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An Electroencephalography Study of Adult Attention Deficit Hyperactivity Disorder and its Association with Insomnia

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ABSTRACT

Background: The present study aimed to find the electroencephalography (EEG) characteristics of adult attention-deficit/hyperactivity disorder (ADHD) and to examine if these EEG indices are associated with insomnia in adult ADHD.
Methods: Twenty-six participants were included in the adult ADHD group, and 26 sex-, age-, and education-matched participants formed the control group. Between-group differences in the resting-state EEG indices and the score of insomnia scale were assessed. Correlational analysis between these EEG indices and the score of insomnia scale was conducted.
Results: The adult ADHD group had more insomnia problems and showed increased power over 4 frequency bands at electrodes frontal area, Cz, and Pz, except for alpha band at electrode frontal area. Furthermore, some EEG indices, especially over fast frequency bands, are associated with the score of insomnia scale.
Conclusion: The findings of this study reveal that adult ADHD shows a distinct EEG pattern during the resting state. The correlation between the EEG indices over fast frequency bands in adult ADHD and the score of the insomnia scale may explain the high prevalence of insomnia in adult ADHD.

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INTRODUCTION

In the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), attention-deficit/hyperactivity disorder (ADHD) is categorized as a neurodevelopmental disorder with core symptoms of inattention, hyperactivity, and impulsivity.[1] The symptoms of ADHD persist into adulthood in up to 60% of this population.[2] The prevalence rate of adult ADHD is approximately 3.4%.[3] One meta-analysis of 16 functional magnetic resonance imaging studies of adults with ADHD revealed hypoactivation of the frontoparietal system and hyperactivation of the visual, dorsal attention, and default networks.[4] Research has also indicated that adult ADHD is associated with several deficits in cognitive functions.[5-8] Therefore, adults with ADHD may experience significant functional impairment affecting their work performance, relationship maintenance, and quality of life.[9]

The US Food and Drug Administration approved the use of electroencephalography (EEG) as an assistive tool for diagnosing childhood ADHD in 2013. However, fewer studies employing EEG have been conducted on adult ADHD than on

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childhood ADHD, and the EEG pattern in adult ADHD differs from that of childhood ADHD. Electroencephalography waves can be divided into 4 different frequency bands, each representing a distinct state of neural activity. Delta waves (with a frequency range of 1-4 Hz) primarily occur during deep sleep and are associated with brain function recovery and repair. Theta waves (with a frequency range of 4-8 Hz) are commonly seen in infants, children, and adults during deep relaxation, meditation, or sleep. Alpha waves (with a frequency range of 8-13 Hz) are typically generated during relaxation, resting with eyes closed, or in a focused, relaxed state. Beta waves (with a frequency range of 13-30 Hz) are primarily related to wakefulness and cognitive brain activities. Spectral analysis studies on the resting-state EEG patterns of adults with ADHD have produced mixed results.[10-15] For example, Bresnahan and Barry[11] and Koehler et al[13] have observed elevated alpha activity in adult ADHD groups, but Woltering et al[15] noted decreased alpha activity. For beta band activity, Bresnahan and Barry[11] observed elevated power; Clarke et al[12] reported reduced activity at the midline but increased activity in the right posterior area; and Woltering et al[15] noted reduced beta activity. Regarding delta band activity, Clarke et al[12] observed reduced power across the entire scalp, but Bresnahan and Barry[11] reported elevated global power. With regard to theta band activity, Bresnahan and Barry,[11], Clarke et al,[12] Koehler et al,[13] and Woltering et al[15] all indicated increased activity. However, van Dongen-Boomsma et al[14] observed no significant difference between an adult ADHD group and a control group. One systematic review in 2020 suggested that elevated theta power and alpha activity indicated through resting-state EEG could be potential biomarkers for identifying adult ADHD, but inconsistency among studies remains a barrier.[16]

In addition to the core symptoms of ADHD, emotional dysregulation, sluggish cognitive tempo, and sleep problems are 3 commonly co-occurring conditions.[17] The prevalence of insomnia in the general population is approximately 31%-56%.[18] Insomnia is a common comorbidity in adult ADHD, with a prevalence that is 67%-80%, higher than that in the general population.[19,20] Adults with ADHD tend to report having difficulty falling asleep, frequent nocturnal awakenings, shorter total sleep time, and feeling unrefreshed after waking up compared to adults without ADHD.[21-25] Adult ADHD groups are also associated with longer sleep onset latency and more nocturnal awakenings, which are objective parameters, than their counterparts in control groups.[21,24,26,27]

The disturbance of nocturnal sleep may cause daytime sleepiness, which can exacerbate the core symptoms of ADHD and reduce cognitive performance, further leading to impairment in academic or occupational functions.[28] One study revealed that insomnia in adult ADHD is associated with higher ADHD severity, more physical and mental comorbidities, and a less favorable quality of life.[29] The most widely supported pathophysiological theory of insomnia is the hyperarousal theory. According to the theory, increased cortical activity is one of the characteristics of insomnia.[30] The increased fast frequency EEG activities are considered to be the index of cortical hyperarousal.[31,32] Because adult ADHD and insomnia are 2 highly comorbid disorders that place a considerable burden on society, elucidating their neurophysiological relationship is thus paramount.

In the present study, the following were explored: (a) whether results from resting-state EEG spectral analysis could be used to differentiate an adult ADHD group from a control group, (b) whether the adult ADHD group had a higher prevalence of insomnia symptoms than the control group did, and (c) whether the differences in EEG indices between the 2 groups were correlated with insomnia. Given the mixed results from the literature, there were no predictions about the EEG indices for differentiation between groups. However, it could be predicted that the adult ADHD group presented more sleep problems according to past studies. In addition, it can be hypothesized that the high-frequency band EEG indices were associated with insomnia based on the hyperarousal theory.

MAIN POINTS

- The resting-state electroencephalography index biomarkers of adult attention-deficit/hyperactivity disorder may be power over 4 frequency bands at electrodes frontal area, Cz, and Pz, except for alpha band at electrode frontal area.
- The adult attention-deficit/hyperactivity disorder group had significantly higher sleep distress symptoms and poorer sleep quality than the control group.
- Some electroencephalography indices, especially over fast frequency bands, correlated with the score of insomnia scale in the adult attention-deficit/hyperactivity disorder group.

MATERIAL AND METHODS

Participants

A total of 52 participants were included in this study. The participants comprised 26 adults with ADHD or childhood-onset ADHD, who were classified into the adult ADHD group, and 26 sex-, age-, and education-matched participants without adult ADHD or a history of ADHD, who were classified into the control group. All participants were recruited for the study through advertisements from November 2020 to April 2021. The inclusion criteria were as follows: individuals aged 20-40 years who were native speakers of Mandarin. The exclusion criteria were as follows: individuals with a lifetime substance-use disorder (except for nicotine-use disorder), a history of bipolar spectrum disorder or psychotic spectrum disorder, neurological diseases or injury, or intellectual disability, who were currently using an illicit substance, or who were currently treated with psychotropic medication.

The participants underwent psychiatric evaluation in which the Chinese version of the Mini International Neuropsychiatric Interview (MINI) and a diagnostic psychiatric interview were employed.[33] A psychiatrist determined the diagnosis of adult ADHD and a history of childhood ADHD according to the criteria in DSM-5.

All participants and their parents have signed an informed consent form agreeing to the use of the data provided for this study. This study protocol was reviewed and approved by the Institutional Review Board of Kaohsiung Medical University Chung-Ho Memorial Hospital, (approval number KMUHIRB-SV[II]-20170073, Date: 2018/02/27)

Process

The participants were asked to refrain from smoking for at least 2 hours before the study evaluation. Those who were currently being treated with psychostimulants were also asked not to take their medication on the day of the evaluation (n = 1). After consent was obtained from all participants, they underwent a psychiatric interview to verify the diagnosis of adult ADHD and to assess the exclusion criteria according to the MINI. Then, the participants in both groups completed the 20-item Pittsburgh Insomnia Rating Scale (PIRS_20) and underwent resting-state EEG examination.

Measures

Diagnosis of Adult ADHD: The DSM-5 criteria for diagnosing adult ADHD is the presence of 5 of the 9 symptoms of inattention, namely carelessness, difficulty concentrating, absent-mindedness, not following through on instructions, lacking organizational ability, avoiding tasks requiring sustained attention, distractibility, forgetfulness, and frequent losing objects required for tasks, and/or 5 of the 9 symptoms of hyperactivity or impulsivity, namely fidgeting and squirming in seats, frequently leaving seats, restlessness, inability to engage in activities quietly, always "on the go," talking excessively, blurting out answers, difficulty waiting for turns, and interrupting others.[1] The onset of symptoms must occur before the age of 12 years. The symptoms must occur in at least 2 settings and must last more than 6 months. Functional impairment in social, academic, or occupational areas of life is also required for an ADHD diagnosis.

Twenty-Item Pittsburgh Insomnia Rating Scale: The PIRS_20 is a self-reported scale comprising 20 items scored on a 4-point Likert scale from 0 (not at all bothered) to 3 (severely bothered).[34] The participants completed each item according to their sleep condition over the past 7 days. Twelve items refer to distressing insomnia-related symptoms (e.g., difficulty in maintaining concentration, not feeling refreshed after waking up, and relationship impairment), 4 items refer to sleep parameters (e.g., sleep latency and the time required to fall asleep again after awakening), and 4 items pertain to sleep quality (e.g., satisfying level about insomnia and sleep regularity). The PIRS_20 was further divided into subscales related to sleep distress, quality, and parameters. The PIRS_20 has respectable reliability and validity (Cronbach's α of 0.95 and a test–retest reliability of 0.92).[35]

Electroencephalography Data Acquisition and Analysis

Electroencephalography data were recorded using wireless dry electrode electroencephalogram recordings from 32 channels organized according to the 10-20 system (Artise Biomedical, Hsinchu, Taiwan). Vertical and horizontal electrooculograms were recorded. Two electrodes were placed on the earlobes to serve as a reference. The dry sponge electrodes were pre-soaked in water, gently squeezed out the excess moisture, and set up. The impedance of each electrode was monitored to ensure it stably dropped below 100 kΩ before data collection.[36,37] Data were collected under eyes-closed conditions, with a 147-second duration. Electroencephalography data preprocessing was conducted using the EEGLAB toolbox.[38] Artifacts, such as muscle twitches, were automatically identified and removed. The EEG sampling rate was 500 Hz, with a 1-50 Hz bandpass filter for artifact removal. Regarding artifact rejection, bad periods and bad channels with amplitudes exceeding 100 μV[39] were first visually inspected and removed. Then, artifact rejection on the resting EEG was performed using artifact subspace reconstruction (ASR)[40] with the following parameters: setting the "BurstCriterion" parameter at 20 to automatically extract high-amplitude muscle artifacts and reconstruct the period EEG data (WindowCriterion = 0.25, BurstRejection = off).[40] Then, the EEGLAB "spectopo" function was utilized to calculate the power spectral density (PSD) by applying a Fast Fourier Transform (FFT), and a Hamming window was used with a window length of 1 second and an overlap of 50%. The PSD is the measure of the signal's power strength over a range of frequencies, which describes how the intensity of a time-varying signal is distributed in the frequency domain. The PSD was computed using a fast Fourier transform, and the powers of the delta (1-4 Hz), theta (4-8 Hz), alpha (8-13 Hz), and beta (13-30 Hz) frequency bands were calculated separately.

Statistical Analysis

We analyzed the data in this study by using SPSS20[th] edition (IBM SPSS Corp.; Armonk, NY, USA), and statistical significance was indicated by a P value less than .05. Independent t-tests were used to assess differences in age, education level, PIRS_20 score, and sleep distress, quality, and parameter subscale scores, and chi-square test was used to evaluate the association of gender between the adult ADHD group and control group. Descriptive statistics were presented as mean ± SD. To identify significant EEG indices, the difference in the PSDs of the 4 frequency bands in electrodes at the frontal area (average of Fp1, Fp2, AF3, AF4, Fz, F3, F4, F7, and F8),[12,13,41] at Cz, and at Pz was compared between the adult ADHD group and the control group through independent *t*-tests. For the repeated tests, Benjamini-Hochberg correction was used to control the False Discovery Rate. To determine the associations among EEG indices, PIRS_20 score, and sleep distress, quality, and parameter subscale scores, Spearman's correlation coefficient was used. The above correlation was predicted in this study hypothesis and focused on these scientifically sensible comparisons, which should be viewed as a planned comparison. Therefore, the correction for repeated tests was not needed.

RESULTS

We analyzed the data of the 52 participants (26 in the adult ADHD group and control group) (Table 1). The between-group analysis revealed no difference between the 2 groups in terms of sex, age, and education level. However, the adult ADHD group had a significantly higher PIRS_20 score ($P < .001$), sleep distress subscale score ($P < .001$), and sleep quality subscale score ($P = .009$).

The eyes-closed resting-state EEG analysis revealed significant differences over the delta band in the electrodes at the frontal area ($P = .021$), at Cz ($P = .003$), and at Pz ($P = .006$); over the theta band in the electrodes at the frontal area ($P = .019$), at Cz ($P = .017$), and at Pz ($P = .017$); over the alpha band at electrode Cz ($P = .015$) and Pz ($P = .002$); and over the beta band in the electrodes at the frontal area ($P = .010$), at Cz ($P = .011$), and at Pz ($P = .014$) (Table 2 and Figures 1-3). After the Benjamini-Hochberg correction, the above findings still remained significant. All of the PSDs of the aforementioned EEG indices in the adult ADHD group were significantly higher than those in the control group.

In the analysis of the association between the eyes-closed resting-state EEG indices and PIRS_20 score (Table 3), a significant correlation was observed between PIRS_20 score and the theta and beta bands in the electrodes at the frontal area ($r = 0.333$, $P = .016$; $r = 0.387$, $P = .005$), between PIRS_20 score and the theta, alpha, and beta bands at electrode Cz ($r = 0.322$, $P = .021$; $r = 0.327$, $P = .019$; $r = 0.365$, $P = .008$), and between PIRS_20 score and the delta, theta, alpha, and beta bands at electrode Pz ($r = 0.293$, $P = .039$; $r = 0.449$, $P = .001$; $r = 0.421$, $P = .002$; $r = 0.429$, $P = .002$).

In the analysis of the association between the eyes-closed resting-state EEG indices and sleep distress subscale score (Table 3), a significant correlation was noted between sleep distress subscale score and the theta and beta bands in the electrodes in the frontal area ($r = 0.382$, $P = .005$; $r = 0.430$, $P = .001$), between sleep distress subscale score and the delta, theta, alpha, and beta bands at electrode Cz ($r = 0.315$, $P = .024$; $r = 0.364$, $P = .009$; $r = 0.346$, $P = .013$; $r = 0.425$, $P = .002$), and between sleep distress subscale score and the delta, theta, alpha, and beta bands at electrode Pz ($r = 0.357$, $P = .011$; $r = 0.470$, $P = .001$; $r = 0.405$, $P = .004$; $r = 0.479$, $P = .001$).

In the analysis of the association between the eyes-closed resting-state EEG indices and the sleep quality subscale score (Table 3), a significant correlation was observed between the sleep quality subscale score and the beta band in the electrodes in the frontal area ($r = 0.295$, $P = .034$) and between the sleep quality subscale score and the theta, alpha, and beta bands at electrode Pz ($r = 0.302$, $P = .033$; $r = 0.282$, $P = .047$; $r = 0.323$, $P = .024$).

The analysis of the association between the eyes-closed resting-state EEG indices of the adult ADHD group and PIRS_20 score revealed a significant correlation between PIRS_20 score and the alpha and beta bands at electrode Cz ($r = 0.446$, $P = .025$; $r = 0.408$, $P = .043$) and between

Table 1. Demographic and Clinical Differences Between the Two Groups

	Adult ADHD Group (n=26) Mean (SD)	Control Group (n=26) Mean (SD)	*P*
Age	28.85 (6.17)	29.65 (5.84)	.630
Education level	16.15 (1.43)	16.35 (2.10)	.701
PIRS_20 score	26.54 (10.61)	13.80 (10.25)	**<.001**
Distressing subscale	16.15 (7.19)	6.42 (6.12)	**<.001**
Quality subscale	7.04 (2.85)	4.88 (2.83)	**.009**
Parameter subscale	3.35 (2.53)	2.50 (2.25)	.208
Independent sample *t* tests between the adult ADHD group and the control group			
Sex			
Male (ratio)	23 (50%)	23 (50%)	1.000
Female (ratio)	3 (50%)	3 (50%)	

Chi-square test between the adult ADHD group and the control group.
Bold = significant ($P < .05$).
PIRS_20, Twenty-item Pittsburgh Insomnia Rating Scale.

Table 2. Differences in Electroencephalography Activity Between the Two Groups

	Adult ADHD Group (n = 26) Mean (SD)	Control Group (n = 26) Mean (SD)	*P*
Delta			
Frontal	16.20 (3.83)	13.95 (2.90)	**.021†**
Cz	14.91 (4.28)	11.69 (2.95)	**.003†**
Pz	14.53 (4.41)	11.24 (3.59)	**.006†**
Theta			
Frontal	8.32 (2.78)	6.60 (2.32)	**.019†**
Cz	8.30 (3.30)	6.29 (2.41)	**.017†**
Pz	7.42 (3.32)	5.23(2.90)	**.017†**
Alpha			
Frontal	9.87 (2.59)	8.63 (2.46)	.081
Cz	10.96 (2.30)	9.25 (2.53)	**.015†**
Pz	11.16 (2.18)	8.77 (2.91)	**.002†**
Beta			
Frontal	1.75 (2.39)	.05 (2.15)	**.010†**
Cz	1.82 (2.45)	.02 (2.44)	**.011†**
Pz	1.56 (2.43)	−.18 (2.31)	**.014†**

Independent sample *t* tests between the adult ADHD group and the control group.
Bold = significant ($P < .05$).
EEG, electroencephalography.
†, still significant after Benjamini-Hochberg correction.

PIRS_20 score and the theta, alpha, and beta bands at electrode Pz ($r = 0.405$, $P = .045$; $r = 0.504$, $P = .010$; $r = 0.461$, $P = .020$) (Table 4).

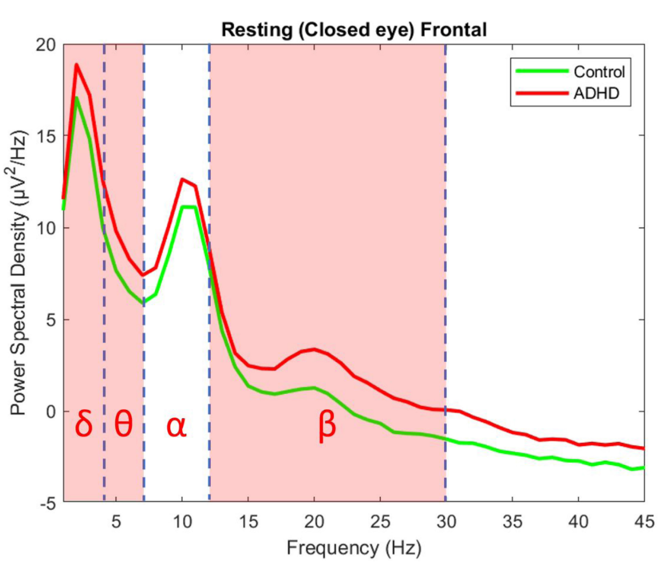

Figure 1. The power spectral density at electrode frontal area for the adult ADHD group (red line) and the control group (green line) of 4 frequency bands in the eye-closed condition. Greater values (red background area) represent significant higher power in the adult ADHD group (*P*-value < .05).

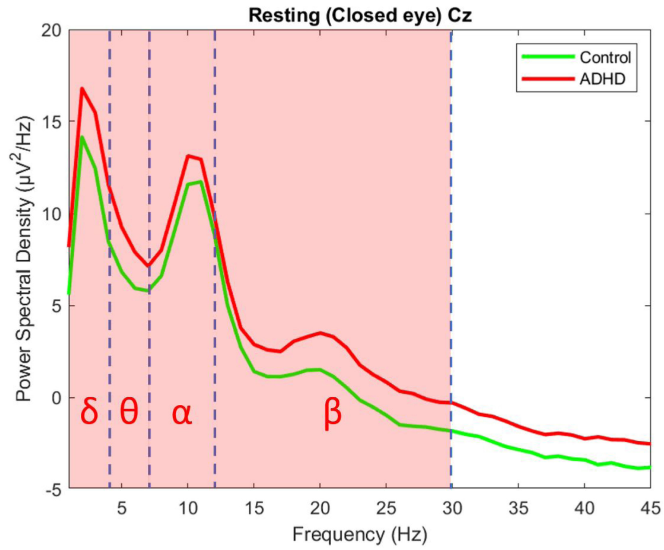

Figure 2. Power spectral density at electrode Cz for the adult ADHD group (red line) and the control group (green line) of 4 frequency bands in the eye-closed condition. Greater values (red background area) represent significant higher power in the adult ADHD group (*P*-value < .05).

The analysis of the association between the eyes-closed resting-state EEG indices of the adult ADHD group and the sleep distress subscale score indicated a significant correlation of the sleep distress subscale score with the beta band in the electrodes in the frontal area ($r = 0.395$, $P = .046$), with the beta band at electrode Cz ($r = 0.402$, $P = .046$), and with the beta band at electrode Pz ($r = 0.402$, $P = .046$) (Table 4).

An analysis of the association between the eyes-closed resting-state EEG indices of the adult ADHD group and sleep

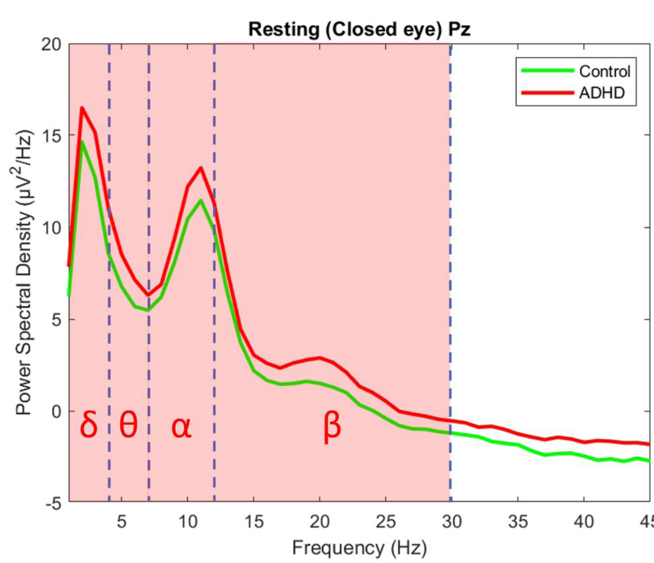

Figure 3. Power spectral density at electrode Pz for the adult ADHD group (red line) and the control group (green line) of 4 frequency bands in the eye-closed condition. Greater values (red background area) represent significant higher power in the adult ADHD group (*P*-value < .05).

Table 3. Correlations Among Measures (All Participants, n=52)

	PIRS_20 (*r*, *P*)	Distressing Subscale (*r*, *P*)	Quality Subscale (*r*, *P*)
Delta			
Frontal	0.138, .331	0.257, .066	0.039, .782
Cz	0.232, .101	**0.315*, .024**	0.162, .476
Pz	**0.293*, .039**	**0.357*, .011**	0.158, .275
Theta			
Frontal	**0.333*, .015**	**0.382**, .005**	0.144, .308
Cz	**0.322*, .021**	**0.364**, .009**	0.143, .317
Pz	**0.449**, .001**	**0.470**, .001**	**0.302*, .033**
Alpha			
Cz	**0.327*, .019**	**0.346*, .013**	0.133, .352
Pz	**0.421**, .002**	**0.405**, .004**	**0.282*, .047**
Beta			
Frontal	**0.387**, .005**	**0.430**, .001**	**0.295*, .034**
Cz	**0.365**, .008**	**0.425**, .002**	0.266, .060
Pz	**0.429**, .002**	**0.479**, .001**	**0.323*, .024**

Spearman's correlation coefficient.
PIRS_20, Twenty-item Pittsburgh Insomnia Rating Scale.
Bold=significant ($P < .05$). *$P < .05$, **$P < .01$.

quality subscale score indicated a significant correlation of sleep quality subscale score with the alpha band at electrode Pz ($r=0.432$, $P=.031$) and with the beta band at electrode Pz ($r=0.398$, $P=.049$) (Table 4).

Table 4. Correlations Among Measures (Participants in the Adult ADHD Group, n=26)

	PIRS_20 (*r*, *P*)	Distressing Subscale (*r*, *P*)	Quality Subscale (*r*, *P*)
Delta			
Frontal	−0.086, .675	0.155, .450	−0.258, .204
Cz	−0.023, .914	0.115, .593	−0.130, .535
Pz	−0.004, .985	0.093, .657	−0.066, .754
Theta			
Frontal	0.320, .111	0.310, .123	0.106, .605
Cz	0.355, .082	0.296, .150	0.180, .389
Pz	**0.405, .045**	0.303, .140	0.346, .090
Alpha			
Cz	**0.446, .025**	0.376, .054	0.276, .101
Pz	**0.504, .010**	0.367, .071	**0.432, .031**
Beta			
Frontal	0.387, .051	**0.395, .045**	0.260, .200
Cz	**0.408, .043**	**0.402, .046**	0.299, .146
Pz	**0.461, .020**	**0.402, .046**	**0.398, .049**

Spearman's correlation coefficient.
PIRS_20, Twenty-item Pittsburgh Insomnia Rating Scale.
Bold=significant ($P < .05$).

DISCUSSION

The adult ADHD group had more distressing sleep problems and lower sleep quality than the control group did. The PSDs of the 4 EEG frequency bands (delta, theta, alpha, and beta) at the frontal area, at Cz, and at Pz were all significantly higher in the adult ADHD group than those in the control group, except for the alpha band at the frontal area. Correlations were also observed between the theta band at Pz, the alpha band at Cz and Pz, and the beta band at the frontal area, Pz, and Cz with PIRS_20 score, sleep distress subscale score, or sleep quality subscale score in the adult ADHD group.

Resting-state EEG is a real-time, inexpensive, and easily accessible tool for clinical diagnosis and treatment evaluation. Its utility in ADHD evaluation, including in the diagnosis of childhood and adolescent ADHD and in neurofeedback, represents a promising non-pharmacological treatment modality. Because few studies have investigated EEG patterns in adults with ADHD and those that have focused on this have reported inconsistent results, the present study investigated reliable EEG indices for identifying adult ADHD. Consistent with results in the literature, it was noted that higher PSDs were associated with slow-wave frequency bands, especially the theta band, in the adult ADHD group. This pattern is also apparent in child and adolescent ADHD, which may explain why some ADHD symptoms persist into adulthood. The globally increased PSD of slow-wave activity may also support the hypoarousal model of ADHD.[42] According to this model, the core symptoms of ADHD result from the hypoarousal of the central nervous system. Studies have provided inconsistent results on fast-wave activity. For the increased PSD of the beta wave, these findings are consistent with those of Bresnahan and Barry,[11] which may explain why most hyperactivity symptoms remit in adulthood and may support the maturational delay model of ADHD. For the increased PSD of the alpha wave, the findings are consistent with those of Bresnahan and Barry[11] and Koehler et al.[13] Poil et al[43] reported the increased PSD of the alpha wave is highly dependent on age and may be related to the distinct brain maturation processes in individuals with ADHD. However, interpreting the results of the current study is difficult because of the inconsistency across studies in the literature.

The adult ADHD group had a significantly higher PIRS_20 score than the control group, indicating that they had a less favorable sleep condition. The subscale scores indicated that the adult ADHD group had significantly higher scores in the sleep distress subscale and sleep quality subscale than the control group. Consistent with the literature, the results revealed that the adult ADHD group had more subjectively bothersome symptoms caused by nocturnal sleep disturbance and a subjectively less satisfying nocturnal sleep quality. Unlike other studies, the present study did not reveal any difference between the 2 groups in sleep parameter subscale aspects such as sleep onset latency or total sleep time. Because sleep apnea, restless leg syndrome, and periodic leg movement disorder are commonly associated with sleep disturbances in ADHD, future studies using objective measures (e.g., polysomnography, multiple sleep latency tests, and actigraphy) are required to further elucidate sleep disturbances as measured through objective sleep parameters and their association with sleep breathing or movement disorders.[44-47]

The present study performed a correlational analysis to determine whether the differences in EEG indices between the 2 groups were associated with sleep disturbance. The within-group analysis revealed that the significant association only existed within the adult ADHD group and not in the control group. It was surmised that the differences in EEG indices between the 2 groups may at least partially result from the sleep problems experienced by the adult ADHD group.

In the within-group correlation analysis, the PIRS_20 score was associated with the alpha and beta bands at Cz and the theta, alpha, and beta bands at Pz; the sleep distress subscale score was associated with the global beta band; and the sleep quality subscale score was associated with the alpha and beta bands at Pz. Similar EEG findings were noted in patients with comorbid depression and insomnia.[48] It was thus inferred that significantly higher high-frequency band activity in adults with ADHD is associated with sleep disturbance. This finding supported the hyperarousal theory of insomnia and may explain why neurofeedback SMR training, which maintains brainwave frequency between 12 and 15 Hz, can be effective for treating insomnia and ADHD core symptoms.[49]

Models have been proposed to explain the relationship between ADHD and insomnia.[50] The first model holds that ADHD symptoms, especially hyperactivity, lead to insomnia. This is supported by 1 study reporting that stimulants, the standard pharmacological treatment for ADHD, can relieve the sleep problems of adults with ADHD.[29] The second model maintains, conversely, that insomnia causes ADHD symptoms. Case reports revealed that daytime ADHD symptoms could be alleviated through managing different sleep problems (e.g., delayed sleep onset, sleep-disordered breathing, and periodic limb movements disorder).[51] Other models suggest that ADHD and insomnia share common or overlapping neurological deficits or that the 2 disorders are coincidentally comorbid. These models propose that ADHD is composed of several different phenotypes. Therefore, assessments of whether adults with ADHD have sleep problems must be incorporated into individualized treatment planning. Further research must be conducted to elucidate the causal relationship between ADHD and insomnia to, in turn determine whether resting-state EEG measurements can be used to differentiate between the phenotypes of adult ADHD. For instance, a study could investigate whether the sleep condition the night before the resting-state EEG examination affects the EEG indices studied in the present research.

The current study has several limitations. First, the sample size was relatively small, with only 26 participants in each group, which may limit generalizability. However, most other studies on EEG presentation in adults with ADHD have had fewer than 50 participants in each group. The reason may be the lower prevalence of adult ADHD compared with that of childhood ADHD. Future studies may need larger sample sizes, more specific diagnostic criteria for a more homogeneous sample component, and ADHD subtype analysis to expand the generalizability of the findings. Second, only eyes-closed resting-state EEG results were investigated. Because eyes-open and eyes-closed EEG may produce different results, the outcomes could not be compared to those of studies that used eyes-open resting-state EEG. Third, 1 participant in the adult ADHD group was treated using stimulants, which may influence the participant's EEG results and sleep condition. However, the participant was asked to refrain from taking the medication at least 24 hours before their EEG examination. To entirely rule out the medication's effects on participants' EEG results, future studies may require participants to refrain the participants from taking the medication for a longer time. Fourth, because of the small sample size, there was no further differentiation of the adult ADHD group into subtypes, as some studies have done. Finally, the self-report PIRS_20 score was used as the sole indicator of sleep problems. If the PIRS_20 score was combined with objective sleep parameter measurements, the results might be more robust. However, traditional objective measurements, such as polysomnography, typically only reflect the individual's sleep condition over 1 night and in an unfamiliar environment. Thus, the use of portable measurement equipment for monitoring participants at home could be considered.

To the knowledge of the authors, the present study is the first to use eyes-closed resting-state EEG to examine the correlation between EEG indices and insomnia in adult ADHD. In conclusion, this study revealed the globally increased PSDs of the delta, theta, alpha (except for the frontal area), and beta bands that occur in adults with ADHD in an eyes-closed resting-state condition. Regarding the association between insomnia and adult ADHD, the findings are consistent with those of studies reporting that adults with ADHD have more bothersome sleep problems and lower sleep quality than those without ADHD. Moreover, the eyes-closed resting-state EEG indices in this study, especially the alpha and beta bands at Cz and Pz, are correlated with insomnia in adult ADHD. The findings of the present study provide scientific evidence for the crucial nature of managing sleep problems in adult ADHD and also support the use of neurofeedback SMR training in simultaneously alleviating core symptoms of ADHD and insomnia. Future research may further explore distinct EEG patterns using event-related potentials to broaden the understanding of the neurophysiological characteristics of adult ADHD; they may employ a longitudinal design to further elucidate the causal relationship between insomnia and EEG findings in adult ADHD.

Data Availability Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethics Committee Approval: This study was approved by the Ethics Committee of Kaohsiung Medical University Chung-Ho Memorial Hospital (Approval no.: KMUHIRB-SV[II]-20170073; Date: 2018/02/27).

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

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