Extracting Stress-Related EEG Patterns From Pre-Sleep EEG for Forecasting Slow-Wave Sleep Deficiency

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***Abstract*— Sleep is vital to our daily activity. Lack of proper sleep can impair functionality and overall health. While stress is known for its detrimental impact on sleep quality, the precise effect of pre-sleep stress on subsequent sleep structure remains unknown. This study introduced a novel approach to study the pre-sleep stress effect on sleep structure, specifically slow-wave sleep (SWS) deficiency. To achieve this, we selected forehead resting EEG immediately before and upon sleep onset to extract stress-related neurological markers through power spectra and entropy analysis. These markers include beta/delta correlation, alpha asymmetry, fuzzy entropy (FuzzEn) and spectral entropy (SpEn). Fifteen subjects were included in this study. Our results showed that subjects lacking SWS often exhibited signs of stress in EEG, such as an increased beta/delta correlation, higher alpha asymmetry, and increased FuzzEn in frontal EEG. Conversely, individuals with ample SWS displayed a weak beta/delta correlation and reduced FuzzEn. Finally, we employed several supervised learning models and found that the selected neurological markers can predict subsequent SWS deficiency. Our investigation demonstrated that the classifiers could effectively predict varying levels of slow-wave sleep (SWS) from pre-sleep EEG segments, achieving a mean balanced accuracy surpassing 0.75. The SMOTE-Tomek resampling method could improve the performance to 0.77. This study suggests that stress-related neurological markers derived from pre-sleep EEG can effectively predict SWS deficiency. Such information can be integrated with existing sleep-improving techniques to provide a personalized sleep forecasting and improvement solution.**

***Index Terms*— Electroencephalography, sleep, stress, slow-wave sleep, entropy, supervised learning.**

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I. Introduction

SLEEP is a crucial aspect of our lives. A healthy adult needs 6-8 hours of sleep per day, accounting for one-third of their life. In addition to cognitive impairment [1], lack of sleep and poor sleep quality are known to contribute to a wide range of issues, including compromised immunity [2], [3], obesity [4], [5], and cardiovascular disorders [6]. During sleep, the human brain undergoes several stages of activity, which can be distinguished through electroencephalography (EEG). These stages can be roughly classified into two major stages: non-rapid-eye movement (NREM) sleep and rapid-eye-movement (REM) sleep. NREM sleep can be further divided into three stages: NREM1 (N1), NREM2 (N2), and NREM3 (N3). N1 is the lightest stage, while N3 is the deepest. As human sleep progresses from N1 to N3, the amplitude of EEG gradually increases while the frequency decreases. N3 sleep is also known as slow-wave sleep (sleep), because its EEG pattern in

mostly in delta frequency Healthy adult sleep cycles through different stages, starting with N1, then progressing to N2 and N3, before transitioning to REM and back to N1. Each sleep stage has its own cognitive and physiological importance. For example, N2 sleep is associated with synapse formation and learning, while SWS is associated with brain metabolism. Therefore, healthy sleep should include sufficient time spent in all stages. While the nature of sleep and its structure have been studied extensively in recent decades, predicting sleep quality or patterns remains challenging. Previous studies on sleep quality or pattern prediction have used either wrist-mounted accelerometers [7] or gait sensors embedded in carpets [8]. The latter method correlated 0.71 with the Pittsburgh Sleep Quality Index (PSQI) questionnaire reported by subjects.

Stress is the body's response to potential threats and challenges. While stress can help us to adapt to changing environments and situations, it can also lead to various health problems, including sleep difficulties [9], [10], [11]. Previous studies have provided conflicting results regarding how stress affects sleep structure. On the one hand, stress from social conflict has consistently been reported to induce NREM sleep and SWS in rodent models [12], [13], [14], [15], [16]. On the other hand, the anticipation of future stress, sometimes referred to as repetitive negative thinking [17] or cognitive arousal, has been reported to negatively impact sleep quality and reduce NREM sleep [18], [19], [20]. Several large population studies of sleep patterns during the COVID-19 pandemic have reported that the stress caused by involuntary lifestyle changes and fear of infection have led to increased sleep onset latency (SOL) and decreased subjective sleep quality [21], [22], [23]. Regarding sleep patterns, research has shown that anticipating early awakening or a challenging workday can lead to decreased NREM sleep [24] or SWS [25]. Other studies that artificially induce cognitive arousal before sleep have found that anticipated stress before sleep can cause an increase in sleep onset latency and reduced slow-wave activity during SWS [26], [27]. A recent study by Beck et al. [28] also reported that anticipatory stress resulted in reduced SWS and sleep spindle in naps, even when the subjects did not report decreased subjective sleep quality. Similarly, rodent studies have also found that both daily electric shock and physical restraint can lead to decreased SWS in rats [29], [30]. On the other hand, studies on various relaxation methods, such as hypnosis [31], [32] and progressive muscle relaxation [33], have found that pre-sleep relaxation increases SWS in human adults. These findings suggest a negative relationship between psychological stress before sleep and SWS, as well as overall sleep quality.

Using EEG to assess stress levels and related emotional responses has been studied extensively over the past two decades. EEG has several advantages over more traditional approaches, such as questionnaires. Firstly, EEG signals are more objective and suffer less from bias caused by cultural differences. Secondly, EEG can be passively recorded with high time resolution, making it possible to track the subject's mental state while performing various tasks. A popular method for estimating stress using EEG is based on early findings by Davidson regarding approach-versus-avoidance behavior [34], [35]. These studies compared participants' left frontal activity with their right frontal activity. Participants with stronger right frontal activity prefer avoidance behavior, whereas those with stronger left frontal activity lean towards approach behavior. As alpha (8-13 Hz) power is inversely related to task-related activity in the frontal lobe, we can measure the asymmetry of left/right frontal activity by observing the absence of alpha power [36], [37], [38]. Another commonly used method is based on the theory of the fear network model theory [39], [40]. According to this theory, anxiety and fear are mediated by cross-linking between the cortical (middle prefrontal cortex) and the subcortical (amygdala and hippocampus) regions. Fast beta-band EEG activity originating from the middle prefrontal cortex correlates with slow delta-band EEG activity originating from the amygdala. Harrewijn et al. [41] showed that subjects who reported higher social anxiety after a stressful social performance task showed a strong negative correlation between these regions. Using these methods, one can estimate subject's stress level with minimal EEG electrodes in the frontal region.

In addition to power spectra, entropy has become an increasingly popular analytical tool for human emotion and mental state. In biomedical signal analysis, entropy is used to assess the complexity and non-stationarity of a given signal. In EEG, this allows for the identification of sudden, short-lived signal signatures that are much harder to detect through power spectrum decomposition. Several variants of entropy have been adapted for emotion detection in the last decade. For instance, Martínez-Rodrigo et al. [42] used several variations of permutation entropy (PE) to discriminate between calm and distress. In another study, García-Martínez et al. [43] employed a variant of Shannon entropy to analyze brain dynamics with and without negative stress. They discovered that psychological stress increases signal entropy in the brain, particularly in the left frontal lobe (F3) and right parietal lobe (P4). This indicates that EEG entropy in the frontal region could be a potential biomarker for stress detection.

The present study examines the relationship between pre-sleep psychological stress and SWS deficiency based on established work on stress and sleep. We took a novel approach by using EEG analysis techniques based on both power spectra and complexity analysis to extract stress-related neurological markers immediately before sleep onset. We then investigated the relationship between these markers and SWS deficiency in subsequent sleep. Finally, we proposed a proof-of-concept SWS deficiency prediction system based on said neurological markers using different supervised learning models (Fig. 1). In doing so, we aimed to test the feasibility of using frontal EEG-based biomarkers to predict sleep structure for personalized sleep monitoring and improvement.

II. Hypothesis & Limitation

This study hypothesizes that subjects with stronger stress-related neural patterns in pre-sleep resting EEG will experience SWS deficiency in the upcoming sleep. Based on this hypothesis, one can build a classification system to predict SWS deficiency with pre-sleep EEG. This study serves as a proof-of-concept, which should be validated with large, diverse samples before broad real-world deployment.

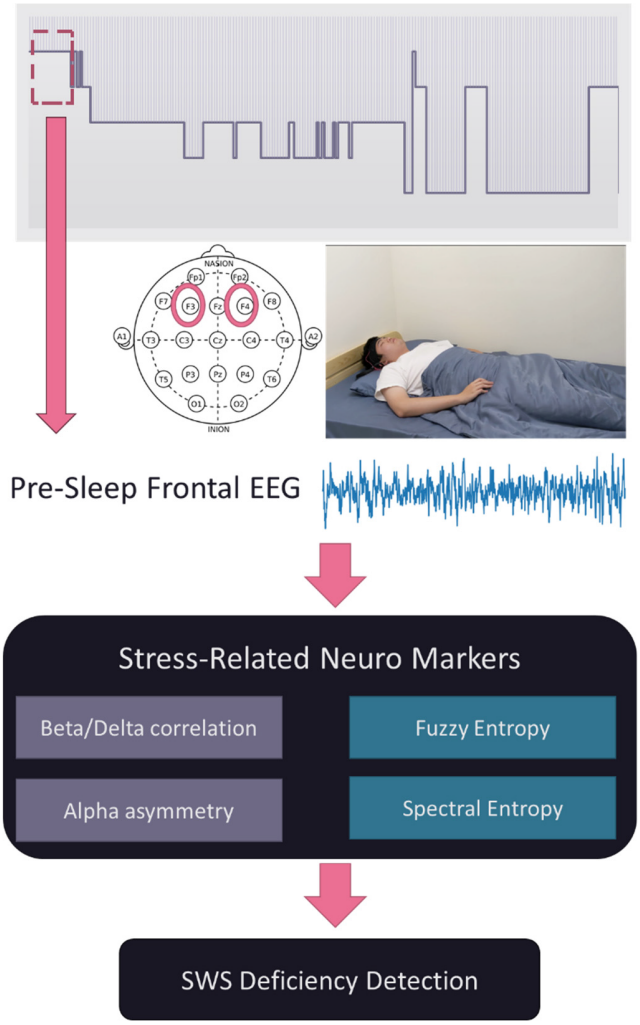

Fig. 1. The proposed approach to predict SWS deficiency. Pre-sleep EEG was collected until sleep onset. We derived stress-related neuromarkers through spectral and complexity analysis to predict potential SWS deficiency.

III. Material & Methods

A. Subject and Data Acquisition

We collected PSG records from eighteen healthy human participants working in a high-stress environment (professional nurses) who had no sleep disorder. The recordings were done with one of the following systems: NicoletOne v44 (Natus Medical, Middleton, Wisconsin, United States) (sampling rate: 125Hz; n=5), Alice 6 LDx (Philips, Amsterdam, Netherlands) (sampling rate: 200Hz; n=6), or Grael PSG (Compumedics, Abbotsford, Victoria, Australia) (sampling rate: 1024Hz; n=7). Sleep technicians reviewed all records and labelled sleep stages. Among the eighteen records, two (No. 8, No. 9) were excluded due to severe muscle noise contamination in pre-sleep EEG and one (No.14) was excluded because it did not capture pre-sleep EEG. The records included three males (age: 31±7) and twelve females (age: 37±9). Table S1 lists the detailed data collection conditions.

To explore the relation between SWS deficiency and pre-sleep resting EEG, we divided these records into three groups based on [44], which indicates that a young adult's normal SWS percentage is around 13-23% per sleep. Therefore, we defined records with less than 13% of SWS as Low SWS subjects, records with 13%-23% of SWS as Mid SWS subjects, and records with over 23% of SWS as High SWS subjects. We calculated the SWS percentage of each PSG record by dividing the total time spent in N3 sleep by the total sleep time (TST). The average TST of the subjects was 5.38±1.75 hours. Among the included records, six were in the Low SWS group, six were in the Mid SWS group, and four were in the High SWS group. No significant differences in TST were found between groups.

We collected the data at Kaohsiung Medical University Hospital after receiving approval from the local ethics board (Institutional Review Board of Kaohsiung Medical University Hospital, Kaohsiung City, Taiwan; approval code: KMUHIRB-E(II)-20190371; approval granted on Jul. 14th, 2021). All records were collected with fully informed consent.

B. EEG Selection and Processing

Figure 2 shows the data selection flowchart. EEG signals from the forehead region (F3 and F4 channels) were used to extract stress-related neurological markers. For each PSG record, 5 minutes (ten 30-second epochs) of EEG data before sleep onset was selected first. If a subject's SOL was shorter than 5 minutes, the first 5 minute of EEG data was selected instead. The selected EEG epochs were down-sampled to 125Hz and filtered with a Butterworth bandpass filter (1.0-35 Hz). Epochs labelled as either NREM or REM sleep were rejected so that only Wake epochs remained. Each Wake epoch was then divided into six non-overlapping 5-second segments. Finally, segments with strong muscle noises in either channel were further rejected by eye. The remaining segments would represent resting EEG before and upon sleep onset. After the data selection process, the Low SWS group had 190 segments, the Mid SWS group had 237 segments, and the High SWS group had 119 segments.

C. Stress-Related EEG Pattern Extraction

This study used spectral power trends such as left-to-right alpha asymmetry and beta-delta correlation in the frontal lobe as neuromarkers related to stress. To do this, we extracted alpha, beta, delta, and theta band power from each 5-second segment through Fast Fourier transform (FFT) using Welch's method [45] with a 1-second window. For delta and beta band, relative power values were derived by dividing the raw power by the sum of total spectral power. Using Pearson's R value, we estimated the correlation between beta and delta power. As for the alpha asymmetry, we subtracted the F4 alpha power from the F3 alpha power:

$$AlphaAsym\,(t) = P_{F4}\,(t) - P_{F3}\,(t) \tag{1}$$

The P_{F4} and P_{F3} are raw alpha power from the F4 and F3 channel respectively. Stronger F4 alpha activity over F3 indicates a lack of stress and vice versa. In a previous study, we formulated the *AlphaAsym* value into a scale of stress levels from 0 to 10 with the following equation [36]:

$$Stress\,(\mathrm{t}) = 5 - AlphaAsym(t)/2 \tag{2}$$

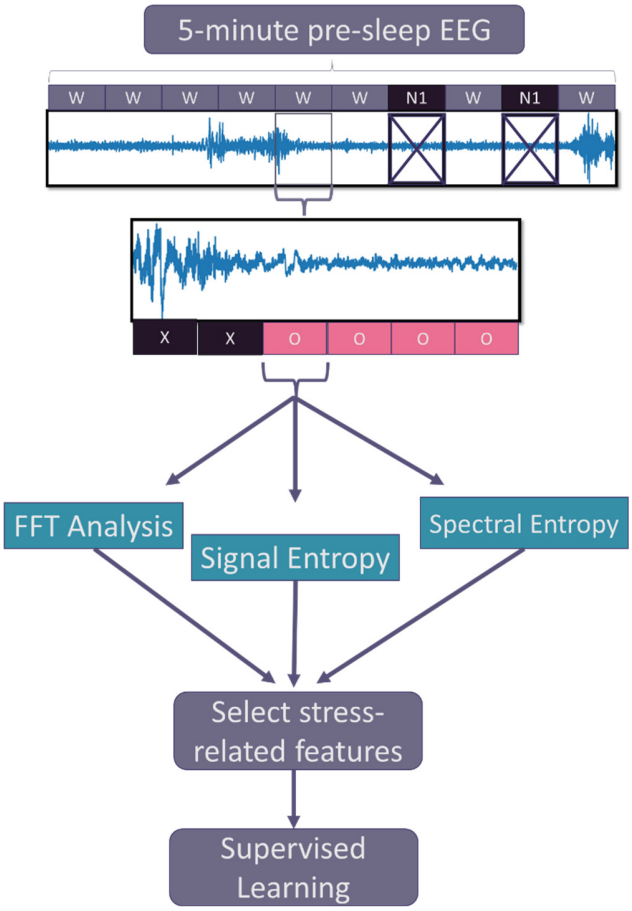

Fig. 2. EEG data selection and processing flowchart. First, the first 5 minutes of EEG data from the F3 and F4 channel were selected and filtered before being divided into 30-second epochs. Epochs labeled as REM or NREM sleep were rejected so that only Wake EEG remained. Then, each epoch was divided into 5-second segments. Finally, segments with heavy muscle noises were rejected. The remaining segments were used to derive power spectra, signal entropy and spectral entropy.

Aside from power spectra, we also calculated the entropy of each segment to see the differences in signal and spectral complexity between groups. For signal entropy, we used Fuzzy Entropy (FuzzEn) [46] to estimate the complexity of each segment. We chose FuzzEn because it is less sensitive to noise by introducing a fuzzy membership function. FuzzEn depends on two variables: embedding dimension (m) and time delay (Tau). We set m at 2 and Tau at 12 (equivalent to a shifting window of 0.096 seconds, in the alpha-band range). A previous study [43] observed the asymmetry of signal entropy at the forehead increased in stressful scenarios. Therefore, we also calculated the FuzzEn asymmetry using the following equation:

$$FuzzEnAsym\,(t) = FuzzEn_{F4}\,(t) - FuzzEn_{F3}\,(t) \quad (3)$$

For spectral entropy, we selected alpha, beta, and delta band spectra for analysis [47], [48]. A power distribution was derived through Welch's FFT for each EEG segment and then normalized. The normalized distribution is treated as a probability distribution. Then, Shannon entropy was estimated from the distribution of alpha, beta, theta, and gamma band. In spectral entropy, higher entropy values indicate relatively random, white-noise-like signal, while lower values indicate a more complex signal containing more information.

Signal pre-processing and spectral decomposition were achieved with Scipy (1.9.0, The Scipy project) [49]. Fuzzy entropy and spectral entropy estimation were conducted through EntropyHub (v0.2, Matthew W. Flood) [50].

D. Statistics Analysis

For frontal beta/delta power correlation, we used Pearson's R value to measure the mean correlation of each group. For comparisons of other power spectra, entropy values, and sleep structure, since most of these results were not uniformly distributed amongst different groups, we employed nonparametric Krustal-Wallis test (for 3-group comparison) or Mann-Whitney U test (for 2-group comparison) to calculate the significance of inter-group differences. We implemented all statistical analyses using Scipy (v1.9.0, The Scipy project) [49].

E. Supervised Learning Models

We used various supervised models to determine whether the band power and complexity features we selected were capable of predicting the amount of SWS in subsequent sleep. The applied models were k-nearest neighbours (KNN) [51], random forest (RF), linear support vector machine (SVM), and Gaussian SVM. For each 5-second EEG segment, spectral power and entropy patterns were extracted as features. The prediction target was the degree of SWS deficiency in the PSG record that an EEG segment came from. Three-fourths of included EEG segments were randomly selected as training data, while the rest were used as testing data. All supervised learning models were implemented using Scikit-learn (v. 1.11.2, scikit-learn developers) [52].

Because there are participant imbalances between different groups, we conducted a parallel training routine using over-sampling and under-sampling techniques to balance the training data [53]. First, additional data points of minority classes were generated through the synthetic minority over-sampling technique (SMOTE) [54] so that their number matched the largest class. Then, noisy and borderline data points were removed using Tomek's links [55]. We then trained supervised classification models using the re-sampled training data and evaluated them using unprocessed testing data. The SMOTE-Tomek technique was implemented with Imbalanced-learn (0.10.1, The imbalanced-learn developers) [56].

IV. Results

A. Sleep Structure

Table I shows the general sleep structures of each group. There was no significant difference in TST (318.40±88.74 vs. 316.67±112.13 vs. 337.50±119.89 minutes; $p>0.05$), indicating that the sleep opportunity was the same among the groups. There was no significant difference in WASO either (29.10±17.49 vs. 28.90±17.78 vs. 31.63±18.72 minutes, $p>0.05$). Curiously, we observed that the degree of SWS deficiency was negatively related to sleep onset latency

TABLE I
GENERAL SLEEP STRUCTURE OF INCLUDED PSG RECORDS GROUPED BY SWS PERCENTAGE

	Low SWS (n=5)	Mid SWS (n=6)	High SWS (n=4)
TST (min)	318.40±88.74	316.67±112.13	337.50±119.89
SOL (min)	10.40±7.16	7.33±4.30	2.40±1.24
SWS (%)	10.02±2.88	16.88±1.80	34.14±8.69
WASO (min)	29.10±17.49	28.90±17.78	31.63±18.72

(10.40±7.16 vs. 7.33±4.30 vs. 2.40±1.24 minutes, p=0.048). Specifically, the Low SWS group showed a significantly longer SOL than the High SWS group (p=0.036). This suggests that the High SWS group had an easier time falling asleep than the Low SWS group. On the other hand, the correlation between SOL and SWS percentage was not significant among the subjects (p=0.180). This suggests that the reduced SOL is not directly related to increased SWS. Overall, while the High SWS group showed shorter SOL, no significant between-group differences in sleep duration exist.

B. Power Analysis

Figure 3 shows the results of the power spectrum analysis. When evaluating the frontal beta/delta correlation, we found that segments from the Low SWS group exhibited the strongest negative correlation in the F3 channel (r=−0.66). In contrast, segments from the Mid SWS and High SWS groups showed much weaker correlations (r=−0.29 and r=−0.33, respectively) (Fig. 3a). A similar phenomenon was observed in the F4 channel (Fig. 3b). The low SWS group showed the strongest negative correlation (r=−0.46). The Mid and High SWS groups showed progressively weaker correlations (−r=−0.34 vs. −0.24). These differences suggest strong cortical-subcortical crosslinks in subjects lacking SWS but not those with sufficient SWS. Our findings indicate that the fear network was most active in the Low SWS group, while it was least active in the High SWS group.

Regarding the stress level derived from frontal alpha asymmetry, we found that the Low SWS group showed the highest stress level. In contrast, the Mid and High SWS groups showed progressively lower levels (4.74±0.76 vs. 4.55±0.91 vs. 4.53±0.73, respectively, p<0.05) (Fig. 3c). Upon analyzing individual subjects, we observed a trend where the Low SWS subjects tended to have higher stress levels In contrast, the Mid & High SWS subjects tended to show lower values (Fig. 3d). These results are consistent with the beta/delta correlation findings. The group with less SWS showed higher stress levels, while the group with more SWS showed the opposite.

In addition to examining the well-defined alpha asymmetry and beta/delta correlations, we also investigated the power of other EEG bands to identify potential biomarkers. Aside from differences in the alpha, beta, and delta bands, we found that activities in the theta band also differed among groups. The Low SWS group exhibited significantly stronger theta

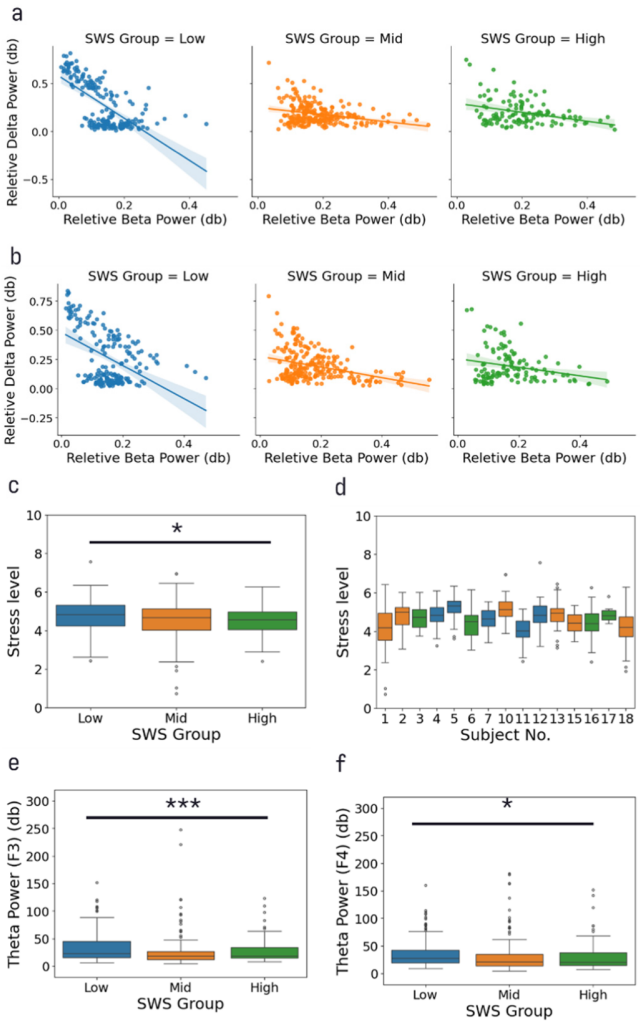

Fig. 3. Pre-sleep EEG power characteristics analysis between SWS groups. (a) Correlation between relative delta and beta power in the F3 channel. The Low SWS group showed a stronger correlation than the other groups (b) Correlation between relative delta and beta power in the F4 channel. The Low SWS group showed the strongest correlation, while the High SWS group showed the weakest. (c) Estimated stress level. The Low SWS group showed the highest stress level among the three groups (p<0.05). (d) Estimated stress levels of each subject. (e) Theta power of the F3 channel. The Low SWS group showed stronger theta activity than the other groups (p<0.01) (f) Theta power of the F4 channel. The Low SWS group showed stronger theta activity than the other groups (p<0.05).

power than the Mid and High SWS groups in the F3 channel (32.85±25.79 vs. 24.45±46.85 vs. 26.75±20.32, p<0.005) (Fig. 3e). A similar phenomenon also existed in the F4 channel (36.07±25.27 vs. 29.95±28.02 vs. 30.28±24.70, p<0.05) (Fig. 3f). Theta activity in the frontal lobe is known to be related to motor inhibition and conflict [57]. The higher theta activity in Low SWS subjects may be linked to restlessness and the need to consciously inhibit body movement before falling asleep, which is consistent with their longer SOL. Overall, the power analysis shows that subjects who experienced SWS deficiency tended to show stronger signs of pre-sleep stress, while those with sufficient SWS showed the opposite.

C. Entropy Analysis

Figure 4 shows the results of the signal entropy analysis. The Low SWS group showed significantly higher signal

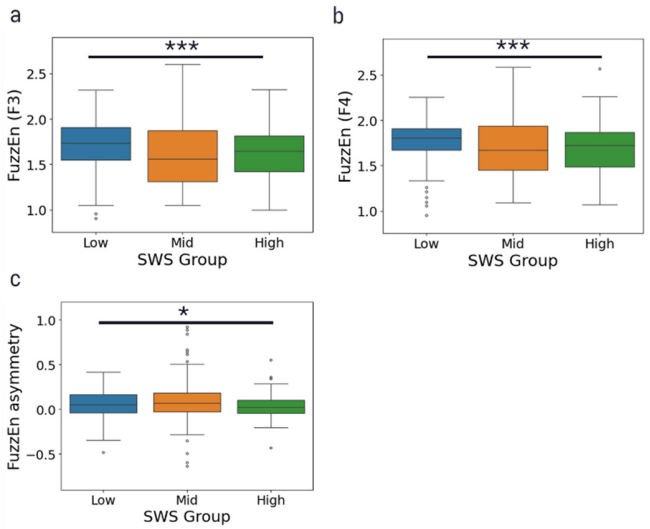

Fig. 4. Signal entropy of pre-sleep EEG between SWS groups. (a) The signal entropy in the F3 channel. The Low SWS group showed much higher signal entropy than the other two groups ($p<0.001$) (b) The signal entropy in the F4 channel. The Low SWS group showed much higher signal entropy than the other two groups ($p<0.001$) (c) The signal entropy right-over-left asymmetry. The Low and Mid SWS group showed higher entropy in the right frontal region than in the left ($p<0.05$).

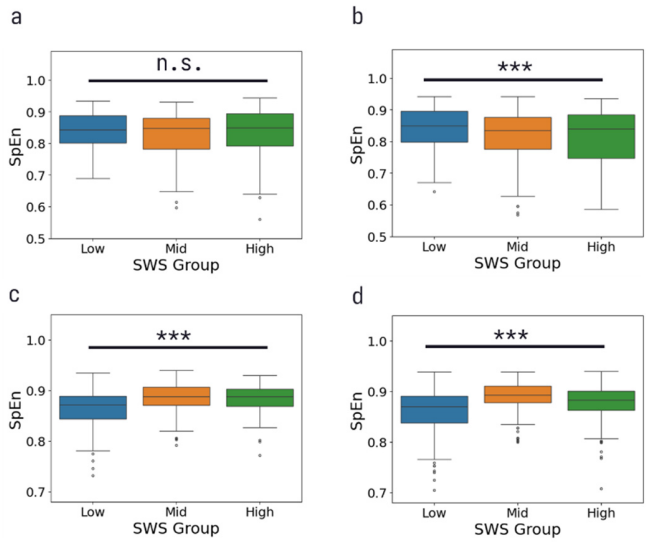

Fig. 5. Spectral entropy of pre-sleep EEG between SWS groups. (a) The spectral entropy of alpha band in the F3 channel. No significant difference existed between groups (b) The spectral entropy of alpha band in the F4 channel. The Low SWS group showed higher spectral entropy than the other two groups ($p<0.001$) (c) The spectral entropy of theta band in the F3 channel. The Low SWS group showed lower entropy than the other two groups ($p<0.001$) (d) The spectral entropy of theta band in the F4 channel. The Low SWS group showed lower entropy than the other two groups ($p<0.001$).

entropy in the F3 channel, while the Mid and High SWS group showed lower values (1.71±0.25 vs. 1.61±0.34 vs. 1.64±0.30, $p<0.005$) (Fig. 4a). The F4 channel also exhibited the same trend, with the Low SWS group showing significantly higher signal entropy compared to the Mid and High SWS groups (1.76±0.22 vs. 1.70±0.30 vs. 1.67±0.31) ($p<0.005$) (Fig. 4b). In addition, we observed that in the Low and Mid SWS groups, the right frontal region showed a higher signal entropy than the left frontal region. In contrast, the left-right signal entropy difference in the High SWS group is much smaller. (0.06±0.16 vs. 0.10±0.21 vs. 0.04±0.13, respectively; $p<0.05$) (Fig. 4c). The increased FuzzEn values in the Low SWS group might be related to the stronger cortical and subcortical cross-linking we found in the power analysis. In contrast, the High SWS group had weaker cross-linking, which resulted in a lower entropy value and decreased FuzzEn asymmetry.

Figure 5 shows the results of the spectral entropy analysis. We found significant differences in the alpha and theta bands between the groups. Specifically in the alpha band, while there was no significant difference in the F3 channel (0.83±0.06 vs. 0.83±0.07 vs. 0.83±0.08, $p>0.05$) (Fig. 5a), we observed that the Low SWS group showed higher SpEn than the other groups in the F4 channel (0.85±0.06 vs. 0.82±0.08 vs. 0.82±0.08, $p<0.005$) (Figure 5b). This indicates that the Low SWS group showed less frequent alpha activity in the right frontal cortex. In the theta band, we found that the Low SWS group showed significantly lower SpEn values both in the F3 channel (0.87±0.04 vs. 0.89±0.03 vs. 0.88±0.03, $p<0.005$) (Fig. 5c) and the F4 channel (0.86±0.05 vs. 0.89±0.03 vs. 0.88±0.04, $p<0.005$) (Fig. 5d).

D. Supervised Learning

To investigate the potential for predicting SWS deficiency using pre-sleep frontal EEG, we selected several neural

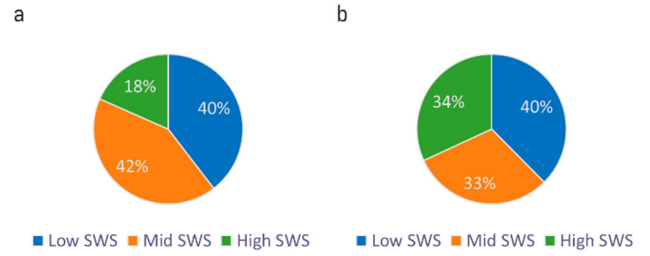

Fig. 6. Group size percentages during training with and without resampling. (a) Percentages without resampling. The Mid SWS group had the most samples, while the Low SWS group had the least. (b) Percentages with resampling. The percentages were more balanced between groups after SMOTE-Tomek resampling.

markers that showed significant inter-group differences: relative beta power, beta/delta power ratio, stress level, raw Theta power, FuzzEn, FuzzEn asymmetry, and SpEn (theta). We conducted two parallel training routines, one with SMOTE-Tomek resampling and one without. In the routine without resampling, the group size percentages between Low:Mid:High SWS groups were 40:42:18 (Fig 6a). In the routine with resampling, the percentages were 40:33:34 (Fig 6b), which was more balanced.

Fig. 7 shows the confusion matrices resulting from supervised learning without over-under sampling. The accuracies of the classifiers for the Mid SWS group, which contains the most samples, ranged from 0.75 (in linear SVM) to 0.85 (in Gaussian SVM). The accuracies for the Low SWS group ranged from 0.74 (in KNN) to 0.81 (in the random forest). These results demonstrate that the selected markers can effectively identify EEG segments collected from SWS-deficient subjects. On the other hand, EEG segments from

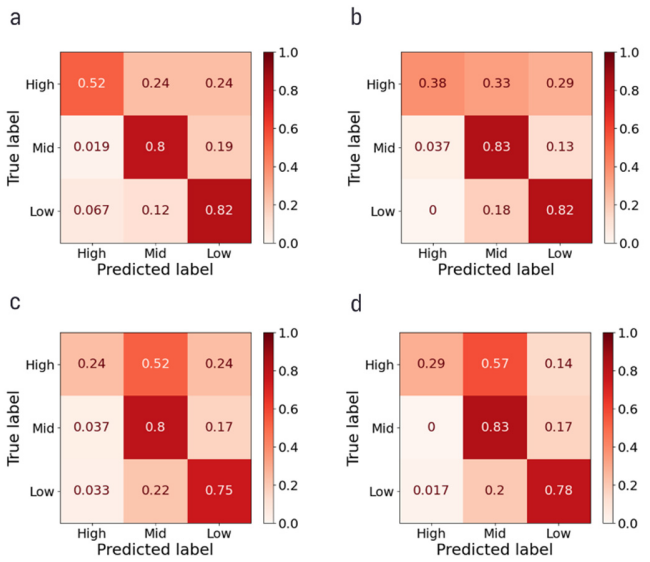

Fig. 7. Classifications results with no resampling. (a) KNN classifier (b) random forest classifier (c) linear SVM classifier (d) Gaussian SVM classifier.

the High SWS group were poorly classified, with accuracies ranging from 0.24 to 0.52. In both linear SVM and Gaussian SVM, segments from the High SWS group were largely misclassified as coming from the Mid SWS group. The suboptimal classification result for the High SWS group might be because of sample imbalance.

Fig. 8a-d shows the confusion matrices using the over-under sampling routine. All classifiers showed increased accuracy in identifying the High SWS class but at the expense of decreased accuracy for the Mid SWS and the Low SWS classes. In particular, the KNN classifier (Fig. 8a) and the linear SVM classifier (Fig. 8c) showed accuracies below 0.7 for the Mid and Low SWS classes. On the other hand, the random forest classifier (Fig. 8b) showed the smallest decrease in the Low SWS group (from 0.83 to 0.76) while showing significantly increased accuracy for the High SWS group (from 0.38 to 0.76). Fig. 8e shows the overall performance of all classifiers. With the SMOTE-Tomek routine, both the random forest classifier and the Gaussian SVM showed increased Cohen's Kappa values. (Fig. 8e). The random forest classifier also showed an increased balanced accuracy of 0.77. Overall, our results show that stress-related neural markers can be used to predict SWS deficiency.

V. Discussion

The importance and quality of sleep have garnered attention in the recent decades. Psychological stress is a major factor affecting sleep quality is stress, as it is known to cause decreased subjective sleep quality and prolonged SOL in humans. Previous studies have consistently reported that negative anticipation stress can lead to disrupted sleep patterns and reduced SWS. These impacts are especially detrimental to individuals working in high-stress environments with limited sleep opportunities and those who are experiencing traumatic events, such as the COVID-19 pandemic [3], [21], [22], [23]. While most studies on stress and sleep relied on subjective questionnaires to assess stress levels, recent

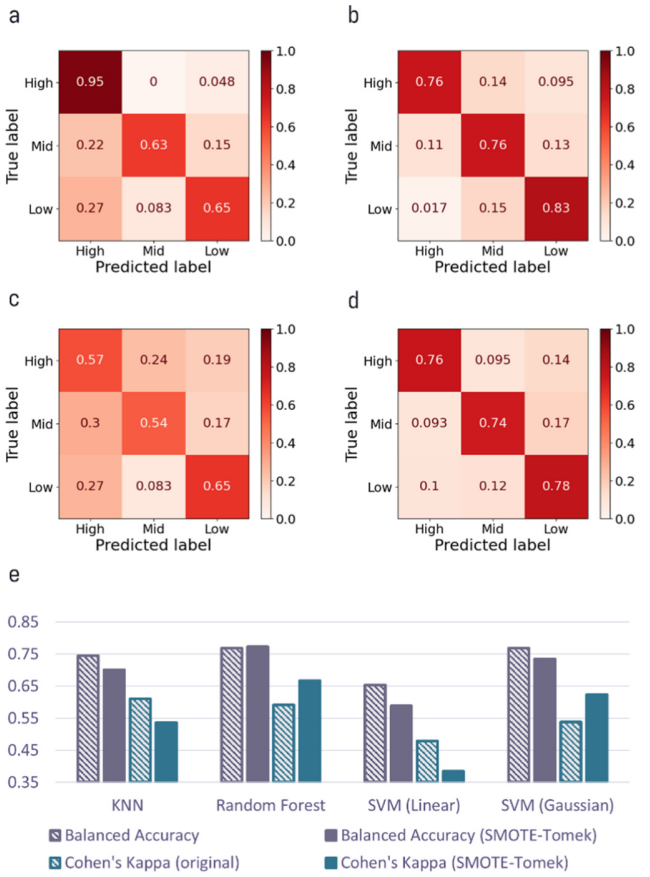

Fig. 8. Classifications results with SMOTE-Tomek resampling. (a) KNN classifier, (b) random forest classifier, (c) linear SVM classifier, (d) Gaussian SVM classifier, and (e) accuracy and Cohen's Kappa values of various classifiers with and without resampling. Linear SVM showed reduced performance after resampling, while Gaussian SVM and random forest classifiers showed improved accuracy and Kappa value.

advances in cognitive science and EEG analysis have allowed a more objective way to measure stress levels in human subjects. This approach also opens the possibility of using stress-related neuromarkers to predict changes in sleep patterns and, potentially, overall sleep quality. This study took a novel approach combining mental state assessment and sleep study by extracting stress-related neural markers in pre-sleep EEG. We found that subjects with SWS deficiency (<13%) exhibited higher stress levels and signs of restlessness in pre-sleep resting frontal EEG, while subjects with abundant SWS showed the opposite. These results suggest that an individual's pre-sleep mental state is linked to subsequent sleep structure and SWS percentage. Based on these findings, we show that neural markers from pre-sleep EEG can be used to predict SWS deficiency, with the highest accuracy of 0.77. Our result implies that pre-sleep mental state may affect more than just SOL and that pre-sleep EEG may be used to predict sleep quality.

A. Stress-Related Power Differences Corresponds With Insufficient SWS

The frontal lobe is crucial to many cognitive functions of the human brain. When comparing frontal spectra, we found that subjects showed SWS-deficient subjects exhibited several

differences compared to other subjects. The first difference is the correlation between beta and delta power. We observed that EEG segments from SWS-deficient subjects showed a stronger correlation, while subjects with abundant SWS showed a weaker correlation. This difference may have resulted from the increased competition between the middle prefrontal cortex and subcortical regions, as suggested by traditional fear network theory [39], [40]. The increased correlation in the Low SWS group suggests a more active fear network, which could lead to experiences of stress and restlessness. Indeed, the Low SWS subjects showed the highest average stress level, while the High SWS subjects showed the lowest. Besides spectral signatures related to stress, we also found that Low SWS subjects tended to show stronger theta band activity than High SWS subjects. Theta activity in the prefrontal cortex (particularly the middle prefrontal cortex) is known to be related to motor and impulse inhibition [58], [59]. The increased motor inhibition suggests that the Low SWS subjects may have experienced greater restlessness and a greater need to inhibit body movement. The spectral differences suggest that the Low SWS group may have had more difficulty falling asleep, as reflected in their longer SOL. Overall, our findings in spectral power differences are consistent with previous studies showing that the presence of stress is negatively related to the SWS ratio.

B. SWS Deficiency Is Related to Increased EEG Signal Complexity

Previous studies have shown that EEG signal complexity would increase during periods of stress [43] and decrease during relaxation [60], [61] in healthy humans. These findings suggest that entropy can be a biomarker for detecting psychological stress. Our study found that subjects in the Low SWS group tended to show higher FuzzEn values. The increased entropy value was more prominent in the F4 channel than in the F3 channel, consistent with a previous study by García-Martínez et al. [43]. Additionally, we observed that while the Low and Mid SWS groups showed a right-over-left bias in FuzzEn, the High SWS group did not. A previous study by Ren et al. suggests that right-over-left complexity asymmetry indicates the presence of negative emotion, while a lack of asymmetry indicates calmness [62]. Therefore, the absence of entropy asymmetry in the High SWS group indicates they were more composed and less agitated when falling asleep.

Aside from signal entropy, we also noted differences in spectral entropy in the theta and alpha bands. The spectral entropy value is inversely proportional to how frequently certain rhythms occur in a given signal. The Low SWS group showed decreased SpEn in the theta band, consistent with their overall increased theta activity. Curiously, the Low SWS group showed higher SpEn in right frontal cortex. This contrasts with their higher stress level, which is derived from right-to-left alpha power. A possible explanation is that while the alpha rhythm occurred less frequently in the right frontal cortex in the Low SWS subjects, it occurred in higher magnitude. This also implies that EEG in the Low SWS subjects was less stationary than in other subjects, which may be linked to increased fear network activity in these subjects.

C. Potential Biological Link Between Sleep and Stress

While slow waves during NREM sleep are most prominent in the neocortex, several studies have suggested that they are likely regulated by the thalamus [63], [64], [65]. An earlier study by David et al. found that blocking the thalamic signal to the neocortex greatly diminished slow-wave activity in rats [64]. A later study by Gent et al. also found that centromedial thalamus (CMT) neurons are phase-advanced to cortical slow waves in mice [65]. Traditionally, the thalamus had been viewed as a passive relay for sensory inputs and subcortical signals to the cerebral cortex. However, studies in the last decade have revealed that the thalamus is critical in mediating complex cognitive functions in the human brain [66], [67], [68]. While most studies have associated the stress response with the hypothalamic–pituitary–adrenal (HPA) axis, recent animal studies suggest that paraventricular thalamus is also involved in the stress response, including increased alertness [69] and binge eating [70]. This may provide a biological link between psychological stress and decreased slow-wave activity in mammals, although more studies are needed to confirm this hypothesis.

D. Pre-Sleep Frontal EEG Predicts Lack of SWS for Sleep Monitoring and Improvement

Based on our observations in the frontal EEG power spectra and complexity, we employed several supervised models (KNN, random forest, linear SVM, Gaussian SVM) to validate if the neurological metrics could discriminate EEG segments from subjects with SWS deficiency. Initially, all classifiers could identify EEG segments from the Low SWS subjects (accuracy ranging from 0.74 to 0.81) and the Mid SWS subjects (accuracy ranging from 0.75 to 0.85). However, the classifiers showed suboptimal performance in classifying High SWS subjects (accuracy ranging from 0.24 to 0.52). This is likely because of imbalanced sample sizes. Specifically, the High SWS group's size was less than half the size of the Mid SWS group. We used the SMOTE-Tomek resampling technique to address the imbalance issue As shown in Fig. 7b, the three groups became more balanced after the re-sampling procedure. In Fig. 8e, the Gaussian SVM and the random forest classifier showed improved performance. In particular, the random forest showed an accuracy of 0.77 in distinguishing EEG segments from subjects with different SWS levels. Overall, our study showed that EEG from the frontal lobe contains valuable information for predicting sleep structure. A proper hyperparameter tuning strategy and resampling strategy could further enhance the performance in the future.

E. Stress Evaluation and SWS Deficiency Prediction May Be Utilized to Enhance Immediate Sleep Quality and Long-Term Brain Health

Studies on mammal brain metabolism in the last decade have suggested that SWS is crucial in removing excessive materials from the brain [71], [72]. Moreover, an animal model of Parkinson's disease found that enhancing SWS promotes the removal of misfolded alpha-Syn protein and the expression of enzymes that prevent misfolding [73]. Psychological stress

may have a larger implication on the brain's overall health as it diminishes SWS and crucial material exchange. On the other hand, various relaxation methods have been proposed to promote SWS in human adults, including aromatherapy [74], muscle relaxation [33], and acoustic hypnosis [31]. These methods offer opportunities to supplement SWS artificially in users experiencing a deficiency.

The present study establishes pre-sleep EEG as a reliable predictor of SWS deficiency. Its simplicity allows seamless integration with our team's existing frontal-EEG-based sleep stage classification algorithms [75], [76], paving the way for an IoT system. This IoT system would assess stress levels and predict potential SWS deficiency, then, automatically activate activating relaxation protocols (e.g., speakers, aroma dispensers) when needed. Such automation promises a significant advancement in personalized sleep profiling and improvement.

F. Limitations

Firstly, the study was done on a relatively small population who are majorly female. The presented findings need to be validated with a larger sample size to determine whether they are gender dependent. Secondly, this study did not use a questionnaire to evaluate the subjects' subjective stress levels. We omitted the questionnaire because we preferred to use the objective information from the subjects' EEG, which may differ from their conscious feelings. For future studies, we intend to increase our subject number so that we may develop a machine learning system that can automatically detect stress in real-time and develop it into a solution for sleep monitoring and profiling.

VI. Conclusion

This study presents a novel approach to predict SWS deficiency through stress-related neurological markers in resting EEG. We did so by explicitly selecting frontal resting EEG immediately before sleep onset and extracting stress-related neurological markers through PSD and complexity analyses. Our study found that subjects with SWS deficiency showed longer SOL than others. They also showed strong beta/delta correlation, higher alpha asymmetry, increased theta band activity, higher signal complexity, and decreased spectral entropy in the theta band. These signs indicate restlessness and stress in these subjects. On the other hand, subjects with sufficient SWS showed the opposite trend in these neural markers. Based on these findings, we used several supervised learning classifiers to predict SWS deficiency. The classifiers can detect segments from EEGs collected from subjects with SWS deficiency. The classifiers' performance can be further enhanced through the SMOTE-Tomek over-under-sampling technique, with the best model showing a mean balanced accuracy of 0.77. Overall, our results show that stress-related neurological markers in pre-sleep forehead EEG can be used as predictors for SWS deficiency. Future applications can use this information to realize personalized sleep monitoring and improvement.

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