Sleep Health: Journal of the National Sleep Foundation 11 (2025) 335–343

Contents lists available at ScienceDirect

Sleep Health: Journal of the National Sleep Foundation

journal homepage: www.sleephealthjournal.org

Sleep homeostasis occurs in a naturalistic setting

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ARTICLE INFO

Article history:
Received 24 September 2024
Received in revised form 16 December 2024
Accepted 12 January 2025

Keywords:
Synaptic homeostasis
Slow wave
NREM
Sleep pressure
Spectral exponents

ABSTRACT

Background: Sleep, especially non-rapid eye movement sleep depth is homeostatically regulated, as sleep pressure builds up during wakefulness and diminishes during deep sleep. Previous evidence from this phenomenon, however, mainly stems from experimental studies which may not generalize to an ecologically valid setting.
Methods: In the current study, we used a dataset of 246 individuals sleeping for at least seven nights each with a mobile electroencephalography headband according to their ordinary daily schedule to investigate the effect of time spent in wakefulness on sleep characteristics.
Results: Increased time in wakefulness prior to sleep was associated with decreased sleep onset latency, increased sleep efficiency, a larger percentage of N3 sleep, and higher delta activity. Moreover, increased sleep pressure resulted in an increase in both the slope and the intercept of the sleep electroencephalography spectrum. As predicted, power spectral density effects were most prominent in the earliest hours of sleep.
Conclusion: Our results demonstrate that experimental findings showing increased sleep depth after extended wakefulness generalize to ecologically valid settings, and that time spent awake is an important determinant of sleep characteristics on the subsequent night. Our findings are evidence for the efficacy of sleep restriction, a behavioral technique already widely used in clinical settings, as a simple but powerful method to improve the objective quality of sleep in those with sleep problems.

Introduction

The concept of homeostasis covers the innate mechanisms of organisms that enable them to maintain steady internal states in a changing external environment.[1] The concept of sleep homeostasis was coined to explain empirical evidence suggesting that several aspects of sleep depend on the recent sleep-wake history of the organism, thus sleep characteristics could be involved in maintaining the regulated balance between sleep and waking.[2] A now widely received complete model of sleep characteristics is the two-process model.[3,4] In the two-process model, both homeostatic and circadian processes operate to regulate sleep and wakefulness. Homeostatic pressure for sleep builds up during wakefulness, while the phase of the circadian process determines the sleep threshold at which sleep is initiated or terminated. An explanatory model, known as the synaptic homeostasis hypothesis, highlights the overall level of synaptic strengths within the brain as the neurophysiological mechanism behind sleep homeostasis.[5] In this model, sleep pressure reflects the overall level of synaptic strengths within the central nervous system, whereas the function of sleep is to downscale these strengths. According to this model, synaptic potentiation is an inevitable consequence of wakefulness, whereas deep sleep (and more specifically, the slow waves that occur in deep sleep) serve synaptic downscaling, enabled by low levels of noradrenergic activity.

Typical studies investigating the homeostatic regulation of sleep focus on recovery sleep after an experimentally scheduled extension of prior wakefulness[6,7] or the overnight dynamics of sleep as reflecting dissipating sleep pressure.[8,9] Other commonly used research designs to unravel sleep homeostasis are the experimental extension of sleep opportunity by scheduled daytime naps[10,11] or enforced bedrest[12] and the non-24-hour day protocols known as forced desynchrony settings.[13] In each of these designs, changes in sleep metrics after the experimental manipulation of the duration of wakefulness are interpreted as evidence for sleep homeostasis.

Based on experimental research, several features of sleep macrostructure and electroencephalography (EEG) indeed exhibit finely graded homeostatic regulation. The propensity to initiate sleep (sleep onset latency - SOL)[14,15] total sleep time (TST),[16] non-rapid eye movement (NREM) sleep duration,[16] sleep efficiency,[15,17] slow wave sleep time and percentage,[15,18,19] and particularly slow wave activity (SWA, spectral power in the 0.75-4.5 Hz range)[20] were shown to reflect at

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https://doi.org/10.1016/j.sleh.2025.01.007

least in part the sleep-wake history of the organism. SWA is an indicator of sleep intensity that reflects a restorative function of sleep.[2] Recent evidence indicates a gradual leveling off of excess EEG power along the frequency scale, with lower frequencies showing stronger wake time-dependent upregulation. Consequently, sleep-wake history was best reflected by the steepness of the spectral slope of aperiodic activity in the sleep EEG.[21] In other words, a steeper (downward) slope of the sleep EEG spectrum toward the higher frequencies, indicating higher SWA, is an indicator of sleep pressure.

Despite the strong focus on sleep homeostasis in academic research,[3,4] virtually all evidence for it stems from experimental studies. While experimental studies are powerful tools to demonstrate causality, they may suffer from deficiencies in ecological validity and their findings may not generalize to real-world settings.[22] In the specific case of sleep homeostasis, as recordings of undisturbed, natural sleep were virtually never analyzed to demonstrate this phenomenon, it is possible that the relatively drastic manipulations of wake time introduced in experimental studies are poor models of the much more modest day-to-day variability of sleep pressure, and in real-world settings sleep parameters are more robust to slight changes in the characteristics of the preceding wakefulness.

Here we aimed to fill this gap by analyzing the within-person covariation of wakefulness duration and subsequent sleep in a sample of healthy volunteers using wearable EEG-headbands on consecutive nights in an ecologically valid naturalistic setting. Our goal was to translate the concept of sleep homeostasis, conceived in laboratory-based intervention studies, to an ecologically valid setting and to demonstrate that small natural fluctuations in wake duration also influence sleep characteristics in line with the two-process model.

Our hypothesis was that natural variations in the duration of wakefulness result in changes in subsequent sleep comparable to those observed in experimental studies. Specifically, we hypothesized that increased time in wakefulness results in (1) increased overall sleep propensity, shown by reduced SOL and sleep fragmentation as well as an increase in the relative proportion of deep sleep, (2) increased low-frequency activity in the sleep EEG as a marker of homeostatic restorative processes, especially in the SWA range and in the first hours of sleep, and (3) in line with the previous hypothesis, a steeper spectral slope.

Methods

Participants

We used data from the Budapest Sleep, Experiences and Traits Study (BSETS) a multiday observational study. The full protocol of BSETS has been published separately.[23] In short, BSETS participants were community-dwelling volunteers who tracked their life for at least seven consecutive days, including an array of demographic, psychological and psychiatric questionnaires, diaries about daily and nightly experiences, and an EEG recording of each night sleep, in order to discover the relationship between sleep and daily experiences in a causally informative time-lagged within-participant design exploiting natural variation in daily experiences and sleep patterns. BSETS was designed to be radically ecologically valid, hence, minimal inclusion criteria were applied. Participants were recruited by branched diffusive convenience sampling in nonclinical settings. Participants, often students of Semmelweis University, were encouraged to include family members, friends and romantic partners to participate. No experimental interventions or behavioral limitations were given, participants were free to schedule their daily activities and their sleep as they chose. The mean age of the sample was 29.15 years (SD = 12.82), with 55% females and 45% males, with a distinctly bimodal age distribution with a peak at the age of students (< 25 years old) and their parents (46-55 years old).

The current study relies on night EEG recordings and subjective sleep quality reported the following morning. Data availability depended on the outcome of interest in each model. After discarding first-night recordings (see Statistical analysis) and artifactual EEG data (see EEG) 1358 nights of sleep macrostructure from 247 participants, 1316 nights of subjective sleep quality from 245 participants, and 1031 nights of quantitative EEG from 219 participants was available.

The Institutional Review Board (IRB) of Semmelweis University, as well as the Hungarian Medical Council (under 7040-7/2021/ EÜIG "Vonások és napi események hatása az alvási EEG-re" [“The effect of traits and daily activities and experiences on the sleep EEG”]), approved BSETS as compliant with the latest revision of the Declaration of Helsinki. All participants gave written informed consent on a form reviewed and approved by the IRB.

Electroencephalography

BSETS participants were issued a Dreem2 mobile EEG headband[24,25] and trained in its use to record their sleep at their homes for at least 7 consecutive nights. Dreem2 uses dry silicone electrodes to record brain activity with a 250 Hz sampling frequency and bandpass filtering at 0.4-18 Hz, and a validated complimentary algorithm[24,25] uses the resulting waveforms to score the vigilance state of participants. Based on these scorings, the following sleep macrostructure metrics were extracted: SE, TST, SOL, wake after sleep onset (WASO), the number of awakenings, and the latency, duration, and percentage of N1, N2, N3, and rapid eye moevment (REM) sleep. Sleep macrostructure metrics are often bounded at perfect values (e.g., SOL at 0 and SE at 100%), resulting in a skewed distribution. In order to make these variables more appropriate for linear analyses, we applied an outlier exclusion using the generalized form of Grubb’s test (implemented with the isoutlier() MATLAB function).[26] We briefly report on findings without this data cleaning procedure (see Results).

For quantitative EEG (qEEG) analysis, we used the channel F7-O1 based on preliminary analyses[23] showing a good tradeoff between this channel’s data quality and its ability to record topographically widespread activity such as slow waves. For qEEG analyses, we used a complimentary algorithm to automatically detect artifactual EEG epochs on a 2-second basis. Based on preliminary analyses[23] and in line with published BSETS protocols[23,26] epochs were discarded if this algorithm estimated artifact probability as greater than 25%. If the proportion of nonartifactual epochs for a night was below 20%, the night was discarded entirely. Data availability is illustrated on Fig. 1.

We used the periodogram() function in MATLAB EEGLab with 2-second nonoverlapping epochs and Hamming windows to perform spectral analysis. Power spectral density (PSD) estimates were averaged across epochs for each night and log10-transformed. PSDs of each night were visually inspected for abnormalities, and if they were detected, the night was discarded.[23] Band-wise spectral values were calculated by averaging power values within the following frequency ranges: delta (0.5-4 Hz), theta (4-7 Hz), alpha (7-10 Hz), low sigma (10-13 Hz), high sigma (13-16 Hz), and beta (16-25 Hz) by averaging.

Spectral parametrization

We decomposed absolute spectra into periodic and aperiodic components using FOOOF 1.1.0 (“Fitting Oscillations & One Over f,” available at https://github.com/fooof-tools/fooof).[27] The power law function was estimated in the 3-20 Hz range. We allowed periodic components (spectral peaks) with a width of 1-4 Hz, a minimum peak height of 1 SD, a maximum of 10 peaks, and “fixed” aperiodic mode. We discarded nights where periodic and aperiodic components accounted for less than 95% of the variance in the power

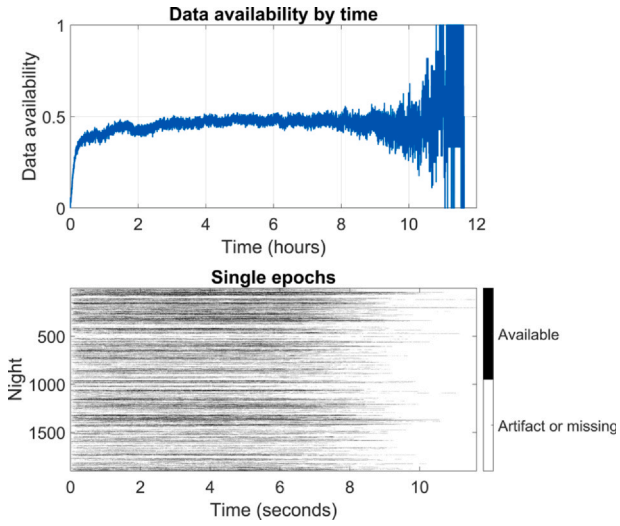

Fig. 1. Data availability in BSETS. The top panel illustrates the proportion of data epochs surviving artifact detection criteria relative to all recorded EEG epochs as a function of time since recording start. The bottom panel illustrates single epochs for all nights. Epochs are marked black if data was available and white if either they did not survive artifact detection criteria or no data was available for that night. EEG, electroencephalography

spectrum (N = 6). We used the spectral intercept and spectral slope as variables of interest indicating increased sleep pressure.

Subjective sleep quality and dreaming

Upon awakening, participants reported their subjectively rated sleep quality using the Hungarian version of the Groningen Sleep Quality Scale (GSQS). Higher scores on this instrument indicate worse sleep. GSQS total were used as the main estimate of subjective sleep quality. In line with previous analyses,[26] we also specify two alternative metrics, the first unscored question of the GSQS ("I had a deep sleep last night"), treated as a binary variable, and an additional question prompting participants to rate their subjective level of restedness on a Likert scale of 1 to 10, which was treated as a continuous variable.

Participants also reported in this diary whether they recall a dream from the night. We used this variable to attempt to replicate previous research[15] which indicated that dream recall is less frequent under increased sleep pressure.

Time spent awake as the indicator of sleep pressure

Sleep pressure builds up steadily during wakefulness.[2] Thus, we estimated the amount of sleep pressure at the beginning of the night as the time since the previous night's last sleep epoch and the current night's first sleep epoch (henceforth referred to as "time spent awake" or "time awake"). The clock time of both was based on EEG measurement.

EEG recordings were only performed at night, so sleep pressure may be overestimated if daytime naps are not considered. Participants recorded in their evening diary if they napped during the day and self-reported the total duration of napping. 297 daytime naps with an average duration of 70.34 minutes were recorded.

Because nap duration may be misreported and naps may be shallower than nighttime sleep, simply excluding self-reported naps from time awake may introduce bias. Therefore, in two alternative specification of the analyses we calculated the effect of time spent awake on sleep variables both with and without excluding naps.

Statistical analysis

We investigated the relationship between sleep pressure and objective and subjective sleep metrics using multilevel models,[28,29] implemented using the fitglme() MATLAB function. Multilevel models are statistical tools which can handle dependent observations (such as multiple nights recorded from the same participant) and can estimate if the effect of a predictor exists at the within-person level (level 1) or the between-person level (level 2), also allowing controlling for covariates at both levels. Within-person effects are more in line with a causal interpretation (see limitations below) as they reflect that the same person's sleep changes after increased sleep pressure, as opposed to between-person effects which merely reflect a correlation observed between participants.[26]

We modeled the effect of sleep pressure by first creating a between-person and a within-person specification of sleep pressure.[28,29] For the between-person specification, the mean amount of time spent awake (across all days) was entered for all observations from the same person. For the within-person specification, deviations from the individual mean were used, with each person's data centered around 0. A separate model was fitted for each sleep macrostructure variable, EEG power band, and subjective sleep metric as dependent variables. For binwise qEEG analyses, a separate model was fitted for each frequency bin. Each model used a random intercept per participant, both the between-person and the within-person specification of sleep pressure as predictors, together with age, sex, a dummy variable for weekend/weekday, and lagged outcomes (the previous day's value of the dependent variable) to control models for these potential confounders. In Wilkinson notation, the equation was:

$$sleep\_metric \sim 1 + weekend + age + sex + time\_awake_{means} + time\_awake_{devs} + sleep\_metric_{lag} + (1|ID)$$

that is, each sleep metric was expressed as a function of a fixed intercept, control variables age, sex and weekend, individual mean wakefulness duration and within-individual deviations from this value, the previous night's sleep metric, and a random intercept per participant.

From each model, the critical result was the within-person regression coefficient: the estimated causal effect of an additional hour of time spent awake on a given person's sleep. Within-person regression coefficients cannot be automatically interpreted as a causal effect of wakefulness duration on sleep due to the possibility of confounding.[30] For example, it is possible that it is not extended wakefulness, but events leading to it (e.g., increased social activity) that cause changes in sleep. Within-person regression coefficients in a time-lagged analysis such as ours, however, severely constrain the possible causal paths compared to cross-sectional and/or between-person analyses. First, because wakefulness duration always precedes sleep, reverse causation (sleep causing increased wakefulness) is impossible. Second, due to the within-person nature of the analyses any confounding must be time-variant. For example, sex, age or personality characteristics cannot confound the associations we found because we show that sleep is altered in the same person after increased wakefulness: anything that confounds this relationship must be something that also varies from one day to the other. Third, our analyses are well-suited to falsify causal associations (with cautions about reductions in power to detect indirect associations). While positive findings can be due to time-variant confounding (the causes of wakefulness, not wakefulness itself, changing sleep), negative findings indicate that wakefulness duration, either directly or via its causes, is not related to sleep characteristics.

The *p*-values of regression coefficients were corrected for multiple testing using the Benjamini-Hochberg method of false discovery rate correction.[31] All nominally significant ($p < .05$) *p*-values

reported in the manuscript also survive this correction unless indicated otherwise.

Because time spent awake (calculated since the end of the first night's sleep) could only be estimated starting on the second night, first night recordings could not be directly used for our analyses. We did use first night recordings, however, to control for lagged outcomes to eliminate spillover effects from the previous nights. For example, a model estimating the effect of time spent awake on WASO is controlled for WASO on the previous night. Controls for lagged outcomes was another separate reason for not using first-night recordings.

Data availability

Raw data is available at https://osf.io/2p8hj/.

Results

Descriptive statistics

The mean time spent awake was 17.05 hours (SD = 2.03). Intraclass coefficients ranged from 0.076 (time spent awake) to 0.718 (alpha power). Detailed descriptive statistics (valid Ns, means, SDs, intraclass correlation coefficients and within- and between-participant correlations are available in the Supplementary File 1).

Sleep macrostructure

Increased time spent awake during the previous day was associated with deeper and less disturbed sleep (Fig. 2). For each additional hour spent awake, SOL was reduced by 0.62 minute and WASO by 1.31 minutes, SE was increased by 0.23% points and 0.5 fewer awakenings were observed.

TST was reduced after increased sleep pressure: however, this is expected as longer wakefulness results in less time to sleep due to the normal obligations of the following day. Consequently, increased sleep pressure was associated with later bedtimes (32 minutes for each additional hour spent awake). Controlling for bedtime reduced the association between sleep pressure and TST by 70% to 4.13 minutes, but it remained statistically significant (p = .002).

The analysis of sleep composition revealed that – mirroring reductions in TST – time spent in N1, N2 and REM was significantly reduced after longer wakefulness, but N3 duration remained unchanged. When we used relative durations, the percentage of N3 sleep was increased, N2 sleep was reduced, while N1 and REM were unchanged.

All effects replicated with only slight variation in the effect sizes after naps were excluded (Table 1).

Quantitative EEG: PSD

Increased time spent awake led to NREM sleep EEG PSD increases in the delta through the low sigma frequency range (Table 2).

In more detailed analyses, we calculated a multilevel model for each 0.5 Hz frequency bin. Fig. 3 illustrates the expected change in the PSD of each frequency bin per one of our additional wakefulness. Increased PSD was most prominent in the ~1-2 Hz frequency range corresponding to slow waves, but the PSD increase remained at least nominally significant into the high alpha-low sigma frequency ranges (Fig. 3).

Quantitative EEG: Bi-hourly PSD

In the next step, we calculated NREM PSD separately from bi-hourly periods (with 1 hour of overlap up to the fifth hour) of recordings to test how homeostatic effects are distributed across the night.

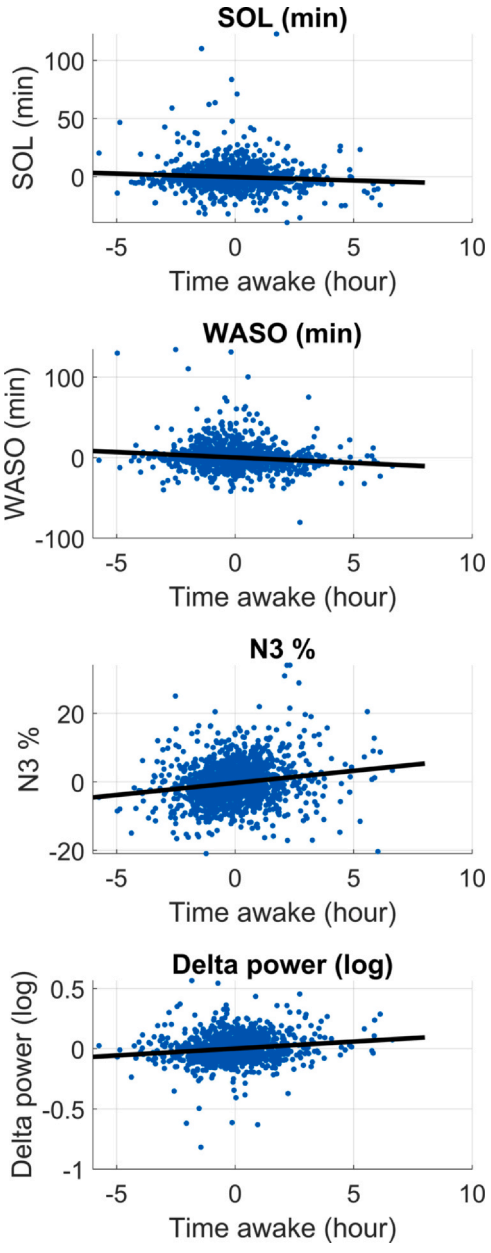

Fig. 2. Selected effects of increased sleep pressure. The scatterplots show the within-participant relationship between time spent awake (horizontal axis) and sleep onset latency (SOL, r = 0.08), wake after sleep onset (WASO, r = − 0.13), sleep stage N3% (N3%, r = 0.17) and delta power (r = 0.15). Both variables are expressed as residuals controlling for age, sex, and day of the week and consequently centered on zero. Pearson correlations displayed before have also been controlled for these covariates. log, logarithm

As the duration of recordings and the distribution of artifact-free NREM sleep was nonuniform across the nights, the total number of available EEG nights varied by hour. We limited analyses for the first 5 hours of recordings in 2-hour overlapping batches. For these batches, a total of 1632, 1606, and 1508 nights (including first nights) were available. Statistical modeling was performed in a manner identical to full-night recordings.

For all periods, a trend for increased low-frequency activity as a function of increased time spent awake was observed. This trend, however, only reached significance across a broad frequency range in the first 2 hours of sleep. After the first 2 hours of sleep, significance was limited to a single isolated bin in the theta ranges.

The results are summarized in Fig. 4.

Table 1
Within-person effects of time spent awake on sleep macrostructure

	Naps included			Naps excluded		
	B	SE	*p*	B	SE	*p*
SOL (min)	−0.616	0.217	.005	−0.633	0.213	.003
Sleep efficiency (%)	0.225	0.097	.021	0.268	0.095	.005
Awakenings (count)	−0.488	0.124	< .001	−0.535	0.119	< .001
WASO (min)	−1.313	0.299	< .001	−1.287	0.283	< .001
TST (min)	−13.518	1.394	< .001	−11.005	1.404	< .001
N1 duration (min)	−0.619	0.141	< .001	−0.637	0.135	< .001
N2 duration (min)	−9.122	0.853	< .001	−7.764	0.837	< .001
N3 duration (min)	−0.325	0.380	.392	−0.008	0.376	.983
REM duration (min)	−1.823	0.625	.004	−1.818	0.614	.003
N1% (%)	0.010	0.031	.755	−0.018	0.030	.541
N2% (%)	−0.785	0.120	< .001	−0.686	0.117	< .001
N3% (%)	0.630	0.122	< .001	0.647	0.119	< .001
REM percentage (%)	0.181	0.115	.117	0.088	0.113	.438

Abbreviations: SOL, sleep onset latency; TST, total sleep time; WASO, wake after sleep onset.
In each line, we show the expected effect of one additional hour spent awake during the previous day on the sleep macrostructure indicators in the first column (B), the standard error (SE) and the p-value of this coefficient. Values are expressed in natural units (in parentheses). All models are controlled for age, sex and day of the week (weekday/weekend). The left half of the table shows effects without and the right half with the inclusion of self-reported naps from time spent awake.

Table 2
Within-person effects of time spent awake on absolute spectral power of the NREM sleep electroencephalography (EEG)

	Naps included			Naps excluded		
	B	SE	*p*	B	SE	*p*
Delta	0.011	0.003	< .001	0.010	0.003	< .001
Theta	0.008	0.002	< .001	0.008	0.002	< .001
Alpha	0.008	0.002	< .001	0.008	0.002	< .001
Low sigma	0.005	0.002	.002	0.004	0.002	.016
High sigma	0.003	0.002	.047 ±	0.002	0.002	.208
Beta	0.003	0.002	.199	0.002	0.002	.262

In each line, we show the expected effect of one additional hour spent awake during the previous day on the power spectral density (PSD) of the EEG band indicated in the first column (B), the standard error (SE) and the p-value of this coefficient. PSD values were log10-transformed before analyses. All models are controlled for age, sex, and day of the week (weekday/weekend). The left half of the table shows effects without and the right half with the inclusion of self-reported naps from time spent awake.
± Indicates a nominally significant ($p < .05$) effect which does not survive correction for multiple testing.

Quantitative EEG: Spectral slopes and intercepts

We found that both spectral slopes and intercepts were significantly increased after increased time spent awake, mirroring results about PSD (Table 3).

Fig. 5 shows the predicted spectral intercept and slope across a range of plausible wakefulness durations.

Subjective sleep quality

We found no significant effect of time spent awake on self-reported subjective sleep quality, either considering GSQS total scores, the first item of the GSQS not counted toward the total score, or self-reported restedness on a Likert scale (Table 4). Thus, although increased sleep pressure resulted in objectively deeper and more efficient sleep, this was not translated into a perception of higher sleep quality the following morning.

Dream recall

We found no compelling evidence that increased sleep pressure reduces the chance of reporting a dream in the morning diary. While the effect sizes were in this direction both with (within-participant odds ratio = 0.94 per hour of wakefulness, $p = .12$) and without (odds ratio = 0.95, $p = .11$) the exclusion of naps, they were not statistically significant. The failure to replicate previous findings may be due to differences in the setting (home vs. laboratory) or the magnitude of additional wakefulness.

Between-participant effects

Our research focused on within-participant effects, that is, how a given participant's sleep changed after varying degrees of wakefulness. However, between-participant effects (associations between average wake duration and average sleep characteristics across participants) are also of interest.

Between-participant effects of wakefulness were very similar to within-participant effects, although less precisely estimated due to fewer observations (Fig. 6).

The duration of sleep and specific sleep stages was much more strongly associated with wakefulness, which is unsurprising as these are zero-sum variables at the between-participant level (on average, a given participant spends a certain amount of time sleeping and the rest awake). However, longer typical wakefulness was also associated with significantly higher delta power, N3%, significantly reduced WASO and awakenings, and a within-participant-like trend for lower SOL, higher SE, higher spectral slopes and intercepts, and higher EEG power even at higher frequency ranges. Notably, despite the zero-sum nature of sleep and wakefulness, typical N3 duration was not affected by typical wake duration, suggesting a compensatory mechanism where the lower absolute amount of sleep is composed of deeper stages. These findings show that homeostatic processes operate on the between-participant level as well, and – even after controlling for age – participants with longer typical wakefulness tend to have increased sleep propensity.

Outlier effects and channel choice

We elected to exclude observations with extreme values to make analyses more amenable for linear analyses (see Methods). In Supplementary File 2, we report regression results without this correction. Outlier exclusion made minimal effect on the results, and except for affecting marginal significance in the case of two between-participant effects it was limited to changes in the signs of otherwise nonsignificant findings. Results were also replicated if non-normally distributed sleep metrics (sleep efficiency, WASO, and SOL) were subjected to a transformation instead of artifact exclusion (see also[32] for a discussion of non-normality in BSETS variables). WASO and SOL were log-transformed while sleep efficiency was reflected (SEt = log10(100-SE)) and then log transformed. Increased sleep duration still had substantial associations with reduced SOL ($B = -0.014$, $p = 10^{-6}$), WASO ($B = -0.02$, $p = 10^{-8}$), while the effect on sleep efficiency was nonsignificant ($B = -0.006$, $p = .068$, note the inversion of the coefficient sign due to reflecting the variable).

Our results were based on the F7-O1 channel. At a reviewer's suggestion, we replicated findings using F8-O2 instead. Using this channel, PSD in all spectral bands was still increased as a function of increased wakefulness duration, with or without excluding naps ($p < .01$, except high sigma and beta [$p = .017$ and $p = .018$ respectively]).

Discussion

Electrophysiological evidence for sleep homeostasis has previously only been sourced from experimental studies. This is potentially problematic as experiments in the biomedical sciences often fail to translate into ecologically valid settings.[33] In the current case, it is possible that while drastic experimental manipulations in

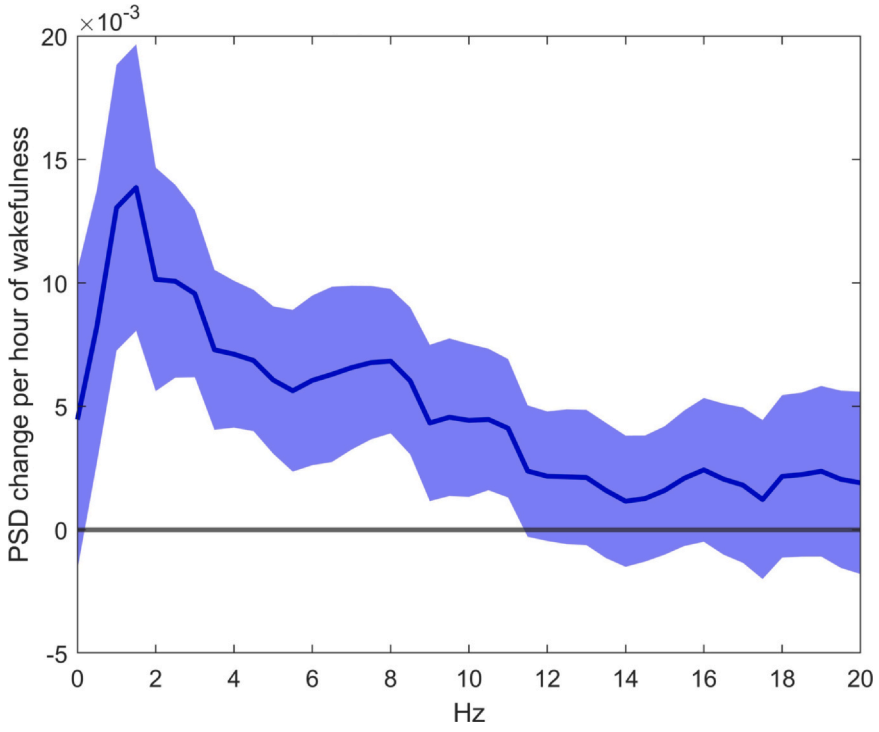

Fig. 3. Within-person effects of time spent awake on NREM sleep electroencephalography (EEG) power spectral density (PSD) as a function of frequency (Hz). The chart shows the within-person estimates of the effect of one additional hour spent awake during the previous day on the NREM sleep EEG PSD of each frequency. PSD values were log10-transformed before analyses. All models are controlled for age, sex, and day of the week (weekday/weekend). The shaded area shows 95% confidence intervals

wake duration can affect sleep characteristics, the effect of much smaller variations in wake duration experienced by a typical person are overpowered by other determinants of sleep, such as stress,[34,35] learning or cognitive load,[36] auditory stimulation,[37] or meals.[38,39] In the current study, we show that experimental studies of sleep homeostasis indeed replicate in an ecologically valid, naturalistic

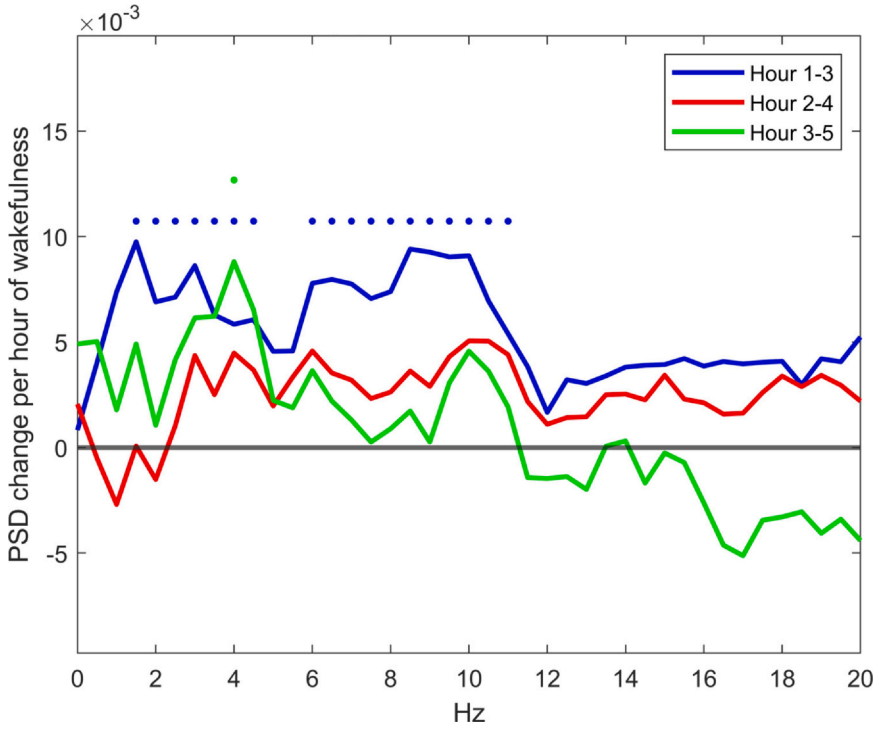

Fig. 4. Within-person effects of time spent awake on NREM sleep electroencephalography (EEG) power spectral density (PSD) as a function of frequency (Hz) and hours of sleep. The chart shows the within-person estimates of the effect of one additional hour spent awake during the previous day on the NREM sleep EEG PSD of each frequency, calculated separately for three bi-hourly, overlapping period of recordings (separate lines). PSD values were log10-transformed before analyses. All models are controlled for age, sex, and day of the week (weekday/weekend). Dots above the lines indicate that the effect is significant after correcting for multiple comparisons in the hour indicated by the line with the matching color

Table 3
Within-person effects of time spent awake on aperiodic spectral parameters

	Naps included			Naps excluded		
	B	SE	*p*	B	SE	*p*
Intercept	0.014	0.004	< .001	0.013	0.004	< .001
Slope	0.009	0.004	.018	0.009	0.004	.011

In each line, we show the expected effect of one additional hour spent awake during the previous day on the spectral intercepts and slopes, as indicated in the first column (B), the standard error (SE) and the p-value of this coefficient. All models are controlled for age, sex, and day of the week (weekday/weekend). The left half of the table shows effects without and the right half with the inclusion of self-reported naps from time spent awake.

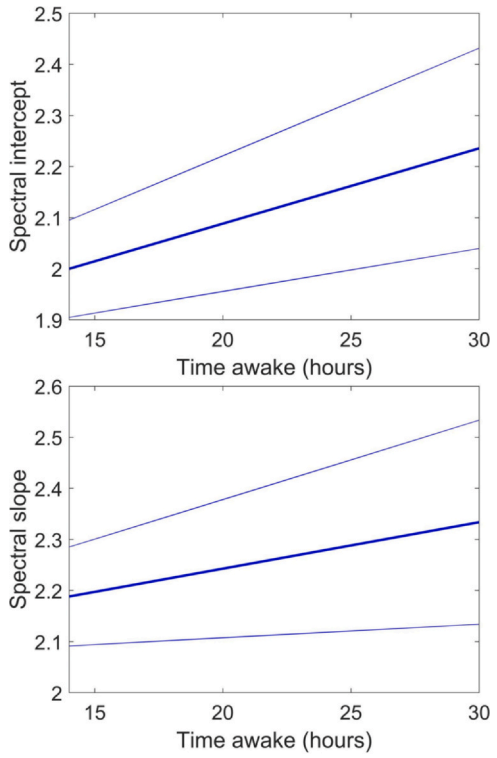

Fig. 5. Predicted values of the spectral intercept (left) and spectral slope (right) as a function of the duration of previous wakefulness

Table 4
Within-person effects of time spent awake on three measures of self-reported sleep quality: Groningen Sleep Quality Scale (GSQS) total score, the first unscored GSQS item, and self-reported restedness

	Naps included			Naps excluded		
	B	SE	*p*	B	SE	*p*
GSQS	0.020	0.057	.722	0.011	0.055	.847
GSQS1	−0.009	0.048	.854	0.003	0.046	.941
Restedness	0.010	0.038	.788	−0.019	0.037	.611

For the first GSQS item, the linear effect is shown for simplicity, although this estimate is derived from logistic regression as this is a binary variable. In each line, we show the expected effect of one additional hour spent awake during the previous day on the sleep quality indicators in the first column (B), the standard error (SE) and the p-value of this coefficient. Values are expressed in raw scores. All models are controlled for age, sex, and day of the week (weekday/weekend). The left half of the table shows effects without and the right half with the inclusion of self-reported naps from time spent awake.

study, and sleep characteristics change as a function of sleep-wake history within the normal range.

Longer wakefulness resulted in an increased propensity for sleep, especially of deep sleep and slow waves. Furthermore, time spent awake translated into an increased propensity for sleep initiation, in coherence with laboratory studies.[6–8] Specifically, longer presleep wakefulness was followed by reduced SOL, increased SE and higher N3% at the expense of other sleep stages, as well as decreased WASO and fewer awakenings. Spectral analyses replicated experimental laboratory studies[6–8] and reveal that increased wakefulness results in increased NREM sleep PSD, most prominently at the lowest frequencies but extending up to the sigma range. Binwise statistics revealed that effects transcend the canonical SWA range,[21] extend over higher frequencies, but increasing EEG frequencies are characterized by decreasing sensitivity to extensions in presleep wakefulness. Moreover, both the NREM sleep EEG spectral slope and intercept values increased as a function of time awake, supporting the relevance of these indices in depicting sleep homeostasis.

Importantly, we found no evidence that increased wakefulness duration also resulted in improvements in subjectively rated sleep quality the next morning. We also could not replicate a previous finding[15] that increased sleep pressure results in a reduced likelihood of dreaming.

Sleep characteristics are to some extent habitual or trait-like.[40] This was also observed in our dataset (Supplementary File 1): on average, 41% of the variance of macrostructure variables, 66% of PSD values, 57% of spectral parameters, and 21% of subjective sleep quality reports was at the within-participant level, showing that the same participant experiences similar sleep across nights. Between-participant analyses using habitual sleep metrics generally replicated the within-participant effects of time awake on sleep propensity and structure. Participants with habitually longer periods of wakefulness were characterized by higher N3%, delta power, as well as by significantly reduced WASO and number of awakenings, with trends in line with within-participant effects for most other sleep metrics as well. These findings suggest that while fluctuations in a person's sleep propensity can reflect fluctuations sleep pressure, habitual sleep patterns may also be partially the result of the habitual duration and intensity of wakefulness.

Our results have important implications for both sleep research and somnology. The replication of laboratory studies of sleep homeostasis provides not only evidence for the two-process model, but also for the feasibility of conducting sleep research outside of the laboratory. Effect sizes were moderate (e.g., within-person correlation between wakefulness duration of delta power r = 0.15, see Fig. 2 for further standardized within-person effects), highlighting the necessity for a large sample in detecting the effect of small fluctuations of wakefulness on sleep. Using wearable EEG devices at home is substantially less cost and labor intensive than standard laboratory PSG studies and this approach may result in larger, better-powered studies to test hypotheses about sleep regulation and function with relatively little compromise toward fidelity. Studying impairments of sleep regulation in a naturalistic setting might especially be useful to understand the mechanisms behind insomnia, depression, and circadian rhythm disorders.

A special case where sleep homeostasis under natural conditions is important is sleep restriction therapy (SRT), commonly used to alleviate symptoms of insomnia. In SRT, patients are instructed to reduce time spent asleep (or attempting to sleep) to increase sleep pressure and the depth and efficiency of subsequent sleep. A recent meta-analysis of SRT randomized controlled trials[41] supported the efficacy of SRT to reduce the insomnia severity index, SOL and WASO, but this evidence was rated as low quality due to the low number of studies, between-study heterogeneity, and lack of long-term follow-up. Our study using a causally informative non-randomized controlled trial design confirms that SRT might be a promising therapy for sleep problems, and that increasing time spent awake may be a highly efficacious way to increase sleep quality. Caution must be warranted, however, as SRT was shown to negatively affect vigilance and sleepiness[42] suggesting that while sleep following SRT is biologically more efficient, its restorative properties might be missing. In line with these observations, we also found that next-morning subjective sleep quality was not significantly improved after longer time spent awake. Wearable

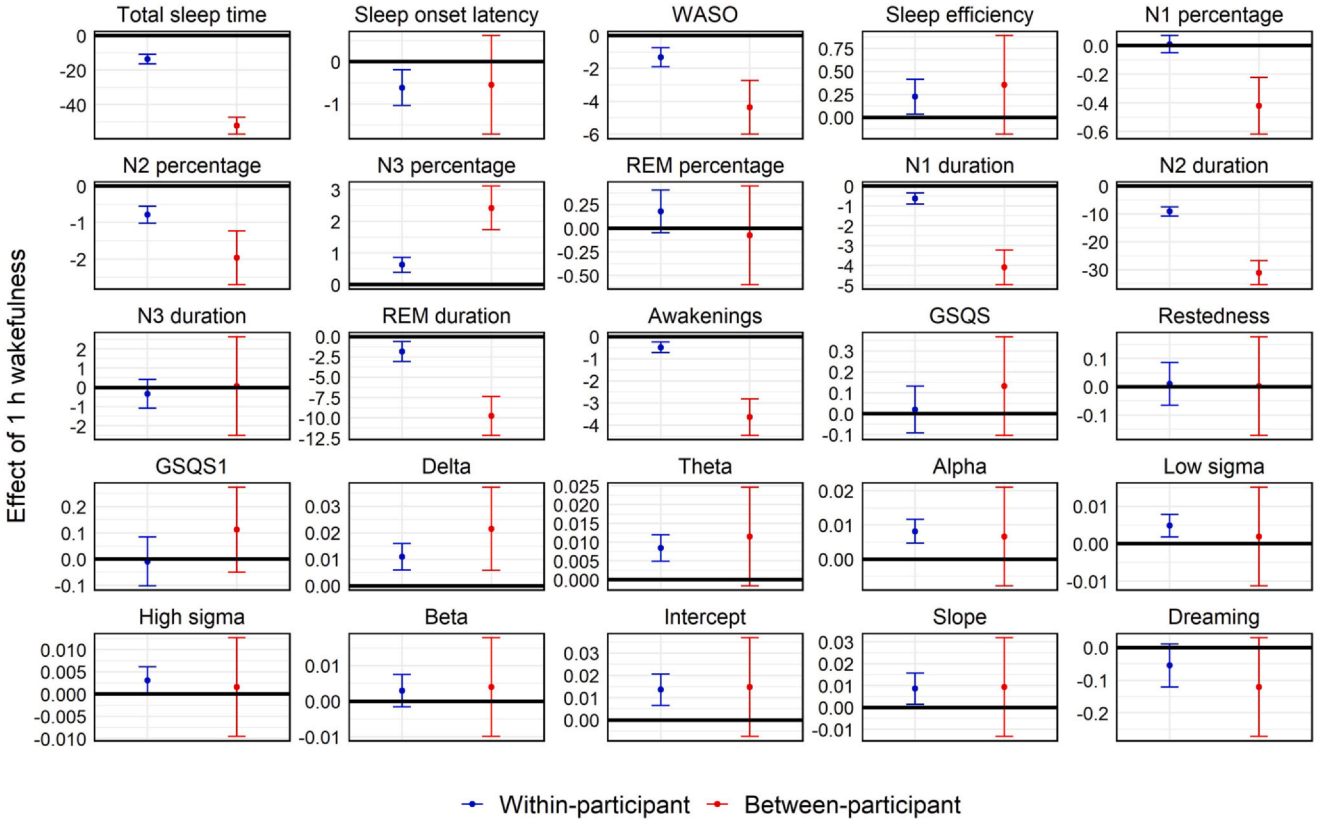

Fig. 6. Comparison of within- and between participants effects. Each tile shows the estimated within- and between-participant effects of 1 hour of wakefulness (without taking into account naps) on sleep characteristics. Estimates originate from multilevel models adjusted for age, sex and day of the week. Error bars show 95% confidence intervals. GSQS, Groningen Sleep Quality Scale; WASO, wake after sleep onset

instruments could be used in clinical settings to fine-tune SRT in way that balances the efficiency and restorative property of sleep in individual patients.

Our work has limitations. First, ours was a relatively young volunteer sample without clinically significant sleep complaints or other medical issues. The findings may not generalize to older populations or those with significant sleep problems, requiring replication. Second, our study relied on a mobile EEG headband instead of gold-standard PSG to measure sleep. While the general reliability of the method has been demonstrated in previous studies, sleep structure may be better determined using PSG. This limitation especially affects qEEG findings, as the Dreem2 headband has strong hardware filtering above 18 Hz and the accurate mapping of higher frequencies is not possible with this method. This especially concerns our analysis of beta-frequency oscillations, which should therefore be treated with caution. Another limitation of our mobile EEG headband is its limited topographical sampling of brain activity. While our findings were robust to the choice of channel, other (e.g., frontal or prefrontal channels with mastoid or earlobe references) channels might be better suited for the analysis of homeostatic effects. Third, we could imperfectly model events in wakefulness which may affect the amount of sleep pressure that was generated. While we had self-reported data on naps which did not substantially affect findings, an ideal study would include actigraphic measures of the day which could precisely model sleep pressure by taking into account the exact duration of napping and physical activity.

In sum, our findings demonstrate that validity of the two-process model in the description of day-to-day natural variations in sleep. Similarly to experimental manipulations, a naturally longer wakefulness period also results in increased sleep propensity and depth but no change in subjective sleep quality, informing clinical interventions aimed to improve sleep.

Author contributions

PPU: Conceptualization, Theoretical framework, Methodology, Writing – original draft, review and edits of revisions, Obtaining funding, parent study data collection. **RB**: Conceptualization, Theoretical framework, Formal analysis, Writing – original draft, review and edits of revisions.

Funding

Research supported by the National Research, Development and Innovation Office – NKFIH (grant number: 138935), by the Culture and Innovation Ministry of Hungary (TKP2021-EGA-25, TKP2021-NKTA-47).

Declaration of conflicts of interest

All authors declare no conflict of interest.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.sleh.2025.01.007.

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