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A Randomized Controlled Trial Comparing Neurofeedback and Cognitive-Behavioral Therapy for Insomnia Patients: Pilot Study

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Abstract

Insomnia is a common disease that negatively affects patients both mentally and physically. While insomnia disorder is mainly characterized by hyperarousal, a few studies that have directly intervened with cortical arousal. This study was conducted to investigate the effect of a neurofeedback protocol for reducing cortical arousal on insomnia compared to cognitive-behavioral treatment for insomnia (CBT-I). Seventeen adults with insomnia, free of other psychiatric illnesses, were randomly assigned to neurofeedback or CBT-I. All participants completed questionnaires on insomnia [Insomnia Severity Index (ISI)], sleep quality [Pittsburgh Sleep Quality Index (PSQI)], and dysfunctional cognition [Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS-16)]. The neurofeedback group showed decreases in beta waves and increases in theta and alpha waves in various areas of the electroencephalogram (EEG), indicating lowered cortical arousal. The ISI and PSQI scores were significantly decreased, and sleep efficiency and sleep satisfaction were increased compared to the pre-treatment scores in both groups. DBAS scores decreased only in the CBT-I group (NF $p = 0.173$; CBT-I $p = 0.012$). This study confirmed that neurofeedback training could alleviate the symptoms of insomnia by reducing cortical hyperarousal in patients, despite the limited effect in reducing cognitive dysfunction compared to CBT-I.

Keywords Hyperarousal · Insomnia · Neurofeedback · Non-pharmacological treatment

Introduction

Insomnia disorder is the most common sleep disorder (Basiri et al., 2017), characterized by difficulties in initiating and maintaining sleep, despite ample opportunity to sleep (Renom-Guiteras et al., 2014). One of the most prominent models playing a key role in explaining the pathophysiology of insomnia is the hyperarousal model (Riemann and Perlis, 2009), which has been characterized by significantly higher sympathetic and lower parasympathetic activity during sleep (Varkevisser, Van Dongen, & Kerkhof, 2005), decreased heart rate variability (Spiegelhalder et al, 2011; Bonnet & Arand, 1995; Haynes et al., 1981; Stepanski et al., 1994), increases in body temperature (Chiu et al., 2000), and increases in the basal metabolic rate (Bonnet & Arand, 1995).

EEG studies have also shown that patients with insomnia have high cortical activation (Molen et al., 2014). Various EEG studies have shown that the increased high-frequency EEG activity ranged from 15 to 40 Hz in insomnia patients (Fernandez-Mendoza et al., 2012; Kwan et al., 2018). In an EEG study conducted in the resting state, patients with insomnia had higher EEG power in the frontal, temporal, and parietal lobes in the eyes-closed (EC) state compared to non-insomnia controls (Colombo et al., 2016). Other EEG studies also showed that high-frequency EEG activity predominated in the left-frontal and anterior-middle areas during sleep and incubation periods in insomnia patients (Kay & Buysse, 2017). These various studies suggest that cortical hyperarousal in insomnia patients was seen as increased high-frequency activity (Kay & Buysse, 2017; Kwan et al., 2018).

Yunna Kwan and Soyoung Yoon made equal contributions and are co-first authors.

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Neurofeedback training for insomnia patients may increase theta waves, increase Sensorimotor rhythm (SMR; 12–15 Hz) waves, and individualized protocols using Z scores. Theta wave-increasing training is similar in mechanism to relaxation therapy, which is widely used in the treatment of insomnia disorders (Batty et al, 2006). However, a study showed that neurofeedback training to reduce beta waves was more effective in treating insomnia than relaxation training (Kim et al., 2017).

SMR wave-enhancing training helps sleep by increasing sleep spindles at 12–14 Hz, a frequency band similar to that during sleep (Cortoos et al., 2010). However, there is still controversy over whether insomnia is due to a lack of sleep spindles (Bastien et al, 2009), and studies have shown that SMR wave-enhancing training is ineffective in insomnia patients with high arousal. Furthermore, several researchers have classified SMR as a high-frequency band (beta-1) rather than a low-frequency one, based on studies showing that SMR was associated with optimal cognitive performance (Egner & Gruzelier, 2001, 2004; Vernon et al., 2003). Taking this into account, it is difficult to rule out the risk that the SMR enhancing protocol will increase arousal.

Finally, there is a neurofeedback training protocol that is personalized using Z scores. This is a treatment that uses statistical techniques using standard deviations to determine how different the brainwaves of a patient are from normal, and then normalizes the abnormal EEG patterns seen in individual patients (Hammer et al., 2011). However, there is a limitation that differences in anatomical characteristics will affect the individual absolute power variance of the EEG (Nam & Choi, 2020) and the inconvenience of attaching electrodes from several channels in each session.

Previous neurofeedback protocols have rarely shown attempts to directly modulate beta waves. Neurofeedback interventions work through a mechanism to alleviate symptoms by normalizing abnormal brain waves in a particular group of patients (Yucha & Montgomery, 2008). It is necessary to determine whether reducing beta waves can help alleviate insomnia symptoms, considering the evidence that the major physiological characteristic of insomnia is cortical hyperarousal, which is manifested by an increase in beta waves. However, we could identify one study on beta reduction training. The study found that a beta wave-reducing protocol an advantage over SMR-enhancing and relaxation training protocols despite the small number of samples (Kim et al., 2017). Beta-reducing neurofeedback may be effective in reducing hyperarousal in insomnia patients who show neurophysiological abnormalities. Thus, this study aimed to compare the efficacy of neurofeedback training for reducing beta waves by comparing it to an already existing effective treatment, namely cognitive-behavioral therapy for insomnia (CBT-I), which is an evidence-based treatment for insomnia disorder. CBT-I is an effective non-pharmacological treatment for insomnia and is considered the first-line of treatment for insomnia disorder (Qaseem et al., 2016).

This study aimed to compare neurofeedback to CBT-I in 17 insomnia patients through a randomized controlled trial. The study utilized a randomized, parallel-group, single-blind experimental design. We hypothesize that neurofeedback and CBT-I will similarly decrease insomnia symptom severity and increase sleep quality. Also, neurofeedback training is hypothesized to be effective in reducing beta wave amplitude.

Methods

Participants

The participants applied to an insomnia treatment program conducted by the Department of Psychology at a university in Seoul, South Korea from 2017 through 2018. They participated in the research by seeing the recruitment announcement on the Social Network Service or the advertisement on the department's bulletin board. A total of 64 applicants applied to participate in the study and all of them signed informed consent before starting the study. The participants were screened based on the following process: (1) a brief telephone interview to exclude individuals taking medication or working as a shift worker, (2) a cutoff score on the ISI of above 15, and (3) a diagnosis made using Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association, 5th edition (DSM-5) criteria based on a detailed clinical interview by a licensed clinical psychologist. For additional psychiatric co-morbidities, a structured clinical interview for DSM-IV (SCID) was used, which had been validated for DSM-IV (First, Spitzer, Gibbon, & Williams, 1997).

Although the overall sample size was small, we tried to minimize the possibility of confounders that could affect the treatment and assign an unbiased sample to each treatment group through random assignment. Therefore, a total of 17 patients with insomnia and free of other psychiatric illnesses illness were randomly assigned to the neurofeedback or CBT-I group. The average age of the participants was 25 years, with 6 men and 11 women.

Inclusion/Exclusion Criteria

Participants qualified for the study if they met the diagnostic criteria for insomnia disorder according to the DSM-5 and also met the following criteria: (1) PSQI score > 6, ISI score > 15 (Morin et al., 2011), (2) free of other psychiatric illnesses as determined by a psychological screening interview (DSM-IV; SCID-I). (3) free of obstructive sleep apnea

(OSA) syndrome screened by the Berlin questionnaire, (4) not taking medication for insomnia disorder, and (5) was not a shift worker or had experienced jet lag within the past six months.

Some participants were excluded from the study. Thirteen did not meet the PSQI and ISI criteria, 14 were excluded due to comorbidities, six were excluded because they were at high risk for OSA, six were excluded due to taking medications, one was a shift worker, and one experienced jet lag within six months. Finally, five people voluntarily withdrew from the study.

Intervention

Neurofeedback treatment was performed using Thought Technology's Procomp 5 at Duksung Women's University. The protocol was used in previous studies as neurofeedback treatment to reduce the absolute power of the beta waves (18–30 Hz), indicating cortical arousal, and maintain the sigma wave (12–15 Hz), known as a sleep-protective mechanism (Cortoos et al., 2010). Based on the results of a previous study (Corsi-Cabrera et al., 2012) that many beta wave activities appeared in the left frontal region of patients with insomnia, the procedure was carried out in F3 and F7 (according to a 10–20 electrode arrangement). The average sigma value of each participant was calculated from the pre-measured EEG values and set to a fixed threshold. The beta waveband is relatively broad compared to other frequency bands. According to the evidence that high and low beta bands are associated with different mental activities (Díaz et al., 2019; Lim et al., 2019), we divided the bands into narrower segments to fine-tune the neurofeedback target of beta-2 (15–18 Hz), beta-3 (18–25 Hz), and beta-4 (25–30 Hz). The threshold values were set according to performance level to allow the participants to receive 80% rewards during neurofeedback training. The sound of nature was rewarded when the beta-2, 3, and 4 values fell below the threshold while the sigma values rose above the threshold. A total of 10 sessions (30 min per session) were conducted.

CBT-I was based on the program introduced by Perlis et al. (2005). Structured sessions for sleep education, sleep restriction and sleep hygiene, stimulation control, relaxation therapy, and relapse prevention were conducted by two master-level student therapists who were trained in CBT-I. They were supervised by a licensed clinical psychologist who was a behavioral sleep medicine specialist. CBT-I consisted of a total of six weekly sessions (50 min per session). Assessments were conducted at baseline and post-treatment.

Measures

Korean Version of the Pittsburgh Sleep Quality Index (PSQI-K)

The sleep quality of the participants was measured using the Korean version of the PSQI. The PSQI-K consists of 19 questions distributed across the seven components of sleep quality, sleep onset latency, sleep duration, sleep efficiency, sleep disturbances, the use of sleeping medications, and daytime dysfunction. Each item is scored from 0 to 3, and the total scores of the seven components are referred to as the global PSQI score, which ranges from 0 to 21. A score of 6 or more reflects poor sleep quality (Sohn et al., 2012), which has a strong correlation with sleep efficiency and subjective sleep quality.

Insomnia Severity Index (ISI)

The ISI is a seven-item self-reported measure of insomnia symptoms and related distress over the past month (Bastien, 2001). The measure consists of seven items targeting sleep disturbance severity, sleep-related satisfaction, the degree of daytime functional impairment, impairment perception and distress, and concerns related to sleeping problems. Each item is rated on a 5-point Likert scale (0 to 4) and summed to provide a total score ranging from 0 to 28. Higher scores reflect more severe insomnia symptoms. A score above 15 points is considered a clinically significant insomnia disorder, and a score below 8 points is considered remission following treatment (Sohn et al., 2012).

Korean Version of the Epworth Sleepiness Scale (ESS)

The Epworth Sleepiness Scale (ESS; Johns, 1991) is an 8-item self-reported questionnaire that measures the propensity to sleep and feel sleepy during the day in everyday situations. It consists of a 4-point Likert scale, which is rated between 0 and 3 points for eight questions on the possibility of dozing or falling asleep in certain everyday situations (Cho et al., 2009).

Pre-sleep Arousal Scale (PSAS)

The PSAS consists of 16 items and assesses arousal before falling asleep, making the distinction between physical and cognitive arousal.

Glasgow Sleep Effort Scale (GSES)

This scale consists of seven items and was developed by Broomfield and Espie (2005). The GSES assesses the present state of sleep effort. The responses are recorded on a 3-point Likert scale from not at all (0), to some extent (1), and very much (2). Higher scores on the scale indicate higher degrees of effort to control sleep (Kim, 2014). The reliability of the Korean version of K-GSES, which was revised and validated by Kim (2014), was 0.76, and the test–retest reliability was 0.83.

Dysfunctional Beliefs and Attitudes about Sleep Scale 16 (DBAS 16)

Morin et al. (1993) developed a scale of 30 questions to identify irrational beliefs and attitudes about sleep. Morin, Vallires, & Ivers (2007) composed and validated 16 questions. There are four factors on a 10-point Likert scale: false attribution to the causes and consequences of insomnia, loss of control over sleep, worries about sleep, wrong expectations about sleep, and attitudes toward sleeping pills. Yoo et al. (2009) translated and validated the degree of dysfunctional beliefs and attitudes toward sleep (K-DBAS 16) and the reliability was 0.85.

Beck Depression Index-II (BDI-II). This is a self-reported inventory developed by Beck et al. (1996) designed to measure the severity of depressive symptomatology. It is a 21-item inventory. Each item is rated on a 4-point scale from 0 to 3 with summed scores ranging from 0 to 63. The reliability of the Korean version of the BDI, which was revised and validated by Sung et al. (2008) was 0.834, with a sensitivity of 0.935 and a specificity of 0.981. The reliability determined by Lim, Hwang, Hong, & Kim (2014) was 0.89 and the correlation between the items ranged from 0.25 to 0.57.

Beck Anxiety Index (BAI)

This scale is a self-reported measure of anxiety. It is a 21-item inventory with an internal consistency of 0.92 and a 1-week test–retest reliability of 0.75 (Beck et al., 1988). The reliability of the Korean version of the Beck Anxiety Scale translated by Cho and Kim (2004) showed validity and reliability of 0.90. Lee et al. (2016) found that the reliability was 0.91 and the correlation between the items was between 0.18 and 0.64.

Berlin questionnaire (BQ). The BQ (Netzer et al., 1999) incorporates questions on snoring (category 1), daytime somnolence (category 2), and hypertension and body mass index (BMI) (category 3). Positive responses in two or more of the three categories indicate patients at high risk for OSA (Kang et al., 2011). The reliability of the Korean version of the Berlin questionnaire revised by Kang et al (2013) was 0.64 to 0.78.

Sleep Diary

A sleep diary, which was revised and validated by Kim (2014) was used for total sleep time (TST), time in bed (TIB), sleep efficacy (SE), sleep latency (SL), and sleep satisfaction.

Consumer Sleep Technology–Wearable Device

Additionally, smart wearable devices (Xiaomi Mi band2) were used to measure total sleep time (Xie et al., 2018). Unlike the sleep diary, which records the subject's sleep problems, a smart wearable device can objectively measure sleep-related indicators. It is worn on the wrist to evaluate sleep or arousal status using motion detection and the activity index of the participant during sleep (Sadeh & Acebo, 2002). Compared to traditional actigraphs, the Mi-Band has shown a good level of accuracy and is often used in sleep studies because of its low cost and ease of use (Kubala et al., 2020).

Resting-State EEG

A reference electrode and ground electrode were attached to the left and right earlobes, respectively, and 19 areas according to the 10–20 international electrode placement method using the Brain Master Discovery 20 and Electro Cap (FP1, F3, F7, Fz, FP2, F4, F8, T3, C3, Cz, T4, C4, T5, P3, O1, Pz T6, P4, and O2) were recorded. Measurements were recorded for five minutes, one time each in eye open (EO) and eye closed (EC) conditions since patients with insomnia exhibited different characteristics in EO and EC conditions in previous studies (Colombo et al., 2016; Kwan et al., 2018). Normally, the alpha frequency band appears strongly as the visual stimulus is blocked in the EC condition. However, there was evidence that insomnia patients had an increase in high-frequency amplitude in the overall cortical area compared to the healthy control group, which was thought to reflect physiological hyperarousal. In contrast, a previous study on resting-state EEG in the EO condition of insomnia patients found increased high-frequency amplitude in the left central area. That is, insomnia patients were thought to exhibit focal hyperarousal in the region that included the cortical network utilized for attention or higher-order cognitive function in the EO condition in which relatively aroused and visual stimuli are input.

Pre-measurement, post-measurement, and follow-up resting brain waves were measured using NeuroGuide (NeuroGuide, Applied Neuroscience, Inc., St. Petersburg, FL, USA) software to remove artifacts and were analyzed. Artifacts due to body movement or blinking eyes were eliminated by auto-editing. After the removal of artifacts, the split-half reliability was at least 0.95 and the test–retest reliability was at least 0.90. The delta (1 – 4 Hz), theta (4–8 Hz), alpha (8–12 Hz), sigma (12–15 Hz), beta-2 (15–18 Hz), beta-3 (18–25 Hz), beta-4 (25–30 Hz), and gamma (30–40 Hz) = frequency bands were designated via fast Fourier transform (FFT). Topographic color mapping was performed using Neurorosat to visually identify the difference in absolute power before and after treatment and the absolute power difference between the after-treatment and follow-up brain waves.

Variables related to sleep and EEG were measured pre-, post-, and 2 weeks after (follow-up) treatment. Every participant in the study completed all measurements.

Statistical Analysis

All statistical analyzes were performed with SPSS 21.0 (IBM Corporation, Armonk, NY, USA). In this study, non-parametric statistics were performed because the sample size did not satisfy normal distribution. Before treatment, the Mann–Whitney test was performed on the demographic variables, resting EEG, insomnia scale, and sleep data to confirm homology between the neurofeedback group and the CBT-I group. To verify the effectiveness of treatment, Wilcoxon's signed-rank test was used to compare the pre- and post-treatments in each group.

Table 1 Homogeneity test and statistics of demographic and heart rate features

	NF(n = 9) M(SD)	CBT-I(n = 8) M(SD)	Z	p-value
Age	25.78(4.02)	23.63(3.38)	− 1.863	.062
Education degree	15.44(.88)	14.75(.71)	− 1.741	.082
IQ	107.71(13.11)	103.79(8.93)	− .194	.846
BMI	21.04(2.2)	21.74(3.5)	− .289	.773
BQ	2.67(1.87)	1.38(.74)	− 1.751	.08
Heart rate	75.84(19.52)	70.6(6.05)	− .481	.630
LF	34.39(17.75)	27.91(12.15)	− .962	.336
HF	43.08(17.52)	46.15(17.12)	− .192	.847
LF/HF	1.48(1.76)	1.07(1.08)	− .577	.564

BMI Body Mass Index, *BQ* Berlin questionnaire, *LF* low frequency, *HF* high frequency

Table 2 Homogeneity test and insomnia symptoms of pre-measurement

	NF(n = 9) M(SD)	CBT-I(n = 8) M(SD)	Z	p-value
ISI	18.67(2.73)	17.63(2.72)	− 7.88	.430
PSQI	12.11(3.79)	10(2.88)	− 1.065	.287
DBAS 16	98(31.24)	85.88(20.12)	− 1.204	.229
ESS	10.22(4.21)	7.75(4.65)	− 1.061	.289
PSAS	53.89(10.2)	57.88(11.89)	− .916	.359
GSES	8.22(3.77)	7.63(1.3)	− .436	.663
TST	387(77.97)	368.91(86.32)	− .578	.563
SL	43.22(20.58)	56.04(34.41)	− .434	.665
WASO	12.78(15.36)	17.04(17.46)	− .339	.735
Sleep efficacy	84.07(6.55)	76.9(12.63)	− 1.348	.178
Sleep satisfictioin	4.92(1.27)	5(1.58)	− .145	.885

ISI Insomnia severity index, *PSQI* pittsburgh sleep quality index, *DBAS 16* dysfunctional beliefs and attitudes about sleep scale, *ESS* epworth sleepiness scale, *PSAS* pre sleep arousal scale, *GSES* glasgow sleep effort scale, *TST* total sleep time, *SL* sleep latency, *WASO* wake after sleep onset

Results

Demographic Characteristics

The Mann–Whitney test was performed on demographic variables, EEG, heart rate and heart rate variability, insomnia scale, and sleep data variables to determine the presence of heterogeneous variables that may affect the treatment outcomes. No differences were found between the two groups (Tables 1 and 2).

EEG Changes from Baseline to Post-treatment

The neurofeedback group showed a significant increase in theta and alpha waves and a significant decrease in beta waves in many areas after treatment (Fig. 1, Supplementary Table 1). The CBT-I group also showed a significant increase in theta and alpha waves and a significant decrease in beta waves (Fig. 2, Supplementary Table 2).

Sleep Changes from Baseline to Post-treatment

All sleep scales except DBAS-16 and ESS were significantly decreased in the neurofeedback treatment group. CBT-I treatment group showed a significant decrease in all sleep scales except ESS (Table 3). The changes in sleep date in the neurofeedback and CBT-I populations were as follows (Table 4). In the neurofeedback treatment group, total sleep time (TST) after treatment ($z = -2.666$, $p = 0.008$), sleep efficiency (SE) ($z = -2.547$, $p = 0.011$), and sleep satisfaction ($z = -2.549$, $p = 0.011$) significantly increased,

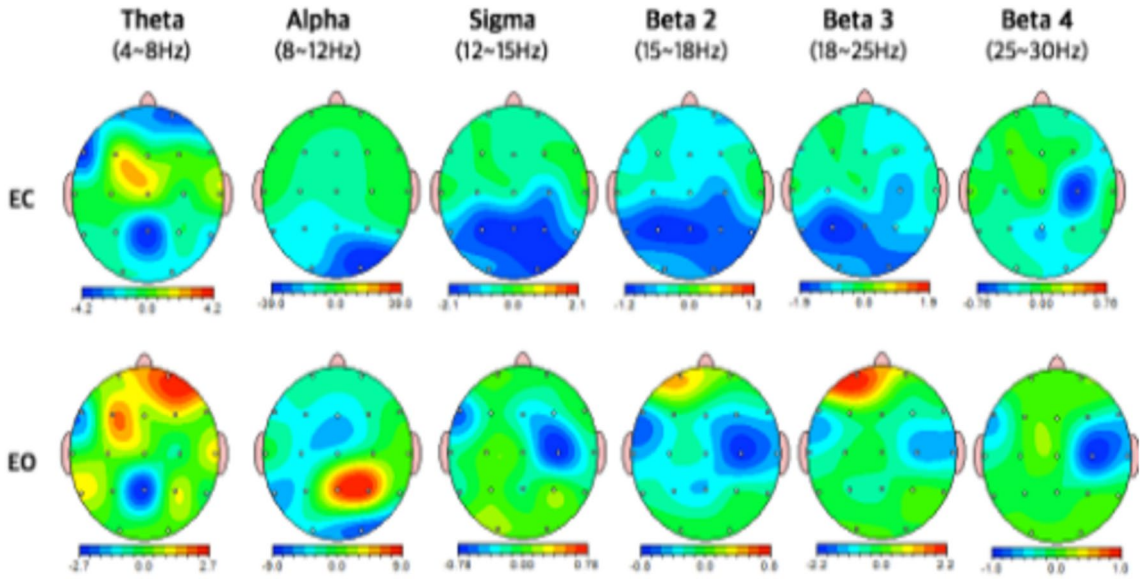

Fig. 1 Topographical maps of pre-to-post EEG change of neurofeedback group. *EC* Eye closed, *EO* eye open

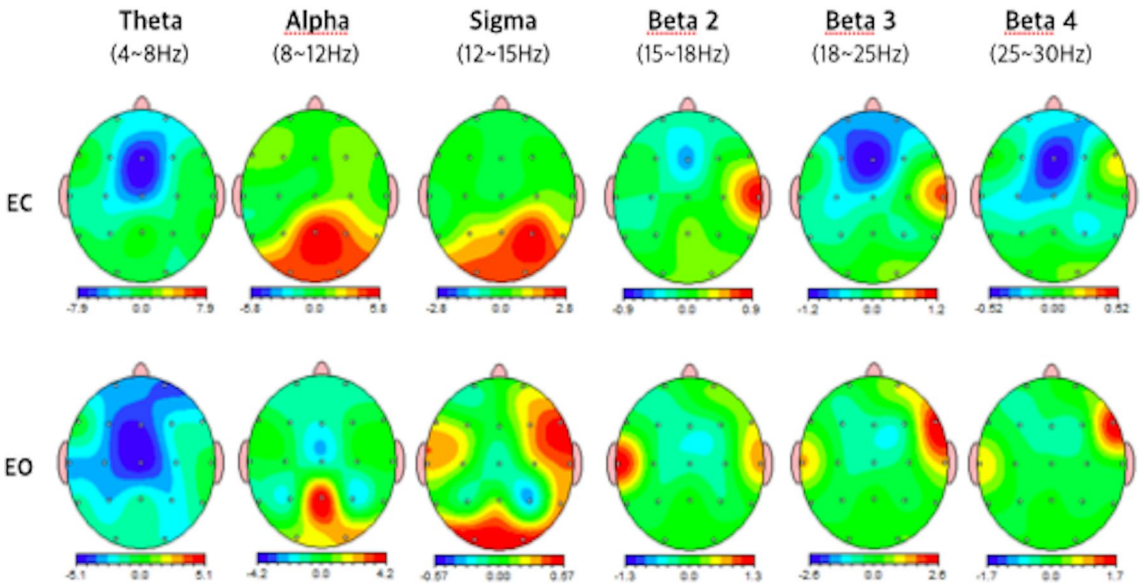

Fig. 2 Topographical maps of pre-to-post EEG change of CBT-I group. *EC* Eye closed, *EO* eye open

and sleep latency (SL) ($z = -2.057$, $p = 0.038$) significantly decreased. In the CBT-I treatment group, sleep efficiency (SE) ($z = -2.547$, $p = 0.012$) and sleep satisfaction ($z = -2.521$, $p = 0.012$) significantly increased and waking after sleep onset (WASO) ($z = -2.103$, $p = 0.035$) significantly decreased.

Comparison with Post and Follow-up Measurements

The Wilcoxon's signed-rank test was performed on the post-treatment data and follow-up data to confirm that EEG, the insomnia scale scores, and sleep data were maintained after treatment. In the neurofeedback group, beta waves increased and alpha waves decreased, resulting in an increase in hyperactivity (Fig. 3, Supplementary Table 3). The CBT-I group showed a small increase in beta waves (Fig. 4, Supplementary Table 4). No changes were found in the other variables.

Between-Group Comparison

According to the Mann–Whitney test, EEG after treatment was significantly different between the two groups (Table 5).

Table 3 Wilcoxon's signed-ranks test of sleep scales pre-post changes

Group	Pre-mean(SD)	Post-mean(SD)	Z	P
ISI				
NF	18.67(2.73)	9.79(5.05)	− 2.677	.007**
CBT-I	17.63(2.72)	5.75(5.2)	− 2.533	.011*
PSQI				
NF	12.11(3.79)	8.67(1.94)	− 2.533	.011*
CBT-I	10(2.88)	5.5(3.85)	− 2.530	.011*
DBAS 16				
NF	98(31.24)	85.33(22.99)	− 1.364	.173
CBT-I	85.88(20.12)	37.36(13.88)	− 2.524	.012*
ESS				
NF	10.22(4.21)	8.33(3.97)	− 1.071	.284
CBT-I	7.75(4.65)	5.63(4.37)	− 1.268	.205
PSAS				
NF	53.89(10.2)	44.11(9.88)	− 2.527	.012*
CBT-I	57.88(11.89)	34.38(14.37)	− 2.521	.012*
GSES				
NF	8.22(3.77)	5.89(2.98)	− 2.687	.007**
CBT-I	7.63(1.3)	3.5(2.33)	− 2.383	.017*

ISI insomnia severity index, *PSQI* Pittsburgh sleep quality index, *DBAS 16* dysfunctional beliefs and attitudes about sleep scale, *ESS* Epworth sleepiness scale, *PSAS* pre sleep arousal scale, *GSES* Glasgow sleep effort scale

Table 4 Wilcoxon's signed-ranks test of sleep data pre-post changes

Group	Pre-mean(SD)	Post-mean(SD)	Z	P
TST				
NF	387(77.97)	459.28(95.48)	− 2.666	.008**
CBT-I	368.91(86.32)	419.29(47.58)	− 1.682	.092
TIB				
NF	456.79(81.38)	493.49(116.12)	− 1.599	.110
CBT-I	503.69(53.18)	453.67(25.13)	− 2.103	.035*
SE				
NF	84.07(6.55)	92.46(3.57)	− 2.547	.011*
CBT-I	76.9(12.63)	92.42(9.29)	− 2.521	.012*
SL				
NF	43.22(20.58)	25.89(16.4)	− 2.057	.038*
CBT-I	56.04(34.41)	31.13(21.74)	− 1.262	.207
WASO				
NF	12.78(15.36)	7.22(7.63)	− .845	.398
CBT-I	17.04(17.46)	3.25(6.82)	− 2.103	.035*
Sleep satisfaction				
NF	4.92(1.27)	6.5(1.04)	− 2.549	.011*
CBT-I	5(1.58)	6.46(.46)	− 2.521	.012*

TST total sleep time, *TIB* time in bed, *SE* sleep efficacy, *SL* sleep latency, *WASO* wake after sleep onset

The insomnia scale PSQI and DBAS 16 scores were significantly lower in the CBT-I group compared to the neurofeedback group (Table 6).

Discussion

The purpose of this study was to investigate the effects of neurofeedback training, which controls cortical hyperarousal, the main physiological mechanism of insomnia. Participants who complained of insomnia were recruited and assigned to the neurofeedback group and the CBT-I group for insomnia treatment. Both groups showed a decrease in EEG high-frequency bands and an increase in EEG low-frequency bands after the intervention. This means reduced arousal and increased relaxation occurred at the same time. In addition, both groups showed alleviated insomnia symptoms, decreased physical and cognitive awakening, and increased overall satisfaction with sleep. The total sleep time increased by an average of 72.28 min in the neurofeedback group, while time spent in bed and awakening after sleep onset decreased in the CBT-I group.

The results of this study are described in more detail as follows. First, in physiological indicators, beta waves, indicating hyperarousal, decreased, and theta and alpha waves, indicating relaxation, increased. Previous studies (Corsi-Cabrera et al., 2012; Wolynczyk-Gmaj & Szelenberger, 2011) found increased high-frequency band power in the left frontal, frontal center, and occipital regions in the brainwaves measured during awakening. The high-frequency beta waves in F3, C3, P3, C4, P4, F8, and T4 decreased and the theta waves in T6 and Pz and the alpha waves in Fp1, F3, C3, P3, T3, T5, Fz, C4, P4, T6, and Cz increased after neurofeedback training, which showed that this was an effective intervention for reducing hyperarousal. High-frequency waves decreased in C4, 02, and T6 and low-frequency waves increased in Cz and P4 in the CBT-I treatment group, consistent with a previous study (Cervena et al., 2004) that reported that slow-wave activity increased and beta activity decreased in polysomnography (PSG) measurements made after therapeutic CBT-I intervention. Both treatment groups were effective in reducing cortical hyperarousal, but the neurofeedback group showed more changes than the CBT-I group. This is thought to be because neurofeedback training directly affected cortical hyperarousal. The heart rate did not change significantly in either group. Conflicting results were reported in studies on insomnia patients, showing a higher heart rate than normal sleepers (Varkevisser, Van Dongen, & Kerkhof, 2005) and no differences compared to normal sleepers (Fang et al., 2008; Spiegelhalder et al., 2011; Varkevisser et al., 2005). Further studies are needed on the heart rate of patients with insomnia.

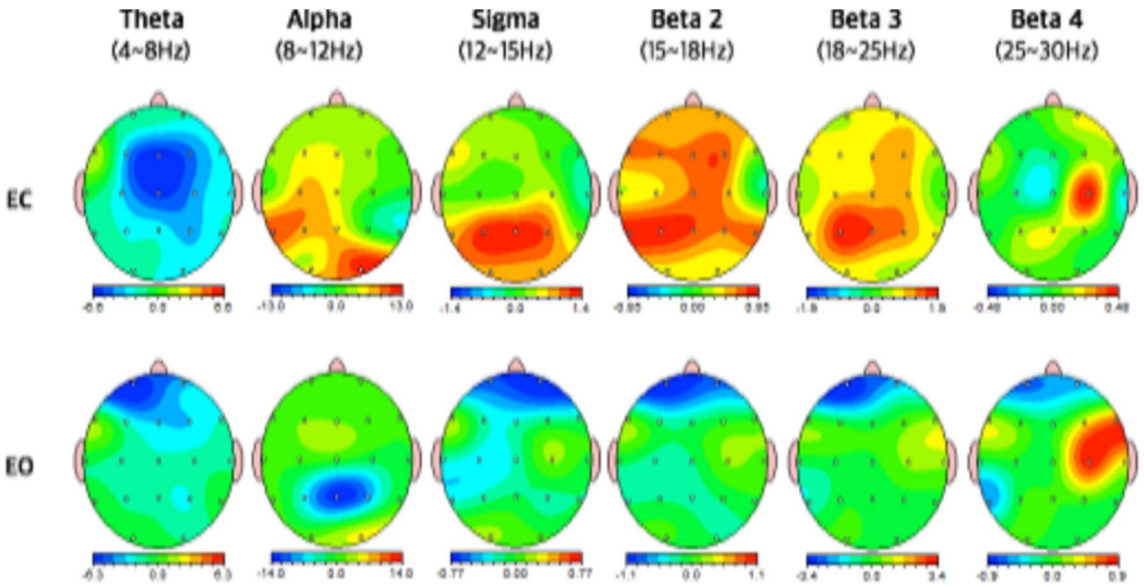

Fig. 3 Topographical maps of post-to-follow up EEG change of neurofeedback group. *EC* Eye closed, *EO* eye open

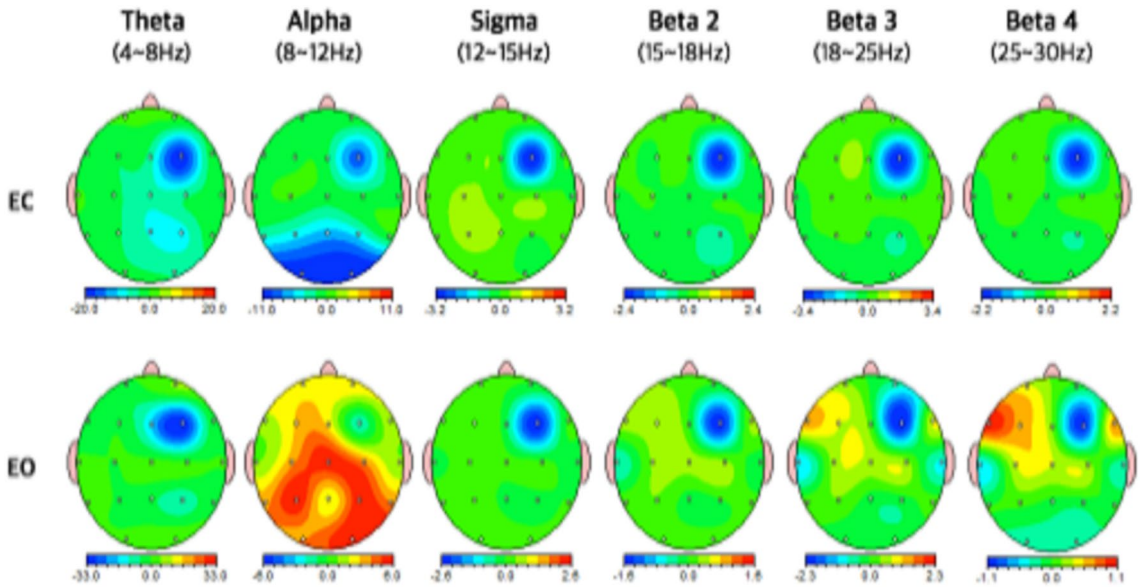

Fig. 4 Topographical maps of post-to-follow up EEG change of CBT-I group. *EC* eye closed; *EO* eye open

Table 5 Mann–Whitney test of EEG after treatment

Location	Frequency	EC/EO	Z	P	
T6	Theta	EC	− 2.023	.043*	NF > CBT
T6	Beta 2	EC	− 2.698	.007*	NF > CBT
T5	Beta 3	EC	− 2.023	.043*	NF > CBT
T3	Theta	EO	− 2.023	.043*	NF < CBT
C4	Beta 3	EO	− 2.023	.043*	NF < CBT

A sign of inequality is difference about the absolute power of EEG between two groups

Next, the overall insomnia symptoms were alleviated in both groups. The severity of insomnia and the quality of sleep and sleep efficacy were improved compared to before treatment. There was a significant decrease in ISI and PSQI scores after treatment as a measure of the severity of insomnia. The post-treatment neurofeedback group had an ISI score of 9.8 and a PSQI score of 8.7, and the CBT-I group had an ISI score of 5.8 and a PSQI score of 5.5. This was a similar range of scores as the post-treatment scores in previous studies on neurofeedback and CBT-I. In a previous study

Table 6 Mann–Whitney test of sleep scales

	NF Mean (SD)	CBT-I Mean (SD)	Z	P	
ISI	9.78(5.04)	5.75(5.2)	− 1.788	.074	
PSQI	8.67(1.94)	5.5(3.84)	− 2.331	.02*	NF < CBT
DBAS 16	85.33(22.99)	37.38(13.88)	− 3.226	.001**	NF < CBT
ESS	44.11(9.88)	34.38(14.37)	− 1.529	.111	
PSAS	5.89(2.98)	3.5(2.33)	− 1.742	.082	

A sign of inequality is difference about mean score between two groups

(Hammer et al., 2011) that conducted neurofeedback for the treatment of insomnia disorder, the after-treatment ISI was 6.56 and the PSQI was 4.5, and 8.4 and 8.6, respectively, in another study. In addition, after treatment with CBT-I, the ISI decreased to 12.5 (Falk & Hagesund, 2016) and the PSQI score was 7.9 (Falk & Hagesund., 2016). These results suggest that both treatments were effective in improving insomnia symptoms. Significant effects were also found in the participants' sleep patterns, with both groups showing higher sleep efficacy at 92.46% (neurofeedback) and 92.42% (CBT-I) after treatment, with the total sleep time ranging from 387 to 459 min in the neurofeedback group. This was more than a previous study in which 447 min of sleep and 89.7% sleep efficiency were seen after beta wave neurofeedback treatment (Jeon and Choi, 2017) and SMR neurofeedback increased the average sleep time after treatment by 60 min (Hammer et al., 2011).

Neurofeedback training could be considered an easy and effective treatment for insomnia. It relieves insomnia symptoms by inducing relaxation by directly affecting cortical hyperactivity, without changing the patients' dysfunctional beliefs about sleep, which are difficult to modify. Faster sleep onset and an increase in total sleep time reflected effective reductions in arousal resulting by neurofeedback training (Perlis et al., 1997). It is expected to be more effective when applied to insomnia patients whose cortical arousal is prominent because relaxation has been induced in many other areas besides the training regions. The results of this study showed that CBT-I was effective in improving overall insomnia symptoms by correcting distorted beliefs about sleep. The time in bed and waking after sleep onset were reduced, and thought to be the result of behavioral modification, which was part of the treatment. CBT-I can be expected to be more effective when applied to insomnia patients whose distorted beliefs related to sleep are strong or whose in affective sleeping habits are prominent.

Clinical Implications

The implications of this study are as follows. First, the hyperarousal neurofeedback treatment showed significant effects in treating insomnia after 10 short training sessions. Neurofeedback treatment for insomnia was conducted using 28 sessions of Z score-based SMR neurofeedback (Hammer et al., 2011), 15 sessions of individualized neurofeedback (Hammer et al., 2011), and 20 sessions of SMR neurofeedback (Basiri et al., 2017). Although short sessions, the neurofeedback in this significantly reduced cortical hyperarousal in a variety of areas, including the frontal central and occipital areas, as well as the left frontal area (F3 and F7), which was the training area. In addition, the decreased PSAS and SL results are thought to be effective in reducing conditioned arousal, which is pre-sleep cognitive and physical arousal. The severity of insomnia and quality of sleep were effectively changed. Therefore, neurofeedback treatment can be a rapid therapeutic intervention with the same effect as existing treatment from only a short training session.

Second, a new non-pharmacological therapeutic option that is relatively easy to implement and can increase treatment compliance was provided. This study identified that neurofeedback was not inferior in therapeutic effects to CBT-I, which is considered the gold standard for the non-pharmacological treatment of insomnia. Since CBT fundamentally aims to alleviate symptoms by modifying dysfunctional beliefs, CBT-I utilizes a variety of cognitive and behavioral techniques to correct the dysfunctional beliefs about sleep. This results in some limitations, that it should target patients with sufficient cognitive capacity. Patient compliance is important since various tasks are given and is a more effortful treatment from the patient's point of view (Agnew et al., 2021). In contrast, neurofeedback has been effective enough without behavioral interventions that prevent the use of cell phones on the bed or irrational beliefs about sleep. This is because neurofeedback adopts a more behavioral approach, which can target the unintentional or unconscious modifications of specific physiological functions simply by providing the selective reinforcement of physiological activities (Yucha & Montgomery, 2008).

This brings us to the third clinical implication of this study. Neurofeedback was demonstrated to treat insomnia by correcting physiological hyperarousal without intervening in the dysfunctional beliefs of insomnia patients. In accordance with drug treatment therapeutic mechanisms, which reduce physiological hyperarousal (Morin et al., 2001),

neurofeedback to decrease beta waves, which improves insomnia symptoms by lowering hyperarousal, was similar to drug treatment that modulates excitatory neuronal activity. Therefore, neurofeedback to decrease beta waves is expected to be a safe treatment without side effects and an alternative new non-pharmaceutical treatment for patients.

Limitations

The limitations of this study are as follows. First, due to the small number of samples, it is difficult to generalize the results of this study to all patients with insomnia. The process of selecting participants who only complained of pure insomnia without co-morbid disorders was not easy because insomnia is associated with many other mental and physical disorders and other sleep disorders, especially OSA. In this study, 22% of the participants were excluded due to other disorders and 9% were classified as having OSA. In addition, 9% of the participants taking a drug were excluded because drugs can affect the EEG. As 40% of the participants who wished to participate in this study were excluded for this reason, it was not easy to recruit those with pure insomnia. However, this study is thought to be of significance because only people with pure insomnia were recruited and the effectiveness of this safe and alternative new non-drug treatment was verified, expanding the options for insomnia disorder treatment. Second, the follow-up period was short. In this study, after the post-intervention measurements, follow-up measurements were made to ensure that the treatment effect was maintained. However, a two-week period is a relatively short time to confirm that a therapeutic effect is maintained. In both treatments, the outcomes were preserved, but the beta waves were observed to increase after the end of treatment. This suggests the possibility that 10 sessions may not be sufficient to prevent relapse. Therefore, it is necessary to have longer and other follow-up periods. Third, we measured a relatively large number of dependent variables for a relatively small sample size. This was useful for examining the various effects of the novel non-pharmacological treatment protocol on sleep but had a limitation in that it was difficult to perform strict statistical corrections. Although some researchers do not recommend statistical corrections in early exploratory studies as it is important to reduce the likelihood of a type II error that fails to detect a phenomenon that is evidently present in nature (Rothman, 1990), future studies will require a study design that takes these limitations into account.

Conclusion

This study demonstrated that the novel neurofeedback protocol proposed in this study was effective for insomnia disorder through 10 short training sessions and was not inferior in therapeutic effects to CBT-I, which is considered the gold standard for the non-pharmacological treatment of insomnia. It is necessary to increase the number of samples and determine how well the treatment effect is maintained through a longer follow-up period in future studies. Also, a future important task will be to examine the treatment effect of drugs, CBT-I, and neurofeedback treatment in a variety of insomnia symptoms.

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Author Contribution SY designed the study, conducted the literature review, treatment, and statistical analysis, and wrote the manuscript. YK wrote, revised, and checked the entire manuscript. SS reviewed the related articles and checked the entire manuscript. SC designed the study, supervised the overall course of treatment, and checked the entire manuscript. All authors reviewed and approved the final version of the manuscript.

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Data Availability Not applicable.

Code Availability Not applicable.

Declarations

Conflict of interest Not applicable.

Ethical Approval This study was conducted with the approval of the Institutional Ethics Committee of Duksung Women's University (No. 2017-09-02-1).

Informed Consent This study was conducted with the written consent of all participants.

Consent for Publication This study obtained written consent from all participants stating that the results will be published without the disclosure of personally identifiable information.

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