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# Spectral EEG markers of the drives for wake and sleep in afternoon naps as the correlates of subjective and objective measures of daytime sleepiness

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**Abstract** Despite our capability to consciously perceive sleepiness, the consensus on the method of its objective measurement is not reached. The complex nature of this transitional state between wake and sleep states might be one of explanations of the failure to validate subjective sleepiness self-assessments against objective sleepiness measures, such as spectral electroencephalographic (EEG) powers. Our previous pilot study showed that the disconnect between excessive daytime sleepiness (EDS) assessed with Epworth Sleepiness Scale (ESS) and measured as Latency to Sleep Onset (SOL) can be linked to their differential relationship with the opposing drives for sleep and wake. To try to replicate this result, we enlarged the sample of nap study from 27 to 80 university students and found again that ESS score and min of SOL were differentially linked to scores on the first and second principal components of the EEG spectrum that are the indicators of these two opposing sleep–wake regulatory processes. Thus, a stronger sleep drive and a weaker opposing wake drive might be the major contributors to the subjective and objective indicator of EDS, respectively. The results are discussed in light of findings of questionnaire studies indicating a relatively high prevalence of ESS-based EDS among university students.

## 1 Introduction

Complex systems are occasionally switching between several qualitatively different modes of behavior, even in the absence of external influences [1]. An example of such mode-switching behavior of a complex system is a sequence of changes in sleep stages observed on approximately 90-min interval of sleep cycle [2, 3]. Therefore, the concepts from complex systems can be applied to the physiology of sleep–wake regulation [4]. It seems that even the transitional states between wake and sleep states, such as sleepiness, have complex nature. Indeed, sleepiness is a paradoxical vigilance substate for which the consensus on the method of its objective measurement has not been reached so far, despite the universal ability of humans to consciously perceive sleepiness [5, 6].

Research of the subjective concept of sleepiness produced many controversial questions, including such questions as whether the physiological component of subjective feeling of drowsiness can be precisely defined in scientific terms, and what can be its reliable physiological marker [7–11]. Despite this, standardized questionnaires for sleepiness self-assessment are often used in clinical studies as an alternative of the well-established method of objective evaluation of sleepiness, the Multiple Sleep Latency Test (MSLT), that seem to be impracticable due to the requirement of attendance at a sleep laboratory for many hours [12, 13].

The 8-item Epworth Sleepiness Scale (ESS) [14] serves as the most popular questionnaire tool for diagnostic of excessive daytime sleepiness (EDS) in clinical populations [12, 15]. For instance, an umbrella review of the

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literature on sleepiness as a complex construct revealed that the ESS was the first among 6 most frequently cited sleepiness assessment tools (from 99 tools in total) [16]. However, the physiologic underpinnings of this scale are not well understood [17, 18]. In particular, one of the consistent findings of the studies aimed on validation of the ESS against objective measures of sleepiness is a lack of the expected close association between subjective sleepiness assessed as a score on the ESS and objective sleep propensity measured in MSLT that is mean Latency to Sleep Onset (SOL) [19–25].

The complexity of the transitional state of sleepiness might, at least partly, explain the disconnect between two most popular subjective and objective indicators of EDS, ESS score and SOL. In a number of recent publications, this complexity was recognized in the concept of multi-dimensionality of sleepiness construct [18, 26–37].

Besides, it can be hypothesized that one of dimensions of sleepiness is closer related to one of several different components of the complex mechanism of sleep–wake regulation, while another dimension of sleepiness is closer related to another component of this mechanism. Originally, the authors of two-process model of sleep–wake regulation postulated that two—circadian and homeostatic—processes are underlying the alternations between two major—wake and sleep—states of the sleep–wake cycle [38, 39] and that slow-wave activity (SWA) can serve as a spectral electroencephalographic (EEG) indicator of the homeostatic process or, in other terms, the drive for sleep [40–42]. It was also proposed that the circadian process simply modulates the time course of this indicator [43, 44]. However, several researchers in the field of chronobiology also postulated that the circadian process can be interpreted as the wake drive that opposes the sleep drive, i.e., the homeostatic process, in determination of the pattern of cyclicity of wake and sleep states [45, 46]. The hypothetical mechanism proposed by biological rhythm research resembles the complex neurobiological mechanism postulated by sleep researchers. This mechanism is designed to control the transitions between different substates of the sleep–wake continuum that, ultimately, might be delineated as alternations between opposing processes, i.e., the processes promoting arousal and inhibiting sleep and the processes promoting sleep and inhibiting arousal [47–49]. Moreover, it was hypothesized that there are two opposing processes of sleep–wake regulation, the drives for wake and sleep, which parameters are modulated by the circadian clock [50–52]. The time courses of two spectral EEG indicators of these drives, scores on the 1st and 2nd principal components of the EEG spectrum, can be traced across sleep cycles [52–54], multiple nap episodes [51, 55], night sleep episode [11, 56, 57], afternoon napping attempts [58, 59], extension of wakefulness behind habitual sleep time [50, 60], and transitions between wake and sleep states after various intervals of preceding wakefulness [61–63].

Previously, we addressed the issue of disagreement between an objective sleep propensity measured by SOL and subjective sleepiness assessed with the ESS asking the following question: what can be the underlying causes of this disagreement and can be these causes defined by measurement of spectral sleep EEG indexes during afternoon napping attempt? [58] We hypothesized that these two sleepiness assessments are disconnected due to their differential relationship with the antagonistic drives for wake and sleep, respectively, measured with the spectral EEG indicators of these drives (i.e., scores on the first and second principal components of the EEG spectrum, respectively). In a pilot study of 50-min napping attempts of 27 university students, this hypothesis was supported. We showed that the spectral EEG indicators of the sleep and wake drives, scores on the first and second principal components, respectively, are positively correlated with ESS score and min of SOL, respectively. It was concluded that a stronger drive for sleep (i.e., a larger first principal component score) and a weaker opposing drive for wake (i.e., a larger second principal component score) significantly contribute to a larger ESS score and a longer SOL, respectively [58].

Here, we increased the size of sample of nap study to 80 university students to test replicability of this preliminary result. Our hypothesis was that, during 50 min of afternoon napping attempt, EDS determined by ESS score and minutes of SOL is differentially associated with scores on the first and second principal component of the EEG spectrum that are the spectral EEG markers of the sleep and wake drive, respectively. If ESS score is positively associated with score on the first rather than second principal component, SOL is positively associated with score on the second rather than first principal component.

## 2 Methods

### 2.1 Participants

Unpaid volunteers of this study were 40 male and 40 female university students enrolled in physiology courses with mean age ± standard deviation of 20.40 ± 1.56 and 20.25 ± 1.14 years, respectively.

The structured interview preceded the invitation to participate in the study and to choose the dates of three afternoon naps. The interview was focused on the following exclusion criteria. Age either younger than 18 or older than 23 years, history of mental or sleep disorder, any complaints about poor physical condition and functioning, current mild cold and missing classes due to any sickness in two previous weeks, involvement in shift or night work and crossing several time zones in the previous month, irregularity of sleep–wake schedule exemplified by more

than one-h difference in weekday bedtimes, frequent sleep reduction exemplified by, at least, one night of partial sleep deprivation in the previous week. The exclusion criteria for female students additionally included pregnancy or breastfeeding, and they were also asked about the day of last menstruation and usual cycle's length.

### 2.2 Study protocol

Each visit to a sleep laboratory was preceded and followed by attending classes in the same university building. During one month, each study participant was invited to have a napping attempt three times. The intervals between naps varied from three days to three weeks. Each visit to the sleep laboratory was scheduled at the same afternoon hour (not earlier than 12:30 and not later than 15:30). Each visit lasted at least one hour. Since the first napping attempt was regarded an adaptation nap, the analysis of the polysomnographic records was limited to Nap 2 and Nap 3.

### 2.3 Polysomnographic recordings

During the preparation to polysomnographic recordings and throughout the time interval for recording, a participant was lying in bed in a room of the sleep laboratory. He/she was instructed to try to relax and fall sleep after light off to sleep for, at least, 50 min. The recordings were performed using a Neurovisor BMM-36 (Medical Computer Systems LLC, Moscow), the MCScap Sleep electrode helmet, and the NeoRec 1.4 software. The electrodes were applied in accord with the standard monitoring montage known as the International 10–20 system of electrode placement [64].

The EEG signals were obtained from 19 channels connected by a monopolar 10–20 scheme with two reference electrodes on the mastoid bones. Other than the EEG signals, the polysomnographic recording included the signals from two electrooculogram channels, one electromyogram channel, and one electrocardiogram channel. The EEG signals were conditioned by the high-pass, low-pass, and notch filters (0.5 Hz, 35 Hz, and 50 Hz frequencies, respectively). The sampling frequency was 1000 Hz.

### 2.4 Sleep scoring

In accord with the conventional scoring procedure [65] (https://aasm.org/clinical-resources/scoring-manual/), visual scoring on 30-s epochs of each record was performed independently by two experienced scorers. The initial disagreement, depending upon a stage, varied from 10% (N1) to 2% (N3). In order to finally produce consensus scores, the scorers reexamined together all intervals with discrepant scores. Since only the unique number was assigned to each analyzed record, the scorers were uninformed about order of nap and name or ID of study participant. The 30-s epochs were classified into 5 stages: wake stage (W), three stages of NREM sleep (N1, N2, N3), and rapid-eye-movement (REM) sleep (R).

### 2.5 Spectral analysis of the EEG signals

The spectral EEG power densities were calculated from data on the EEG signals recorded from electrodes placed at 5 derivations (Fz, F4, Cz, Pz, and O2 referenced to the ear mastoid sites, M1/M2). The records of the signals from each of these derivations were visually inspected on 1-s epochs to remove all epochs containing artifacts from further analysis. Spectral power densities for the artifact-free epochs were computed using the FFTW (Fastest Fourier Transform in the West) package [66] (see also www.fftw.org for more detail). With few exceptions, mean spectra for each 30-s epoch were obtained by averaging over as many as 20–30 one-sec spectra. Only very rare, the number of averaged spectra was lower, between 5 and 20, due to excluding spectra affected by movements.

Further analysis of these spectra was limited to the first 16 single-Hz frequency bandwidths, between 1 and 16 Hz (i.e., 0.50–1.49 Hz for 1 Hz, 1.50–2.49 Hz for 2 Hz, 2.50–3.49 Hz for 3 Hz, ..., 15.50–16.49 Hz for 16 Hz). These sets of 16 single-Hz power densities were averaged within each 30-s interval of EEG records, ln-transformed, and assigned to their stages. For statistical analysis of EEG signals for separate sleep stages, the individual sets of spectral powers (100 per each derivation of each of two napping attempts) were further averaged over derivations. Moreover, these 16 ln-transformed single-Hz power densities were finally averaged within four 4-Hz frequency ranges, delta (1–4 Hz), theta (5–8 Hz), alpha (9–12 Hz), and sigma (13–16 Hz).

### 2.6 Analysis of principal component structure of the EEG spectrum

The $SPSS_{23.0}$ statistical software package (IBM, Armonk, NY, USA) was used for all analyses including principal component analysis of the sets of 16 ln-transformed single-Hz power densities (1–16 Hz) from each of 5 derivations.

Scores on the 1st and 2nd principal components of variation in the EEG power spectra were calculated and averaged in a similar way as the sets of 16 ln-transformed single-Hz powers and spectral powers in 4 frequency ranges.

Using the 16 ln-transformed single-Hz power densities, scores on the 1st and 2nd principal components were calculated. The 16 ln-transformed power densities were weighted and summed:

$$\text{PC score} = \Sigma\, w_i * p_i, \tag{1}$$

where $p_i$ is a power density for a i-th frequency bin, and $w_i$ is a loading of this principal component on this i-th frequency bin (i = 1 Hz, 2 Hz, ..., and 16 Hz).

Depending upon derivation, the variation explained by the 1st and 2nd principal components varied from 50 to 55% and from 19 to 32%, and their eigenvalues varied from 7.9 to 8.8 and from 3.1 to 5.1, respectively.

The pattern of loadings of 16 single-Hz spectral powers on each of principal components was almost identical for 5 derivations. The loadings of alpha frequencies on both components were positive, the loadings of delta frequencies on the 1st or the 2nd principal component were positive and negative, respectively, the loadings of sigma and theta frequencies on the 1st principal component resembled those for delta frequencies (positive), and the loadings of sigma frequencies on the 2nd principal component resembled those for alpha frequencies (positive).

### 2.7 Questionnaire

Before each napping attempt, the study participants completed the 8-item Epworth Sleepiness Scale (ESS) for the determination of level of daytime sleepiness [14]. The ESS [14] quantifies the likelihood to fall asleep in each of 8 different daily life situations with a scale ranging from 0 to 3, where 0 corresponds to none and 3 to the situation when dozing off is the most likely.

The total score ranges from 0 to 24. Values above 10 are considered to be indicative for significant (excessive) sleepiness. The psychometric properties of the ESS have been investigated on multiple occasions (e.g., [67]). Its internal consistency (Cronbach's Alpha) varies between 0.73 and 0.90 [68]. Cronbach's alphas from clinical and nonclinical groups in 46 publications (63 estimates comprising 92,503 participants) was about 0.82 [69]. Test–retest reliability of the Epworth Sleepiness Scale in clinical trial settings was found to attain the values of 0.82 for weeks 14 and 26/27, 0.85 for weeks 26/27 and 39/40 (n = 463), and 0.78 for weeks 14 and 39/40 (n = 463) [70].

### 2.8 Statistical analysis

Three- and four-way repeated measure ANOVAs (rANOVAs) were run to test significance of main effect of independent factor "EDS group" (without and with EDS diagnosed with either ESS score or SOL), independent factor "Sex" (male and female students), and repeated measure "Nap" (the 2nd and 3rd nap). "Time" (5 10-min intervals of nap) was additional repeated measure in four-way rANOVAs. Mauchly's test was conducted to assess the sphericity and, if necessary, the Greenhouse-Geiser correction was used to adjust the degrees of freedom, but the original degrees of freedom are reported in Table 1. The results of these rANOVAs are also illustrated in Figs. 1, 2 and 3.

## 3 Results

### 3.1 Correlation between two EDS indicators

For the 2nd and 3rd napping attempts, test–retest reliability of ESS score and SOL (Spearman's rho) attained the values of 0.928 and 0.814, respectively ($p < 0.001$). Correlation of ESS score with SOL (Spearman's rho) in these 2nd and 3rd napping attempts was non-significant (−0.117 and −0.122, respectively). Figure 2C, D illustrates ESS score and SOL in min calculated for ESS scores $< 10$ and $\geq 10$ and for SOL $< 10$ and $\geq 10$ min, that are the divisions applied in the present analysis of principal component scores (Table 1; Figs. 1, 2A, B).

### 3.2 Association of two EDS indicators with two largest principal component scores

The results of rANOVAs of scores on the first and second principal component scores suggested that ESS score and min of SOL were differentially linked to these scores (Table 1). Significant main effect of ESS-based EDS (ESS score $\geq 10$) was found for the first principal component of the EEG spectrum that is an indicator of the strength of the sleep drive. In contrast, significant main effect of SOL-based EDS (SOL $< 10$ min) was found for the second principal components that is an indicator of the opposing wake drive (Table 1; Figs. 1, 2). Moreover, there was

**Table 1** F-ratio from three-way and four-way rANOVAs

| Factors and interactions | 1. "ESS"/"SOL" | | 2. "Sex" | | 3. "Nap" | | 1. × 2. "Sex" | | 1. × 3. "Nap" | |
|---|---|---|---|---|---|---|---|---|---|---|
| The 1st factor | "ESS" | "SOL" | "ESS" | "SOL" | "ESS" | "SOL" | "ESS" | "SOL" | "ESS" | "SOL" |
| Three-way rANOVAs of sleepiness measures and sleep–wake stages | | | | | | | | | | |
| ESS score | 152.5*** | 5.1* | 3.7 | 1.7 | 1.5 | 1.4 | 0.1 | 0.0 | 0.4 | 0.1 |
| SOL, min | 0.2 | 53.9*** | 0.0 | 1.5 | 0.1 | 0.0 | 0.2 | 1.0 | 0.3 | 0.9 |
| W, min | 0.9 | 53.5*** | 0.1 | 0.8 | 0.3 | 0.2 | 0.6 | 0.1 | 0.3 | 0.3 |
| N1, min | 4.1* | 2.7 | 0.3 | 7.5** | 4.1* | 4.6* | 1.3 | 1.0 | 4.1* | 2.6 |
| N2, min | 0.5 | 12.1** | 4.9* | 4.5* | 2.3 | 1.8 | 0.4 | 0.1 | 0.1 | 1.2 |
| N3, min" | 3.6 | 16.3*** | 0.4 | 2.3 | 0.4 | 0.9 | 0.6 | 0.5 | 3.6 | 0.1 |
| Four-way rANOVAs of scores on the 1st and 2nd principal components (PC1 and PC2 score) | | | | | | | | | | |
| PC1 score | 4.1* | 1.0 | 5.1* | 4.8* | 0.6 | 0.2 | 0.9 | 0.8 | 0.2 | 2.9 |
| × 4. "Time" | 0.5 | 12.9*** | 2.2 | 2.4* | 2.1 | 2.1 | 1.9 | 1.0 | 0.5 | 0.4 |
| PC2 score | 0.3 | 40.5*** | 1.2 | 3.4 | 1.3 | 1.0 | 0.1 | 3.4 | 0.0 | 0.8 |
| × 4. "Time" | 0.9 | 4.3** | 0.8 | 0.9 | 1.0 | 1.1 | 0.4 | 0.2 | 0.6 | 0.8 |

Notes. Upper part: Three-way rANOVAs of sleepiness measures (ESS score and SOL, min) and sleep–wake stages (Stages W, N1, N2, and N3). 1. "ESS" /"SOL" (either ESS scores $< 10$ and $\geq 10$ or SOL $< 10$ and $\geq 10$ min) is the 1st independent factor. 2. "Sex" (Male and Female) is another independent factor. 3. "Nap" (Nap 2 and Nap 3) is the repeated measure. Lower part: Four-way rANOVAs of scores on the 1st and 2nd Principal Components of the EEG spectrum (PC1 and PC2) with the additional repeated measure 4. "Time" (10 5-min intervals of polysomnographic record). For scoring subjective daytime sleepiness, the 8-item Epworth Sleepiness Scale (Johns,1991) was administered prior to each of napping attempts. Other measures were obtained by the analysis of 50-min interval of polysomnographic record during these attempts. Mauchly's test was conducted to assess the sphericity and, if necessary, the Greenhouse-Geiser correction was used to adjust the degrees of freedom, but the original degrees of freedom are reported in this table. Level of significance for $F_{9/684}$ (any of interactions with 4. "Time") or for $F_{1/76}$ (all other interactions and main effects of each of factors): *$p < 0.05$, **$p < 0.01$, ***$p < 0.01$

a significant interaction of SOL-based EDS with time interval of napping attempt (Table 1). As expected, scores more rapidly increased when SOL was shorter (Fig. 1).

### 3.3 Association of two EDS indicators with amount of sleep stages

Such a rapid increase of scores after shorter SOL was, also as expected, associated with a larger amount of sleep in the napping attempt (Table 1; Fig. 3). The expected increase of stage 3 sleep in study participants with higher ESS score did not reach significant level (Table 1; Fig. 3).

## 4 Discussion

Sleepiness is viewed as a paradoxical vigilance substate for which the consensus on the method of its objective measurement is not reached, despite our capability to consciously perceive sleepiness. The dominating approach

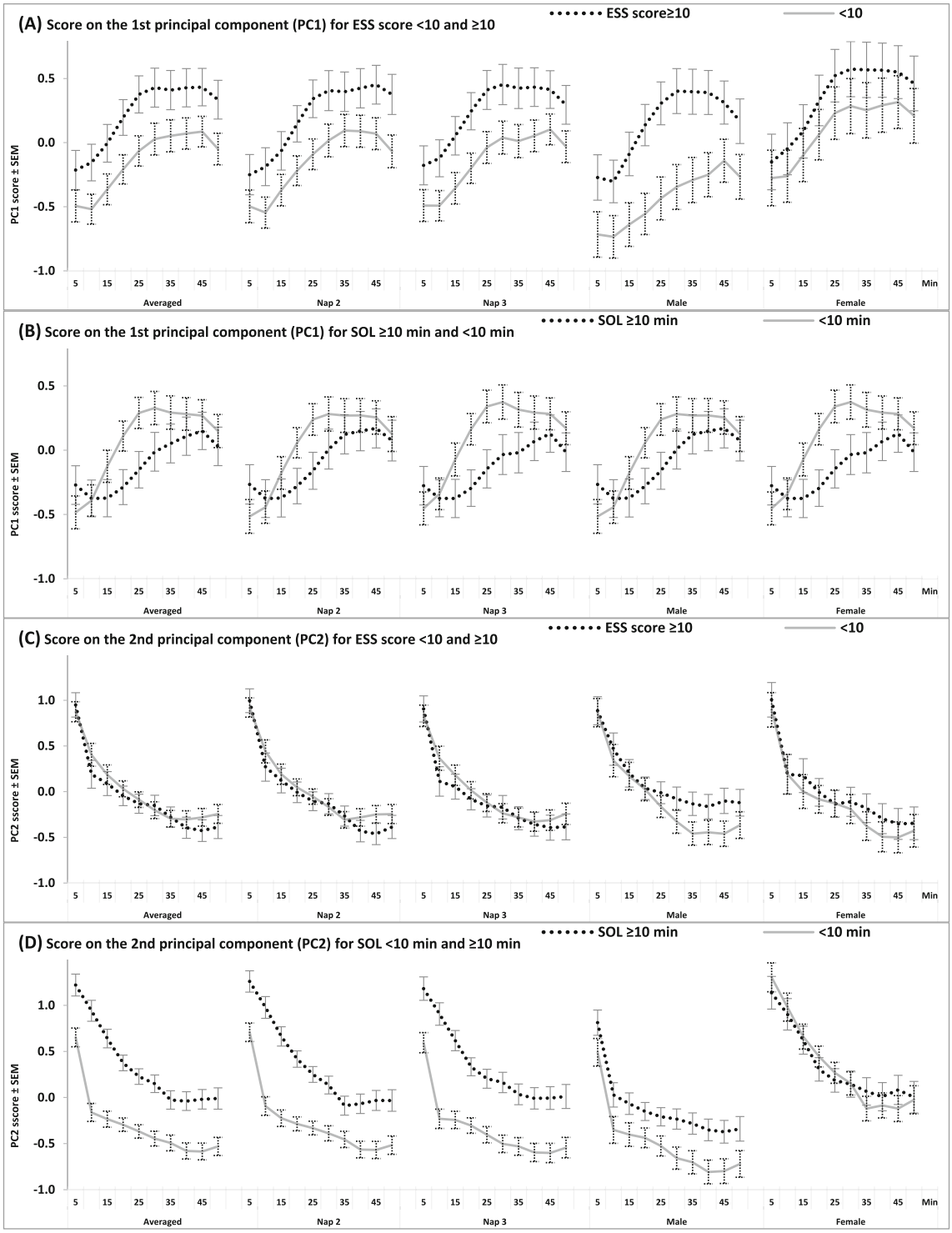


**Fig. 1** Time courses of two principal component scores across 5-min intervals. Scores on the 1st and 2nd principal components were calculated by applying principal component analysis to the whole set of 1600 30-s spectra (i.e., two 100-epoch records of each nap of 80 study participants). The scores obtained for 30-s spectra were averaged within 10 5-min intervals of each record and further averaged by running four-way rANOVA with the 1st independent factor "ESS" /"SOL" (either ESS scores $< 10$ and $\geq 10$ or SOL $< 10$ and $\geq 10$ min) and 10 5-min intervals as repeated measure (Table 1, lower part). The 1st principal component score for ESS scores $< 10$ and $\geq 10$ (**A**) and SOL $< 10$ and $\geq 10$ (**B**). The 2nd principal component score for ESS scores $< 10$ and $\geq 10$ (**C**) and for SOL $< 10$ and $\geq 10$ min (**D**)

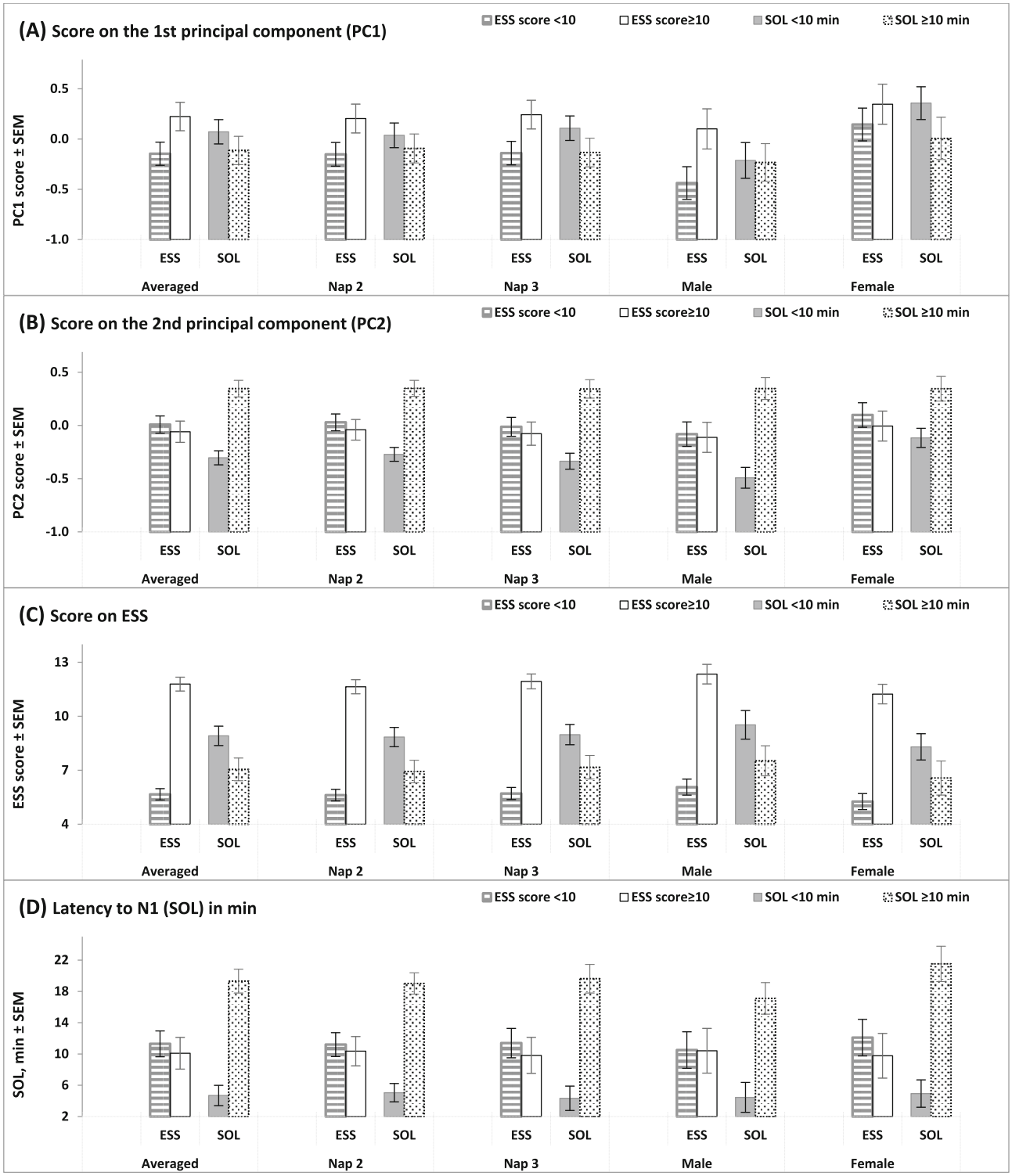


**Fig. 2** Two principal component scores, ESS score and SOL. **A**–**D** The 1st and the 2nd principal component score, ESS score and SOL in min, respectively, for ESS scores $< 10$ and $\geq 10$ and for SOL $< 10$ and $\geq 10$ min. Calculated by running four-way rANOVAs with the 1st independent factor "ESS"/"SOL" (Table 1). See also the legend to Fig. 1

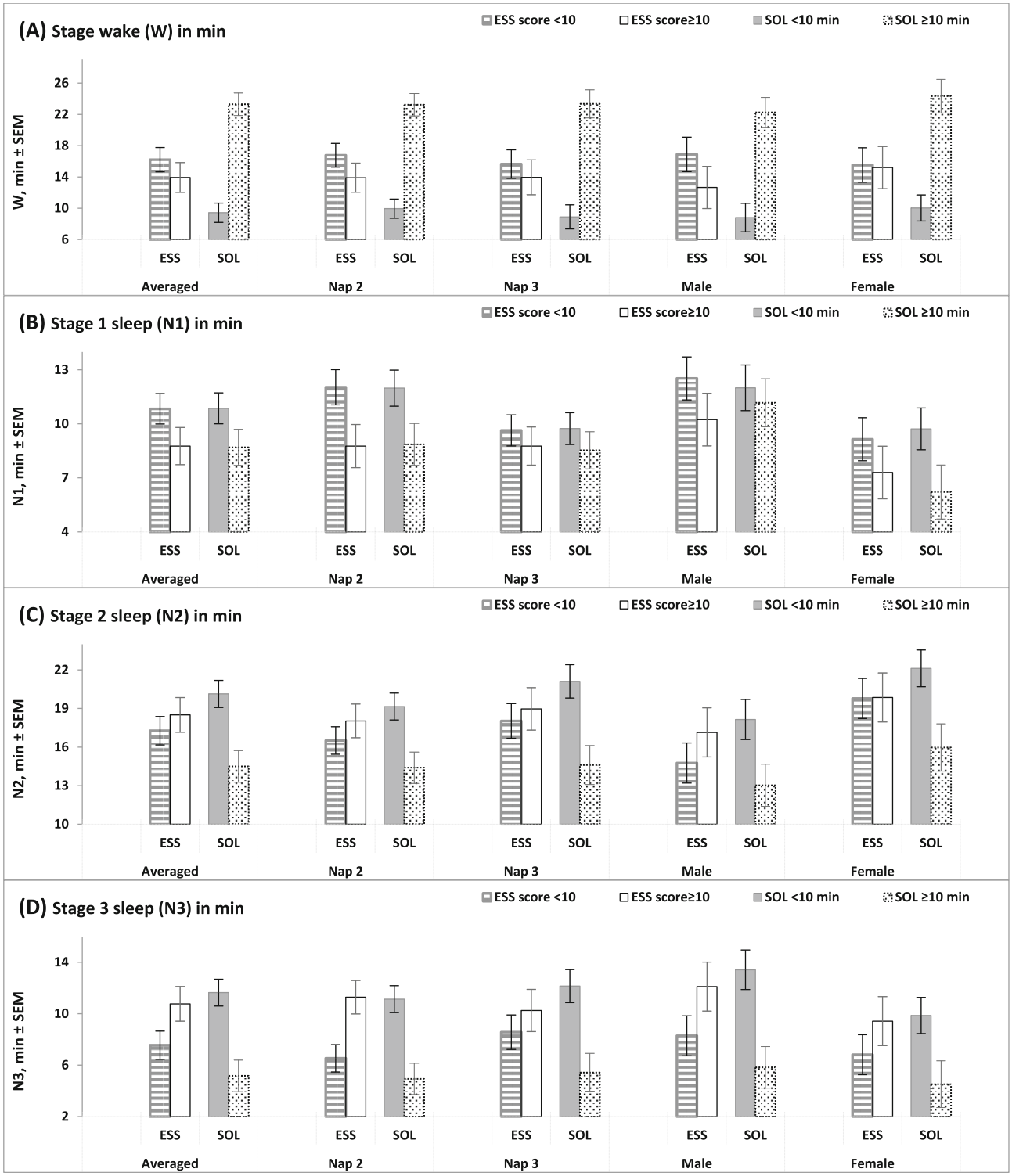


**Fig. 3** Four sleep–wake stages. **A**–**D** Wake (W), Stage 1 sleep (N1), Stage 2 sleep (N2), and Stage 3 sleep (N3), respectively, for ESS scores $< 10$ and $\geq 10$ and for SOL $< 10$ and $\geq 10$ min. Calculated by running three-way rANOVAs with the 1st independent factor "ESS"/"SOL" (Table 1, upper part). See also the legend to Fig. 1

to evaluation of sleepiness—sleepiness self-assessment,—based on our ability to perceive it, contrasts with the conventional methods of elaboration of the well-discriminated sub-states of sleep—their scoring based on polysomnographic measurement. The attempts to validate the subjective sleepiness self-assessments against objective (e.g., spectral EEG) sleepiness measures were often unsuccessful. This can be explained by complex nature of this transitional state between wake and sleep states. Here, we tested the hypothesis that the disconnect between the ESS-based and SOL-based measurements of EDS can be explained by the differential relationship of these subjective and objective indicators of sleepiness, respectively, with the antagonistic drives for sleep and wake, respectively. The following results of the nap study of 80 university students seem to support this hypothesis. We demonstrated that such a subjective measure of EDS as ESS score was significantly related to the EEG marker of the sleep dive strength rather than to the EEG marker of the wake drive strength, while the EEG marker of the wake dive strength rather than the EEG marker of the sleep dive strength was significantly related to such an objective measure of EDS as SOL. Therefore, the disconnect between the ESS and SOL might be, at least partly, explained by such differential relationship of these sleepiness measures with the sleep and wake drives.

Although spectral power density in delta range is regarded the classical marker of the sleep drive, [40–42] the present and previous findings [51–63] supported an interpretation of this index as a marker of mutual influence of the drives for wake and sleep, i.e., an increase/decrease of power density in delta rage might be explained by either a weakening/strengthening of the former or a strengthening/weakening of the latter or both. Such interpretation of the spectral EEG indexes is in line with the theoretic framework of sleep regulation research postulating that the transitions between different substates of the sleep–wake continuum are governed by the complex neurobiological mechanism that, ultimately, might be delineated as alternations between opposing processes promoting sleep and inhibiting arousal and promoting arousal and inhibiting sleep, respectively [47–49].

There is an important practical aspect of the methodology for subjective assessment of sleepiness and interpretation of results of such assessment in populations of university students. A method of quick and user-friendly evaluation of sleepiness has obvious implications for health care [12, 13]. However, the question arises: can we trust the results of such a self-assessment in light of reliable findings indicating low correlation between subjective and objective measures of sleepiness? The present results of nap study provided support for a significant association of a higher ESS score, a subjective indicator of EDS, and such an objective indicator of a higher sleep pressure as a higher score on the first principal component score in the afternoon napping attempt.

Moreover, the results of the present nap study agree with the results of our previously published online survey of these students [71]. In this survey with a much larger number of participants from the same population, we addressed the following question: what are the possible causes of high rate of EDS reported by university students? The results indicated that chronic sleep deprivation on weekdays is a significant contributor to an elevated ESS score. Therefore, we concluded that, although it is known that individual variation in ESS score shows the expected impact of genetic component (i.e., [72]), the results of questionnaire studies in a population of university students pointed at weekday sleep insufficiency rather than an abnormally high prevalence of predisposition to pathological ESD as the most reasonable explanation of a relatively high prevalence of ESS score $> 10$ among university students.

Consequently, the finding of questionnaire studies indicating high rates of the ESS-based EDS among university students can be trusted because such rates seem to reflect chronical sleep deprivation during the days of attending classes leading to an elevated level of sleep pressure during afternoon nap.

There are limitations to our dataset. The profound change in sleep–wake characteristics across ages does not allow the generalization of the results to the whole lifespan. Since all participants were Russian university students enrolled in physiology courses, the present results require confirmation with samples representing university students of other faculties, younger (school and college) students, workers and older adults from different countries around the globe.

## 5 Conclusions

Here, we tried to replicate the results of our preliminary study of differential relationship of subjective and objective indexes of EDS with the spectral EEG indicators of the drives for sleep and wake (i.e., scores on the first and second principal components of the EEG spectrum, respectively). We enlarged the sample of nap study of university students ($n = 80$) to confirm that an ESS score and min of SOL are differentially linked to these spectral EEG indicators of these two opposing sleep–wake regulatory processes. As in the previous pilot study ($n = 27$), the present results pointed at a difference between the possible underlying causes of two distinct aspects of sleepiness measured with the ESS and SOL. If a stronger sleep drive might be suggested to underlie a higher ESS score (i.e., a subjective indicator of EDS), a stronger opposing drive for wake might be suggested to underlie a longer SOL (i.e., an objective indicator of EDS).

## Author contributions

Conceptualization AAP; Funding acquisition DSS, and VBD; Data curation DSS, DES, ANP, NVL, AOT, ONT, EOG, AEM, EAC, IAY, ZVB, EBY, OVM, VIT, and VBD, Resources AAP, DSS, and VBD; Project administration AAP and VBD; Supervision AAP; Software DSS, DES, ANP, and AAP; Investigation DSS, VBD, ANP, and AAP; Methodology AAP, VBD, and ANP; Sleep scoring: ANP and EOG; Validation AAP; Visualization AAP; Writing, review & editing AAP, DSS, DES, ANP, NVL, AOT, ONT, EOG, AEM, EAC, IAY, ZVB, EBY, OVM, VIT, and VBD.

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**Data availability** The datasets analyzed during the current study are available from the corresponding author on reasonable request.

**Declarations**

**Conflict of interest** The authors declare no competing interests. The funders had no role in the design of the study, the collection, analyses, or interpretation of data, the writing of the manuscript, and the decision to publish the results.

**Informed consent** The study participants were informed in detail about the nap study and informed consent was obtained from each participant.

**Institutional Review Board** The study was conducted in accordance with the Declaration of Helsinki, and approved by the Ethics Committee of the Institute of Higher Nervous Activity and Neurophysiology (Approval#12402-02-7112 from 03.06.2019).

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