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Resting-state electroencephalography (EEG) microstates of healthy individuals following mild sleep deprivation

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Mild sleep deprivation is widespread in many societies worldwide. Electroencephalography (EEG) microstate analysis provides information on spatial and temporal characteristics of resting brain network, serving as an indicator of neurophysiological activities at rest. This study seeks to investigate potential neural markers in EEG following mild sleep deprivation of a single night using EEG microstate analysis. Six-minute resting EEG was conducted on thirty healthy adults within 6 hours of waking in the morning and after at least 18 h of sleep deprivation. Translated and validated Malay language Karolinska Sleepiness Scale was used to assess the participants' degree of sleepiness. Microstate characteristics analysis was conducted on the final 24 subjects based on four standard microstate maps. Microstate C shows a significant increase in mean duration, coverage and occurrence, while microstate D has significantly higher occurrence after sleep deprivation. This study demonstrates notable changes in resting state EEG microstates following mild sleep deprivation. Present findings deepen our understanding of the brain's spatiotemporal dynamics under this condition and suggest the potential utility of neural markers in this domain as components of composite markers for sleep deprivation.

Keywords Electroencephalography, EEG, Electroencephalogram, Microstate, Sleep deprivation

Sleep plays a crucial role in human health, impacting both brain function and overall physiological well-being. Adequate sleep is of paramount importance in all age groups, as it influences crucial aspects such as cognitive processes, mood regulation, metabolism and hormone function[1]. Recognizing the variations in the ideal duration of sleep throughout different life stages, the American Academy of Sleep Medicine (AASM) and Sleep Research Society (SRS) guidelines advocate for adults to aim for a minimum of seven hours of sleep per night, allowing for 17 h of wakefulness each day[2]. Prolonged wakefulness beyond this recommended duration may lead to sleep deprivation, consequently, disrupting the balance of sleep–wake cycle.

Sleep deprivation is a state marked by a lack of adequate sleep or the inability to meet the necessary quantity or quality of sleep[3,4]. Mild sleep deprivation occurs when an individual experiences sleep loss of only a few hours at night[5]. In contrast to extreme sleep deprivation, the prevalence of mild sleep deprivation is more common, with a substantial portion of the global population routinely experiencing such conditions, particularly during the transition from standard time to daylight saving time[6], during long distance air travel[7] or as a consequence of shift work[8,9]. In real-world scenarios, the elevated risk of culpable crash involvement due to the mild sleep deprivation[10] underscores the higher ecological validity of this state compared to extreme sleep deprivation[6]. Previous studies also indicated that mild sleep deprivation results in diminished performance[11,12], deficits in emotional processing[13] and reductions in decision-making capacity, as well as motor preparation and execution[9].

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Electroencephalography (EEG) is a non-invasive tool used to detect and measure brain electrical fields using electrodes placed on scalp. By measuring the voltage fluctuations generated by neural circuits, EEG is a suitable tool to study the functional and anatomical configuration of the brain[14]. Despite the well-documented impacts of mild sleep deprivation on various aspects of performance and safety, there is a limited amount of research investigating the neural markers and spatiotemporal properties of resting-state EEG following mild sleep loss. Most studies reported EEG activities of chronic or extreme sleep deprivation following prolonged periods of wakefulness[15–17], and many others conduct EEG analysis in brain frequency domain such as spectral power analysis[18,19]. Furthermore, many studies also focus on specific populations such as drivers[20–22], medical professionals[23] or patients with brain pathology[24,25]. Therefore, specific characteristics of resting-state EEG in dynamic system domain following short duration of sleep deprivation among general population remain unclear.

The dynamic system approach to EEG analysis involves studying the spatiotemporal patterns of brain activity during periods of rest. In this approach, the state of brain activity represents the collective impact of all EEG signal variables defining the EEG system at a specific moment, while the system dynamics illustrate the changes in brain state over time[26]. EEG microstate analysis is one of the popular methods to study patterns in brain state and dynamics. The brain state dynamics is characterized by organised patterns of scalp potential topographies that remains stable over short period of time (80–120 ms) before rapidly switching to the next microstate[26]. Microstates arise from the coordinated activity of neural assemblies in the brain, generating distinct maps in a non-random fashion. Therefore, a shift in topographical map signifies a change in the distribution of underlying active dipoles that contribute to the topography[27]. The dynamic transitions between microstates thus indicate the sequential activation of different neural networks over time[28] and reflect the combined neuronal activities across brain regions rather than activity specific to a particular frequency band, which offer insight into the overall coordination and stability of neuronal activities when the brain is at rest[29].

The EEG microstate analysis is typically performed during resting state to investigate patterns of neuronal activities referred to as the resting-state network (RSN). The RSN is a spontaneous and ongoing global brain activity that occurs when the brain is at rest, characterized by highly organised spatiotemporal patterns resulting from the interplay between the network's dynamics and structure[30]. It originates from the concept that functionally connected areas in brain maintain their inherent activities in an organised manner to facilitate optimal readiness for upcoming inputs[31]. These discrete mental operations giving rise to conscious mentation can be studied and quantified using EEG microstate analysis.

To date, the evidence on EEG microstates during sleep deprivation is scarce. Back in 1999, Cantero et al. explored EEG microstates on eight male and female subjects[32]. The study was one of the earliest to compare EEG microstates during the state of alertness, drowsiness and in sleep. They discovered that drowsiness microstate displayed a shorter duration compared to rested wakefulness. However, the study focused mainly on brain activities during sleep, and the duration of subjects' wakefulness was not specified. Furthermore, the microstate was not divided into topography, precluding deeper understanding of the physiology behind. In 2021, Ke et al. conducted microstate analysis on 27 healthy males after 24 h of sleep deprivation[33]. They found a significant decrease in presence of microstate A and increase in presence of microstate D, and correlated them to disrupted phonological information processing and altered attention resource allocation respectively. When correlated with sleepiness level, similar changes were observed in these microstates, indicating the vulnerability of brain microstates to sleepiness. On the other hand, An et al.[34] conducted EEG microstate analysis on 30 males after 24 h and 36 h of sleep deprivation[34]. They found that the presence of microstate B increased significantly especially after 36 h of sleep deprivation, and inferred that a structure corresponding to microstate B, namely insula, underwent augmented passive activation. Despite the inconsistent findings, both of these recent studies attributed these microstate changes to compensatory mechanisms that help sustain wakefulness and mitigate the negative effects of sleep deprivation.

Present evidence has demonstrated dynamic alteration in spatiotemporal activities of brain during sleep deprivation. Nonetheless, the results were very inconsistent. The findings from recent studies also lack generalizability due to extended period of sleep deprivation and exclusion of female participants. This leaves an evidence gap for microstate changes in mild sleep deprivation among wider population. Based on the established evidence that mild sleep deprivation induces significant change to cognition and behaviour, this study hypothesizes that even mild sleep deprivation (only a few hours) of a single night would be sufficient to induce significant changes to EEG microstates. By addressing the existing gap in the literature, we aim to characterize the EEG patterns during mild sleep deprivation using dynamic system approach within a cohort of clinically validated healthy individuals recruited from general population. This study hopes to contribute additional knowledge and insights into brain dynamics among healthy population experiencing mild sleep loss.

Methodology

Study subjects

The study subjects were healthy volunteers recruited from healthy subject's database in Clinical Research Centre, Sarawak General Hospital. 30 subjects aged between 21 to 40 years old participated the study. Six subjects were subsequently excluded due to presence of significant amount of artifact as detected by QEEG Pro software and issue with EEG format incompatibility with the software. Demographic of participants are shown in Table 1. Among the final 24 subjects, 16 (66.7%) were female. The majority of participants were of Malay descent, comprising 15 (62.5%) individuals, followed by 3 (12.5%) each of Chinese, Iban, and Bidayuh ethnicities. Their age group was categorized by decade: 13 (54.2%) were aged between 21 to 30 years old (third decade), and 11 (45.8%) were aged between 31 to 40 years old (fourth decade)[35].

The health status of participants was confirmed through blood investigations involving complete blood count, renal profile, liver profile and fasting glucose level as well as vital signs assessment by attending medical doctors.

Demographic	n (%)
Age (year)	
21–30	13 (54.2%)
31–40	11 (45.8%)
Gender	
Male	8 (33.3%)
Female	16 (66.7%)
Ethnicity	
Malay	15 (62.5%)
Chinese	3 (12.5%)
Iban	3 (12.5%)
Bidayuh	3 (12.5%)

Table 1. Distribution of 24 participants included in EEG microstate analysis according to age group, gender and ethnicity.

Medical history and physical examination were conducted to screen out subjects with history of chronic medical illness, mental disorders, sleep disorders and brain pathology. Subjects who were smokers, consumed alcohol on regular basis, on long term medications or drug use, and had difficulty staying awake for extended period were excluded. Subjects were refrained from smoking one week prior to the experiment and from consuming food containing caffeine and nicotine 24 h prior as well as throughout the study. They were required to have minimum seven hours of sleep for at least two days prior to study.

Experiment protocol

The experiment commenced when a study participant was instructed to stay awake for at least 18 h, starting from 4 a.m. on the day of experiment. The first EEG was recorded within 6 h of waking and the second recording was carried out after 18 h of waking. During the wakefulness period, subjects were allowed to resume their routine work and activities. To ensure wakefulness during the day, subjects were required to respond to messages sent intermittently every few hours. All participants were required to report their level of sleepiness using the Malay language translated and validated Karolinska Sleepiness Scale (KSS-MAS) before and after the sleep deprivation.

Questionnaire translation and validation

Karolinska Sleepiness Scale (KSS) assesses the degree of sleepiness which best represent an individual's psychophysical state over the past 10 min, serving as a physiological indicator of sleepiness[36–38]. It employs a 9-point Likert scale with corresponding labels: 1 = extremely alert, 3 = alert, 5 = neither sleepy or alert, 7 = sleepy, but no difficulty remaining awake, and 9 = extremely sleepy, fighting sleep). KSS is a valuable tool to assess sleepiness due to its sensitivity to sleep deprivation and its ability to predict vigilance[39,40]. As the KSS was not available in Malay, the national language in Malaysia, translation and validation of Malay language KSS (KSS-MAS) were performed.

The Malay translation and validation of KSS began with content validity evaluation for contextual appropriateness, followed by independent professional forward and backward translation process and internal pilot testing involving 15 staffs from our Clinical Research Centre, with further improvement to the scale statements following the pilot test. Finally, the validation of KSS-MAS was performed with fieldwork involving 30 healthy subjects. The criterion validity was tested by having the participants complete the questionnaire at baseline and after at least 18 h of wakefulness period[41–43].

EEG recording

Six-minute EEG recording was conducted in a quiet room in the research centre. During the procedure, subjects were placed in relaxed, sitting position, and were asked to look at a dot placed in front of them. The EEG recording was conducted using 19-channel Nicolet EEG Amplifier version 5.94. The electrode placement was carried out on the scalp according to the International 10–20 system. The Ten20 paste and Nuprep EEG skin preparation gel were used for scalp preparation. The brain electrical activity was measured in absolute power (microvolt, μV^2) and hertz (frequency). The impedance was set to 5 kΩ, sampling rate was 1 kHz, low pass filter of 0.3 Hz and a high pass filter of 50 Hz and notch setting at 45–55 Hz. The electrical activity from the brain was recorded using the Average Reference Montage.

EEG processing and analysis

The EEG analysis in this study was limited to open-eye condition to avoid drowsiness particularly during baseline recording. The analysis was performed with QEEG Pro software. Artifact removal was conducted with Standardized Artifact Rejection Algorithm on QEEG Pro software, which uses finite impulse response (FIR) filtering functions of EEGLAB[44]. The montage for microstate analysis was set against average reference and EEGs were band-pass filtered between 2 and 17 Hz. The QEEG Pro software employs algorithms to fit EEG data of each subject into four standard class of microstate topographies as proposed by Koenig et al., namely microstate A – left occipital to right frontal orientation, microstate B – right occipital to left frontal, microstate C – symmetric occipital to prefrontal and microstate D – symmetric frontocentral to occipital axis orientation[45].

The parameters of EEG microstate of each subject were then extracted for statistical analysis. The mean duration (in milliseconds) is the average length of time that a microstate exhibits sustained activation before transitioning to the subsequent microstate. The duration of a microstate mirrors the stability of neuronal activities at the region, with a reduction indicating an inadequacy in the resting state network[46]. On the other hand, coverage refers to the proportion of total recording time during which a specific microstate is dominant[47], while occurrence is defined as the average number of times a microstate appears per second[48]. Lastly, global explained variance refers to the portion of EEG point topography that can be accounted for by the four archetypes (microstate A to D)[26].

Statistical analysis

Kolmogorov–Smirnov test was used to test for normality assumption of the data. As there was no serious violation in parametric assumption, parametric test was employed in statistical analysis. Parameters of microstate were compared before and after sleep deprivation using paired sample t-test. All analyses were carried out using SPSS 19.0 (IBM Corp. Released 2019. IBM SPSS Statistics for Windows, Version 26.0. Armonk, NY: IBM Corp).

Ethics approval and informed consent

This study is registered with the National Medical Research Register (NMRR) Malaysia, with registration number NMRR-21–630-59,231. Ethical approval from Ministry of Health's Medical Research & Ethics committee (MREC) was obtained. All methods were performed in accordance with Declaration of Helsinki and institutional guidelines. Participants were informed about the purpose of study, study procedures, potential risks and benefits of the study. Written informed consent was obtained from all participants prior to study commencement. All personal information is kept confidential.

Results

Thirty healthy adults were recruited to explore neural markers of waking EEG in mild sleep deprivation of a single night using dynamic system approach. Validation of KSS-MAS was successfully conducted on all 30 subjects. EEG microstate analysis was conducted for the final 24 subjects.

Questionnaire translation and validation

The Karolinska Sleepiness Scale was successfully translated and validated to Malay (KSS-MAS). During the pilot phase of translation process, highly satisfactory results were obtained, with only minor adjustments needed to ensure semantic alignment with the original version. All the 30 subjects underwent the fieldwork phase in validation of KSS-MAS. Their mean age was 29.1 (SD ± 5.3) years with the majority female (66.7%), and Malay (63.3%). The baseline mean score for KSS-MAS was 1.63 (SD ± 1.0) and increased to 7.80 (SD ± 1.5) following more than 18 h of sleep deprivation, with $p < 0.001$. This shows that the Malay-translated version of Karolinska Sleepiness Scale (KSS-MAS), is a reliable and valid scale to assess the level of subjective sleepiness in individuals.

The questionnaire scores of the 24 subjects included in the microstate analysis were examined. At baseline (within six hours of waking in the morning), 13 subjects (54.2%) rated themselves as 1 (extremely alert), 10 subjects (41.7%) rated themselves as 2, while one subject (4.1%) rated 6. Following at least 18 h of sleep deprivation, 13 subjects (54.2%) rated themselves as 8, 8 subjects (33.4%) rated themselves as 9 (extremely sleepy), 1 subject (4.1%) rated as 7 (sleepy but with no difficulty remaining awake), and 2 subjects (8.3%) rated themselves as 3 (alert).

EEG microstate

The distribution of EEG microstates' mean duration, occurrence and coverage for all subjects before and after sleep deprivation are displayed in Fig. 1 and Supplementary Table S1.

Global explained variance

The global explained variance of our subjects was 0.69 (SD ± 0.05) at baseline and 0.68 (SD ± 0.05) after mild sleep deprivation ($p = 0.497$). This suggests that microstates A to D predominantly represent the microstate composition of individuals both in awake and sleep-deprived state.

Occurrence and coverage

Present results for both microstate C and D showed significant increase in occurrence after sleep deprivation: occurrence of microstate C rose from 3.09 Hz (SD ± 0.63) to 3.50 Hz (SD ± 0.69) per second, $p = 0.018$, while microstate D increased from 4.15 Hz (SD ± 0.51) to 4.40 Hz (SD ± 0.45), $p = 0.019$, implying an increased in neuro-electrical activity of the neuronal assemblies over these regions after sleep deprivation.

Similar to occurrence, coverage provides an indication of level of the neuronal activation in a microstate. In this study, coverage of microstate C exhibits a notable increase from 19.19% (SD ± 4.25) to 22.62% (SD ± 5.03), $p = 0.008$, following sleep deprivation. The coverage of other microstates does not show significant changes.

Mean duration

After sleep deprivation, it is found that microstate C shows a borderline significant increase in mean duration from 62.20 ms (SD ± 6.77) to 64.68 ms (SD ± 7.49), $p = 0.039$. The mean duration of other microstates does not reach statistical significance.

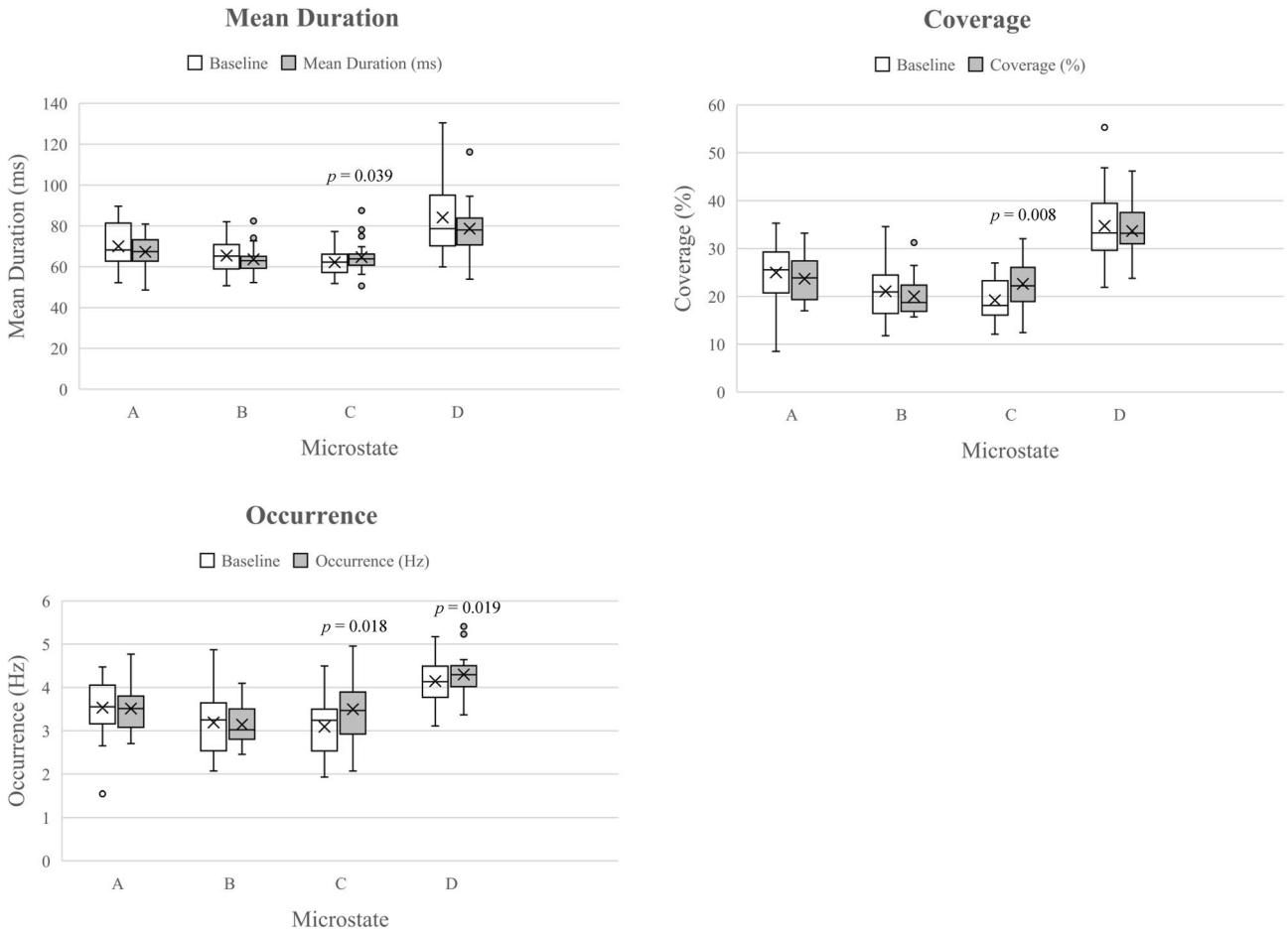

Figure 1. Distributions of microstate parameters before and after sleep deprivation for final 24 subjects. *P* value obtained with paired sample t-test.

Degree of sleepiness vs EEG microstate

In order to better depict and illustrate the changes in EEG microstates between the states of alertness and drowsiness, microstate analysis was further carried out solely on participants who were alert at baseline and were sleepy after sleep deprivation (n = 21). One participant who scored 6 on KSS-MAS before sleep deprivation and two participants who scored 3 (alert) after sleep deprivation were excluded from the analysis.

The results reveal that the microstate parameters showing significant changes remain largely similar to previous findings. An increase is observed in the occurrence of microstate C and microstate D, as well as the coverage of microstate C, as shown in Supplementary Table S2. Specifically, the occurrence of microstate C rose from 3.13 Hz (SD ± 0.63) to 3.50 Hz (SD ± 0.62), $p = 0.042$, while the occurrence of microstate D increased from 4.17 Hz (SD ± 0.50) to 4.32 Hz (SD ± 0.37), $p = 0.042$. The coverage of microstate C increased from 19.31% (SD ± 3.96) to 22.62% (SD ± 4.40), $p = 0.021$. However, the mean duration of microstate C no longer reaches statistical significance ($p = 0.054$).

Discussion

Present study reveals significant changes in microstate C and D following mild sleep deprivation of one night. In order to further understand how does sleep deprivation of this duration lead to the changes observed in the microstates, it is worth considering the potential underlying brain structures and networks at each topography. Several studies have sought to establish EEG microstates with specific brain structures and networks, albeit using vastly different methods. Britz et al. (2010) utilised k-means clustering to analyse canonical four EEG microstates, and spatially correlated them with blood oxygenation level dependent (BOLD) activation patterns on functional MRI (fMRI) resting state network, and discovered that microstate C correlates to dorsal anterior cingulate cortex, bilateral inferior frontal cortices and the right insular area, while microstate D correlates with right superior and middle frontal gyrus as well as the right superior and inferior parietal lobule[49]. The same study also attributed distinct network to each microstate: auditory network (microstate A), visual network (microstate B), salience network (microstate C), attention network (microstate D)[49]. However, when Custo et al.[50] extended their topography mapping to seven microstates, they discovered that although microstate D is highly concordant with the study by Btriz et al. (2010), microstate C more accurately corresponds to posterior cingulate cortex and precuneus, whilst the microstate C as reported by Britz et al. (2010) is actually microstate F. They concluded that

only using four microstate maps for analysis (instead of seven) may easily cause spatially correlated microstates to be combined into one[50]. This finding is supported by Yuan et al.[29], who utilized a data driven approach based on temporal independent component analysis in their microstate analysis, and found that each microstate is associated with at least one or more combinations of networks, implying that extension of microstate analysis into more clusters provide more accurate depiction of the underlying networks[29].

Therefore, following the microstate mapping by Custo et al.[50], the microstate C, which displays increased occurrence and coverage after sleep deprivation in this study, likely consists of posterior components of default mode network, namely posterior cingulate cortex and precuneus[50]. Default mode network is widely recognized for its high activation during rest and tendency to decrease activation below baseline levels during cognitively demanding tasks[51]. Of particular significance to our investigation, subregions in default mode network display a pattern of balance and synchrony in their activity, whereby posterior cingulate cortex and precuneus demonstrate higher activities in the evening/night in order to adapt and compensate for the decrease in activity of medial temporal lobe as the day goes on[52]. Therefore, the increase in occurrence and coverage of microstate C in this study can be explained by the enhanced activity in posterior cingulate cortex and precuneus as part of their diurnal rhythm throughout the day[52].

On the other hand, as our subjects only underwent sleep loss of a few hours, heightened activity in microstate C may suggest that posterior cingulate cortex and precuneus are the primary locus of neural activity that uphold the functions of default mode network in the state of mild sleep deprivation. This hypothesis can be supported by several studies that show a decrease in functional connectivity in posterior cingulate cortex and precuneus after sleep deprivation, highlighting the impact of sleep deprivation on these areas[53,54]. As posterior cingulate cortex and precuneus are important in serving as a fundamental brain function in addition to their more specific roles such as memory recollection[55,56], enhanced neuronal activity in these areas in response to mild sleep deprivation may indicate their greater vulnerability to sleep deprivation compared to other networks in the brain. More importantly, the susceptibility of default mode network to sleep deprivation sheds light on the potential role of sleep deprivation as one of the etiologies of neuropsychiatric disorders marked by disrupted activity in the default mode network, such as Alzheimer's disease[57]. Therefore, further investigations are warranted to study the reversibility of changes in default mode network noted in this study and their possible clinical association with neuropsychiatric disorders.

Additionally, our study reveals an increase in the occurrence of microstate D following sleep deprivation, with its topography correlating to the right superior and middle frontal gyrus as well as the right superior and inferior parietal lobule[49,50]. These structures constitute components of the dorsal and ventral attention networks[49,58]. The attention network plays a crucial role in regulating a broad spectrum of cognitive functions relating to attention domain[59], and it is often observed to undergo varying degrees of activation during cognitive task[60]. However, in this investigation, we observed an increase in neural activity of attention network during resting-state EEG following a few hours of sleep loss. This discovery is intriguing given that the subjects were not engaged in cognitive tasks during the EEG recording, thus the increased activity in attention network compared to baseline was unexpected. This finding implies that mild sleep deprivation may impose a physiological demand akin to that experienced during cognitively demanding tasks, thereby putting this network in a state of 'work' rather than 'rest'. The influence of sleep deprivation on the attention network is evident, with numerous studies noting increased activation of this network during tasks following sleep deprivation when compared to well-rested state[61,62]. More importantly, this sheds light on the adverse effect of sleep deprivation on the attention network, which is a crucial component for cognitive performance as reported in other studies[63,64].

Conversely, the increased activation of the attention network following mild sleep deprivation could be a prelude to a decrease in its activation following prolonged sleep deprivation. Several studies involving participants subjected to sleep deprivation exceeding 24 h have observed that critical regions of the attention network fail to sustain their activity levels after prolonged sleep deprivation[65,66], which indicate the possibility that the initial activation of the attention network observed in this study may precede a subsequent decline, likely serving as a compensatory mechanism for the network to sustain homeostatic regulation during drowsiness[67]. Therefore, present study illustrates the vulnerability of the attention network to sleep deprivation, even with mild sleep restriction over a single night. Hence, an in-depth exploration of the neural mechanisms responsible for the altered activation of the attention network following mild sleep deprivation warrants further research, as such exploration may facilitate a deeper comprehension of the neurophysiological alterations in this network and offer insights into the decline in cognitive performance frequently observed following sleep deprivation[68,69].

The average duration of EEG microstates refers to the time spent by a group of neural assemblies in an active state and reflects the pace at which a topography transitions to a subsequent topography. Examining the temporal dimension of resting-state EEG microstates provides valuable information about the time during which diverse neural assemblies, situated in various cortical and subcortical structures, collaborate and actively coordinate to produce a unified function[26]. Consequently, the mean duration of a microstate serves as a metric indicating the stability of neural assemblies within a given topography[28].

Present study noted a borderline significant increase in the duration of microstate C ($p = 0.039$). This finding was surprising, as one would expect the stability of a microstate to reduce after neuronal stress. Nonetheless, the ability of the microstates to sustain their duration indicates that mild sleep deprivation does not lead to instability of neural assemblies in serving a common function. This finding is similar to the previous studies by Ke et al.[33] and An et al.[34], which found that duration of microstates was not negatively affected by sleep deprivation[33,34]. On the other hand, the study by Cantero et al.[32] observed a noteworthy reduction in microstate duration during drowsiness[32]. Despite the lack of details on wakefulness period and microstate subtyping, the finding suggests that more research is needed to confirm the impact of sleep deprivation on microstate duration. This is crucial as microstate duration has been identified as one of the key indicators of various neuropsychiatric conditions[31]. For example, a meta-analysis conducted by Rieger et al.[70] revealed a significant reduction in the mean duration

of microstate D among patients with schizophrenia compared to healthy controls[70]. More importantly, the mean duration of microstate D exhibited a noteworthy increase when healthy controls were administered a low dose of antipsychotic medication[71], underscoring the influence of decreased duration in the disease mechanism and the efficacy of medications in mitigating this anomaly. Given the significance of microstate duration in brain pathology, verifying the impact of lack of sleep on microstate duration could provide valuable insights into neuronal responses under various stressful conditions.

During the state of drowsiness, it has been observed that the alpha peak frequency progressively decreases[72,73], while delta and theta power increases with the level of sleepiness[74,75]. Since EEG microstates primarily derive from alpha waves, factors influencing alpha wave activity, such as the state of alertness and drowsiness, are likely to affect microstate parameters. Hence, additional analysis was done to focus exclusively on participants who were alert before sleep deprivation and experienced drowsiness afterward. The findings revealed an increase in the occurrence and coverage of microstate C, as well as in the occurrence of microstate D, consistent with previous observations in this study. However, the mean duration of microstate C no longer yielded significant results after excluding three subjects from the analysis, likely due to reduced sample size.

The rise in the occurrence of microstate D during drowsiness aligns with the findings by Ke et al.[33], who assessed their subjects' sleepiness using the Visual Analogue Scale (VAS)[33]. This finding corroborates other research to indicate that the attention network is affected by sleepiness even during rest[76], which may in turn explain the attention deficit observed when one is sleepy[77]. Aside from microstate D, Ke et al.[33] discovered no change in microstate C, and instead found inverse correlation between sleepiness level and presence of microstate A (bilateral superior and middle temporal gyrus and left middle frontal gyrus)[33,49]. However, the evidence of correlation between drowsiness and these brain structures is still inconsistent[78–81]. Nonetheless, by demonstrating the effect of sleepiness on microstates, we show that our study design effectively captured subjects during the state of alertness at baseline and drowsiness post-sleep deprivation. More importantly, it underscores that sleep loss of a few hours is sufficient to induce a significant level of drowsiness and alter dynamic of neural activities in healthy adults.

Conclusion

This study employs EEG microstates to investigate the temporal and spatial characteristics of the resting brain network following mild sleep deprivation among healthy adults recruited from the general population. The findings reveal enhanced activation of microstate C (default mode network) and D (attention network) after sleep deprivation, without significant loss in the stability of any microstate. This study presents an initial exploration of the dynamic properties of neural activity in the brain following mild sleep deprivation, offering novel insights in this area. Furthermore, the alterations in EEG microstates after 18 h of sleep deprivation could serve as a suitable neural marker for identifying individuals experiencing early effects of sleep deprivation, particularly in work environments where maintaining optimal alertness is critical. Lastly, our results lay the groundwork for future investigations aimed at exploring potential composite markers of sleep deprivation that encompass various domains, including frequency, dynamics, and cognition, thus enhancing the precision and reliability of neurophysiological markers for detecting sleep deprivation.

Limitation

Firstly, sleep disorders, ideally ruled out using sleep study, are ruled out with medical history taking. Furthermore, this study does not perform cognitive assessments among participants, which could offer a measure of vigilance after mild sleep deprivation. Moreover, as participants were permitted to resume their usual activities on the day of the experiment and were tasked with responding to messages at regular intervals, the confirmation of their wakefulness was not done under direct observation and therefore remains uncertain. However, regular communication with participants likely mitigated the risk of daytime napping. Lastly, afterwork-thoughts and stress may also influence the EEG findings, which warrant further investigations in future studies.

Data availability

Raw data for the study is available at: https://github.com/khoosingyee/EEG-Microstate-After-Sleep-Deprivation/tree/main/Qeegreport.

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Author contributions

Study was designed and conceived by SAT, FAYY, LWH, KSY; translation and validation of Karolinska Sleepiness Scale by MAB, KSY, OSH and OYY; EEG data processing by KSY, LWC and NBHS, statistical analysis by KSY and MAB; manuscript writing by KSY, LWH, OSH and OYY; overall study supervised by SAT and FAYY.

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Competing interests

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Additional information

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