren E. J. W. Brain mechanisms of insomnia: new perspectives on causes and consequences. *Physiological reviews*. 2021. Vol. 101, No. 3. P. 995–1046. DOI: <https://doi.org/10.1152/physrev.00046.2019>.
7. Dressler R. J., Riemann D. Hyperarousal in insomnia disorder: Current evidence and potential mechanisms. *Journal of Sleep Research*. 2023. Vol. 32, No. 6. Art. e13928. DOI: <https://doi.org/10.1111/jsr.13928>.
8. Werner G. G., Schabus M., Blechert J., Wilhelm F. H. Differential Effects of REM Sleep on Emotional Processing: Initial Evidence for Increased Short-term Emotional Responses and Reduced Long-term Intrusive Memories. *Behavioral sleep medicine*. 2021. Vol. 19, No. 1. P. 83–98. DOI: <https://doi.org/10.1080/15402002.2020.1713134>.

9. Rho Y. A., Sherfey J., Vijayan S. Emotional Memory Processing during REM Sleep with Implications for Post-Traumatic Stress Disorder. *The Journal of neuroscience*. 2023. Vol. 43, No. 3. P. 433–446. DOI: <https://doi.org/10.1523/JNEUROSCI.1020-22.2022>.
10. Repantis D., Wermuth K., Tsamitros N. et al. REM sleep in acutely traumatized individuals and interventions for the secondary prevention of post-traumatic stress disorder. *European Journal of Psychotraumatology*. 2020. Vol. 11, No. 1. Art. 1740492. DOI: <https://doi.org/10.1080/20008198.2020.1740492>.
11. Machado, R. B., & Suchecki, D. (2016). Neuroendocrine and Peptidergic Regulation of Stress-Induced REM Sleep Rebound. *Frontiers in endocrinology*, 7, 163. <https://doi.org/10.3389/fendo.2016.00163>
12. Pavlova I., Rogowska A. M. Exposure to war, war nightmares, insomnia, and war-related posttraumatic stress disorder: A network analysis among university students during the war in Ukraine. *Journal of affective disorders*. 2023. Vol. 342. P. 148–156. DOI: <https://doi.org/10.1016/j.jad.2023.09.003>.
13. Rogowska A. M., Pavlova I. A path model of associations between war-related exposure to trauma, nightmares, fear, insomnia, and posttraumatic stress among Ukrainian students during the Russian invasion. *Psychiatry research*. 2023. Vol. 328. Art. 115431. DOI: <https://doi.org/10.1016/j.psychres.2023.115431>.
14. Boiko D. I. Sleep problems in combatants with posttraumatic stress disorder and its acceptance. *Visnyk Problem Biologii i Medytsyny*. 2024. Vol. 2, No. 173. P. 165–174. DOI: <https://doi.org/10.29254/2077-4214-2024-2-173-165-174>.
15. Kurapov A., Schabus M., Kahveci S. et al. Explaining post-traumatic stress symptoms and sleep disturbance in Ukrainian civilians: perceived threat versus objective war exposure. *European journal of psychotraumatology*. 2024. Vol. 15, No. 1. Art. 2381371. DOI: <https://doi.org/10.1080/20008066.2024.2381371>.
16. Chinoy E. D. et al. Performance of seven consumer sleep-tracking devices compared with polysomnography. *Sleep*. 2021. Vol. 44, No. 5. Art. zsa291. DOI: <https://doi.org/10.1093/sleep/zsa291>.
17. Kainec K. A., Caccavaro J., Barnes M. et al. Evaluating Accuracy in Five Commercial Sleep-Tracking Devices Compared to Research-Grade Actigraphy and Polysomnography. *Sensors*. 2024. Vol. 24, No. 2. Art. 635. DOI: <https://doi.org/10.3390/s24020635>.
18. Herberger S., Aurnhammer C., Bauerfeind S. et al. Performance of wearable finger ring trackers for diagnostic sleep measurement in the clinical context. *Sci Rep*. 2025. Vol. 15. Art. 9461. DOI: <https://doi.org/10.1038/s41598-025-93774-z>.
19. de Zambotti M. et al. Rigorous performance evaluation for informed use of new technologies for sleep health measurement. *Sleep Health*. 2022. Vol. 8, No. 3. P. 263–269. DOI: <https://doi.org/10.1016/j.sleh.2022.02.006>.
20. Schyvens A. M., Peters B., Van Oost N. C. et al. A performance validation of six commercial wrist-worn wearable sleep-tracking devices for sleep stage scoring compared to polysomnography. *Sleep advances*. 2025. Vol. 6, No. 2. Art. zpaf021. DOI: <https://doi.org/10.1093/sleepadvances/zpaf021>.
21. Trinder J., Kleiman J., Carrington M. et al. Autonomic activity during human sleep as a function of time and sleep stage. *Journal of Sleep Research*. 2001. Vol. 10. P. 253–264. DOI: <https://doi.org/10.1046/j.1365-2869.2001.00263.x>.
22. Halász P., Terzano M., Parrino L., Bódizs R. The nature of arousal in sleep. *Journal of Sleep Research*. 2004. Vol. 13. P. 1–23. DOI: <https://doi.org/10.1111/j.1365-2869.2004.00388.x>.
23. Stein P. K., Pu Y. Heart rate variability, sleep and sleep disorders. *Sleep medicine reviews*. 2012. Vol. 16, No. 1. P. 47–66. DOI: <https://doi.org/10.1016/j.smrv.2011.02.005>.
24. Walch O., Huang Y., Forger D., Goldstein C. Sleep stage prediction with raw acceleration and photoplethysmography heart rate data derived from a consumer wearable device. *Sleep*. 2019. Vol. 42, No. 12. Art. zsz180. DOI: <https://doi.org/10.1093/sleep/zsz180>.
25. Chen C. X., Li S. X., Ho C. S. et al. Associations of psychological resilience with macro- and microstructures in NREM and REM sleep in adolescents. *International journal of clinical and health psychology*. 2025. Vol. 25, No. 2. Art. 100570. DOI: <https://doi.org/10.1016/j.ijchp.2025.100570>.
26. Ren W., Zhang N., Sun Y. et al. The REM microarousal and REM duration as the potential indicator in paradoxical insomnia. *Sleep medicine*. 2023. Vol. 109. P. 110–117. DOI: <https://doi.org/10.1016/j.sleep.2023.06.011>.
27. Attoh-Mensah, E., Igor-Gaez, I., Vincent, L., Bessot, N., Nathou, C., & Etard, O. (2023). Cardiorespiratory changes associated with micro-arousals during naps. *Neurobiology of Sleep and Circadian Rhythms*, 14, 100093. <https://doi.org/10.1016/j.nbscr.2023.100093>

REFERENCES

1. Liu, P. Y., & Reddy, R. T. (2022). Sleep, testosterone and cortisol balance, and ageing men. *Reviews in Endocrine & Metabolic Disorders*, 23(6), 1323–1339. <https://doi.org/10.1007/s11154-022-09755-4>
2. Feeney, S. P., McCarthy, J. M., Petruconis, C. R., & Tudor, J. C. (2025). Sleep loss is a metabolic disorder. *Science Signaling*, 18(881), eadp9358. <https://doi.org/10.1126/scisignal.adp9358>
3. Franken, P., & Dijk, D. J. (2024). Sleep and circadian rhythmicity as entangled processes serving homeostasis. *Nature Reviews Neuroscience*, 25, 43–59. <https://doi.org/10.1038/s41583-023-00764-z>

4. Xu, Y., Schneider, A., Wessel, R., et al. (2024). Sleep restores an optimal computational regime in cortical networks. *Nature Neuroscience*, 27, 328–338. <https://doi.org/10.1038/s41593-023-01536-9>
5. Tempesta, D., Soccia, V., De Gennaro, L., & Ferrara, M. (2018). Sleep and emotional processing. *Sleep Medicine Reviews*, 40, 183–195. <https://doi.org/10.1016/j.smrv.2017.12.005>
6. Van Someren, E. J. W. (2021). Brain mechanisms of insomnia: New perspectives on causes and consequences. *Physiological Reviews*, 101(3), 995–1046. <https://doi.org/10.1152/physrev.00046.2019>
7. Dressle, R. J., & Riemann, D. (2023). Hyperarousal in insomnia disorder: Current evidence and potential mechanisms. *Journal of Sleep Research*, 32(6), e13928. <https://doi.org/10.1111/jsr.13928>
8. Werner, G. G., Schabus, M., Blechert, J., & Wilhelm, F. H. (2021). Differential effects of REM sleep on emotional processing: Initial evidence for increased short-term emotional responses and reduced long-term intrusive memories. *Behavioral Sleep Medicine*, 19(1), 83–98. <https://doi.org/10.1080/15402002.2020.1713134>
9. Rho, Y. A., Sherfey, J., & Vijayan, S. (2023). Emotional memory processing during REM sleep with implications for post-traumatic stress disorder. *The Journal of Neuroscience*, 43(3), 433–446. <https://doi.org/10.1523/JNEUROSCI.1020-22.2022>
10. Repantis, D., Wermuth, K., Tsamitros, N., et al. (2020). REM sleep in acutely traumatized individuals and interventions for the secondary prevention of post-traumatic stress disorder. *European Journal of Psychotraumatology*, 11(1), 1740492. <https://doi.org/10.1080/20008198.2020.1740492>
11. Machado, R. B., & Suchecki, D. (2016). Neuroendocrine and peptidergic regulation of stress-induced REM sleep rebound. *Frontiers in Endocrinology*, 7, 163. <https://doi.org/10.3389/fendo.2016.00163>
12. Pavlova, I., & Rogowska, A. M. (2023). Exposure to war, war nightmares, insomnia, and war-related posttraumatic stress disorder: A network analysis among university students during the war in Ukraine. *Journal of Affective Disorders*, 342, 148–156. <https://doi.org/10.1016/j.jad.2023.09.003>
13. Rogowska, A. M., & Pavlova, I. (2023). A path model of associations between war-related exposure to trauma, nightmares, fear, insomnia, and posttraumatic stress among Ukrainian students during the Russian invasion. *Psychiatry Research*, 328, 115431. <https://doi.org/10.1016/j.psychres.2023.115431>
14. Boiko, D. I. (2024). Sleep problems in combatants with posttraumatic stress disorder and its acceptance. *Visnyk Problem Biologii i Medytsyny* (Bulletin of Problems in Biology and Medicine), 2(173), 165–174. <https://doi.org/10.29254/2077-4214-2024-2-173-165-174>
15. Kurapov, A., Schabus, M., Kahveci, S., et al. (2024). Explaining post-traumatic stress symptoms and sleep disturbance in Ukrainian civilians: Perceived threat versus objective war exposure. *European Journal of Psychotraumatology*, 15(1), 2381371. <https://doi.org/10.1080/20008066.2024.2381371>
16. Chinoy, E. D., et al. (2021). Performance of seven consumer sleep-tracking devices compared with polysomnography. *Sleep*, 44(5), zsa291. <https://doi.org/10.1093/sleep/zsa291>
17. Kainec, K. A., Caccavaro, J., Barnes, M., et al. (2024). Evaluating accuracy in five commercial sleep-tracking devices compared to research-grade actigraphy and polysomnography. *Sensors*, 24(2), 635. <https://doi.org/10.3390/s24020635>
18. Herberger, S., Aurnhammer, C., Bauerfeind, S., et al. (2025). Performance of wearable finger ring trackers for diagnostic sleep measurement in the clinical context. *Scientific Reports*, 15, 9461. <https://doi.org/10.1038/s41598-025-93774-z>
19. de Zambotti, M., et al. (2022). Rigorous performance evaluation for informed use of new technologies for sleep health measurement. *Sleep Health*, 8(3), 263–269. <https://doi.org/10.1016/j.sleh.2022.02.006>
20. Schyvens, A. M., Peters, B., Van Oost, N. C., et al. (2025). A performance validation of six commercial wrist-worn wearable sleep-tracking devices for sleep stage scoring compared to polysomnography. *Sleep Advances*, 6(2), zpaf021. <https://doi.org/10.1093/sleepadvances/zpaf021>
21. Trinder, J., Kleiman, J., Carrington, M., et al. (2001). Autonomic activity during human sleep as a function of time and sleep stage. *Journal of Sleep Research*, 10(4), 253–264. <https://doi.org/10.1046/j.1365-2869.2001.00263.x>
22. Halász, P., Terzano, M., Parrino, L., & Bódizs, R. (2004). The nature of arousal in sleep. *Journal of Sleep Research*, 13(1), 1–23. <https://doi.org/10.1111/j.1365-2869.2004.00388.x>
23. Stein, P. K., & Pu, Y. (2012). Heart rate variability, sleep and sleep disorders. *Sleep Medicine Reviews*, 16(1), 47–66. <https://doi.org/10.1016/j.smrv.2011.02.005>
24. Walch, O., Huang, Y., Forger, D., & Goldstein, C. (2019). Sleep stage prediction with raw acceleration and photoplethysmography heart rate data derived from a consumer wearable device. *Sleep*, 42(12), zsz180. <https://doi.org/10.1093/sleep/zsz180>
25. Chen, C. X., Li, S. X., Ho, C. S., et al. (2025). Associations of psychological resilience with macro- and microstructures in NREM and REM sleep in adolescents. *International Journal of Clinical and Health Psychology*, 25(2), 100570. <https://doi.org/10.1016/j.ijchp.2025.100570>
26. Ren, W., Zhang, N., Sun, Y., et al. (2023). The REM microarousal and REM duration as the potential indicator in paradoxical insomnia. *Sleep Medicine*, 109, 110–117. <https://doi.org/10.1016/j.sleep.2023.06.011>
27. Attoh-Mensah, E., Igor-Gaez, I., Vincent, L., Bessot, N., Nathou, C., & Etard, O. (2023). Cardiorespiratory changes associated with micro-arousals during naps. *Neurobiology of sleep and circadian rhythms*, 14, 100093. <https://doi.org/10.1016/j.nbscr.2023.100093>

Чернолевський Б. О., Севериновська О. В.

ДИНАМІКА ВІДНОВЛЕННЯ АРХІТЕКТУРИ СНУ ТА ВЕГЕТАТИВНІ РЕАКЦІЇ ПІСЛЯ НІЧНОГО ВОЄННОГО СТРЕСУ

Сон є ключовим механізмом емоційної регуляції та нейрофізіологічної адаптації, при цьому REM-фаза відіграє провідну роль у переробці стресового досвіду. В умовах воєнних конфліктів цивільне населення зазнає гострого нічного стресу, що може порушувати стабільність сну. Водночас об'єктивні дані щодо мікроструктурних змін REM-сну в природних умовах залишаються обмеженими, особливо у випадках, коли макроархітектура сну залишається відносно стабільною.

Метою дослідження було проаналізувати нейрофізіологічні закономірності відновлення сну після гострого нічного стресу та оцінити можливість використання кардіовегетативних показників як непрямих маркерів мікроструктурних порушень сну.

Для оцінки основних структурних та вегетативних показників сну було проведено проспективне натуралістичне дослідження із використанням носимих пристроїв для багатоденного моніторингу сну. Проаналізовано п'ятиденні цикли, що включали фонову ніч, ніч стресової події та період відновлення. Для перевірки інформативності кардіовегетативних сигналів в рамках дослідницького спостереження за окремим випадком було використано паралельну реєстрацію з портативною ЕЕГ-системою.

Встановлено, що макроархітектура REM-сну, зокрема його загальна тривалість, залишалася відносно стабільною після стресової події, тоді як на мікроструктурному рівні спостерігалася її дестабілізація. Обґрунтовано можливість використання кардіовегетативних реакцій як проксі-маркерів мікроароузлів, що відкриває перспективи дистанційної оцінки фрагментації сну без застосування полісомнографії.

Виявлено, що відновлення структури сну після гострого стресу має інерційний характер: найбільш виражені порушення спостерігаються у перші дві ночі, тоді як нормалізація функціональних взаємозв'язків відбувається на третю ніч. Підтверджено, що носимі пристрої можуть бути використані для довготривалого моніторингу вегетативної динаміки сну, однак мають обмеження у детекції нічних пробуджень. Отримані результати можуть бути використані для розробки підходів до раннього виявлення порушень сну, зумовлених стресом.

Ключові слова: *нейрофізіологія сну; REM-фаза; мікроароузали; мікроструктура сну; вегетативна регуляція; носимі смарт-трекери; постстресове відновлення.*

Надійшла до редакції / Received: 08.04.2026

Схвалено до друку / Accepted: 11.05.2026